

# Roads and Maritime Services

# **F6 Extension Stage 1** New M5 Motorway at Arncliffe to President Avenue at Kogarah

# **Environmental Impact Statement**

Appendix F Human Health Technical Report



(blank page)

# Contents

Glos	sary of	terms and	abbreviation	v				
Exec	cutive S	ummary		x				
1	Introduction							
	1.1	Overview of the project						
	1.2	Project location						
	1.3	Purpose	e of this report	1-1				
	1.4	SEARs	and Agency comments	1-2				
2	The P	roject		2-1				
	2.1	Project features						
	2.2	Constru	uction	2-3				
		2.2.1	Construction activities	2-3				
		2.2.2	Construction boundary	2-3				
		2.2.3	Construction program	2-5				
3	Asses	sment me	ethodology	3-1				
	3.1	What is	a risk or impact assessment?	3-1				
		3.1.1	Risk	3-1				
		3.1.2	Defining risk and impacts	3-1				
	3.2	Overall	approach	3-1				
		3.2.1	General	3-1				
		3.2.2	Data evaluation and issue identification	3-2				
		3.2.3	Exposure assessment	3-2				
		3.2.4	Hazard assessment	3-3				
		3.2.5	Risk characterisation	3-3				
		3.2.6	Features of the risk assessment	3-3				
	3.3	Incorpo	ration of health issues into the project design	3-4				
4	Comr	nunity pro	file	4-1				
	4.1	Genera	ﺎ	4-1				
	4.2	Surrour	nding area and population	4-2				
	4.3	Sensitiv	/e receptors	4-2				
	4.5	Populat	tion profile	4-5				
	4.8	Existing	g health of the population	4-8				
		4.8.1	General	4-8				
		4.8.2	Health related behaviours	4-8				
		4.8.3	Health indicators	4-10				
5	Comr	nunity cor	ncerns	5-1				
6	Asses	sment of	changes in air quality on community health	6-1				
	6.1	Genera	ﺎ	6-1				
	6.2	Existing air quality6						
	6.3	6.3 Overview of air quality impact assessment						

		6.3.1	Construction	6-3
		6.3.2	Operation	6-6
	6.4	Assessn	nent scenarios	6-9
		6.4.1	Overview	6-9
		6.4.2	Assessment scenarios evaluated in the health risk assessment.	6-10
	6.5	Vehicle	emissions	6-10
	6.6	Assessn	nent of volatile organic compounds and polycyclic aromatic hydro	carbons6-10
		6.6.1	General	6-10
		6.6.2	Volatile organic compounds	6-11
		6.6.3	Polycyclic aromatic hydrocarbons	6-11
		6.6.4	Assessment of health impacts	6-13
	6.7	Assessn	nent of carbon monoxide	6-21
	6.8	Assessn	nent of nitrogen dioxide	6-22
		6.8.1	Approach	6-22
		6.8.2	Assessment of total exposures	6-23
		6.8.3	Assessment of incremental exposures	6-24
	6.9	Assessn	nent of particulate matter	6-29
		6.9.1	Particle size	6-29
		6.9.2	Health effects	6-31
		6.9.3	Approach to the assessment of particulate exposures	6-32
		6.9.4	Assessment of total exposures	6-33
		6.9.5	Changes in air quality associated with project	6-35
	6.10	Assessn	nent of regulatory worst-case scenario	6-49
	6.11	Sensitiv	ity analysis	6-51
	6.12	Valuing	particulate impacts	6-52
7	Assess	sment of i	n-tunnel air quality	7-1
	7.1	General		7-1
	7.2	Carbon	monoxide	7-3
	7.3	Nitroger	n dioxide	7-4
		7.3.1	Health effects of short-duration exposures to nitrogen dioxide	7-5
		7.3.2	Further consideration of potential exposures within tunnels	7-6
	7.4	Particula	ate matter	7-10
		7.4.1	Review of short duration exposure to particles	7-11
	7.5	Carbon	dioxide issues	7-12
	7.6	Overall	assessment	7-12
8	Assess	sment of a	changes in noise and vibration impacts on community health	8-1
	8.1	General		8-1
	8.2	Existing	noise environment	8-1
		8.2.1	General	8-1
		8.2.2	Ambient noise monitoring	8-1
		8.2.3	Background noise levels	8-1

	8.3	Noise as	ssessment criteria	8-2
		8.3.1	General	8-2
		8.3.2	Construction noise criteria	8-2
		8.3.3	Ground-borne noise criteria	8-3
		8.3.4	Vibration criteria	8-3
		8.3.5	Operational noise criteria	8-4
	8.4	Overvie	w of noise and vibration assessment	8-5
		8.4.1	Construction impacts	8-5
		8.4.2	Operational impacts	8-6
	8.5	Health c	outcomes relevant to noise	8-7
		8.5.1	General	8-7
		8.5.2	Health impacts from traffic noise	8-9
	8.7	Assessn	nent of noise impacts from project	8-13
		8.7.1	Construction noise	8-14
		8.7.2	Operational noise	8-14
9	Public	safety an	d contamination	9-1
	9.1	General		9-1
	9.2	Public s	afety	9-1
		9.2.1	Construction	9-1
		9.2.2	Operation	9-3
	9.3	Contam	ination	9-4
		9.3.1	Construction	9-5
		9.3.2	Operation	9-6
10	Assess	ment of c	changes in social aspects on community health	10-1
	10.1	General		10-1
	10.2	Change	s in traffic	10-1
		10.2.1	Construction	10-1
		10.2.2	Operations	10-2
		10.2.3	Public transport	10-2
		10.2.4	Pedestrian and cycle access	10-2
		10.2.5	Impacts on health and emergency services	10-3
	10.3	Property	/ acquisitions	10-3
	10.4	Green s	pace	10-4
	10.5	Change	s in community access and connectivity	10-5
	10.6	Visual c	hanges	10-5
	10.7	Equity		10-6
	10.8	Constru	ction fatigue	10-6
	10.9	Econom	ic aspects	10-8
		10.9.1	Road tolling	10-8
	10.10	Stress a	nd anxiety issues	10-8
	10.11	Overall	assessment	10-10

11	Uncerta	inties		11-1					
	11.1	General		11-1					
	11.2	Populatio	on health data	11-1					
	11.3	e concentrations and levels	11-1						
		11.3.1	Traffic modelling	11-1					
		11.3.2	Air quality	11-1					
		11.3.3	Noise assessment	11-2					
	11.4	Approach	n to the assessment of risk for particulates	11-2					
		11.4.1	General	11-2					
		11.4.2	Exposure-response functions	11-2					
		11.4.3	Shape of exposure-response function	11-6					
	11.5	Diesel pa	articulate matter evaluation	11-6					
	11.6	Co-pollut	tants	11-6					
	11.7	Selected	health outcomes	11-7					
	11.8	Exposure	e time/duration	11-7					
	11.9	Changing	g population size and demographics	11-7					
	11.10	Application	on of exposure-response functions to small populations	11-7					
	11.11	Overall e	valuation of uncertainty	11-8					
12	Referer	nces		12-1					
Annex	kure A –	Approach	n to Risk Assessment using exposure response relationships	A					
Annex	kure B –	Approach	n to assessment of cancer risk	B					
Annex	kure C –	Acceptab	ble risk levels	C					
Annex	kure D –	Risk calc	ulations: Nitrogen dioxide	D					
Annex	kure E –	Populatio	on incidence calculations: Nitrogen dioxide	E					
Annex	kure F –	Risk calc	ulations: Particulate matter	F					
Annex	Annexure G – Population incidence calculations: Particulate matterG								
Annex	kure H –	Risk calc	culations: Particulate matter exposures for elevated receptors	H					
Annex	kure I – I	Noise cato	chment areas	I					

# **Glossary of terms and abbreviation**

Term	Definition
ABL	Assessment background noise level
ABS	Australian Bureau of Statistics
ACTAQ	NSW Government Advisory Committee on Tunnel Air Quality
Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days)
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems
ATSDR	Agency for Toxic Substances and Disease Register
AAQ	Ambient air quality
ANZECC	Australia and New Zealand Environment and Conservation Council
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
BaP	Benzo(a)pyrene
Biodegradation	Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).
Body burden	The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.
BTX	Benzene, toluene and total xylenes
Carcinogen	A substance that causes cancer.
CASA	Civil Aviation Safety Authority
CBD	Central business district
CCME	Canadian Council of Ministers of the Environment
CCTV	Closed Circuit Television
CEMP	Construction Environmental Management Plan
CHD	Coronary heart disease
Chronic exposure	Contact with a substance or stressor that occurs over a long time (more than one year) [compare with acute exposure and intermediate duration exposure].
СО	Carbon monoxide
COPD	Chronic Obstructive Pulmonary Disease
CPI	Consumer Price Index
CNVG	Construction Noise and Vibration Guideline (Roads and Maritime, 2016)
СТАМР	Construction Traffic Management and Access Plan
dB(A)	Decibels (A-weighted)
DE	Diesel exhaust
DECCW	NSW Department of Environment, Climate Change and Water
DEFRA	Department for Environment, Food & Rural Affairs
DEH	Australian Department of Environment and Heritage
Detection limit	The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Term	Definition
DIRDC	Department of Infrastructure, Regional Development and Cities
Do Minimum	Air quality, noise and traffic modelling scenario with the full WestConnex (Stages 1, 2 and 3) King St Gateway and Sydney Gateway are complete but the project, Western Harbour Tunnel and Beaches Link are not built
Do Something	Air quality, noise and traffic modelling scenario with the full WestConnex (Stages 1, 2 and 3) King St Gateway, Sydney Gateway and with the project but without the Beaches Link and Western Harbour Tunnel
Do Something - cumulative	Air quality, noise and traffic modelling scenario with the full WestConnex (Stages 1, 2 and 3), the project, the Beaches Link and Gore Hill Freeway Connection, King St Gateway, Sydney Gateway and the Western Harbour Tunnel
Dose	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An 'exposure dose' is how much of a substance is encountered in the environment. An 'absorbed dose' is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
DPM	Diesel particulate matter
DSI	Detailed site investigation
EC	European Commission
ED	Emergency department
EIS	Environmental Impact Statement
EP&A Act	Environmental Planning and Assessment Act 1979 (NSW)
EU	European Union
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Also includes contact with a stressor such as noise or vibration. Exposure may be short term [acute exposure], of intermediate duration, or long term [chronic exposure].
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.
Exposure pathway	The route a substance takes from its source (where it began) to its endpoint (where it ends), and how people can come into contact with (or get exposed) to it. An exposure pathway has five parts: a source of contamination (such as chemical leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.
Genotoxic carcinogen	These are carcinogens that have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement). Where this occurs, the damage may be sufficient to result in the initiation of cancer at some time during a lifetime.
GRAL	Graz Lagrangian Model
GRAMM	GRAZ Mesoscale Model
GSP	NSW State Gross Product

Term	Definition
Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the NSW Department of Environment and Conservation (DEC) or institutions such as the National Health and Medical Research Council (NHMRC), Australia and New Zealand Environment and Conservation Council (ANZECC) and World Health Organization (WHO)), that is used to identify conditions below which no adverse effects, nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines would have different names, such as investigation level, trigger value and ambient guideline.
HHRA	Human health risk assessment
HI	Hazard Index
IARC	International Agency for Research on Cancer
ICNG	Interim Construction Noise Guideline (NSW DECC 2009)
IHD	Ischaemic heart disease
Inhalation	The act of breathing. A hazardous substance can enter the body this way [see route of exposure].
Intermediate exposure Duration	Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].
LA1	A-weighted sound level exceeded for 1% of the measurement period
LA10	A-weighted sound level exceeded for 10% of the measurement period
LA90	A-weighted sound level exceeded for 90% of the measurement period
LAeq	A-weighted equivalent sound level
LAmax	A-Weighted, maximum sound level
LGA	Local Government Area
LOAEL	Lowest observed adverse effect level – The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.
LOR	Limit of Reporting
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
NCAs	Noise catchment areas
NCG	Noise Criteria Guideline (various, as referenced in the report)
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NMG	Noise Mitigation Guideline (various, as referenced in the report)
NML	Noise management level
NPfl	NSW Noise Policy for Industry
NO2	Nitrogen dioxide
NOx	Nitrogen oxides
NOAEL	No-observed-adverse-effect-level – The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.
NSW	New South Wales

Term	Definition					
NSW EPA	NSW Environment Protection Authority					
OEH	NSW Office of Environment and Heritage					
ОЕННА	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA)					
OLS	Obstacle limitation surface					
РАН	Polycyclic aromatic hydrocarbon					
PANS-OPS	Procedures for air navigation systems operations					
PIARC	Name of the World Road Association					
PM	Particulate matter					
PM1	Particulate matter below one micron in diameter, often termed very fine particles					
PM2.5	Particulate matter of aerodynamic diameter 2.5 µm and less					
PM10	Particulate matter of aerodynamic diameter 10 $\mu m$ and less					
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].					
Population	A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).					
ррbv	Parts per billion by volume					
ppm	Parts per million					
RAP	Remedial action plan					
RBL	Rating background level					
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].					
Risk	The probability that something would cause injury or harm.					
RNP	Road Noise Policy					
Roads and Maritime	NSW Roads and Maritime Services					
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].					
RWR	Residential, worker and recreational receptors					
SA	Statistical area					
SEIFA	Socio-Economic Index for Areas					
SO2	Sulfur dioxide					
Т90	Distillation temperature where 90% of the fuel is evaporated					
TCEQ	Texas Commission on Environmental Quality					
TEQ	Toxicity equivalent					
Toxicity	The degree of danger posed by a substance to human, animal or plant life.					
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on based on available toxicity studies relevant to humans and/or animals and relevant safety factors.					
Toxicological profile	An assessment that examines, summarises, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.					

Term	Definition
Toxicology	The study of the harmful effects of substances on humans or animals.
TSP	Total suspended particulates
Uncertainty factor	Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure would cause harm to people [also sometimes called a safety factor].
ultrafines	Particulate matter below 0.1 microns in diameter
UK	United Kingdom
US	United States
USEPA	United States Environmental Protection Agency
VDV	Vibration dose values
VOC	Volatile organic compound
WHO	World Health Organization
WRTM	WestConnex Road Traffic Model
β coefficient	Beta coefficient
µg/m3	Micrograms per cubic metre

# **Executive Summary**

# The project

Approval is being sought under Part 5, Division 5.2 of the EP&A Act for a new multi-lane road between the New M5 Motorway at Arncliffe and President Avenue at Kogarah (F6 Extension – Stage 1 (the project)). The project would connect underground with the New M5 Motorway tunnel and to a new surface level intersection at President Avenue, Kogarah.

# The purpose of this report

The purpose of this report is to support the environmental impact statement for the project. This report presents a Health Impact Assessment (HIA) associated with impacts identified in relation to air quality, noise and vibration and social aspects, to address the Secretary's Environmental Assessment Requirements (SEARs).

# Method

A Health Impact Assessment is a way of deciding now, what the consequences to health (both positive and negative) of some future action (such as this project) may be. It draws on previous experience about impacts from road tunnels and their potential effects on people who live or work around them. It uses this information to predict the impacts of the project on community health.

In this case, this report includes a detailed review of what impacts may occur, who may be exposed to these impacts and whether there is potential for these impacts to result in adverse health effects or positive benefits within the local community. The Health Impact Assessment presented in this report has been conducted in accordance with national guidance (enHealth 2001, 2012b; Harris 2007), which has involved the following:

- Review of predicted impacts to air quality, noise and vibration during construction and operation of the project. In some cases, the issues identified, such as those during construction, are short-term and can be mitigated/managed through the implementation of specific management measures. For other impacts, such as those from operations or for extended periods of construction from a number of projects, the impacts may occur over a longer period of time and require a more detailed assessment of how these impacts affect health
- Identification and characterisation of the community (including the presence of sensitive receptors such as childcare centres, aged care centres, schools and hospitals) who may be affected by these impacts
- Assessment of air quality impacts on health including:
  - Reviewing the key air pollutants (associated with vehicle emissions) that are predicted from the operation of the project (within the tunnel and outside the tunnel)
  - Identifying guidelines that are based on protection of the health of all members of the population for exposure to these pollutants over a short period of time as well as all day, every day
  - Comparing the predicted impacts with the health based guidelines
  - Undertaking a more detailed assessment of potential risks of changes in nitrogen dioxide and particulates, including fine particulate matter or PM2.5 (particulate matter of aerodynamic diameter 2.5 microns (µm) and less) and coarse particulate matter or PM10 (particulate matter of aerodynamic diameter 10 µm and less). The assessment has addressed specific health effects (or health endpoints) associated with exposures to these pollutants. The assessment conducted has evaluated the impact of the project on these health endpoints within the local community
  - Assessment of the potential for health issues for users of the tunnel, as well as users of the wider tunnel network
  - Valuing/costing the impacts on health relevant to particulate matter based on the NSW Environment Protection Authority (NSW EPA) methodology.

- Assessment of noise and vibration impacts on health including:
  - Reviewing the impacts that are predicted from the construction and operation of the project
  - Identifying guidelines that are based on the protection of the health and wellbeing (including sleep disturbance) during all phases of the project, both construction and operation
  - Comparing predicted impacts with the health based guidelines. Where the health based guidelines cannot be met, consideration of the implementation of mitigation/management measures
- Assessment of public safety and contamination

This has involved a qualitative assessment, providing an overview of the potential hazards that may affect public safety during construction and operation, including contamination. This review has considered the implementation of mitigation/management measures and whether these can minimise risks to the community

• Assessment of social changes on health associated with the project:

This has involved a qualitative assessment. Aspects of the project that have the potential to result in impacts or changes in the community (including traffic, pedestrian and cycle access, property acquisitions and access, visual changes, community access/cohesion and economic impacts) have been evaluated with respect to potential effects on health and well-being. In addition, the equity of changes associated with the project has also been evaluated within the community.

An assessment of construction fatigue, related to community exposure to a number of concurrent construction projects, has also been undertaken.

# Conclusions

### Air Quality

In relation to air quality impacts the following conclusion are made:

- Impacts associated with dust generated from construction activities require management to
  ensure impacts to community health are minimised. Measures required to be implemented to
  minimise dust impacts are to be detailed in a Dust Management Plan, forming part of the
  Construction Air Quality Management Plan, as detailed in the Air quality technical report (ERM,
  2018)
- Impacts in the community outside the tunnel: the project is expected to result in an overall
  decrease in total pollutant levels in the community. The project is expected to result in a
  redistribution of impacts associated with vehicle emissions, specifically in relation to emissions
  derived from vehicles using surface roads. For much of the community this would result in no
  change or a small improvement (ie decreased concentrations and health impacts), however for
  some areas located near key surface roads, a small increase in pollutant concentration may
  occur. Potential health impacts associated with changes in air quality (specifically nitrogen dioxide
  and particulates) within the local community have been assessed and are considered to be
  tolerable / acceptable
- For the project, future development of land (including re-zonings) in the vicinity of the ventilation facilities require planning controls to be developed to ensure future developments at heights above 30 metres are not adversely impacted by the ventilation outlets. Development of planning controls would be supported by detailed modelling addressing all relevant pollutants and averaging periods
- Impacts within the tunnel: while concentrations of pollutants from vehicle emissions are higher within the tunnel (compared with outside the tunnel), and with the completion of a number of tunnel projects (approved or proposed) there is the potential for exposures to occur within a network of tunnels over varying periods of time, depending on the journey. The assessment of potential exposures inside these tunnels, has indicated:
  - Where windows are up and ventilation is on recirculation, exposures to nitrogen dioxide inside vehicles is expected to be below the current health based guidelines. In congested conditions inside the tunnels, it is not considered likely that significant adverse health effects would occur. Placing ventilation on recirculation is also expected to minimise exposures to particulates during travel through the tunnels

For motorcyclists, where there is no opportunity to minimise exposure through the use of ventilation, there is the potential for higher levels of exposure to nitrogen dioxide. These exposures, under normal conditions, are not expected to result in adverse health effects. When the tunnels are congested it is expected that motorcyclists would spend less time in the tunnels than passenger vehicles and trucks due to lane filtering, limiting the duration of exposure and the potential for adverse health effects.

# **Noise and Vibration**

In relation to noise and vibration the following conclusions are made:

### Construction

A number of receptors have been identified as highly affected from standard and out of hours construction noise, especially around the Rockdale Construction Ancillary Facility, Cut and Cover Construction and President Avenue, Princes Highway Intersection works and along the powerline installation route. These noise impacts are predicted to be of a significant volume to cause sleep disturbances. Health effects from these noise impacts are likely without the intervention of mitigation measures. The detailed design for the mitigation measures will be outlined in the Construction Noise and Vibration Management Plan (CNVMP) and include architectural treatment for those properties that are also identified as being impacted by operational noise. The aim of the mitigation measures should be to reduce noise and vibration to levels that comply with the management goals established in this assessment. If it is not possible to achieve compliance with these goals, health impacts for the affected community are likely.

Construction road traffic noise was estimated to be generally compliant with guideline levels except for roads around the Rockdale construction ancillary facility (especially Wickham Street) during night time periods where increased traffic noise was predicted to be up to 7.3 dB(A) and during the powerline construction works. The impact around the Rockdale construction ancillary facility is considerable and night-time haulage should be avoided during night-time off-peak traffic periods to minimise noise impacts. Powerline construction works also result in considerable impacts and mitigation measures need to be undertaken to reduce noise impacts.

### Operation

The noise assessment predicts that noise criteria will be exceeded at a number of properties adjacent to the project without mitigation measures, with 109 properties considered appropriate for mitigation measures due to operational noise. These properties are primarily along Princes Highway and President Avenue. While 109 properties have been identified as appropriate for mitigation measures, many of these properties currently experience elevated noise levels so mitigation measures may provide a net benefit to those receptors.

Mitigation measures should be applied at the source where possible with in-property architectural treatments for noise only considered when all other options are exhausted. It is noted that in-property architectural treatments are associated with several limitations.

# Public safety and contamination

A review of the potential risks posed to public safety, associated with the project, from issues such as dangerous goods, subsidence, contamination and road safety was undertaken. For both construction and operational aspects of the project no issues were identified that had the potential to result in significant safety risks to the community.

# **Social**

Changes in the urban environment associated with the project have the potential to result in a range of impacts on health and wellbeing of the community. The potential for changes to result in impacts on health and wellbeing is complex. Changes that may occur have the potential to result in both positive and negative impacts. Positive impacts include economic benefits, changes in traffic levels in some areas and increased pedestrian and cycle access. Negative impacts may occur as a result of traffic changes during construction, property acquisitions, visual changes, noise impacts and changes in access/cohesion of local areas. These may result in increased levels of stress and anxiety. In many cases the impacts identified are either short term (associated with construction only) and/or mitigation/management measures have been identified to minimise the impacts on the community.

# 1 Introduction

The project would comprise a new multi-lane road between the New M5 Motorway at Arncliffe and President Avenue at Kogarah. The project would connect underground with the New M5 Motorway tunnel and to a new surface level intersection at President Avenue, Kogarah.

# **1.1 Overview of the project**

Key components of the project would include:

- An underground connection to the existing stub tunnels at the New M5 at Arncliffe
- Twin motorway tunnels (around four kilometres in length) between the New M5 at Arncliffe and President Avenue, Kogarah
- A tunnel portal and entry and exit ramps connecting the tunnels to a surface intersection with President Avenue
- Intersection improvements at the President Avenue / Princes Highway intersection
- Mainline tunnel stubs to allow for connections to future stages of the F6 Extension
- Shared pedestrian and cycle pathways connecting Bestic Street, Rockdale to Civic Avenue, Kogarah via Rockdale Bicentennial Park (including an on-road cycleway)
- An Operational Motorway Control Centre to be located off West Botany Street, Rockdale
- Ancillary infrastructure and operational facilities for signage (including electronic signage), ventilation structures and systems at Rockdale, fire and safety systems, and emergency evacuation and smoke extraction infrastructure
- A permanent power supply connection from the Ausgrid Canterbury subtransmission substation
- Temporary construction ancillary facilities and temporary works to facilitate the construction of the project.

Once complete, the F6 Extension Stage 1 would improve connections and travel times between Sydney and the Princes Highway and enhance connections for residents and businesses within the broader regional area as well as promote and support economic development in areas to the south, such as Sutherland and the Illawarra.

Approval for the project is being sought under Part 5, Division 5.1 of the EP&A Act. Future stages of the F6 Extension would be subject to separate planning applications and assessments would be undertaken accordingly.

The configuration and design of the project will be further developed to take into consideration the outcomes of community and stakeholder engagement.

# **1.2 Project location**

This project would be generally located within the Bayside local government area. The project commences about 8 kilometres south west of the Sydney central business district (CBD). The proposed President Avenue intersection would be located about 11 kilometres south east of the Sydney CBD.

# **1.3 Purpose of this report**

The purpose of this report is to support the environmental impact statement for the project. This report presents a Health Impact Assessment (HIA) associated with impacts identified in relation to air quality, noise and vibration and social aspects, to address the Secretary's Environmental Assessment Requirements (SEARs). The report has been prepared in accordance with the relevant guidelines as outlined in **Section 3.2.1**.

# 1.4 SEARs and Agency comments

# Table 1-1: SEARs – Health Impact Assessment

As	sessme	nt requirements	Where addressed							
Req	uirements	, as per Section 3 of the key issues SEARs (Health and Safety)	Section where addressed in report							
1.	The Properation	ponent must assess the potential health impacts from the construction and n of the project.	Section 6 to 10							
2.	The ass	essment must:								
	а.	describe the current known health status of the potentially affected population	Section 4							
	b.	describe how the design of the proposal minimises adverse health impacts and maximises health benefits	Section 3.3							
	C.	assess human health impacts from the operation and use of the tunnel under a range of conditions, including worst case operating conditions and the potential length of existing and committed future motorway tunnels in Sydney	Section 6 to 8							
	d.	human health risks and costs associated with the construction and operation of the proposal, including those associated with air quality, odours, noise and vibration (including residual noise following application of mitigation measures), construction fatigue and social impacts (including from acquisitions) on the adjacent and surrounding areas, as well as opportunity costs (such as those from social infrastructure and active transport impacts) during the construction and operation of the proposal	Section 6 to 10							
	e.	include both incremental changes in exposure from existing background pollutant levels and the impacts of project specific pollutant levels at the location of the most exposed receivers and other sensitive receptors (including public open space areas, sportsgrounds, child care centres, schools, hospitals and aged care facilities)	Section 6							
	f.	assess the likely risks of the project to public safety, paying particular attention to pedestrian safety, subsidence risks, flood risks and the handling and use of dangerous goods	Section 9							
	g.	assess the opportunities for health improvement	Section 6 to 10							
	h.	assess the distribution of the health risks and benefits	Section 6 to 10							
	i.	include a cumulative human health impact assessment inclusive of in- tunnel users, local and regional impacts due to the operation of and potential continuous travel through existing and committed future motorway tunnels and surface roads	Section 6 to 8							

# 2 The Project

# 2.1 **Project features**

The project would comprise a new multi-lane underground road link between the New M5 Motorway and a surface intersection at President Avenue, Kogarah.

Key components of the project would include:

- Twin mainline tunnels. Each mainline tunnel would be around 2.5 kilometres in length, sized for three lanes of traffic, and line marked for two lanes as part of the project
- A tunnel-to-tunnel connection to the New M5 Motorway southern extension stub tunnels, including line marking of the New M5 Motorway tunnels from St Peters interchange to the New M5 Motorway stub-tunnels
- Entry and exit ramp tunnels about 1.5 kilometres long (making the tunnel four kilometres in length overall) and a tunnel portal connecting the mainline tunnels to the President Avenue intersection
- An intersection with President Avenue including entry and exit ramps and the widening and raising of President Avenue
- Upgrade of the President Avenue / Princes Highway intersection to improve intersection capacity
- Shared cycle and pedestrian pathways connecting Bestic Street, Brighton-Le-Sands to Civic Avenue, Kogarah (including an on-road cycleways)
- Three motorway operation complexes:
  - Arncliffe, including a water treatment plant, substation and fitout (mechanical and electrical) of a ventilation facility currently being constructed as part of the New M5 Motorway project
  - Rockdale (north), including a motorway control centre, deluge tanks, a workshop and an office
  - Rockdale (south), including a ventilation facility, substation and power supply.
- Reinstatement of Bicentennial Park and recreational facilities
- In-tunnel ventilation systems including jet fans and ventilation ducts connecting to the ventilation facilities
- Drainage infrastructure to collect surface water and groundwater inflows for treatment
- Ancillary infrastructure for electronic tolling, traffic control and signage (both static and electronic signage)
- Emergency access and evacuation facilities (including pedestrian and vehicular cross and long passages); and fire and life safety systems
- New service utilities, and modifications and connections to existing service utilities.

The project does not include ongoing motorway maintenance activities during operation or future upgrades to other intersections in the vicinity during operation. These works are permitted under separate existing approvals and are subject to separate assessment and approval in accordance with the EP&A Act.

The key features of the project are shown on Figure 2-1.

#### Appendix F - Human Health Technical Report



# 2.2 Construction

## 2.2.1 Construction activities

The proposed construction activities for the project would include:

- Preparatory investigations
- Site establishment and enabling work
- Tunnelling
- Surface earthworks and structures
- Construction of motorway operations complexes
- Drainage and construction of operational water management infrastructure
- Construction of the permanent power supply connection
- Road pavement works
- Finishing works.

These activities would generally be undertaken within the following construction ancillary facilities:

- Arncliffe construction ancillary facility (C1) at Arncliffe, within the Kogarah Golf Course currently being used for the construction of the New M5 Motorway
- Rockdale construction ancillary facility (C2) at Rockdale, within a Roads and Maritime depot at West Botany Street
- President Avenue construction ancillary facility (C3) at Rockdale, north and south of President Avenue within Rockdale Bicentennial Park and part of Scarborough Park North, and a site west of West Botany Street
- Shared cycle and pedestrian pathways construction ancillary facilities (C4 and C5) at Brighton-le-Sands, within the recreation area between West Botany Street and Francis Avenue, near Muddy Creek
- Princes Highway construction ancillary facility (C6), on the north-east corner of the President Avenue and Princes Highway intersection.

# 2.2.2 Construction boundary

The area required for project construction is referred to as the 'construction boundary'. This comprises the surface construction works area, and construction ancillary facilities (refer to **Figure 2-2**). Utility works to support the project would occur within and outside the construction boundary (refer to **Chapter 7** (Construction) of the EIS).

In addition to these works, the underground construction boundary (including mainline tunnel construction and temporary access tunnels) is also shown on **Figure 2-2**.

#### **C1**

- Tunnelling and spoil handling
- Construction of MOC1 (Water treatment plant, substation)
- Fitout, testing and commissioning of tunnels and MOC 1

#### **C**2

- Construction of the decline tunnel
- Tunnelling and spoil handling
- Pavement works for internal access road
- Construction of MOC2
- Reconfiguration of the site to enable ongoing/future use for maintenance activities

#### **C**3

- Demolition of buildings and vegetation clearing and removal
- · Relocation of utilities
- Temporary stockpiling of spoil and fill materials
- Management of any contaminated land, including acid sulphate soils
- Construction of cut-and-cover structures
- Construction of MOC3 (Rockdale ventilation facility and substation)
- President Avenue intersection
   upgrade works
- Construction of shared pedestrian and cyclist path and overpass

#### C4/C5

- Site establishment
- Vegetation clearing and removal, topsoil stripping areas and landform shaping
- Temporary stockpiling of materialsConstruction of the shared
- Construction of the shared pedestrian and cyclist path
  Finishing works including
- Finishing works including lighting, line marking and signage installation

#### **C**6

- Property adjustment and demolition
- Relocation of utilities, stormwater infrastructure, underground storage tanks and substation
- Laydown and parking of construction vehicles and equipment
- · Reinstatement of site



#### LEGEND

- Surface works
- Construction boundary
- Cut-and-cover structures
- Underground construction
- Construction ancillary facility — Permanent power supply line
  - Permanent power supply line
- Figure 2-2 Construction boundary and construction ancillary facilities

New M5 Tunnel

Road

Waterway

Railway line

T Railway station

Parks and recreation

# 2.2.3 Construction program

The project would be constructed over a period expected to be around four years, including commissioning which would occur concurrently with the final stages of construction (refer to **Figure 2-3**).

The project is expected to be completed towards the end of 2024.

	1	20	20	1	2	021			20	22	1	-	20	23		20	024	
Construction activity	Q1	Q2	Q3 0	4 Q	02	Q3	Q4	Q1	Q2	Q3	Q4	01	Q2	Q3	Q4 Q1	Q2	03	Q4
C1 Arncliffe construction ancillary facility	1	-				-		-				-						
Site establishment	1		(	ж					-									
Tunnelling works and spoil handling	-			C	-	-			_	_			_		0	-		
Construction of Motorway Operations Complex 1 (Surface Buildings)														0	-	-0		
Rehabilitation and landscaping																	0	0
C2 Rockdale construction ancillary facility	1		la al															
Site establishment			(	>	-0													
Tunnelling works and spoil handling				C	-				_				_	_	0	-	1	
Construction of Motorway Operations Complex 2 (Surface Buildings)														0		0		
Rehabilitation and landscaping																	0	-0
C3 President Avenue construction ancillary facil	ity				1													
Site establishment			(	ж														
Excavation and construction of cut-and-cover structure				C	-							0						
Rehabilitation and landscaping												0	_	-0				
Construction of Motorway Operations Complex 3 (Surface Buildings)												0			-0			
Relocation of utilities/services along President Avenue			(	~		-0												
President Avenue widening works						0			-		-	0		-				
Rehabilitation and landscaping											0		0					
Construction of shared cycle and pedestrian bridge									-	0			-	-0				
C4/C5 Shared cycle and pedestrian pathways																		
Site establishment					-	0												
Construction of shared cycle and pedestrian pathways							0					0						
Rehabiltation and landscaping						-							0					
C6 Princes Highway construction ancillary facili	ty																	
Property demolition, rehabilitation and adjustment						0		0										1
Relocation of utilities, stormwater infrastructure and substation								1	0			0						
Pavement works along Princes Highway and President Avenue													0-		-0			
Rehabilitation and landscaping																0		

Figure 2-3: Indicative construction program

# 3 Assessment methodology

# 3.1 What is a risk or impact assessment?

## 3.1.1 Risk

Risk assessment is used extensively in Australia and overseas to assist in decision making on the acceptability of the risks associated with the presence of contaminants or stressors in the environment and assessment of potential risks to the public. Risk is commonly defined as the chance of injury, damage, or loss. Therefore, to put oneself or the environment 'at risk' means to participate, either voluntarily or involuntarily, in an activity or activities that could lead to injury, damage, or loss.

Voluntary risks are those associated with activities that we decide to undertake such as driving a vehicle, riding a motorcycle and smoking cigarettes. Involuntary risks are those associated with activities that may happen to us without our prior consent or forewarning. Acts of nature such as being struck by lightning, fires, floods and tornados, and exposures to environmental contaminants are examples of involuntary risks.

# 3.1.2 Defining risk and impacts

Risks to the public and the environment are determined by direct observation or by applying mathematical models and a series of assumptions to infer risk. No matter how risks are defined or quantified, they are usually expressed as a probability of adverse effects associated with a particular activity. Risk is typically expressed as a likelihood of occurrence and/or consequence (such as negligible, low or significant) or quantified as a fraction of, or relative to, an acceptable risk number.

Risks or impacts from a range of facilities (eg industrial or infrastructure) are usually assessed through qualitative and/or quantitative risk assessment techniques. In general, risk or impact assessments seek to identify all relevant hazards; assess or quantify their likelihood of occurrence and the consequences associated with these events occurring; and provision of an estimate of the risk levels for people who could be exposed, including those beyond the perimeter boundary of a facility. In this report, quantitative risk is assessed in terms of acceptable, tolerable or unacceptable risk. A full explanation of these terms can be found in Annexure C of this report.

# 3.2 Overall approach

# 3.2.1 General

The methodology adopted for the conduct of the HIA is in accordance with national and international guidance that is endorsed/accepted by Australian health and environmental authorities, and includes:

- Harris, P., Harris-Roxas, B., Harris, E. & Kemp, L., Health Impact Assessment: A Practical Guide, Centre for Health Equity Training, Research and Evaluation (CHETRE). Part of the UNSW Research Centre for Primary Health Care and Equity. University of NSW, Sydney (Harris 2007)
- Health Impact Assessment Guidelines. Published by the Environmental Health Committee (enHealth), which is a subcommittee of the Australian Health Protection Committee (AHPC) (enHealth 2001)
- Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazards, 2012 (enHealth 2012b)
- Schedule B8 Guideline on Community Engagement and Risk Communication, National Environment Protection (Assessment of Site Contamination) Measure, 1999 (National Environment Protection Council (NEPC 1999 amended 2013a))
- National Environmental Protection (Air Toxics) Measure, Impact Statement for the National Environment Protection (Air Toxics) Measure, 2003 (NEPC 2003)
- Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment), EPA-540-R-070-002, January 2009 (United States Environment Protection Agency (USEPA 2009a)).

More specifically, in relation to the assessment of health impacts associated with exposure to nitrogen dioxide and particulate matter, guidelines available from the NEPC ((Burgers & Walsh 2002; NEPC 1998, 2002, 2003, 2009, 2010)), World Health Organization (WHO) (Ostro 2004; WHO 2003, 2006b, 2006a, 2013b) and the USEPA (USEPA 2005b, 2009b) have been used as required.

In addition, the following has been considered:

- NSW Health, Building Better Health, Health considerations for urban development and renewal in the Sydney Local Health District (NSW Health 2016)
- NSW Health, Healthy Urban Development Checklist, A guide for health services when commenting on development policies, plans and proposals, 2009
- Methodology for Valuing the Health Impacts of Changes in Particle Emissions (EPA 2013)
- Air Quality in and Around Traffic Tunnels (NHMRC 2008)
- State Environmental Planning Policy (SEPP) 33 Hazardous and Offensive Development

These guidelines have been used to evaluate health impacts associated with the project that relate to:

- Changes in air quality in the tunnels (as presented in section 7)
- Changes in air quality around the tunnels (within the community) during construction and operation (as presented in section 6)
- Changes in the noise environment during construction and operation (as presented in section 8)
- Impacts on public safety (as presented in section 9)
- Changes in the social environment, including an overview of the positive and negative impacts of the project on health (as presented in section 10).

In following this guidance, the following tasks have been completed and are presented in this technical report.

### 3.2.2 Data evaluation and issue identification

This task involves a review of all available information that relates to the proposed design and outcomes from relevant specialist studies undertaken in relation to air quality within the tunnel itself, air quality within the surrounding community, noise and vibration. Specifically, the assessment has considered existing conditions (in relation to air quality and noise) and estimation of short term (acute) and long term (chronic) impacts during construction and operation of the project.

This aspect of the assessment also considers the available guidelines for air quality and noise, whether these guidelines are based on the protection of community health, and if a more detailed evaluation of specific impacts is required. The HIA has considered a more detailed evaluation of exposures to nitrogen dioxide and particulate matter emissions within the surrounding community from the operation of the project. Other pollutants have also been considered that include volatile organic compounds, polycyclic aromatic hydrocarbons and carbon monoxide. In addition, a review of health risk impacts associated with air quality within the tunnel itself has been included.

### 3.2.3 Exposure assessment

This involves the identification of populations located in the project study area (see **section 4**) which may be exposed to impacts from the project. The existing air and noise environments as well as the health of the existing population has been considered in relation to the key health effects (with specific health effects termed health endpoints) consideration in this assessment. The assessment has considered both acute and chronic inhalation exposures relevant to the project.

### 3.2.4 Hazard assessment

The objective of the hazard or toxicity assessment is to identify the adverse health effects and quantitative toxicity values or exposure-response relationships that are associated with the key pollutants and stressors that have been identified and evaluated as part of this assessment. This has been applied to the assessment of exposures to particulate matter where the following steps have been undertaken:

- Identify the adverse health effects associated with exposure to the pollutants or stressors. Based on the available information, the most robust health endpoints (effects or outcomes) have been identified. The most robust health endpoints are where a relationship has been firmly (based on sound studies and statistical analysis) established between exposure to particulate matter and a specific health endpoint (effect/outcome)
- Identify the most relevant and robust exposure-response relationship for the quantitative assessment of exposure. The exposure-response relationships are derived from published peer reviewed sources and relate to the identified health endpoints (effects/outcomes)
- The health endpoints and associated exposure-response relationships adopted for this assessment, in particular those associated with nitrogen dioxide and particulate matter derived from combustion sources (such as petrol and diesel vehicles) have been discussed with NSW Health prior to the completion of this assessment.

For other pollutants and stressors, national guidelines based on the protection of health have been adopted.

### 3.2.5 Risk characterisation

Risks have been characterised using quantitative and qualitative assessment methods. For the assessment of nitrogen dioxide and particulate matter, the quantitative assessment involved identification of an exposure concentration that relates to the project (ie the change in particulate concentration associated with the project), use of relevant exposure-response relationships (for the health endpoints/effects assessed) to calculate health impacts. This enabled an assessment of an increased annual risk and an increased incidence of the effect occurring within the population of concern.

In some cases, a qualitative assessment has been undertaken. A qualitative assessment does not specifically require the quantification of risk or exposure. Rather, the assessment provides a relative or comparative evaluation of whether the exposure or impact considered is positive or negative and where there may be a negative impact, whether this impact is acceptable or unacceptable in the local population.

The assessment presented has also considered the level of uncertainty associated with the concept design, and all aspects of the technical studies relied on for the conduct of the HIA and within the HIA. The final determination of risks to human health was based on the quantification of risks as well as consideration of these uncertainties.

### 3.2.6 Features of the risk assessment

The HIA has been carried out in accordance with international best practice and general principles and methodology accepted in Australia by groups/organisations such as National Health and Medical Research Committee (NHMRC), NEPC and enHealth. There are certain features of risk assessment methodology that are fundamental to the assessment of the outputs and to drawing conclusions on the significance of the results. These are summarised below:

- The assessment has relied on assessments completed in other technical reports, specifically in relation to traffic, air quality, noise and vibration, economic and social impacts
- A risk assessment is a tool (that is systematic) that addresses potential exposure pathways based on an understanding of the nature and extent of the impact assessed and the uses of the local area by the general public. The risk assessment is based on an estimation of maximum, or worst case, impacts (air quality, noise and vibration) in the local community and hence is expected to overestimate the actual risks
- Conclusions can only be drawn with respect to emissions to air, noise and vibration derived from the project as outlined in the respective technical reports

- Available statistics in relation to the existing health status of the existing community are
  presented. However, the HIA does not provide an evaluation of the overall health status of the
  community or any individuals. Rather, it is a logical process of calculating and comparing potential
  exposure concentrations (acute and chronic) in surrounding areas (associated with the project)
  with regulatory and published acceptable air concentrations that any person may be exposed to
  over a lifetime without unacceptable risk to their health. It can also involve calculating an
  incremental impact that can be evaluated in terms of an acceptable level of risk
- The risk assessment reflects the current state of knowledge regarding the potential health effects
  of chemicals identified and evaluated in this assessment. This knowledge base may change as
  more insight into biological processes is gained.

This assessment has focused on key impacts on air quality, noise and vibration and social changes. Other impacts relevant to the health of the community, as outlined in the SEARs have also been considered.

# 3.3 Incorporation of health issues into the project design

The design of the project has been undertaken with changes made to various aspects of the design to minimise impacts on the community, including on health and wellbeing. Some of the key design changes that have been incorporated into the project to minimise impacts to community health include:

- Selection of a road tunnel instead of the development of a surface road, thereby reducing potential air quality impacts to residents along the reserved corridor and providing increased use of existing surface roads for cyclists, pedestrians and public transport
- Tunnel and portals designed to minimise impacts on surrounding parkland.

In addition, the tunnel ventilation system has been designed to meet the in-tunnel air quality criteria, ensure emissions are dispersed so that there are minimal or no effects on air quality and does not require portal emissions. The design considerations included ensuring the location, height, diameter and emission ventilation rate minimises local air quality impacts.

Noise mitigation measures (road pavement treatments, noise barriers and/or architectural treatments where necessary) have also been identified to address predicted exceedances of operational noise traffic.

Refer to Chapter 5 (Project alternatives and options) of the environmental impact statement for additional details on design considerations.

# 4 Community Profile

# 4.1 General

This section provides an overview of the communities potentially impacted by the project. The key focus of the assessment presented is the local community evaluated in relation to the project, referred to as the study area. The proposed F6 extension will comprise of a new multi-lane road between the New M5 at Arncliffe and President Avenue at Kogarah. The project will connect underground with the New M5 tunnel and to a new surface level intersection at President Avenue, Kogarah. The study area, illustrated in **Figure 4-1**, identifies the area over which impacts to air quality has been considered (referred to as GRAL domain). A smaller area, within this larger area, has been considered for the assessment of noise, soil and vibration impacts.



Figure 4-1 HIA study area

In reviewing key aspects of the local communities that are relevant to the conduct of the HIA, information has been obtained from the Australian Bureau of Statistics (ABS) Census 2016. Information has also been collected from relevant to local government areas (LGAs) and health districts (in particular South Eastern Sydney and Sydney local health districts). In some cases, where local data is lacking, information has been obtained (or compared with) data from larger population areas of Sydney and/or NSW.

# 4.2 Surrounding area and population

The population considered in this assessment include those who live or work within the vicinity of the construction compounds, interchanges (ie where the tunnel interfaces with the surface road network), ventilation facilities and the road network, related to the F6 extension as well as the combined WestConnex project.

The study area covers a large number of individual suburbs that sit within the following LGAs:

- Bayside (amalgamated from Botany and Rockdale LGAs)
- Sydney
- Inner West (amalgamated from Ashfield, Leichhardt and Marrickville LGAs)
- Canterbury Bankstown
- Georges River

The above list reflects the current LGAs as defined following amalgamations. Some data used is only available for the former LGAs.

# 4.3 Sensitive receptors

The assessment of potential impacts on the surrounding community, particularly in relation to air quality, has considered the location where maximum impacts from the project may occur. In addition, impacts in the wider community have also been considered. Within the wider community, a number of additional locations, referred to as community receptors, have been identified in the suburbs close to the project. Community receptors are representative locations in the local community where more sensitive members of the population, such as infants and young children, the elderly or those with existing health conditions or illnesses, may spend a significant period of time. These locations comprise hospitals, child care facilities, schools and aged care homes/facilities. **Table 4-1** presents a list of the community receptors included in this assessment. It is noted that this is representative only and is not intended to comprise an exhaustive list of community receptors in the study area.

The location of the sensitive or community receptors is presented in Figure 4-2.

In addition to these community receptors, 17,509 individual receptors (residential, workplace and recreational [RWR] receptors also shown in **Figure 4-2**) have been modelled in the streets/suburbs located in the study area. These individual RWR receptors represent a range of uses including residential, workplaces or recreational (open space) areas in the surrounding community, as detailed in **Table 4-2**. The RWR include all other sensitive community receptors located in the study area, not only those included in **Table 4-1**.

All these individual receptors have also been considered in this report, so that all sensitive receptors have been adequately addressed.

	Receptor name	Type of receptor	Suburb	LGA
CR1	St Finbar's Primary School	Primary School	Sans Souci	Georges River
CR2	St George Christian School Infants	Primary School	Sans Souci	Georges River
CR3	Ramsgate Public School	Primary School	Ramsgate Beach	Bayside
CR4	Estia Health	Community Home	Kogarah	Bayside
CR5	Wesley Hospital Kogarah	General Hospital	Kogarah	Georges River
CR6	St George School	Special School	Kogarah	Bayside
CR7	St George Hospital	General Hospital	Kogarah	Georges River
CR8	Brighton-Le-Sands Public School	Primary School	Brighton Le-Sands	Bayside
CR9	Kogarah Public School	Primary School	Kogarah	Georges River
CR10	St George Girls High School	High School	Kogarah	Georges River
CR11	St Thomas More's Catholic School	Primary School	Brighton Le-Sands	Bayside
CR12	Jenny-Lyn Nursing Home	Community Home	Brighton Le-Sands	Bayside
CR13	Huntingdon Gardens Aged Care Facility	Community Home	Bexley	Bayside
CR14	Rockdale Public School	Primary School	Rockdale	Bayside
CR15	Scalabrini Village Nursing Home-Bexley	Community Home	Bexley	Bayside
CR16	Rockdale Nursing Home	Community Home	Rockdale	Bayside
CR17	Arncliffe Public School	Primary School	Arncliffe	Bayside
CR18	Athelstane Public School	Primary School	Arncliffe	Bayside
CR19	Al Zahra College	Combined Primary- Secondary School	Arncliffe	Bayside
CR20	Cairnsfoot School	Special School	Brighton Le-Sands	Bayside
CR21	Undercliffe Public School	Primary School	Earlwood	Canterbury- Bankstown
CR22	Ferncourt Public School	Primary School	Marrickville	Inner West
CR23	Tempe High School	High School	Tempe	Inner West
CR24	St Peters Public School	Primary School	St Peters	Inner West
CR25	St Pius' Catholic Primary School	Primary School	Enmore	Inner West
CR26	Frobel Alexandria Early Learning Centre	Child Care Centre	Alexandria	Sydney
CR27	Little Learning School - Alexandria	Child Care Centre	Alexandria	Sydney
CR28	Active Kids Mascot	Child Care Centre	Mascot	Bayside
CR29	Mascot Public School	Primary School	Mascot	Bayside
CR30	Hippos Friends	Child Care Centre	Botany	Bayside

# Table 4-1 Community receptors included in health risk assessment



Figure 4-2 :Community receptors and RWR receptors evaluated in HIA

#### Table 4-2 Summary of RWR receptor types

Receptor type	Number	% of total
Aged care	32	0.18%
Child care / pre-school	21	0.12%
Commercial	1,359	7.76%
Community	3	0.02%
Further education	4	0.02%
Hospital	7	0.04%
Industrial	355	2.03%
Mixed use	617	3.52%
Other	445	2.54%
Park / sport / recreation	174	0.99%
Residential	14,408	82.28%
School	84	0.48%
Total	17,509	100.00%(a)

Total of receptor types does not add up to exactly 100 per cent due to rounding.

# 4.5 **Population profile**

The population within the study area consists of residents and workers as well as those attending schools, day care centres, hospitals and recreational areas. The composition of the populations located within the study area is expected to be generally consistent with population statistics for the larger individual suburbs that are wholly or partially included in the study area. Population statistics for the LGAs are available from the ABS for the Census year 2016 and are summarised in **Table 4-3**. For the purpose of comparison, the population statistics presented also include the statistics for larger statistical population groups in the area (defined by the ABS SA4) and the larger statistical areas of Greater Sydney and the rest of the NSW (excluding Greater Sydney) (as defined by the ABS).

**Table 4-4** presents a summary of a selected range of demographic measures relevant to the population of interest with comparison to statistical areas of Greater Sydney and the rest of NSW (excluding Greater Sydney).

Leastion	Total population		% Population by key age groups							
Location	Male	Female	0–4	5–19	20–64	65+*	1–14*	30+*		
Local government areas										
Botany #	23,229	23,420	6.2	16.5	64.3	13.0	15.7	59.8		
Rockdale #	54,079	55,325	6.1	14.8	63.8	15.3	14.6	61.5		
Sydney	107,852	100,530	3.3	7.4	81.0	8.2	5.9	57.6		
Inner West	88,736	93,302	5.9	13.2	68.7	12.2	14.1	63.8		
Canterbury – Bankstown	172,327	173,977	7.2	19.6	59.2	13.9	19.2	58.4		
Georges River	71,755	75,086	5.8	17.0	61.8	15.3	15.7	60.8		
Larger local statistical areas (SA4 – includes local government areas)										
Sydney - City and Inner South	161,061	154,483	4.1	9.6	76.9	9.4	8.6	58.9		
Sydney – Inner West	142,436	150,867	5.9	14.5	66.1	13.5	14.6	61.9		
Sydney – Inner South West	282,753	288,670	6.7	18.1	60.7	14.6	17.5	59.6		
Statistical areas of Sydney and NSW										
Greater Sydney	2,376,766	2,447,221	6.4	18.2	61.4	13.9	17.4	60.4		
Rest of NSW (excluding Greater Sydney)	1,301,717	1,341,813	5.8	18.5	55.1	20.6	17.3	64.6		

Ref: Australian Bureau of Statistics, Census Data 2016 SA = statistical area

\* Age groups specifically relevant to the characterisation of risk

# (Now amalgamated and known as Bayside Council)

Comparing the populations of the study area to that of Greater Sydney the following is noted:

- Sydney City and Inner South have a lower proportion of children (0-19 years), a higher proportion of working aged individuals and a lower proportion of individuals aged over 65 years
- Sydney Inner West have a slightly lower proportion of children and slightly higher proportion of working age individuals.
- At a local government area level:
  - Sydney have a lower proportion of young children (0-4 years)
  - Botany, Rockdale, Sydney, Inner West, and Georges River have a lower proportion, while Canterbury-Bankstown have a higher proportion of children (5-19 years)
  - Canterbury-Bankstown have a lower proportion while Botany, Rockdale, Sydney and Inner West, have a higher proportion of working age individuals
  - Sydney and Inner West have a lower proportion while Rockdale and Georges River have a higher proportion of individuals aged over 65 years.

The estimated population growth from 2011 to 2036 for these areas are (NSW Planning & Environment 2016):

- Botany: 75.2 per cent growth
- Rockdale: 50.2 per cent growth
- Sydney: 72.0 per cent growth
- Inner West: 28.7 per cent growth
- Canterbury Bankstown: 49.7 per cent growth
- Georges River: 28.5 per cent growth.

Location	Median age	Median household income (\$/week)	Median mortgage repayment (\$/month)	Median rent (\$/week)	Average household size (persons)	Unemployment rate (%)		
Local government areas								
Botany #	35	1,626	2,400	460	2.7	5.6		
Rockdale #	35	1,575	2,167	460	2.7	6.2		
Sydney	32	1,926	2,499	565	2.0	6.0		
Inner West	36	2,048	2,600	480	2.4	4.8		
Canterbury – Bankstown	35	1,298	2,000	380	3.0	8.2		
Georges River	37	1,654	2,167	450	2.9	6.5		
Larger local statistical areas (SA4 – includes local government areas)								
Sydney - City and Inner South	33	1,894	2,500	550	2.2	5.7		
Sydney – Inner West	36	1,964	2,500	500	2.6	5.5		
Sydney – Inner South West	35	1,431	2,167	415	2.9	7.4		
Statistical areas of Sydney and NSW								
Greater Sydney	36	1,750	2,167	440	2.8	6.0		
Rest of NSW (excluding Greater Sydney)	43	1,168	1,590	270	2.4	6.6		

Table 4-4 Selected demographics of population of interest

Source: Australian Bureau of Statistics, Census Data 2016

# (Now amalgamated and known as Bayside Council)

The social demographics of an area have some influence on the health of the existing population. As shown in **Table 4-4**, comparing the populations of the study area to that of Greater Sydney:

- Botany, Rockdale, Canterbury-Bankstown and Georges River have a lower, while Sydney, and Inner West have a higher median income
- Botany, Sydney and Inner West have higher, while Canterbury-Bankstown have lower monthly mortgage repayments
- Sydney has higher and Canterbury-Bankstown has lower median weekly rental costs
- Sydney and Inner West have a smaller average household size
- Canterbury-Bankstown has higher and Inner West have lower unemployment rates.

# 4.8 Existing health of the population

### 4.8.1 General

The assessment presented in this report has focused on key pollutants that are associated with construction and combustion sources (from vehicles), including volatile organic compounds, polycyclic aromatic hydrocarbons, carbon monoxide, nitrogen dioxide and particulate matter (namely  $PM_{2.5}$  and  $PM_{10}$ ). For these pollutants, there are a large number of sources in the study area including other combustion sources (wood-fired heating, domestic cooking, industrial emissions), non-combustion sources including other local construction/earthworks. Other aspects that affect the health of an individual include personal exposures (such as smoking) and risk taking behaviours.

When considering the health of a local community there are a large number of factors to consider. The health of the community is influenced by a complex range of interacting factors including age, socioeconomic status, social networks, behaviours, beliefs and lifestyle, life experiences, country of origin, genetic predisposition and access to health and social care. Hence, while it is possible to review existing health statistics for the local areas surrounding the project and compare them to the Greater Sydney area and NSW, it is not possible or appropriate to be able to identify a causal source, particularly individual or localised sources.

Information relevant to the health of populations in NSW is available from NSW Health for populations grouped by local health districts (where the project area is located in the South Eastern Sydney Local Health District and Sydney Local Health District). Not all of the health data is available for all of these areas.

Most of the health indicators presented in this report are not available for each of the smaller suburbs/statistical areas surrounding the site. Health indicators are only available from a mix of larger areas (that incorporate the study area), namely the South Eastern Sydney Local Health District and the Sydney Local Health District. There are few health statistics that are reported for the smaller local government areas relevant to this project. The health statistics for these larger areas (and in some cases data for the Greater Sydney area) are assumed to be representative of the smaller population located within these districts and areas.

### 4.8.2 Health related behaviours

Information in relation to health related behaviours (that are linked to poorer health status and chronic disease including cardiovascular and respiratory diseases, cancer, and other conditions that account for much of the burden of morbidity and mortality in later life) is available for the larger populations within the local health districts in Sydney and NSW. This includes risky alcohol drinking, smoking, consumption of fruit and vegetables, being overweight or obese, and adequate physical activity. The study population is located within the South Eastern Sydney Local Health District and the Sydney Local Health District. The incidence of these health-related behaviours in these districts, compared with other districts in NSW, and the state of NSW (based on NSW Health data from 2015 and 2016) is illustrated in **Figure 4-3.** 

Review of this data indicates the population in the South Eastern Sydney and Sydney local health districts (that include the study area) have lower rates of physical inactivity and of being overweight and obese compared with NSW.



Note: these health related behaviours include those where the behaviour/factor may adversely affect health (eg alcohol drinking, smoking, being overweight/obese and inadequate physical activity) and others where the behaviour/factor may positively affect (enhance) health (eg adequate fruit and vegetable consumption). Study area is located in the South Eastern Sydney Local Health District and Sydney Local Health District

Figure 4-3 Summary of incidence of health-related behaviours (Source: HealthStats NSW 2018)

## 4.8.3 Health indicators

**Figure 4-4** presents a comparison of the rates of the key mortality indicators based on data from 2011 to 2015 (depending on the available data) for all causes, potentially avoidable, cardiovascular disease, lung cancer and chronic obstructive pulmonary disease (COPD), reported in the larger South Eastern Sydney and Sydney local health districts, with comparison to other NSW local health districts (in urban and regional areas) as well as NSW as a whole.

**Figure 4-5** present a comparison of the rates of the hospitalisations for key health effects based on data from 2015-2016 for diabetes, cardiovascular disease, asthma (5–34 years) and COPD (65+ years) reported in the larger South Eastern Sydney and Sydney local health districts, with comparison to other NSW local health districts (in urban and regional areas) as well as NSW as a whole.

It is noted that the data reported in these figures is based on statistics that are publicly available from NSW Health. Hence some of the statistics for mortality and hospitalisations relate to slightly different health endpoints and/or different age groups. The statistics are included for general comparison and discussion. Actual health statistics considered in the characterisation of risk are presented in **Table 4-5**.



Figure 4-4 Summary of mortality data 2011–2015 (Source: HealthStats NSW 2018)

Review of the figures presented above indicate that the rate of mortality for the indicators presented in the South Eastern Sydney and Sydney local health districts are significantly lower than that reported for NSW, except for lung cancer which was not significant for Sydney Local Health District.


Figure 4-5 Summary of hospitalisation data 2015–2016 (Source: HealthStats NSW 2018)

Review of the figures presented above indicate that the rate of hospitalisations for the indicators presented in the South Eastern Sydney and Sydney local health districts is significantly lower than that reported for NSW, with the exception for cardiovascular disease hospitalisations in South Eastern Sydney, which is similar to the rate for NSW.

In relation to mental health, data from NSW Health indicates the following for adults:

- The rate of high or very high psychological distress reported in 2015 in the Sydney Local Health District (13.9 per cent) is a little higher, and South Eastern Sydney local health districts (9.3 per cent) a little lower than the state average (11.8 per cent), however none were significantly different.
- The rate of high or very high psychological distress in Sydney Local Health District has varied between 10 and 15 per cent between 2003 and 2015 while in the South Eastern Sydney Local Health District, the rate has declined from around 14 per cent in 2003 to less than 10 per cent in 2015.

In relation to some more specific health indicators **Table 4-5** presents the available data for the slightly smaller population areas in the LGAs in the study area. These have been compared with available data for the South Eastern Sydney Local Health District, Sydney Local Health District, Sydney and NSW. It is noted that health statistics are not available for the LGAs for all the health endpoints considered in this assessment. Where available, they have been presented for the purpose of comparison with statistics from Sydney and NSW.

The health indicators presented in the table include those that are specifically relevant to the quantification of exposure to nitrogen dioxide and particulate matter presented in **section 6**.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

#### Table 4-5: Summary of key health indicators

lealth indicator Data available for population areas (rate per 100,000 population)										
	Botany LGA	Rockdale LGA	Sydney LGA	Inner West LGA	Canterbury- Bankstown LGA	Georges River LGA	South Eastern Sydney LHD	Sydney LHD	Sydney (wider metro area)*	MSN
Mortality										
All causes – all ages	523.8 C	534.5 C	508.0 C	534.2 C	490.6 C	465.5 C	493.0 C	477.4 C		546.0 C
All causes (non-trauma) ≥30 years									976.5	
All causes ≥30 years									1026	
Cardiopulmonary ≥30 years									412	
Cardiovascular – all ages	150.0 C	150.0 C	138.9 C	146.4 C	139.2 C	131.3 C	134.7 C	128.7 C	191.8	155.7 C
Respiratory – all ages							37.8 A	39.9A	51.5	46.8 A
Hospitalisations										
Coronary heart disease	713.4 B	467.6 B	378.1 B	276.5 B	448.0 B	436.3 B	611.9 E	328.5 E		525.7 E
COPD >65 years							928.5 E	1147.3 E		1462.8 E
COPD All ages	191.3 B	155.3 B	243.4 B	195.9 B	199.4 B	128.8 B	142.4 E	187.3 E		242.2 E
Cardiovascular disease										
All ages	1988.6 B	1499.1 B	1435.3 B	1329.3 B	1613.6 B	1372.4 B	1772.1 E	1372.4 E	1976	1713.3 E
>65 years									9235	
Respiratory disease										
All ages							1441.8 E	1494.3 <sup>E</sup>	2003	1731.3 E
>65 years									3978	
Asthma										
Asthma hospitalisations (ages 5–34 years)							124.0 E	137.6 E		171.1 E
Asthma emergency department hospitalisations (1–14 years)									1209	
Asthma prevalence (current) for children aged 2–15 years							10.2% C	6.2% C		13.5% C
Current asthma for ages 16 and over							9.0% D	9.7% D		11.3% D

\* Data for Sydney Metropolitan area for 2010 based on hospital statistics as reported for 2010 and population data from the ABS for 2011 (relevant to each age group considered) used in review of exposure and risks to inform recommendations for updating the National Environment Protection Measure (NEPM) Ambient Air Quality (AAQ) (Golder 2013)

All other data has been obtained from Health Statistics New South Wales, where: A: 2013–2015 data B: 2014-15 to 2015-16 data C: 2014-2015 or 2015 data D: 2016 data E: 2015-2016 data

-- No data available Bold and shaded: Data used in the characterisation of risk

#### Table 4-6: Summary of key health indicators: Mental health

Age group	Number of prescriptions for antidepressants per 100,000 people, by LGA in 2014-2015								
	Botany	Sydney Inner City	Marrickville – Sydenham- Petersham	Canterbury	Kogarah - Rockdale	NSW average			
17 years and under	4,988	7,284	6,531	3,294	3,502	8,187			
18 to 64 years	65,100	76,303	79,279	54,776	58,780	90,959			
65 years and over	149,818	159,584	158,224	143,705	152,210	179,771			

Data from Australian Atlas of Healthcare Variation, Atlas 2015 (note that the Atlas 2017 did not include mental health data)

Review of the data presented in **Table 4-5** generally indicates that for the population in project area, the health statistics (including mortality rates and hospitalisation rates for most of these categories) are variable but generally similar to those reported in the larger local health districts of South Eastern Sydney, Sydney and the wider Sydney metropolitan area and slightly lower than the whole of NSW.

For the assessment of potential health impacts from the project, where specific health statistics for the smaller populations within the project area is not available (and not reliable due to the small size of the population), adopting health statistics from the whole of NSW is considered to provide a representative, if not cautious (eg over estimating existing health issues), summary of the existing health of the population of interest.

The rate of antidepressant medication prescriptions is an indicator that can be used to review changes in stress and anxiety levels within a community, and these are presented in **Table 4-6**. While these data were not directly used in the HIA, to evaluate specific impacts, the data is relevant to assist in ongoing monitoring of potential indicators of changes that increase or decrease stress and anxiety in the community. In relation to the rate of medication prescriptions for antidepressants it is noted that all local government areas have lower rates of prescription, for all age groups, than the state average.

# 5 Community Concerns

A range of community engagement activities have been and continue to be undertaken as part of the F6 Extension – Stage 1 project, as outlined in Chapter 15 (Social and economic) of the environmental impact statement. Issues raised during community consultation have covered a range of different aspects of the project, with the following **Table 5-1** showing the key issue categories.

Issue	Project stage		Detail		
	Construction	Operation			
Property	~		Concern about the scale of impacts to property, particularly the potential loss of homes and/or green space		
	✓	~	Concern about impacts on property values		
	✓	~	Concern about whether compensation would be offered to those properties affected by the project		
Accessibility and parking	✓	✓	Concern about local traffic impacts and congestion due to increased traffic volumes on local roads		
		✓	Concern about the project encouraging rat runs and dangerous driving behaviour		
		$\checkmark$	Concern about project cost and tolling		
		~	Concern about risk of increased collisions between vehicles and general decrease in road safety		
	✓		Concern about the relocation of bus stops in the vicinity of the project		
	✓	~	Concern about the impact on access to green space and community facilities such as local sporting fields		
	✓	~	Concern about the removal of parking and the impacts this would have on side streets		
-	✓	~	Concern about pedestrian safety due to increased traffic movements, particularly for children		
	✓		Concern about road closures during construction affecting pedestrian, cycle and vehicle movements		
		~	Request to build cycling and pedestrian infrastructure to improve safety and connectivity for active transport users in the area		
Amenity	✓	~	Concern about the noise, vibration and pollution impacts of heavy vehicles and increased traffic		
		✓	Concern that the project would decrease the liveability of the area		
		✓	Concern about a decrease in the amenity of the existing public transport infrastructure in the area		
		~	Concern about the visual and amenity impacts of ventilation outlets and noise walls		
	✓		Concern about the removal of trees and vegetation		
	✓		Concern regarding the management of and disruption caused by spoil removal		
	✓		Concern regarding the disturbance of contaminants		
	✓		Concern about the amenity impacts (especially resulting from tunnelling) of construction including pollution, noise and vibration		
		~	Concern about air quality impacts of the project, including the health impacts of exhaust emissions		

 Table 5-1 Feedback provided by community and key stakeholders

Issue	Project stage		Detail		
	Construction	Operation			
		~	Concern about the health, safety and environmental impact of the ventilation outlets and suggestions that they should be filtered, particularly as they are in close proximity to playing fields and parks		
		~	Suggestion that the ventilation outlets be constructed within industrial areas and equally distanced along the project corridor, so that no residents are subjected to air pollution disproportionately		
Community facilities	✓		Concern about impacts on the dog park at Patmore Swamp		
	~		Concern about impacts to the Memorial Playing Fields, used by local schools and sporting groups		
	~		Concern over impacts to the local playgrounds and recreational facilities within Rockdale Bicentennial Park		
	~		Concern over the impact on Kogarah playing fields and local sporting clubs, including use of community facilities, membership and relocation logistics		
	~	~	Concern about the cumulative impacts to open green space which would make exercising more difficult		
	~	~	Concern about the impacts to Brighton-Le-Sands Public School (due to close proximity to the President Avenue intersection and tunnel portals) and Arncliffe Public School		
	~		Concern that the F6 Extension would delay the reinstatement of Kogarah Golf Course		
		$\checkmark$	Proximity of ventilation facilities to playing fields and parks		
		$\checkmark$	Request for separate cycle paths, pedestrian bridges and extended cycle paths		
Environment	~	~	Concern about the impacts to ecological values and water quality of the Rockdale Wetlands and Rockdale Bicentennial Park, including threatened and migratory species		
	~	~	Concern about impacts to water quality and hydrology within Scarborough Park		
	~	~	Concern about the impacts to areas and properties of historical significance, such as settlement as a result of tunnelling		
Business impacts	~	~	Concern about impacts to local businesses including the loss of parking		
	✓	~	Concern about changes in access to local businesses		
	~	~	Some concern that traffic congestion would affect business deliveries while others perceived business deliveries would be more efficient as a result of the project		
	<b>√</b>		Concern over the impact of road closures resulting in disruptions to businesses, particularly those relying on passing trade (e.g. vehicle related businesses)		

None of the issues raised and grouped as above directly refer to health concerns, however issues such as air and water quality, noise and road safety are related to health. In addition, a number of other issues raised may also indirectly affect health and wellbeing.

# 6 Assessment of changes in air quality on community health

# 6.1 General

The characterisation of changes in air quality as a result of the project is complex. Full details of the assessment undertaken are presented in **Appendix E** (Air quality technical report) of the EIS conducted by ERM (2018). This section presents an overview of the key aspects of the assessment undertaken and an assessment of potential health impacts associated with the predicted changes in air quality in the local community.

# 6.2 Existing air quality

When predicting the impact of any new or modified source of air pollution, it is necessary to take into account the way in which the emissions from the source would interact with existing pollutant levels. Defining these existing levels and the interactions can be challenging, especially in a large urban area such as Sydney where there is a complex mix of sources. It is important to consider both the temporal and spatial variation in pollutant concentrations; these fluctuate a great deal on short time scales, but also show cyclical variations. Moreover, in large urban areas there is usually a complex mix of pollution sources, and substantial concentration gradients. Short term meteorological conditions and local topography are also important.

Air quality in the Sydney region has improved over the last few decades. The improvements have been attributed to initiatives to reduce emissions from industry, motor vehicles, businesses and residences.

Historically, elevated levels of carbon monoxide were generally only encountered near busy roads, but concentrations have fallen as a result of improvements in motor vehicle technology. Since the introduction of unleaded petrol and catalytic converters in 1985, peak carbon monoxide concentrations in central Sydney have significantly reduced, and the last exceedance of the air quality standard for carbon monoxide in NSW was recorded in 1998 (NSW DECCW 2010).

While levels of nitrogen dioxide, sulfur dioxide  $(SO_2)$  and carbon monoxide continue to be below national standards, levels of ozone and particulate matter (PM) can exceed the standards adopted in NSW (NSW EPA 2016) from time to time.

Ozone and PM levels are affected by:

- The annual variability in the weather
- Natural events such as bushfires and dust storms, as well as hazard reduction burns
- The location and intensity of local emission sources, such as wood heaters, transport and industry (NSW OEH 2015).

The project lies within an urbanised area of Sydney and hence it is important that the background air quality considered is representative of existing conditions in the local area.

Assessment of background air quality, including meteorological data, requires the use of data that has been collected from equipment that complies with Australian Standards (to ensure that data is reliable and comparable).

The NSW OEH operates a number of monitoring stations in the Sydney area (see **Figure 6-1**), with the closest stations being located at Earlwood and Randwick. The OEH stations at Chullora and Rozelle were further away but were still considered important in terms of characterising air quality in the Sydney region, as were the stations at Lindfield, Liverpool, Macquarie Fields and Prospect.

In addition, Roads and Maritime Services has established several long-term monitoring stations in response to community concerns relating to the ventilation outlet of the M5 East Tunnel, and to monitor operational compliance of the tunnel with ambient air quality standards. Four of the Roads and Maritime stations (shown on **Figure 6-1** as CBMS, T1, U1, X1) were in the vicinity of the M5 East ventilation outlet. Two Roads and Maritime stations (shown on **Figure 6-1** as CBMS, T1, U1, X1) were in the vicinity of the M5 East ventilation outlet. Two Roads and Maritime stations (shown on **Figure 6-1** as F1 and M1) were much closer to busy roads near the M5 East Motorway tunnel portals. Other Roads and Maritime ambient air modelling locations established as part of the NorthConnex project and near the intersection of Epping Road and Longueville Road (to assess impacts form the Lane Cove Tunnel) were also considered.

Sydney Motorway Corporation (SMC) has established a WestConnex monitoring network to address some of the gaps in the OEH and Roads and Maritime monitoring in terms of pollutants and locations, and SMC has engaged Pacific Environment to operate and maintain the network. The WestConnex network includes monitoring stations at both urban background and near-road stations. Five new monitoring stations were introduced in the M4 East area, seven new stations in the New M5 area, and two new stations in the M4-M5 Link area to support the development and assessment of the respective projects. Some of the WestConnex monitoring stations were subsequently relocated or decommissioned.

Two project-specific monitoring stations were established for the F6 Extension by Roads and Maritime in 2017. One of these was at a background location, and the other at a roadside location. Given the date of deployment, the time period covered was too short for these to be included in the development of background concentrations and model evaluation.



#### Figure 6-1 Locations of air quality monitoring stations

Background air quality relevant to the assessment of carbon monoxide, nitrogen dioxide and particulate matter were determined in **Appendix E** (Air quality technical report) on the basis of data from these monitoring stations. The background air quality considered in **Appendix E** (Air quality technical report) related to air quality in areas away from major roadways.

In relation to the background air quality considered in **Appendix E** (Air quality technical report) for the project area, the following is noted:

- **Carbon monoxide**: background air concentrations (as one hour and eight hour averages) were below the current air quality guidelines both at any of the background air monitoring stations. A general downward trend in background air concentrations was observed.
- **Nitrogen dioxide**: background air concentrations (as one hour and annual averages) were below the current air quality guidelines both at all background air monitoring stations and at roadside monitoring locations. The concentration of nitrogen dioxide has been observed to be generally stable to trending downward over time.
- **Ozone**: background air concentrations (as one hour and four hour averages) exceeded the current air quality guidelines on a few occasions. The most number of times a station exceeded the guideline per year was eighteen, with many of the stations not exceeding more than 5 times per year. Annual ozone concentrations were stable between 2004 2016.
- PM<sub>10</sub>: background concentrations of PM<sub>10</sub> (as an annual average) were below the current air quality guidelines. However, there were exceedances of the 24 hour average criterion, most notably in the warm and dry year 2009.
- PM<sub>2.5</sub>: Long term measurement of annual PM<sub>2.5</sub> concentrations has only occurred at three OEH stations Chullora, Earlwood and Liverpool. Concentrations at these stations showed a broadly similar pattern, with a systematic reduction between 2004 and 2012 being followed by a substantial increase in 2013. The main reason for the increase was a change in the measurement method (as the reporting of PM<sub>2.5</sub> in air varies depending on the type of equipment used). The increases meant that background PM<sub>2.5</sub> concentrations in the study area during 2014 and 2015 were already very close to or above the annual average criterion of eight micrograms per cubic metre. There have been a number of exceedances of the 24 hour average criterion of 25 micrograms per cubic metre.
- Air toxics: A number of campaigns have been undertaken to determine the levels of air toxics around Sydney. All have found the concentrations remain low and under the respective Air Toxic NEPM investigation levels.

# 6.3 Overview of air quality impact assessment

## 6.3.1 Construction

**Appendix E** (Air quality technical report) evaluated impacts on air that may occur during construction. The assessment considered impacts that may occur during tunnelling activities and surface works and involved a semi quantitative assessment approach. The assessment of construction activities addressed two different construction scenarios or areas, as outlined below.

Compound	Description	Indicative construction period
Zone 1	C1	Q4 2020 to Q2 2023
Zone 2	C2,3,4,5	Q4 2020 to Q4 2023

#### Table 6-1: F6 Extension Stage 1 construction compounds

(a) (Q) Quarters refer to the calendar year

(b) C = Construction Boundaries and facilities. See Figure 2-2 in Technical report – Air quality

The assessment identified the range of activities during construction, potential emissions from these activities and the location of these activities in relation to sensitive receptors. **Figure 6-2**: illustrates the location of the sensitive receptors considered in **Appendix E** (Air quality technical report) during construction works. The figure also shows the location of the zones considered in each of the construction sites. **Appendix E** (Air quality technical report) did not identify the construction of the powerline as a significant source of dust that required impact assessment.



Figure 6-2: Location of sensitive human receptors near the construction of the F6 Extension

It is noted that for demolition activities, the Work Health and Safety Regulation 2011 (NSW) requires that all hazardous materials are properly removed from buildings prior to any demolition works occurring. This is to prevent workers and the public from being exposed these materials and contaminants during the demolition and other construction works. Hence there is no need to further assess the presence of hazardous building materials during construction activities.

This approach then allocated a risk associated with the generation of dust and impacts on human health in the adjacent community. This approach considered the proximity to the source area and the number and type of receptors present. Impacts associated with nuisance dust, health impacts on the community were evaluated. For all demolition, earthworks, construction and track-out activities, where no mitigation measures are implemented, the risk of impacts on human health were evaluated and considered in terms of the location of sensitive receptors. Risk ratings that varied from low to high were adopted in the review presented in **Appendix E** (Air quality technical report). In relation to health impacts, the following levels of risk were identified for the following sites (see **Table 6-1** for zone details):

- Zone 1: Low risk for construction, medium risk for earthworks and track-out with no applicable risk for demolition
- Zone 2: High risk for all activities.

On this basis, appropriate mitigation measures are required to minimise impacts on the local community during construction.

For almost all construction activities, the aim should be to prevent significant impacts on receptors through the use of effective mitigation. Experience from similar construction projects shows that this is normally possible. Hence, where mitigation measures are appropriately implemented, **Appendix E** (Air quality technical report) concluded that the residual risk level would normally be 'not significant'.

However, even with a rigorous Dust Management Plan in place, it is not possible to guarantee that the dust mitigation measures will be effective all the time. There is the risk that nearby residences, commercial buildings, hotel, cafés and schools in the immediate vicinity of the construction zone, might experience some occasional dust soiling impacts. This does not imply that impacts are likely, or that if they did occur, that they would be frequent or persistent. Overall construction dust is unlikely to represent a serious ongoing problem. Any effects would be temporary and relatively short-lived, and would only arise during dry weather with the wind blowing towards a receptor, at a time when dust is being generated and mitigation measures are not being fully effective. The likely scale of this would not normally be considered sufficient to change the conclusion that with mitigation the effects will be 'not significant'.

A Construction Air Quality Management Plan will be produced to cover all construction stages of the project. These measures include site management, monitoring, preparing and maintaining the construction sites, maintenance and controls on vehicles and machinery and construction. Chapter 9 of **Appendix E** (Air quality technical report) provides additional details on the dust management measures proposed.

Issues related to health impacts from construction fatigue, where the community may be located close to construction facilities for extended periods of time, as a result of the number of construction projects being undertaken for WestConnex, are further addressed in Section 10.8.

Acid sulphate soils exposed to air has the potential to release the odorous hydrogen sulfide gas (H2S) into the atmosphere impacting nearby receptors. The assessment of odour impacts from the disturbance of acid sulphate soils during construction activities, stockpiling and treatment north of President Avenue was undertaken. This assessment did not find odour impacts, however it recommended either onsite odour measurements be carried out once construction operations begin so that site-specific odour emission rates can be determined, or site odour audits could be carried out to determine the actual impacts at the nearest receptors. It is noted that acid sulphate soil management plans will be developed for all excavation works within the ancillary facilities and along the Shared cycle and pedestrian pathway construction area to minimise any odour impacts from acid sulphate soils.

### 6.3.2 Operation

The assessment of changes in air quality associated with the operation of the project has been undertaken on the basis of the tunnel designs specifications and forecasts of tunnel and surface road traffic volumes (and speeds) as outlined in the Strategic Motorway Project Model (SMPM). The project does not include portal emissions (ie emissions from the tunnel entrances and exits), hence emissions associated with the operation of the tunnel relate to the discharge of air from within the tunnel to atmosphere via seven ventilation outlets (not all for the F6 Extension Stage 1 project) outlined below, and shown on **Figure 6-3**:

- Existing facility
  - Outlet A: M5 East tunnel outlet at Turrella
- Facilities currently under construction for New M5 Motorway
  - Outlet B: New M5 Motorway facility at Arncliffe
  - Outlet C: New M5 Motorway facility at St Peters Interchange
- Facility proposed for M4-M5 link
  - Outlet D: M4-M5 Link facility at St Peters Interchange
- Facilities proposed for F6 Extension Stage 1
  - Outlet E: F6 Extension Stage 1 facility at Arncliffe
  - Outlet F: F6 Extension Stage 1 facility at Rockdale
- Facility proposed for F6 Extension Section B
  - Outlet G: F6 Extension Section B facility at Rockdale.

The ventilation outlets that would be specific to the F6 Extension Stage 1 are E and F. The remaining outlets (A, B, C, D and G) were included to assess potential cumulative impacts only. Apart from outlet A (M5 East), each ventilation outlet had four 'sub-outlets' for air. Further details of the project ventilation facilities, including the locations and surrounding environments, are provided in **Chapter 6** (Project description) of the EIS.



#### Figure 6-3 Locations of all tunnel ventilation outlets included in the assessment of air quality

A description of the GRAMM-GRAL model system can be found in **Appendix E** (Air quality technical report). The model has also been used to evaluate the cumulative air quality impacts associated with other tunnel projects in the study area. The air modelling domain (study area – GRAL domain) considered for the project is shown in **Figure 4-1**.

The modelling considered meteorology relevant to a larger area (red box, or GRAMM (Graz Mesoscale Model) domain, on **Figure 6-4**) that includes the study area, local terrain, and project-specific emission sources.



Figure 6-4 Modelling domains for GRAMM and GRAL

The emission sources relevant to the project addressed in the modelling included the following:

- Emissions from existing and proposed tunnel ventilation outlets
- Emissions from the traffic on the surface road network, including any new roads associated with the project.

The assessment of cumulative impacts, from the operation of all road tunnel projects, evaluated changes in air quality in the study area from all changes in surface traffic and ventilation outlets associated with all projects in the wider area.

When determining the potential emissions to air that may require ventilation from the tunnel the assessment has considered a range of factors associated with the tunnel design, traffic volumes, vehicle mix and age. In addition, in-tunnel air quality limits have also been considered as discussed further in **Section 7**. These have been taken to be limits/criteria that are required to be met under all operational circumstances (except emergencies such as fire). The tunnel ventilation system and tunnel operational parameters have been designed to ensure the in-tunnel concentration limits are not exceeded.

The assessment of air quality impacts involved estimation of emissions from vehicles using the tunnel, and other road tunnels under expected traffic conditions (ie operating normally with traffic volumes fluctuating over the day with peak and out of peak traffic loads). In addition, a regulatory worst case scenario has been evaluated. The regulatory worst case relates to modelling of emissions from the ventilation facilities at the limit expected to be set by the regulators. This is an upper limit that would essentially mean the tunnel is always full of vehicles and trucks. This is not a realistic scenario, but it is required to demonstrate compliance with regulatory air quality objectives.

Additional details on the assessment scenarios and the emission sources considered in **Appendix E** (Air quality technical report) are summarised in the following sections.

## 6.4 Assessment scenarios

#### 6.4.1 Overview

The assessment of impacts on air quality associated with operation of the project has considered a range of scenarios that include the existing situation, construction works and various future operational scenarios both with and without the project. In addition, a cumulative scenario, associated with impacts from all the road tunnel projects was assessed.

In all of the air modelling scenarios considered, changes in emissions to air from the surface road network as well as the ventilation facilities (as relevant to each scenario) have been included.

The air modelling scenarios have included the following:

- **2016 Base Year**: This represents the current road network with no new projects/upgrades, and was used to establish existing conditions. The main purpose was to enable the dispersion modelling methodology to be verified against actual air quality monitoring data
- **2026 Do Minimum**: The 2026 'Do minimum' case assumes that the WestConnex, King St Gateway and Sydney Gateway projects are complete, but the F6 Extension, Western Harbour Tunnel and Beaches Link are not built. It is called 'do minimum' rather than 'do nothing' as it assumes that some improvements would be made to the broader transport network to improve capacity and cater for traffic growth
- 2026 Do Something: As for the 2026 Do Minimum, but with the F6 Extension Stage 1 also completed
- 2036 Do Minimum: As for the 2026 Do Minimum, but 10 years after project opening and without the project. This took into account changes in traffic and the emission behaviour of the fleet with time
- 2036 Do Something: As for the 2026 Do Something, but 10 years after project opening
- **2036 Do Something Cumulative**: As for the 2036 Do Something, but with all stages of the F6 Extension, Western Harbour Tunnel and Beaches Link also completed.

More specific details associated with each of these scenarios are outlined in **Appendix D** of the EIS (Traffic and transport technical report).

#### 6.4.2 Assessment scenarios evaluated in the health risk assessment

Health impacts that may be associated with changes in air quality that are associated with the project have been assessed for the following years and scenarios:

- 2026: project operations (ie do something)
- 2036: project operations (ie do something) and cumulative impacts (ie do something cumulative).

The assessment has considered total impacts (ie background or existing air quality plus the project) and changes in air quality associated with the project. The assessment of changes in air quality is based on the predicted air quality impacts for all the local roads plus the project (the 'do something scenario') minus the air quality impacts for all the local roads without the project (the 'do minimum' scenario). The net change in air quality assessed relates to emissions directly from the project as well as changes in emissions on surface roads.

In relation to the operation of the project considered in each of the above scenarios the air quality modelling has been undertaken to consider expected traffic volumes within the tunnel. The number of vehicles moving through the tunnel varies depending on the hour of the day. Air modelling predictions associated with the expected traffic movements through the tunnel have been used for the assessment of long term/chronic exposures in the local community.

# 6.5 Vehicle emissions

Emissions from vehicles using the tunnel have been estimated based on an emissions inventory model developed by the NSW EPA (as described in **Appendix E** (Air quality technical report)).

# 6.6 Assessment of volatile organic compounds and polycyclic aromatic hydrocarbons

#### 6.6.1 General

**Appendix E** (Air quality technical report) has considered emissions of volatile organic compounds (VOCs) and polycyclic aromatic hydrocarbons (PAHs) to air from the project. Both VOCs and PAHs refer to a group of compounds with a mix of different proportions and toxicities. It is the individual compounds within the group that are of importance for evaluating adverse health effects. The composition of individual compounds in the VOCs and PAHs evaluated would vary depending on the source of the emissions. Hence it is important that the key individual compounds present in emissions considered for this project are speciated (ie identified and quantified as a percentage of the total VOCs or total PAHs) to ensure that potential impacts associated with exposure to these compounds can be adequately assessed.

VOCs in air in Sydney (OEH 2012) are primarily derived from domestic/commercial sources (54 per cent) with on-road vehicles contributing approximately 24 per cent, industrial emissions eight per cent with the remainder from off-road mobile sources and other commercial sources.

VOCs and PAHs from the project are associated with emissions from vehicles assumed to be using the tunnel (and approaches) and surface roads. The makeup of the VOCs and PAHs emissions would depend on the mix of vehicles considered as these pollutants would be emitted in different proportions from petrol and diesel powered vehicles. In addition, the age and the fuel used by the vehicle fleet would affect these emissions. The vehicle fleet mix considered in this project is summarised in Table 6-2.

	% of VOC					
Pollutant/metric	Petrol I	ight duty	Diesel light duty	Diesel heavy duty		
	Petrol (E0)	Petrol (E10)	Diesei light duty			
Benzene	4.95	4.54	1.07	1.07		
PAHs (as B(a)P) (a)	0.03	0.03	0.08	0.08		
Formaldehyde	1.46	1.82	9.85	9.85		
1,3-Butadiene	1.27	1.20	0.40	0.40		

#### Table 6-2: Volatile organic compounds speciation profile for vehicle emissions

Based on a combination of PAH fraction of THC from NSW EPA (2012b) and the B(a)P fraction of PAH of 4.6 per cent from Environment Australia (2003)

#### 6.6.2 Volatile organic compounds

VOCs have been modelled in **Appendix E** (Air quality technical report) based on emissions from all vehicles considered. The proportion of each of the individual VOCs that may be present in the air is then estimated based on the assumed composition of the vehicle fleet during the different years and the type of fuel used.

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) (EPA 2012). From a toxicity perspective the key VOCs that have been considered for the vehicle emissions are BTX, 1,3-butadiene, acetaldehyde and formaldehyde (consistent with those identified and targeted in studies conducted in Australia on vehicle emissions (*Australian* Department of Environment and Heritage (DEH 2003; EPA 2012)).

The proportion of each of the key VOCs considered are derived from the 2008 Calendar Year Air Emissions Inventory for the Greater Metropolitan Region in NSW (EPA 2012), for the vehicle fleet assessed in **Appendix E** (Air quality technical report) (as summarised above). In relation to passenger vehicles it has been assumed that 60 per cent<sup>1</sup> of fuel used is E10. It is conservatively assumed that the composition of VOCs in vehicle emissions remains the same over time and does not improve with enhanced vehicle emissions technology.

**Table 6-3** presents a summary of the weighted mass fraction for these VOCs considered for the project in 2026 and 2036.

NOC	Weighted % of total VOC estimate				
VOC	2026	2036			
Benzene	3.9	3.4			
Toluene	7.1	5.9			
Xylenes	5.9	4.9			
1,3-butadiene	1.1	0.9			
Formaldehyde	3.4	4.6			
Acetaldehyde	1.6	2.0			

Table 6-3 Weighted volatile organic compounds speciation profile for vehicle emissions

#### 6.6.3 Polycyclic aromatic hydrocarbons

PAHs have been considered in **Appendix E** (Air quality technical report) as key pollutants that may be derived from diesel powered heavy goods vehicles. The total PAH concentration that may be derived from the project has been determined on the basis of a proportion of the total VOCs. While not all of the PAHs would be volatile the approach adopted provides an estimate of potential levels of total PAHs that may be in air, as a result of the change in emissions derived from the project.

For the year 2026 and 2036 total PAHs have been estimated to comprise 0.79 and 0.95 per cent respectively of the total VOCs.

In relation to the toxicity of PAHs, this differs significantly for the different individual PAHs that may be present. The detailed review of the potential health impacts associated with exposures to PAHs in air from the project requires an assessment of the key individual PAHs.

<sup>1</sup> The value of 60 per cent of ethanol in total fuel volume sales comes from the requirement that a minimum of 6% ethanol in the total volume of petrol sold in NSW as outlined in the *Biofuels Act 2007* (NSW). This equates to selling 60% E10 fuel.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

The presence of PAHs in diesel exhaust has been found to be more a function of the PAH content of the fuel than of engine technology. For a given refinery and crude oil, diesel fuel PAH levels correlate with total aromatic content and T90 (distillation temperature where 90 per cent of the fuel is evaporated). Representative data on aromatic content for diesel fuels in Australia is limited, however emissions tests have been conducted on a range of light and heavy vehicles under different traffic congestion conditions (DEH 2003). The data presented from these emissions tests is assumed to include fuels commonly used in Australia and are considered to provide an indication of the likely proportions of individual PAHs in diesel exhaust.

The PAHs reported in diesel exhaust by the DEH (now the Australian Department of Environment and Energy) (DEH 2003) comprise the 16 most commonly reported (and highest proportion) PAHs present in exhaust. The data available from this study is dated (from vehicles manufactured from 1990 to 1996) and use of this data is likely to provide an overestimation of PAH emissions from current (and future) diesel vehicles. The evaluation of potential health impacts associated with exposure to PAHs from the project requires consideration of the 16 individual PAHs, present at the highest levels in exhaust and which have the most information on chronic health effects.

The toxicity of individual PAHs varies significantly, with some considered to be carcinogenic while others are not carcinogenic. For the carcinogenic PAHs, these are commonly assessed as a group with the total carcinogenic PAH concentration calculated using weighting factors that relate the toxicity of individual carcinogenic PAHs to the most well studied PAH, benzo(a)pyrene. For the carcinogenic PAHs the weighting factors presented by the Canadian Council of Ministers of the Environment (CCME 2010) have been adopted. Other PAHs that are not carcinogenic have been considered separately.

On the basis of this approach the speciation of individual PAHs (as per cent of total PAHs) has been calculated based on the data from DEH (2003). The data presented relates to emissions that occur in congested or stop/start traffic. This data has been used to be representative of the worst case situation of heavy congested traffic in the project area and is considered to be conservative for expected traffic conditions in the motorway tunnels. The proportion of these individual PAHs, derived from the older data presented by DEH (2003), is considered to be sufficiently representative for the purpose of this assessment. It should be noted that the calculated risks posed by these non-carcinogenic PAHs is very low (refer to **Table 6-9** and **Table 6-10**) and any likely variation in the proportioning of these individual PAHs (even if the proportioning was out by 100%) will not change the outcome of the health impact assessment undertaken for this project.

**Table 6-4** presents a summary of the PAH speciation profile considered in this assessment for the above traffic conditions.

Per cent of total PAH emissions (PAHs)					
Used to evaluate emissions in 2026 and 2036					
70					
4.9					
2.0					
5.0					
3.4					
0.49					
0.45					
0.71					
4.6					

Table 6-4 Polycyclic aromatic hydrocarbon speciation profile for diesel vehicle emissions

### 6.6.4 Assessment of health impacts

The change in VOC and PAH concentrations associated with the project is a decrease for most receptors, however in some areas there is an increase in concentrations. These changes relate to the redistribution of emissions from vehicles, primarily associated with surface roads. The following evaluation has been undertaken to assess the potential health impacts associated with the maximum increases predicted.

The assessment of potential health impacts associated with exposure to changes in VOCs and PAHs concentrations (calculated for individual VOCs and PAHs based on the speciation outlined above) in air within the community has been assessed on the basis of the following:

- For VOCs and PAHs that are considered to be genotoxic carcinogens (consistent with guidance provided by enHealth (enHealth 2012b)) an incremental lifetime carcinogenic risk has been calculated. For the VOCs and PAHs evaluated in this assessment a carcinogenic risk calculation has been adopted for the assessment of maximum potential (incremental) increase in benzene, 1,3-butadiene and carcinogenic PAHs (as a benzo(a)pyrene toxicity equivalent or TEQ). The assessment undertaken has adopted the calculation methodology outlined in **Annexure B**, adopting the inhalation unit risk values presented in .
- For other VOCs and PAHs, where the health effects are associated with a threshold (ie a level below which there are no effects), the maximum predicted concentration from all sources (ie background plus the project) of individual VOCs and PAHs associated with the project have been compared against published peer-reviewed health based guidelines that are relevant to acute and chronic exposures (where relevant). The health based guidelines adopted (identified on the basis of guidance from enHealth 2012) are relevant to exposures that may occur to all members of the general public (including sensitive individuals) with no adverse health effects. The guidelines available relate to the duration of exposure and the nature of the health effects considered where:
  - Acute guidelines are based on exposures that may occur for a short period of time (typically between an hour or up to 14 days). These guidelines are available to assess peak exposures (based on the modelled one hour average concentration) that may be associated with volatile organic compounds in the air, and are presented in **Table 6-5**.
  - Chronic guidelines are based on exposures that may occur all day, every day for a lifetime. These guidelines are available to assess long term exposures (based on the modelled annual average concentration) that may be associated with volatile organic compounds and PAHs in the air, and are presented in **Table 6-6**.

Compound assessed	Acute health based guideline (µg/m <sup>3</sup> )	Basis
Volatile organ	ic compound	ls
Benzene	580	Acute 1 hour health based guideline, based on depressed peripheral lymphocytes from Texas Commission on Environmental Quality (TCEQ) evaluation (TCEQ 2013d).
Toluene	15000	Acute 1 hour health based guideline, based on eye and nose irritation, increased occurrence of headache and intoxication in human male volunteers from TCEQ evaluation (TCEQ 2013c).
Xylenes	7400	Acute 1 hour health based guideline, based on mild respiratory effects and subjective symptoms of neurotoxicity in human volunteers from TCEQ evaluation (TCEQ 2013b).
1,3-Butadiene	660	Acute 1 hour health based guideline, based on developmental effects derived by the California Office of Environmental Health Hazard Assessment (OEHHA 2013). The guideline developed is lower than developed by TCEQ (TCEQ 2007) based on the same critical study.
Formaldehyde	50	Acute 1 hour health based guideline, based on eye and nose irritation in human volunteers from TCEQ evaluation (TCEQ 2014). This guideline is noted to be lower than the acute guideline available from the WHO (WHO 2000d, 2010) of 100 $\mu$ g/m <sup>3</sup> for formaldehyde.
Acetaldehyde	470	Acute 1 hour health based guideline, based on effects on sensory irritation, bronchoconstriction, eye redness and swelling derived by the California OEHHA (OEHHA 2013).

#### Table 6-5 Adopted acute inhalation based on protection of public health

Table 6-6 Adopted chronic guidelines and carcinogenic unit risk values based on protection of public health

Compound assessed	Chronic health based guideline (µg/m <sup>3</sup> )	Basis
Threshold guide	elines for vol	atile organic compounds
Benzene	30	The most significant chronic health effect associated with exposure to benzene is the increased risk of cancer, specifically leukaemia, which is assessed separately (below). The assessment of other health effects (other than cancer) has been undertaken using a chronic guideline derived by the USEPA (USEPA 2002a) based on haematological effects in an occupational inhalation study (converted to public health value using safety factors). This is the most current evaluation of effects associated with chronic inhalation exposure to toluene and is consistent with the value used to derive the NEPM (NEPC 1999 amended 2013b) health based guidelines.
Toluene	5000	Chronic guideline derived by the USEPA (USEPA 2005a) based on neurological effects in an occupational study (converted to public health value using safety factors). This is the most current evaluation of effects associated with chronic inhalation exposure to toluene and is consistent with the value used to derive the NEPM (NEPC 1999 amended 2013b) health based guidelines.
Xylenes	220	Chronic guideline derived by the Agency for Toxic Substances and Disease Register (ATSDR) (ATSDR 2007) based on mild subjective respiratory and neurological symptoms in an occupational study (converted to public health value using safety factors).
Formaldehyde	3.3	Formaldehyde is classified by IARC as carcinogenic to humans. The guideline developed by TCEQ (TCEQ 2013a) is derived on the basis of irritation of the eyes and airway discomfort in humans, with review of carcinogenic and other non-carcinogenic effects found to be adequately protected by this guideline. The guideline is more conservative than derived by the WHO (WHO 2010).
Acetaldehyde	9	Chronic guideline derived by the USEPA (USEPA IRIS) based on nasal effects (in a rat study) (converted to a public health value using safety factors). Value is more conservative that more recent evaluations from WHO and Californian OEHHA.
Threshold guide	elines for pol	ycyclic aromatic hydrocarbons
Naphthalene	3	Chronic guideline from USEPA (USEPA 1998) based on nasal effects (in a mice study) (converted to a public health value using safety factors) and is consistent with the value used to derive the NEPC (NEPC 1999 amended 2013b) health based guidelines.
Acenaphthylene	200#	These are the non-carcinogenic PAHs. Guideline available from the USEPA (USEPA IRIS). Chronic guidelines are based on criteria derived from oral studies (for critical effects on the liver, kidney and haematology) which are then converted to an inhalation value (relevant for the pretestion of public health including the use of eaferty factors) for use in this essentiated to be a statement of public health including the use of eaferty factors) for use in this essentiated to be a statement of public health including the use of eaferty factors) for use in this essentiated to be a statement of public health including the use of eaferty factors of public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of eaferty factors of the public health including the use of thealth including the use of the public health including the
Acenaphthene	200	The value presented in the above table has been converted from an acceptable dose in mg/kg/day to an acceptable air concentration assuming a body weight of 70 kg and inhalation
Fluorene	140	of 20 m³/day (as per (USEPA 2009a).
Phenanthrene	140#	# No guideline available for individual PAHs, hence a surrogate compound has been used for the purpose of assessment. The surrogate compound is a PAH of similar structure and toxicity. In relation to the surrogates adopted in this evaluation, acenaphthene has been
Anthracene	1000	adopted as a surrogate for acenaphthylene, fluoranthene has been adopted as a surrogate for phenanthrene.
Fluoranthene	140	
Pyrene	100	

Compound assessed	Chronic health based guideline (µg/m <sup>3</sup> )	Basis
Carcinogenic in	halation unit	risk values adopted for carcinogenic risk calculation
Benzene	6x10 <sup>-6</sup> (μg/m³)-1	Benzene is classified as a known human carcinogen by the International Agency for Research on Cancer (IARC). Inhalation unit risk value is from the WHO (WHO 2000d, 2010) and is based on excess risk of leukaemia from epidemiological studies.
1,3-Butadiene	5x10 <sup>-7</sup> (µg/m³) <sup>-1</sup>	1,3-Butadiene is classified as a known human carcinogen by the International Agency for Research on Cancer (IARC). Inhalation unit risk values are available from a number of agencies, including the WHO, USEPA and TCEQ. The most current evaluation has been undertaken by TCEQ (TCEQ 2013e). This has considered the same studies as WHO and USEPA, but included more recent studies and more relevant dose-response modelling.
Benzo(a)pyrene TEQ	0.087 (µg/m³)-1	BaP is classified by IARC as a known human carcinogen, which relates to BaP as well as all the other carcinogenic PAHs assessed as a BaP toxicity equivalent (TEQ) value. Inhalation unit risk value is from the WHO (WHO 2010) and is based on protection from lung cancer for an occupational study associated with coke oven emissions, which are very different from those from diesel emissions, and is expected to be conservative. It is noted that carcinogenic risks associated with lung cancer from diesel particulate matter (which is dominated by the presence of carcinogenic PAHs) is also assessed as outlined in section 6.9.5 and Annexure B).

**Table 6-7** to **Table 6-11** present a summary of the maximum predicted one hour or annual average concentrations of VOCs and PAHs assessed on the basis of a threshold with comparison against acute and chronic health based guidelines. The table also presents a Hazard Index (HI) which is the ratio of the maximum predicted concentration to the guideline. Each individual HI is added up to obtain a total HI for all the threshold VOCs and PAHs considered. The total HI is a sum of the potential hazards associated with all the threshold VOCs and PAHs together assuming the health effects are additive, and is evaluated as follows (enHealth 2012b):

- A total HI less than or equal to one means that all the maximum predicted concentrations are below the health based guidelines and there are no additive health impacts of concern
- A total HI greater than one means that the predicted concentrations (for at least one individual compound) are above the health based guidelines, or that there are at least a few individual VOCs or PAHs where the maximum predicted concentrations are close to the health based guidelines such that there is the potential for the presence of all these together (as a sum) to result in adverse health effects.

The assessment of acute exposures, presented in **Table 6-7** and **Table 6-8**, has compared the maximum predicted total (background plus existing roads and project) one-hour average concentration against the relevant acute guidelines. This is the maximum one-hour average concentration reported anywhere in the project area, regardless of land use.

The assessment of chronic exposures, presented in **Table 6-9** and **Table 6-10**, has compared the maximum predicted total annual average concentration relevant to residential land use against the relevant chronic guidelines. For exposures in other areas, **Table 6-9** and **Table 6-10** also presents the maximum calculated HI relevant to exposures in commercial/industrial areas, where the maximum change in VOC concentrations is predicted. The calculated HI takes into account that these exposures occur for eight hours per day over 240 days per year.

**Table 6-11** and **Table 6-12** presents a summary of the calculated incremental lifetime carcinogenic risk associated with exposure to the maximum predicted change in concentrations of benzene, 1,3-butadiene and carcinogenic PAHs (as benzo(a)pyrene TEQ) in residential areas. The calculation presented assumes residents are exposed to these pollutants all day, every day for a lifetime. The calculated carcinogenic risk for these compounds has been summed, in accordance with enHealth guidance where the following has been considered (enHealth 2012b). The tables present the impact due to tunnel ventilation along with the total changes due to the project (tunnel ventilation and road emissions). The table also presents the calculated total carcinogenic risk relevant to exposures in commercial/industrial areas, where the maximum change in VOCs and PAHs is predicted to occur. This calculated risks are considered in conjunction with what are considered negligible, tolerable/acceptable and unacceptable risks as outlined in Annexure C.

The values presented in the tables have been rounded to two significant figures for individual calculations and one significant figure for the total HI and total carcinogenic risk, reflecting the level of uncertainty in the calculations presented.

The following evaluation is based on the maximum predicted concentration in air for the relevant assessment scenarios for 2026 and 2036 as modelled in **Appendix E** (Air quality technical report). Concentrations in all other areas of the surrounding community are lower than the maximum as evaluated in this assessment. In many locations, the change due to the project is a lowering of VOC and PAH concentrations in air (ie a benefit).

Table 6-7 Assessment of acute exposures to VOCs - maximum impacts in community associatded with project: 2026

Key VOC	Maximum predicted 1 hour average concentration associated with project (background plus project) and calculated HI						
	2026: Without project		2026: With project				
	Maximum concentration (µg/m <sup>3</sup> )	HI	Maximum concentration (µg/m <sup>3</sup> )	HI			
Benzene	9.7	0.017	7.7	0.013			
Toluene	17.8	0.0012	14.0	0.00093			
Xylenes	14.6	0.0020	11.5	0.0016			
1,3-Butadiene	2.6	0.0039	2.0	0.0031			
Formaldehyde	8.0	0.16	6.3	0.13			
Acetaldehyde	3.8	0.0082	3.0	0.0064			
	Total HI	0.19	(	).15			

Table 6-8: Assessment of acute exposures to VOCs - maximum impacts in community associated with project: 2036

Key VOC	Maximum predicted 1 hour average concentration associated with project (background plus project) and calculated HI									
	2036: Without proj	ect	2036: With project		2036: Cumulative					
	Maximum concentration (µg/m <sup>3</sup> )	HI	Maximum concentration (µg/m <sup>3</sup> )	HI	Maximum concentration (µg/m <sup>3</sup> )	HI				
Benzene	5.4	0.0093	4.6	0.0080	5.0	0.0086				
Toluene	9.4	0.00062	8.1	0.00054	8.7	0.00058				
Xylenes	7.7	0.0010	6.7	0.0009	7.2	0.0010				
1,3-Butadiene	1.5	0.0022	1.3	0.0019	1.4	0.0021				
Formaldehyde	7.0	0.14	6.0	0.12	6.5	0.13				
Acetaldehyde	3.1	0.0066	2.7	0.0057	2.9	0.0061				
	Total HI	0.16		0.14		0.15				

Table 6-9: Assessment of chronic exposures to VOCs and PAHs - maximum impacts in community associated with project 2026

Key VOCs and PAHs	Maximum predicted annual average concentration associated with project (background plus project) and calculated HI – Residential exposures							
	2026: Without project		2026: With project					
	Max concentration (µg/m <sup>3</sup> )	HI	Max concentration (µg/m <sup>3</sup> )	HI				
Benzene	0.51	0.017	0.48	0.016				
Toluene	0.92	0.0002	0.87	0.0002				
Xylenes	0.76	0.003	0.72	0.003				
Formaldehyde	0.42	0.13	0.40	0.12				
Acetaldehyde	0.20	0.022	0.19	0.021				
Naphthalene	0.069	0.023	0.065	0.022				
Acenaphthylene	0.0048	2.4 x10 <sup>-5</sup>	0.0046	2.3 x10 <sup>-5</sup>				
Acenaphthene	0.0020	9.8 x10 <sup>-6</sup>	0.00186	9.3 x10 <sup>-6</sup>				
Fluorene	0.0049	3.5 x10 <sup>-5</sup>	0.0047	3.3 x10 <sup>-5</sup>				
Phenanthrene	0.0033	2.4 x10 <sup>-5</sup>	0.0032	2.3 x10 <sup>-5</sup>				
Anthracene	0.00048	4.8 x10 <sup>-7</sup>	0.00046	4.6 x10 <sup>-7</sup>				
Fluoranthene	0.00044	3.2 x10 <sup>-6</sup>	0.00042	3.0 x10 <sup>-6</sup>				
Pyrene	0.00070	7.0 x10 <sup>-6</sup>	0.00066	6.6 x10 <sup>-6</sup>				
	Total HI – Residential	0.18		0.17				
Max	Max HI – Commercial/Industrial 0.039 0.036							

Table 6-	10: /	Assessment	of	chronic	exposures	to	VOCs	and	PAHs	-	maximum	impacts	in
commun	ity a	ssociated wi	th p	project: 2	036							-	

Key VOCs and PAHs	Maximum predicted annual average concentration associated with pro (background plus project) and calculated HI – Residential exposures					ject
	2036: Do minimal		2036: With project		2036: Cumulative	
	Max concentration (µg/m <sup>3</sup> )	HI	Max concentration (µg/m <sup>3</sup> )	HI	Max concentration (µg/m <sup>3</sup> )	HI
Benzene	0.33	0.011	0.33	0.011	0.32	0.011
Toluene	0.58	0.0001	0.57	0.00011	0.56	0.00011
Xylenes	0.48	0.002	0.47	0.002	0.46	0.002
Formaldehyde	0.43	0.13	0.42	0.13	0.42	0.13
Acetaldehyde	0.19	0.0212	0.19	0.0209	0.18	0.020
Naphthalene	0.063	0.021	0.062	0.021	0.061	0.020
Acenaphthylene	0.0044	2.2 x10⁻⁵	0.0043	2.2 x10 <sup>-5</sup>	0.0042	2.1 x10 <sup>-5</sup>
Acenaphthene	0.0018	9.0 x10⁻ <sup>6</sup>	0.0018	8.8 x10 <sup>-6</sup>	0.0017	8.7 x10 <sup>-6</sup>
Fluorene	0.0045	3.2 x10⁻⁵	0.0044	3.2 x10 <sup>-5</sup>	0.0043	3.1 x10⁻⁵
Phenanthrene	0.0030	2.2 x10⁻⁵	0.0030	2.1 x10 <sup>-5</sup>	0.0029	2.1 x10 <sup>-5</sup>
Anthracene	0.00044	4.4 x10 <sup>-7</sup>	0.00043	4.3 x10 <sup>-7</sup>	0.00042	4.2 x10 <sup>-7</sup>
Fluoranthene	0.00040	2.9 x10 <sup>-6</sup>	0.00040	2.8 x10 <sup>-6</sup>	0.00039	2.8 x10 <sup>-6</sup>
Pyrene	0.00064	6.4 x10 <sup>-6</sup>	0.00063	6.3 x10 <sup>-6</sup>	0.00061	6.1 x10 <sup>-6</sup>
Тс	otal HI – Residential	0.17		0.17		0.17
Max HI – Co	mmercial/Industrial	0.038		0.038		0.037

# Table 6-11 Assessment of incremental lifetime carcinogenic risk – maximum impacts in community associated with project: 2026

Key VOC	Maximum predicted change in annual average associated with project and cancer risk – Resid	concentration dential			
	2026: With project				
	Maximum concentration (µg/m <sup>3</sup> )	ILCR			
Changes due to tunnel ventil	ation				
Benzene	0.0026	6 x 10 <sup>-9</sup>			
1,3-Butadiene	0.00069	1 x 10 <sup>-10</sup>			
Benzo(a)pyrene TEQ	0.000023	8 x 10 <sup>-7</sup>			
	Total carcinogenic risk – Residential	8 x 10 <sup>-7</sup>			
	Maximum carcinogenic risk – Commercial/Industrial	2 x 10 <sup>-7</sup>			
Total changes due to project	(tunnel ventilation and road emissions)				
Benzene	0.061	2 x 10 <sup>-7</sup>			
1,3-Butadiene	0.0162	3 x 10 <sup>-9</sup>			
Benzo(a)pyrene TEQ	0.00054	2 x 10 <sup>-5</sup>			
	Total carcinogenic risk – Residential	2 x 10 <sup>-5</sup>			
Maximum carcinogenic risk – Commercial/Industrial 4 x 10-6					

Note: ILCR = incremental lifetime carcinogenic risk (refer to Annexure B for calculation methodology and Table 5-5 for inhalation unit risk values)

# Table 6-12 Assessment of incremental lifetime carcinogenic risk – maximum impacts in community associated with project: 2036

Key VOC	Maximum predicted change in annual average concentration associated w project and cancer risk – Residential							
	2036: With project		2036: Cumulative					
	Maximum concentration (µg/m³)	ILCR	Maximum concentration (µg/m <sup>3</sup> )	ILCR				
Changes due to tunn	el ventilation							
Benzene	0.0021	5 x 10-9	0.0024	6 x 10 <sup>-9</sup>				
1,3-Butadiene	0.00057	1 x 10 <sup>-10</sup>	0.00067	1 x 10 <sup>-10</sup>				
Benzo(a)pyrene TEQ	0.000026	9 x 10 <sup>-7</sup>	0.000030	1 x 10 <sup>-6</sup>				
Total ca	rcinogenic risk – Residential	9 x 10 <sup>-7</sup>	1 x 10 <sup>-6</sup>					
٨	/aximum carcinogenic risk – Commercial/Industrial	2 x 10 <sup>-7</sup>		2 x 10 <sup>-7</sup>				
Total changes due to	project (tunnel ventilation and	d road emissions)						
Benzene	0.044	1 x 10 <sup>-7</sup>	0.052	1 x 10 <sup>-7</sup>				
1,3-Butadiene	0.012	2 x 10 <sup>-9</sup>	0.014	3 x 10 <sup>-9</sup>				
Benzo(a)pyrene 0.00055 TEQ		2 x 10 <sup>-5</sup>	0.00065	2 x 10 <sup>-5</sup>				
Total ca	rcinogenic risk – Residential	2 x 10 <sup>-5</sup>	2 x 10 <sup>-5</sup>					
Λ	/aximum carcinogenic risk – Commercial/Industrial	4 x 10 <sup>-6</sup>	5 x 10 <sup>-6</sup>					

Note: ILCR = incremental lifetime carcinogenic risk (refer to Annexure B for calculation methodology and Table 5-5 for inhalation unit risk values)

For the assessment of acute exposures to VOCs (**Table 6-7** and **Table 6-8**), the calculated HI associated with exposure to the maximum concentrations predicted is less than one for 2026, 2036 and the cumulative scenario. On this basis, there are no acute risk issues in the local community associated with the project.

For the assessment of chronic exposures to VOCs and PAHs (**Table 6-9** to **Table 6-12**), the calculated HI associated with exposure to the maximum concentrations predicted is less than or equal to one for 2026, 2036 and the cumulative scenario. The calculated lifetime cancer risks associated with the maximum change in benzene, 1,3-butadiene and carcinogenic PAHs (as benzo(a)pyrene TEQ) are less than or equal to  $2x10^{-5}$  and are considered to be tolerable. It is noted that the calculations undertaken for PAHs is based on a conservative estimate of the fraction of emissions from vehicles that comprises PAHs (as a percentage of total VOCs). The approach adopted is expected to be a conservative upper limit estimate.

On this basis, there are no chronic risk issues in the local community associated with the project.

When comparing the calculated risks due to tunnel ventilation outlets versus the total changes due to the project (tunnel ventilation outlets and road emissions) (**Table 6-11** and **Table 6-12**), the risks are approximately 2 to 100 times lower for the tunnel ventilation changes outlets only. This means that the road emissions are driving the cancer risk for VOC and PAH exposures for the project in most cases.

# 6.7 Assessment of carbon monoxide

Motor vehicles are the dominant source of carbon monoxide in air (DECCW 2009). Adverse health effects of exposure to carbon monoxide are linked with carboxyhaemoglobin (COHb) in blood. In addition, association between exposure to carbon monoxide and cardiovascular hospital admissions and mortality, especially in the elderly for cardiac failure, myocardial infarction and ischemic heart disease, and some birth outcomes (such as low birth weights) have been identified (NEPC 2010).

Guidelines are available in Australia from NEPC (NEPC 2003) and NSW EPA that are based on the protection of adverse health effects associated with carbon monoxide. Review of these guidelines by NEPC (2010) identified additional supporting studies<sup>2</sup> for the evaluation of potential adverse health effects and indicated that these should be considered in the current review of the National Ambient Air Quality NEPM (no interim or finalisation date available). The air guidelines currently available from NEPC are consistent with health based guidelines currently available from the WHO (2005) and the USEPA (2011)<sup>3</sup>, specifically listed to be protective of exposures by sensitive populations including asthmatics, children and the elderly). On this basis, the current NEPC guidelines are considered appropriate for the assessment of potential health impacts associated with the project.

The NEPC ambient air quality guideline for the assessment of exposures to carbon monoxide has considered lowest observed adverse effect level (LOAEL) and no observed adverse effect level (NOAEL) associated with a range of health effects in healthy adults, people with ischemic heart disease and foetal effects. In relation to these data, a guideline level of carbon monoxide of nine parts per million (ppm) by volume (or ten milligrams per cubic metre or 10,000 micrograms per cubic metre) over an eight-hour period was considered to provide protection (for both acute and chronic health effects) for most members of the population. An additional 1.5-fold uncertainty factor to protect more susceptible groups in the population was included. On this basis, the NEPC (and the USEPA) guideline is protective of adverse health effects in all individuals, including sensitive individuals.

The NSW EPA has also established a guideline for 15-minute average (100 milligrams per cubic metre) and one-hour average (30 milligrams per cubic metre) concentrations of carbon monoxide in ambient air. These guidelines are based on criteria established by the WHO (WHO 2000c) using the same data used by the NEPC to establish the guideline (above) with extrapolation to different periods of exposure on the basis of known physiological variables that affect carbon monoxide uptake.

**Table 6-13** presents a summary of the maximum predicted cumulative one-hour average and eighthour average concentrations of carbon monoxide for the assessment years 2026 and 2036, without the project, with the project and for the cumulative scenario.

Scenario	Maximum concentratio	1-hour n of CO (mg	average /m³)	Maximum 8 hour average concentration of CO (mg/m³)			
	Without project	With project	Cumulative	Without project	With project	Cumulative	
2026							
Maximum	5.3	5.3		3.7	3.7		
2036							
Maximum	5.0	4.7	4.8	3.5	3.3	3.3	
Relevant health based guideline		30			10		

Table 6-13 Review of potential acute and chronic health impacts – carbon monoxide (CO)

<sup>2</sup> Many of the more current studies are epidemiology studies that relate to a mix of urban air pollutants (including particulate matter) where it is more complex to determine the effects that can be attributed to carbon monoxide exposure only.

<sup>3</sup> Most recent review of the Primary National Ambient Air Quality Standards for Carbon Monoxide published by the USEPA in the Federal Register Volume 76, No. 169, 2011, available from: <u>http://www.gpo.gov/fdsys/pkg/FR-2011-08-31/html/2011-21359.htm</u>.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

All the concentrations of carbon monoxide presented in the above table are below the relevant health based guidelines. On the basis of the assessment undertaken there are no adverse health effects expected in relation to exposures (acute and chronic) to carbon monoxide in the local area surrounding the project footprint.

# 6.8 Assessment of nitrogen dioxide

#### 6.8.1 Approach

Nitrogen oxides (NOx) refers to nitrogen oxide and nitrogen dioxide, which are highly reactive gases containing nitrogen and oxygen. Nitrogen oxide gases form when fuel is burnt. Motor vehicles, along with industrial, commercial and residential (eg gas heating or cooking) combustion sources, are primary producers of nitrogen oxides.

In Sydney, the OEH (2012) estimated that on-road vehicles account for about 62 per cent of emissions of nitrogen oxides, industrial facilities account for 12 per cent, other mobile sources account for about 22 per cent, with the remainder from domestic/commercial sources.

In terms of health effects, nitrogen dioxide is the only oxide of nitrogen that may be of concern (WHO 2000a). Nitrogen dioxide can cause inflammation of the respiratory system and increase susceptibility to respiratory infection. Exposure to elevated levels of nitrogen dioxide has also been associated with increased mortality, particularly related to respiratory disease, and with increased hospital admissions for asthma and heart disease patients (WHO 2013a). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of nitrogen dioxide (Morgan et al. 2013; NEPC 2010). The health effects associated with exposure to nitrogen dioxide depend on the duration of exposure as well as the concentration.

Guidelines are available from the NSW EPA and NEPC (NEPC 2003) which indicate acceptable concentrations of nitrogen dioxide. These guidelines are based on protection from adverse health effects following both short term (acute) and longer term (chronic) exposure for all members of the population including sensitive populations like asthmatics, children and the elderly. Recently these guidelines have been reviewed by NEPC (Golder 2013; NEPC 2010, 2014). The review identified additional supporting studies for the evaluation of potential adverse health effects. The reviews undertaken to date have not recommended any change to the existing health based guidelines.

When reviewing the available literature on the health effects associated with exposure to nitrogen dioxide it is important to consider the following:

- Whether the evidence suggests that associations between exposure to nitrogen dioxide concentrations and effects on health are causal. The most current review undertaken by the USEPA (USEPA 2015) specifically evaluated evidence of causation. The review identified that a causal relationship existed for respiratory effects (for short term exposure with long term exposures also likely to be causal). All other associations related to exposure to nitrogen dioxide (specifically cardiovascular effects, mortality and cancer) were considered to be suggestive
- Whether the reported associations are distinct from, and additional to, those reported and assessed for exposure to particulate matter. Co-exposures to nitrogen dioxide and particulate matter complicates review and assessment of many of the epidemiology studies as both these air pollutants occur together in urban areas. There is sufficient evidence (epidemiological and mechanistic) to suggest that some of the health effect associations identified relate to exposure to nitrogen dioxide after adjustment/correction for co-exposures with particulate matter (COMEAP 2015)
- Whether the assessment of potential health effects associated with exposure to different levels of nitrogen dioxide can be undertaken on the basis of existing guidelines, or whether specific risk calculations are required to be undertaken. The current guidelines in Australia for the assessment of nitrogen dioxide in air relate to cumulative (total) exposures, and adopt criteria that are considered to be protective of short and long term exposures. Hence, it is relevant that these guidelines be considered in this assessment

In addition, it is noted that in areas of high traffic congestion (as is the case with the project area evaluated in this assessment) background levels of nitrogen dioxide may already be elevated such that use of the existing guideline is limited for the purpose of assessing health impacts from a particular project or activity. For these situations, it is relevant to also evaluate the impact on community health of the change in nitrogen dioxide concentration in the local community using appropriate risk calculations. For the conduct of risk assessments in relation to exposure to nitrogen dioxide, the WHO (WHO 2013a) identified that the strongest evidence of health effects related to respiratory hospitalisations and to a lesser extent mortality (associated with short term exposures) and recommend that these health endpoints should be considered in any core assessment of health impacts associated with exposure.

On the basis of the above, potential health effects associated with exposure to nitrogen dioxide would be undertaken for this project using both comparison with guidelines (assessing total exposures) and an assessment of incremental impacts on health (associated with changes in air quality from the project).

#### 6.8.2 Assessment of total exposures

#### Assessment of acute exposures

The NEPC ambient air quality guideline for the assessment of acute (short term) exposures to nitrogen dioxide relates to the maximum predicted total (cumulative) one-hour average concentration in air. The guideline of 246 micrograms per cubic metre (or 120 parts per billion by volume) is based on a LOAEL of 409–613 micrograms per cubic metre derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of two to protect susceptible people (ie asthmatic children) was applied to the LOAEL (NEPC 1998). On this basis, the NEPC (and Environment Protection Authority) acute guideline is protective of adverse health effects in all individuals, including sensitive individuals.

**Table 6-14** presents a summary of the maximum predicted cumulative one-hour average concentration of nitrogen dioxide the modelled scenarios.

Location and	Maximum 1-hour average concentration of NO₂ (μg/m³)						
Scenano	Without the project	With the project	Cumulative				
2027							
Maximum	348.5	307.9					
2037							
Maximum	375.1	334.9	321.5				
Acute health based guideline	246	246	246				

#### Table 6-14 Review of potential acute health impacts – nitrogen dioxide (NO<sub>2</sub>)

The maximum cumulative concentrations of nitrogen dioxide presented in the above table exceed the acute NEPC guideline of 246 micrograms per cubic metre for all the scenarios, with and without the project. The elevated levels listed above are not considered to be representative of exposure concentrations that would occur within the study area. This is due to the combined effect of the approach adopted for converting NOx to nitrogen dioxide (that overestimates short-term one-hour average concentrations), and the use of a contemporaneous assessment of background and project impacts. The contemporaneous approach assumes that the highest background concentrations may occur during the same hour as the maximum incremental change from the project. This results in a very high estimate of total nitrogen dioxide concentrations that is not likely to ever occur (refer to **Appendix E** (Air quality technical report) for more detailed discussion). As a result, the magnitude of the maximum total concentrations reported for nitrogen dioxide over a one-hour average cannot be used to evaluate the potential for adverse health effects in the community.

As assessment of total concentrations of nitrogen dioxide cannot be used to determine the potential for adverse health impacts in the community, and because there is no clear threshold established for community exposures to nitrogen dioxide, the assessment of incremental exposures is of most relevance. This assessment is presented in **Section 6.8.3**.

#### Assessment of chronic exposures

The NEPC ambient air quality guideline for the assessment of chronic (long term) exposures to nitrogen dioxide relates to the maximum predicted total (cumulative) annual average concentration in air. The guideline of 62 micrograms per cubic metre (or 30 ppbv [parts per billion by volume]) is based on a LOAEL of the order of 40–80 parts per billion by volume (around 75–150 micrograms per cubic metre) during early and middle childhood years which can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent 'colds', a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school. An uncertainty factor of two was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 parts per billion by volume (38–75 micrograms per cubic metre) (NEPC 1998). On this basis, the NEPC (and OEH) chronic guideline is protective of adverse health effects in all individuals, including sensitive individuals. **Table 6-15** presents a summary of the maximum predicted cumulative annual average concentration of nitrogen dioxide for the modelled scenarios.

Location ar	nd Maximum annual av	Maximum annual average concentration of NO2 (µg/m3)						
scenario	Without the project	With the project	Cumulative					
2027								
Maximum	42.5	40.7						
2037								
Maximum	44.8	42.7	42.2					
Chronic health based guideline	62							

#### Table 6-15 Review of potential chronic health impacts – Nitrogen dioxide (NO<sub>2</sub>)

All the concentrations of nitrogen dioxide presented in the above table are below the chronic NEPC guideline of 62 micrograms per cubic metre. In addition, the concentrations of nitrogen dioxide are lower with the project (in both assessment years) and for the cumulative scenario. Hence there are no adverse health effects expected in relation to chronic exposures to nitrogen dioxide in the local area surrounding the project.

## 6.8.3 Assessment of incremental exposures

The evidence base supports quantification of effects of short term exposure to nitrogen dioxide, using the averaging time as in the relevant studies. The strongest evidence is for respiratory effects, in particular exacerbation of asthma, with some support also for all-cause mortality. These health endpoints have been evaluated in relation to changes in nitrogen dioxide concentrations in air associated with the project within the local community in 2026 and 2036.

The approach adopted for the assessment of incremental exposures is consistent with that adopted for particulates as outlined in **section 6.9.5**. This involves the calculation of a change in individual risk, as well as the change in incidence, or the number of cases, that occur in the community as a result of the project.

**Table 6-16** presents a summary of the health endpoints considered in this assessment, the  $\beta$  coefficient relevant to the calculation of a relative risk (refer to Annexure A for details on the calculation of a  $\beta$  coefficient from published studies). The coefficients adopted for the assessment of impacts on mortality and asthma emergency department admissions are derived from the detailed assessment undertaken for the current review of health impacts of air pollution undertaken by NEPC (Golder 2013) and are considered to be robust.

Table 6-16 Adopted exposure-responses relationships for assessment of changes in nitrogen dioxide concentrations

Health endpoint	Exposure period	Age group	$\begin{array}{lll} Adopted & \beta \\ coefficient (also \\ as per cent) for \\ 1 \ \mu g/m^3 \ increase \\ in \ NO_2 \end{array}$	Reference
Mortality, all causes (non- trauma)	Short term	All ages*	0.00188 (0.19%)	Relationship derived for from modelling undertaken for 5 cities in Australia and 1 day lag (EPHC 2010; Golder 2013)
Mortality, respiratory	Short term	All ages*	0.00426 (0.43%)	Relationship derived for from modelling undertaken for 5 cities in Australia and 1 day lag (EPHC 2010; Golder 2013)
Asthma emergency department (ED) admissions	Short term	1–14 years	0.00115 (0.11%)	Relationship established from review conducted on Australian children (Sydney) for the period 1997 to 2001 (Golder 2013; Jalaludin et al. 2008)

Note: \* Relationships established for all ages, including young children and the elderly

It is noted that while the maximum concentrations of nitrogen dioxide are lower in the local community with the operation of the project, the concentrations at individual receptors vary. While the concentrations at most receptors decrease with the operation of the project, there are some receptors where there is an increase, associated with the redistribution of emissions from vehicles using surface roads.

**Table 6-17** presents the change in individual risk associated with changes in nitrogen dioxide at the maximum impacted receptors relevant to the various land use in the community, as well as the community receptors, for the operational years 2026 and 2036, including the cumulative scenario (refer to Annexure A for methodology for the calculation of individual risks). The assessment assumes an individual is exposed at each maximum impacted location over all hours of the day, regardless of the land use. This has been undertaken to address any future changes in land use that may occur. Risks for all other receptors (including other sensitive receptors) are lower than the maximums presented.

All risks are presented to one significant figure, reflecting the level of uncertainty associated with the calculations presented.

Figure 6-5 presents a summary of the calculated change in individual risk associated with changes in nitrogen dioxide concentrations at each community receptor location evaluated.

Annexure C presents a discussion on levels of the levels of risk that are considered to be negligible, tolerable/acceptable and unacceptable. A summary of these risk levels is included in **Table 6-17**.

Calculations relevant to the characterisation of risks associated with changes in nitrogen dioxide concentrations in the community are presented in Annexure D.

**Table 6-18** present a summary of the calculated change in incidence of the relevant health effects for the population living in the LGAs within the study area, associated with changes in nitrogen dioxide concentrations for 2026 and 2036. All calculations relevant to the LGAs, including calculation for each individual suburb considered in the LGAs, are presented in Annexure E.

Scenario and receptor	Maximum change in individual risk from short term exposure to nitrogen dioxide for the following health endpoints								
	Mortality: Al ages)	l causes (all	Mortality: Re ages)	spiratory (all	Asthma ED A 14 years)	dmissions (1–			
	Ventilation only	Roads and ventilation	Ventilation only	Roads and ventilation	Ventilation only	Roads and ventilation			
2026 – with project									
Maximum residential	8 x 10 <sup>-8</sup>	2 x 10 <sup>-5</sup>	2 x 10 <sup>-8</sup>	3 x 10 <sup>-6</sup>	1 x 10 <sup>-7</sup>	2 x 10 <sup>-5</sup>			
Maximum workplace	1 x 10 <sup>-7</sup>	1 x 10 <sup>-5</sup>	2 x 10 <sup>-8</sup>	2 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	2 x 10 <sup>-5</sup>			
Maximum childcare and schools	2 x 10 <sup>-8</sup>	7 x 10⁻	3 x 10 <sup>-9</sup>	1 x 10 <sup>-6</sup>	3 x 10 <sup>-8</sup>	1 x 10 <sup>-5</sup>			
Maximum aged care	4 x 10 <sup>-9</sup>	4 x 10 <sup>-6</sup>	7 x 10 <sup>-10</sup>	7 x 10 <sup>-7</sup>	6 x 10 <sup>.9</sup>	5 x 10 <sup>-6</sup>			
Maximum hospitals/medical	6 x 10 <sup>-9</sup>	2 x 10 <sup>-6</sup>	1 x 10 <sup>-9</sup>	4 x 10 <sup>-7</sup>	9 x 10 <sup>.9</sup>	3 x 10 <sup>-6</sup>			
Maximum open space	3 x 10 <sup>-8</sup>	4 x 10 <sup>-6</sup>	5 x 10 <sup>-9</sup>	7 x 10 <sup>-7</sup>	4 x 10 <sup>-8</sup>	5 x 10⁻ <sup>6</sup>			
Maximum from all receptors	1 x 10 <sup>-7</sup>	2 x 10 <sup>-5</sup>	2 x 10 <sup>-8</sup>	3 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	2 x 10 <sup>-5</sup>			
Maximum from sensitive receptors	1 x 10-6	4 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	7 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>	6 x 10 <sup>-6</sup>			
2036 – with project									
Maximum residential	2 x 10 <sup>-6</sup>	1 x 10 <sup>-5</sup>	4 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>	3 x 10⁻	2 x 10 <sup>-5</sup>			
Maximum workplace	2 x 10 <sup>-6</sup>	1 x 10 <sup>-5</sup>	3 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>	2 x 10 <sup>-6</sup>	2 x 10 <sup>-5</sup>			
Maximum childcare and schools	1 x 10 <sup>-6</sup>	6 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	1 x 10-6	2 x 10 <sup>-6</sup>	9 x 10 <sup>-6</sup>			
Maximum aged care	7 x 10 <sup>-7</sup>	3 x 10-6	1 x 10 <sup>-7</sup>	5 x 10 <sup>-7</sup>	1 x 10 <sup>-6</sup>	4 x 10-6			
Maximum hospitals/medical	8 x 10 <sup>-7</sup>	4 x 10 <sup>-6</sup>	1 x 10 <sup>-7</sup>	7 x 10 <sup>-7</sup>	1 x 10 <sup>-6</sup>	6 x 10-6			
Maximum open space	9 x 10 <sup>-7</sup>	5 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	1 x 10 <sup>-6</sup>	1 x 10 <sup>-6</sup>	8 x 10-6			
Maximum from all receptors	2 x 10 <sup>-6</sup>	1 x 10 <sup>-5</sup>	4 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>	3 x 10 <sup>-6</sup>	2 x 10 <sup>-5</sup>			
Maximum from sensitive receptors	1 x 10 <sup>-6</sup>	4 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	7 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>	5 x 10-₀			
2036 – cumulative									
Maximum residential	1 x 10-6	9 x 10 <sup>-6</sup>	3 x 10-7	2 x 10 <sup>-6</sup>	2 x 10-6	1 x 10 <sup>-5</sup>			
Maximum workplace	1 x 10 <sup>-6</sup>	1 x 10 <sup>-5</sup>	2 x 10 <sup>-7</sup>	3 x 10-6	2 x 10 <sup>-6</sup>	2 x 10 <sup>-5</sup>			
Maximum childcare	1 x 10 <sup>-6</sup>	7 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	1 x 10-6	2 x 10 <sup>-6</sup>	1 x 10 <sup>-5</sup>			
Maximum aged care	7 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>	1 x 10 <sup>-7</sup>	4 x 10 <sup>-7</sup>	1 x 10-6	3 x 10-6			
Maximum hospitals/medical	7 x 10 <sup>-7</sup>	9 x 10 <sup>-7</sup>	1 x 10 <sup>-7</sup>	2 x 10 <sup>-7</sup>	1 x 10 <sup>-6</sup>	1 x 10 <sup>-6</sup>			
Maximum open space	1 x 10 <sup>-6</sup>	6 x 10 <sup>-6</sup>	2 x 10 <sup>-7</sup>	1 x 10 <sup>-6</sup>	2 x 10 <sup>-6</sup>	9 x 10 <sup>-6</sup>			
Maximum from all receptors	1 x 10 <sup>-6</sup>	2 x 10 <sup>-5</sup>	3 x 10 <sup>-7</sup>	3 x 10-6	2 x 10 <sup>-6</sup>	2 x 10 <sup>-5</sup>			
Maximum from sensitive receptors	1 x 10-6	3 x 10-6	2 x 10 <sup>-7</sup>	5 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>	4 x 10 <sup>-6</sup>			
Negligible risks			<1	x 10 <sup>-6</sup>					
Tolerable/acceptable risks			≥1 x 10-6	and ≤1 x 10 <sup>-4</sup>					
Unacceptable risks	>1 x 10 <sup>-4</sup>								

Table 6-17 Maximum calculated risks associated with short term exposure to changes in nitrogen dioxide concentrations with operation of the project



Figure 6-5 Change in calculated risk for key health endpoints associated with total changes in nitrogen dioxide concentrations at community receptors (2026 and 2036)

LGA	Change in population incidence – number of cases									
		2026			2036					
	Mortality – All Causes	Mortality – Respiratory	Morbidity – Asthma ED Admissions	Mortality – All Causes	Mortality – Respiratory	Morbidity – Asthma ED Admissions				
	All ages	All ages	1-14 years	All ages	All ages	1–14 years				
With Project										
Strathfield - Burwood - Ashfield LGA	-0.00026	-0.000050	-0.000078	-0.00011	-0.000022	-0.000034				
Sydney Inner City LGA	-0.000057	-0.000010	-0.0000049	-0.00078	-0.00014	-0.000067				
Marrickville - Sydenham - Petersham LGA	-0.00093	-0.00016	-0.00018	-0.0013	-0.00023	-0.00026				
Canterbury LGA	-0.0000089	-0.0000016	-0.0000026	-0.00018	-0.000034	-0.000053				
Botany LGA	-0.0024	-0.00041	-0.00053	-0.0041	-0.00071	-0.00091				
Kogarah - Rockdale LGA	0.0011	0.00018	0.00021	0.00030	0.000051	0.000060				
Hurstville LGA	0.000024	0.0000041	0.0000049	0.000031	0.0000053	0.0000063				
Total for all LGAs	-0.0026	-0.00045	-0.00058	-0.0062	-0.0011	-0.0013				
Cumulative										
Strathfield - Burwood - Ashfield LGA				-0.000061	-0.000012	-0.000018				
Sydney Inner City LGA				-0.00063	-0.00011	-0.000054				
Marrickville - Sydenham - Petersham LGA				-0.00018	-0.000031	-0.000035				
Canterbury LGA				-0.000043	-0.000080	-0.000013				
Botany LGA				-0.0033	-0.00057	-0.00073				
Kogarah - Rockdale LGA				-0.0056	-0.00094	-0.0011				
Hurstville LGA				-0.0000052	-0.00000088	-0.0000011				
Total for all LGAs				-0.0098	-0.0017	-0.0020				

Table 6-18 Calculated changes in incidence of health effects in population associated with changes in NO<sub>2</sub> concentrations

Negative value indicates that there is a decrease in incidence associated with the project

Review of the individual risks calculated for changes in nitrogen dioxide levels associated with the F6 Extension Phase 1, indicates the following:

- The maximum risks calculated for exposures in residential areas are less than 1x10<sup>-4</sup> and are therefore considered to be tolerable/acceptable
- The maximum risks calculated for exposures in commercial/industrial areas are less than 1x10<sup>-4</sup> and are therefore considered to be tolerable/acceptable
- All maximum risks calculated for continuous exposures in childcare centres, schools, aged care homes and open space areas are below 1x10<sup>-4</sup> and considered to be tolerable/acceptable
- All risks calculated for exposures at community receptors are below 1x10<sup>-4</sup> and considered to be tolerable/acceptable. It is noted that for most community receptors the impact of the project is a lowering of risk (negative risk values presented in Figure 6-4).
- When comparing the maximum risk from the ventilation outlets only compared to the risks from roads and ventilation outlets (**Table 6-17**) the risk is dominated by the road component in most cases.

Review of the calculated impacts in terms of the change in incidence of the relevant health effects associated with exposure to nitrogen dioxide in the community, indicates the following:

- The total change in the number of cases relevant to the health effects evaluated, for both 2026 and 2036 is negative, meaning a decrease in incidence as a result of the project. The number of cases, however is small, with a decrease of less than one case. These changes would not be measurable within the community
- Most individual LGAs show a total decrease in health incidence. There are a two LGAs (Kogarah
   – Rockdale and Hurstville) where there is an increase. These increases and decreases are also
   small and as a result these changes would not be measurable in the community
- The incidence calculations presented in **Table 6-18** are the totals for each LGA. Within these LGAs are a number of smaller suburbs. The calculated change in incidence relevant to each of these suburbs has also been evaluated, as presented in Annexure E. Review of the incidence calculated for the individual suburbs indicates that these predominantly relate to small decreases in health incidence with some suburbs showing an increase. There are no individual suburbs within the LGAs where there is a change incidence that is of significance or would be measurable.

# 6.9 Assessment of particulate matter

#### 6.9.1 Particle size

Particulate matter is a widespread air pollutant with a mixture of physical and chemical characteristics that vary by location (and source). Unlike many other pollutants, particulate matter includes a broad class of diverse materials and substances, with varying morphological, chemical, physical and thermodynamic properties, with sizes that vary from less than 0.005 micrometres (or microns) to greater than 100 microns. Particles can be derived from natural sources such as crustal dust (soil), pollen and moulds, and other sources that include combustion and industrial processes. Secondary particulate matter is formed via atmospheric reactions of primary gaseous emissions. The gases that are the most significant contributors to secondary particulates include nitrogen oxides, ammonia, sulfur oxides, and certain organic gases (derived from vehicle exhaust, combustion sources, agricultural, industrial and biogenic emissions).

Numerous epidemiological studies<sup>4</sup> have reported significant positive associations between particulate air pollution and adverse health outcomes, in particular mortality as well as a range of adverse cardiovascular and respiratory effects.

<sup>4</sup> Epidemiology is the study of diseases in populations. Epidemiological evidence can only show that this risk factor is associated (correlated) with a higher incidence of disease in the population exposed to that risk factor. The higher the correlation the more certain the association. Causation (ie that a specific risk factor actually causes a disease) cannot be proven with only epidemiological studies. For causation to be determined a range of other studies need to be considered in conjunction with the epidemiology studies.

The potential for particulate matter to result in adverse health effects is dependent on the size and composition of the particulate matter. The common measures of particulate matter that are considered in the assessment of air quality and health risks are:

- Total suspended particulates (TSP): This refers to all particulate matter with an equivalent aerodynamic particle<sup>5</sup> size generally below 50 to 100 microns in diameter<sup>6</sup>. It is a fairly gross indicator of the presence of dust with a wide range of sizes. Larger particles (termed 'inspirable', comprise particles around 10 microns and larger) are of less concern and more of a nuisance as they would deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory system<sup>7</sup> and do not reach the lungs. Smaller particles (smaller than 10 microns, termed 'respirable') tend to be transported further from the source and are of greater concern with respect to human health as these particles can penetrate into the lungs (see following point). Hence not all of the dust characterised as total suspended particulates is relevant for the assessment of health impacts, and total suspended particulates as a measure of impact, has not been further evaluated in this assessment. The assessment has only focused on particulates of a size where significant associations have been identified between exposure and adverse health effects.
- PM<sub>10</sub> (particulate matter below 10 microns in diameter), PM<sub>2.5</sub> (particulate matter below 2.5 microns in diameter), PM<sub>1</sub> (particulate matter below one micron in diameter, often termed very fine particles) and ultrafines (particulate matter below 0.1 microns in diameter): These particles are small and have the potential to penetrate beyond the body's natural clearance mechanisms of cilia and mucous in the nose and upper respiratory system, with smaller particles able to further penetrate into the lower respiratory tract<sup>8</sup> and lungs. Once in the lungs adverse health effects may result (OEHHA 2002).

Evaluation of size alone as a single factor in determining the potential for particulate toxicity is difficult since the potential health effects are not independent of chemical composition. There are certain particulate size fractions that tend to contain certain chemical components, such as metals in fine particulates (less than  $PM_{2.5}$ ) and crustal materials (like soil) in the coarse mode ( $PM_{2.5}$  to  $PM_{10}$ ). In addition, different sources of particulates have the potential to result in the presence of other pollutants in addition to particulate matter. For example, combustion sources, prevalent in urban areas, result in the emission of particulate matter (more dominated by  $PM_{2.5}$ ) as well as gaseous pollutants (such as nitrogen dioxide and carbon monoxide). This results in what is referred to as co-exposure, and is an issue that has to be accounted for when evaluating studies that come from studying health effects in large populations exposed to pollution from many sources (as is the case in urban air).

Where co-exposure is accounted for, the available science supports that exposure to fine particulate matter (less than 2.5 microns,  $PM_{2.5}$ ) is associated (and shown to be causal in some cases) with health impacts in the community (USEPA 2012). A more limited body of evidence suggests an association between exposure to larger particles,  $PM_{10}$  and adverse health effects (USEPA 2009b; WHO 2003).

It is noted that when assessing potential health impacts associated with changes in particulate matter concentrations the studies relied upon for establishing associations (between changes in concentrations in air and health effects) are large epidemiological studies. These studies relate changes in health indicators with changes in measured concentrations of particulate matter. As a result, the particle size fractions addressed in these studies relate to the fractions measured in the urban air environment studies. In relation to measuring particulate matter in urban air, the following should be noted:

• The measurement of particulate matter in urban air most commonly reports PM<sub>10</sub>. This is the concentration of particulate matter less than or equal to 10 microns in diameter (and includes the

<sup>5</sup> The term equivalent aerodynamic particle is used to reference the particle to a particle of spherical shape and particle of density one gram per cubic metre.

<sup>6</sup> The size, diameter, of dust particles is measured in micrometers (microns).

<sup>7</sup> The upper respiratory tract comprises the mouth, nose, throat and trachea. Larger particles are mostly trapped by the cilia and mucosa and swept to the back of the throat and swallowed.

<sup>8</sup> The lower respiratory tract comprises the smaller bronchioles and alveoli, the area of the lungs where gaseous exchange takes place. The alveoli have a very large surface area and absorption of gases occurs rapidly with subsequent transport to the blood and the rest of the body. Small particles can reach these areas, be dissolved by fluids and absorbed.
smaller fractions of  $PM_{2.5}$  and very fine particles). The measurement techniques for  $PM_{10}$  are well established and provide stable, robust, verifiable data that is considered to be consistently reported across all countries. In addition, there is a longer and more extensive history/database of  $PM_{10}$  data. This means this data on  $PM_{10}$  collected in different parts of a city, in different parts of a country and by different countries can be compared against each other. This is the key reason why many of the epidemiological studies have looked at associations between  $PM_{10}$  and various health effects

- The measurement of PM<sub>2.5</sub> is becoming more common in urban environments. This is the concentration of particulate matter less than or equal to 2.5 microns in diameter (and includes the smaller fractions of very fine particles and ultrafines). The measurement techniques used for PM<sub>2.5</sub> are less well established resulting in data that varies depending on the type of equipment used and how it is set up and maintained. Due to either a lack of monitoring data or the inconsistency of monitoring data some epidemiology studies have assessed associations between PM<sub>2.5</sub> and health effects by using PM<sub>10</sub> data and assuming that a certain percentage of PM<sub>10</sub> comprises PM<sub>2.5</sub>. Some studies have directly used measurements of PM<sub>2.5</sub> in urban air. Even where these measurement issues are considered, the studies still clearly show strong relationships between changes in PM<sub>2.5</sub> concentrations and health effects
- The measurement of ultrafine particles is difficult (using equipment that is less robust/stable and provides variable data) and has not been undertaken in most urban air environments. As a result, there are no robust epidemiological studies that relate changes in ultrafine particle levels and health effects that can be used in a risk assessment. There is sufficient data available to confirm that motor vehicles are a key source of ultrafine particles. Available studies in animals and humans have identified a range of adverse health effects associated with exposure to ultrafine particulates. However the studies do not show that short term exposure to ultrafine particulates have effects that are significantly different from those associated with exposure to PM<sub>2.5</sub> (HEI 2013).

When assessing health impacts from fine particulates, the robust associations of effects (that are based on large epidemiology studies primarily from the US and Europe) have been determined on the basis of  $PM_{2.5}$ , as  $PM_{2.5}$  is what is commonly measured in urban air. No robust associations (that can be used in a quantitative assessment) are available for  $PM_1$  and the current science is inconclusive in relation to ultrafine particulates. The associations developed for  $PM_{2.5}$  would include a significant contribution from  $PM_1$  (as  $PM_1$  comprises a significant proportion of  $PM_{2.5}$ ) and hence health effects observed for  $PM_1$  would be captured in the studies that have been conducted on the basis of  $PM_{2.5}$ . It is important that the quantitative evaluation of potential health impacts adopts robust health effects associations and utilises particulate matter measures that are collected in the urban air environment. Hence the further assessment of exposure to fine particulate matter has focused on particulates reported/evaluated as  $PM_{2.5}$ .

## 6.9.2 Health effects

Adverse health effects associated with exposure to particulate matter have been well studied and reviewed by Australian and International agencies. Most of the studies and reviews have focused on population-based epidemiological studies in large urban areas in North America, Europe and Australia, where there have been clear associations determined between health effects and exposure to  $PM_{2.5}$  and to a lesser extent,  $PM_{10}$ . These studies are complemented by findings from other key investigations conducted in relation to the characteristics of inhaled particles; deposition and clearance of particles in the respiratory tract; animal and cellular toxicity studies; and studies on inhalation toxicity by human volunteers (NEPC 2010).

Particulate matter has been linked to adverse health effects after both short term exposure (days to weeks) and long term exposure (months to years). The health effects associated with exposure to particulate matter vary widely (with the respiratory and cardiovascular systems most affected) and include mortality and morbidity effects.

In relation to mortality, for short term exposures in a population this relates to the increase in the number of deaths due to existing (underlying) respiratory or cardiovascular disease. For long term exposures in a population this relates to mortality rates over a lifetime, where long term exposure is considered to accelerate the progression of disease or even initiate disease.

In relation to morbidity effects, this refers to a wide range of health indicators used to define illness that have been associated with (or caused by) exposure to particulate matter. In relation to exposure to particulate matter, effects are primarily related to the respiratory and cardiovascular system and include (Morawska et al. 2004; USEPA 2009b):

- Aggravation of existing respiratory and cardiovascular disease (as indicated by increased hospital admissions and emergency room visits)
- Changes in cardiovascular risk factors such as blood pressure
- Changes in lung function and increased respiratory symptoms (including asthma)
- Changes to lung tissues and structure
- Altered respiratory defence mechanisms.

These effects are commonly used as measures of population exposure to particulate matter in community epidemiological studies (from which most of the available data in relation to health effects is derived), and are more often grouped (through the use of hospital codes) into the general categories of cardiovascular morbidity/effects and respiratory morbidity/effects. The available studies provide evidence for increased susceptibility for various populations, particularly older populations, children and those with underlying health conditions (USEPA 2009b).

There is consensus in the available studies and detailed reviews that exposure to fine particulates,  $PM_{2.5}$ , is associated with (and causal to) cardiovascular and respiratory effects and mortality (all causes) (USEPA 2012). Similar relationships have also been determined for  $PM_{10}$ , however, the supporting studies do not show relationships as clear as those shown with  $PM_{2.5}$  (USEPA 2012).

There are a number of studies that have been undertaken where other health effects have been evaluated. These studies are suggestive (but do not show effects as clearly as the effects noted above) of an association between exposure to  $PM_{2.5}$  and reproductive and developmental effects as well as cancer, mutagenicity and genotoxicity (USEPA 2012). IARC (2013) has classified particulate matter as carcinogenic to humans based on data relevant to lung cancer.

Other studies have been reviewed to determine relationships/associations between particulate matter exposure (either  $PM_{10}$  or  $PM_{2.5}$ ) and a wide range of other health effects and health measures including mortality (for different age groups), chronic bronchitis, medication use by adults and children with asthma, respiratory symptoms (including cough), restricted work days, work days lost, school absence and restricted activity days (Anderson et al. 2004; EC 2011; Ostro 2004; WHO 2006a). While these relationships/associations have been identified the exposure-response relationships established are not as strong as those discussed above. Also, the available baseline data does not include information for many of these health effects which means it is not possible to undertake a quantitative assessment.

### 6.9.3 Approach to the assessment of particulate exposures

In relation to the assessment of exposures to particulate matter there is sufficient evidence to demonstrate that there is an association between exposure to  $PM_{2.5}$  (and to a lesser extent  $PM_{10}$ ) and effects on health that are causal. In addition, the effects related to exposures to  $PM_{2.5}$  (or  $PM_{10}$ ) alone (ie without co-exposures).

The available evidence does not suggest that there is a threshold below which health effects do not occur. Hence there are likely to be health effects associated with background levels of  $PM_{2.5}$  and  $PM_{10}$ , even where the concentrations are below the current guidelines. Guidelines are currently available for the assessment of  $PM_{2.5}$  and  $PM_{10}$  in New South Wales (DEC 2005) and Australia (NEPC 2002, 2003). These guidelines are not based on any acceptable level of risk, rather they are based on levels that are desirable in the community to balance background/urban sources with lowering impacts on health and cost savings in the health system.

The air quality goals relate to average or regional exposures by populations from all sources, not to localised 'hot-spot' areas such as locations near industry, busy roads or mining. They are intended to be compared against ambient air monitoring data collected from appropriately sited regional monitoring stations. In some cases, there may be local sources (including busy roadways and industry) that result in background levels of  $PM_{10}$  and  $PM_{2.5}$  that are close to, equal to, or in exceedance of the air quality goals. Where impacts are being evaluated from a local source it is important to not only consider total impacts associated with the project (undertaken using the current air quality goals) but also evaluate the impact of changes in air quality within the local community.

This assessment has therefore been undertaken to consider both cumulative exposure impacts (refer to **Section 6.9.4**) and incremental exposure impacts associated with changes in  $PM_{2.5}$  and  $PM_{10}$  concentrations that are associated with the project (refer to **Section 6.9.5**).

## 6.9.4 Assessment of total exposures

The assessment of cumulative exposures to  $PM_{2.5}$  and  $PM_{10}$  is based on a comparison of the total concentrations predicted in 2026 and 2036 (ie without the project ('Do Minimum'), with the project and for the cumulative scenario, all of which include background exposures) with the relevant air quality guidelines/standards available from the NEPC and NSW EPA. The current NEPC and NSW EPA air quality goals and guidelines/standards for particulate matter are presented in **Table 6-19**. These guidelines/standards are for cumulative impacts and should also be considered in conjunction with incremental impact calculations presented in **Section 6.9.5**.

Pollutant	Averaging period	Criteria (µg/m³)	Reference
PM <sub>10</sub>	24 hour	50	(NEPC 2016; NSW EPA 2016)
	Annual	25	(NSW EPA 2016)
PM <sub>2.5</sub>	24 hour	25 with goal of 20 by 2025	(NEPC 2016)
	Annual	8 with goal of 7 by 2025	

Tuble V 19. An quality guidennes/standards for particulates	Table 6-19: A	ir quality	guidelines/standards	for	particulates
---	---------------	------------	----------------------	-----	--------------

In relation to the current NEPC guidelines, the following is noted (NEPC 1998, 2010, 2014):

- The guideline was derived through a review of appropriate health studies by a technical review panel of the NEPC where short term exposure-response relationships for PM and mortality and morbidity health endpoints were considered
- Mortality health impacts were identified as the most significant and were the primary basis for the development of the guideline
- On the basis of the available data for key air sheds in Australia, the criteria listed in Table 6-19
  was based on analysis of the number of premature deaths that would be avoided and associated
  cost savings to the health system (using data from the US). The development of the goal is not
  based on any acceptable level of risk
- The assessment undertaken considered exposures and issues relevant to urban air environments that are expected to also be managed through the PM guideline. These issues included emissions from vehicles and wood heaters.

**Table 6-20** presents a comparison of the NEPC guidelines with those established (following more recent reviews) by the WHO (WHO 2005), the EU and the USEPA (2012). The standards established by the NEPC for  $PM_{2.5}$  (and adopted in this assessment) are similar to but slightly more conservative (health protective) than those provided by the WHO, EU and the USEPA. The NEPC and NSW OEH  $PM_{10}$  guidelines are also similar to those established by the WHO and EU, however the guidelines are significantly lower than the 24-hour average guideline available from the USEPA.

Pollutant	Averaging			Criteria/guidelines/goals	
	period	NEPC and NSW OEH	WHO (2005)	EU #	USEPA (2012)
PM10	24 hour	50 µg/m³	50 µg/m³	50 $\mu$ g/m <sup>3</sup> as limit value with 35	150 µg/m³
				exceedances permitted each year	(not to be exceeded more than once per year on average over 3 years)
	Annual	25 µg/m³	20* µg/m³	40 μg/m <sup>3</sup> as limit value	NA
PM <sub>2.5</sub>	24 hour	25 μg/m³ (with goal of 20 by 2025)	25 µg/m³	NA	35 μg/m <sup>3</sup> (98th percentile, averaged over 3 years)

### Table 6-20 Comparison of particulate matter air quality goals

Pollutant	Averaging			Criteria/guidelines/goals					
	Annual	8 μg/m <sup>3</sup> (with goal of 7 by 2025)	10* µg/m³	<ul> <li>25 μg/m<sup>3</sup> as target value from 2010 and limit value from 2015.</li> <li>20 μg/m<sup>3</sup> as a 3 year average (average exposure indicator) from 2015 with requirements for ongoing percentage reduction and target of 18 μg/m<sup>3</sup> as 3 year average by 2020</li> </ul>	12 μg/m <sup>3</sup> (annual mean averaged over 3 years)				

# Current EU Air Quality Standards available from http://ec.europa.eu/environment/air/quality/standards.htm

\* The WHO Air Quality guidelines are based on the lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95 per cent confidence in response to PM<sub>2.5</sub> in the ACS study (Pope et al. 2002). The use of a PM<sub>2.5</sub> guideline is preferred by the WHO (WHO 2005).

The NEPM air quality standards for  $PM_{2.5}$  and  $PM_{10}$  relate to total concentrations in the air (from all sources including the project). The background air quality data that has been used in **Appendix E** (Air quality technical report) for this project is summarised in **Section 6.2** and generally relates to urban air quality in areas located away from major roadways. The background data includes a contribution of PM that is derived from vehicles that utilise the existing road network (but not representative of locations adjacent to main roadways). Hence use of this background data would result in some double counting of the contribution of vehicle emissions to air quality in the local area, as the project has then modelled emissions from surface roads and added these to the background.

**Table 6-21** and **Table 6-22** present a summary of the maximum total 24-hour average and annual average concentrations of  $PM_{2.5}$  and  $PM_{10}$  relevant to the assessment of emissions in 2026 and 2036, for the project and for the cumulative case.

Location and scenario	Maximum concentra	24 hour tion (µg/m	average PM <sub>2.5</sub> ³)	Maximum 24 hour average $PM_{10}$ concentration (µg/m <sup>3</sup> )				
	Without project	With project	Cumulative	Without project	With project	Cumulative		
2026			•					
Maximum	39.1	39.0		70.6	69.0			
Maximum residential	31.2	30.4		58.6	56.1			
Maximum commercial	31.9	31.9		58.8	58.3			
2036	1	1	I			I		
Maximum	42.0	39.8	38.3	74.1	71.7	70.5		
Maximum residential	31.8	30.3	30.8	58.3	56.9	56.8		
Maximum commercial	34.0	34.1	34.1	61.1	62.7	61.5		
			•		•			
Guideline	25			50				
	20 by 2025 (	goal)						

### Table 6-21 Review of total PM concentrations – 24-hour average

Location and scenario	Maximum concentra	annual ition (µg/m	average PM <sub>2.5</sub> <sup>3</sup> )	Maximum annual average $PM_{10}$ concentration (µg/m <sup>3</sup> )				
	Without project	With project	Cumulative	Without project	With project	Cumulative		
2026								
Maximum	16.1	15.6		30.3	29.5			
Maximum residential	12.5	12.1		24.5	23.8			
Maximum commercial	12.4	12.4		24.7	24.4			
2036								
Maximum	17.1	16.3	16.1	31.8	30.9	30.7		
Maximum residential	12.6	12.1	12.1	24.8	23.9	23.9		
Maximum commercial	13.0	12.7	12.8	24.9	25.0	25.2		
					•			
Guideline	8 7 by 2025 (g	oal)		25				

### Table 6-22 Review of total PM concentrations – annual average

The maximum total/cumulative concentrations of  $PM_{2.5}$  are above the guidelines for both a 24-hour average and an annual average (including the 2025 goal). This is due in large part to the existing levels of  $PM_{2.5}$  in air within the existing urban environment. These elevated background levels would be present in the community regardless of the construction and operation of the project. Concentrations of total  $PM_{2.5}$ , however, are essentially unchanged within the local community with the operation of the project, as well as the construction and operation of all road tunnel projects.

The maximum cumulative concentrations of  $PM_{10}$  presented in the above tables are above the 24-hour average and annual average guidelines. The maximum concentrations in residential areas are below the annual average guideline. The elevated levels of total  $PM_{10}$  are due to the existing levels of  $PM_{10}$  in air within the existing urban environment. These elevated background levels would be present in the community regardless of the construction and operation of the project. Concentrations of total  $PM_{10}$ , however, are essentially unchanged within the local community with the construction and operation of the project, as well as the operation of all road tunnel projects in NSW to date.

To further address potential risks to human health that may be associated with localised changes (or redistribution) in exposures to  $PM_{2.5}$  and  $PM_{10}$  that relate to the project, an assessment of incremental impacts has been undertaken and are presented in **Section 6.9.5**.

### 6.9.5 Changes in air quality associated with project

### Methodology for assessment of PM<sub>2.5</sub> and PM<sub>10</sub>

A detailed assessment of potential health effects associated with exposure to changes in air quality as a result of the project has been undertaken. As no threshold has been determined for exposure to  $PM_{2.5}$  or  $PM_{10}$  the assessment of impacts on health has utilised robust, published, quantitative relationships (exposure-response relationships) that relate a change in  $PM_{2.5}$  or  $PM_{10}$  concentration with a change in a health indicator. Annexure A presents an overview of the methodology adopted for using exposure-response relationships for the assessment of health impacts in a community.

This report has presented an assessment of changes in individual risk associated the predicted changes in air quality, as well as a change in population health impacts (as would be measured by changes in mortality statistics or hospital admissions) related to changes in exposures to particulates in the surrounding community.

For the assessment of changes in particulate matter exposures in the community the assessment has focused on health effects and exposure-response relationships that are robust and relate to  $PM_{2.5}$ , being the more important particulate fraction size relevant for emissions from combustion sources. Assessment of  $PM_{10}$  has also been included.

The specific health effects (or endpoints) evaluated in this assessment have been identified and include the following:

### Primary health endpoints:

- Long term exposure to PM<sub>2.5</sub> and changes in all-cause mortality (equal or greater than 30 years of age)
- Short term exposure and changes to the rate of hospitalisations with cardiovascular and respiratory disease (equal or greater than 65 years of age).

### Secondary health endpoints (to supplement the primary assessment):

- Short term exposure to PM<sub>10</sub> and changes in all-cause mortality (all ages)
- Long term exposure to PM<sub>2.5</sub> and changes in cardiopulmonary mortality (equal or greater than 30 years of age)
- Short term exposure to PM<sub>2.5</sub> and changes in cardiovascular and respiratory mortality (all ages)
- Short term exposure to PM<sub>2.5</sub> and changes in emergency department admissions for asthma in children aged 1–14 years.

**Table 6-23** presents a summary of the health endpoints considered in this assessment, the relevant health impact functions (from the referenced published studies) and the associated  $\beta$  coefficient relevant to the calculation of a relative risk (refer to Annexure A for details on the calculation of a  $\beta$  coefficient from published studies).

The health impact functions presented in this table are considered to be the most current and robust values, and are appropriate for the quantification of potential health effects for the health endpoints considered in this assessment.

Health endpoint	Exposure period	Age group	Published relative risk [95 confidence interval] per 10 µg/m <sup>3</sup>	Adopted β coefficient (as per cent) for 1 μg/m <sup>3</sup> increase in PM	Reference
Primary assess	sment health	endpoint	S	F	
PM <sub>2.5</sub> : Mortality, all causes	Long term	≥30yrs	1.06 [1.04-1.08]	0.0058 (0.58)	Relationship derived for all follow-up time periods to the year 2000 (for approx. 500,000 participants in the US) with adjustment for seven ecologic (neighbourhood level) covariates (Krewski et al. 2009). This study is an extension (additional follow- up and exposure data) of the work undertaken by Pope (2002), is consistent with the findings from California (1999–2002) (Ostro et al. 2006) and is more conservative than the relationships identified in a more recent Australian and New Zealand study (EPHC 2010).
PM <sub>2.5</sub> : Cardiovascular hospital admissions	Short term	≥65yrs	1.008 [1.0059– 1.011]	0.0008 (0.08)	Relationship established for all data and all seasons from US data for 1999 to 2005 for lag 0 (exposure on same day) (strongest effect identified) (Bell, M. L. 2012; Bell, Michelle L. et al. 2008)
PM <sub>2.5</sub> : Respiratory hospital admissions	Short term	≥65yrs	1.0041 [1.0009– 1.0074]	0.00041 (0.041)	Relationship established for all data and all seasons from US data for 1999 to 2005 for lag 2 (exposure 2 days previous) (strongest effect identified) (Bell, M. L. 2012; Bell, Michelle L. et al. 2008)
Secondary ass	sessment hea	Ith endpo	pints		

### Table 6-23 Adopted health impact functions and exposure-responses relationships

Health endpoint	Exposure period	Age group	Published relative risk [95 confidence interval] per 10 µg/m <sup>3</sup>	Adopted β coefficient (as per cent) for 1 μg/m <sup>3</sup> increase in PM	Reference
PM10: Mortality, all causes	Short term	All ages*	1.006 [1.004–1.008]	0.0006 (0.06)	Based on analysis of data from European studies from 33 cities and includes panel studies of symptomatic children (asthmatics, chronic respiratory conditions) (Anderson et al. 2004)
PM <sub>2.5</sub> : Mortality, all causes	Short term	All ages*	1.0094 [1.0065– 1.0122]	0.00094 (0.094)	Relationship established from study of data from 47 US cities for the years 1999 to 2005 (Zanobetti & Schwartz 2009)
PM <sub>2.5</sub> : Cardio- pulmonary mortality	Long term	≥30yrs	1.14 [1.11–1.17]	0.013 (1.3)	Relationship derived for all follow-up time periods to the year 2000 (for approx. 500,000 participants in the US) with adjustment for seven ecologic (neighbourhood level) covariates (Krewski et al. 2009).
PM <sub>2.5</sub> : Cardiovascular mortality	Short term	All ages*	1.0097 [1.0051– 1.0143]	0.00097 (0.097)	Relationship established from study of data from 47 US cities for the years 1999 to 2005 (Zanobetti & Schwartz 2009)
PM <sub>2.5</sub> : Asthma (emergency department admissions)	Short term	1–14 years	-	0.00148 (0.148)	Relationship established from review conducted on Australian children (Sydney) for the period 1997 to 2001 (Jalaludin et al. 2008)
PM <sub>2.5</sub> : Respiratory mortality (including lung cancer)	Short term	All ages*	1.0192 [1.0108– 1.0278]	0.0019 (0.19)	Relationship established from study of data from 47 US cities for the years 1999 to 2005 (Zanobetti & Schwartz 2009)

Note: \* Relationships established for all ages, including young children and the elderly

The assessment of health impacts for a population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004) (also outlined in **Annexure A**) where the exposure-response relationships (presented in **Table 6-23**) have been directly considered.

A change in relative risk has then been calculated on the basis of the following:

- Estimates of the changes in PM<sub>2.5</sub> and PM<sub>10</sub> exposure levels due to the project in 2026 and 2036 (as provided in **Appendix E** (Air quality technical report)) for the scenarios assessed with the project as well as the cumulative impacts from all road tunnel projects at each of the community receptors (see **Figure 4-2**) as well as the maximum off-site residential and workplace receptors from the RWR receptors
- Baseline incidence of the key health endpoints that are relevant to the population exposed (see **Table 4-5**)
- Exposure-response relationships expressed as a percentage change in health endpoint per micrograms per cubic metre change in particulate matter exposure (see **Table 6-23**).

The change in incidence of each health indicator relevant to changes in  $PM_{2.5}$  exposures in the local community (for the population exposed) has been calculated on the basis of the following:

- The relative risk has been calculated for a population weighted annual average incremental increase in PM<sub>2.5</sub> concentrations (using the approach outlined above). The population weighted average change in concentration has been calculated on the basis of the smallest statistical division provided by the Australian Bureau of Statistics within a suburb (ie mesh blocks which are small blocks that cover an area of about 30 urban residences). For each mesh block in a suburb, the average change in PM<sub>2.5</sub> concentration has been calculated and multiplied by the population living in the mesh block (data available from the ABS for the 2016 census year). The weighted average has been calculated by summing these calculations for each mesh block in a suburb and dividing by the total population in the suburb (ie in all the mesh block)
- A change in the number of cases associated with the change in PM<sub>2.5</sub> impact evaluated in the population within the study area has been calculated (refer to Annexure A for details on the methodology). The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered (see **Table 4-5**) and the population (for the relevant age groups) present in the suburb (see **Table 4-3**).

### Methodology for assessing exposure to diesel particulate matter

In addition to the above exposure-response relationships, potential exposure to diesel particulate matter (DPM) derived from the project has been evaluated.

Diesel exhaust (DE) is emitted from 'on-road' diesel engines (vehicle engines) and can be formed from the gaseous compounds emitted by diesel engines (secondary particulate matter). After emission from the exhaust pipe, diesel exhaust undergoes dilution and chemical and physical transformations in the atmosphere, as well as dispersion and transport in the atmosphere. The atmospheric lifetime for some compounds present in diesel exhaust ranges from hours to days.

Available evidence indicates that there are human health hazards associated with exposure to diesel particulate matter. The hazards include acute exposure-related symptoms, chronic exposure related non-cancer respiratory effects, and lung cancer. The non-cancer health effects associated with exposure to DPM are adequately addressed on the basis of the current  $PM_{2.5}$  and  $PM_{10}$  guidelines. However, the potential for exposure to DPM to result in an increased risk of lung cancer in the community requires further consideration. Annexure B presents the methodology adopted for the assessment of lung cancer risks associated with exposure to DPM. In summary, the following has been assumed/undertaken:

- It has been conservatively assumed that 100 per cent of PM<sub>2.5</sub> predicted in the local community is derived from diesel vehicles and comprises DPM
- An incremental lifetime risk of lung cancer has been calculated (refer to Annexure B for methodology) on the basis of the inhalation toxicity value available from the World Health Organization (WHO 1996).

### Acceptability of health impacts

Based on the methodology outlined above, potential health impacts associated with the project have been assessed on the basis of two calculations:

- Calculation of an annual risk for each health endpoint. This is a change in risk that differs from the baseline risk (or incidence) of the effect occurring for any member of the population, where exposed to the change in particulate matter concentration estimated
- Calculation of a change in incidence of the health effect occurring within the population exposed. This calculates the change in the number of cases (mortality or hospitalisations) that may occur for the population assumed to be exposed to the changes in particulate matter concentration estimated.

To determine if the calculated annual risk or change in incidence within a population associated with particulate matter impacts from the project may be considered to be acceptable a number of factors need to be considered. These are discussed further in Annexure C.

It is noted that the change in risk and health incidence calculated in this assessment includes negative values (where there is a lower risk and incidence of health effects in the community with the operation of the project) and positive values (where there is an increase in risk and health incidence in the community with the operation of the project).

Any negative values are related to improved health impacts in the community and are considered acceptable. The following discussion relates to the evaluation of positive values.

#### <u>Risk:</u>

While it is not possible to provide a rigid definition of acceptable risk due to the complex and contextdriven nature of the challenge, it is possible to propose some general guidelines as to what might be an acceptable risk for specific development projects.

If a level of less than  $10^{-6}$  (one chance in a million) were retained as a level of increased risk that would be considered as a negligible risk in the community, then the level of risk that could be considered to be tolerable would lie between this level and an upper level that is considered to be unacceptable.

While there is no guidance available on what level of risk is considered to be unacceptable in the community, a level in excess of 10<sup>-4</sup> for increased risk (one chance in 10,000) has been generally adopted by health authorities as a point where risk is considered to be unacceptable. This level has been adopted in the development of drinking water guidelines (that impact on whole populations) (for exposure to carcinogens as well as for annual risks of disease (Fewtrell & Bartram 2001)) and in the evaluation of exposures from pollutants in air (NSW DEC 2005).

Between an increased risk level considered negligible (less than  $10^{-6}$ ) and unacceptable (greater than  $10^{-4}$ ) lie risks that may be considered to be tolerable or even acceptable. Tolerable risks are those that can be tolerated (and where the best available, and most appropriate, technology has been implemented to minimise exposure) in order to realise some wider community benefit.

In a societal context, risks are inevitable and any new development would be accompanied by risks which are not amenable or economically feasible to reduce below a certain level. It is not good policy to impose an arbitrary risk level to such developments without consideration of the many factors that should be considered to determine what is 'tolerable' or 'acceptable'.

Hence for this project the calculated risks have been considered to be tolerable when in the range of greater than or equal to  $10^{-6}$  and less than or equal to  $10^{-4}$  of increased risk and where the increased incidence of the health impacts are considered to be insignificant.

#### Population incidence:

The assessment of changes in incidence of particular health indicators in the community results in the calculation of a change in the number of cases (of mortality, hospital or emergency department admissions) within the population evaluated.

As discussed in Annexure C, where changes in air quality associated with this project are well below 10 cases per year they are considered to be within the normal variability of health statistics, and these changes would not be measurable in any health statistics for the area. For evaluating impacts from this project a more conservative tenfold margin of safety has been included to determine what changes in incidence may be considered negligible within the study population.

This means that changes in the population incidence of any health effect evaluated that is less than one case per year are considered negligible.

### Calculated risks and population incidence for operation of the project

Review of the changes in particulate matter concentrations predicted in 2026 and 2036 indicates that for a number of receptors in the local community the project results in a decrease in the concentration of  $PM_{2.5}$  and  $PM_{10}$ . For a number of receptors there is an increase in the concentration of  $PM_{2.5}$  and  $PM_{10}$ , which relates to the redistribution of emissions on surface roads in the study area, not from emissions from the ventilation facilities (as discussed in **Appendix E** (Air quality technical report)). This is illustrated in **Figure 6-6** that presents a contour plot of the change in annual average  $PM_{2.5}$  concentrations associated with the project in the assessment year 2036. For a number of areas, the change is negative (ie a decrease in  $PM_{2.5}$  concentrations due to the project) however for some areas adjacent to some roadways (President Ave, Princes Hwy and O'Connell St) the change is positive (ie an increase in  $PM_{2.5}$  concentrations due to the project).



Figure 6-6 Contour plot showing change in annual average  $\text{PM}_{\rm 2.5}$  concentrations associated with the project in 2036

Based on the methodology outlined above, **Table 6-24** to **Table 6-25** present the calculated individual risk associated with changes in  $PM_{2.5}$  and  $PM_{10}$  concentrations from the ventilation facilities only as well from the total project (ventilation plus roadway emissions) at the maximum impacted residential, childcare, schools, aged care, hospital, commercial/industrial and open space areas as well as the maximum impacted community receptor, for the operational years 2026 and 2036. The change in  $PM_{2.5}$  and  $PM_{10}$  concentration considered in the risk calculations are also included in the tables.

The calculated change in risk at the maximum receptors represents the worst case impact associated with the project. Risks for all other receptors would be lower than calculated for the maximum receptors.

**Table 6-7** shows the calculated risks for each of the community receptors, associated with the primary health endpoints evaluated in this assessment for the project's operations in 2026 and 2036.

All calculated individual risks are presented in Annexure F.

**Table 6-27** and **Table 6-28** present a summary of the calculated change in incidence of the relevant health effects for the population living in the LGAs within the study area, associated with changes in  $PM_{2.5}$  concentrations for 2026 and 2036. All calculations relevant to the LGAs, including calculation for each individual suburb considered in the LGAs, are presented in Annexure G.

Receptor	Chan	nge in	Calculated risks for health endpoints										
	average concentration (µg/m <sup>3</sup> )		PM2.5: Mortality, all causes	PM2.5: Cardiovascular hospitalisations	PM2.5: Respiratory hospitalisations	PM10: Mortality, all causes	PM2.5: Mortality, all causes	PM2.5: Mortality, cardiopulmonary	PM2.5: Mortality, cardiovascular	PM2.5: Mortality, respiratory	PM2.5: Asthma emergency department hospitalisations	DPM Lung cancer	
	PM <sub>10</sub>	PM <sub>2.5</sub>	long-term	short-term	short-term	short- term	short- term	long-term	short-term	short-term	short-term	long-term	
			≥30 yrs	≥65 yrs	≥65 yrs	all	all	≥30 yrs	all	all	1–14 yrs	all	
2026 with project – cha	anges froi	m ventilat	ion facilities										
Maximum residential	0.17	0.11	7X10 <sup>-6</sup>	8X10 <sup>-6</sup>	2X10 <sup>-6</sup>	5X10 <sup>-7</sup>	5X10 <sup>-7</sup>	6X10-6	1X10 <sup>-7</sup>	8X10 <sup>-8</sup>	2X10 <sup>-6</sup>	4X10 <sup>-6</sup>	
Maximum childcare	0.04	0.028	2X10 <sup>-6</sup>	2X10 <sup>-6</sup>	5X10 <sup>-7</sup>	1X10 <sup>-7</sup>	1X10 <sup>-7</sup>	1X10-6	4X10 <sup>-8</sup>	2X10 <sup>-8</sup>	5X10 <sup>-7</sup>	1X10 <sup>-6</sup>	
Maximum schools	0.082	0.059	4X10 <sup>-6</sup>	4X10 <sup>-6</sup>	1X10 <sup>-6</sup>	2X10 <sup>-7</sup>	3X10 <sup>-7</sup>	3X10-6	8X10 <sup>-8</sup>	4X10 <sup>-8</sup>	1X10 <sup>-6</sup>	2X10 <sup>-6</sup>	
Maximum aged care	0.054	0.038	2X10-6	3X10-6	6X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10-6	5X10 <sup>-8</sup>	3X10 <sup>-8</sup>	7X10 <sup>-7</sup>	1X10-6	
Maximum hospital	0.067	0.046	3X10-6	3X10-6	8X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10-6	6X10- <sup>8</sup>	3X10 <sup>-8</sup>	8X10 <sup>-7</sup>	2X10 <sup>-6</sup>	
Maximum commercial/ industrial	0.12	0.083	5X10-6	6X10 <sup>-6</sup>	1X10 <sup>-6</sup>	4X10 <sup>-7</sup>	4X10 <sup>-7</sup>	4X10 <sup>-6</sup>	1X10 <sup>-7</sup>	6X10- <sup>8</sup>	1X10 <sup>-6</sup>	3X10-₀	
Maximum open space	0.073	0.049	3X10-6	4X10 <sup>-6</sup>	8X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10 <sup>-7</sup>	3X10-6	6X10 <sup>-8</sup>	4X10 <sup>-8</sup>	9X10 <sup>-7</sup>	2X10-6	
Maximum community receptors	0.08	0.06	4X10-6	5X10 <sup>-6</sup>	1X10 <sup>-6</sup>	2X10 <sup>-7</sup>	3X10 <sup>-7</sup>	3X10 <sup>-6</sup>	8X10 <sup>-8</sup>	5X10 <sup>-8</sup>	1X10 <sup>-6</sup>	2X10 <sup>-6</sup>	
2026 with project – cha	anges froi	m ventilat	ion facilities a	and roadway emis	ssions								
Maximum residential	0.68	0.44	3X10-5	3X10-5	7X10-6	2X10-6	2X10-6	2X10-5	6X10 <sup>-7</sup>	3X10 <sup>-7</sup>	8X10-6	1X10-5	
Maximum childcare	0.08	0.05	3X10-6	4X10-6	8X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10 <sup>-7</sup>	3X10-6	7X10 <sup>-8</sup>	4X10 <sup>-8</sup>	9X10 <sup>-7</sup>	2X10-6	
Maximum schools	0.25	0.17	1X10-5	1X10 <sup>-5</sup>	3X10-6	7X10 <sup>-7</sup>	8X10-7	9X10-6	2X10 <sup>-7</sup>	1X10 <sup>-7</sup>	3X10-6	6X10-6	
Maximum aged care	0.08	0.12	7X10-6	9X10-6	2X10-6	2X10 <sup>-7</sup>	6X10 <sup>-7</sup>	6X10-6	2X10 <sup>-7</sup>	9X10 <sup>-8</sup>	2X10-6	4X10-6	
Maximum hospital	0.15	0.11	7X10-6	8X10-6	2X10-6	4X10 <sup>-7</sup>	5X10 <sup>-7</sup>	6X10-6	1X10 <sup>-7</sup>	8X10 <sup>-8</sup>	2X10-6	4X10-6	
Maximum commercial/ industrial	0.52	0.35	2X10-5	3X10 <sup>-5</sup>	6X10-6	2X10-6	2X10-6	2X10 <sup>-5</sup>	5X10-7	3X10 <sup>-7</sup>	6X10 <sup>-6</sup>	1X10 <sup>-5</sup>	
Maximum open space	0.15	0.12	7X10 <sup>-6</sup>	9X10⁻ <sup>6</sup>	2X10 <sup>-6</sup>	4X10 <sup>-7</sup>	5X10 <sup>-7</sup>	6X10-6	2X10 <sup>-7</sup>	9X10 <sup>-8</sup>	2X10 <sup>-6</sup>	4X10 <sup>-6</sup>	
Maximum community receptors	0.20	0.19	1X10 <sup>-5</sup>	1X10 <sup>-5</sup>	3X10-6	6X10 <sup>-7</sup>	9X10 <sup>-7</sup>	1X10 <sup>-5</sup>	2X10 <sup>.7</sup>	1X10 <sup>-7</sup>	3X10 <sup>-6</sup>	6X10-6	
	Negligik	ole risks					<	<1 x 10 <sup>-6</sup>					
Tolerable	/acceptat	ole risks					≥1 x 10	<sup>-6</sup> and ≤1 x 10 <sup>-4</sup>					
Ur	nacceptat	ole risks					>	>1 x 10 <sup>-4</sup>					

### Table 6-24 Calculated individual risk associated with changes in PM<sub>2.5</sub> and PM<sub>10</sub> concentrations – project operations in 2026

Receptor	Chan	ige in	Calculated risks for health endpoints									
	average concentration (µg/m <sup>3</sup> )		PM2.5: Mortality, all causes	PM2.5: Cardiovascular hospitalisations	PM2.5: Respiratory hospitalisations	PM10: Mortality, all causes	PM2.5: Mortality, all causes	PM2.5: Mortality, cardiopulmonary	PM2.5: Mortality, cardiovascular	PM2.5: Mortality, respiratory	PM2.5: Asthma emergency department hospitalisations	DPM Lung cancer
	PM <sub>10</sub>	PM <sub>2.5</sub>	long- term	short-term	short-term	short- term	short- term	long-term	short-term	short-term	short-term	long-term
			≥30 yrs	≥65 yrs	≥65 yrs	all	all	≥30 yrs	all	all	1–14 yrs	all
2036 with project – cha	nges fron	n ventilati	ion facilities	S								
Maximum residential	0.20	0.14	8X10-6	1X10 <sup>-5</sup>	2X10 <sup>-6</sup>	6X10 <sup>-7</sup>	6X10 <sup>-7</sup>	7X10-6	2X10 <sup>-7</sup>	1X10 <sup>-7</sup>	3X10 <sup>-6</sup>	5X10 <sup>-6</sup>
Maximum childcare	0.051	0.038	2X10-6	3X10-6	6X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10-6	5X10 <sup>-8</sup>	3X10 <sup>-8</sup>	7X10 <sup>-7</sup>	1X10 <sup>-6</sup>
Maximum schools	0.10	0.075	4X10 <sup>-6</sup>	6X10 <sup>-6</sup>	1X10 <sup>-6</sup>	3X10 <sup>-7</sup>	3X10 <sup>-7</sup>	4X10-6	1X10 <sup>-7</sup>	6X10 <sup>-8</sup>	1X10 <sup>-6</sup>	3X10⁻ <sup>6</sup>
Maximum aged care	0.071	0.046	3X10-6	3X10-6	8X10 <sup>-7</sup>	2X10-7	2X10-7	2X10-6	6X10 <sup>-8</sup>	3X10 <sup>-8</sup>	8X10 <sup>-7</sup>	2X10-6
Maximum hospital	0.08	0.055	3X10-6	4X10-6	9X10 <sup>-7</sup>	2X10-7	3X10-7	3X10-6	7X10 <sup>-8</sup>	4X10 <sup>-8</sup>	1X10-6	2X10-6
Maximum commercial/ industrial	0.13	0.097	6X10-6	7X10 <sup>-6</sup>	2X10 <sup>-6</sup>	4X10-7	4X10-7	5X10 <sup>-6</sup>	1X10 <sup>-7</sup>	7X10 <sup>-8</sup>	2X10 <sup>-6</sup>	3X10-₀
Maximum open space	0.089	0.062	4X10 <sup>-6</sup>	5X10-6	1X10 <sup>-6</sup>	3X10 <sup>-7</sup>	3X10 <sup>-7</sup>	3X10 <sup>-6</sup>	8X10 <sup>-8</sup>	5X10 <sup>-8</sup>	1X10 <sup>-6</sup>	2X10 <sup>-6</sup>
Maximum community receptors	0.10	0.07	4X10 <sup>-6</sup>	5X10 <sup>-6</sup>	1X10 <sup>-6</sup>	3X10 <sup>-7</sup>	3X10 <sup>-7</sup>	4X10 <sup>-6</sup>	9X10 <sup>-8</sup>	5X10⁻ <sup>8</sup>	1X10 <sup>-6</sup>	2X10 <sup>-6</sup>
2036 with project - cha	nges fron	n ventilati	ion facilities	s and roadway en	nissions							
Maximum residential	0.65	0.39	2X10-5	3X10-5	6X10-6	2X10-6	2X10-6	2X10-5	5X10 <sup>-7</sup>	3X10-7	7X10-6	1X10-5
Maximum childcare	0.14	0.04	2X10-6	3X10-6	6X10 <sup>-7</sup>	4X10 <sup>-7</sup>	2X10-7	2X10-6	5X10 <sup>-8</sup>	3X10 <sup>-8</sup>	6X10 <sup>-7</sup>	1X10-6
Maximum schools	0.21	0.23	1X10 <sup>-5</sup>	2X10 <sup>-5</sup>	4X10 <sup>-6</sup>	6X10 <sup>-7</sup>	1X10-6	1X10-5	3X10 <sup>-7</sup>	2X10 <sup>-7</sup>	4X10-6	8X10-6
Maximum aged care	0.09	0.08	5X10-6	6X10-6	1X10 <sup>-6</sup>	3X10-7	4X10-7	4X10-6	1X10 <sup>-7</sup>	6X10 <sup>-8</sup>	1X10 <sup>-6</sup>	3X10-6
Maximum hospital	0.16	0.12	7X10-6	9X10 <sup>-6</sup>	2X10-6	5X10-7	6X10-7	6X10-6	2X10 <sup>-7</sup>	9X10 <sup>-8</sup>	2X10-6	4X10-6
Maximum commercial/ industrial	0.52	0.30	2X10-5	2X10 <sup>-5</sup>	5X10-6	2X10-6	2X10-6	2X10 <sup>-5</sup>	4X10 <sup>-7</sup>	2X10 <sup>-7</sup>	5X10 <sup>-6</sup>	1X10 <sup>-5</sup>
Maximum open space	0.20	0.15	9X10-6	1X10 <sup>-5</sup>	2X10 <sup>-6</sup>	6X10 <sup>-7</sup>	8X10 <sup>-7</sup>	8X10-6	2X10 <sup>-7</sup>	1X10 <sup>-7</sup>	3X10 <sup>-6</sup>	5X10 <sup>-6</sup>
Maximum community receptors	0.18	0.14	9X10-6	1X10 <sup>-5</sup>	2X10-6	5X10-7	8X10-7	8X10-6	2X10 <sup>-7</sup>	1X10 <sup>-7</sup>	3X10 <sup>-6</sup>	5X10-₀
							I			I		
	Nealiait	ole risks						<1 x 10 <sup>-6</sup>				
Tolerable	acceptat	ole risks					≥1 x 1	0 <sup>-6</sup> and ≤1 x 10 <sup>-4</sup>				
ıU	nacceptak	ole risks						>1 x 10 <sup>-4</sup>				

### Table 6-25 Calculated individual risk associated with changes in PM<sub>2.5</sub> and PM<sub>10</sub> concentrations – project operations in 2036

Receptor	Chan	ige in	Calculated risks for health endpoints										
	average concentration (µg/m <sup>3</sup> )		PM2.5: Mortality, all causes	PM2.5: Cardiovascular hospitalisations	PM2.5: Respiratory hospitalisations	PM10: Mortality, all causes	PM2.5: Mortality, all causes	PM2.5: Mortality, cardiopulmonary	PM2.5: Mortality, cardiovascular	PM2.5: Mortality, respiratory	PM2.5: Asthma emergency department hospitalisations	DPM Lung cancer	
	PM <sub>10</sub>	PM <sub>2.5</sub>	long- term	short-term	short-term	short- term	short- term	long-term	short-term	short-term	short-term	long-term	
			≥30 yrs	≥65 yrs	≥65 yrs	all	all	≥30 yrs	all	all	1–14 yrs	all	
2036 cumulative - chan	ges from	ventilatio	n facilities										
Maximum residential	0.25	0.16	1X10 <sup>-5</sup>	1X10 <sup>-5</sup>	3X10-6	7X10 <sup>-7</sup>	7X10 <sup>-7</sup>	9X10 <sup>-6</sup>	2X10 <sup>-7</sup>	1X10 <sup>-7</sup>	3X10-6	5X10-6	
Maximum childcare	0.08	0.051	3X10-6	4X10-6	8X10 <sup>-7</sup>	2X10 <sup>-7</sup>	2X10-7	3X10-6	7X10 <sup>-8</sup>	4X10 <sup>-8</sup>	9X10 <sup>-7</sup>	2X10 <sup>-6</sup>	
Maximum schools	0.14	0.096	6X10-6	7X10-6	2X10-6	4X10-7	4X10-7	5X10-6	1X10 <sup>-7</sup>	7X10 <sup>-8</sup>	2X10-6	3X10-6	
Maximum aged care	0.096	0.066	4X10-6	5X10-6	1X10-6	3X10-7	3X10-7	4X10-6	9X10-8	5X10-8	1X10-6	2X10-6	
Maximum hospital	0.11	0.077	5X10-6	6X10-6	1X10-6	3X10-7	4X10-7	4X10-6	1X10-7	6X10-8	1X10-6	3X10-6	
Maximum commercial/ industrial	0.17	0.12	7X10-6	9X10-6	2X10-6	5X10-7	6X10-7	6X10-₀	2X10-7	9X10⁻ <sup>8</sup>	2X10-6	4X10-6	
Maximum open space	0.12	0.076	5X10-6	6X10-6	1X10 <sup>-6</sup>	4X10 <sup>-7</sup>	4X10 <sup>-7</sup>	4X10-6	1X10 <sup>-7</sup>	6X10 <sup>-8</sup>	1X10-6	3X10-6	
Maximum community receptors	0.13	0.09	5X10-₀	6X10 <sup>-6</sup>	1X10 <sup>-6</sup>	4X10 <sup>-7</sup>	4X10 <sup>-7</sup>	5X10⁻ <sup>6</sup>	1X10 <sup>-7</sup>	7X10 <sup>-8</sup>	2X10-6	3X10-6	
2036 cumulative – char	nges from	ventilatio	on facilities	and roadway em	issions								
Maximum residential	0.50	0.37	2X10-5	3X10-5	6X10-6	1X10-6	2X10-6	2X10-5	5X10 <sup>-7</sup>	3X10 <sup>-7</sup>	7X10-6	1X10 <sup>-5</sup>	
Maximum childcare	0.14	0.08	4X10-6	6X10-6	1X10 <sup>-6</sup>	4X10-7	3X10 <sup>.7</sup>	4X10 <sup>-6</sup>	1X10 <sup>-7</sup>	6X10 <sup>-8</sup>	1X10-6	3X10-6	
Maximum schools	0.11	0.10	6X10-6	7X10-6	2X10-6	3X10-7	5X10 <sup>-7</sup>	5X10-6	1X10 <sup>-7</sup>	8X10 <sup>-8</sup>	2X10-6	3X10-6	
Maximum aged care	0.09	0.07	4X10-6	5X10-6	1X10 <sup>-6</sup>	3X10-7	3X10-7	4X10-6	9X10 <sup>-8</sup>	5X10 <sup>-8</sup>	1X10-6	2X10-6	
Maximum hospital	0.10	0.06	4X10-6	5X10-6	1X10 <sup>-6</sup>	3X10-7	3X10-7	3X10-6	8X10 <sup>-8</sup>	5X10 <sup>-8</sup>	1X10-6	2X10-6	
Maximum commercial/ industrial	0.46	0.23	1X10 <sup>-5</sup>	2X10 <sup>-5</sup>	4X10-6	1X10-6	1X10-6	1X10 <sup>-5</sup>	3X10 <sup>-7</sup>	2X10-7	4X10 <sup>-6</sup>	8X10-6	
Maximum open space	0.25	0.11	6X10-6	8X10-6	2X10-6	7X10 <sup>-7</sup>	5X10 <sup>-7</sup>	6X10-6	1X10 <sup>-7</sup>	8X10 <sup>-8</sup>	2X10 <sup>-6</sup>	4X10 <sup>-6</sup>	
Maximum community receptors	0.16	0.09	5X10-6	7X10 <sup>-6</sup>	1X10-6	5X10-7	4X10-7	5X10-6	1X10 <sup>-7</sup>	7X10 <sup>-8</sup>	2X10 <sup>-6</sup>	3X10-₀	
•								•					
	Negligik	ole risks						<1 x 10 <sup>-6</sup>					
Tolerable	acceptak	ole risks					≥1 x 1	0-6 and ≤1 x 10-4					
U	nacceptat	ole risks						>1 x 10-4					

Table 6-26 Calculated individual risk associated with changes in PM<sub>2.5</sub> and PM<sub>10</sub> concentrations - project operations in 2036 Cumulative





# Figure 6-7 Calculated change in individual risk at community receptors from total change in $PM_{2.5}$ concentrations (primary health end points) - project in 2026 and 2036

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

LGA			Change i	n population ir	cidence – number	of cases				
		Primary indicate	ors	Secondary indicators						
	Mortality – All Causes	Hospitalisations – Cardiovascular	Hospitalisations – Respiratory	Mortality – All causes	Mortality – Cardiopulmonary	Mortality – Cardiovascular	Mortality – Respiratory	Morbidity – Asthma ED admissions		
	≥30 years	≥65 years	≥65 years	All ages	≥30 years	All ages	All ages	1–14 years		
With Project										
Strathfield - Burwood - Ashfield LGA	-0.0039	-0.00092	-0.00020	-0.00032	-0.0035	-0.000098	-0.000096	-0.00026		
Sydney Inner City LGA	-0.00036	-0.000064	-0.000014	-0.000053	-0.00033	-0.000015	-0.000010	-0.000011		
Marrickville - Sydenham - Petersham LGA	-0.013	-0.0030	-0.00066	-0.0016	-0.011	-0.00045	-0.00031	-0.00084		
Canterbury LGA	-0.00012	-0.000036	-0.0000080	-0.000013	-0.00011	-0.0000039	-0.0000033	-0.000012		
Botany LGA	-0.028	-0.0076	-0.0017	-0.0037	-0.025	-0.0010	-0.00074	-0.0022		
Kogarah - Rockdale LGA	0.015	0.0047	0.0010	0.0015	0.014	0.00044	0.00039	0.0011		
Hurstville LGA	0.00033	0.00010	0.000023	0.000039	0.00030	0.000011	0.0000085	0.000024		
Total for all LGAs	-0.030	-0.0068	-0.0015	-0.0041	-0.027	-0.0011	-0.00077	-0.0022		

Table 6-27 Calculated changes in incidence of health effects in population associated with changes in PM<sub>2.5</sub> concentrations – project in 2026

Negative value indicates that there is a decrease in incidence associated with the project

LGA		Change in population incidence – number of cases										
		Primary Indicat	ors		Sec	ondary Indicators						
	Mortality – All Causes	Hospitalisations – Cardiovascular	Hospitalisations – Respiratory	Mortality – All causes	Mortality – Cardiopulmonary	Mortality – Cardiovascular	Mortality – Respiratory	Morbidity – Asthma ED Admissions				
	≥30 years	≥65 years	≥65 years	All ages ≥30 years		All ages	All ages	1–14 years				
With Project												
Strathfield - Burwood - Ashfield LGA	-0.0017	-0.00041	-0.000090	-0.00014	-0.0015	-0.000043	-0.000043	-0.00011				
Sydney Inner City LGA	-0.0089	-0.0016	-0.00035	-0.0013	-0.0080	-0.00037	-0.00024	-0.00027				
Marrickville - Sydenham - Petersham LGA	-0.018	-0.0043	-0.00094	-0.0023	-0.0023 -0.016		-0.00044	-0.0012				
Canterbury LGA	-0.0025	-0.00074	-0.00016	-0.00027	-0.0022	-0.000079	-0.000067	-0.00025				
Botany LGA	-0.049	-0.013	-0.0029	-0.0065	-0.044	-0.0018	-0.0013	-0.0038				
Kogarah - Rockdale LGA	0.0052	0.0016	0.00036	0.00053	0.0047	0.00015	0.00013	0.00037				
Hurstville LGA	0.00042	0.00013	0.000029	0.000050	0.00038	0.000014	0.000011	0.000030				
Total for all LGAs	-0.074	-0.018	-0.0041	-0.010	-0.067	-0.0027	-0.0019	-0.0053				
Cumulative												
Strathfield - Burwood - Ashfield LGA	-0.00092	-0.00022	-0.000048	-0.000077	-0.00083	-0.000023	-0.000023	-0.000061				
Sydney Inner City LGA	-0.0069	-0.0012	-0.00027	-0.0010	-0.0062	-0.00029	-0.00019	-0.00021				
Marrickville - Sydenham - Petersham LGA	-0.0025	-0.00060	-0.00013	-0.00032	-0.0023	-0.000090	-0.000062	-0.00017				
Canterbury LGA	-0.00060	-0.00018	-0.000039	-0.000065	-0.00054	-0.000019	-0.000016	-0.000059				
Botany LGA	-0.038	-0.010	-0.0023	-0.0051	-0.035	-0.0014	-0.0010	-0.0030				
Kogarah - Rockdale LGA	-0.074	-0.023	-0.0051	-0.0076	-0.067	-0.0022	-0.0019	-0.0053				
Hurstville LGA	-0.000072	-0.000022	-0.0000049	-0.000086	-0.000065	-0.0000024	-0.0000018	-0.0000051				
Total for all LGAs	-0.12	-0.036	-0.0078	-0.014	-0.11	-0.0040	-0.0032	-0.0088				

Table 6-28 Calculated changes in incidence of health effects in population associated with changes in PM<sub>2.5</sub> concentrations – project in 2036

Negative value indicates that there is a decrease in incidence associated with the project

Review of the calculated changes in risk indicates the following in relation to impacts associated with the expected operation of the project in 2026 and 2036, including the cumulative scenario:

- A number of the calculated individual risks as shown in **Figure 6-7** for the community receptors are negative, meaning that the operation of the project would result in lower levels of risk, when compared with the situation where the project is not operating
- The maximum risks calculated for exposures in residential areas are less than 1x10<sup>-4</sup> and considered to be tolerable/acceptable
- The maximum risks calculated for exposures in commercial/industrial areas are less than 1x10<sup>-4</sup> and considered to be tolerable/acceptable
- All maximum risks calculated for continuous exposures in childcare centres, schools, aged care homes and open space areas are below 1x10<sup>-4</sup> and considered to be tolerable/ acceptable
- Depending on the scenario the risks from changes in PM<sub>2.5</sub> and PM<sub>10</sub> exposures may be driven by the ventilation facilities or the roadway emissions (**Table 6-24** to **Table 6-26**)
- In relation to impacts on the health of the population in the local community, the calculated change in incidence of the health indicators evaluated shows that the increased incidence of the evaluated health effects occurring in the population in the study area ranges from 0.001 to 0.11 cases per year, which would not be measurable and is considered to be negligible.

Review of the calculated impacts in terms of the change in incidence of the relevant health effects for  $PM_{2.5}$  in the community, indicates the following:

- The total change in the number of cases relevant to the health effects evaluated, for both 2026 and 2036 is negative, meaning a decrease in incidence as a result of the project. The number of cases, however is very small, less than one for all health effects considered. As a result, these changes would not be measurable within the community
- Most individual LGAs show a total decrease in health incidence. There are two LGAs (Kogarah-Rockdale and Hurstville) where there is an increase. These increases and decreases are also very small, less than one for all health effects considered. As a result, these changes would not be measurable in the community
- The incidence calculations presented in **Table 6-27** and **Table 6-28** are the totals for each LGA. Within these LGAs are a number of smaller suburbs. The calculated change in incidence relevant to each of these suburbs has also been evaluated, as presented in Annexure G. Review of the incidence calculated for the individual suburbs indicates that these predominantly relate to small decreases in health incidence with some suburbs showing an increase. The largest increase in health incidence for any individual suburb is less than 0.1 case. Hence there are no individual suburbs within the LGAs where there is a change incidence that is of significance or would be measurable.

### **Elevated receptors**

The calculations presented in the above relate to inhalation exposures that may occur at ground level (ie within typical low to medium density residential homes and commercial/industrial properties).

**Appendix E** (Air quality technical report) has conducted a screening assessment of potential issues related to exposures that may occur at elevated receptors, close to ventilation outlets, to identify areas that may need to have more detailed analysis and where future development controls may be required for high-rise buildings. This has been undertaken on the basis of evaluating predicted concentrations of  $PM_{2.5}$  at 10 metres, 20 metres, 30 metres and 45 metres above the ground level, representative of potential exposures that may occur in multi-storey buildings. The assessment undertaken has evaluated impacts at 10 metres, 20 metres, 30 metres and 45 metres across the whole study area, regardless of whether a multi-storey building is present or not. Impacts that are derived from changes in emissions from surface roads are expected to decrease with height above the roadway, however in areas closest to the ventilation outlets there is the potential for increased impacts with height.

The assessment of potential impacts at 10 metres, 20 metres, 30 metres and 45 metres height has focused on the cumulative scenario in the year 2036 where impacts from the F6 Extension, Western Harbour Tunnel and Warringah Freeway Upgrade, Beaches Link and Gore Hill Connection, Sydney Gateway and WestConnex projects are included. The maximum change in  $PM_{2.5}$  relevant to this scenario has been evaluated. As the approach adopted in **Appendix E** (Air quality technical report) is a screening level assessment no other pollutants have been evaluated.

**Table 6-29** presents the calculated risks associated with the maximum predicted change in  $PM_{2.5}$  concentrations at a height of 10 metres, 20 metres, 30 metres and 45 metres above ground level throughout the study area. It is noted that these maximum impacts do not relate to existing multi-storey buildings, rather these are the maximum impacts anywhere in the study area and have been included to evaluate potential future development.

Table 6-29 Calculated individual risk associated with changes in  $PM_{2.5}$  concentrations - cumulative scenario in 2036 for elevated receptors

Health endpoint	Maximum calculated					
	10 m	20 m	30 m	45 m		
	height	height	height	height		
Annual average concentration						
PM <sub>2.5</sub> (µg/m³)	1.4	0.23	0.30	1.6		
Primary health indicators: PM <sub>2.5</sub>						
Mortality all causes (long term effects, ages 30+)	8 x 10 <sup>-5</sup>	1 x 10 <sup>-5</sup>	2 x 10 <sup>-5</sup>	1 x 10 <sup>-4</sup>		
Cardiovascular hospitalisations (short term effects, ages 65+)	1 x 10 <sup>-4</sup>	2 x 10 <sup>-5</sup>	2 x 10 <sup>-5</sup>	1 x 10 <sup>-4</sup>		
Respiratory hospitalisations (short term effects, ages 65+)	2 x 10 <sup>-5</sup>	4 x 10 <sup>-6</sup>	5 x 10⁻	3 x 10 <sup>-5</sup>		
Secondary health indicators: PM <sub>2.5</sub>	1					
Mortality all causes (short term effects, all ages)	6 x 10-6	1 x 10 <sup>-6</sup>	1 x 10 <sup>-6</sup>	7 x 10 <sup>-6</sup>		
Mortality, cardiopulmonary (long term effects, ages 30+)	7 x 10-5	1 x 10 <sup>-5</sup>	2 x 10 <sup>-5</sup>	9 x 10 <sup>-5</sup>		
Mortality, cardiovascular (short term effects, all ages)	2 x 10⁻	3 x 10 <sup>-7</sup>	4 x 10 <sup>-7</sup>	2 x 10 <sup>-6</sup>		
Mortality, respiratory (short term effects, all ages)	1 x 10⁻	2 x 10 <sup>-7</sup>	2 x 10 <sup>-7</sup>	1 x 10 <sup>-6</sup>		
Asthma emergency department hospitalisations (1–14 years)	3 x 10-5	4 x 10-6	5 x 10-6	3 x 10-5		
Negligible risks		<1	x 10 <sup>-6</sup>			
Tolerable/acceptable risks	≥1 x 10 <sup>-6</sup> and ≤1 x 10 <sup>-4</sup>					
Unacceptable risks	>1 x 10 <sup>-4</sup>					

The calculations presented in Table 6-29 indicate the following:

- The maximum change in PM<sub>2.5</sub> decreases by around 5 fold with increasing height from 10 to 30 metres. PM<sub>2.5</sub> concentrations increase at 45 metres.
- All calculated risks at elevated receptors, at 10 metres, 20 metres and 30 metres height are considered to range of tolerable/acceptable.
- At 45 metres height the calculated risk is equal to the level above which risks are considered to be unacceptable. Review of the maximum impacts predicted indicates that these are close to the ventilation outlets. There are currently no multi-storey buildings located close to the proposed ventilation outlets and hence the maximum calculated risks presented are hypothetical at this stage.

To address the potential health impacts identified, planning controls should be developed in the vicinity of the proposed ventilation facilities to ensure future developments at heights above 30 metres are not adversely impacted by the ventilation outlets. Development of planning controls would be supported by detailed modelling addressing all relevant pollutants and averaging periods.

## 6.10 Assessment of regulatory worst-case scenario

A regulatory worst-case scenario has been evaluated in **Appendix E** (Air quality technical report). This is based on the situation where emissions to air from the tunnel ventilation outlets occur at the maximum discharge limits at all hours of the day. This may occur in the event of a breakdown or accident and may result in a short period of time where emissions from the tunnel ventilation facility are higher than during normal operations. Such situations are not planned and where they occur the duration of the event is not expected to last for longer than a few hours.

The assumptions underpinning the all regulatory worst-case scenarios were conservative and resulted in contributions from project ventilation outlets that were much higher than those that could ever occur under any operational conditions in the tunnel.

In relation to impacts on health a worst-case situation results in short-term changes in air quality. Hence health effects identified and evaluated in this assessment that relate to changes in short-term concentrations of  $PM_{2.5}$  require further assessment. The assessment of short-term health impacts has utilised the methodology outlined in Annexure A with the parameters selected to be relevant to a one-hour or 24-hour exposure period (as relevant to each pollutant). The assessment has considered short-term change in air concentrations associated with maximum emissions from the ventilation outlets from the project tunnels in 2036 for the cumulative scenario.

Risk calculations can be undertaken for the short-term change in air quality associated with each of these scenarios. How often these events occur during any one year may result in some contribution to the total annual individual risk calculated for the expected operation of the project. The frequency of a worst-case traffic scenario occurring is not known, hence for the purpose of this assessment some conservative assumptions have been adopted.

**Table 6-30** presents the calculated change in individual risk associated with residential exposure to worst-case emissions of  $PM_{2.5}$ . The table includes the assumptions adopted for the assessment.

# Table 6-30 Maximum calculated risk associated with short-term residential exposure changes in PM2.5 concentrations: regulatory worst case 2036 cumulative scenaria

Scenario	Maximum change in individual risk for the following short-term health endpoints								
	Cardiovascular hospitalisations (65 years+)	Respiratory hospitalisations (65 years +)	Mortality all causes (all ages)	Mortality cardiovascular (all ages)	Mortality respiratory (all ages)	Asthma ED admissions (1–14 years)			
The project									
Maximum annual risk – expected operations	3 x 10 <sup>-5</sup>	6 x 10 <sup>-6</sup>	1 x 10 <sup>-6</sup>	5 x 10 <sup>-7</sup>	3 x 10 <sup>-7</sup>	7 x 10 <sup>-6</sup>			
Increase in risk for 1 day of worst-case emissions (24 hours which is highly conservative)	4 x 10 <sup>-7</sup>	8 x 10 <sup>-8</sup>	2 x 10 <sup>-8</sup>	7 x 10 <sup>.9</sup>	5 x 10-9	9 x 10 <sup>-8</sup>			
Increase in risk assuming worst-case event occurs 1 day each week (52 days per year)*	2 x 10 <sup>-5</sup>	4 x 10 <sup>-6</sup>	1 x 10 <sup>-6</sup>	3 x 10 <sup>-7</sup>	2 x 10 <sup>-7</sup>	5 x 10-6			
Maximum annual risk – expected conditions plus worst-case event**	5 x 10 <sup>-5</sup>	1 x 10 <sup>-5</sup>	2 x 10 <sup>-6</sup>	8 x 10 <sup>-7</sup>	5 x 10 <sup>-7</sup>	1 x 10 <sup>-5</sup>			
Negligible risks	< 1 x 10 <sup>-6</sup>								
Tolerable/acceptable risks	$\geq$ 1 x 10 <sup>-6</sup> and $\leq$ 1 x 10 <sup>-4</sup>								
Unacceptable risks	> 1 x 10-4								

\* Assumes that the maximum predicted impact occurs at the same location (receptor) every day the worst-case event occurs. With changes in meteorology in the local area the 24-hour maximum concentration is expected to change in concentration and location over different days. Hence this assumption is conservative

\*\* Assumes the maximum annual average impact and maximum short-term change occur that the same location (receptor) 1 day per week

Review of the maximum calculated changes in risk associated with short-term changes in  $PM_{2.5}$  (**Table 6-30**) concentration under the worst-case scenarios evaluated indicates the following:

 The maximum change in short-term risk associated with worst-case scenarios occurring on any one day is negligible

- Where it is conservatively assumed that the worst-case scenario occurs one day each week (and the maximum changes impact occurs at the same receptor location every time), the maximum individual risk increases
- The total maximum individual risk increases to but does not exceed 1x10<sup>-4</sup> and hence there are no unacceptable risks identified in the community surrounding the project
- The calculated maximum individual risks are in the range 1x10<sup>-6</sup> to 1x10<sup>-4</sup> and are considered to range from negligible to tolerable/acceptable.

On the basis of the above, emissions from the ventilation outlets during a worst-case scenario (such as a breakdown or accident) has the potential to increase individual risks, however the maximum individual risks (even where conservative assumptions are adopted) are considered to be tolerable/acceptable.

# 6.11 Sensitivity analysis

A sensitivity analysis was undertaken to determine the impact from emissions where the emission limit for the ventilation outlets were reached for at least 1 hour every day. **Figure 6-8** shows the different contributions to  $PM_{2.5}$  concentrations for the expected traffic conditions (for background plus traffic), the sensitivity test (1 hour per day  $PM_{2.5}$  concentrations reach the emission limit) and regulatory worse case (24 hours per day of  $PM_{2.5}$  concentrations reaching the emission limit) for the 2036 do something cumulative scenario. This figure essentially shows that all assumptions for ventilation outlets result in relatively small contributions compared with the total.



# Figure 6-8 Results of sensitivity tests for ventilation outlets - total annual mean PM2.5 concentration at RWR receptors (2036-DSC scenaria)

In relation to potential impacts on health, risk calculations have been undertaken for the change in  $PM_{2.5}$  (for the primary health endpoints) and  $NO_2$ . These risk calculations have been undertaken for the 2036 cumulative scenario, consistent with the scenario evaluated in the Air Quality Impact Assessment.

**Table 6-31** presents the maximum calculated risk, from all receptors, associated with the change in  $PM_{2.5}$  and  $NO_2$ , for the expected traffic conditions and the sensitivity test.

Table 6-31 Calculated individual risk associated with maximum changes in PM2.5 and NO2 concentrations: sensitivity test – 2036 cumulative scenario

Health endpoint	Max	kimum calculated
	Expected traffic	Sensitivity test
Primary health indicators: PM <sub>2.5</sub>		
Mortality all causes (long term effects, ages 30+)	2 x 10 <sup>-5</sup>	6 x 10 <sup>-5</sup>
Cardiovascular hospitalisations (short term effects, ages 65+)	3 x 10 <sup>-5</sup>	7 x 10 <sup>-5</sup>
Respiratory hospitalisations (short term effects, ages 65+)	6 x 10 <sup>.6</sup>	2 x 10 <sup>-5</sup>
Health indicators: NO <sub>2</sub>		
Mortality all causes (short term effects, all ages)	1 x 10 <sup>-5</sup>	2 x 10 <sup>-5</sup>
Mortality, respiratory (short term effects, all ages)	3 x 10-6	4 x 10 <sup>-6</sup>
Asthma emergency department hospitalisations (1–14 years)	2 x 10 <sup>-5</sup>	3 x 10-5
	1	
Negligible risks		<1 x 10 <sup>-6</sup>
Tolerable/acceptable risks	≥1	x 10 <sup>-6</sup> and ≤1 x 10 <sup>-4</sup>
Unacceptable risks		>1 x 10 <sup>-4</sup>

Review of the maximum calculated changes in risk associated with changes in  $PM_{2.5}$  and  $NO_2$  concentrations relevant to the sensitivity test scenario evaluated indicates the following:

- For NO<sub>2</sub> the sensitivity test shows a very small increase in the maximum calculated risks. The calculated risks, however remain low and are considered tolerable/acceptable
- For PM<sub>2.5</sub> the sensitivity test shows a small increase in the maximum calculated risks. The calculated risks, however remain low and are considered tolerable/acceptable.

On the basis of the above, emissions from the ventilation outlets, where the sensitivity test scenario is considered, has the potential result in a small increase in  $NO_2$  and  $PM_{2.5}$  risks, however the maximum individual risks associated with  $PM_{2.5}$  and  $NO_2$  are considered to be tolerable/acceptable.

## 6.12 Valuing particulate impacts

The SEARs (as outlined in **Section 1.4**) requires the assessment of health impacts to also evaluate costs to the community. More specifically the SEARs have indicated that costs should be evaluated on the basis of the following guidance document:

• Methodology for Valuing the Health Impacts of Changes in Particle Emissions (EPA 2013).

This guideline has developed an approach for use in Australia that is based on the approach developed in the UK. The approach adopted is simplistic, relating health costs in the community to changes in total tonnes of  $PM_{2.5}$  emitted. This calculation has generalised the health impacts associated with changes in  $PM_{2.5}$  exposures as emitted to air and does not specifically address how people are exposed to these emissions (this is assumed to occur). **Appendix E** (Air quality technical report) has calculated the tonnes of  $PM_{2.5}$  relevant to each of the scenarios evaluated for this project. This relates to the total tonnes of  $PM_{2.5}$  emitted to air and this shows a small decrease in  $PM_{2.5}$  with the project in 2026 and increase in 2036, including the 2036 cumulative scenario.

The assessment of potential health effects associated with the change in  $PM_{2.5}$  concentrations the community are exposed to, however are different, and as discussed in **section 6.9.5**, **Table 6-27** and **Table 6-28**, the project is associated with a decrease in incidence, or the number of cases, relevant to mortality and hospitalisations (ie a health benefit). These impacts, ie the change in number of cases, ideally should be those that are considered in valuing the health impacts. Where this is considered a reduction in health costs should be calculated. However, that is not the case with the methodology outlined by NSW EPA (2013) which is only based on the change in total tonnes of  $PM_{2.5}$  emitted. As a result, the calculations presented are not considered representative of health costs related to the project.

When applying the NSW EPA (2013) methodology, the project area has been assumed to be urban large (noting there are no definitions in the guidance in relation to determining this), where the damage costs listed are \$593,617 per tonne of  $PM_{2.5}$  in 2011 prices. In today's prices, based on the inflation calculator from the Reserve Bank of Australia<sup>9</sup> the damage cost is \$664,773 per tonne of  $PM_{2.5}$ . Following this approach, the damage costs / saving associated with changes in  $PM_{2.5}$  are calculated to be minus \$1,329,546 (saving) in 2026 and \$664,773 (cost) in 2033, with the cumulative scenario resulting in a cost that is \$1,329,546 (cost) in 2036. As noted above these costs are not considered to be representative for the project.

<sup>&</sup>lt;sup>9</sup> http://www.rba.gov.au/calculator/annualDecimal.html

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

# 7 Assessment of in-tunnel air quality

# 7.1 General

The in-tunnel air quality has been evaluated for the following reasons:

- To design and control ventilation systems. Tunnel builders and operators aim to minimise the significant costs involved in providing active ventilation. As a result, systems are designed, built and operated to provide sufficient ventilation to maintain acceptable air quality in the tunnel, but at reasonable cost (NHMRC 2008)
- To manage in-tunnel exposure to air pollution
- To manage external air pollution.

Traditionally, the approach to considering air quality within tunnels was based on managing carbon monoxide levels. However, modern petrol fuelled cars now have low levels of carbon monoxide emissions, and with an increasing proportion of diesel fuelled cars, a number of countries are considering the use of nitrogen dioxide concentrations for tunnel ventilation design.

Another important consideration for tunnel ventilation design is visibility. Consideration of visibility criteria in the design of the tunnel ventilation system is required due to the need for visibility levels that exceed the minimum vehicle stopping distance at the design speed. Visibility is reduced by the scattering and absorption of light by PM suspended in the air. The amount of light scattering or absorption is dependent upon the particle composition (dark particles, such as soot, are particularly effective), diameter (particles need to be larger than around 0.4 micrometres), and density. Particles causing a loss of visibility also have an effect on human health, and so monitoring visibility also provides the potential for an alternative assessment of the air quality and health risk within a tunnel. However, such an assessment is limited by the short duration of exposure in tunnels compared with the longer exposure times (24 hours and one year) for which the health effects of ambient particles have been established. Moreover, there is no safe minimum threshold for particles, and so visibility cannot reliably be used as a criterion for health risk (NHMRC 2008). Hence visibility limits within the tunnel have not been further evaluated.

The operational in-tunnel limits for carbon monoxide and nitrogen dioxide in several Sydney road tunnels are shown in **Table 7-1**. With the current pollution limits, and for the assessment years of the F6 extension project,  $NO_2$  would be the pollutant that determines the required air flows and drives the design of ventilation for in-tunnel pollution.

Tunnel	(1	CO concentration opm, rolling average	NO₂ concentration (ppm)		
	3 min	15 min	30 min	15 min	
Cross City Tunnel	200	87	50	N/A	
Lane Cove Tunnel	-	87	50	N/A	
M5 East Tunnel	200	87	50	N/A	
NorthConnex					
WestConnex M4 East	<b>200</b> (a)	<b>07</b> (h)	FO(b)	0.5 <sup>(b)</sup>	
WestConnex New M5	200(8)	0709	50(%)		
M4 M5 Link					

Table 7-1	Operational	limits in	Sydney	road	tunnels
	Operational	11111115 111	Syune	y i Uau	luiineis

(a) In-tunnel single point exposure limit

(b)In-tunnel average limit along tunnel length

Sources: NHMRC (2008), Longley (2014c), PIARC (visibility), NSW Government (2015, 2016a, 2016b)

In February 2016, the NSW Government Advisory Committee on Tunnel Air Quality (ACTAQ) issued a document entitled 'In-tunnel air quality (nitrogen dioxide) policy' (ACTAQ, 2016). That document further consolidated the approach taken earlier for the NorthConnex, M4 East and New M5 projects. The policy wording requires tunnels to be 'designed and operated so that the tunnel average nitrogen dioxide (NO2) concentration is less than 0.5 ppm as a rolling 15 minute average'.

For the F6 tunnel the 'tunnel average' has been interpreted as a 'route average', being the 'length-weighted average pollutant concentration over a portal-to-portal route through the system'. Tunnel average  $NO_2$  has been assessed north and southbound from the New M5 to President Ave as highlighted in **Table 7-2**.

St	art at	Finis	Approximate length		
Southbound dire	ection				
New M5	St Peters	F6 Extension	President Ave	6.7 km	
New M5	M4-M5 link interface	F6 Extension	President Ave	6.7 km	
Northbound dire	ction				
F6 Extension	President Ave	New M5	St Peters	6.8 km	
F6 Extension	President Ave	New M5	M4-M5 link interface	6.7 km	

### Table 7-2 List of routes assessed

The tunnel ventilation system would be designed and operated so that the in-tunnel air quality limits, consistent with those in the conditions of approval for NorthConnex and other approved WestConnex projects are not exceeded.

A number of factors have been considered in this assessment. Firstly, concentrations in the tunnel are expected to vary depending on the location within the main alignment tunnels and ventilation facilities. Concentrations of pollutants would gradually increase from the tunnel entrance to the next offtake to a ventilation outlet. Second, the concentration of pollutants within the vehicle itself would be lower, particularly where all windows are closed when inside the tunnel, as most vehicles have filters on the air intake. Where the air conditioning/ventilation in the car is set to recirculation this would limit the contribution of air derived from within the tunnel to the air within the vehicle. Measurements conducted by NSW Health in relation to the M5 East Tunnel (NSW Health 2003) identified that closing car windows and switching the ventilation to recirculation can reduce exposures by about 70-75 per cent for carbon monoxide and nitrogen dioxide, 80 per cent for fine particulates and 50 per cent for volatile organic compounds. Further testing of the reduction in nitrogen dioxide levels inside vehicles using road tunnels was commissioned by Roads and Maritime in 2016 (PEL 2016), where recirculation was found to reduce exposures by around 70 per cent. Finally, there may be individuals who utilise the network of tunnels in the Sydney area on a frequent basis, throughout the day. This includes taxi drivers, courier drivers and some truck drivers and use other tunnel systems in conjunction with the F6 extension. More frequent and cumulative exposures in these tunnels are considered below.

The following provides further discussion on the range of concentrations predicted within the F6 extension tunnel.

# 7.2 Carbon monoxide

**Table 7-3** presents the maximum in-tunnel concentration of carbon monoxide predicted in the F6 Extension. The table presented is for the year 2036 cumulative scenario, that is with all tunnels in consideration.

Time Period	CO	30 minute CO criteria (ppm)	
	Southbound	Northbound	
7am – 9am	4.3	1.2	48.7*
9am – 3pm	5.1	0.8	48.7
3pm – 6pm	7.8	0.7	48.7
6pm – 7am	2.9	0.5	48.7

#### Table 7-3 Maximum estimated in-tunnel air quality for CO based on expected traffic in 2036

\* The modelling has been undertaken without consideration of CO background concentrations of 1.3 ppm. Therefore 1.3 ppm is subtracted from the 30 minute criteria of 50 ppm

In relation to the carbon monoxide concentrations predicted within the tunnel, the following is noted:

- The maximum one hour average concentration of carbon monoxide in the tunnels is predicted to be less than 10 ppm in both direction for all times of the day. These concentrations are lower than the health based guideline of 25 ppm (one-hour average) established by the WHO (WHO 2010) and 34 ppm established by the USEPA (NHMRC 2008). The concentrations are lower than PIARC in-tunnel limits (Longley 2014)
- The NHMRC (2008) has published measured concentrations of carbon monoxide from a range of tunnels in Sydney and around the world. The measured concentrations come from a number of different studies where the averaging time for the collection of the data varies significantly. This makes it difficult to directly compare the range of reported concentrations with the concentrations predicted in this assessment (ie not comparing data reported over similar averaging/exposure periods). While noting this difficulty in comparing the data, a range of average concentrations of carbon monoxide have been reported from six to 38 ppm (NHMRC 2008). The predicted hourly average concentration in the project tunnel is within the range reported in other tunnels

On the basis of the above, there are no health issues of concern related to in-tunnel exposures to carbon monoxide. This relates to exposures that may occur in the F6 Extension Stage 1.

# 7.3 Nitrogen dioxide

**Table 7-4** presents the maximum route average concentration of nitrogen dioxide predicted in the F6 Extension, while travelling in both directions. The table presented is for the year 2036 cumulative scenario, that is with all tunnels in consideration. Stacey Agnew have stated that a previous in-tunnel assessment undertaken for the M4-M5 link, that considered all possible tunnel travel routes (including the F6 extension) remain valid. This assessment showed that the in-tunnel nitrogen dioxide concentrations for all trips fell below the 0.5 ppm criteria.

Time Period		Criteria (ppm)			
	St Peters to President Ave	M4-M5 to President Ave	President Ave to St Peters		
7am – 9am	0.14	0.18	0.12	0.11	0.47*
9am – 3pm	0.15	0.20	0.07	0.07	0.47
3pm – 6pm	0.19	0.23	0.05	0.05	0.47
6pm – 7am	0.07	0.10	0.03	0.03	0.47

Table 7-4: Maximum estimated in-tunnel air quality for CO based on expected traffic in 2036

\* The modelling has been undertaken without consideration of NO<sub>2</sub> background concentrations of 1.3 ppm. Therefore 0.03 ppm is subtracted from the 50 ppm criteria

In relation to the nitrogen dioxide concentrations predicted within the F6 Extension tunnel, the following is noted:

- The maximum concentrations in the F6 tunnel vary throughout the day, with the maximum concentration predicted at any time of the day less than 0.5 ppm.
- The NHMRC (2008) has published measured concentrations of nitrogen dioxide from a range of tunnels in Sydney and around the world. The measured concentrations come from a number of different studies where the averaging time for the collection of the data varies significantly. This makes it difficult to directly compare the range of reported concentrations with the concentrations predicted in this assessment (ie not comparing data reported over similar averaging/exposure periods). While noting this difficulty in comparing the data, the NHMRC (2008) have reported a range of average concentrations of nitrogen dioxide in tunnels that range from 0.05 to 0.3 ppm with levels up to 0.4 ppm reported during peak periods. These levels are based on data with averaging times that vary from 30 seconds during travel through a tunnel, six minute averages, to long term data with (unspecified averaging times). At the downstream end of a tunnel (where exposure is very short, ie minutes) levels up to 0.8 ppm have been reported.

The concentrations discussed above relate to nitrogen dioxide levels inside the tunnels, not inside the vehicles. A study of nitrogen dioxide concentrations inside vehicles travelling in Sydney and using existing road tunnels was commissioned by Roads and Maritime in 2016 (PEL 2016) to better understand the relationship between nitrogen dioxide outside the vehicle, and inside the vehicle. The study involved a range of vehicles considered representative of the existing vehicle fleet, travelling through existing tunnels in Sydney and simulating travel times between 45 minutes and 60 minutes over a distance of 30 kilometres.

The concentration of nitrogen dioxide that entered a vehicle depended on the concentration outside the vehicle as well as the air exchange rate relevant to the individual vehicle. The air exchange rate depends on the ventilation, whether on recirculation or not, and a range of factors relevant to the vehicle air tightness, or leakiness.

Within existing tunnels utilised in the study, concentrations of nitrogen dioxide were generally less than 0.15 ppm, however during periods of high traffic volume and a high proportion of heavy vehicles, the concentrations inside existing tunnels exceeded 0.5 ppm, with levels up to 0.7 ppm. Inside these tunnels with high external concentrations of nitrogen dioxide, the average concentrations inside the vehicles, when ventilation was on recirculation was less than 0.2 ppm.

The study found that the use of ventilation on recirculation can significantly reduce concentrations of nitrogen dioxide inside vehicles. The ratio of indoor to outdoor concentrations ranged from 0.06 to 0.32. This is consistent with the findings from a NSW Health study on vehicles using the M5 East tunnel, where an indoor to outdoor ratio of 0.25 to 0.3 was determined for nitrogen dioxide where ventilation is set to recirculation. When ventilation was not set to recirculation the concentration of nitrogen dioxide was higher inside the vehicles, and in some cases accumulated inside the vehicle after travelling through short tunnels.

### 7.3.1 Health effects of short-duration exposures to nitrogen dioxide

Short term exposure to nitrogen dioxide has been shown to cause respiratory health effects and is suspected of causing other health impacts such as cardiovascular effects (US EPA 2016). The concentration at which these impacts occur was subject to a review in 2015 (Jalaludin 2015). This review, which has been used to develop the NSW NO<sub>2</sub> in tunnel guideline, evaluated available studies in relation to health effects from in-tunnel and short term exposures to nitrogen dioxide. The review evaluated studies associated with exposures that occur for less than 30 minutes as well as those with exposures of more than 60 minutes.

In relation to the available studies (18 studies) that relate to exposures of 30 minutes or less, the review identified the following (Jalaludin 2015):

- There were no effects identified in relation to lung function for individuals exposed to nitrogen dioxide between 0.12 and 0.5 ppm
- The results for inflammatory markers (physiological measures that indicate the respiratory system or other systems in the body are dealing with inflammation) are mixed
- An effect of exposure to nitrogen dioxide and airway responsiveness was identified in individuals with asthma
- There is no clear evidence of a dose-response relationship for exposure and airway responsiveness for nitrogen dioxide levels at or below 0.5 ppm
- The effects observed for airway responsiveness may be transient. There is no clear evidence that repeated exposure to nitrogen dioxide leads to cumulative effects.

In relation to the available studies (14 studies) that relate to exposures of 60 minutes or more, the review identified the following (Jalaludin 2015):

- There were no effects identified in relation to lung function for individuals exposed to nitrogen dioxide between 0.3 and 4 ppm
- The results for inflammatory markers are mixed, however overall, inflammatory markers increased after exposure to nitrogen dioxide
- An effect of exposure to nitrogen dioxide and airway responsiveness was identified
- Insufficient data is available to determine any cardiovascular effects (or otherwise)
- One study indicated the effects were attenuated with repeated exposures.

In relation to the available studies (eight studies) from road tunnels, busy roads and subways, the review identified the following (Jalaludin 2015):

- Exposures to nitrogen dioxide were in the range of less than 0.2 ppm (in seven studies) to 0.5 ppm (in one study)
- There were no effects identified in relation to lung function
- Both upper and lower respiratory symptoms were commonly reported after exposure to road tunnel and subway environments
- The results for inflammatory markers are mixed
- The effects on airway responsiveness were unclear.

More recently, another review (EnRiskS 2018) was undertaken to consider NO<sub>2</sub> exposures of up to 60 minutes. This review supported the conclusions of the Jalaludin report, even for exposures of NO<sub>2</sub> up to 60 minutes. It found that for NO<sub>2</sub> exposures 0.5 ppm or less, the strongest evidence for effects were seen on airways responsiveness, and generally in asthmatics. These effects, if detected were small and not defined to be clinically relevant.

However, there were limitations in the studies, in particular the small number of participants and the lack of subjects who are more sensitive to effects of nitrogen dioxide. Further, when considering the studies conducted in road tunnels, busy roadways and in subways it is important to note that nitrogen dioxide is only part of a complex mixture of air pollution, including PM<sub>2.5</sub>, and determining health effects that may be only related to nitrogen dioxide is difficult.

For the assessment of short duration exposures to nitrogen dioxide in road tunnels, Australia along with a number of other jurisdictions, have established guidelines. These guidelines are based on the available short term studies which have been considered in the review presented by (Jalaludin 2015) and (EnRiskS 2018).

**Table 7-5** presents a summary of the available guidelines for the assessment of short duration exposures to nitrogen dioxide within tunnels.

Jurisdiction/Project	Guideline	Averaging period	Nature of guideline (tunnel design or compliance)
NSW (ACTAQ 2016)	0.5 ppm tunnel average	15 minutes	Design and compliance
NorthConnex and WestConnex	0.5 ppm tunnel average	15 minutes	Design and compliance
Brisbane City Council/Clem 7 and LegacyWay tunnels	1 ppm tunnel average	NA	Design
PIARC	1 ppm tunnel average	NA	Design
New Zealand	1 ppm	15 minutes	Design
Belgium	0.5 ppm tunnel average	<20 minutes	Design
France	0.4 ppm tunnel average	15 minutes	Design
Norway	0.75 ppm at midpoint in tunnel	15 minutes	Design and compliance
Hong Kong	1 ppm	5 minutes	Design

 Table 7-5: Summary of nitrogen dioxide guidelines for in-tunnel exposures

## 7.3.2 Further consideration of potential exposures within tunnels

The average concentration of nitrogen dioxide has been calculated for the north and south bound trips through the F6 Extension. However, users of the tunnel network are likely to travel further in the connecting tunnel networks. A previous in-tunnel assessment undertaken for the M4-M5 link that considered all possible tunnel travel routes including those that emanated or ended through the F6 extension. In the current in-tunnel assessment (Annexure K to **Appendix E** Air quality technical Report) it is confirmed that;

The ventilation system of New M5 and F6 Extension, as outlined in this report, meets or exceeds the functional performance requirements of the M4-M5 Link EIS. As such, the integrated analysis of the overarching tunnel network completed as part of the M4-M5 Link EIS remains valid.

The following points are drawn from the work from the in-tunnel report for the M4-M5 link EIS.

- In the M4-M5 Link EIS the average concentration of nitrogen dioxide was calculated for all sections of tunnels within the combined (cumulative) tunnel network for different hours of the day, travelling in different directions. These were estimates of the average concentration of nitrogen dioxide inside each of the tunnel segments and for a range of different trips that may take place within the tunnel network (including through the F6 Extension). These estimates were presented for expected traffic conditions (varying by hour of the day and the presence of congested traffic, particularly during peak travel times) as well as an extreme congestion case where traffic travels at an average spend of 20 kilometres per hour.
- With windows up and ventilation on recirculation the concentrations that may be present inside vehicles would be lower. The concentration of nitrogen dioxide inside the vehicle is the point of exposure and what should be considered in relation to the potential for health effects.

- In relation to assessing exposures within vehicles using the tunnels, in-vehicle nitrogen dioxide levels were taken to be equal to the in-tunnel average for the segment travelled multiplied by 0.3, the upper end of the range of ratios for indoor:outdoor nitrogen dioxide levels from the studies undertaken.
- For individuals using other modes of transport, the following was noted:
  - Individuals using motorbikes would not have the opportunity to reduce exposure inside the tunnel through the use of ventilation controls. However, the time spent inside tunnels would be less than for other users, particularly in heavy traffic, as motorcyclists can lane filter when traffic is travelling at 35 kilometres per hour and slower. This would limit the amount of time that motorcyclists spend inside the tunnel, even during worst case congested conditions
  - Individuals travelling in buses may also be exposed to nitrogen dioxide inside the bus. It is understood that NSW buses have air conditioning and ventilation systems that include recirculation, with new buses10 allowing a minimum of 10 per cent fresh air at all times to maximum passenger comfort and minimise excess levels of carbon dioxide. Buses may also be leakier than passenger vehicles, resulting in more outdoor air entering the bus. However, the volume of air inside a bus is much greater than in a passenger vehicle and hence air entering from outdoors would be mixed in a larger volume. No data is available for the air exchange rates in Sydney buses. Published data suggests highly variable values in the range of 2.6 to 4.55 air changes per hour for more modern school buses and 16 air exchanges per hour for an older (pre-1998) bus (Knibbs et al. 2009). Adopting the nitrogen dioxide model established by Roads and Maritime (PEL 2016), a well ventilated older bus with 16 air exchanges per hour results in an indoor:outdoor ratio for nitrogen dioxide of 0.3, the same as measured for the older/leakier vehicles considered in the Roads and Maritime study. A lower ratio is calculated for a tighter modern bus. Hence the adjustment of 0.3 to calculate indoor air concentrations of nitrogen dioxide inside passenger vehicles can also be applied to busses.
- Table 7-6 and Table 7-7 present a summary of the maximum (by time of the day) predicted average concentrations of nitrogen dioxide for the most prominent routes of travel using the F6 Extension and different parts of the tunnel system (assuming all tunnel projects are completed in 2033), for expected traffic within the tunnel. Average nitrogen dioxide levels in some of the travel routes have also been calculated for the extreme congestion scenario of traffic at 20 kilometres per hour. The tables also present the estimated worst case in-cabin or inside concentration of nitrogen dioxide, where windows are up and ventilation is on recirculation.

<sup>&</sup>lt;sup>10</sup> <u>http://www.transport.nsw.gov.au/sites/default/files/b2b/busreform/bus-specification-double-deck-two-door-city.pdf</u>

Path No.	ath Travel o.				Tunnels used for travel along path			Average NO <sub>2</sub> concentration (ppm) – Maximum from travel over all hours of the day					
					M4-			Expected traffic		Expected traffic Hour of day for		Extreme congestion	
				M4	M5	New	F6	In-tunnel	In-vehicle	maximum:	In-tunnel	In-vehicle	
	Enter at	Exit at	Distance	East	Link	M5	Extension*		(recirculation)	expected traffic		(recirculation)	
1F	M4 East	F6 Extension	19.5 km	Х	Х	Х	Х	0.25	0.076	7am			
1M	Concord Rd	F6 Extension	18.4 km	Х	Х	Х	Х	0.26	0.079	7am	0.39	0.12	
1R	Wattle St	F6 Extension	13 km		Х	Х	Х	0.25	0.074	4pm	0.38	0.11	
1U	Western Harbour Tunnel	F6 Extension	13 km		Х	Х	Х	0.23	0.068	4pm	0.34	0.10	
1W	St Peters	F6 Extension	6.9 km			Х	Х	0.22	0.066	4pm			
1AA	Iron Cove	F6 Extension	13.4 km		Х	Х	Х	0.22	0.066	4pm	0.33	0.10	
1AD	City West Link	F6 Extension	12.1 km		Х	Х	Х	0.24	0.073	4pm	0.36	0.11	
					NO2	guideline	: 15 minute av	erage = 0.5 ppn	n				

### Table 7-6 Average nitrogen dioxide levels for different trips using completed tunnel network 2033: To F6 Extension

٠

Path No.			Tunnels used for travel along path				Average NO <sub>2</sub> concentration (ppm) – Maximum from travel over all hours of the day					
					M4-			Expected traffic		Hour of day for	Extrem	e congestion
				M4	M5	New	F6	In-tunnel	In-vehicle	maximum	In-tunnel	In-vehicle
	Enter at	Exit at	Distance	East	Link	M5	Extension*		(recirculation)			(recirculation)
2F	F6 Extension	St Peters	7.1 km			Х	Х	0.05	0.02	7am		
2G	F6 Extension	Western Harbour Tunnel	12.8 km		х	Х	Х	0.13	0.04	7am		
2H	F6 Extension	Wattle St	14.3 km		Х	Х	Х	0.14	0.04	7am		
2J	F6 Extension	Concord Rd	18.5 km	Х	Х	Х	Х	0.19	0.06	7am		
2K	F6 Extension	M4 East	19.7 km	Х	Х	Х	Х	0.24	0.07	7am	0.41	0.12
2AA	F6 Extension	Iron Cove	13.6 km		Х	Х		0.13	0.04	7am	0.39	0.12
2AB	F6 Extension	City West Link	12.3 km		Х	Х		0.12	0.04	7am	0.35	0.11
				NO	2 guideline	e: 15 min	ute average =	0.5 ppm				

### Table 7-7 Average nitrogen dioxide levels for different trips using completed tunnel network 2033: From F6 Extension

- In relation to the trips emanating and exiting from the F6 Extension these trips including the extreme congestion scenario, these trips have been found to be below the 0.5ppm guideline and therefore it is unlikely that significant health effects would occur
- It is noted that the NO2 guideline may not protective of all health effects for all individuals. There is the potential for severe asthmatic individuals, especially if they utilise motorbikes, to experience some change in respiratory response after using the tunnels, particularly when congested
- Repeated use of tunnels also requires consideration. The available data on health effects associated with short-duration exposures indicates the effects are transient, ie only relate to the peak exposure that has occurred. Repeated exposures that may occur as a result of morning peak and afternoon peak travel, have not been considered to be additive. Provided the average nitrogen dioxide concentrations that occur during the travel times in the vehicle are below the health based guidelines, which is expected to be the case for the expected traffic conditions, then no significant adverse health effects are expected
- For individuals involved in occupations that may require more regular use of the road network, such as taxi and courier drivers, there is the potential for these individuals to make more frequent and varied trips over different travel segments in any one day. For these drivers, it is important that they keep their window up and ventilation on recirculation to minimise exposures throughout the day.

# 7.4 Particulate matter

There are no health based guidelines available for the assessment of short-duration exposures to particulate matter (PM) within a tunnel. In-tunnel criteria relate to visibility (and safety in using the tunnel). It is expected that the concentration of PM within the tunnel would be higher than ambient air concentrations, and the concentration of PM would increase with increasing distance travelled through the tunnel.

Potential concentrations of PM were not considered in the ventilation and in-tunnel air quality report ( Annexure K to **Appendix E:** Air quality technical report). However potential concentrations of PM for cumulative tunnel exposures were considered in the M4-M5 EIS. The following is taken from the M4-M5 EIS.

Potential concentrations of PM inside the tunnel are derived from exhaust as well as non-exhaust sources. Non-exhaust sources include tyre and break wear and dust from surface road wear and the resuspension of road dust. The modelling of PM and visibility issues within the tunnel did consider both sources. **Table 7-8** presents a summary of the peak concentrations of PM estimated inside the tunnels in 2023, for the expected traffic conditions.

Scenario/Tunnel segment	Peak PM concentration (mg/m <sup>3</sup> )			
	Exhaust	Non-exhaust sources		
	Cumulative	Cumulative		
To F6 Extension				
New M5 including F6 Extension	0.08	0.64		
From F6 Extension				
New M5 including F6 Extension	0.03	0.2		

Table 7-8 Predicted	noak concentrations of	narticulate matt	or instunnal: 2022
I able 1-0 Fleuicleu	peak concentrations or	particulate matte	51 III-luiiiei. 2023

The characteristics of PM derived from exhaust and non-exhaust sources would be different.

The available evidence suggests that non-exhaust particles are generally larger than exhaust particles. It is likely that non-exhaust particles are greater than 10 micrometres in diameter, however this is not well characterised. Where the particles are larger than 10 micrometres in diameter they are of less importance in terms of potential health effects, as these relate to the finer particles that are less than 10 micrometres in diameter, with stronger health effects relevant to exposure to particles less than 2.5 micrometres in diameter. The tunnel design and air quality assessment is based on non-exhaust PM emission factors that relate to PM<sub>10</sub> and PM<sub>2.5</sub> from relevant emissions studies.

PM from exhaust is expected to be largely fine particulates, ie  $PM_{10}$  and  $PM_{2.5}$  that are of importance to health.

In relation to the PM concentrations predicted within the tunnel, the following is noted:

- The in-tunnel concentrations for PM are taken to be PM10 concentrations where concentrations
  of PM2.5 are likely to comprise a significant portion of the PM10 concentration, particularly for
  exhaust emissions
- PM10 concentrations within the tunnels are dominated by non-exhaust sources
- The maximum concentrations of PM10 in the tunnels evaluated are up to 0.7 milligrams per cubic metre. The average concentration in the tunnels would be lower than the peak concentration predicted, potentially up to 50 per cent of that reported as the peak concentration. When windows are up and ventilation is on recirculation the average level of PM10 inside a vehicle would be lower, potentially up to 0.07 milligrams per cubic metre
- As a significant proportion of in-tunnel particulate matter is non-exhaust, regular cleaning of tunnel roadways may reduce these levels

### 7.4.1 Review of short duration exposure to particles

In relation to assessing potential short-duration exposures to particles, the following should be noted:

- The NHMRC (2008) has published measured concentrations of particulates (as PM2.5 and PM10) from a range of tunnels in Sydney and around the world. The measured concentrations come from a number of different studies where the sampling methodology and averaging time for the collection of the data varies significantly. This makes it difficult to directly compare the range of reported concentrations with the concentrations predicted in this assessment (ie not comparing data reported over similar averaging/exposure periods). While noting this difficulty in comparing the data, the range of average concentrations of PM2.5 reported typically range from around 0.03 to 0.343 milligrams per cubic metre (AMOG 2012; NHMRC 2008). These levels are based on data with averaging times that vary from one hour averages, peak hour averages, daytime averages to 24 hour averages
- The exposure-response relationships for particulate matter that have been established on the basis of adverse health effects from short term exposures relate to changes in the health effects associated with variability in 24 hour average concentrations of PM2.5 in urban air. They do not relate to much shorter variations in PM2.5 exposure that may occur within a 24 hour period, where there may be exposures over a few minutes to higher levels of PM2.5. No guidelines are currently available for assessing potential health effects that may occur as a result of exposures to particulates that may occur for minutes (or even an hour)
- Recent review (WHO 2013a) of available studies in relation to short duration (less than 24 hour) exposures to particulates indicates the following:
  - Epidemiological and clinical studies have demonstrated that sub-daily exposures to elevated levels of particulate matter can lead to adverse physiological changes in the respiratory and cardiovascular system, in particular exacerbation of existing disease. This is generally consistent with the outcome of studies reviewed and considered by the USEPA (USEPA 2009b)
  - The studies available do not cover a range of exposure concentrations, nor do they adequately address other variables such as co-pollutants (gases) or repeated short-duration exposures
  - The studies have not determined if a one hour exposure would lead to a different response than a similar dose spread over 24 hours, or if an exposure-response can be determined

- Exposures that occur during the use of various transportation methods (such as in-vehicles) have been found to contribute to and affect 24 hour personal exposures.

The urban epidemiology studies (upon which exposure-response relationships are based and have been used in this assessment) utilise health data for adverse health effects from an urban population, where the urban population would have been exposed to ambient levels of particulate matter (as measured by air monitoring stations) as well as fluctuations that occur throughout the day during various daily activities including in-vehicle exposures (and others such as cooking). These large urban studies have related health effects to regional ambient (urban) air concentrations. They have not measured daily (or longer term) personal exposures to particulate matter, but such fluctuations would occur within the population exposed and would be expected to be accounted for within the health data considered in the epidemiology studies. Specific health effects from the short duration variations in particulate exposures throughout any specific day cannot be determined from these studies. It is therefore important to consider if exposures to PM<sub>2.5</sub> in the project tunnels would be consistent with other tunnels or in-vehicle exposures (during commuting in an urban environment), where the following can be considered:

- Exposure to particulate matter within vehicles varies with the intensity of the traffic, the age of the vehicle the choice of ventilation used within the vehicle and the type of fuel used (Knibbs et al. 2010). Levels of PM2.5 reported in vehicles in Europe (ETC 2013) vary from 0.022 to 0.085 milligrams per cubic metre for passenger cars and 0.026 to 0.13 milligrams per cubic metre for bus travel
- Levels of PM2.5 that have been measured within cars while commuting in Sydney (where tunnel travel was not part of the study) range from 0.009 to 0.045 milligrams per cubic metre (NSW Health 2004)
- Keeping windows closed and switching ventilation to recirculation has been shown to reduce exposures to particulates inside the vehicle by up to 80 per cent (NSW Health 2003). While noting no guidelines are availability for very short duration exposures, this would further reduce exposure to motorists.

# 7.5 Carbon dioxide issues

To minimise exposures in-vehicle to nitrogen dioxide and particulates the above assessment has relied on Roads and Maritime providing advice to motorists using the proposed tunnels to wind up windows and place ventilation in recirculation. Health issues that may arise from such advice relate to the potential build-up of carbon dioxide inside the vehicle. An assessment of in-cabin levels of carbon dioxide and potential effects on the health and safety of drivers travelling through tunnels over varying distances and times, has been completed by Roads and Maritime in 2017 (enRiskS 2017). Based on this study for vehicles that may include between one and five occupants, travelling through tunnels for up to an hour, the levels of carbon dioxide were not expected to adversely affect driver safety.

Assessment of potential exposures that may occur for periods of time up to two hours, where ventilation is left on recirculation indicates that there may be levels of carbon dioxide inside a vehicle where there are one or more passengers that may affect an already fatigued driver.

It is noted that there is a general lack of guidance or regulations in terms of the design or use of ventilation systems in vehicles in Australia. Hence there is currently no advice to drivers on the suitable use of ventilation in various circumstances, to minimise the potential for effects on already fatigued drivers.

Where Roads and Maritime provides specific advice to drivers entering road tunnels to put ventilation on recirculation, it is recommended that further advice is provided that recirculation should be switched off at some point after using the tunnel network and not left on for an extended period of time.

## 7.6 Overall assessment

Impacts within the tunnel: while concentrations of pollutants from vehicle emissions are higher within the tunnel (compared with outside the tunnel), and with the completion of a number of tunnel projects (approved or proposed) there is the potential for exposures to occur within a network of tunnels over varying periods of time, depending on the journey. The assessment of potential exposures inside these tunnels, has indicated:

- Where windows are up and ventilation is on recirculation, exposure to nitrogen dioxide inside vehicles is expected to be below the current health based guidelines. In congested conditions inside the tunnels, it is not considered likely that significant adverse health effects would occur. Placing ventilation on recirculation is also expected to minimise exposures to particulates during travel through the tunnels
- For motorcyclists, where there is no opportunity to minimise exposures through the use of ventilation, there is the potential for higher levels of exposure to nitrogen dioxide are particulates. These exposures, under normal conditions, are not expected to result in adverse health effects. When the tunnels are congested it is expected that motorcyclists would spend less time in the tunnels than passenger vehicles and trucks, limiting the duration of exposure and the potential for adverse health effects
- For individuals who regularly use tunnels for commuting or as part of their employment there is the potential for repeated exposures to higher levels of nitrogen dioxide and particulates during the day. While these exposures are not likely to be additive, in terms of potential health effects, it is important that these road users utilise ventilation on recirculation whenever they are using the tunnels
- Where advice is provided to place ventilation on recirculation when using the tunnel or the proposed network of tunnels, it is not expected to result in carbon dioxide levels inside the vehicle that may adversely affect driver safety. However, where Roads and Maritime provides specific advice to drivers entering road tunnels to put ventilation on recirculation, it is recommended that further advice is provided that recirculation should be switched off at some point after using the tunnel network and not left on for an extended period of time.
# 8 Assessment of changes in noise and vibration impacts on community health

# 8.1 General

A detailed assessment of noise and vibration impacts associated with the project is presented in **Appendix G** of the EIS (Noise and vibration: Technical report).

**Appendix G** (Noise and vibration: Technical report) has been reviewed to determine if the predicted impacts have the potential to affect the health of the surrounding community, and if impacts are predicted, if they can be effectively mitigated. The assessment of noise has considered impacts at a number of different receptors (termed noise receivers, or receivers within the Technical report.

The assessment of noise during construction and operations involved consideration of impacts at 17 noise catchment areas (NCAs) presented in the figures in Annexure I. A NCA is defined by what is considered a similar noise environment. Thus receptors belonging to the same NCA are assigned the same background noise level and noise management level.

# 8.2 Existing noise environment

### 8.2.1 General

The study area includes a mixture of urban and suburban noise and vibration sensitive receivers (such as, residential properties, educational establishments, hospitals and recreational areas), commercial and industrial properties, and major roads and railway lines. The existing ambient noise environment can be divided into three sections, northern, central and southern end of the study area. The north is dominated by heavy traffic flows and aircraft noise, the central part is dominated by local traffic with aircraft and railway movements, while the south is dominated by heavy traffic, railway and industry noise.

To undertake the noise assessment required for the project, the existing background noise quality needed to be assessed as the guidelines that relate to noise impacts from a specific project are based on levels allowable above background.

### 8.2.2 Ambient noise monitoring

Existing ambient noise was measured at 16 locations (refer to Annexure I for locations) at one of 3 time periods (June 2015, November / December 2017 or February 2018). Monitoring was undertaken by a noise logger. A noise logger measures the noise level over the sample period and then determines  $L_{A1}$ ,  $L_{A10}$ ,  $L_{A90}$ ,  $L_{Amax}$  and  $L_{Aeq}$  levels of the noise environment. The A-weighting is a frequency filter applied to represent how the human ear hears sound. The  $L_{A1}$ ,  $L_{A10}$  and  $L_{A90}$  levels are the levels exceeded for 1 per cent, 10 per cent and 90 per cent of the sample period respectively. The  $L_{Amax}$  level is the maximum noise levels due to individual noise events. The  $L_{A90}$  level is taken as the background noise level also known as the Rated Background Level (RBL). The  $L_{Aeq}$  level is the energy averaged noise level over a defined period and is known as Ambient Noise Level (ANL).

### 8.2.3 Background noise levels

Based on the monitoring undertaken the RBL has been calculated for use in the noise assessment. The RBLs calculated relate to specific time periods (namely daytime, evening and night-time) and were used to define the appropriate construction noise management levels, consistent with the Interim Construction Noise Guideline (ICNG) (DECC, 2009). They were also used to define the applicable noise criteria for fixed ancillary facilities such as the ventilation and tunnel support facilities, in accordance with the Noise Policy for Industry (NPfI) (NSW EPA 2017).

The RBL were determined for the assessment of construction noise for different periods of the day: daytime (7.00 am to 6.00 pm), evening (6.00 pm to 10.00 pm) and night-time (10.00 pm to 7.00 am). The RBLs determined at each of the monitoring locations varied from 38 to 66 decibels (dB(A)) during the daytime, 37 to 66 dB(A) during the evening and 31 to 56 dB(A) during the night-time.

The ANL were also determined for the assessment of noise for different periods of the day: daytime (7.00 am to 6.00 pm), evening (6.00 pm to 10.00 pm) and night-time (10.00 pm to 7.00 am). The ANLs determined at each of the monitoring locations varied from 49 to 72 decibels (dB(A)) during the daytime, 47 to 70 dB(A) during the evening and 44 to 68 dB(A) during the night-time.

# 8.3 Noise assessment criteria

### 8.3.1 General

Noise issues in NSW are managed by the NSW EPA. The NSW EPA has prepared a number of guidance documents with regard to the types of noise that are considered in relation to construction and operation of the project. The *NSW Noise Policy for Industry (NPfI)* (NSW EPA 2017), the *NSW Road Noise Policy* (RNP) (NSW DECCW 2011), and the ICNG (NSW DECC 2009) are all relevant to the assessment of noise generated by this project. In all these policies, there is discussion of the need to balance the economic and social benefits of activities that may generate noise with the protection of the community from the adverse effects of noise. The noise assessment criteria adopted relate to levels of noise that can be tolerated or permitted above background before some adverse effect (annoyance, discomfort, sleep disturbance or complaints) occurs.

The Roads and Maritime *Construction Noise and Vibration Guideline*, August 2016 (CNVG) outlines Roads and Maritime's approach to assessing and mitigating construction noise. The Roads and Maritime *Noise Mitigation Guide* applies to the assessment and management of noise during operations. These guidelines are considered in addition to the other relevant policy and guidelines from the NSW EPA.

For the assessment of noise impacts from the project a range of guidelines and criteria have been adopted for the assessment of:

- Construction including ground-borne noise, vibration and blasting
- Operations relevant to road noise and fixed facilities.

The following sections provide an overview of the guidelines adopted for each of these aspects. In particular, the basis for the guidelines and relevance to the protection of health and wellbeing is noted.

### 8.3.2 Construction noise criteria

People are usually more tolerant to noise and vibration during the construction phase of projects than during normal operation. This response results from recognition that the construction emissions are of a temporary nature – especially if the most noise-intensive construction impacts occur during the less sensitive daytime period. For these reasons, acceptable noise and vibration levels are normally higher during construction than during operations.

Construction often requires the use of heavy machinery which can generate high noise and vibration levels at nearby buildings and receptors. For some equipment, there is limited opportunity to mitigate the noise and vibration levels in a cost-effective manner and hence the potential impacts should be minimised by using feasible and reasonable management techniques.

At any particular location, the potential impacts can vary greatly depending on factors such as the relative proximity of sensitive receptors, the overall duration of the construction works, the intensity of the noise and vibration levels, the time at which the construction works are undertaken, and the character of the noise or vibration emissions.

**Appendix G** (Noise and vibration: Technical report) has considered construction noise impacts associated with construction activities for Stage 1 of the F6 extension. There are some areas within the community were construction impacts from a number of road projects are proposed, with these works occurring over a longer period of time, potentially up to eight years. Further discussion on issues related to these longer duration impacts, ie construction fatigue, are further addressed in the **Section 10.8**.

The ICNG has been adopted for the assessment of noise during construction works (NSW DECC 2009). These guidelines require that noise impacts from the project be predicted at sensitive receptors. These noise levels are then compared with the project specific criteria, referred to as management levels, which are based on an increase above background levels. Where an exceedance occurs, the guidelines require that the proponent must apply all feasible and reasonable work practices to minimise impacts. The management levels are based on levels of noise above background that may result in reactions (or complaints) by the community. The levels are based on some reaction (noise affected) and a strong reaction (highly noise affected).

Levels of noise allowable outside standard work hours, particularly at night, are lower than those permitted during normal work hours. Where construction works are planned to extend over more than two consecutive nights a sleep disturbance assessment is required to be undertaken. Based on the available information on the levels of noise that result in sleep disturbance the following has been adopted:

- A maximum internal noise level below 50–55 dB(A) is considered unlikely to cause awakening;
- External noise levels of 60–65 dB(A) are unlikely to result in awakening reactions.

The project has considered that an open window provides up to 10 dB(A) attenuation of noise from outdoors to indoors.

The assessment of noise impacts during construction has been undertaken based on 17 noise catchment areas (assumed to have background noise levels consistent with the background noise monitoring location within each catchment area).

The ICNG does not provide direct reference to an appropriate criterion to assess the noise arising from construction traffic on public roads. However, it does refer to the Road Noise Policy which presents a discussion on assessing feasible and reasonable mitigation measures. In assessing feasible and reasonable mitigation measures, an increase of up to 2 dB(A) represents a minor impact that is considered barely perceptible to the average person. Therefore, the noise goal applied to traffic movements on public roads generated during the construction phase of the project is an increase in existing road traffic noise levels of no more than 2 dB(A).

Where construction would be undertaken during the night-time period the potential for sleep disturbance should be assessed. The current approach to identifying potential sleep disturbance impacts is to predict maximum noise levels and assess against a screening criterion 15 dB(A) above the RBL during the night-time period (10.00 pm–7.00 am).

### 8.3.3 Ground-borne noise criteria

The ICNG provides residential NMLs for ground-borne noise, which are applicable when ground-borne noise levels are higher than the corresponding airborne construction noise levels such as might occur during tunnelling. The ICNG provides ground-borne noise levels at residences for evening and night-time periods only, as the objectives are to protect the amenity and sleep of people when they are at home. The following ground-borne noise levels are applicable for residences:

- Evening 40 dB(A) LAeq (15 minute)
- Night-time 35 dB(A) LAeq (15 minute).

These guidelines are applicable during tunnelling and other construction activities.

### 8.3.4 Vibration criteria

The effects of vibration on buildings can be divided into three main categories:

- Human comfort: Those in which the occupants or users of the building are inconvenienced or
  possibly disturbed. These guidelines are of most relevance to the assessment of community
  health. Intermittent vibration has been evaluated on the basis of the NSW EPA guideline
  Assessing Vibration: A Technical Guideline (NSW DEC 2006), which is based on vibration dose
  values (VDV). The criteria for VDV are based on the potential for annoyance (based on the level
  of vibration over the assessment period). Guidelines for continuous and impulsive vibration are
  dependent on the time of day they occur and the activity taking place that could be affected
- Building contents: Those where the building contents may be affected. As people perceive floor vibration well before levels are likely to cause damage to building contents and structures, for

most areas controlling vibration to manage human comfort would also address damage to building contents. No separate criteria are adopted to evaluate this aspect.

Structural damage: Those in which the integrity of the building or the structure itself may be
prejudiced (structural damage). Most commonly specified 'safe' structural vibration limits are
designed to minimise the risk of threshold or cosmetic surface cracks, and are set well below the
levels that have potential to cause damage to the main structure. The assessment of potential
structural damage has been undertaken in accordance with Australian Standard AS2187, British
Standard BS 7385 and German Standard DIN 4150:Part 3-1999 (DIN 1999). These guidelines
include criteria relevant to addressing blasting activities.

### 8.3.5 Operational noise criteria

Operational noise impacts have been evaluated on the basis of the RNP, with additional guidance and criteria provided within Roads and Maritime's *Noise Criteria Guideline* (NCG) and *Noise Mitigation Guideline* (NMG) (NSW DECCW 2011; NSW Roads and Maritime 2015). The principles underlying the guidance documents are:

- Criteria are based on the road development type a residence is affected by due to the road project
- Adjacent and nearby residences should not have significantly different criteria for the same road
- Criteria for the surrounding road network are assessed where a road project generates an increase in traffic noise greater than 2 dB(A) on the surrounding road network
- Existing quiet areas are to be protected from excessive changes in amenity due to traffic noise.

The project consists of both new and redeveloped roads or road sections according to the definitions in the guidance documents and so both road types need to be considered in developing project-specific limits.

For residential areas, criteria are established for properties near either freeway/arterial/sub-arterial roads or local roads. These criteria relate to noise levels during the daytime (7.00 am to 10.00 pm) and night-time (10.00 pm to 7.00 am). Night-time noise criteria are aimed at minimising sleep disturbance. Criteria are also available to assessed noise exposures in other types of buildings, including schools, places of worship, open space, childcare, aged care and hospital facilities.

Operational traffic noise from the surrounding road network also required some consideration, with criteria (e.g. noise criteria is exceeded and an increase by more than 2 dB(A) is predicted) established to determine if such impacts need to be further considered for mitigation measures.

Guidelines are also available to evaluate maximum noise levels from roadways, such as those from individual vehicles or trucks that have the potential to disturb sleep. While no specific criterion is set to address this specific issue, a number of guidance points may be used to qualify if the maximum noise level is likely to be an issue. These include calculation of maximum noise levels, the extent to which the maximum noise levels for individual vehicle pass-bys exceed the  $L_{Aeq}$  noise level for each hour of the night, and the number of times the maximum noise levels for individual vehicle pass-bys exceed the  $L_{Aeq}$  noise level for each hour of the night.

The assessment has also evaluated noise from the operation of fixed facilities, namely the jet-fans within the tunnels, ventilation facilities, substations and water treatment plants. Noise from these facilities have been assessed on the basis of criteria in NSW Noise Policy for Industry. This assessment considers short term intrusive noise impacts for residents, as well as noise level amenity for residents and other land uses, with the lower of the two impacts informing the noise criteria. This policy established criteria for daytime, evening and night-time noises, as well as criteria relevant to the assessment of sleep disturbance.

The current approach to assessing potential sleep disturbance is to apply an initial screening criterion of:

- LAeq,15min 40 dB(A) or the prevailing RBL plus 5 dB, whichever is the greater, and/or
- LAFmax 52 dB(A) or the prevailing RBL plus 15 dB, whichever is the greater,

and to undertake further analysis if the screening criterion cannot be achieved. The further analysis should cover the maximum noise level, the extent to which the maximum noise level exceeds the rating background noise level, and the number of times this happens during the night-time period.

Other guidelines that contain additional advice relating to potential sleep disturbance impacts should also be considered, including the RNP (NSW DECCW 2011). The RNP provides a review of research into sleep disturbance. From the research to date, the RNP concludes that:

- Maximum internal noise levels of 50–55 dB(A) LAFmax are unlikely to awaken people from sleep
- One or two events per night, with maximum internal noise levels of 65 to 70 dB(A) LAFmax, are not likely to affect health and wellbeing significantly.

It is generally accepted that internal noise levels in a dwelling, with the windows open are 10 dB(A) lower than external noise levels. Based on a worst case minimum attenuation, with windows open, of 10 dB(A), the first conclusion above suggests that short term external noises of 60 dB(A) to 65 dB(A) are unlikely to cause awakening reactions. The second conclusion suggests that one or two noise events per night with maximum external noise levels of 75 dB(A) to 80 dB(A)  $L_{AFmax}$  are not likely to affect health and wellbeing significantly.

# 8.4 **Overview of noise and vibration assessment**

### 8.4.1 **Construction impacts**

### **Construction Noise**

Applicable NSW legislation and guidelines have been used to inform the construction noise modelling and assessment. Noise mitigation has been recommended in accordance with these guidelines. These guidelines have been developed taking into consideration current international practices, health impacts of noise and to protect vulnerable people.

Noise that may be generated during construction has been modelled based on the type of equipment to be used, where the equipment is to be used in relation to the community receptors, the hours of work, the duration of the activities undertaken and the local terrain. Modelling was undertaken at a number of construction sites within the project area.

The assessment has considered a range of standard noise mitigation measures, ie those that would be a standard requirement for a range of construction activities. In some situations, impacts from construction noise and vibration may be unavoidable, particularly where works are undertaken in close proximity to the community. Where this occurs the Roads and Maritime CNVG includes a range of additional mitigation measures to manage these impacts. These measures include actions to notify and provide warning to the community and/or to offer respite or alternate accommodation.

Overall, a worst case assessment has been used in accordance with the ICNG, assuming no additional mitigation measures are implemented. For each area assessed, the noise levels at the most affected receptor have been used to represent the whole noise catchment area.

The noise modelling, which included mitigation measures such as acoustic sheds and noise barriers, identified noise impacts in excess of the criteria for standard and out of hours construction period (refer to **Appendix G** (Noise and vibration: Technical report) for further detail).

Overall over hundred receptors have been identified as exceeding the day or night noise management level by over 25 dB(A), during various phases of construction works at the different construction sites. Further several hundred receptors have been identified as exceeding the sleep disturbance criteria, including the criteria for awakening.

To address the noise impacts identified, mitigation measures have been identified. These include work scheduling, temporary noise walls or hoarding, respite, plant selection and equipment and traffic management. However an evaluation of implementing these mitigation measures has not been undertaken to determine the effectiveness of these measures.

The assessment has also addressed the impact of simultaneous construction works on noise from a number of different infrastructure projects. An identification of developments planned in the area along with current developments was undertaken. It was estimated that the cumulative construction noise impact may increase by as much as 3 dB(A). An assessment of consecutive construction works was also undertaken and is further discussed in **section 10.8**.

A separate assessment of the noise impact from the construction of a power line from the Ausgrid Canterbury subtransmission substation to the Rockdale (south) motorway operations complex was undertaken. Using the Roads and Maritime Construction Noise Estimator tool it was predicted that noise impacts would exceed the noise management levels by up to 36 dB(A), with maximum  $L_{AEQ}$  noise levels predicted up to 84 dB(A). Receptors experiencing these noise levels are likely to be highly affected and it is predicted that these impacts could last up to a few weeks.

### Potential noise impacts from movement of construction vehicles

Potential increases in noise for sensitive receptors due to construction traffic has been assessed separately from the assessment of noise from other construction activities. Heavy vehicles involved in construction are expected to travel via existing major roadways with minimal use of local roads. For most areas evaluated, there are no noticeable increases in noise from construction traffic on the proposed routes during the daytime or night-time. The exceptions were at Bruce Street during the day time period (increased traffic noise predicted at 2.4 dB(A)) and roads around the Rockdale construction ancillary facility (especially Wickham Street) during night time periods (increased traffic noise predicted up to 7.3 dB(A)). For Bruce Street the modelling was considered conservative and unlikely to result in impact predicted. However, the predicted night time impacts are considerable and night-time haulage should be avoided during night-time off-peak traffic periods to minimise noise impacts.

### Ground-borne construction noise

Ground-borne noise occurs when works are being undertaken under the ground surface or in some other fashion that results in the vibrations from noise moving through the ground rather than the air. When vibrations reach a building they enter the foundations, which are subject to a coupling loss and are then transmitted into the walls and ceiling. The excitation of the walls and ceiling results in the generation of low-frequency noise which could be audible if the vibration levels are high enough. The noise is typically considered to be a low 'rumble'.

For this project vibration would be generated during tunnelling from the operation of road headers. Blasting has been suggested but is currently not proposed. This project involves tunnelling where activities are expected to occur 24 hours per day.

Tunnelling would typically progress around a maximum of seven metres per day. It is likely that ground-borne noise would be discernible for up to five days at each affected receiver with exceedances occurring for up to two days. Only one receptor is predicted to exceed the ground-borne noise criteria. This exceedance would be up to 1 dB(A) during the night-time period.

### Vibration impacts

A range of the equipment to be used in construction have the potential to cause unacceptable levels of vibration. Managing the potential for such vibration to actually cause discomfort or structural damage at sensitive receptor locations is based on ensuring suitable separation distances between the equipment and the receptor locations.

The assessment did not identify any receptors that would exceed the vibration criteria for human comfort, and concluded that the structural damage criteria would not be exceeded by the tunnelling activities.

### 8.4.2 **Operational impacts**

Assessment of operational noise impacts has been undertaken by modelling noise associated with the project. The assessment evaluated impacts on the community at locations surrounding the surface works on President Avenue and Princes Highway as well as a higher level assessment of noise impacts from the surrounding road network. The study area was defined by an area impacted by at least 2.0 dB(A) due to the project.

The noise modelling took into consideration both the physical design changes and additional traffic generated by the project. Two separate years, in addition to three separate traffic scenarios were assessed. The assessment of road traffic noise has been completed in accordance with the relevant guidelines (RNP, the NCG and the NMG). An assessment was undertaken to determine how well the model estimated noise impacts based on a current scenario. The modelled and measured results were found to be within acceptable tolerances, which are +/- 2 dB(A).

To assess the potential impact of the project on noise sensitive receivers, the following steps were completed:

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

- Existing road traffic noise levels modelled with existing (2017/2018) road traffic volumes
- Future road traffic noise levels modelled for the No Build (without project), Build (with project), and Cumulative (with other major infrastructure projects) scenarios for the year of opening (2026) and design year (2036).

The modelling identified 109 receptors that exceed the applicable noise criteria and were eligible for consideration of noise mitigation measures. It has been estimated that noise barriers and road surfaces changes are not considered reasonable, and therefore architectural treatment of houses should be considered.

The assessment of the surrounding road network also found significant noise impacts with increases in noise levels of greater than 5 dB(A), with the highest predicted values on O'Connell Street at 6 dB(A). Mitigation measures would need to be considered and traffic calming measures may reduce this noise impact to acceptable levels, however the diversion of traffic may also increase noise impacts on other roads. It has been suggested that an updated road traffic model be presented during the detailed design phase of the project.

No estimation was made of the likely number of maximum noise level events. With the potential increase in traffic, the maximum noise level events are likely to decrease due to the increased typical noise, however the increase in traffic will bring an increased number of engine breaking events by heavy vehicles.

In relation to the permanent operation facilities (Arncliffe and Rockdale), no noise exceedances are predicted to occur. However, the assumptions made in the assessment should be confirmed at the detailed design stage to ensure this is still the case.

# 8.5 Health outcomes relevant to noise

### 8.5.1 General

Environmental noise has been identified (I-INCE 2011; WHO 2011) as a growing concern in urban areas because it has negative effects on quality of life and wellbeing and it has the potential for causing harmful physiological health effects. With increasingly urbanised societies impacts of noise on communities have the potential to increase over time.

Deciding on the most effective noise management options in a specific situation is not just a matter of defining noise control actions to achieve the lowest noise levels or meeting arbitrarily chosen criteria for exposure to noise. The goal should be designed to achieve the best available compromise between the benefits to society of reduced exposure to community noise versus the costs and technical feasibility of achieving the desired exposure levels given the project. On the one hand, there are the rights of the community to enjoy an acceptably quiet and healthy environment. On the other hand there are the needs of the society for new or upgraded facilities, industries, roads and recreation opportunities, all of which typically produce more community noise (I-INCE 2011; WHO 2011).

Sound is a natural phenomenon that only becomes noise when it has some undesirable effect on people or animals. Unlike chemical pollution, noise energy does not accumulate either in the body or in the environment but it can have both short term and long term adverse effects on people. These health effects include (WHO 1999, 2011):

- Sleep disturbance (sleep fragmentation that can affect psychomotor performance, memory consolidation, creativity, risk-taking behaviour and risk of accidents)
- Annoyance
- Hearing impairment
- Interference with speech and other daily activities
- Children's school performance (through effects on memory and concentration)
- Cardiovascular health.

Other effects for which evidence of health impacts exists, but for which the evidence is weaker, include:

• Effects on mental health (usually in the form of exacerbation of existing issues for vulnerable populations rather than direct effects)

- Tinnitis (which can also result in sleep disturbance, anxiety, depression, communication and listening problems, frustration, irritability, inability to work, reduced efficiency and a restricted participation in social life)
- Cognitive impairment in children (including deficits in long term memory and reading comprehension)
- Some evidence of indirect effects such as impacts on the immune system.

Within a community the severity of the health effects of exposure to noise and the number of people who may be affected are schematically illustrated in **Figure 8-1**.



# Figure 8-1: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011)

Often, annoyance is the major consideration because it reflects the community's dislike of noise and their concerns about the full range of potential negative effects, and it affects the greatest number of people in the population (I-INCE 2011; WHO 2011).

There are many possible reasons for noise annoyance in different situations. Noise can interfere with speech communication or other desired activities. Noise can contribute to sleep disturbance, which can obviously be very annoying and has the potential to lead to long term health effects. Sometimes noise is just perceived as being inappropriate in a particular setting without there being any objectively measurable effect at all. In this respect, the context in which sound becomes noise can be more important than the sound level itself (I-INCE 2011; WHO 2011).

Different individuals have different sensitivities to types of noise and this reflects differences in expectations and attitudes more than it reflects any differences in underlying auditory physiology. A noise level that is perceived as reasonable by one person in one context (eg in their kitchen when preparing a meal) may be considered completely unacceptable by that same person in another context (eg in their bedroom when they are trying to sleep). In this case the annoyance relates, in part, to the intrusion from the noise. Similarly, a noise level considered to be completely unacceptable by one person, may be of little consequence to another even if they are in the same room. In this case, the annoyance depends almost entirely on the personal preferences, lifestyles and attitudes of the listeners concerned (I-INCE 2011; WHO 2011).

Perceptible vibration (eg from construction activities) also has the potential to cause annoyance or sleep disturbance and so adverse health outcomes in the same way as airborne noise. However, the health evidence available relates to occupational exposures or the use of vibration in medical treatments. No data is available to evaluate health effects associated with community exposures to perceptible vibrations (I-INCE 2011; WHO 2011).

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

It is against this background that regulators in various communities have established sound level criteria above which noise is deemed to be unacceptable and below which it is deemed to be acceptable. Any assessment of noise impacts needs to consider the relevant criteria established for a new or existing (or upgraded) facility or activity. Where there are impacts in excess of these guidelines, an assessment of noise mitigation is required to be undertaken.

### 8.5.2 Health impacts from traffic noise

Road traffic noise is caused by the combination of rolling noise (noise from tyres on the roadway) and propulsion noise (from engine, exhaust and transmission).

A number of large international studies are available that have specifically evaluated health impacts associated with exposure to road traffic noise. Where exposure to road traffic noise is associated with, or can be shown to be causal, adverse health effects an exposure-response relationship is often established. The main health effects that have been studied in these types of investigations in relation to road traffic noise are annoyance, sleep disturbance, cardiovascular disease, stroke and memory/concentration (cognitive) effects. These are further discussed below.

### Cardiovascular effects

There is substantial evidence that hypertension and more importantly blood pressure measurements are an independent risk for cardiovascular disease. Cardiovascular diseases are the class of diseases that involve the heart or blood vessels, both arteries and veins. These diseases can be separated by end target organ and health outcomes. Strokes reflecting cerebrovascular events and ischaemic heart disease (IHD) or coronary heart disease (CHD) are the most common representation of cardiovascular disease.

A link between noise and hypertension is relatively well established in the relevant literature. Whilst there is no consensus on the precise causal link between the two, there are a number of credible hypotheses. A leading hypothesis is that exposure to noise could lead to triggering of the nervous system (autonomic) and endocrine system which may lead to increases in blood pressure, changes in heart rate, and the release of stress hormones. Depending on the level of exposure to excess noise, the duration of the exposure and certain attributes of the person exposed, this can cause an imbalance in the person's normal state (including blood pressure) which can then lead to other cardiovascular diseases (DEFRA 2014). This hypothesis is illustrated in **Figure 8-2**.



### Figure 8-2: Noise reaction model/hypothesis (Babisch 2014)

The available studies regarding road traffic noise and cardiovascular disease risk largely involve metaanalysis (ie statistical analysis that combines the results of multiple scientific studies). A number of studies have been published by Babisch (Babisch 2002, 2006, 2008, 2014; van Kempen & Babisch 2012) have provided the basis for a number of exposure-response relationships adopted for the assessment of cardiovascular health effects associated with road traffic noise.

In relation to hypertension the most relevant recent study (van Kempen & Babisch 2012) involved analysis of 27 studies between 1970 and 2010, where a relationship between road traffic noise and hypertension was determined. This relates to the incidence of hypertension in the population and has been adopted by the European Commission for the assessment of health impacts of road noise in Europe (EEA 2014).

Relationships have also been established between road traffic noise (as  $L_{den}$ )<sup>11</sup> and ischemic heart disease (Babisch 2014; Vienneau et al. 2015). The study by Babisch (2014) involved analysis of 14 studies related to road traffic noise. The study by Vienneau et al (2015) involved analysis of 10 studies related to both air and road transport. The study by Babisch (2014) was more directly relevant to road traffic noise and has been adopted in this assessment.

<sup>&</sup>lt;sup>11</sup> L<sub>den</sub> = average noise level across day, evening and night (ie 24 hour period)

Meta-analysis involves more detailed statistical analysis of large numbers of individual epidemiological studies. In relation to the risk of stroke from exposure to noise, there are limited meta-analysis type studies available and the studies available combine the risks from noise from road and air transport. A more specific study that just investigated the link between road traffic noise and cardiovascular disease/mortality has been undertaken in London (Halonen et al. 2015). This was a large epidemiological study that identified statistically significant associations between road traffic noise (as modelled to residential dwellings) and hospital admissions for stroke and all-cause mortality. The relationships identified related to exposure to day and evening noise as  $L_{Aeq,16h}$ . The study corrected for confounders<sup>12</sup> such as PM<sub>2.5</sub> and NO<sub>2</sub> exposures and has been considered suitable for use in this assessment. The relative risk identified for hospital admissions for stroke is equivalent to that identified for a meta-analysis of air and road noise (Houthuijs et al. 2014).

The relationships determined in the above studies relate to noise exposures in excess of a threshold. The threshold for where these effects are of significance are generally equal to or above the noise criteria adopted for the assessment of operational noise impacts. It is noted, however that in areas already affected by noise at levels above these thresholds, the guidelines relate to an increase in noise attributed to the project, with a guideline of 2 dB(A) adopted. An increase in noise by 2 dB would not be associated with unacceptable cardiovascular risks (where the above exposure-response relationships were considered).

### Annoyance and sleep disturbance

Changes in annoyance and sleep disturbance associated with noise are considered to be pathways for the key health indicators listed above. However, these issues are of importance to the local community and so it is relevant to evaluate the changes in levels of annoyance and sleep disturbance as a result of noise from the operation of the project within the community.

### <u>Annoyance</u>

Annoyance is a feeling of displeasure associated with any agent or condition known or believed by an individual or group to adversely affect them. Annoyance following exposure to prolonged high levels of environmental noise may also result in a variety of other negative emotions, for example feelings of anger, depression, helplessness, anxiety and exhaustion (EEA 2014).

Annoyance levels can be reliably measured by means of an International Organisation for Standardization/Technical Standard (ISO/TS) 15666:2003 defined questionnaire, which has enabled the identification of relationships between annoyance and noise sources. The European Commission (EC 2002) conducted a review of the available data and provided recommendations on relationships that define the percentage of persons annoyed (%A) and the percentage of persons highly annoyed (%HA) to total levels of noise reported as  $L_{DEN}$  (ie average noise levels during the day, evening and night). These relationships were established for exposure to aircraft noise, road traffic noise and rail traffic noise, and have been adopted by the UK and European Environment Agency (DEFRA 2014; EEA 2010, 2014). The recommended relationships between noise exposure and annoyance are based on the data from a large set of field studies in which data on noise exposure and noise annoyance (as reported by individuals) were collected.

The available noise guidelines have been developed to address noise annoyance within the community. Hence the increase in noise permitted as a result of the project is small. In many cases the change in noise exposure is reduced as a result of the project. However where noise level changes of 2 dB occur, this has the potential to result in an increase in individuals highly annoyed by noise by 2 per cent, which is well below the level of annoyance of 5 per cent considered to be of concern (or likely to be perceived) by residents (Schomer 2005).

<sup>&</sup>lt;sup>12</sup> Confounders are variables (not the ones being studied) that can affect the same health measures/outcomes, and make it appear that an observed exposure is associated with an outcome. These variables can distort the presence of, and magnitude of a relationship that is established between an exposure and an effect/outcome. Good studies try to correct for confounders, however not all of these are known and the way in which the correction is applied can vary.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

### Sleep disturbance

It is relatively well established that night time noise exposure can have an impact on sleep (WHO 2009, 2011). Noise can cause difficulty in falling asleep, awakening and alterations to the depth of sleep, especially a reduction in the proportion of healthy rapid eye movement sleep. Other primary physiological effects induced by noise during sleep can include increased blood pressure, increased heart rate, vasoconstriction, changes in respiration and increased body movements (WHO 2011). Exposure to night-time noise also may induce secondary effects, or so-called after effects. These are effects that can be measured the day following exposure, while the individual is awake, and include increased fatigue, depression and reduced performance.

Studies are available that have evaluated awakening by noise, increased mortality (i.e. increase in body movements during sleep), self-reported chronic sleep disturbances and medication use (EC 2004). The most easily measurable outcome indicator is self-reported sleep disturbance, where there are a number of epidemiological studies available. From these studies the WHO (2009, 2011) identified an exposure-response relationship that relates to the percentage of persons sleep disturbed and highly sleep disturbed to total levels of noise reported as  $L_{night}$  (ie average noise levels during night, which is an eight-hour time period, as measured outdoors). The relationship adopted relates to the assessment of road-traffic noise, with other relationships for air and rail traffic noise. These relationships have been adopted by the WHO (2009, 2011), UK and European Environment Agency (DEFRA 2014; EEA 2010, 2014).

The available noise guidelines include criteria to address sleep disturbance that are based on the above studies and relationships. Hence compliance with these guidelines will address health impacts associated with sleep disturbance in the community.

### Cognitive effects

There is evidence for effects of noise on cognitive performance in children such as lower reading performance (WHO 2011). A major study was undertaken in the EU – RANCH – and this study was reviewed in WHO (2011). The study found an exposure-response relationship between noise and cognitive performance in children for aircraft noise but the relationship between performance and noise for road traffic was much less clear (Stansfeld et al. 2005a; Stansfeld et al. 2005b; WHO 2011). The same study showed that road traffic alone did not show an association between road traffic noise and adverse changes in children's cognitive functions studied (reading comprehension, episodic memory, working memory, prospective memory or sustained attention), nor with sustained attention, self-reported health, or mental health.

### Individual road noise events

It is noted that noise impacts can also occur because of individual noise events, such as engine braking or loud exhausts. The noise measures adopted above for the assessment of the health effects of noise relate to an average/equivalent sound level over different time periods, which, when measured, would include individual noise events. This is the preferred approach for evaluating annoyance and other health effects related to noise (NSW DECCW 2011). Individual noise events are of most significance in relation to the assessment of sleep disturbance. The available research indicates that one or two individual noise events per night, with a maximum indoor noise level of 65-70 dB(A) are not likely to affect health and wellbeing (NSW DECCW 2011). Criteria have been adopted to address maximum noise events, however it is noted that it is not possible to model all individual noise events as these relate to individual vehicles or trucks and individual driving behaviour that cannot be predicted.

# 8.7 Assessment of noise impacts from project

In relation to this project, potential noise impacts have been assessed against Australian (more specifically NSW) criteria that have been established on the basis of the relationship between noise and health impacts. The criteria developed for use in the assessment for control of noise come from policy documents developed by the NSW Government including the Noise Policy for Industry (NPfI) (EPA, 2017), the NSW Interim Construction Noise Policy, and the RNP (NSW DECC 2009; NSW DECCW 2011; NSW EPA 2000). All of these policies are based on the health effects of noise outlined in the reviews published by the following organisations:

- World Health Organization Guidelines on Community Noise Health effects of noise (WHO 1999)
- World Health Organization Night Noise Guidelines for Europe (WHO 2009)
- International Institute of Noise Control Engineering Guidelines for Community Noise Impact Assessment and Mitigation (I-INCE 2011)
- Environmental Health Council of Australia The health effects of environmental noise other than hearing loss (enHealth 2004).

Various attempts have been made to assess the effect (measured by average reported annoyance, sleep disturbance or a similar type of effect) from community noise (measured by long term average sound levels) to develop exposure-response relationships. As individual reactions to noise are so varied, these studies need large sample sizes to obtain reasonable correlation between the noise exposure and the response. Any dose-response relationship determined from large studies over a range of communities and cultures will not necessarily represent the reaction of individuals or small communities. These exposure-response relationships are of value for macro-scale (ie whole urban environment scale) strategic assessment purposes where individual differences are not important; however, they are not as useful when considering potential impacts on a small population located close to a specific project/activity. Hence these macro-scale relationships cannot be easily applied (in any meaningful way) in this assessment.

For a number of the noise guidelines (including the RNP), the criteria has been established on the basis of noise annoyance, which is considered to be the more sensitive effect and an effect that precedes the physiological effects. As a result, these guidelines are designed to be protective of all adverse health effects. Other guidelines are based on specific sensitive health effects such as sleep disturbance for the assessment of night-time noise.

As guidelines/criteria that are based on the protection of health are available to assess construction and operational noise impacts associated with this project, the assessment of potential health impacts has focused on whether the guidelines/criteria established can be met. Where the guidelines cannot be met then there is the potential for the above adverse health effects to occur in the community adjacent to the project.

In most cases, when developing management limits for the project, it has been assumed that there is a 10 dB(A) difference between noise inside and outside of a building with windows open. This assumption is sourced from the RNP. Further consideration of this assumption raises a number of issues including:

- Internal noise levels are defined in the RNP as those measured in the centre of a habitable room so if activities (like sleeping or concentrating) happen at the edge of a room they may be more impacted by noise than might be expected
- The RNP refers to windows being open sufficient to provide adequate ventilation as discussed in the Building Code of Australia. The Building Code of Australia does not require that residential buildings have significant levels of ventilation and, as a result, opening a window sufficient to provide the minimum ventilation required is unlikely to mean that the window is completely open or even that more than one window in a room is opened. Sufficient ventilation may result from the existing drafts in a building (with no windows open) or the opening of two windows only for the entire building. Assuming that the 10 dB(A) change in noise applies for all situations where windows are open is not appropriate
- Consequently, the use of this assumption in setting noise management limits for this project may need to be reviewed when designing property specific noise mitigation measures (to be undertaken in consultation with the property owner).

### 8.7.1 Construction noise

A number of receptors have been identified as highly affected from standard and out of hours construction noise, especially around the Rockdale Construction Ancillary Facility, Cut and Cover Construction and President Avenue, Princes Highway Intersection works and along the powerline installation route. These noise impacts are predicted to be of a significant volume to cause sleep disturbances. Health effects from these noise impacts are likely without the intervention of mitigation measures. The detailed design for the mitigation measures will be outlined in the Construction Noise and Vibration Management Plan (CNVMP) as discussed in **Appendix G** (Noise and vibration: Technical report) and include architectural treatment for those properties that are also identified as being impacted by operational noise. The aim of the mitigation measures should be to reduce noise and vibration to levels that comply with the management goals established in this assessment. If it is not possible to achieve compliance with these goals, health impacts for the affected community are likely.

Construction road traffic noise was estimated to be generally compliant with guideline levels except for roads around the Rockdale construction ancillary facility (especially Wickham Street) during night time periods where increased traffic noise was predicted to be up to 7.3 dB(A). This impact is considerable and night-time haulage should be avoided during night-time off-peak traffic periods to minimise noise impacts.

### 8.7.2 Operational noise

The worst case assessment predicts that noise criteria will be exceeded at a number of properties adjacent to the project without mitigation measures, with 109 properties considered appropriate for mitigation measures due to operational noise.

The worst-case levels estimated are sufficiently high for some receptors that health impacts are likely to occur. These properties are along Princes Highway and President Ave the impacts shown in **Table 8-1** and **Figure 8-3**.

ID	Address	Use	Scenario	L <sub>Aeq (period)</sub> dB(A) <sup>1</sup>				Reason for non-compliance
				Criteria	No Build	Build	Change	
1056	17 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.9	Exceeds cumulative noise limit
1057	21 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.9	Exceeds cumulative noise limit
1058	23 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	75	0.8	Exceeds cumulative noise limit
1060	1 Traynor Ave, Kogarah	Residential	2036 Cumulative Day	60	64	65	0.6	Exceeds cumulative noise limit
1075	59 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	76	1.0	Exceeds cumulative noise limit
1076	61 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	1.0	Exceeds cumulative noise limit
1077	3 Civic Ave, Kogarah	Residential	2036 Build Night	55	58	60	1.4	Exceeds cumulative noise limit
1133	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	62	62	-0.4	Exceeds cumulative noise limit
1135	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	60	59	-0.6	Exceeds cumulative noise limit
1136	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	62	63	0.5	Exceeds cumulative noise limit
1137	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	59	59	-0.5	Exceeds cumulative noise limit
1138	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	67	66	-0.5	Exceeds cumulative noise limit
1139	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	69	68	-0.4	Exceeds cumulative noise limit
1140	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	74	75	0.8	Exceeds cumulative noise limit
1141	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	76	76	0.6	Exceeds cumulative noise limit
1144	800 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	66	65	-0.7	Exceeds cumulative noise limit
1162	732 Princes Hwy, Kogarah	Residential	2036 Cumulative Day	60	74	74	-0.1	Exceeds cumulative noise limit
1163	2 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	75	0.4	Exceeds cumulative noise limit
1164	4-6 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	74	0.4	Exceeds cumulative noise limit
1165	12-14 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.6	Exceeds cumulative noise limit
1166	18 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.7	Exceeds cumulative noise limit
1167	20 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.6	Exceeds cumulative noise limit
1168	22-24 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.6	Exceeds cumulative noise limit

### Table 8-1: Receptors eligible for consideration of additional mitigation measures

ID	Address	Use	Scenario	L <sub>Aeq (period)</sub> dB(A) <sup>1</sup>				Reason for non-compliance
				Criteria	No Build	Build	Change	
1169	34-36 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.7	Exceeds cumulative noise limit
1170	38-40 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	75	0.8	Exceeds cumulative noise limit
1171	42 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.8	Exceeds cumulative noise limit
1172	7-9 Cross St, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.7	Exceeds cumulative noise limit
1173	74 President Ave, Kogarah	Residential	2036 Cumulative Day	60	76	77	1.3	Exceeds cumulative noise limit
2302	35 Crawford Rd, Brighton-Le- Sands	School	2036 Cumulative Day	53 <sup>2</sup>	51	54	2.6	Exceeds the RNP and increases by more than 2.0 dB(A)
2329	146 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	76	1.4	Exceeds cumulative noise limit
2330	156 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	76	1.5	Exceeds cumulative noise limit
2331	160 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	75	1.1	Exceeds cumulative noise limit
2332	162 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	74	0.9	Exceeds cumulative noise limit
2334	62 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	60	66	5.8	Exceeds the RNP and increases by more than 2.0 dB(A) and exceeds cumulative noise limit
2335	52 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	58	62	4.5	Exceeds the RNP and increases by more than 2.0 dB(A)
2336	50 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	57	62	4.7	Exceeds the RNP and increases by more than 2.0 dB(A)
2516	137 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	-0.2	Exceeds cumulative noise limit
2517	139 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.0	Exceeds cumulative noise limit
2518	141 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.1	Exceeds cumulative noise limit
2519	143 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.2	Exceeds cumulative noise limit
2520	145 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.3	Exceeds cumulative noise limit
2521	147 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.4	Exceeds cumulative noise limit
2522	149 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.3	Exceeds cumulative noise limit

ID	Address	Use	Scenario	L <sub>Aeq (period)</sub> dB(A) <sup>1</sup>				Reason for non-compliance
				Criteria	No Build	Build	Change	
2523	151 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.3	Exceeds cumulative noise limit
2524	153 President Ave, Monterey	Residential	2036 Cumulative Day	60	72	73	0.3	Exceeds cumulative noise limit
2525	155 President Ave, Monterey	Residential	2036 Cumulative Day	60	73	73	0.2	Exceeds cumulative noise limit
2526	157-159 President Ave, Monterey	Residential	2036 Cumulative Day	60	72	73	0.4	Exceeds cumulative noise limit
2527	157-159 President Ave, Monterey	Residential	2036 Cumulative Day	60	73	73	0.4	Exceeds cumulative noise limit
2528	161-163 President Ave, Monterey	Residential	2036 Cumulative Day	60	73	73	0.4	Exceeds cumulative noise limit
2529	165-169 President Ave, Monterey	Residential	2036 Cumulative Day	60	74	74	0.3	Exceeds cumulative noise limit
2893	1A Civic Ave, Kogarah	Residential	2036 Cumulative Day	60	67	68	1.3	Exceeds cumulative noise limit
3127	1 Lachal Ave, Kogarah	Residential	2036 Build Night	55	59	60	1.0	Exceeds cumulative noise limit
3128	19 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.9	Exceeds cumulative noise limit
3129	25 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	76	0.7	Exceeds cumulative noise limit
3130	27 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.6	Exceeds cumulative noise limit
3158	2 Traynor Ave, Kogarah	Residential	2036 Cumulative Day	60	66	66	0.5	Exceeds cumulative noise limit
3161	1 Oakdale Ave, Kogarah	Residential	2036 Cumulative Day	60	66	66	0.5	Exceeds cumulative noise limit
3170	57 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	1.1	Exceeds cumulative noise limit
3171	63 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	76	1.1	Exceeds cumulative noise limit
3173	1 Civic Ave, Kogarah	Residential	2036 Cumulative Day	60	64	65	1.4	Exceeds cumulative noise limit
3302	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	77	77	-0.2	Exceeds cumulative noise limit
3303	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	61	61	0.3	Exceeds cumulative noise limit
3304	750 Princes Hwy, Kogarah	School	2036 Cumulative Day	53 <sup>2</sup>	75	75	0.5	Exceeds cumulative noise limit
3314	8-10 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.5	Exceeds cumulative noise limit
3315	30-32 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.7	Exceeds cumulative noise limit
3316	48 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	75	0.7	Exceeds cumulative noise limit
3317	50 President Ave, Kogarah	Residential	2036 Cumulative Day	60	73	74	0.7	Exceeds cumulative noise limit

ID	Address	Use	Scenario	L <sub>Aeq (period)</sub> dB(A) <sup>1</sup>				Reason for non-compliance
				Criteria	No Build	Build	Change	
3318	52 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	74	0.7	Exceeds cumulative noise limit
3319	54 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	75	0.7	Exceeds cumulative noise limit
3320	56 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	74	0.8	Exceeds cumulative noise limit
3321	58 President Ave, Kogarah	Residential	2036 Cumulative Day	60	74	74	0.8	Exceeds cumulative noise limit
3323	62 President Ave, Kogarah	Residential	2036 Cumulative Day	60	73	73	0.7	Exceeds cumulative noise limit
3324	64 President Ave, Kogarah	Residential	2036 Cumulative Day	60	77	77	0.9	Exceeds cumulative noise limit
3325	66 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	75	0.8	Exceeds cumulative noise limit
3326	68 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	75	0.9	Exceeds cumulative noise limit
3327	70 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	76	1.0	Exceeds cumulative noise limit
3328	72 President Ave, Kogarah	Residential	2036 Cumulative Day	60	75	76	1.1	Exceeds cumulative noise limit
3329	467 W Botany St, Kogarah	Residential	2036 Cumulative Day	60	71	72	1.2	Acute
3332	730 Princes Hwy, Kogarah	Residential	2036 Cumulative Day	60	74	74	-0.3	Exceeds cumulative noise limit
3333	726-728 Princes Hwy, Kogarah	Residential	2036 Cumulative Day	60	74	74	-0.4	Exceeds cumulative noise limit
3334	726-728 Princes Hwy, Kogarah	Residential	2036 Cumulative Day	60	74	74	-0.4	Acute
3648	35 Crawford Road, Brighton-Le- Sands	School	2036 Cumulative Day	53 <sup>2</sup>	51	53	2.1	Exceeds the RNP and increases by more than 2.0 dB(A)
3649	35 Crawford Rd, Brighton-Le- Sands	School	2036 Cumulative Day	53 <sup>2</sup>	52	54	2.2	Exceeds the RNP and increases by more than 2.0 dB(A)
3650	35 Crawford Rd, Brighton-Le- Sands	School	2036 Cumulative Day	53 <sup>2</sup>	52	54	2.1	Exceeds the RNP and increases by more than 2.0 dB(A)
3651	35 Crawford Rd, Brighton-Le- Sands	School	2036 Cumulative Day	53 <sup>2</sup>	51	53	2.4	Exceeds the RNP and increases by more than 2.0 dB(A)
3664	49 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	57	62	4.1	Exceeds the RNP and increases by more than 2.0 dB(A)
3665	51 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	59	63	4.0	Exceeds the RNP and increases by more than 2.0 dB(A)
3666	51 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	58	61	3.7	Exceeds the RNP and increases by more than 2.0 dB(A)

ID	Address	Use	Scenario	L <sub>Aeq (period)</sub> dB(A) <sup>1</sup>				Reason for non-compliance
				Criteria	No Build	Build	Change	
3667	51 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	57	61	3.6	Exceeds the RNP and increases by more than 2.0 dB(A)
3671	148 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	76	1.7	Exceeds cumulative noise limit
3672	150 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	76	1.6	Exceeds cumulative noise limit
3673	152 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	76	1.6	Exceeds cumulative noise limit
3674	154 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	76	1.5	Exceeds cumulative noise limit
3675	158 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	75	1.3	Exceeds cumulative noise limit
3676	164 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	73	73	0.7	Exceeds cumulative noise limit
3677	166 President Ave, Brighton-Le- Sands	Residential	2036 Cumulative Day	60	74	74	0.6	Exceeds cumulative noise limit
3680	66 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	62	67	5.3	Exceeds the RNP and increases by more than 2.0 dB(A) and exceeds cumulative noise limit
3681	64 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	59	66	6.4	Exceeds the RNP and increases by more than 2.0 dB(A) and exceeds cumulative noise limit
3682	60 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	57	64	6.7	Exceeds the RNP and increases by more than 2.0 dB(A)
3683	58 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	59	65	5.8	Exceeds the RNP and increases by more than 2.0 dB(A) and exceeds cumulative noise limit
3684	56 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	57	63	6.4	Exceeds the RNP and increases by more than 2.0 dB(A)
3685	54 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	58	63	5.1	Exceeds the RNP and increases by more than 2.0 dB(A)
3686	48 O'Neill St, Brighton-Le-Sands	Residential	2036 Cumulative Day	60	58	62	3.7	Exceeds the RNP and increases by more than 2.0 dB(A)
3827	38 Gladstone St, Kogarah	Residential	2036 Cumulative Day	60	67	67	-0.5	Exceeds cumulative noise limit
3886	1-5 Hogben St, Kogarah	Other	2036 Cumulative Day	60	66	65	-0.7	Exceeds cumulative noise limit

ID	Address	Use	Scenario	L <sub>Aeq (period)</sub> dB(A) <sup>1</sup>				Reason for non-compliance
				Criteria	No Build	Build	Change	
3903	69 Gladstone St, Kogarah	Residential	2036 Cumulative Day	60	71	70	-0.4	Exceeds cumulative noise limit
3904	58 Premier St, Kogarah	Residential	2036 Cumulative Day	60	65	65	-0.2	Exceeds cumulative noise limit
3907	63-67 Gladstone St, Kogarah	Residential	2036 Cumulative Day	60	68	67	-0.5	Exceeds cumulative noise limit
3936	79-103 Princes Hwy, Kogarah	Other	2036 Cumulative Day	60	79	78	-0.8	Exceeds cumulative noise limit

Notes:

1 Daytime parameter is L<sub>Aeq(15 hr</sub>). Night-time parameter is L<sub>Aeq(9 hr</sub>). Schools are L<sub>Aeq(1 hr</sub>), during school hours.

2 The applicable school classroom criteria is L<sub>Aeq(1 hr)</sub> 40dB(Å), internal. Assuming a conservative external to internal reduction of 10 dB(Å) for the purpose of this assessment, this makes an equivalent 50 dB(Å) external. The noise predictions add façade reflection of 2.5 dB(Å) to the overall noise level on the façade, which is not included when calculating the internal noise level. To account for the façade reflection included in the predictions, 2.5 dB(Å) has been added to the criteria. No correction has been applied from a L<sub>Aeq(15 hr)</sub> to L<sub>Aeq(1 hr)</sub>. Due to traffic flows hours of use (9am to 3pm), the L<sub>Aeq(1 hr)</sub> peak noise levels do not significantly diverge from the L<sub>Aeq(15 hr)</sub> noise levels.



F6 Extension S1

Receiver exceeds the Cumulative Noise Limit in any scenario

Receiver exceeds the RNP Criteria and Increases >2dB(A) in any scenario

#### Acute

### Figure 8-3: Receivers eligible for the consideration of noise mitigation

The criteria for consideration of noise mitigation from operational noise was either if the noise criteria was exceeded by 2.0 dB(A) OR the cumulative noise exceeded the noise criteria by 5 dB(A) and the receiver is impacted by the project, regardless of the increment. As it can be seen in table 6.1, many of the receivers are already above the noise criteria, so mitigation measures may actually provide a net benefit to those receptors.

Mitigation measures considered during operation would principally involve the use of low noise pavement, noise mounds and noise barriers, where possible. However, for 109 buildings these mitigation measures do not provide sufficient or appropriate mitigation, and so have been identified for in-property treatment.

The use of in property treatment have a number of downsides, and therefore treatment at or near the source should be the preferred option. In property treatment downsides include:

- Loss of use of outdoor areas. In urban areas particularly where existing levels of noise are dominated by road traffic noise, access to outdoor green space areas that are not (perceived to be) impacted by noise (eg where there is a quiet side of a specific property or there is access to a quiet green space areas close to the residential home) have been found to significantly improve wellbeing and lower levels of stress (Gidlöf-Gunnarsson & Öhrström 2007). Impacts on the use and enjoyment of outdoor areas due to increased noise may result in increased levels of stress at individual properties
- The requirement that residents take up in-property treatment measures and where they do, they keep external windows and doors shut and have minimal use of outdoor areas. Where specific residents/properties do not take up recommended in-property treatments to mitigate noise indoors there is the potential for noise levels at these properties to exceed the relevant guidelines/criteria. In these situations, there is the potential for adverse health effects, particularly annoyance and sleep disturbance, to occur.

Community consultation will be an important part of the process in addressing noise impacts for the project as there are a number of individual homes where in-property treatment will be required to enable the noise criteria to be met, and minimise the potential for adverse health effects associated with the project. However, such treatments may have other effects (as discussed above) which will also need to be managed/considered.

# 9 Public safety and contamination

## 9.1 General

This section provides a review of the potential risks posed to public safety, associated with the project. This section also presents a review of health impacts associated with the presence and management of contamination (in soil or water) relevant to the project.

This section only addresses risks to the community, ie risks that only have the potential to adversely affect the community. Issues relevant to workplace health and safety during construction (including contamination remediation) and operation have not been further discussed or addressed.

Evaluation of public safety has considered the hazard and risk assessment, presented in **Chapter 10** of the EIS (Health, safety and hazards). This assessment was undertaken in accordance with the State Environmental Planning Policy No.33 (The Policy) *Hazardous and Offensive Developments*, that identified and addresses risks during construction and operation. Pedestrian safety aspects are addressed in detail in the Traffic and Transport assessment. Issues from these assessments specifically relevant to public health and safety have been further detailed in this section.

Health impacts associated with contamination have been assessed on the basis of **Appendix J** of the EIS (Contamination technical report).

Health impacts associated with subsidence have been assessed on the basis of **Chapter 17** of the EIS (Groundwater and geology) and **Chapter 14** of the EIS (Property and land use).

# 9.2 Public safety

### 9.2.1 Construction

A range of potential hazards have been identified that have the potential to affect public safety during construction. These are outlined in **Table 9-1**, along with discussion on the risks that may be posed by these hazards. Not all the hazards identified in the Hazard and Risk assessment have been included in the table, only those where there is the potential for risks to public safety.

Table 9-1: Overview of	public safet	y hazards and	risks:	Construction
------------------------	--------------	---------------	--------	--------------

Hazard: Public safety	Risk to public safety	Management measures
Storage and handling of dangerous goods on construction sites that may impact on the off-site community	Low The storage would comply with screening thresholds prescribed under SEPP 33.	All materials will be stored in accordance with appropriate Acts, Standards and Code that includes the use of bunding and ventilation of areas where gases are stored, maintaining a register and inventory.
Transport of dangerous goods and hazardous substances on public roads within the community	Low The transportation would comply with screening thresholds prescribed under SEPP 33.	All materials are to be transported in accordance with the Storage and Handling of Dangerous Goods Code of Practice (WorkCover NSW 2005), Dangerous Goods (Road and Rail Transport) Act 2008 (NSW), Dangerous Goods (Road and Rail Transport) Regulation 2014 (NSW) and relevant Australian Standards.
Tunnel collapse, that may affect community areas overlying the tunnel	Low	All tunnelling to be undertaken under a permit to tunnel system that requires consideration of ground support performance, geotechnical and groundwater conditions for each tunnel section.
Acid sulfate soil, that may result in acidification and the mobilisation of metals, adversely impacting groundwater that can then migrate off-site	Low	Standard construction and mitigation measures would be applied to mitigate the potential risks associated with the disturbance of acid sulfate soils.

Hazard: Public safety	Risk to public safety	Management measures
Contamination, specifically the presence of hazardous materials such as asbestos and works in areas where contamination is present in soil, which may result in contaminants migrating off-site and affecting the community	Low	Removal of asbestos is required to be undertaken in accordance with procedures detailed in the Asbestos Management Plan as part of the Construction Environmental Management Plan (CEMP) for the project, which reflect national legislation and guidance. Other contaminants would be managed using relevant guidelines.
Flooding issues that extend outside the construction areas into the community	Low as flooding risks to off-site areas evaluated have been considered to be minor.	Design to minimise the potential for off-site flooding impacts.
Damage to underground utilities, affecting roadways and services provided to the community	Low	A preliminary assessment of utilities in the area has been undertaken as well as consultation with utilities and service infrastructure providers to mitigate the risk of unplanned or unexpected disturbance of utilities.
Bushfire or fire risks that may spread off-site and affect neighbouring	Low	The project is in a highly urbanised area that is not in or near a bushfire prone area.
properties		Management of construction facilities and activities involving flammable materials and ignition sources will be undertaken to minimise fire risks. High risk construction activities, such as welding and metal work, would be subject to a risk assessment on total fire ban days, and restricted or ceased as appropriate.
Aviation risks, specifically works that may affect the safety of aircraft using Sydney Airport	Low	Construction activities would be carried out to ensure that equipment such as cranes and materials do not intrude into the obstacle limitation surface (OLS) or procedures for air navigation systems operations (PANS-OPS) for the airport. The Civil Aviation and Safety Authority (CASA) and Department of Infrastructure, Regional Development and Cities (DIRDC) are being consulted to ensure construction works are undertaken in line with the Airports (Protection of Airspace) Regulations 1996 (Commonwealth), in a manner that satisfies the requirements of CASA. This includes compliance with CASA requirements for lighting.
Traffic and trucks on surface roads and the potential for changes in public safety	Low Changes to the surface road network may require temporary traffic detours. Construction road traffic volumes are expected to be low compared with existing traffic volumes, which is not expected to substantially impact on road safety.	A Construction Traffic and Access Management Plan (CTAMP) will be prepared to manage these impacts.
Pedestrian and cycle safety	Low Pedestrian and cycle safety assess as negligible.	Alternate safe pedestrian and cycle access is to be provided where it is practical and safe to do so. This will be addressed in the Construction Traffic Management and Access Plan (CTAMP).

Hazard: Public safety	Risk to public safety	Management measures
Subsidence	Low	Monitoring of settlement throughout the construction program would be included as part of the Construction Environmental Management Plan (CEMP) and may include the installation of settlement markers or inclinometers. Pre-construction condition surveys of property and infrastructure that could be impacted by settlement would be undertaken before the commencement of construction activities. Groundwater inflow control measures to also be undertaken.

On the basis of the above there are no issues related to construction that have the potential to result in significant safety risks to the community.

#### 9.2.2 **Operation**

A range of potential hazards have been identified that have the potential to affect public safety during the operation of the project, principally in relation to traffic accidents. These are outlined in Table 9-2, along with discussion on the risks that may be posed by these hazards. Not all the hazards identified in the Hazard and Risk assessment have been included in the table, only those where there is the potential for risks to public safety.

•		•
Hazard: Public safety	Risk to public safety	Management measures
Storage, handling and transport of dangerous goods required for maintenance of the project, that may impact on the off-site community	Low The storage would comply with screening thresholds prescribed under SEPP 33.	All materials will be stored and transported in accordance with the relevant legislation and codes.
Transport of dangerous goods and hazardous substances in project tunnels	Low The transport of these materials will be prohibited within the tunnels (as per Road Rules 2014, 300-2 NSW rule: carriage of dangerous goods in	The transport of dangerous goods and hazardous substances will be prohibited through the mainline tunnels and entry and exit ramps during operation. Signage will be provided near tunnel entry portals advising of the restrictions to ensure compliance.

Table 9-2: Overview of public	safety hazards	and risks: C	peration
-------------------------------	----------------	--------------	----------

	prohibited areas).	
Traffic accidents in the tunnel	Low to moderate All use of public roadways carries an inherent risk of vehicle collision. The project has been designed to minimise these risks for travel within the tunnels. The project also provides fire and life safety requirements.	Use of height detection systems prior to tunnel entry portals, tunnel barrier gates to prevent access if the tunnel is closed, CCTV throughout the tunnel, adjustable speed signs, provision of breakdown bays and emergency phones, provision of pedestrian cross-passages to enable safe evacuation from the tunnel, automated fire detection, longitudinal ventilation to push smoke in the direction of traffic flow away from the fire source towards a ventilation facility or portal, water deluge system that can be activated manually or automatically.

Hazard: Public safety	Risk to public safety	Management measures
Traffic accidents on surface roads (including pedestrian and cycle safety)	Moderate, however the risk is considered to be reduced with the project	The design of the project has been developed to inherently minimise the likelihood of incidents and crashes. The project will involve a reduction in traffic on some roadways, which has the potential to reduce crash rates, improve pedestrian and cyclist safety.
EMF from new substations	Low	The project substations will be designed to ensure that the exposure limits for the general public detailed in by the Draft Radiation Standard (Australian Radiation Protection and Nuclear Safety Agency 2006) will not be exceeded at the boundary of the substation sites.
Bushfire risks	Low	The project is in a highly urbanised area that is not in or near a bushfire prone area. Operational infrastructure is largely invulnerable to bushfires as it is not combustible.
Aviation risks, specifically works that may affect the safety of aircraft using Sydney Airport	Low	The project design has considered airspace protection and associated risk and hazards. This includes the design of lighting and the ventilation facilities to ensure they meet the safety requirements set by DIRDC and CASA.
Subsidence	Low to moderate risk of damage to buildings Predicted movements up to 50mm. Damage to buildings and structures can have subsequent impacts on human health (stress and anxiety)	A geotechnical model of representative geological and groundwater conditions would be prepared during the detailed design phase prior to the commencement of tunnelling. The model would be used to assess predicted settlement impacts and ground movement during the construction and operation of the project. Any damage to buildings would be repaired. Any stress and anxiety experieicned by property owners are expected to be temporary.

On the basis of the above there are no issues related to the operation of the project that have the potential to result in significant safety risks to the community.

# 9.3 Contamination

Contamination risk issues to the community are more relevant to the construction phase of the project because exposure to contaminated soil or groundwater would most likely occur during the excavation and construction phase, if not appropriately managed. The interaction with contamination and the community during the operations phase is primarily related to spills and accidents associated with the completed motorway. **Appendix J** (Contamination technical report) has considered the location of the construction activities in relation to known areas of contamination in soil and groundwater, as well as issues associated with the impact of construction on the environment, where the community may be exposed.

### 9.3.1 Construction

In relation to construction works, the following areas have been identified, and ranked as posing a medium or high risk<sup>13</sup>) that require further investigation and management:

- Identified areas ancillary facilities and construction footprint:
  - Arncliffe ancillary facility (medium)
  - Rockdale construction ancillary facility (medium)
  - President Avenue construction ancillary facility, specifically parts of Bicentennial Park, Memorial Playing Fields (high), Rockdale ventilation facility construction area (427 to 441 West Botany Street, Rockdale), West Botany Street (medium), substation within St. George TAFE (medium), shared cycle and pedestrian pathways, including the bridge over President Avenue (high) and acquired houses along northern side of President Avenue (medium)
  - Shared cycle and pedestrian pathways construction ancillary facilities (medium)
  - Princes Highway construction ancillary facility (high)
  - Permanent power supply connection (power line) (medium)
- Identified areas with a medium or high risk of groundwater contamination due to historical land uses, that could impact on groundwater quality along the tunnel alignment:
  - Kogarah Golf Course and surrounding filled land to the south
  - Former Tempe Bus Depot in Arncliffe
  - Up-gradient former and current commercial/ industrial properties (mainly mechanics and workshops) along Princes Highway, Arncliffe
  - Former Goodfellow Dry Cleaners at 122 Cameron St, Rockdale
  - Rockdale industrial area
  - Bicentennial Park and surrounding filled land

There is also a potential that contamination arising from tunnel construction and associated project works, such as stockpiling of contaminated soil and acid sulfate soils and storage and use of fuel and chemicals, could adversely impact soil, sediment, groundwater and surface water if not managed appropriately.

The areas identified as medium and high risk within the construction footprint would be further investigated. **Appendix J** (Contamination technical report) outlines the measures required to be adopted during construction to manage soil and water contamination. These are to be outlined in detail in the Construction Environmental Management Plan (CEMP). For sites where remediation is required a remedial action plan (RAP) would be required. In some cases, where limited information is currently available on contamination a detailed site investigation (DSI) is required. A DSI and RAP, and all remediation works are required to be undertaken in accordance with guidance from the NSW EPA, including obtaining approved by an independent NSW EPA accredited site auditor. This process is required to ensure assessment and remedial works adequately address and prevent risks to human health, including the surrounding community.

During tunnelling works, groundwater would be extracted and would be collected, treated and discharged in accordance with the adopted site guidelines. Specifically, water would be treated onsite, at the Arncliffe water treatment plant or at the temporary construction water treatment plants and sedimentation ponds at the Rockdale construction ancillary facility and President Avenue construction ancillary facility. The surface water receiving body from the Arncliffe water treatment plant would be the Cooks River. in the vicinity of the project that have the potential to be impacted if groundwater disposal is not effectively addressed include Cooks River. Discharged water would be required to comply with the NSW Water Quality Objectives. Meeting these guidelines would ensure that discharged water would not affect the health of the community using these waterways for recreation.

<sup>&</sup>lt;sup>13</sup> The level of risk depends on the likelihood of contamination being present, including the concentrations that may be present, and the likelihood that the community or an environment may be exposed to the contamination, as a result of the project, prior to any control measures in place.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

### 9.3.2 Operation

During operation, groundwater seepage would be required to be extracted from the tunnels, treated and discharged to the receiving water bodies. The groundwater quality maybe impacted along parts of the tunnel alignment due to overlying contamination sources.

Tunnel drainage infrastructure will be designed to accommodate a combination of water ingress events including groundwater ingress, stormwater ingress at portals, tunnel wash-down water, fire suppressant deluge or fire main rupture and spillage of flammable and other hazardous materials. Tunnel drainage would be pumped to an operational water treatment plant at Arncliffe Motorway Operations Complex, with treated flows ultimately discharged to the Cooks River.

The site will be managed under an Operational Plan. **Appendix J** (Contamination technical report) outlines the measures required to be adopted in this operational plan. That includes storage of chemicals and products associated with the operation of the project as well as groundwater and surface water impacts.

# 10 Assessment of changes in social aspects on community health

# 10.1 General

The World Health Organization defines health as 'a (dynamic) state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity'. Hence the assessment of health should include both the traditional/medical definition that focuses on illness and disease as well as the broader social definition that includes the general health and wellbeing of a population.

The assessment of changes in air quality and noise on the health of the local community (presented in **sections 6, 7** and **8**) addressed key aspects that have the potential to directly affect health.

This section has more specifically evaluated changes in the community that have the potential to indirectly affect the health and wellbeing of the community. This section also provides a review of whether there are any impacts that are likely to be more significant in any section of the community, and if these areas may result in inequitable impacts on the health of the population. This may affect population groups that may be advantaged or disadvantaged based on age, gender, socioeconomic status, geographic location, cultural background, aboriginality, current health status or existing disability. The evaluation presented in this section provides a qualitative evaluation of potential health impacts on the community.

Within an urban environment there are a wide range of complex factors (acting and interacting at different scales) that can affect health and wellbeing. This is conceptualised in **Figure 10-1** (presented by the International Council for Science and similar to that defined by the WHO) (ICSU 2011), that also presents a summary of the outcomes of this assessment. The broad range of factors identified may result in either positive or negative impacts on health and wellbeing. It is noted that no single element or determinant acts in isolation. Health and wellbeing in the urban environment depends on the sum of the total interactions between many factors. It is within this complex model that changes associated with the F6 Extension project have been evaluated in relation to impacts on health and wellbeing.

Chapter 15 - Social and economic of the environmental impact statement provides details in relation to many of the social impacts associated with the project. Aspects that are specifically relevant to potential impacts on the health and wellbeing of the community, either positive or negative, have been further highlighted in this section.

# **10.2** Changes in traffic

The study area broadly encompasses an area extending from St Peters in the north to Kogarah in the south. It is predominantly focused on the corridor between St Peters and Kogarah, as well as the surface road networks around the President Avenue and St Peters interchanges.

The President Ave intersection and surrounding area include the key roads of Princes Highway (classified highway), The Grand Parade, President Ave and Bay St (classified as main roads) and the regional roads of West Botany St, Chuter Ave / O'Connell St.

The St Peters interchange and surrounding area includes links with the M5 East Motorway corridor that provides the main passenger, commercial and freight connection between South West Sydney and the Sydney CBD, Sydney Airport and Port Botany. It is centred on the area bounded by Princes Highway, Canal Road, Burrows Road and Campbell Road. Key roads in the vicinity of the project include Princes Highway / King Street / Canal Road / Gardeners Road / Burrows Road / Campbell Road. Campbell St / Euston Road and Bourke Road.

### 10.2.1 Construction

A number of changes to local roads are proposed during the construction phase of works. While it is expected that access to all properties on the local roads would be maintained during the construction works, some permanent and temporary closures or reduced capacity of some local roads may affect the movement of local traffic through the area. In relation to traffic changes in the project area during construction, most of the issues that are relevant to community health relate to public safety, which is addressed in **Section 9**.

In addition to safety risks to the public, construction works are expected to result in some increases in travel times for motorists, bus travel, pedestrians and cyclists. These changes have the potential to result in increased levels of stress and anxiety in the local community (as discussed below). These impacts, however, are expected to occur during the period of construction only.

A CTAMP would be prepared for the project, detailing temporary road closures and including traffic control procedures, signage requirements, construction traffic management requirements of the relevant Roads and Maritime manuals and procedures and Australian Standards. For the partial road closures that would occur due to the construction of the permanent power supply line a Traffic Control Plan (TCP) and Road Occupancy Licence (ROL) would be submitted for approval to the relevant authorities prior to works in several construction areas along the route.

### 10.2.2 Operations

Once the project is complete, it is expected to result in reductions in vehicle delays in a number of areas. There are some areas, however, where traffic volumes would increase, mainly around the President Ave corridor.

Traffic congestion and long commuting times can contribute to increased levels of stress and fatigue, more aggressive behaviour and increased traffic and accident risks on residential and local roads as drivers try to avoid congested areas (Hansson et al. 2011). Increased travel times reduce the available time to spend on heathy behaviours such as exercise, or engage in social interactions with family and friends. Long commute times are also associated with sleep disturbance, low self-rated health and absence from work (Hansson et al. 2011). Reducing travel times and road congestion is expected to reduce these health impacts.

### **10.2.3 Public transport**

Access to public transport is important, particularly for people who cannot or are unable to drive (such as the elderly and those with disabilities). Lack of good access to public transport for these individuals can result in increased feelings of isolation, helplessness and dependence.

During construction of the project, public transport in the project corridor and surrounding areas will be temporarily affected. The construction of the F6 Extension would not directly affect heavy rail or light rail services however passenger access to stations may be affected by temporary traffic changes and congestion arising from the presence of construction works. Most impacts related to the project relate to bus travel, where construction activities would result in the relocation of some bus stops and increased travel times.

From a public transport network perspective, the project, once complete, is expected to slightly increase bus travel times in 2026 AM peak periods around President's Ave intersection, with minimal time changes over other periods. Minimal changes in bus travel times are predicted around the St. Peters interchange and surrounds.

### **10.2.4** Pedestrian and cycle access

Walking and cycling have many health benefits including maintaining a healthy weight and improved mental status (Hansson et al. 2011; Lindström 2008; Wen & Rissel 2008; WHO 2000b).

There is currently a network of cycle paths in the area, comprising a mixture of separated cycleways and on road paths in areas of medium to high difficulty for on road cyclists.

During construction, temporary alterations and diversions to pedestrian and cyclist networks have the potential to affect commuter departure times, travel durations, movement patterns and accessibility. Construction and operation of the project would result in changes to pedestrian and cycle access, including temporary and permanent closures or diversions of some pathways and pedestrian bridges, especially along Presidents Avenue and Rockdale wetlands. While the opportunity to walk or cycle in the project area would be addressed in a Construction Traffic and Access Management Plan (CTAMP), the alterations and changes to amenity may detract from the experience of an environment and potentially deter people from enjoying an active lifestyle or feeling connected with their community. Hence it is important that the diversions and detours are safe, and perceived by the community to be a safe alternative.

Once completed, the project would deliver new pedestrian and cyclist infrastructure project in the form of Shared cycle and pedestrian pathways. The Shared cycle and pedestrian pathways would be developed from Bestic Street, Brighton-le-Sands south to Civic Avenue, Kogarah through the reinstated Rockdale Bicentennial Park. As part of the corridor a dedicated shared overpass would be built over President Avenue.

Improvements in the active transport network, including improvements in transport connections, will have a positive benefit on community health. Where active transport opportunities are improved and offer safe alternatives to driving and public transport, they can encourage more active recreation and commuting activities.

### **10.2.5** Impacts on health and emergency services

The existing arterial roads and the local road network are currently used by emergency services to travel to and from call-outs. Construction of the project may require temporary traffic diversions, road occupation, temporary road closures and alternative property access arrangements. Comprehensive communication of changes to roads or paths to emergency services will be an integral part of the CTAMP.

# **10.3 Property acquisitions**

The project has been designed and developed to minimise the need for surface property acquisition. Where property acquisition could not be avoided, impacts have been balanced by maximising opportunities for the beneficial re-use of land that is required for construction of the project but not operation.

Notwithstanding, the project does require 13 property acquisitions as well as other temporary and permanent impacts on land use.

The acquisition and relocation of households and businesses due to property acquisition can disrupt social networks and affect health and wellbeing due to raised levels of stress and anxiety. This includes increased levels of stress and anxiety during the process of negotiating reasonable compensation. The purchase of and moving into a house can be one of the most significant events in a person's life. Both a house and a workplace are central to daily routine with the location of these premises influencing how a person may travel to/from work or study, the social infrastructure and businesses they visit and the people they interact with.

Impacts associated with property acquisition would be managed through a property acquisition support service that would provide the following:

- Affected households would have access to a counselling service that would assist people through the property acquisition process and, where necessary, provide referrals to more specialised experts
- A property acquisition factsheet that outlines the process and provides further information for concerned residents is to be prepared and made available online and in hard copy at project information centres
- An independent service would be provided to vulnerable households (eg elderly, those suffering an illness) to assist with relocation. Assistance could include finding a suitable house for relocation (purchase or rent), arranging removalists, disconnecting services and attending appointments with solicitors or other representatives
- A community relations support toll-free telephone line is to be established to respond to any community concerns or requests for translation services.

All acquisition required for the project would be undertaken in accordance with the Land Acquisition (Just Terms Compensation) Act 1991 (NSW), the Land Acquisition Information Guide (NSW Government 2014) and the land acquisition reforms announced by the NSW Government in 2016 (2016 reform).

# 10.4 Green space

Green space within urban areas includes green corridors (paths, rivers and canals), grassland, parks and gardens, outdoor sporting facilities, playing fields and children play areas. Epidemiological studies have been undertaken that show a positive relationship between green space and health and wellbeing (de Vries et al. 2003; Health Scotland 2008; Kendal et al. 2016; Maas et al. 2006; Mitchell & Popham 2007). The outcomes of these international studies from the literature did depend on the quality of the available green space. They showed that green space areas in low socio-economic areas often had poor facilities, higher levels of graffiti, vacant/boarded up buildings and lower levels of safety. These studies showed that such spaces had few health benefits.

The health benefits of green space in urban areas include the following (Health Scotland 2008; Kendal et al. 2016; Lee & Maheswaran 2011):

- Green space areas that include large trees and shrubs can protect people from environmental exposures associated with flooding, air pollution, noise and extreme temperature (by regulating microclimates and reducing the urban heat island effect)
- Reduced morbidity
- Improved opportunities for physical activity and exercise. The benefits depend on a range of factors including the distance, ease of access, size of green space, location in relation to connectivity to residential or workplace areas, attractiveness, available facilities (particularly where used by specific sporting clubs) and multi-use (ie including children play areas, garden, seating, sporting facilities that can be used by a wide range of the community for different purposes)
- Improved mental health and feelings of wellbeing, particularly lower stress levels
- Improve opportunities for social interactions.

Green space areas in urban areas may also present some hazards, such as attracting antisocial behaviours (particularly in isolated areas), providing areas for drug or sexual activity and unintentional injuries from sports or use of playground equipment. It has also been found that individuals from ethnic or minority groups and those with disabilities are less frequent users of use green spaces areas.

The construction works will remove two sporting pitches in Memorial Fields as well as an existing car park in the north west corner of Bicentennial Park. **Table 10-1** provides a summary of the open space areas impacted by construction and operation.

Impacts to green space as a result of the project may reduce opportunities for physical activity and exercise, social interactions and increase in stress levels for the community. A reduction in green spaces with trees and shrubs (for example, parts of Rockdale Bicentennial Park) may also reduce the protection offered by these green spaces from air pollution, noise and extreme temperatures.

### Table 10-1: Impacts to green space during construction and operation

Construction impacts to open space	Operational impacts to open space			
Rockdale Bicentennial Park				
Acquisition of approximately 1.1 hectares plus temporary lease of 7.6 hectares. Works would temporarily restrict access to much of Rockdale Bicentennial Park and the recreational facilities located within the park including the Rockdale Skate Park and disability playground. These facilities would be temporarily relocated to a nearby area of open space to allow the community to continue to benefit from their use during the construction period. The Bicentennial East soccer fields would be temporarily relocated and Brighton Memorial Playing Fields, may be reconfigured at their current location to allow the community to continue to benefit from their use during the construction period.	Intension that much of the space would be reinstated as parkland and would include landscaping works. A concept design for urban design and landscaping works at Bicentennial Park has been prepared (refer to <b>Appendix C</b> (Place making and urban design)).			
Scarborough Park North				
Acquisition of approximately 0.5 hectares plus temporary lease of 0.5 hectares	Nil, during operation the project infrastructure (shared cycle and pedestrian pathways) would continue to function as open space			

Construction impacts to open space	Operational impacts to open space			
Kogarah Golf Course				
Acquisition of approximately 0.7 hectares plus temporary lease of 6 hectares. Reduction of golf course from 18 holes to 9 holes	Loss of approximately 0.7 hectares of the Golf Course.			
Bardwell Valley Golf Course				
The installation of an underground power cable from Canterbury subtransmission station to the project in Rockdale would require temporary occupation of some parts of the course. It is expected that impacts would be limited to discrete construction areas and would not require the full closure of any particular holes, though some may be temporarily shortened during construction.	Nil			

# **10.5** Changes in community access and connectivity

Roads and freeways can divide residential communities hindering social contact. The presence of busy roads inhibits residents from socialising and children from playing, or accessing nearby recreational areas. Heavy traffic also affects child development (WHO, 2000). Children learn how to make responsible decisions, how to behave in different situations and develop a relationship with their environment and community through independent mobility. Where children have the opportunity to be able to play in local streets or safely access local parks they have been found to have twice as many social contacts as those where such activities are prevented by heavy traffic or unsafe conditions.

Social connectedness and relationships are important aspects of feeling safe and secure. Streets with heavy traffic have been associated with fewer neighbourhood social support networks and has been linked to adverse health outcomes (WHO 2000b). Any temporary and permanent changes to the access to social infrastructure, community resources or to other desirable locations (such as employment, study, friends and family) and safety to movement may affect community networks and in turn trigger community severance.

Community severance effects often occur during major transportation projects (during construction and operation) due to detours in the local road network, changes to active and public transport routes, and connector roads receiving an increase or decrease in traffic movements. The changes to the road networks may contribute to feelings of community severance and disconnection. The project is not introducing new major roadways that would change existing conditions.

Construction of the project would involve the temporary disruption of pedestrian and cycleway routes especially around Rockdale Bicentennial Park. This reduced connectivity may deter people from participating in community activities or active transport, potentially reducing the connection to an environment and feeling of community cohesion.

# 10.6 Visual changes

Visual amenity can be described as the pleasantness of the view or outlook of an identified receptor or group of receptors (eg residences, recreational users). Visual amenity is an important part of an area's identity and offers a wide variety of benefits to the community in terms of quality of life, wellbeing and economic activity. For some individuals, changes in visual amenity can increase levels of stress and anxiety. These impacts, however, are typically of short duration as most people adapt to changes in the visual landscape, particularly within an already urbanised area. As a result, most changes in visual impacts are not expected to have a significant impact on the health of the community.

During construction, visual amenity throughout the project area has the potential to be affected by factors such as the removal of established vegetation, the installation of construction hoardings and/or the visual appearance of construction sites. In some areas, the acoustic sheds and hoardings required to manage noise impacts during construction are large and may cause overshadowing. Further factors may include the alteration of view corridors to heritage, open space, water bodies or the city skyline.

The operational project would include changes to local visual amenity due to the presence of new and amended infrastructure (including ventilation facilities, water treatment plants, substations, bridges and drainage channels), landscaping and urban design features.

# 10.7 Equity

The health effects associated with impacts related to transport projects are not equally distributed across the community. Groups at higher risk, or more sensitive to impacts, include:

- Elderly
- Individuals with pre-existing health problems
- Infants and young children
- Individuals with disabilities
- Individuals who live in areas of higher levels of air or noise pollution.

Often the impacts can accumulate in the same areas, which may already have poorer socio-economic and health status, most commonly due to the affordability of housing in areas that are closer to main roads, industry or rail infrastructure. Disadvantaged urban areas are commonly characterised by high traffic volumes, higher levels of air and noise pollution, feelings of insecurity and lower levels of social interactions and physical activity in the community.

To further evaluate potential equity issues associated with the project, the location of impacts identified in relation to air quality, noise and traffic were reviewed individually and in combination, in conjunction with available information on the location of sensitive community groups.

It is noted that in many urban areas housing prices are lower on main roadways. The median house prices in the study area are variable, however in most areas they are consistent with the Sydney average. Some public housing is located in the study area; however, these properties are mixed in with privately owned property such that there are no specific areas with higher populations of public housing tenants. Hence there are no social equity issues identified in relation to the change in air quality in the local community. However, there is an alignment of noise and air impacts along President Avenue and Princes Highway that coincide with increased traffic volumes.

Canterbury Bankstown is the only local government areas in the study area have been identified as disadvantaged, based on the 2016 Census Data - Socio-Economic Index for Australia (SEIFA). However, it is noted that the major air and noise impacts are not located in this local government area. Therefore, the major impacts from the project are not impacting a low socioeconomic local government area.

In relation to broader equity aspects the F6 Extension, along with approved WestConnex projects (M4-M5 Link, M4 East and New M5) are aimed at improving access to the area from outer lying areas in the south and west. The SEIFA for populations in the outer south and west are lower, indicating they are more disadvantaged, than populations in the study area. Improving access and travel times for these more disadvantaged populations provides the potential for health benefits such as those that are derived from improved employment opportunities, decreased travel times (and potentially more time available for other active, family or community activities) and reduced levels of stress and anxiety.

# 10.8 Construction fatigue

Construction fatigue relates to receptors that experience construction impacts from a variety of projects over an extended period of time with few or no breaks between construction periods. Construction fatigue typically relates to traffic and access disruptions, noise and vibration, air quality, visual amenity and social impacts from projects that have overlapping construction phases or are back to back. Construction impacts on that occur in this manner are no longer considered to be transient and/or short-term.

The assessment of construction fatigue in this report includes the construction impacts of the New M5 / WestConnex project that may overlap with the timing of the construction of the F6 Extension – Stage 1 project. It is noted that construction fatigue is particularly relevant for the community surrounding the Arncliffe construction ancillary facility, a facility anticipated to be used for both New M5 and F6 Extension projects. Other potential construction fatigue risk areas identified include:

- Rockdale construction ancillary facility
- President Avenue construction ancillary facility
- Princes Highway / President Avenue intersection upgrade

The area is also subject to ongoing urban development, with many of the LGAs in the study area projected to have significant population growth (refer to section 4.4) driven by increased development density in the Arncliffe, Banksia, Rockdale and Kogarah areas, as well as the proposed Cooks Cove development.

**Appendix E** (Air Quality technical report) has not specifically addressed impacts to air from longer duration construction activities. The approach adopted evaluates risk on the basis of the type and scale of activity and potential for dust to be generated, and the location of sensitive receptors in the vicinity of these works. Hence the dust management measures identified to minimise dust impacts and health risks during construction would be need to be applied through the duration of the works, consistent with standard construction management practices. Such measures would need to then be applied across all construction projects, for major infrastructure and other construction activities (including building works) to minimise impacts in the long-term and would be subject to the requirements of approvals for those projects.

**Appendix G** (Noise and vibration technical report) has included an assessment of noise impacts that may occur where there are construction activities from a number of road or other infrastructure projects that occur consecutively (one after another) and result in exposure to construction noise impacts for a longer period of time. It identified construction noise of up to 8 years surrounding the area of the Arncliffe ventilation facility, currently being built as part of the New M5 Motorway project. However, while the current New M5 Motorway project is expected to operate 60 heavy vehicle movements an hour, the F6 Extension is expecting 26 heavy vehicle movements an hour. It has been suggested that construction fatigue be managed through discussions with the affected community and where practicable noise attenuation and respite provided.

There are other impacts associated with construction that affect the health and wellbeing of the community. This includes:

- Traffic and transport:
  - Congestion on surface roads from the movement of construction vehicles including heavy vehicles (for spoil haulage) and light vehicles (such as worker access to construction ancillary facility sites)
  - Temporary access disruption to private properties including residences and businesses
  - Partial and/or complete closure of roads, active transport links (ie pedestrian and cyclist paths, including provision of alternate links), and potential loss of street parking
  - Changes to the location of bus stops
- Visual amenity
  - Views of temporary noise barriers and construction hoarding, plant and equipment
  - Alteration of views through removal of landscaping.

Where these impacts occur for extended periods of time, there is the potential that increased levels of stress and anxiety may also continue for extended periods of time. Health effects associated with stress and anxiety are further discussed in **section 10.10**.

# **10.9 Economic aspects**

The construction expenditure of the project would be of significant benefit to the economy. This expenditure would inject economic stimulus benefits into the local, regional and state economies. Ongoing or improved economic vitality brings significant health benefit to the community. Employment opportunities would grow in the region through the potential increase in business customers and through the increase in demand for construction workers. The increase in demand for labour may increase wages in the region, particularly for construction workers, who would be in high demand.

It is noted that some local businesses will be adversely impacted by both construction and operational activities, along with other businesses marked for acquisition. This can cause stress for the impacted individuals and lead to health impacts if not appropriately managed. To minimise these impacts the project would include development of a Business Management Plan. This plan should include ways to minimise stress to impacted individuals.

Freight and commercial vehicle movements are an important component of the economy. Numerous industries are dependent upon efficient transport to service operational requirements. Transport for NSW estimated that freight and logistics contributed \$66 billion to NSW State Gross Product (GSP) in 2011, this represented 13.8 per cent of NSW GSP at the time.

An objective of the F6 Extension project is to encourage heavy and commercial vehicle movements into the tunnel, increasing efficiencies and reducing *'freight costs through increased travel speeds and reliability and reduced travel distances'*.

The transport modelling undertaken for the project highlighted that the project would result in substantial potential benefits for freight and commercial vehicle movements. Improvements in the efficiency and reliability of these transport networks would likely result in increased productivity, reduced costs and broader economic benefits for these workforces.

### 10.9.1 Road tolling

The implementation of road tolls can have direct impacts on the management of congestion, which has an impact on economic productivity, and social elements such as stress, time with family and friends, cost and environmental amenity such as reduced traffic emissions.

One impact is the potential to increase congestion volumes on surrounding roads as a result of toll avoidance. The use of a toll road can also increase the cost of living and can exacerbate social inequality. Specifically, the impact of roads tolls on households can be assessed as a function of household income, urban spatial structure, and available mobility choices. Depending on the travel routes of individuals, and the individual economic situation, there may be a proportion of the population that avoid the use of tollways due to affordability.

An evaluation of road tolling has been undertaken in Chapter 15 - Social and economic of the environmental impact statement found an overall positive impact from the toll road. However, this is undertaken on a regional scale and individual benefits would vary.

### **10.10** Stress and anxiety issues

A number of changes within the community (discussed in **sections 10.2** to **10.9**) have the potential to affect levels of stress and anxiety. Some changes may result in a lowering of feelings of stress and anxiety, and there are others that may result in higher levels within the community. In addition, construction fatigue (as discussed in **section 10.8**) from the combined road tunnel projects, other infrastructure projects and ongoing urban developments associated with urban growth, may result in elevated levels of stress and anxiety for extended periods of time.

Chronic and persistent negative stress, or distress, can lead to many adverse health problems including physical illness and mental, emotional and social problems. Response to stress will vary between individuals with genetic inheritance and personal/environmental experiences of importance (Schneiderman et al. 2005).

An acute stressful event results in changes to the nervous, cardiovascular, endocrine and immune systems, more commonly known as the "fight or flight" response (Schneiderman et al. 2005). Unless there is an accident or other significant event, such acute stress events are not expected to be associated with construction or operation of the M4-M5 Link project.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

For shorter-term events, stress causes the immune system to release hormones that trigger the production of white blood cells that fight infection and other disease-fighting elements. This response is important for fighting injuries and acute illness. However, this activity within the body is not beneficial if it occurs for a long period of time. Hormones released during extended or chronic stress can inhibit the production of cytokines (the messengers that allow cells to talk together to fight infection) lowering the body's ability to fight infections. This makes some individuals more susceptible to infections, and may also experience more severe infections. It can also trigger a flare up of pre-existing autoimmune diseases (which are a range of diseases where the immune system gets confused and starts attacking healthy cells) (Mills et al. 2008; Schneiderman et al. 2005).

Other physiological effects associated with chronic stress include (Brosschot et al. 2006; McEwen, Bruce S. 2008; McEwen, B. S. & Stellar 1993; Mills et al. 2008; Moreno-Villanueva & Bürkle 2015):

- Digestive disorders, with hormones released in response to stress causing a number of people to
  experience stomach ache or diarrhoea, with appetite also affected in some individuals (resulting
  in under-eating or over-eating).
- Chronic activation of stress hormones can raise an individual's heart rate, cause chest pain and/or heart palpitations and increase blood pressure and blood lipid (fat) levels. Sustained high levels of cholesterol and other fatty substances can lead to atherosclerosis and other cardiovascular disease and sometimes a heart attack (Pimple et al. 2015; Seldenrijk et al. 2015).
- Cortisol levels, release at higher levels with stress, play a role in the accumulation of abdominal fat, which has been linked to a range of other health conditions.
- Stress can cause muscles to contract or tighten, cause tension aches and pains (Ortego et al. 2016).

Some individuals respond to elevated levels of stress by taking up or continuing unhealthy stress coping strategies such as smoking, drinking or overeating, all of which are associated with significant health risks. Chronic levels of stress have also been found to cause or exacerbate existing mental health issues, including mood disorders such as depression and anxiety, cognitive problems, personality changes and problem behaviours. It can also affect individuals with pre-existing bipolar disorders.

By-products of stress hormones can act as sedatives (chemical substances which cause us to become calm or fatigued). When such hormone by-products occur in large amounts (which will happen under conditions of chronic stress), they may contribute to a sustained feeling of low energy or depression. Habitual patterns of thought which influence appraisal and increase the likelihood that a person will experience stress as negative (such as low self-efficacy, or a conviction that you are incapable of managing stress) can also increase the likelihood that a person will become depressed. It is normal to experience a range of moods, both high and low, in everyday life. While some "down in the dumps" feelings are a part of life, sometimes, people fall into depressing feelings that persist and start interfering with their ability to complete daily activities, hold a job, and enjoy successful interpersonal relationships (Mills et al. 2008; Schneiderman et al. 2005).

Some people who are stressed may show relatively mild outward signs of anxiety, such as fidgeting, biting their fingernails, tapping their feet, etc. In other people, chronic activation of stress hormones can contribute to severe feelings of anxiety (eg racing heartbeat, nausea, sweaty palms, etc.), feelings of helplessness and a sense of impending doom. Thought patterns that lead to stress (and depression, as described above) can also leave people vulnerable to intense anxiety feelings (Mills et al. 2008).

Anxiety or dread feelings that persist for an extended period of time; which cause people to worry excessively about upcoming situations (or potential situations); which lead to avoidance; and cause people to have difficulty coping with everyday situations may be symptoms of one or more anxiety disorders (Mills et al. 2008).

More generally, it must be noted that urbanisation, or increased urbanisation, regardless of specific projects has been found to affect levels of stress and mental health (Srivastava 2009). These impacts are greater where there is urbanisation without improvements in infrastructure to improve equitable access to employment and social areas/communities (Srivastava 2009).
The role of either acute or long-term environmental stress on the health of any community, in general and for specific project(s), including the F6 Extension project, cannot be quantified. There are a wide range of complex factors that influence health and wellbeing, specifically mental health. It is not possible to determine any specific outcomes that may occur as a result of a specific project, or number of projects. However, it is noted that within any urban environment there will be a wide range of stressors present from infrastructure projects as well as other urban developments that may or may not contribute to the health effects outlined above.

It is noted that the F6 Extension project along with the other approved WestConnex projects aim to improve infrastructure, connections and access within the urban environment. Hence on a broader scale, the longer-term projects, while requiring long-term management to minimise construction impacts, may assist in reducing stress and associated physiological and mental health impacts within the urban environment.

### 10.11 Overall assessment

Within an urban environment there are a wide range of complex factors (acting and interacting at different scales) that can affect health and wellbeing. This is conceptualised in **Figure 10-1** (presented by the International Council for Science and similar to that defined by the WHO) (ICSU 2011). The factors identified may result in either positive or negative impacts on health and wellbeing. It is noted that no single element or determinant acts in isolation. Health and wellbeing in the urban environment depends on the sum of the total interactions between many factors.

Potential impacts related to this project are summarised on the figure, showing both positive and negative impacts. The figure illustrates the complexity of making definitive conclusions in relation to health impacts in the community. However, it is noted that where negative impacts have been identified, impacts to the community are minimised through the implementation of appropriate mitigation or management measures.



Figure 10-1: Conceptual framework for determinants of health and wellbeing in the urban environment and potential impacts from project (ICSU 2011)

## 11 Uncertainties

### 11.1 General

Any assessment of health risk or health impact incorporates data and information that is associated with some level of uncertainty. In most cases, where there is uncertainty in any of the key data or inputs into an assessment of health risk or health impact, a conservative approach is adopted. This approach is adopted to ensure that the assessment presents an overestimation of potential health impacts, rather than an underestimation. It is therefore important to provide some additional information on the key areas of uncertainty for the HIA to support the conclusions presented.

### **11.2 Population health data**

There are limitations in the use of this data for the quantification of impact and risk. This data is 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 may 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 data considered in this assessment is only a general indicator (not a precise measure) of the incidence of these health endpoints.

### **11.3** Exposure concentrations and levels

The concentration of various pollutants in air (ie exposure concentrations) and noise levels relevant to different locations in the community have been calculated on the basis of a range of input assumptions and modelling. Details of these are presented within the relevant technical reports.

### 11.3.1 Traffic modelling

Assessment of impacts of the project on air and noise has relied on the modelling of traffic changes (refer to **Appendix E** (Air quality technical report)). The traffic modelling has population growth projections over the Sydney metropolitan area and relies on these predictions in its modelling.

### 11.3.2 Air quality

An assessment on the scale of the project is a complex, multi-step process which involves various different assumptions, inputs, models, and post-processing procedures. There is an inherent uncertainty in each of the methods used to estimate emissions and concentrations, and there are clearly limits to how accurately any impacts in future years can be predicted. Conservatism is built into predictions to ensure that a margin of safety is applied (ie to minimise the risk that any potential impacts are underestimated).

The operational air quality assessment for the project has been conducted, as far as possible, with the intention of providing 'accurate' or 'realistic' estimates of pollutant emissions and concentrations. The general approach has been to use inputs, models and procedures that are as accurate as possible, except where the context dictates that a degree of conservatism is sensible. An example of this is the estimation of the maximum one hour NO<sub>2</sub> concentration during a given year. Any method which provides a 'typical' or 'average' one hour NO<sub>2</sub> concentration would tend to result in an underestimate of the likely maximum concentration, and therefore a more conservative approach is required. However, the scale of the conservatism can often be quite difficult to define, and this can sometimes result in some assumptions being overly conservative. Skill and experience is required to estimate impacts that err on the side of caution but are not unreasonably exaggerated or otherwise skewed. By demonstrating that a deliberate overestimate of impacts is acceptable, it can be confidently predicted that the actual impacts that are likely to be experienced in reality would also lie within acceptable limits.

A number of conservative assumptions and approaches have been adopted in the assessment of air quality impacts, which include:

Emissions model adopted overestimate emissions and concentrations within the tunnels (by a factor of 1.7 to 3.3).

Assessment of total concentrations at receptor locations has adopted a contemporaneous approach. For the assessment of impacts it is assumed that the background concentration estimated occurs at the same time as the maximum predicted air quality impact from the project. It is unlikely that this would occur, and as a result the predicted maximum total concentration will be an overestimate. It is noted that it is not possible to know the true total (background plus project) concentration at any location.

A comparison of modelled and measured air concentrations was undertaken to evaluate the performance of the modelling approach adopted (as presented in Annexure H of Air quality technical report (ERM, 2018)). It found the modelling approach to have provided conservative estimates of exposure concentrations throughout the study area. Specifically,

- For PM<sub>10</sub> the results suggested that the use of modelling should give good (and slightly conservative) estimates of the annual PM<sub>10</sub> concentration; and
- For NOx the results suggest that the estimated total annual mean and short-term NO<sub>X</sub> concentrations ought to be quite conservative for most of the modelling domain.

### 11.3.3 Noise assessment

The noise impact assessment incorporates information on traffic volumes and composition from the traffic model and other information on the design of the F6 Extension – Stage 1 project. The modelling also incorporates measured background noise levels and a range of inputs and assumptions in relation to noise generated from the project. The model used in the assessment was validated based on existing information and traffic information and found to predict noise impacts within acceptable levels of variability, namely the difference between measured and modelled noise levels is  $\pm$  two dB(A).

### **11.4** Approach to the assessment of risk for particulates

### 11.4.1 General

The available scientific information provides a sufficient basis for determining that exposure to particulate matter (particularly  $PM_{2.5}$  and smaller) is associated with adverse health effects in a population. The data is insufficient to provide a thorough understanding of all of the potential toxic properties of particulates to which humans may be exposed. Over time it is expected that many of the current uncertainties would be refined with the collection of additional data, however some uncertainty would be inherent in any estimate. The influence of the uncertainties may be either positive or negative.

Overall, the epidemiological and toxicological data on which the assessment presented in this technical report are based on current and robust information for the assessment of risks to human health associated with the potential exposure to particulate matter from combustion sources.

### 11.4.2 Exposure-response functions

The choice of exposure-response functions for the quantification of potential health impacts is important. For mortality health endpoints, many of the exposure-mortality functions have been replicated throughout the world. While many of these have shown consistent outcomes, the calculated relative risk estimates for these studies do vary. This is illustrated by **Figure 11-1**:, **Figure 11-2**: and **Figure 11-3**: that show the variability in the relative risk estimates calculated in published studies for the US (and Canadian) population that are relevant to the primary health endpoints considered in this assessment (USEPA 2012). A similar variability is observed where additional studies from Europe, Asia and Australia/New Zealand are considered.



Figure 11-1: All-cause mortality relative risk estimates for long term exposure to PM<sub>2.5</sub> (USEPA 2012, note studies in red are those completed since 2009)

Study	Location	Age	Lag	Mean	98 <sup>th</sup>		
						All CVI	0
Metzger et al. (2004)	Atlanta, GA	All	0-2	17.8			
Tolbert et al. (2007)	Atlanta, GA	A11	0-2	17.1			
Zanobettietal (2009)*	26US Communities	65+	0-1	153			
Ito et al. (2011)	New York NV	40+	Ó	14.4			
Bell et al (2008)	202 U.S. Counties	65+	0	120	34.2		
Shughteret al (2005)	Spokane WA	A 11	1	10.8	20.6		
Slaughteret al. (2005)	Spokane, WA	All	0.1	10.6	29.0		
Kioogeral (2012)	New England	0.5+	0-1	9.0			
Killeral (2012)	Denver, CO	All	0-2 DL	8.0			
Rumattatal (1000)	Terrete CAN	A 11	0.1	19.0		IHI	)
Dumeneral (1999)	Detect MI	All	0-1	10.0			
10 (2003)	Detroit, MI	05+	1	18.0			
Metzger et al. (2004)	Atlanta, GA	All	0-3	17.8			
Dominici et al. (2000)	204 U.S. Counties	05+	0-2 DL	13.5	34.8		
Pope et al. (2006)	Utah Valley, UT	All	0	10.1-11.3			
						м	т
Zanobetti et al. (2009)*	26US Communities	65+	0-1	153			•
Sullivan et al (2005)	King County WA	21-08	24 h	128			
Betern et al. (2001)	Reston MA	61.65	24 h	12.0	202		
Zanobetti & Schwartz (2005)	Boston MA	01.0a	241	11.1**	20.2		
Stich at al (2000)*	7 Canadian aitian	A 11	0	67.0.9			
SHED ET AL (2009)	/ Canadan Chies	All	U	0.7-9.0	-		
Burnett at a1 (1000)	Toronto CAN	A11	0.2	18.0		CHI	F
Ite (2002)	Detroit MI	65.	1	18.0			
Motager et al. (2004)*	Atlanta CA	A11	0.2	17.0			
Metzger et al. (2004)	Adama, GA	All	0-2	17.0			
Symonset al. (2006)	Baltimore, MD	All	2	10.0			
Zanobettiet al (2009)*	20 U.S. Communities	00+	0-1	15.3			
Dominici et al (2006)	204 U.S. Counties	05+	0	13.3	34.8		
Haley et al. (2009)	New York State	All	0-2	11.1-15.5			
Pope et al (2008)	Utah	All	0-13 DL	10.8	44.5		
Stieb et a1 (2009)*	7 Canadian cities	All	0	6.7-9.8	-		
						CBVI	D
Metzger et al. (2004)*	Atlanta, GA	All	0-2	17.8			
Dominici et al (2006)	204 U.S. Counties	65+	0	13.3	34.8		
Kloog et al. (2012)	New England	65+	0-1	9.6			
						Hypertension	a
Szyszkowicz et al. (2012)*	Edmonton_CAN	All	0-3	8.5			
						4.0 -2.0 0.0 2.0 4.0 6.0 8.0 10.0 12.0 14.0 16.0 18.0 20.0 22.0 24.0 26.0 28.0 30.0 32.0	
						% Increase	

Figure 11-2: Per cent increase in cardiovascular-related hospital admissions for a 10 microgram per cubic metre increase in short term (24 hour average) exposure to PM<sub>2.5</sub> (USEPA 2012, note studies in red are those completed since 2009)

(Note: CVD = cardiovascular disease; IHD = ischemic heart disease; MI = myocardial infarction; CHF = congestive heart failure; CBVD = cerebrovascular disease)



Figure 11-3: Per cent increase in respiratory-related hospital admissions for a 10 micrograms per cubic metre increase in short term (24 hour average) exposure to PM<sub>2.5</sub> (USEPA 2012, note studies in red are those completed since 2009)

These figures illustrate the 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 particulate matter to which the population is exposed.

Based on the available data, and the detailed reviews undertaken by organisations such as the USEPA (USEPA 2010, 2012) and WHO (WHO 2003, 2006b, 2006a) and discussions with NSW Health, the adopted exposure-response estimates are considered to be current, robust and relevant to the characterisation of impacts from  $PM_{2.5}$ .

### **11.4.3** Shape of exposure-response function

The shape of the exposure-response function and whether there is a threshold for some of the effects endpoints remains an uncertainty. Reviews of the currently available data (that includes studies that show effects at low concentrations) have not shown evidence of a threshold. However, as these conclusions are based on epidemiological studies, discerning the characteristics of the particulates responsible for these effects and the observed shape of the dose-response relationship is complex. For example, it is not possible to determine if the observed no threshold response is relevant to exposure to particulates from all sources, or whether it relates to particulates from combustion sources only.

Most studies have demonstrated that there is a linear relationship between relative risk and ambient concentration however for long term exposure-related mortality a log-linear relationship is more plausible and should be considered where there is the potential for exposure to very high concentrations of pollution. In this assessment, the impact considered is a localised impact with low level incremental increases in concentration. At low levels, the assumption of a linear relationship is considered appropriate.

### **11.5 Diesel particulate matter evaluation**

The assessment of exposure to diesel particulate matter has assumed that 100 per cent of the  $PM_{2.5}$  associated with the project is derived from diesel sources. This is a conservative assumption.

The health hazard conclusions associated with exposure to diesel particulate matter are based on studies that are dominated by exhaust emissions from diesel engines built prior to the mid-1990s. With current engine use including some new and many older engines (engines typically stay in service for a long time), the health hazard conclusions, in general, are likely to be applicable to engines currently in use.

However as new and cleaner diesel engines, together with different diesel fuels, replace a substantial number of existing engines; the general applicability of the health hazard conclusions may require further evaluation. The NEPC (NEPC 2009) has established a program to reduce diesel emissions from the Australian heavy vehicle fleet. This is expected to lower the potential for all diesel emissions over time

### 11.6 **Co-pollutants**

For the assessment of nitrogen dioxide, particulates and noise, the exposure-response relationships used in this assessment are based on large epidemiology studies where exposures have occurred in urban areas. These exposures do not relate to only one pollutant or exposure (noise) but a mix of these, and others including occupational and smoking. While many of the studies have endeavoured to correct for other pollutants and exposures, no study can fully correct for these and there would always be some level of influence from other exposures on the relationships adopted.

In relation to air quality, many of the pollutants evaluated come from a common source (eg fuel combustion) so the use of only particulate matter (or nitrogen dioxide) as an index for the mix of pollutants that is in urban air at the time of exposure is reasonable but conservative.

In relation to the assessment of cardiovascular effects from road traffic noise, these effects are also associated with (and occur together with) increased exposures to vehicle emissions, specifically particulate exposures.

For this reason, it is important the health risks and incidence evaluations presented for exposure to nitrogen dioxide, particulates and noise should not be added together as these effects are not necessarily additive as the relationships already include co-exposures to all these aspects (and others).

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

### **11.7** Selected health outcomes

The assessment of risk has utilised exposure-response functions and relative risk values that relate to the more significant health endpoints where the most significant and robust positive associations have been identified. The approach does not include all possible subsets of effects that have been considered in various published studies. However, the assessment undertaken has considered the health endpoints/outcomes that incorporate many of the subsets, and has utilised the most current and robust relationships.

### **11.8 Exposure time/duration**

The assessment of potential exposure and risk to changes in air quality and noise levels associated with the project has assumed that all areas evaluated are residential and people may be at home for 24 hours of the day for 365 days of the year, for a lifetime. This is a conservative assumption to ensure that all members of the public are adequately addressed in the assessment of health impacts, including the elderly and those with disabilities who may not leave the home very often. As a result, the quantification of risk and health incidence is expected to be an overestimation.

### 11.9 Changing population size and demographics

The assessment presented has utilised information on the size of the population and distribution of the population in relevant ages from the ABS Census data from 2016. As discussed in **section 3** the population in the study area is projected to increase significantly by 2036. In addition, many of the LGAs are expecting a significant increase in the proportion of the population aged 65 years and over.

The increase in population size and distribution does not affect the calculation of an individual risk. The key aspect that does affect this calculation is the baseline incidence of the health effects within the population. Based on statistics from NSW Health the baseline incidence of the health effects evaluated in this assessment have been relatively stable or decreasing over time (with improvements in health care). Hence changes in the population over time are not expected to result in any increase in the calculated individual risk.

For the calculation of the change in incidence in the community the size and distribution of the population is important. However, as the project is associated with an overall improvement (ie decrease in incidence) in the health endpoints evaluated, and increase in population would not change this outcome.

It is noted that population growth has been included in the forecast of traffic volumes predicted for the project and hence these changes have, by default, be incorporated into all subsequent impact assessment, including assessments associated with changes in air quality, noise and vibration.

## 11.10 Application of exposure-response functions to small populations

The exposure-response functions have been developed on the basis of epidemiological studies from large urban populations where associations have been determined between health effects (health endpoints) and changes in ambient (regional) particulate levels. Typically, these exposure-response functions are applied to large populations for the purpose of establishing/reviewing air guidelines or reviewing potential impacts of regional air quality issues on large populations. When applied to small populations (less than larger urban centres such as the whole of Greater Sydney) the uncertainty increases.

In addition, it is noted that the exposure-response functions relate changes in health endpoints with changes in regional air quality measurements. They do not relate to specific local sources (which occur within a regional airshed), or daily variability in exposure that may occur as a result of various different activities that may occur in any one day.

### **11.11 Overall evaluation of uncertainty**

Overall the assessment of health impacts presented in this report has incorporated a range of assumptions and models that will have resulted in an overestimation of impacts. The most significant factors that result in the assessment providing conservative outcomes are as follows:

- Modelling of air quality impacts this has included a range of conservative assumptions about the type of vehicles and the emissions to air that may come from these vehicles over time. The assessment has also utilised a model to predict ground level concentrations (i.e. concentrations in the community) that are expected to be conservative. Overall the air modelling may have overestimated air concentrations in the community by a factor of 2 fold.
- Community exposures there are a number of assumptions adopted in the characterisation of exposure that will have overestimated exposure:
  - It is assumed that the maximum changes in air quality, regardless of where this may occur (e.g. industrial area, in a roadway, open space area or residential area), affects a resident.
  - All exposures to changes in air quality and noise that occur, in all areas, assume that all residents are at home all day, every day for a lifetime.
- Exposure-response the relationships utilised in this assessment are based on the most current, robust studies that are relate to health effects from exposure to changes in nitrogen dioxide and particulates. The relationships adopted come from large epidemiology studies that include a number of co-pollutants (i.e. exposure occurs to a wide range of factors not just the pollutant being evaluated) and confounding factors that can result in more conservative relationships being developed. In addition, it is assumed the relationships adopted are linear and apply to small changes in air quality, at levels that would not be measurable with air monitoring equipment.

## 12 References

ACTAQ 2016, *In-Tunnel Air Quality (Nitrogen Dioxide) Policy*, NSW Advisory Committee on Tunnel Air Quality.

AMOG 2012, M5 East Tunnel Filtration Trial Evaluation Program - Review of Operational Performance, Independent Review Role M5 East Air Filtration Project, prepared for Roads and Maritime Services.

Anderson, CH, Atkinson, RW, Peacock, JL, Marston, L & Konstantinou, K 2004, *Meta-analysis of time-series studies and panel studies of Particulate Matter (PM) and Ozone (O3), Report of a WHO task group*, World Health Organisation.

ATSDR 2007, *Toxicological Profile for Xylene*, US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry.

<http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=296&tid=53>.

Attfield, MD, Schleiff, PL, Lubin, JH, Blair, A, Stewart, PA, Vermeulen, R, Coble, JB & Silverman, DT 2012, 'The Diesel Exhaust in Miners study: a cohort mortality study with emphasis on lung cancer', *Journal of the National Cancer Institute*, vol. 104, no. 11, Jun 6, pp. 869-883.

Babisch, W 2002, 'The Noise/Stress Concept, Risk Assessment and Research Needs', *Noise Health,* vol. 4, no. 16, pp. 1-11.

Babisch, W 2006, 'Transportation noise and cardiovascular risk: updated review and synthesis of epidemiological studies indicate that the evidence has increased', *Noise Health*, vol. 8, no. 30, Jan-Mar, pp. 1-29.

Babisch, W 2008, 'Road traffic noise and cardiovascular risk', *Noise Health,* vol. 10, no. 38, Jan-Mar, pp. 27-33.

Babisch, W 2014, 'Updated exposure-response relationship between road traffic noise and coronary heart diseases: A meta-analysis', *Noise and Health*, vol. 16, no. 68, January 1, 2014, pp. 1-9.

Bell, ML, Ebisu, K, Peng, RD, Walker, J, Samet, JM, Zeger, SL & Dominici, F 2008, 'Seasonal and Regional Short-term Effects of Fine Particles on Hospital Admissions in 202 US Counties, 1999–2005', *American Journal of Epidemiology*, vol. 168, no. 11, December 1, 2008, pp. 1301-1310.

Bell, ML 2012, 'Assessment of the health impacts of particulate matter characteristics', *Research report*, no. 161, Jan, pp. 5-38.

Brosschot, JF, Gerin, W & Thayer, JF 2006, 'The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health', *Journal of Psychosomatic Research*, vol. 60, no. 2, 2006/02/01/, pp. 113-124.

Burgers, M & Walsh, S 2002, *Exposure Assessment and Risk Characterisation for the Development of a PM2.5 Standard*, NEPC.

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

COMEAP 2015, Statement on the Evidence for the Effects of Notrogen Dioxide on Health, Committee on the Medical Effects of Air Pollutants.

de Vries, S, Verheij, RA, Groenewegen, PP & Spreeuwenberg, P 2003, 'Natural Environments— Healthy Environments? An Exploratory Analysis of the Relationship between Greenspace and Health', *Environment and Planning A*, vol. 35, no. 10, October 1, 2003, pp. 1717-1731.

DEC 2005, Approved Methods for the Modelling and Assessment of Air Pollutants in NSW, NSW Department of Environment and Conservation.

DEFRA 2014, *Environmental Noise: Valuing impacts on: sleep disturbance, annoyance, hypertension, productivity and quiet*, UK Department of Environment, Food & Rural Affairs.

DEH 2003, *Technical Report No. 1: Toxic Emissions from Diesel Vehicles in Australia*, Environment Australia.

DIN 1999, Structural Vibration - Effects of vibration on structures. DIN 4150-3, German Institute for Standardisation.

EC 2002, Position paper on dose response relationships between transportation noise and annoyance, Office for Official Publications of the European Communities, Luxembourg.

EC 2004, *Position Paper on Dose-Effect Relationships for Night Time Noise*, European Commission Working Group on Health and Socio-Economic Aspects.

EC 2011, *Final report on risk functions used in the case studies*, Health and Environment Integrated Methodology and Toolbox for Scenario Development (HEIMTSA).

EEA 2010, Good practice guide on noise exposure and potential health effects, EEA Technical report No 11/2010, European Environment Agency, Copenhagen.

EEA 2014, Noise in Europe 2014, EEA Report No 10/2014, European Environment Agency, Luxembourg.

enHealth 2001, *Health Impact Assessment Guidelines*, Commonwealth Department of Health and Aged Care.

enHealth 2004, *The health effects of environmental noise – other than hearing loss*, enHealth Council, Department of Health and Ageing.

enHealth 2012a, *Australian Exposure Factors Guide*, Commonwealth of Australia, Canberra. <<u>http://www.health.gov.au/internet/main/publishing.nsf/Content/health-publicat-environ.htm</u>>.

enHealth 2012b, Environmental Health Risk Assessment, Guidelines for assessing human health risks from environmental hazards, Commonwealth of Australia, Canberra. <<u>http://www.health.gov.au/internet/main/publishing.nsf/content/804F8795BABFB1C7CA256F1900045</u> 479/\$File/DoHA-EHRA-120910.pdf >.

enRiskS 2017, Review of In-Cabin Carbon Dioxide Levels, Report prepared for NSW RMS.

EnRiskS 2018, Literature Review and Risk Characterisation of Nitrogen Dioxide - Long and Heavily Trafficked Road Tunnels A Report prepared for the NSW Raods and Maritime Services

EPA 2012, Air Emissions Inventory for the Greater Metropolitan Region in New South Wales, 2008 Calendar Year, On-Road Mobile Emissions:Results, NSW Environment Protection Authority, Sydney.

EPA 2013, *Methodology for Valuing the Health Impacts of Changes in Particle Emissions*, Prepared by PAEHolmes on behalf of NSW Environment Protection Authority.

EPHC 2010, *Expansion of the multi-city mortality and morbidity study, Final Report*, Environment Protection and Heritage Council.

ETC 2013, Assessment of population exposure to air pollution during commuting in European cities, ETC/ACM Technical Paper 2013/2, European Topic Centre on Air Pollution and Climate Change Mitigation.

Fewtrell, L & Bartram, J 2001, *Water quality: Guidelines, standards and health, Assessment of risk and risk management for water-related infectious disease,* WHO. <<u>http://www.who.int/water sanitation health/dwq/whoiwa/en/</u>>.

Gidlöf-Gunnarsson, A & Öhrström, E 2007, 'Noise and well-being in urban residential environments: The potential role of perceived availability to nearby green areas', *Landscape and Urban Planning*, vol. 83, no. 2–3, pp. 115-126.

Golder 2013, Exposure Assessment and Risk Characterisation to Inform Recommendations for Updating Ambient Air Quality Standards for PM2.5, PMN10, O3, NO2, SO2, Golder Associates for National Environment Protection Council Service Corporation.

Halonen, JI, Hansell, AL, Gulliver, J, Morley, D, Blangiardo, M, Fecht, D, Toledano, MB, Beevers, SD, Anderson, HR, Kelly, FJ & Tonne, C 2015, 'Road traffic noise is associated with increased cardiovascular morbidity and mortality and all-cause mortality in London', *European Heart Journal*, vol. 36, no. 39, 2015-10-14 00:00:00, pp. 2653-2661.

Hansson, E, Mattisson, K, Björk, J, Östergren, P-O & Jakobsson, K 2011, 'Relationship between commuting and health outcomes in a cross-sectional population survey in southern Sweden', *BMC Public Health*, vol. 11, no. 1, p. 834.

Harris, P, Harris-Roxas, B., Harris, E. & Kemp, L. 2007, *Health Impact Assessment: A Practical Guide*, Centre for Health Equity Training, Research and Evaluation (CHETRE). Part of the UNSW Research Centre for Primary Health Care and Equity. University of New South Wales.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

Health Scotland 2008, *Health Impact Assessment of greenspacen, A Guide*, Health Scotland, greenspace scotland, Scottish Natural Heritage and Institute of Occupational Medicine.

HEI 2013, Understanding the Health Effects of Ambient Ultrafine Particles, HEI Review Panel on Ultrafine Particles, HEI Perspectives 3, Health Effects Institute, Boston.

Higson, DJ 1989, Risks to Individuals in NSW and in Australia as a Whole, Nuclear Science Bureau,

Hoffman, HJ 1988, *Survey of risks : Memorandum to the docket, Memorandum to the docket: OAQPS 79-3, Part 1*, EPA, Washington D.C.

Houthuijs, DJM, van Beek, AJ, Swart, WJR & van Kempen, EEMM 2014, *Health implication of road, railway and aircraft noise in the European Union, Provisional results based on the 2nd round of noise mapping, RIVM Report 2014-0130*, National Institute for Public Health and the Environment.

I-INCE 2011, *Guidelines for Community Noise Impact Assessment and Mitigation, I-INCE Publication Number: 11-1*, International Institute of Noise Control Engineering (I-INCE) Technical Study Group on Community Noise: Environmental Noise Impact Assessment and Mitigation.

IARC 2012, IARC: Diesel Engine Exhaust Carcinogenic, World Health Organisation.

ICSU 2011, Report of the ICSU Planning Group on Health and Wellbeing in the Changing Urban Environment: a Systems Analysis Approach, International Council for Science, Paris.

Jalaludin, B, Khalaj, B, Sheppeard, V & Morgan, G 2008, 'Air pollution and ED visits for asthma in Australian children: a case-crossover analysis', *Int Arch Occup Environ Health,* vol. 81, no. 8, Aug, pp. 967-974.

Jalaludin, B 2015, *Review of experimental studies of exposures to nitrogen dioxide*, Centre for Air quality and health Research and evaluation, Woolcock Institute of Medical Research.

Kelly, KE 1991, 'The Myth of 10-6 as a Definition of Acceptable Risk', 84th Annual Meeting, Air & Waste Management Association, Air & Waste Management Association.

Kendal, D, Lee, K, Ramalho, C, Bower, K & Bush, J 2016, *Benefits of Urban Green Space in the Australian Context*, Clean Air and Urban Landscapes Hub, National Environmental Science Programme.

Knibbs, LD, de Dear, RJ & Atkinson, SE 2009, 'Field study of air change and flow rate in six automobiles', *Indoor Air,* vol. 19, no. 4, Aug, pp. 303-313.

Knibbs, LD, de Dear, RJ & Morawska, L 2010, 'Effect of cabin ventilation rate on ultrafine particle exposure inside automobiles', *Environmental science & technology*, vol. 44, no. 9, May 1, pp. 3546-3551.

Krewski, D, Jerrett, M, Burnett, RT, Ma, R, Hughes, E, Shi, Y, Turner, MC, Pope, CA, 3rd, Thurston, G, Calle, EE, Thun, MJ, Beckerman, B, DeLuca, P, Finkelstein, N, Ito, K, Moore, DK, Newbold, KB, Ramsay, T, Ross, Z, Shin, H & Tempalski, B 2009, 'Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality', *Research report*, no. 140, May, pp. 5-114; discussion 115-136.

Lee, ACK & Maheswaran, R 2011, 'The health benefits of urban green spaces: a review of the evidence', *Journal of Public Health*, vol. 33, no. 2, June 1, 2011, pp. 212-222.

Lindström, M 2008, 'Means of transportation to work and overweight and obesity: A population-based study in southern Sweden', *Prev Med*, vol. 46.

Longley, I 2014, *TP11: Criteria for In-Tunnel and Ambient Air Quality*, NSW Advisory Committee on Tunnel Air Quality.

Maas, J, Verheij, RA, Groenewegen, PP, de Vries, S & Spreeuwenberg, P 2006, 'Green space, urbanity, and health: how strong is the relation?', *J Epidemiol Community Health*, vol. 60.

Martuzzi, M, Galasso, C, Ostro, B, Forastiere, F & Bertollini, R 2002, *Health Impact Assessment of Air Pollution in the Eight Major Italian Cities*, World Health Organisation, Europe.

McEwen, BS & Stellar, E 1993, 'Stress and the individual: Mechanisms leading to disease', *Archives of Internal Medicine*, vol. 153, no. 18, pp. 2093-2101.

McEwen, BS 2008, 'Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators', *European Journal of Pharmacology*, vol. 583, no. 2, 2008/04/07/, pp. 174-185.

Mills, H, Reiss, N & Dombeck, M 2008, *Stress Reduction and Management*, Mental Help, <<u>https://www.mentalhelp.net/articles/introduction-and-the-nature-of-stress/</u>>.

Mitchell, R & Popham, F 2007, 'Greenspace, urbanity and health: relationships in England', *Journal of Epidemiology and Community Health*, vol. 61, no. 8, August 1, 2007, pp. 681-683.

Morawska, L, Moore, MR & Ristovski, ZD 2004, *Health Impacts of Ultrafine Particles, Desktop Literature Review and Analysis*, Australian Government, Department of the Environment and Heritage.

Moreno-Villanueva, M & Bürkle, A 2015, 'Molecular consequences of psychological stress in human aging', *Experimental Gerontology*, vol. 68, 2015/08/01/, pp. 39-42.

Morgan, G, Broom, R & Jalaludin, B 2013, *Summary for Policy Makers of the Health Risk Assessment on Air Pollution in Australia*, Prepared for National Environment Protection Council by the University Centre for Rural Health, North Coast, Education Research Workforce, A collaboration between The University of Sydney, Southern Cross University, The University of Western Sydney, The University of Wollongong, Canberra.

NEPC 1998, National Environment Protection (Ambient Air Quality) Measure - Revised Impact Statement, National Environment Protection Council.

NEPC 1999 amended 2013a, National Environment Protection (Assessment of Site Contamination) Measure Schedule B8 Guideline on Community Engagement and Risk Communication, National Environment Protection Council,

NEPC 1999 amended 2013b, Schedule B1, Guideline on Investigation Levels For Soil and Groundwater, National Environment Protection (Assessment of Site Contamination) Measure, National Environment Protection Council. <<u>http://scew.gov.au/nepms/assessment-site-contamination</u>>.

NEPC 2002, National Environment Protection (Ambient Air Quality) Measure, Impact Statement for PM2.5 Variation Setting a PM2.5 Standard in Australia, National Environment Protection Council.

NEPC 2003, National Environment Protection (Ambient Air Quality) Measure, National Environment Protection Council.

NEPC 2009, National Environment Protection (Diesel Vehicle Emissions) Measure, NEPC Service Corporation.

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

NEPC 2014, Draft Variation to the National Environment, protection (Ambient Air Quality) Measure, Impact Statement, National Environment Protection Council.

NEPC 2016, National Environment Protection (Ambient Air Quality) Measure, Federal Register of Legislative Instruments F2016C00215.

NHMRC 2008, *Air Quality in and Around Traffic Tunnels, Systematic Literature Review*, National Health and Medical Research Council.

NSW DEC 2005, Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales, Department of Environment and Conservation NSW (DEC),

NSW DEC 2006, Assessing vibration: a technical guideline, NSW Department of Environment and Conservation. <<u>http://epa.nsw.gov.au/noise/vibrationguide.htm</u>>.

NSW DECC 2009, *Interim Construction Noise Guideline*, NSW Department of Environment and Climate Change. <<u>www.environment.nsw.gov.au/resources/stormwater/0801soilsconststorm2a.pdf</u>>.

NSW DECCW 2010, Current air quality in New South Wales, A technical paper supporting the Clean Air Forum 2010, Sydney.

NSW DECCW 2011, *NSW Road Noise Policy*, NSW Department of Environment, Climate Change and Water, Sydney.

NSW EPA 2000, *NSW Industrial Noise Policy*, NSW Environment Protection Authority. <<u>http://epa.nsw.gov.au/noise/industrial.htm</u>>.

NSW EPA 2016, Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales, State of NSW and Environment Protection Authority, Sydney.

NSW EPA 2017, Noise Policy for Industry, NSW Environment Protection Authority,

NSW Health 2003, *M5 East Tunnels Air Quality Monitoring Project*, South Eastern Sudney Public Health Unit & NSW Department of Health.

NSW Health 2004, Comparison of personal exposures to air pollutants by commuting mode in Sydney, BTEX & NO<sub>2</sub>, NSW Department of Health, Sydney.

NSW Health 2016, Building Better Health, Health considerations for urban development and renewal in the Sydney Local Health District, NSW Health, Sydney Local Health District.

NSW OEH 2015, *New South Wales Air Quality Statement 2014*, NSW and Office of Environment and Heritage, Sydney.

NSW Planning 2011, Risk Criteria for Land Use Safety Planning, Hazardous Industry Planning Advisory Paper No 4, Sydney.

NSW Planning & Environment 2016, *Population projections, 2016 NSW population and household projections*. <<u>http://www.planning.nsw.gov.au/projections</u>>.

NSW Roads and Maritime 2015, *Noise Criteria Guideline*, NSW Roads and Maritime Services. <<u>http://www.rms.nsw.gov.au/documents/about/environment/noise-criteria-guideline-book.pdf</u>>.

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

OEHHA 2002, Staff Report: Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates, Office of Environmental Health Hazard Assessment.

OEHHA 2013, *Individual Acute, 8-hour, and Chronic Reference Exposure Level Summaries*, California Office of Environmental Health Hazard Assessment.

Ortego, G, Villafañe, JH, Doménech-García, V, Berjano, P, Bertozzi, L & Herrero, P 2016, 'Is there a relationship between psychological stress or anxiety and chronic nonspecific neck-arm pain in adults? A systematic review and meta-analysis', *Journal of Psychosomatic Research*, vol. 90, 2016/11/01/, pp. 70-81.

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

Ostro, B, Broadwin, R, Green, S, Feng, WY & Lipsett, M 2006, 'Fine particulate air pollution and mortality in nine California counties: results from CALFINE', *Environmental health perspectives*, vol. 114, no. 1, Jan, pp. 29-33.

PEL 2016, Road tunnels: reductions in nitrogen dioxide concentrations in-cabin using vehicle ventilation systems, Prepared by Pacific Environment Limited for NSW Roads and Maritime Services.

Pimple, P, Shah, AJ, Rooks, C, Douglas Bremner, J, Nye, J, Ibeanu, I, Raggi, P & Vaccarino, V 2015, 'Angina and mental stress-induced myocardial ischemia', *Journal of Psychosomatic Research*, vol. 78, no. 5, 2015/05/01/, pp. 433-437.

Pope, CA, 3rd, Burnett, RT, Thun, MJ, Calle, EE, Krewski, D, Ito, K & Thurston, GD 2002, 'Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution', *JAMA : the journal of the American Medical Association*, vol. 287, no. 9, Mar 6, pp. 1132-1141.

Schneiderman, N, Ironson, G & Siegel, SD 2005, 'STRESS AND HEALTH: Psychological, Behavioral, and Biological Determinants', *Annual review of clinical psychology*, vol. 1, pp. 607-628.

Schoeny, R 2008, 'Acceptable Risk Levels at EPA', in BoR U.S Department of the Interior (ed), *Workshop* on *Tolerable Risk Evaluation*. <<u>http://www.usbr.gov/ssle/damsafety/jointventures/tolerablerisk/07Schoeny.pdf</u>>.

Schomer, PD 2005, 'Criteria for assessment of noise annoyance', *Noise Control Engineering Journal*, vol. 53, no. 4, //, pp. 125-137.

Seldenrijk, A, Vogelzangs, N, Batelaan, NM, Wieman, I, van Schaik, DJF & Penninx, BJWH 2015, 'Depression, anxiety and 6-year risk of cardiovascular disease', *Journal of Psychosomatic Research*, vol. 78, no. 2, 2015/02/01/, pp. 123-129.

Silverman, DT, Samanic, CM, Lubin, JH, Blair, AE, Stewart, PA, Vermeulen, R, Coble, JB, Rothman, N, Schleiff, PL, Travis, WD, Ziegler, RG, Wacholder, S & Attfield, MD 2012, 'The Diesel Exhaust in Miners study: a nested case-control study of lung cancer and diesel exhaust', *Journal of the National Cancer Institute*, vol. 104, no. 11, Jun 6, pp. 855-868.

Sjoberg, K, Haeger-Eugensson, M, Forsberg, B, Astrom, S, Hellsten, S, Larsson, K, Bjork, A & Blomgren, H 2009, *Quantification of population exposure to PM2.5 and PM10 in Sweden 2005*, Swedish Environmental Research Institute.

Srivastava, K 2009, 'Urbanization and mental health', *Industrial Psychiatry Journal*, vol. 18, no. 2, Jul-Dec, pp. 75-76.

Stansfeld, S, Berglund, B, Clark, C, Lopez-Barrio, I, Fischer, P, Ohrstrom, E, Haines, MM, Head, J, Hygge, S, van Kamp, I & Berry, BF 2005a, 'Aircraft and road traffic noise and children's cognition and health: a cross-national study', *Lancet*, vol. 365, no. 9475, Jun 4-10, pp. 1942-1949.

Stansfeld, S, Berglund, B, Ohstrom, E, Lebert, E & Lopez Barrio, I 2005b, *Executive Summary. Road traffic and aircraft noise exposure and children's cognition and health: exposure-effect relationships and combined effects*, European Network on Noise and Health. <<u>https://ec.europa.eu/research/quality-of-life/ka4/pdf/report\_ranch\_en.pdf; www.ennah.eu</u>>.

TCEQ 2007, 1,3-Butadiene, TEXAS COMMISSION ON ENVIRONMENTAL QUALITY.

TCEQ 2013a, *Development Support Document, Formaldehyde*, Texas Commission on Environmental Quality.

TCEQ 2013b, *Development Support Document, Xylenes*, Texas Commission on Environmental Quality. <<u>https://www.tceq.texas.gov/toxicology/dsd/final.html</u>>.

TCEQ 2013c, *Development Support Document, Toluene*, Texas Commission on Environmental Quality. <<u>https://www.tceq.texas.gov/toxicology/dsd/final.html</u>>.

TCEQ 2013d, *Development Support Document, Benzene*, Texas Commission on Environmental Quality. <<u>https://www.tceq.texas.gov/toxicology/dsd/final.html</u>>.

TCEQ 2013e, *1,3-Butadiene, Development Support Document*, Texas Commission on Environmental Quality.

TCEQ 2014, Formaldehyde, 24-hour Ambient Air Monitoring Comparison Value, Development Support Document, Texas Commission on Environmental Quality.

USEPA 1998, Toxicological Review of Naphthalene (CAS No. 91-20-3), In Support of Summary Information on the Integrated Risk Information System (IRIS), U.S. Environmental Protection Agency, Washington.

USEPA 2002a, Toxicological Review of Benzene (Noncancer Effects) (CAS NO. 1330-20-7), In Support of Summary Information on the Integrated Risk Information System (IRIS), U.S. Environmental Protection Agency.

USEPA 2002b, *Health Assessment Document For Diesel Engine Exhaust*, United States Environmental Protection Agency.

USEPA 2005a, Toxicological Review of Toluene (CAS No. 108-88-3), In Support of Summary Information on the Integrated Risk Information System (IRIS), U.S. Environmental Protection Agency, Washington.

USEPA 2005b, *Particulate Matter Health Risk Assessment For Selected Urban Areas*, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards.

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

USEPA 2009b, *Integrated Science Assessment for Particulate Matter*, United States Environmental Protection Agency. <<u>http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546#Download</u>>.

USEPA 2010, Toxicological Review of Carbon Tetrachloride. In Support of Summary Information on the Integrated Risk Information System (IRIS), US Environmental Protection Agency. <a href="http://epa.gov/iris/>http://epa.gov/iris/">http://epa.gov/iris/</a>>.

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, U.S. Environmental Protection Agency.

USEPA 2015, Integrated Science Assessment for Oxides of Nitrogen–Health Criteria, Second External Review Draft, National Center for Environmental Assessment-RTP Division, Office of Research and Development, U.S. Environmental Protection Agency.

USEPA IRIS, Integrated Risk Information System (IRIS), United States Environmental Protection Agency. <<u>http://www.epa.gov/iris/</u>>.

van Kempen, E & Babisch, W 2012, 'The quantitative relationship between road traffic noise and hypertension: a meta-analysis', *J Hypertens*, vol. 30, no. 6, Jun, pp. 1075-1086.

Vienneau, D, Schindler, C, Perez, L, Probst-Hensch, N & Roosli, M 2015, 'The relationship between transportation noise exposure and ischemic heart disease: a meta-analysis', *Environ Res,* vol. 138, Apr, pp. 372-380.

Wen, LM & Rissel, C 2008, 'Inverse associations between cycling to work, public transport, and overweight and obesity: findings from a population based study in Australia', *Prev Med*, vol. 46, no. 1, Jan, pp. 29-32.

WHO 1996, *Diesel Fuel and Exhaust Emissions*, Environmental Health Criteria 171, World Health Organisation.

WHO 1999, Guidelines for Community Noise, World Health Organisation, Geneva.

WHO 2000a, WHO air quality guidelines for Europe, 2nd edition, 2000 (CD ROM version), World Health Organisation.

WHO 2000b, *Transport, environment and health*, WHO Regional Publications, European Series, No. 89.

WHO 2000c, Guidelines for Air Quality, World Health Organisation, Geneva.

WHO 2000d, *Air Quality Guidelines for Europe, Second Edition*, Copenhagen. <<u>http://www.euro.who.int/en/publications/abstracts/air-quality-guidelines-for-europe</u>>.

WHO 2003, Health Aspects of Air Pollution with Particulate Matter, Ozone and Nitrogen Dioxide, Report on a WHO Working Group, World Health Organisation.

WHO 2005, WHO air quality guidelines global update 2005, Report on a Working Group meeting, Bonn, Germany, 18-20 October 2005, World Health Organisation.

WHO 2006a, *Health risks or particulate matter from long-range transboundary air pollution*, World Health Organisation Regional Office for Europe.

WHO 2006b, WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide, Global Update, Summary of risk assessment, World Health Organisation.

WHO 2009, Night Noise Guidelines for Europe, World Health Organisation Regional Office for Europe.

WHO 2010, WHO Guidelines for Indoor Air Quality, Selected Pollutants, WHO Regional Office for Europe.

WHO 2011, Burden of disease from environmental noise, Quantification of healthy life years lost in *Europe*, World Health Organisation and JRC European Commission.

WHO 2013a, *Review of evidence on health aspects of air pollution - REVIHAAP Project, Technical Report*, World Health Organization, Regional Office for Europe.

WHO 2013b, Health Effects of Particulate Matter, Policy implications for countries in eastern Europe, Caucasus and central Asia, WHO Regional Office for Europe.

Zanobetti, A & Schwartz, J 2009, 'The effect of fine and coarse particulate air pollution on mortality: a national analysis', *Environmental health perspectives*, vol. 117, no. 6, Jun, pp. 898-903.

# Annexure A – Approach to Risk Assessment using exposure response relationships

### Mortality and morbidity health endpoints

A quantitative assessment of risk for these endpoints uses a mathematical relationship between an exposure concentration (ie concentration in air) and a response (namely a health effect). This relationship is termed an exposure-response relationship and is relevant to the range of health effects (or endpoints) identified as relevant (to the nature of the emissions assessed) and robust (as identified in the main document). An exposure-response relationship can have a threshold, where there is a safe level of exposure, below which there are no adverse effects; or the relationship can have no threshold (and is regarded as linear) where there is some potential for adverse effects at any level of exposure.

In relation to the health effects associated with exposure to nitrogen dioxide and particulate matter, no threshold has been identified. Non-threshold exposure-response relationships have been identified for the health endpoints considered in this assessment.

The assessment of potential risks associated with exposure to particulate matter involves the calculation of a relative risk (RR). For the purpose of this assessment the shape of the exposure-response function used to calculate the relative risk is assumed to be linear<sup>14</sup>. The calculation of a relative risk based on the change in relative risk exposure concentration from baseline/existing (ie based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

### Equation 1 RR = $exp[\beta(X-X0)]$

Where:

X-X0 = the change in particulate matter concentration to which the population is exposed  $(\mu g/m^3)$ 

 $\beta$  = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 µg/m<sup>3</sup> increase in particulate matter exposure.

Based on this equation, where the published studies have derived relative risk values that are associated with a 10 micrograms per cubic metre increase in exposure, the  $\beta$  coefficient can be calculated using the following equation:

 $\beta = \frac{\ln(RR)}{10}$ 

Where:

Equation 2

RR = relative risk for the relevant health endpoint as published ( $\mu g/m^3$ ) 10 = increase in particulate matter concentration associated with the RR (where the RR is

associated with a 10  $\mu$ g/m<sup>3</sup> increase in concentration).

<sup>14</sup> Some reviews have identified that a log-linear exposure-response function may be more relevant for some of the health endpoints considered in this assessment. Review of outcomes where a log-linear exposure-response function has been adopted (Ostro 2004) for PM<sub>2.5</sub> identified that the log-linear relationship calculated slightly higher relative risks compared with the linear relationship within the range 10–30 micrograms per cubic metre, (relevant for evaluating potential impacts associated with air quality goals or guidelines) but lower relative risks below and above this range. For this assessment (where impacts from a particular project are being evaluated) the impacts assessed relate to concentrations of PM<sub>2.5</sub> that are well below 10 micrograms per cubic metre and hence use of the linear relationship is expected to provide a more conservative estimate of relative risk.

### **Quantification of impact and risk**

The assessment of health impacts for a particular population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004)<sup>15</sup> where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

The calculation of changes in health endpoints associated with exposure to nitrogen dioxide and particulate matter as outlined by the WHO (Ostro 2004) has considered the following four elements:

- Estimates of the changes in particulate matter exposure levels (ie incremental impacts) due to the project for the relevant modelled scenarios
- Estimates of the number of people exposed to particulate matter at a given location
- Baseline incidence of the key health endpoints that are relevant to the population exposed
- Exposure-response relationships expressed as a percentage change in health endpoint per microgram per cubic metre change in NO<sub>2</sub> or particulate matter exposure, where a relative risk (RR) is determined (refer to Equation 1).

From the above, the increased incidence of a health endpoint corresponding to a particular change in particulate matter concentrations can be calculated using the following approach:

The attributable fraction/portion (AF) of health effects from air pollution, or impact factor, can be calculated from the relative risk (calculated for the incremental change in concentration considered as per Equation 1) as:

Equation 3 
$$AF = \frac{RR-1}{RR}$$

The total number of cases attributable to exposure to particulate matter (where a linear dose-response is assumed) can be calculated as:

### Equation 4 $E=AF \times B \times P$

Where:

B = baseline incidence of a given health effect (eg mortality rate per person per year) P = relevant exposed population

The above approach (while presented slightly differently) is consistent with that presented in Australia (Burgers & Walsh 2002), US (OEHHA 2002; USEPA 2005b, 2010) and Europe (Martuzzi et al. 2002; Sjoberg et al. 2009).

<sup>15</sup> For regional guidance, such as that provided for Europe by the WHO WHO 2006a, Health risks or particulate matter from long-range transboundary air pollution regional background incidence data for relevant health endpoints are combined with exposure-response functions to present an impact function, which is expressed as the number/change in incidence/new cases per 100,000 population exposed per microgram per cubic metre change in particulate matter exposure. These impact functions are simpler to use than the approach adopted in this assessment, however in utilising this approach it is assumed that the baseline incidence of the health effects is consistent throughout the whole population (as used in the studies) and is specifically applicable to the sub-population group being evaluated. For the assessment of exposures in the areas evaluated surrounding the project it is more relevant to utilise local data in relation to baseline incidence rather than assume that the population is similar to that in Europe (where these relationships are derived).

The calculation of an increased incidence (ie number of cases) of a particular health endpoint is not relevant to a specific individual, rather this is relevant to a statistically relevant population. This calculation has been undertaken for populations within the suburbs surrounding the proposed project. When considering the potential impact of the project on the population, the calculation has been undertaken using the following:

- Equation 1 has been used to calculate a relative risk. The relative risk has been calculated for a population weighted annual average incremental increase in concentrations. The population weighted average has been calculated on the basis of the smallest statistical division provided by the Australian Bureau of Statistics within a suburb (ie mesh blocks which are small blocks that cover an area of about 30 urban residences). For each mesh block in a suburb the average incremental increase in concentration has been calculated and multiplied by the population living in the mesh block (data available from the ABS for the 2011 census year). The weighted average has been calculated by summing these calculations for each mesh block in a suburb and dividing by the total population in the suburb (ie in all the mesh block)
- Equation 3 has been used to calculate an attributable fraction
- Equation 4 has been used to calculate the increased number of cases associated with the incremental impact evaluated. The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered and the population (for the relevant age groups) present in the suburb.

The above approach can be simplified (mathematically, where the incremental change in particulate concentration is low, less than one microgram per cubic metre) as follows:

Equation 5 
$$E=\beta \times B \times \sum_{mesh} (\Delta X_{mesh} \times P_{mesh})$$

Where:

 $\beta$  = slope coefficient relevant to the per cent change in response to a 1  $\mu$ g/m<sup>3</sup> change in exposure concentration

*B* = baseline incidence of a given health effect per person (eg annual mortality rate)

 $\Delta$ Xmesh = change (increment) in exposure concentration in  $\mu$ g/m<sup>3</sup> as an average within a small area defined as a mesh block (from the ABS – where many mesh blocks make up a suburb)

Pmesh = population (residential – based on data form the ABS) within each small mesh block

An additional risk can then be calculated as:

Equation 6 Risk=
$$\beta x \Delta X x B$$

Where:

 $\beta$  = slope coefficient relevant to the per cent change in response to a 1 µg/m<sup>3</sup> change in exposure  $\Delta X$  = change (increment) in exposure concentration in µg/m<sup>3</sup> relevant to the project at the point of exposure

*B* = baseline incidence of a given health effect per person (eg annual mortality rate)

This calculation provides an annual risk for individuals exposed to changes in air quality from the project at specific locations (such as the maximum, or at specific sensitive receptor locations). The calculated risk does not take into account the duration of exposure at any one location and hence is considered to be representative of a population risk.

### **Quantification of short and long term effects**

The concentration-response functions adopted for the assessment of exposure are derived from long and short term studies and relate to short or long term effects endpoints (eg change in incidence from daily changes in nitrogen dioxide or particulate matter, or chronic incidence from long term exposures to particulate matter).

Long term or chronic effects are assessed on the basis of the identified exposure-response function and annual average concentrations. These then allow the calculation of a chronic incidence of the assessed health endpoint.

Short term effects are also assessed on the basis of an exposure-response function that is expressed as a percentage change in endpoint per microgram per cubic metre change in concentration. For short term effects, the calculations relate to daily changes in nitrogen dioxide and particulate matter exposures to calculate changes in daily effects endpoints. While it may be possible to measure daily incidence of the evaluated health endpoints in a large population study specifically designed to include such data, it is not common to collect such data in hospitals nor are effects measurable in smaller communities. Instead these calculations relate to a parameter that is measurable, such as annual incidence of hospitalisations, mortality or lung cancer risks. The calculation of an annual incidence or additional risk can be undertaken using two approaches (Ostro 2004; USEPA 2010):

- Calculate the daily incidence or risk at each receptor location over every 24 hour period of the year (based on the modelled incremental 24 hour average concentration for each day of the year and daily baseline incidence data) and then sum the daily incidence/risk to get the annual risk
- Calculate the annual incidence/risk based on the incremental annual average concentration at each receptor (and using annual baseline incidence data).

In the absence of a threshold, and assuming a linear concentration-response function (as is the case in this assessment), these two approaches result in the same outcome mathematically (calculated incidence or risk). Given that it is much simpler computationally to calculate the incidence (for each receptor) based on the incremental annual average, compared with calculating effects on each day of the year and then summing, this is the preferred calculation method. It is the recommended method outlined by the WHO (Ostro 2004).

The use of the simpler approach, based on annual average concentrations should not be taken as implying or suggesting that the calculation is quantifying the effects of long term exposure.

Hence for the calculations presented in this technical report that relate to the expected use of the project tunnel, for both long term and short term effects, annual average concentrations of nitrogen dioxide and particulate matter have been utilised.

Where short term worst case exposures are assessed (such as those related to a breakdown in the tunnel) short term, daily, calculations have been undertaken to assessed short term health endpoints. This has been undertaken as the exposure being assessed relates to an infrequent short duration event. It would not occur each day of the year and hence it is not appropriate to assess on the basis of an annual average.

## Annexure B – Approach to assessment of cancer risk

Diesel exhaust (DE) is emitted from 'on-road' diesel engines (vehicle engines) and can be formed from the gaseous compounds emitted by diesel engines (secondary particulate matter). After emission from the exhaust pipe, diesel exhaust undergoes dilution and chemical and physical transformations in the atmosphere, as well as dispersion and transport in the atmosphere. The atmospheric lifetime for some compounds present in diesel exhaust ranges from hours to days.

Data from the USEPA (USEPA 2002b) indicates that diesel exhaust as measured as diesel particulate matter made up about six per cent of the total ambient/urban air  $PM_{2.5}$ . In this project, emissions to air from the operation of the tunnel include a significant proportion of diesel powered vehicles. Available evidence indicates that there are human health hazards associated with exposure to diesel particulate matter. The hazards include acute exposure-related symptoms, chronic exposure related non-cancer respiratory effects, and lung cancer.

In relation to non-carcinogenic effects, acute or short term (eg episodic) exposure to diesel particulate matter can cause acute irritation (eg eye, throat, bronchial), neurophysiological symptoms (eg lightheadedness, nausea), and respiratory symptoms (cough, phlegm). There also is evidence for an immunologic effect-exacerbation of allergenic responses to known allergens and asthma-like symptoms. Chronic effects include respiratory effects. The review of these effects (USEPA 2002b) identified a threshold concentration for the assessment of chronic non-carcinogenic effects. The review conducted by the USEPA also concluded that exposures to diesel particulate matter also consider  $PM_{2.5}$  goals (as these also address the presence of diesel particulate matter in urban air environments). The review found that the diesel particulate matter chronic guideline would also be met if the  $PM_{2.5}$  guideline was met.

Review of exposures to diesel particulate matter (USEPA 2002b) identified that such exposures are 'likely to be carcinogenic to humans by inhalation'. A more recent review by IARC (Attfield et al. 2012; IARC 2012; Silverman et al. 2012) classified diesel engine exhaust as carcinogenic to humans (Group 1) based on sufficient evidence that exposure is associated with an increased risk for lung cancer. In addition, outdoor air pollution and particulate matter (that includes diesel particulate matter) have been classified by IARC as carcinogenic to humans based on sufficient evidence.

Many of the organic compounds present in diesel exhaust are known to have mutagenic and carcinogenic properties and hence it is appropriate that a non-threshold approach is considered for the quantification of lung-cancer endpoints.

In relation to quantifying carcinogenic risks associated with exposure to diesel exhaust, the USEPA (USEPA 2002b) has not established a non-threshold value (due to uncertainties identified in the available data).

WHO has used data from studies in rats to estimate unit risk values for cancer (WHO 1996). Using four different studies where lung cancer was the cancer endpoint, WHO calculated a range of  $1.6 \times 10^{-5}$  to  $7.1 \times 10^{-5}$  per microgram per cubic metres (mean value of  $3.4 \times 10^{-5}$  per microgram per cubic metres). This would suggest that an increase in lifetime exposure to diesel particulate matter between 0.14 and 0.625 microgram per cubic metres could result in a one in one hundred thousand excess risk of cancer.

The California Environmental Protection Agency has proposed a unit lifetime cancer risk of  $3.0 \times 10^{-4}$  per microgram per cubic metres diesel particulate matter (OEHHA 1998). This was derived from data on exposed workers and based on evidence that suggested unit risks between  $1.5 \times 10^{-4}$  and  $15 \times 10^{-4}$  per microgram per cubic metres. This would suggest that an increase in lifetime exposure to diesel particulate matter of 0.033 microgram per cubic metres could result in a one in one hundred thousand excess risk of cancer. This estimate has been widely criticised as overestimating the risk and hence has not been considered in this assessment.

On the basis of the above, the WHO cancer unit risk value (mean value of  $3.4 \times 10^{-5}$  per microgram per cubic metres) has been used to evaluate potential excess lifetime risks associated with incremental impacts from diesel particulate matter exposures. Diesel particulate matter has not been specifically modelled in **Appendix E** (Air quality technical report); rather diesel particulate matter is part of the PM<sub>2.5</sub> assessment. For the purpose of this assessment it has been conservatively assumed that 100 per cent of the incremental PM<sub>2.5</sub> (from the project only) is derived from diesel sources. This is conservative as not all the vehicles using the tunnel (and emitting PM<sub>2.5</sub>) would be diesel powered (as currently there is a mix of petrol, diesel, LPG and hybrid-electric powered vehicles with the proportion of alternative fuels rising in the future).

For the assessment of potential lung cancer risks associated with exposure to diesel particulate matter, a non-threshold cancer risk is calculated. Non-threshold carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential non-threshold carcinogen. The numerical estimate of excess lifetime cancer risk is calculated as follows for inhalation exposures (USEPA 2009a):

#### Equation 7 Carcinogenic Risk (inhalation) = Concentration in Air x Inhalation Unit Risk x AF

### **Exposure adjustment factor (AF):**

The above calculation assumes the receptor is exposed at the same location for 24 hours of the day, every day, for a lifetime (which is assumed to be 70 years). This assumption is overly conservative for residents and workers in the community surrounding the project. Residents do not live in the one home for a lifetime. Guidance from enHealth indicates that an appropriate assumption for the time living in the one home is 35 years (enHealth 2012a). For residents, it is assumed that they may be at home for 20 hours per day for 365 days of the year, for 35 years. This results in an adjustment factor of 0.4 (20/24 hours x 35 years/70 years). This factor has been adopted for the assessment of all exposures regardless of whether these are residential areas, schools, recreational areas or workplaces.

## Annexure C – Acceptable risk levels

### General

The acceptability of an additional population risk is the subject of some discussion as there are currently no guidelines available in Australia, or internationally, in relation to an acceptable level of population risk associated with exposure to particulate matter. More specifically there are no guidelines available that relate to an acceptable level of risk for a small population (associated with impacts from a specific activity or project) compared with risks that are relevant to whole urban populations (that are considered when deriving guidelines). The following provides additional discussion in relation to evaluating calculated risk levels.

'The solution to developing better criteria for environmental contaminants is not to adopt arbitrary thresholds of 'acceptable risk' in an attempt to manage the public's perception of risk, or develop oversimplified tools for enforcement or risk assessment. Rather, the solution is to standardize the process by which risks are assessed, and to undertake efforts to narrow the gap between the public's understanding of actual vs. perceived risk. A more educated public with regard to the actual sources of known risks to health, environmental or otherwise, will greatly facilitate the regulatory agencies' ability to prioritize their efforts and standards to reduce overall risks to public health.' (Kelly 1991).

Most human activities that have contributed to economic progress present also some disadvantages, including risks of different kinds that adversely affect human health. These risks include air or water pollution due to industrial activities (coal power generation, chemical plants, and transportation), food contaminants (pesticide residues, additives), and soil contamination (hazardous waste). Despite all possible efforts to reduce these threats, it is clear that the zero risk objective is unobtainable or simply not necessary for human and environmental protection and that a certain level of risk in a given situation is deemed 'acceptable' as the effects are so small as to be negligible or undetectable. Risk managers need to cope with some residual risks and thus must adopt some measure of an acceptable risk.

Much has been written about how to determine the acceptability of risk. The general consensus in the literature is that 'acceptability' of a risk is a judgment decision properly made by those exposed to the hazard or their designated health officials. It is not a scientifically derived value or a decision made by outsiders to the process. Acceptability is based on many factors, such as the number of people exposed, the consequences of the risk, the degree of control over exposure, and many other factors.

The USEPA (Hoffman 1988) 'surveyed a range of health risks that our society faces' and reviewed acceptable-risk standards of government and independent institutions. The survey found that 'No fixed level of risk could be identified as acceptable in all cases and under all regulatory programs...,' and that: '...the acceptability of risk is a relative concept and involves consideration of different factors'. Considerations may include:

- The certainty and severity of the risk
- The reversibility of the health effect
- The knowledge or familiarity of the risk
- Whether the risk is voluntarily accepted or involuntarily imposed
- Whether individuals are compensated for their exposure to the risk
- The advantages of the activity
- The risks and advantages for any alternatives.

To regulate a technology in a logically defensible way, one must consider all its consequences, ie both risks and benefits.

### 10<sup>-6</sup> as an 'acceptable' risk level?

The concept of  $1 \times 10^{-6}$  ( $10^{-6}$ ) was originally an arbitrary number, finalised by the US Food and Drug Administration (FDA) in 1977 as a screening level of 'essentially zero' or de minimus risk. The term de minimus is an abbreviation of the legal concept, 'de minimus non curat lex: the law does not concern itself with trifles.' In other words,  $10^{-6}$  was developed as a level of risk below which risk was considered a 'trifle' and not of concern in a legal case.

This concept was traced back to a 1961 proposal by two scientists from the National Cancer Institute regarding methods to determine 'safety' levels in carcinogenicity testing. The FDA applied the concept in risk assessment in its efforts to deal with diethylstilboestrol as a growth promoter in cattle. The threshold of one in a million risk of developing cancer was established as a screening level to determine what carcinogenic animal drug residues merited further regulatory consideration. In the FDA legislation, the regulators specifically stated that this level of 'essentially zero' was not to be interpreted as equal to an acceptable level of residues in meat products. Since then, the use of risk assessment and 10<sup>-6</sup> (or variations thereof) have been greatly expanded to almost all areas of chemical regulatory agencies in different countries. What the FDA intended to be a lower regulatory level of 'zero risk' below which no consideration would be given as to risk to human health, for many regulators it somehow came to be considered a maximum or target level of 'acceptable' risk (Kelly 1991).

When evaluating human health risks, the quantification of risk can involve the calculation of an increased lifetime chance of cancer (as is calculated for diesel particulate matter in this assessment) or an increased probability of some adverse health effect (or disease) occurring, over and above the baseline incidence of that health effect/disease in the community (as is calculated for exposure to particulate matter).

In the context of human health risks,  $10^{-6}$  is a shorthand description for an increased chance of 0.000001 in one (one chance in a million) of developing a specific adverse health effect due to exposure (over a lifetime or a shorter duration as relevant for particulate matter) to a substance. The number  $10^{-5}$  represents one chance in 100,000, and so on.

Where cancer may be considered, lifetime exposure to a substance associated with a cancer risk of  $1 \times 10^{-6}$  would increase an individual's current chances of developing cancer from all causes (which is 40 per cent, or 0.4 -the background incidence of cancer in a lifetime) from 0.4 to 0.400001, an increase of 0.00025 per cent.

For other health indicators considered in this assessment, such as cardiovascular hospitalisations for people aged 65 years and older (for example), an increased risk of  $10^{-6}$  (one chance in a million) would increase an individual's (aged 65 years and older) chance of hospitalisation for cardiovascular disease (above the baseline incidence of 23 per cent, or 0.23) from 0.23 to 0.230001, an increase of 0.00043 per cent.

To provide more context in relation to the concept of a one in a million risk, the following presents a range of everyday life occurrences. The activity and the time spent undertaking the activity that is associated with reaching a risk of one in a million for mortality are listed below (Higson 1989; NSW Planning 2011):

- Motor vehicle accident 2.5 days spent driving a motor vehicle to reach one in a million chance of having an accident that causes mortality (death)
- Home accidents 3.3 days spent within a residence to reach a one in a million chance of having an accident at home that causes mortality
- Pedestrian accident (being struck by vehicles) 10 days spent walking along roads to reach a one in a million chance of being struck by a vehicle that causes mortality
- Train accident 12 days spent travelling on a train to reach a one in a million chance of being involved in an accident that causes mortality
- Falling down stairs [1] 66 days spent requiring the use of stairs in day-to-day activities to reach a one in a million chance of being involved in a fall that causes mortality

<sup>[1]</sup> Mortality risks as presented by: <u>http://www.riskcomm.com/visualaids/riskscale/datasources.php</u>.

F6 Extension Stage 1 from New M5 Motorway at Arncliffe to President Avenue at Kogarah Appendix F: Human Health Risk Technical Report

• Falling objects – 121 days spent in day-to-day activities to reach a one in a million chance of being hit by a falling object that causes mortality.

This risk level should also be considered in the context that everyone has a cumulative risk of death that ultimately must equal one and the annual risk of death for most of one's life is about one in 1000.

While various terms have been applied, it is clear that the two ends of what is a spectrum of risk are the 'negligible' level and the 'unacceptable' level. Risk levels intermediate between these are frequently adopted by regulators with varying terms often used to describe the levels. When considering a risk derived for an environmental impact it is important to consider that the level of risk that may be considered acceptable would lie somewhere between what is negligible and unacceptable, as illustrated below.



The calculated individual lifetime **took** of death or illness due to an exposure to a range of different environmental hazards covers many orders of magnitude, ranging from well less than  $10^{-6}$  to levels of  $10^{-3}$  and higher (in some situations). However, most figures for an acceptable or a tolerable risk range between  $10^{-6}$  to  $10^{-4}$ , used for either one year of exposure or a whole life exposure. It is noteworthy that  $10^{-6}$  as a criterion for 'acceptable risk' has not been applied to all sources of exposure or all agents that pose risk to public health.

A review of the evolution of  $10^{-6}$  reveals that perception of risk is a major determinant of the circumstances under which this criterion is used. The risk level  $10^{-6}$  is not consistently applied to all environmental legislation. Rather, it seems to be applied according to the general perception of the risk associated with the source being regulated and where the risk is being regulated (with different levels selected in different countries for the same sources).

A review of acceptable risk levels at the USEPA (Schoeny 2008) points out that risk assessors can identify risks and possibly calculate their value but cannot determine what is acceptable. Acceptability is a value judgment that varies with type of risk, culture, voluntariness and many other factors. Acceptability may be set by convention or law. The review also states that the USEPA aims for risk levels between  $10^{-6}$  and  $10^{-4}$  for risks calculated to be linear at low dose, while for other endpoints, not thought to be linear at low dose, the risk is compared to Reference Dose/Concentrations or guideline levels. The USEPA typically uses a target reference risk range of  $10^{-4}$  to  $10^{-6}$  for carcinogens in drinking water, which is in line with World Health Organization (WHO) guidelines for drinking water quality which, where practical, base guideline values for genotoxic carcinogens on the upper bound estimate of an excess lifetime cancer risk of  $10^{-5}$ .

There are many different ways to define acceptable risk and each way gives different weight to the views of different stakeholders in the debate. No definition of 'acceptable' would be acceptable to all stakeholders. Resolving such issues, therefore, becomes a political (in the widest sense) rather than a strictly health process.

The following is a list of standpoints that could be used as a basis for determining when a risk is acceptable or, perhaps, tolerable. The WHO (Fewtrell & Bartram 2001) address standards related to water quality. They offer the following guidelines for determining acceptable risk. A risk is acceptable when:

- It falls below an arbitrary defined probability
- It falls below some level that is already tolerated
- It falls below an arbitrary defined attributable fraction of total disease burden in the community
- The cost of reducing the risk would exceed the costs saved
- The cost of reducing the risk would exceed the costs saved when the 'costs of suffering' are also factored in
- The opportunity costs would be better spent on other, more pressing, public health problems
- Public health professionals say it is acceptable
- The general public say it is acceptable (or more likely, do not say it is not)
- Politicians say it is acceptable.

In everyday life individual risks are rarely considered in isolation. It could be argued that a sensible approach would be to consider health risks in terms of the total disease burden of a community and to define acceptability in terms of it falling below an arbitrary defined level. A problem with this approach is that the current burden of disease attributable to a single factor, such as air pollution, may not be a good indicator of the potential reductions available from improving other environmental health factors. For diseases such as cardiovascular disease where causes are multifactorial, reducing the disease burden by one route may have little impact on the overall burden of disease.

### Overall

It is not possible to provide a rigid definition of acceptable risk due to the complex and context driven nature of the challenge. It is possible to propose some general guidelines as to what might be an acceptable risk for specific development projects.

If the level of 10<sup>-6</sup> (one chance in a million) were retained as a level of increased risk that would be considered as a negligible risk in the community, then the level of risk that could be considered to be tolerable would lie between this level and an upper level that is considered to be unacceptable.

While there is no guidance available on what level of risk is considered to be unacceptable in the community, a level of 10<sup>-4</sup> for increased risk (one chance in 10,000) has been generally adopted by health authorities as a point where risk is considered to be unacceptable in the development of drinking water guidelines (that impact on whole populations) (for exposure to carcinogens as well as for annual risks of disease (Fewtrell & Bartram 2001)) and in the evaluation of exposures from pollutants in air (NSW DEC 2005).

Between an increased risk level considered negligible  $(10^{-6})$  and unacceptable  $(10^{-4})$  lie risks that may be considered to be tolerable or even acceptable. Tolerable risks are those that can be tolerated (and where the best available, and most appropriate, technology has been implemented to minimise exposure) in order to realise some benefit.

In a societal context, risks are inevitable and any new development would be accompanied by risks which are not amenable or economically feasible to reduce below a certain level. It is not good policy to impose an arbitrary risk level to such developments without consideration of the myriad factors that should be brought into play to determine what is 'tolerable'.

When considering the impacts associated with this project, it is important to note that there are a range of benefits associated with the project and the design of the project has incorporated measures to minimise exposures to traffic-related emissions in the local areas. Hence for this project the calculated risks have been considered to be tolerable when in the range of 10<sup>-6</sup> and 10<sup>-4</sup> of increased risk and where the increased incidence of the health impacts are considered to be insignificant.

### **Determination of significance of population impacts**

The assessment of potential health impacts associated with emissions to air from the project has not only calculated an increased annual risk, relevant to the health endpoints considered, but also a change in the incidence, ie the additional (or saving of) number of cases, of the adverse effects occurring within the population potentially exposed. The calculated change in incidence need to be considered in terms of what may be significant.

In relation to the calculated change in incidence of an adverse health effect occurring in a population, the following is noted for the primary health indicators (based on statistics available from NSW Health):

- In relation to mortality (all causes), the health statistics available show that for the year 2011/2012 the variability in all admissions data reported (based on the 95 per cent confidence interval for data reported in Sydney) is around ± 2.5 per cent. This is the variability in the data reported in one year. Each year the mortality rate also varies with around one per cent variability reported in the mortality rate (number reported for all causes) between 2010/11 and 2011/12. Based on the population considered in this assessment and the baseline incidence, a one per cent variability results in ± 10 cases per year. Changes in mortality within this range would not be detected (above normal variability) in the health statistics
- In relation to cardiovascular disease hospitalisations, the health statistics available show that for the year 2013/2014 the variability in all admissions data reported (based on the 95 percent confidence interval for data reported in Sydney) is around ± two percent. This is the variability in the data reported in one year. Each year the rate of hospitalisations (all ages) also varies with around two to three per cent variability reported in the number of hospitalisations for people aged 65 years and older in each year between 2010/11 and 2013/14. Based on the baseline incidence of cardiovascular hospitalisations considered in this assessment for individuals aged 65 years and the population considered in this assessment a variability of two per cent equates to ± 40 cases per year. Changes in cardiovascular hospitalisations in the population aged 65 years and older within this range would not be detected (above normal variability) in the health statistics
- In relation to respiratory disease hospitalisations, the health statistics available show that for the year 2013/2014 the variability in all admissions data reported (based on the 95 per cent confidence interval for data reported in Sydney) is around ± six per cent. This is the variability in the data reported in one year. Each year the rate of hospitalisations (all ages) also varies with around three to four per cent variability reported in the number of hospitalisations (all ages) in each year between 2011 and 2014. Based on the baseline incidence of respiratory hospitalisations considered in this assessment for individuals aged 65 years and older, and the population evaluated in this assessment, a variability of three per cent equates to ± 25 cases per year. Changes in respiratory hospitalisations in the population aged 65 years and older within this range would not be detected (above normal variability) in the health statistics.

Where changes in air quality associated with this project are well below 10 cases per year they are considered to be within the normal variability of health statistics. For evaluating impacts form this project a 10 fold margin of safety has been included to determine what changes in incidence may be considered negligible within the study population. This means that changes in the population incidence of any health effect evaluated that is less than one case per year are considered negligible.

Annexure D – Risk calculations: Nitrogen dioxide

#### Quantification of Effects - NO<sub>2</sub> F6 Extension

		2026	
Air quality indicator:	NO2	NO2	NO2
Endpoint:	Mortality - All	Mortality -	Asthma - ED
	Causes	Respiratory	Hospital
			admissions
Effect Exposure Duration:	Short-term	Short-term	Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m³ NO2) (as per Table 6-16)	0.00188	0.00426	0.00115
Annual Baseline Incidence (as per Table 4-5)			
Annual baseline incidence (per 100,000)	493	39.9	1209
Baseline Incidence (per person per year)	0.00493	0.000399	0.01209

NO2		NO2	NO2	NO2	
Asthma - ED		Mortality - All	Mortality -	Asthma - ED	
Hospital		Causes	Respiratory	Hospital	
admissions				admissions	
Short-term		Short-term	Short-term	Short-term	
1-14 years		All ages	All ages	1-14 years	
0.00115	_	0.00188	0.00426	0.00115	
1209	-	493	39.9	1209	_
0.01209		0.00493	0.000399	0.01209	
	_				_
Pick	Change in Annual Average NO2	Risk	Risk	Risk	Cha

2036 - Cumulative						
NO2	NO2	NO2				
Mortality - All Causes	Mortality - Respiratory	Asthma - ED Hospital admissions				
Short-term	Short-term	Short-term				
All ages	All ages	1-14 years				
0.00188	0.00426	0.00115				
493	39.9	1209				
0.00493	0.000399	0.01209				

Sensitive Receptors		Change in Annual Average NO2 Concentration (µg/m³)	Risk	Risk	Risk	Change in Annual Average NO2 Concentration (µg/m <sup>3</sup> )	Risk	Risk	Risk	Change in Annual Average NO2 Concentration (µg/m <sup>3</sup> )	Risk	Risk	Risk
Impacts from tunnel ventilation outlets													
Grid receptors: maximum regardless of landuse		1.63	2E-05	3E-06	2E-05	1.37	1E-05	2E-06	2E-05	1.50	1E-05	3E-06	2E-05
Grid receptors: maximum residential		1.63	2E-05	3E-06	2E-05	1.37	1E-05	2E-06	2E-05	0.94	9E-06	2E-06	1E-05
Grid receptors: commercial/industrial		1.23	1E-05	2E-06	2E-05	1.25	1E-05	2E-06	2E-05	1.50	1E-05	3E-06	2E-05
Grid receptors: maximum childcare		0.05	4E-07	8E-08	6E-07	0.11	1E-06	2E-07	1E-06	0.09	8E-07	1E-07	1E-06
Grid receptors: maximum school		0.74	7E-06	1E-06	1E-05	0.61	6E-06	1E-06	8E-06	0.70	7E-06	1E-06	1E-05
Grid receptors: maximum aged care		0.38	4E-06	7E-07	5E-06	0.30	3E-06	5E-07	4E-06	0.22	2E-06	4E-07	3E-06
Grid receptors: maximum hospital and medica		0.23	2E-06	4E-07	3E-06	0.41	4E-06	7E-07	6E-06	0.10	9E-07	2E-07	1E-06
Grid receptors: open space		0.39	4E-06	7E-07	5E-06	0.58	5E-06	1E-06	8E-06	0.68	6E-06	1E-06	9E-06
Community Receptors													
St Finbar's Primary Schoo	Primary Schoo	-0.051	-5E-07	-9E-08	-7E-07	-0.086	-8E-07	-1E-07	-1E-06	-0.236	-2E-06	-4E-07	-3E-06
St George Christian School Infant:	Primary Schoo	0.204	2E-06	3E-07	3E-06	0.162	2E-06	3E-07	2E-06	-0.627	-6E-06	-1E-06	-9E-06
Ramsgate Public Schoo	Primary Schoo	0.172	2E-06	3E-07	2E-06	0.279	3E-06	5E-07	4E-06	-0.117	-1E-06	-2E-07	-2E-06
Estia Health	Community Home	0.284	3E-06	5E-07	4E-06	0.344	3E-06	6E-07	5E-06	-0.885	-8E-06	-2E-06	-1E-05
Wesley Hospital Kogarał	General Hospita	-0.054	-5E-07	-9E-08	-7E-07	0.089	8E-07	2E-07	1E-06	-0.134	-1E-06	-2E-07	-2E-06
St George Schoo	Special Schoo	0.435	4E-06	7E-07	6E-06	0.384	4E-06	7E-07	5E-06	-0.282	-3E-06	-5E-07	-4E-06
St George Hospita	General Hospita	0.092	9E-07	2E-07	1E-06	0.287	3E-06	5E-07	4E-06	-0.034	-3E-07	-6E-08	-5E-07
Brighton-Le-Sands Public Schoo	Primary Schoo	0.046	4E-07	8E-08	6E-07	0.222	2E-06	4E-07	3E-06	0.197	2E-06	3E-07	3E-06
Kogarah Public Schoo	Primary Schoo	0.219	2E-06	4E-07	3E-06	-0.029	-3E-07	-5E-08	-4E-07	-0.153	-1E-06	-3E-07	-2E-06
St George Girls High Schoo	High School	-0.135	-1E-06	-2E-07	-2E-06	0.182	2E-06	3E-07	3E-06	-0.157	-1E-06	-3E-07	-2E-06
St Thomas More's Catholic Schoo	Primary Schoo	-0.042	-4E-07	-7E-08	-6E-07	-0.589	-5E-06	-1E-06	-8E-06	-0.392	-4E-06	-7E-07	-5E-06
Jenny-Lyn Nursing Home	Community Home	-0.156	-1E-06	-3E-07	-2E-06	-0.225	-2E-06	-4E-07	-3E-06	-0.152	-1E-06	-3E-07	-2E-06
Huntingdon Gardens Aged Care Facilit	Community Home	-0.173	-2E-06	-3E-07	-2E-06	0.100	9E-07	2E-07	1E-06	-0.069	-6E-07	-1E-07	-1E-06
Rockdale Public Schoo	Primary Schoo	-0.012	-1E-07	-2E-08	-2E-07	-0.053	-5E-07	-9E-08	-7E-07	0.040	4E-07	7E-08	6E-07
Scalabrini Village Nursing Home-Bexle	Community Home	-0.139	-1E-06	-2E-07	-2E-06	-0.159	-1E-06	-3E-07	-2E-06	-0.230	-2E-06	-4E-07	-3E-06
Rockdale Nursing Home	Community Home	-0.051	-5E-07	-9E-08	-7E-07	-0.066	-6E-07	-1E-07	-9E-07	-0.059	-5E-07	-1E-07	-8E-07
Arncliffe Public School	Primary Schoo	-0.408	-4E-06	-7E-07	-6E-06	-0.189	-2E-06	-3E-07	-3E-06	-0.450	-4E-06	-8E-07	-6E-06
Athelstane Public Schoo	Primary Schoo	-0.069	-6E-07	-1E-07	-1E-06	0.004	4E-08	7E-09	6E-08	-0.069	-6E-07	-1E-07	-1E-06
Al Zahra College	Combined Primary-Secondary Schot	-0.120	-1E-06	-2E-07	-2E-06	-0.014	-1E-07	-2E-08	-2E-07	-0.094	-9E-07	-2E-07	-1E-06
Cairsfoot Schoo	Special Schoo	-0.321	-3E-06	-5E-07	-4E-06	-0.217	-2E-06	-4E-07	-3E-06	-0.281	-3E-06	-5E-07	-4E-06
Undercliffe Public Schoo	Primary Schoo	-0.088	-8E-07	-1E-07	-1E-06	-0.220	-2E-06	-4E-07	-3E-06	-0.303	-3E-06	-5E-07	-4E-06
Ferncourt Public Schoo	Primary Schoo	-0.212	-2E-06	-4E-07	-3E-06	-0.002	-2E-08	-3E-09	-3E-08	-0.014	-1E-07	-2E-08	-2E-07
Tempe High Schoo	High School	0.078	7E-07	1E-07	1E-06	-0.187	-2E-06	-3E-07	-3E-06	-0.365	-3E-06	-6E-07	-5E-06
St Peters Public Schoo	Primary Schoo	-0.191	-2E-06	-3E-07	-3E-06	0.212	2E-06	4E-07	3E-06	0.295	3E-06	5E-07	4E-06
St Pius' Catholic Primary Schoo	Primary Schoo	-0.075	-7E-07	-1E-07	-1E-06	0.200	2E-06	3E-07	3E-06	0.141	1E-06	2E-07	2E-06
Frobel Alexandria Early Learning Centr	Child Care Centre	0.060	6E-07	1E-07	8E-07	0.019	2E-07	3E-08	3E-07	0.173	2E-06	3E-07	2E-06
Little Learning School - Alexandri	Child Care Centre	-0.018	-2E-07	-3E-08	-2E-07	-0.094	-9E-07	-2E-07	-1E-06	-0.204	-2E-06	-3E-07	-3E-06
Active Kids Mascol	Child Care Centre	-0.377	-3E-06	-6E-07	-5E-06	0.183	2E-06	3E-07	3E-06	0.1/1	2E-06	3E-07	2E-06
Mascot Public Schoo	Primary Schoo	-0.356	-3E-06	-6E-07	-5E-06	0.102	9E-07	2E-07	1E-06	+0.336	-3E-06	-6E-07	-5E-06
Hippos Friends	Child Care Centre	0.048	4E-07	8E-08	7E-07	0.092	9E-07	2E-07	1E-06	0.021	2E-07	3E-08	3E-07

## Quantification of Effects - $\ensuremath{\text{NO}_2}$ - Ventilation facilities only F6 Extension

				2026				2036				2036 - Cumulative	,
		Air quality indicator:	NO2	NO2	NO2		NO2	NO2	NO2		NO2	NO2	NO2
		Endpoint:	Mortality - All	Mortality -	Asthma - FD		Mortality - All	Mortality -	Asthma - FD		Mortality - All	Mortality -	Asthma - FD
		Endpoint	Causas	Bocniratory	Hospital		Caucas	Bospiratory	Hospital		Caucas	Bocniratory	Hospital
			Causes	Respiratory	nospital		Causes	Respiratory	nospital		Gauses	Respiratory	nospital
				<b>e</b> t	admissions				admissions				admissions
		Effect Exposure Duration:	Short-term	Short-term	Short-term		Short-term	Short-term	Short-term		Short-term	Short-term	Short-term
		Age Group:	All ages	All ages	1-14 years		All ages	All ages	1-14 years		All ages	All ages	1-14 years
	β (change in effect per 1 µg/m	<sup>3</sup> NO2) (as per Table 6-16)	0.00188	0.00426	0.00115		0.00188	0.00426	0.00115		0.00188	0.00426	0.00115
	Annual Baseline In	cidence (as per Table 4-5)											-
	Annual baselin	no incidence (per 100 000)	40.2	20.0	1200		402	20.0	1200		402	20.0	1200
	Annual basen	ne incluence (per 100,000)	455	0.000200	0.01000		433	0.000200	0.01200		493	0.000200	0.01209
	Baseline Incide	ence (per person per year)	0.00493	0.000288	0.01209	1	0.00493	0.000399	0.01209		0.00493	0.000399	0.01209
			_										
		Change in Annual				Change in Annual				Change in Appual			
		Change III Annual				Change III Annual				Change III Annual			
Sensitive Receptors		Average NO2	Risk	Risk	Risk	Average NO2	Risk	Risk	Risk	Average NO2	Risk	Risk	Risk
		Concentration (µg/m <sup>3</sup> )				Concentration (µg/m <sup>3</sup> )				Concentration (µg/m <sup>3</sup> )			
impacts from tunnel ventilation outlets	1												
Grid receptors: maximum regardless of landuse		0.0109	1E-07	2E-08	2E-07	0.23	2E-06	4E-07	3E-06	0.15	1E-06	3E-07	2E-06
Grid receptors: maximum residential		0.00911	8E-08	2E-08	1E-07	0.23	2E-06	4E-07	3E-06	0.15	1E-06	3E-07	2E-06
Grid receptors: commercial/industrial		0.011	1E-07	2E-08	2E-07	0.17	2E-06	3E-07	2E-06	0.14	1E-06	2E-07	2E-06
Grid receptors: maximum childcare		0.0012	1E-08	2E-09	2E-08	0.057	5E-07	1E-07	8E-07	0.082	8E-07	1E-07	1E-06
Grid receptors: maximum send core		0.002	2E-08	3E-09	3E-08	0.12	1E-06	2E-07	2E-06	0.077	1E-06	2E-07	2E-06
Grid receptors: maximum aged care		0.00041	4E-09	7E-10	6E-09	0.073	/E-0/	1E-07	1E-06	0.077	7E-07	1E-07	1E-06
Grid receptors: maximum nospital and medica		0.00000	0E-09 3E-09	1E-09 5E-09	9E-09	0.087	8E-07	1E-07 2E-07	1E-06	0.08	7E-07 1E-06	1E-07 2E-07	1E-06
Community Bocontors		0.000	02.00	02.00	42.00		02 01	22.01	12 00		12 00	22 01	22.00
Community Receptors		0.010								0.010			
St Findar's Primary School	Primary Schoo	0.013	1E-07	2E-08	2E-07	0.013	1E-07	2E-08	2E-07	0.013	1E-07	2E-08	2E-07
St George Christian School Infant:	Primary Schoo	0.017	9E-08	2E-08	1E-07	0.020	2E-07	3E-08	3E-07	0.027	2E-07	3E-08	4E-07
Fatis Health	Community Home	0.017	2E-07	3E-00	2E-07	0.021	4E 07	4E-08	3E-07	0.055	4E-07	7E-08	9E 07
Wesley Hospital Konarak	General Hospita	0.032	4E-07	3E-00 7E-09	4E-07	0.045	4E=07 3E=07	7E-08	5E-07	0.053	5E-07	9E-08	7E-07
St George Schoo	Special Schoo	0.065	6E-07	1E-07	9E-07	0.066	6E-07	1E-07	9E-07	0.091	8E-07	2E=07	1E-06
St George Hospita	General Hospita	0.065	6E-07	1E-07	9E-07	0.068	6E-07	1E-07	9E-07	0.085	8E-07	1E-07	1E-06
Brighton-Le-Sands Public Schoo	Primary Schoo	0.116	1E-06	2E-07	2E-06	0.123	1E-06	2E-07	2E-06	0.144	1E-06	2E-07	2E-06
Kogarah Public Schoo	Primary Schoo	0.060	6E-07	1E-07	8E-07	0.062	6E-07	1E-07	9E-07	0.083	8E-07	1E-07	1E-06
St George Girls High Schoo	High School	0.037	3E-07	6E-08	5E-07	0.043	4E-07	7E-08	6E-07	0.059	5E-07	1E-07	8E-07
St Thomas More's Catholic Schoo	Primary Schoo	0.062	6E-07	1E-07	9E-07	0.061	6E-07	1E-07	9E-07	0.103	1E-06	2E-07	1E-06
Jenny-Lyn Nursing Home	Community Home	0.029	3E-07	5E-08	4E-07	0.029	3E-07	5E-08	4E-07	0.049	5E-07	8E-08	7E-07
Huntingdon Gardens Aged Care Facilit	Community Home	0.022	2E-07	4E-08	3E-07	0.023	2E-07	4E-08	3E-07	0.034	3E-07	6E-08	5E-07
Rockdale Public Schoo	Primary Schoo	0.053	5E-07	9E-08	7E-07	0.060	6E-07	1E-07	8E-07	0.083	8E-07	1E-07	1E-06
Scalabrini village Nursing Home-Bexte	Community Home	0.018	2E-07	3E-08	3E-07	0.014	1E-07	2E-08	2E-07	0.023	2E-07	4E-08	3E-07
Kockoale Inursing Home	Community Home	0.020	2E-07	3E-08	3E-07	0.023	2E-07	4E-08	3E-07	0.038	4E-07	6E-08	5E-07
Athelatone Dublic School	Primary 30000	0.041	4E-U/ 7E.09	/E-U8	0E-U/ 1E.07	0.035	3E-07	0E-U8 1E-09	3E-U/	0.004	0E-07	1E-0/	9E-07
Atheistane Fubic Schoo	Combined Primany Secondary Schor	0.008	7E-08	1E-08	1E-07	0.009	8E-08	1E-08 1E-09	1E-0/ 9E-00	0.010	2E-07	3E-08	3E-07
Cairefoot Schoo	Special School	0.008	7E-08 3E-07	1E-08 5E-08	1E-07	0.031	3E-09	1E-09 5E-09	0E-09 4E-07	0.025	2E-07 6E-07	4E-08	0E-07
Lindercliffe Public Schoo	Primary Schoo	0.008	9E-09	1E-09	407	0.006	3E-07	1E-09	4E-07	0.002	1E-07	2E-09	9E-07
Ferncourt Public Schoo	Primary Schoo	0.007	6E-08	1E-08	9E-08	0.012	1E-00	2E-08	2E-00	0.008	7E-08	1E-08	1E-07
Tempe High Schoo	High School	0.008	7E-08	1E-08	1E-07	0.002	2E-08	3E-09	3E-08	0.020	2E-07	3E-08	3E-07
St Peters Public Schoo	Primary Schoo	-0.035	-3E-07	-6E-08	-5E-07	-0.054	-5E-07	-9E-08	-7E-07	-0.006	-6E-08	-1E-08	-9E-08
St Pius' Catholic Primary Schoo	Primary Schoo	0.001	5E-09	9E-10	8E-09	-0.007	-7E-08	-1E-08	-1E-07	0.015	1E-07	3E-08	2E-07
Frobel Alexandria Early Learning Centr	Child Care Centre	-0.010	-9E-08	-2E-08	-1E-07	-0.013	-1E-07	-2E-08	-2E-07	0.000	3E-09	6E-10	5E-09
Little Learning School - Alexandri	Child Care Centre	-0.025	-2E-07	-4E-08	-3E-07	-0.030	-3E-07	-5E-08	-4E-07	0.000	-2E-09	-3E-10	-2E-09
Active Kids Mascol	Child Care Centre	-0.003	-3E-08	-5E-09	-4E-08	-0.013	-1E-07	-2E-08	-2E-07	0.005	4E-08	8E-09	7E-08
Mascot Public Schoo	Primary Schoo	0.002	2E-08	4E-09	3E-08	0.001	8E-09	1E-09	1E-08	-0.001	-1E-08	-2E-09	-2E-08
Hippos Friends	Child Care Centre	0.009	8E-08	2E-08	1E-07	0.002	2E-08	4E-09	3E-08	0.009	8E-08	2E-08	1E-07

# Annexure E – Population incidence calculations: Nitrogen dioxide

### Assessment of Increased Incidence - NO2 F6 Extension: 2026

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Strathfield - Burwood - Ashfield LGA			
Total Population in study areas	20160	20160	20160
% population in assessment age-group	100%	100%	19%
total change	-29.18	-29.18	-29.18
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00144742	-0.00144742	-0.00144742
Baseline Incidence (per 100,000) (as per Table 4-5)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Attributoble frection (AF)	0.999997	0.999994	0.999998
Attributable iraction (AF):	-2.7E-06	-6.2E-06	-1.7E-06
Risk:	-0.0003 -1.3E-08	-0.00003 -2.5E-09	-0.00000
Individual subrubs within LGA	1.52 00	2.02 00	2.02.00
Ashfield			
Total Population in study area	1512	1512	1512
% population in assessment age-group	100%	100%	19%
total change	-0.12	-0.12	-0.12
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00007937	-0.00007937	-0.00007937
Baseline Incidence (per 100,000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	1.000000	1.000000	1.000000
Attributable fraction (AF):	-1.5E-07	-3.4E-07	-9.1E-08
Increased number of cases in population	0.0000	0.0000	0.0000
Risk:	-7.1E-10	-1.3E-10	-1.1E-09
Canterbury (North) - Ashbury	7170	7170	7470
I otal Population in study area:	/1/9	/1/9	/1/5
% population in assessment age-group	100%	100%	19%
Deputation weighted Av (ug/m <sup>3</sup> )	-7.71	0.00107207	0.00107207
Population weighted $\Delta x$ (μg/m). Baseline incidence (per 100 000) (as per Table 4.4)	-0.00107397	-0.00107397	-0.00107397
Baseline Incidence (per 100,000) (as per 1 able 4.4)	0.00477	0 00040	0.01208
Balative Rick:	0.00477	0.00040	0.01208
Attributable fraction (AF):	-2.0E-06	-4.6E-06	-1.2E-06
Increased number of cases in population	-0.0001	0.0000	0.0000
Risk:	-9.6E-09	-1.8E-09	-1.5E-08
Dulwich Hill - Lewisham			
Total Population in study areas	11400	11400	11400
% population in assessment age-group	100%	100%	19%
total change	-21.34	-21.34	-21.34
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00187193	-0.00187193	-0.00187193
Baseline Incidence (per 100,000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	0.999996	0.999992	0.999998
Attributable fraction (AF):	-3.5E-06	-8.0E-06	-2.2E-06
Increased number of cases in population	-0.0002	0.0000	-0.0001
Kisk: Haborfield - Summer Hill	-1.7E-08	-3.2E-09	-2.6E-08
Total Population in study area	03	03	60
% population in assessment are-group	100%	100%	10%
total change	-0.012	-0 012	-0.012
Population weighted Ax (ug/m <sup>3</sup> ):	-0.00017391	-0.00017391	-0 00017391
Baseline Incidence (per 100 000) (as per Table 4 4)	477	40	1200
Baseline Incidence (per person)	0 00477	0 00040	0.01200
Relative Risk:	1.000000	0.999999	1.000000
Attributable fraction (AF):	-3.3E-07	-7.4E-07	-2.0E-07
Increased number of cases in population	0.0000	0.0000	0.0000
Risk:	-1.6E-09	-3.0E-10	-2.4E-09

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Sydney Inner City LGA			
Total Population in study area:	29695	29695	29695
% population in assessment age-group	100%	100%	6%
total change	-6	-6	-6
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00020205	-0.00020205	-0.0002020
Baseline Incidence (per 100,000) (as per Table 4-5)	508	40	1209
Baseline Incidence (per person)	0.00508	0.00040	0.01209
Relative Risk:	1.000000	0.999999	1.00000
Attributable fraction (AF):	-3.8E-07	-8.6E-07	-2.3E-07
Increased number of cases in population:	0.000	-0.00001	0.0000
Risk:	-1.9E-09	-3.4E-10	-2.8E-09
Individual subrubs within LGA			
Erskinville - Alexandria			
I otal Population in study area:	11411	11411	1141
% population in assessment age-group	100%	100%	6%
	18.7	18.7	18.
Population weighted $\Delta x$ (µg/m <sup>2</sup> ):	0.00163877	0.00163877	0.0016387
Baseline Incidence (per 100,000) (as per 1 able 4.4)	508	40	120
Baseline Incidence (per person)	0.00508	0.00040	0.01209
Relative Risk:	1.000003	1.000007	1.000002
All Indiable Traction (AF).	3.1E-06	7.0E-00	0.00001
Pick-	1.6E-08	0.00003 2.8E-00	2.35-09
Newtown - Camperdown - Darlington	1.02-00	2.02-03	2.32-00
Total Population in study area:	5225	5225	522
% population in assessment age-group	100%	100%	6%
total change	14.2	14.2	1420%
Population weighted Ax (ug/m <sup>3</sup> ):	0 00271770	0 00271770	0.00271770
Baseline Incidence (per 100 000) (as per Table 4 4)	508	40	1209
Baseline Incidence (per reison)	0.00508	0 00040	0.01209
Relative Risk:	1 000005	1 000012	1 00000
Attributable fraction (AF):	5.1E-06	1.2E-05	3.1E-06
Increased number of cases in population:	0.000	0.000	0.0000
Risk:	2.6E-08	4.6E-09	3.8E-08
Waterloo - Beaconsfield			
Total Population in study area:	13059	13059	13059
% population in assessment age-group	100%	100%	6%
total change	-38.9	-38.9	-38.9
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00297879	-0.00297879	-0.00297879
Baseline Incidence (per 100,000) (as per Table 4.4)	508	40	1209
Baseline Incidence (per person)	0.00508	0.00040	0.01209
Relative Risk:	0.999994	0.999987	0.999993
Attributable fraction (AF):	-5.6E-06	-1.3E-05	-3.4E-06
Increased number of cases in population	0.000	0.000	0.000
Risk:	-2.8E-08	-5.1E-09	-4.1E-08
			1

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Marrickville - Sydenham - Petersham LGA			
Total Population in study area:	35722	35722	35722
% population in assessment age-group:	100%	100%	14%
total change	-93.0	-92.97	-92.97
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00260260	-0.00260260	-0.00260260
Baseline Incidence (per 100,000) (as per Table 4-5)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999995	0.999989	0.999997
Attributable fraction (AF):	-4.9E-06	-1.1E-05	-3.0E-06
Increased number of cases in population:	-0.0009	-0.00016	-0.00018
Risk:	-2.6E-08	-4.4E-09	-3.6E-08
Individual subrubs within LGA			
Marrickville			
Total Population in study area:	25842	25842	25842
% population in assessment age-group:	100%	100%	14%
total change	-28.64	-28.64	-28.64
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00110827	-0.00110827	-0.00110827
Baseline Incidence (per 100,000) (as per Table 4.4)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999998	0.999995	0.999999
Attributable fraction (AF):	-2.1E-06	-4.7E-06	-1.3E-06
Increased number of cases in population.	1 1E 09	1.0000	-0.00000
Petersham - Stanmore	-1.1E-00	-1.9E-09	-1.5E-00
Total Population in study area:	2051	2051	205
% population in assessment age-group:	100%	100%	14%
total change	5.92	5.92	5.92
Population weighted Ax (ug/m <sup>3</sup> ):	0.00288640	0.00288640	0.00288640
Baseline Incidence (per 100 000) (as per Table 4 4)	534	40	1209
Baseline Incidence (per person)	0.00534	0 00040	0.01209
Relative Risk:	1.000005	1.000012	1.000003
Attributable fraction (AF):	5.4E-06	1.2E-05	3.3E-06
Increased number of cases in population:	0.000	0.0000	0.00002
Risk:	2.9E-08	4.9E-09	4.0E-08
Sydenham - Tempe - St Peters			
Total Population in study area:	7829	7829	7829
% population in assessment age-group:	100%	100%	14%
total change	-70.25	-70.25	-70.25
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00897305	-0.00897305	-0.00897305
Baseline Incidence (per 100,000) (as per Table 4.4)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999983	0.999962	0.999990
Attributable fraction (AF):	-1.7E-05	-3.8E-05	-1.0E-05
Increased number of cases in population:	-0.001	-0.0001	-0.0001
Risk:	-9.0E-08	-1.5E-08	-1.2E-07
		1	1

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Canterbury LGA			
Total Population in study areas	12562	12562	12562
% population in assessment age-group	100%	100%	19%
total change	-0.97	-0.97	-0.97
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00007722	-0.00007722	-0.00007722
Baseline Incidence (per 100,000) (as per Table 4-5)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	1.000000	1.000000	1.000000
Attributable fraction (AF):	-1.5E-07	-3.3E-07	-8.9E-08
Increased number of cases in population	0.000	0.00000	0.0000
Risk:	-7.1E-10	-1.3E-10	-1.1E-09
Individual subrubs within LGA			
Canterbury (South) - Campsie			
Total Population in study areas	149	149	149
% population in assessment age-group	100%	100%	19%
total change	0.34	0.34	0.34
Population weighted $\Delta x (\mu g/m^3)$ :	0.00228188	0.00228188	0.00228188
Baseline Incidence (per 100,000) (as per Table 4.4)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	1.000004	1.000010	1.000003
Attributable fraction (AF):	4.3E-06	9.7E-06	2.6E-06
Increased number of cases in population	0.0000	0.0000	0.0000
Risk:	2.1E-08	3.9E-09	3.2E-08
Kingsgrove (North) - Earlwood			
Total Population in study areas	12413	12413	12413
% population in assessment age-group	100%	100%	19%
total change	-1.31	-1.31	-1.31
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00010553	-0.00010553	-0.00010553
Baseline Incidence (per 100,000) (as per Table 4.4)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	1.000000	1.000000	1.000000
Attributable fraction (AF):	-2.0E-07	-4.5E-07	-1.2E-07
Increased number of cases in population	0.0000	0.0000	0.0000
Risk:	-9.7E-10	-1.8E-10	-1.5E-09
Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
---	---	---	---
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Botany LGA			
Total Population in study area:	31386	31386	31386
% population in assessment age-group	100%	100%	16%
total change	-243.3	-243.25	-243.25
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00775027	-0.00775027	-0.00775027
Baseline Incidence (per 100,000) (as per Table 4-5)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
Relative Risk:	0.999985	0.999967	0.999991
All induitable inaction (AF).	-1.5E-05	-3.3E-03	-0.9E-00
Rick	-0.002393	-0.000413	-0.000531
Individual subrubs within I GA	-7.0L-00	-1.32-00	-1.12-07
Botany			
Total Population in study area:	10408	10408	10408
% population in assessment age-group:	100%	100%	16%
total change	-76.52	-76.52	-76.52
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00735204	-0.00735204	-0.00735204
Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
Relative Risk:	0.999986	0.999969	0.999992
Attributable fraction (AF):	-1.4E-05	-3.1E-05	-8.5E-06
Increased number of cases in population:	-0.0008	-0.0001	-0.0002
Risk:	-7.2E-08	-1.2E-08	-1.0E-07
Mascot - Eastlakes			
I otal Population in study area:	20286	20286	20286
% population in assessment age-group:	100%	100%	16%
Bopulation weighted Av (ug/m <sup>3</sup> ):	0.00922101	0.00922101	0.00922101
Baseline Incidence (per 100 000) (as per Table 4.4)	-0.00632101	-0.00632101	-0.00832101
Baseline Incidence (per 100,000) (as per rable 4.4)	0.00524	0 00040	0.01209
Belative Risk:	0.999984	0.999965	0.999990
Attributable fraction (AF):	-1.6E-05	-3.5E-05	-9.6E-06
Increased number of cases in population:	-0.0017	-0.0003	-0.0004
Risk:	-8.2E-08	-1.4E-08	-1.2E-07
Pagewood - Hillsdale - Daceyville			
Total Population in study area:	631	631	631
% population in assessment age-group	100%	100%	16%
total change	2.88	2.88	2.88
Population weighted $\Delta x (\mu g/m^3)$ :	0.00456418	0.00456418	0.00456418
Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
Relative Risk:	1.000009	1.000019	1.000005
Attributable fraction (AF):	8.6E-06	1.9E-05	5.2E-06
Risk:	4 5E-08	7.8E-09	6.3E-08
Svdnev Airport	4.02 00	7.02 00	0.02 00
Total Population in study area:	61	61	61
% population in assessment age-group	100%	100%	16%
total change	-0.786	-0.786	-0.786
Population weighted $\Delta x (\mu q/m^3)$ :	-0.01288525	-0.01288525	-0.01288525
Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
Relative Risk:	0.999976	0.999945	0.999985
Attributable fraction (AF):	-2.4E-05	-5.5E-05	-1.5E-05
Increased number of cases in population:	0.0000	0.0000	0.0000
Risk:	-1.3E-07	-2.2E-08	-1.8E-07
	I		

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Kogarah - Rockdale LGA			
Total Population in study area:	113547	113547	113547
% population in assessment age-group:	100%	100%	15%
total change	104.5	104.51	104.51
Population weighted $\Delta x$ (µg/m <sup>3</sup> ):	0.00092041	0.00092041	0.00092041
Baseline Incidence (per 100,000) (as per Table 4-5)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Attributable fraction (AE):	1.000002	1.00004 3.9E-06	1.000001
Increased number of cases in population:	0.001	0.002	0.0002
Risk:	9.2E-09	1.6E-09	1.3E-08
Individual subrubs within LGA			
Arncliffe - Bardwell Park			
Total Population in study area:	21457	21457	21457
% population in assessment age-group:	100%	100%	15%
total change	-138.2	-138.2	-138.2
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00644079	-0.00644079	-0.00644079
Baseline Incidence (per 100,000) (as per Lable 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Attributable fraction (AF):	0.999988 -1.2E-05	0.999973	0.999993 -7.4E-06
Increased number of cases in population:	-0.0014	-0.0002	-0.00028
Risk:	-6.5E-08	-1.1E-08	-9.0E-08
Bexley			
Total Population in study area:	20002	20002	20002
% population in assessment age-group:	100%	100%	15%
total change	11.06	11.06	11.06
Population weighted $\Delta x (\mu g/m^3)$ :	0.00055294	0.00055294	0.00055294
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Attributable fraction (AE)	1.000001	1.000002	1.000001
Allibutable fraction (AF).	0.0001	2.4E-00	0.42-07
Risk:	5.6E-09	9.4E-10	7.7E-09
Kingsgrove (South) - Bardwell Park	0.02 00	0112 10	
Total Population in study area:	2879	2879	2879
% population in assessment age-group:	100%	100%	15%
total change	-1.38	-1.38	-1.38
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00047933	-0.00047933	-0.00047933
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Relative Risk:	0.999999	0.999998	0.999999
Attributable fraction (AF):	-9.0E-07	-2.0E-06	-5.5E-07
Risk:	-4 8E-09	-8 1E-10	-6 7E-09
Kogarah	4.02 00	0.12 10	0.72 03
Total Population in study area:	11323	11323	11323
% population in assessment age-group:	100%	100%	15%
total change	211.98	211.98	211.98
Population weighted $\Delta x (\mu g/m^3)$ :	0.01872119	0.01872119	0.01872119
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Relative Risk:	1.000035	1.000080	1.000022
Attributable fraction (AF):	3.5E-05	8.0E-05	2.2E-05
Increased number of cases in population:	1.0021	0.00036 3.2E-08	0.00043 2 6E-07
Kogarah Bay - Carlton - Allawah	1.92-07	J.2L-00	2.02-07
Total Population in study area:	10923	10923	10923
% population in assessment age-group:	100%	100%	15%
	79.5	79.5	79.5
Population weighted $\Delta x (\mu g/m^3)$ :	0.00727822	0.00727822	0.00727822
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Relative Risk:	1.000014	1.000031	1.00008
Attributable fraction (AF):	1.4E-05	3.1E-05	8.4E-06
Increased number of cases in population:	0.0008	0.00014	0.00016
KISK: Monterey - Brighton-le-Sands - Kycomagh	1.3E-08	1.2E-08	1.0E-07
Total Population in study area:	13015	13015	13015
% population in assessment age-group	100%	100%	15%
total change	-58.4	-58.4	-58.4
Population weighted $\Delta x$ (µg/m <sup>3</sup> ):	-0.00419691	-0.00419691	-0.00419691
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	0.999992	0.999982	0.999995
Attributable fraction (AF)	-7.9E-06	-1.8E-05	-4.8E-06
Increased number of cases in population	-0.00059	-0.00010	-0.000119
Risk	-4.2E-08	-7.1E-09	-5.8E-08
Rockdale - Banksia	1		
Total Population in study area	19957	19957	19957
% population in assessment age-group	: 100%	100%	15%
total change	-76.6	-76.6	-76.6
Population weighted $\Delta x (\mu g/m^3)$	-0.00383825	-0.00383825	-0.00383825
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	0.999993	0.999984	0.999996
Attributable fraction (AF)	-7.2E-06	-1.6E-05	-4.4E-06
Increased number of cases in population	-0.00077	-0.00013	-0.000155
Risk	-3.9E-08	-6.5E-09	-5.3E-08
Sans Souci - Ramsgate	÷		
Total Population in study area	13091	13091	13091
% population in assessment age-group	: 100%	100%	15%
total change	76.6	76.6	76.6
Population weighted $\Delta x (\mu g/m^3)$	0.00585135	0.00585135	0.00585135
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000011	1.000025	1.000007
Attributable fraction (AF)	1.1E-05	2.5E-05	6.7E-06
Increased number of cases in population	. 0.00077	0.00013	0.000155
Risk	5.9E-08	9.9E-09	8.1E-08
	I		

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16	0.00188	0.00426	0.00115
Hurstville LGA			
Total Population in study area	657	657	657
% population in assessment age-group	: 100%	100%	15%
total change	2.4	2.39	2.39
Population weighted $\Delta x (\mu g/m^3)$	0.00363775	0.00363775	0.00363775
Baseline Incidence (per 100,000) (as per Table 4-5	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000007	1.000015	1.000004
Attributable fraction (AF)	6.8E-06	1.5E-05	4.2E-06
Increased number of cases in population	0.000024	0.000004	0.000005
Risk	3.7E-08	6.2E-09	5.1E-08
Individual subrubs within LGA			
Hurstville	•		
Total Population in study area	96	96	96
% population in assessment age-group	: 100%	100%	15%
total change	0.25	0.25	0.25
Population weighted $\Delta x (\mu g/m^3)$	0.00260417	0.00260417	0.00260417
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000005	1.000011	1.000003
Attributable fraction (AF)	4.9E-06	1.1E-05	3.0E-06
Increased number of cases in population	. 0.0000	0.0000	0.00000
Risk	2.6E-08	4.4E-09	3.6E-08
South Hurstville - Blakehurst			
Total Population in study area	561	561	561
% population in assessment age-group	: 100%	100%	15%
total change	2.13	2.13	2.13
Population weighted Δx (µg/m <sup>3</sup> ):	0.00379679	0.00379679	0.00379679
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000007	1.000016	1.000004
Attributable fraction (AF)	7.1E-06	1.6E-05	4.4E-06
Increased number of cases in population	0.0000	0.0000	0.00000
Risk	3.8E-08	6.5E-09	5.3E-08
Total population incidence - All Suburbs	-0.0026	-0.000451	-0.00058

# Assessment of Increased Incidence - NO2 F6 Extension: 2036

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Strathfield - Burwood - Ashfield LGA			
Total Population in study areas	20160	20160	20160
% population in assessment age-group	100%	100%	19%
total change	-12.75	-12.75	-12.75
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00063244	-0.00063244	-0.00063244
Baseline Incidence (per 100,000) (as per Table 4-5)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	0.999999	0.999997	0.999999
Attributable fraction (AF):	-1.2E-06	-2.7E-06	-7.3E-07
Pick-	-0.0001	-0.00002	-0.00003
	-3.7 -09	-1.12-03	-0.0L-08
Ashfield			
Total Population in study area	1512	1512	1512
% population in assessment age-group	100%	100%	19%
total change	1.87	1.87	1.87
Population weighted $\Delta x (\mu g/m^3)$ :	0.00123677	0.00123677	0.00123677
Baseline Incidence (per 100.000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	1.000002	1.000005	1.000001
Attributable fraction (AF):	2.3E-06	5.3E-06	1.4E-06
Increased number of cases in population	0.0000	0.0000	0.0000
Risk:	1.1E-08	2.1E-09	1.7E-08
Canterbury (North) - Ashbury			
Total Population in study areas	7179	7179	7179
% population in assessment age-group	100%	100%	19%
total change	-8.22	-8.22	-8.22
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00114501	-0.00114501	-0.00114501
Baseline Incidence (per 100,000) (as per I able 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:     Attributable fraction (AE)	0.999998	0.999995	0.999999
Attributable iraction (AF):	-2.2E-06	-4.9E-06	-1.3E-00
Rick-	-0.0001	-1.9E-09	-1 6E-08
Dulwich Hill - Lewisham	-1.02-00	-1.92-03	-1.0L-00
Total Population in study area	11400	11400	11400
% population in assessment age-group	100%	100%	19%
total change	-6.32	-6.32	-6.32
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00055439	-0.00055439	-0.00055439
Baseline Incidence (per 100,000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	0.999999	0.999998	0.999999
Attributable fraction (AF):	-1.0E-06	-2.4E-06	-6.4E-07
Increased number of cases in population	-0.0001	0.0000	0.0000
Risk:	-5.0E-09	-9.4E-10	-7.7E-09
Haberfield - Summer Hill			
Total Population in study areas	69	69	69
% population in assessment age-group	100%	100%	19%
total change	-0.08	-0.08	-0.08
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00115942	-0.00115942	-0.00115942
Baseline Incidence (per 100,000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	0.999998	0.999995	0.999999
Attributable traction (AF):	-2.2E-06	-4.9E-06	-1.3E-06
increased number of cases in population	0.0000	0.0000	0.0000
Risk:	-1.0E-08	-2.0E-09	-1.6E-08
	1	1	1

	-		
Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Sydney Inner City LGA			
Total Population in study area:	29695	29695	29695
% population in assessment age-group	100%	100%	6%
total change	-81.5	-81.5	-81.5
Population weighted Δx ( $\mu$ g/m <sup>3</sup> ):	-0.00274457	-0.00274457	-0.0027445
Baseline Incidence (per 100,000) (as per Table 4-5)	508	40	1209
Baseline Incidence (per person)	0.00508	0.00040	0.01209
Relative Risk:	0.999995	0.999988	0.999997
Attributable fraction (AF):	-5.2E-06	-1.2E-05	-3.2E-06
Increased number of cases in population:	-0.001	-0.00014	-0.000
Risk:	-2.6E-08	-4.7E-09	-3.8E-08
Individual subrubs within LGA			
Erskinville - Alexandria			
I otal Population in study area:	11411	11411	1141
% population in assessment age-group	100%	100%	6%
	-14.9	-14.9	-14.
Population weighted $\Delta x$ (µg/m <sup>2</sup> ):	-0.00130576	-0.00130576	-0.00130570
Baseline Incidence (per 100,000) (as per 1 able 4.4)	508	40	120
Baseline Incidence (per person)	0.00508	0.00040	0.01209
Relative Risk:	0.999998	0.999994	0.999998
All Indiable Traction (AF).	-2.5E-06	-3.0E-00	-1.5E-00
Rick-	-0.0001	-0.00003	-0.000012
Newtown - Camperdown - Darlington	-1.2L-00	-2.2L-09	-1.02-00
Total Population in study area	5225	5225	522!
% population in assessment age-group	100%	100%	6%
total change	-8.97	-8.97	-897%
Population weighted $\Lambda x (ug/m^3)$ :	-0.00171675	-0.00171675	-0.0017167
Baseline Incidence (per 100.000) (as per Table 4.4)	508	40	1209
Baseline Incidence (per person)	0.00508	0 00040	0.01209
Relative Risk:	0.999997	0.999993	0.999998
Attributable fraction (AF):	-3.2E-06	-7.3E-06	-2.0E-06
Increased number of cases in population:	0.000	0.000	0.0000
Risk:	-1.6E-08	-2.9E-09	-2.4E-08
Waterloo - Beaconsfield			
Total Population in study area:	13059	13059	13059
% population in assessment age-group:	100%	100%	6%
total change	-57.64	-57.64	-57.64
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00441381	-0.00441381	-0.0044138
Baseline Incidence (per 100,000) (as per Table 4.4)	508	40	1209
Baseline Incidence (per person)	0.00508	0.00040	0.01209
Relative Risk:	0.999992	0.999981	0.999999
Attributable fraction (AF):	-8.3E-06	-1.9E-05	-5.1E-06
Increased number of cases in population:	-0.001	0.000	0.000
Risk:	-4.2E-08	-7.5E-09	-6.1E-08
		1	1

Health Endpoint:	Mortality - All	Mortality -	Morbidity -
	Causes, Short-	Respiratory,	Asthma ED
	term	Short-term	Admissions,
			Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Marrickville - Sydenham - Petersham LGA			
Total Population in study area:	35722	35722	35722
% population in assessment age-group:	100%	100%	14%
total change	-133.0	-132.97	-132.97
Population weighted $\Delta x$ (µg/m <sup>3</sup> ):	-0.00372236	-0.00372236	-0.00372236
Baseline Incidence (per 100,000) (as per Table 4-5)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999993	0.999984	0.999996
Attributable fraction (AF):	-7.0E-06	-1.6E-05	-4.3E-06
Increased number of cases in population:	-0.0013	-0.00023	-0.00026
Risk:	-3.7E-08	-6.3E-09	-5.2E-08
Individual subrubs within LGA			
Marrickville	050.40	050.40	050.44
I otal Population in study area:	25842	25842	25842
% population in assessment age-group	100%	100%	14%
	-45.0	-45.0	-45.0
Population weighted $\Delta x$ (µg/m):	-0.00176457	-0.00176457	-0.00176457
Baseline Incidence (per 100,000) (as per 1 able 4.4)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999997	0.999992	0.999998
All indiable fraction (AF).	-3.3E-06	-7.5E-00	-2.0E-00
Dicket	-1 8E-08	-0.0001	-0.0000
Petersham - Stanmore	-1.0L-00	-3.02-03	-2.3L-00
Total Population in study area	2051	2051	205
% population in assessment age-group	100%	100%	14%
total change	-4 95	-4 95	-4 95
Population weighted Ax (ug/m <sup>3</sup> ):	-0.00241346	-0.00241346	-0.00241346
Baseline Incidence (per 100 000) (as per Table 4.4)	-0.00241340	-0.00241340	-0.00241340
Baseline Incidence (per 100;000) (ds per 10bie 4.4)	0.00534	0,00040	0.0120
Relative Risk:	0.00004	0.00040	0.01203
Attributable fraction (AF):	-4 5E-06	-1.0E-05	-2 8E-06
Increased number of cases in population:	0.000	0.0000	-0.00001
Risk:	-2.4E-08	-4.1E-09	-3.4E-08
Sydenham - Tempe - St Peters			
Total Population in study area:	7829	7829	7829
% population in assessment age-group	100%	100%	14%
total change	-82.4	-82.4	-82.4
Population weighted $\Delta x (\mu q/m^3)$ :	-0.01052497	-0.01052497	-0.01052497
Baseline Incidence (per 100,000) (as per Table 4.4)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999980	0.999955	0.999988
Attributable fraction (AF):	-2.0E-05	-4.5E-05	-1.2E-05
Increased number of cases in population:	-0.001	-0.0001	-0.0002
Risk:	-1.1E-07	-1.8E-08	-1.5E-07

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Canterbury LGA			
Total Population in study area:	12562	12562	12562
% population in assessment age-group:	100%	100%	19%
total change	-19.80	-19.8	-19.8
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00157618	-0.00157618	-0.00157618
Baseline Incidence (per 100,000) (as per Table 4-5)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	0.999997	0.999993	0.999998
Attributable fraction (AF):	-3.0E-06	-6.7E-06	-1.8E-06
Increased number of cases in population:	0.000	-0.00003	-0.0001
Risk:	-1.5E-08	-2.7E-09	-2.2E-08
Individual subrubs within LGA			
Canterbury (South) - Campsie			
Total Population in study area:	149	149	149
% population in assessment age-group:	100%	100%	19%
total change	0.17	0.17	0.17
Population weighted $\Delta x (\mu g/m^3)$ :	0.00114094	0.00114094	0.00114094
Baseline Incidence (per 100,000) (as per Table 4.4)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	1.000002	1.000005	1.000001
Attributable fraction (AF):	2.1E-06	4.9E-06	1.3E-06
Increased number of cases in population:	0.0000	0.0000	0.0000
Risk:	1.1E-08	1.9E-09	1.6E-08
Kingsgrove (North) - Earlwood			
Total Population in study area:	12413	12413	12413
% population in assessment age-group:	100%	100%	19%
total change	-19.9	-19.9	-19.9
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00160316	-0.00160316	-0.00160316
Baseline Incidence (per 100,000) (as per Table 4.4)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	0.999997	0.999993	0.999998
Attributable fraction (AF):	-3.0E-06	-6.8E-06	-1.8E-06
Increased number of cases in population:	-0.0002	0.0000	-0.0001
Risk:	-1.5E-08	-2.7E-09	-2.2E-08
l	I		I I

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Botany LGA			
Total Population in study area:	31386	31386	31386
% population in assessment age-group	100%	100%	16%
total change	-418.2	-418.2	-418.2
Population weighted $\Delta x (\mu g/m^3)$ :	-0.01332441	-0.01332441	-0.01332441
Baseline Incidence (per 100,000) (as per Table 4-5)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
	0.999975	0.999943	0.999985
All Durable Haction (AF).	-2.3E-03	-0.00711	-0.000913
Risk	-0.004118 -1.3E-07	-0.000711	-0.000913
Individual subrubs within LGA	1.02 07	2.02 00	1.52 07
Botany			
Total Population in study area:	10408	10408	10408
% population in assessment age-group	100%	100%	16%
total change	-113.6	-113.6	-113.6
Population weighted $\Delta x (\mu g/m^3)$ :	-0.01091468	-0.01091468	-0.01091468
Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
Relative Risk:	0.999979	0.999954	0.999987
Attributable fraction (AF):	-2.1E-05	-4.6E-05	-1.3E-05
Increased number of cases in population:	-0.0011	-0.0002	-0.0002
Risk:	-1.1E-07	-1.9E-08	-1.5E-07
Mascot - Eastlakes	20286	20296	20296
I ofal Population in study area:	20286	20286	20286
total change	-301.8	-301.8	-301.8
Population weighted Ax (ug/m <sup>3</sup> ):	-0.01487726	-0.01487726	-0.01487726
Baseline Incidence (per 100 000) (as per Table 4 4)	524	40	1209
Baseline Incidence (per rec, coc) (de per racio 1.1)	0.00524	0 00040	0.01209
Relative Risk:	0.999972	0.999937	0.999983
Attributable fraction (AF):	-2.8E-05	-6.3E-05	-1.7E-05
Increased number of cases in population:	-0.0030	-0.0005	-0.0007
Risk:	-1.5E-07	-2.5E-08	-2.1E-07
Pagewood - Hillsdale - Daceyville			
Total Population in study area:	631	631	631
% population in assessment age-group:	100%	100%	16%
total change	-3.03	-3.03	-3.03
Population weighted $\Delta x$ (µg/m <sup>°</sup> ):	-0.00480190	-0.00480190	-0.00480190
Baseline Incidence (per 100,000) (as per 1 able 4.4)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
	0.999991	0.999980 -2.0E-05	0.999994
Increased number of cases in population	0.0000	0.000	0.000
Risk:	-4.7E-08	-8.2E-09	-6.7E-08
Sydney Airport			
Total Population in study area:	61	61	61
% population in assessment age-group:	100%	100%	16%
total change	0.26	0.26	0.26
Population weighted Δx (µg/m <sup>3</sup> ):	0.00426230	0.00426230	0.00426230
Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209
Baseline Incidence (per person)	0.00524	0.00040	0.01209
Relative Risk:	1.000008	1.000018	1.000005
Attributable fraction (AF):	8.0E-06	1.8E-05	4.9E-06
Increased number of cases in population	0.0000	0.0000	0.0000
RISK:	4.∠⊏-08	1.2E-09	5.9⊑-08
	I	1	I I

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
<u>β (change in effect per 1 μg/m<sup>3</sup> PM) (as per Table 6-16)</u>	0.00188	0.00426	0.00115
Kogaran - Rockdale LGA Total Population in study area:	113547	1135/7	113547
% population in assessment age-group:	100%	100%	15%
total change	29.8	29.8	29.8
Population weighted $\Delta x (\mu g/m^3)$ :	0.00026245	0.00026245	0.00026245
Baseline Incidence (per 100,000) (as per Table 4-5)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Attributable fraction (AF):	1.000000 4 9E-07	1.000001	1.000000 3.0E-07
Increased number of cases in population:	0.000	0.0001	0.0001
Risk:	2.6E-09	4.5E-10	3.6E-09
Individual subrubs within LGA			
Arncliffe - Bardwell Park	04457	01157	04.457
I otal Population in study area:	21457	21457	21457
76 population in assessment age-group.	-161.1	-161.1	-161.1
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00750804	-0.00750804	-0.00750804
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Relative Risk:	0.999986	0.999968	0.999991
Attributable fraction (AF):	-1.4E-05	-3.2E-05	-8.6E-06
Risk:	-0.0016 -7.5E-08	-0.0003 -1 3E-08	-0.00033
Bexley	7.5E 00	1.02 00	1.02 07
Total Population in study area:	20002	20002	20002
% population in assessment age-group:	100%	100%	15%
total change	31.2	31.2	31.2
Population weighted Δx (µg/m <sup>°</sup> ):	0.00155984	0.00155984	0.00155984
Baseline Incluence (per 100,000) (as per 1able 4.4) Baseline Incidence (per person)	0.00535	40	0.01209
Relative Risk:	1.000003	1.00007	1.00002
Attributable fraction (AF):	2.9E-06	6.6E-06	1.8E-06
Increased number of cases in population:	0.0003	0.0001	0.00006
Risk:	1.6E-08	2.7E-09	2.2E-08
Total Population in study area:	2879	2879	2879
% population in assessment age-group:	100%	100%	15%
total change	-4.8	-4.8	-4.8
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00166725	-0.00166725	-0.00166725
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Attributable fraction (AF):	-3 1E-06	-7 1F-06	0.999998 -1 9F-06
Increased number of cases in population:	0.0000	-0.00001	-0.00001
Risk:	-1.7E-08	-2.8E-09	-2.3E-08
Kogarah			
Total Population in study area:	11323	11323	11323
% population in assessment age-group:	100% 231 F	100% 231 F	15% 231 F
Population weighted Ax (ug/m <sup>3</sup> ):	0 02044511	0.02044511	0 02044511
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209
Baseline Incidence (per person)	0.00535	0.00040	0.01209
Relative Risk:	1.000038	1.000087	1.000024
Attributable fraction (AF):	3.8E-05	8.7E-05	2.4E-05
Increased number of cases in population:	0.0023	0.00039	0.00047
Kogarah Bay - Carlton - Allawah	2.1E-07	3.3E-00	2.0E-07
Total Population in study area:	10923	10923	10923
% population in assessment age-group:	100%	100%	15%
total change	75.3	75.3	75.3
Population weighted $\Delta x (\mu g/m^3)$ :	0.00689371	0.00689371	0.00689371
Baseline incidence (per 100,000) (as per 1 able 4.4)	535	40	1209
Baseline Incidence (per person) Relative Rick	0.00535	0.00040	0.01209
Attributable fraction (AF):	1.3E-05	2.9E-05	7.9E-06
Increased number of cases in population:	0.0008	0.00013	0.00015
Risk:	6.9E-08	1.2E-08	9.6E-08
Monterey - Brighton-le-Sands - Kyeemagh	1001-	100:-	1001-
I otal Population in study area:	13915	13915	13915
total change	-67.9	-67.9	-67.9
Population weighted Ax (ug/m <sup>3</sup> ).	-0.00487963	-0.00487963	-0.00487963
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209

	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16	0.00188	0.00426	0.00115
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	0.999991	0.999979	0.999994
Attributable fraction (AF)	-9.2E-06	-2.1E-05	-5.6E-06
Increased number of cases in population	-0.00068	-0.00012	-0.000138
Risk	-4.9E-08	-8.3E-09	-6.8E-08
Rockdale - Banksia			
Total Population in study area	19957	19957	19957
% population in assessment age-group	100%	100%	15%
total change	-124.2	-124.2	-124.2
Population weighted $\Delta x (\mu g/m^3)$	-0.00622338	-0.00622338	-0.00622338
Baseline Incidence (per 100,000) (as per Table 4.4	535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	0.999988	0.999973	0.999993
Attributable fraction (AF)	-1.2E-05	-2.7E-05	-7.2E-06
Increased number of cases in population	-0.00125	-0.00021	-0.000252
Risk	-6.3E-08	-1.1E-08	-8.7E-08
Sans Souci - Ramsgate	1		
Total Population in study area	13091	13091	13091
% population in assessment age-group	100%	100%	15%
total change	49.8	49.8	49.8
Population weighted $\Delta x (\mu g/m^3)$	0.00380414	0.00380414	0.00380414
Baseline Incidence (per 100,000) (as per Table 4.4	535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000007	1.000016	1.000004
Attributable fraction (AF)	7.2E-06	1.6E-05	4.4E-06
Increased number of cases in population	0.00050	0.00008	0.000101
	3.8E-08	6.5E-09	5.3E-08

Health Endpoint	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16	0.00188	0.00426	0.00115
Hurstville LGA			
Total Population in study area	: 657	657	657
% population in assessment age-group	: 100%	100%	15%
total change	e 3.1	3.1	3.1
Population weighted $\Delta x (\mu g/m^3)$	0.00471842	0.00471842	0.00471842
Baseline Incidence (per 100,000) (as per Table 4-5	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000009	1.000020	1.00000
Attributable fraction (AF)	8.9E-06	2.0E-05	5.4E-06
Increased number of cases in population	: 0.000031	0.000005	0.00000
Risk	: 4.7E-08	8.0E-09	6.6E-08
Individual subrubs within LGA			
Hurstville	2		
Total Population in study area	: 96	96	96
% population in assessment age-group	: 100%	100%	15%
total change	0.45	0.45	0.45
Population weighted $\Delta x (\mu g/m^3)$	0.00468750	0.00468750	0.00468750
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000009	1.000020	1.000005
Attributable fraction (AF)	: 8.8E-06	2.0E-05	5.4E-06
Increased number of cases in population	: 0.0000	0.0000	0.00000
Risk	4.7E-08	8.0E-09	6.5E-08
South Hurstville - Blakehurs	t		
Total Population in study area	: 561	561	56
% population in assessment age-group	: 100%	100%	15%
total change	2.62	2.62	2.62
Population weighted $\Delta x (\mu g/m^3)$	0.00467023	0.00467023	0.00467023
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	1.000009	1.000020	1.00000
Attributable fraction (AF)	8.8E-06	2.0E-05	5.4E-06
Increased number of cases in population	: 0.0000	0.0000	0.0000
Risk	4.7E-08	7.9E-09	6.5E-08
Total population incidence - All Suburbs	-0.0062	-0.001075	-0.0012

# Assessment of Increased Incidence - NO2 F6 Extension: 2036 Cumulative

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Strathfield - Burwood - Ashfield LGA			
Total Population in study area:	20160	20160	20160
% population in assessment age-group:	100%	100%	19%
total change	-6.83	-6.83	-6.83
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00033879	-0.00033879	-0.00033879
Baseline Incidence (per 100,000) (as per Table 4-5)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	0.999999	0.999999	1.000000
Attributable fraction (AF):	-6.4E-07	-1.4E-06	-3.9E-07
Rick	-0.0001	-0.00001 -5.8E-10	-0.00002
Individual subrubs within LGA	-3.02-03	-5.02-10	-4.7 2-03
Ashfield			
Total Population in study area:	1512	1512	1512
% population in assessment age-group	100%	100%	19%
total change	-0.14	-0.14	-0.14
Population weighted $\Lambda x (ug/m^3)$ :	-0.00009259	-0.00009259	-0.00009259
Baseline Incidence (per 100.000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	1.000000	1.000000	1.000000
Attributable fraction (AF):	-1.7E-07	-3.9E-07	-1.1E-07
Increased number of cases in population:	0.0000	0.0000	0.0000
Risk:	-8.3E-10	-1.6E-10	-1.3E-09
Canterbury (North) - Ashbury			
Total Population in study area:	7179	7179	7179
% population in assessment age-group	100%	100%	19%
total change	-4.9	-4.9	-4.9
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00068255	-0.00068255	-0.00068255
Baseline Incidence (per 100,000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	0.999999	0.999997	0.999999
Attributable fraction (AF):	-1.3E-06	-2.9E-06	-7.8E-07
Increased number of cases in population	0.0000	0.0000	0.0000
RISK. Dulwich Hill - Lewisham	-0.1E-09	-1.2E-09	-9.5E-08
Total Population in study area:	11400	11400	11400
% population in assessment age-group	100%	100%	19%
total change	-1.87	-1.87	-1.87
Population weighted Ax (ug/m <sup>3</sup> ):	-0.00016404	-0 00016404	-0.00016404
Baseline Incidence (per 100.000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	1.000000	0.999999	1.00000
Attributable fraction (AF):	-3.1E-07	-7.0E-07	-1.9E-07
Increased number of cases in population:	0.0000	0.0000	0.0000
Risk:	-1.5E-09	-2.8E-10	-2.3E-09
Haberfield - Summer Hill			
Total Population in study area:	69	69	69
% population in assessment age-group:	100%	100%	19%
total change	0.11	0.11	0.11
Population weighted $\Delta x (\mu g/m^3)$ :	0.00159420	0.00159420	0.00159420
Baseline Incidence (per 100,000) (as per Table 4.4)	477	40	1209
Baseline Incidence (per person)	0.00477	0.00040	0.01209
Relative Risk:	1.000003	1.000007	1.00002
Attributable fraction (AF):	3.0E-06	6.8E-06	1.8E-06
Increased number of cases in population:	0.0000	0.0000	0.0000
Risk:	1.4E-08	2.7E-09	2.2E-08
	1	1	1

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term		
Age Group:	All ages	All ages	1-14 years		
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115		
Sydney Inner City LGA					
Total Population in study area	29695	29695	29695		
% population in assessment age-group	100%	100%	6%		
total change	-66.4	-66.4	-66.4		
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00223607	-0.00223607	-0.00223607		
Baseline Incidence (per 100,000) (as per Table 4-5	508	40	1209		
Baseline Incidence (per person)	0.00508	0.00040	0.01209		
Relative Risk:	0.999996	0.999990	0.999997		
Attributable fraction (AF):	-4.2E-06	-9.5E-06	-2.6E-06		
Increased number of cases in population	-0.001	-0.00011	-0.0002		
Risk:	-2.1E-08	-3.8E-09	-3.1E-08		
Individual subrubs within LGA					
Erskinvine - Alexandria	11111	11111	1111		
	11411	11411	1141		
% population in assessment age-group	-15	-15	-16		
Benulation weighted Av (ug/m <sup>3</sup> )	0.00121452	0.00121452	0.0012145		
Population weighted ΔX (μg/m). Baseline Incidence (per 100,000) (as per Table 4.4)	-0.00131432	-0.00131432	-0.00131432		
Baseline Incidence (per 100,000) (as per 1able 4.4	0.00508	40	0.0120		
Baseline incidence (per person	0.00308	0.00040	0.01203		
Attributable fraction (AF)	-2.5E-06	-5 6E-06	-1 5E-06		
Increased number of cases in population	-0.0001	-0.00003	-0.000012		
Risk:	-1.3E-08	-2.2E-09	-1.8E-08		
Newtown - Camperdown - Darlington					
Total Population in study area	5225	5225	5225		
% population in assessment age-group	100%	100%	6%		
total change	10.3	10.3	1030%		
Population weighted $\Delta x (\mu g/m^3)$ :	0.00197129	0.00197129	0.00197129		
Baseline Incidence (per 100,000) (as per Table 4.4	508	40	1209		
Baseline Incidence (per person	0.00508	0.00040	0.01209		
Relative Risk:	1.000004	1.00008	1.000002		
Attributable fraction (AF):	3.7E-06	8.4E-06	2.3E-06		
Increased number of cases in population	0.000	0.000	0.0000		
Risk	1.9E-08	3.4E-09	2.7E-08		
Waterloo - Beaconsfield					
I otal Population in study area	13059	13059	13059		
% population in assessment age-group	100%	100%	6%		
total change	-61.8	-61.8	-61.8		
Population weighted $\Delta x$ (µg/m <sup>°</sup> ):	-0.00473237	-0.00473237	-0.00473237		
Baseline Incidence (per 100,000) (as per Table 4.4	508	40	1209		
Baseline Incidence (per person	0.00508	0.00040	0.01209		
Relative Risk:	0.999991	0.999980	0.999995		
Attributable traction (AF):	-8.9E-06	-2.0E-05	-5.4E-06		
Increased number of cases in population	-0.001		-0.000		
RISK.	-4.3⊑-00	-0.0E-09	-0.02-00		
	-				

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115
Marrickville - Sydenham - Petersham LGA			
Total Population in study area:	35722	35722	35722
% population in assessment age-group	100%	100%	14%
total change	-18.0	-18	-18
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00050389	-0.00050389	-0.00050389
Baseline Incidence (per 100,000) (as per Table 4-5)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999999	0.999998	0.999999
Attributable fraction (AF):	-9.5E-07	-2.1E-06	-5.8E-07
Increased number of cases in population:	-0.0002	-0.00003	-0.00004
Risk:	-5.1E-09	-8.6E-10	-7.0E-09
Individual subrubs within LGA			
Marrickville	050.40	050.40	050.44
I otal Population in study area:	25842	25842	25842
% population in assessment age-group	100%	100%	14%
	9.7	9.7	9.7
Population weighted $\Delta x$ (µg/m <sup>2</sup> ):	0.00037536	0.00037536	0.00037536
Baseline Incidence (per 100,000) (as per 1 able 4.4)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Attributoble fraction (AE)	7.15.07	1.00002	1.00000
Increased number of cases in population	0.000	0.000	4.32-07
Risk:	3.8E-09	6.4E-10	5.2E-00
Petersham - Stanmore	0.02 00	0.42 10	0.22 00
Total Population in study area:	2051	2051	2051
% population in assessment age-group	100%	100%	14%
total change	0.028	0.028	0.028
Population weighted $\Delta x (\mu g/m^3)$ :	0.00001365	0.00001365	0.00001365
Baseline Incidence (per 100.000) (as per Table 4.4)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	1.000000	1.000000	1.000000
Attributable fraction (AF):	2.6E-08	5.8E-08	1.6E-08
Increased number of cases in population	0.000	0.0000	0.00000
Risk:	1.4E-10	2.3E-11	1.9E-10
Sydenham - Tempe - St Peters			
Total Population in study area:	7829	7829	7829
% population in assessment age-group	100%	100%	14%
total change	-27.8	-27.8	-27.8
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00355090	-0.00355090	-0.00355090
Baseline Incidence (per 100,000) (as per Table 4.4)	534	40	1209
Baseline Incidence (per person)	0.00534	0.00040	0.01209
Relative Risk:	0.999993	0.999985	0.999996
Attributable fraction (AF):	-6.7E-06	-1.5E-05	-4.1E-06
Increased number of cases in population:	0.000	0.0000	-0.0001
Risk:	-3.6E-08	-6.0E-09	-4.9E-08
	1	1	1

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions,
			Short-term
Age Group:	All ages	All ages	1-14 years
<u>β (change in effect per 1 μg/m<sup>3</sup> PM) (as per Table 6-16)</u>	0.00188	0.00426	0.00115
Canterbury LGA			
Total Population in study areas	12562	12562	12562
% population in assessment age-group	100%	100%	19%
total change	-4.70	-4.7	-4.7
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00037414	-0.00037414	-0.00037414
Baseline Incidence (per 100,000) (as per Table 4-5)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	0.999999	0.999998	1.000000
Attributable fraction (AF):	-7.0E-07	-1.6E-06	-4.3E-07
Increased number of cases in population	0.000	-0.00001	0.0000
Risk:	-3.5E-09	-6.4E-10	-5.2E-09
Individual subrubs within LGA			
Canterbury (South) - Campsie			
Total Population in study areas	149	149	149
% population in assessment age-group	100%	100%	19%
total change	0.37	0.37	0.37
Population weighted Δx (µg/m³):	0.00248322	0.00248322	0.00248322
Baseline Incidence (per 100,000) (as per Table 4.4)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	1.000005	1.000011	1.000003
Attributable fraction (AF):	4.7E-06	1.1E-05	2.9E-06
Increased number of cases in population	0.0000	0.0000	0.0000
Risk:	2.3E-08	4.2E-09	3.5E-08
Kingsgrove (North) - Earlwood			
Total Population in study area:	12413	12413	12413
% population in assessment age-group	100%	100%	19%
total change	-5.1	-5.1	-5.1
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00041086	-0.00041086	-0.00041086
Baseline Incidence (per 100,000) (as per Table 4.4)	491	40	1209
Baseline Incidence (per person)	0.00491	0.00040	0.01209
Relative Risk:	0.999999	0.999998	1.000000
Attributable fraction (AF):	-7.7E-07	-1.8E-06	-4.7E-07
Increased number of cases in population	0.0000	0.0000	0.0000
Risk:	-3.8E-09	-7.0E-10	-5.7E-09
l	I		

Age Group:         All ages         All ages         1.14 years           β (change in effect per 1 μol/m) PM (Jaso per Table 5-10)         0.0018         0.00426         0.00115           Change in effect per 1 μol/m) In study area         31386         31386         31386           % population in assessment age-group         100%         100%         100%           Baseline Incidence (per person         0.00524         0.00446         0.0105804           Baseline Incidence (per person         0.00524         0.00440         0.0105804           Relative Risk         0.999985         0.999985         0.999985           Attributable fraction (PF)         -2.0-5         4.45-05         -1.25-05           Hardivalai subrubs within LGA         0.003276         -0.000565         -0.000727           Risk         -1.0-E-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         -0.010584         -0.000785         -0.000785           Baseline Incidence (per person         0.00524         0.00046         1690           Weight Depulation in study area         10408         10408         10406           Baseline Incidence (per person         0.00524         0.00044         0.0111684           Maread         Baseline Incidence (per pers	Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term	
B (change in effect per 1 µg/m <sup>2</sup> PM) (as per Table 6-16)         0.00178         0.00426         0.00171           Total Population in subsessment age-group.         100%         1109%         1101%         1109%         1101%         1109%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1101%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%         1111%	Age Group:	All ages	All ages	1-14 years	
Botary LGA         Heat           A Total Population in subgrave.         31386         31386         31386           % population in assessment age-group.         100%         100%         100%           Baseline Incidence (per 100.000) (as per Table 4-5         522         0         0.01058904         -0.01058904         -0.01058904         0.000548         0.00058905         0.999985         0.999985         0.999985         0.999985         0.999985         0.999985         0.999985         0.999985         0.999985         0.900565         -1.2E 05         -4.5E-05         -1.2E 05         -4.5E-05         -1.2E 05         -4.5E-05         -1.2E 05         -1.6E-06         -1.5E-07         -1.6E-08         -1.0E-07         -1.6E-08         -1.0E-07         -1.6E-08         -1.0E-07         -1.6E-08         -1.0E-07         -1.6E-08         -1.0E-07         -1.6E-08         -1.0E-07         -1.6E-08         -0.00724         -0.000746         -0.00724         -0.00076         -0.00724         -0.0011005         -0.0011005         -1.0E-08         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.011	β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115	
Total Population in study area:         31386         31386         31386           % population in assessment age-group         100%         100%         100%           Baseline Incidence (per parson)         0.0058904         -0.01058904         -0.01058904           Baseline Incidence (per parson)         0.00524         0.00400         0.01292           Relative Risk         0.99980         0.99985         0.99986         -0.000565           Increased number of cases in population         -0.00376         -0.000665         -0.000726           Risk         1.0E-07         -1.8E-08         -1.8E-07           Individual subrubs within LGA         -0.0111084         -0.0111084         -0.0111084           % population in study area:         10408         10408         10408           % population in sakessment age-group         100%         100%         10468           % population in sakessment age-group         -0.01110884         -0.01110884         -0.01110884           Baseline Incidence (per pron)         0.00524         0.00040         0.01205           Relative Risk         0.999975         0.999975         0.999976         0.999976           Atributable fraction (AF)         2.21E-05         -4.7E-05         -1.3E-06         -1.5E-07	Botany LGA				
% population in assessment age-group:         100%         100%         16%           Baseline Incidence (per 100.000) (as per Table 4-5)         524         40         1205           Baseline Incidence (per person)         0.00524         0.0009865         0.099986           Attributable fraction (AF):         -2.06-05         -4.5E-05         -1.2E-05           Increased number of cases in population         -0.002766         -0.0005865         -0.000726           Increased number of cases in population         -0.002766         -0.0005865         -0.00726           Individual subrubs within LGA         -1.6E-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         -1.06-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         -0.003726         -0.000586         -0.00726           Statistic Risk         0.100%         100%         106%         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.01010         -0.0002         Risk         -1.16-07         -1.6E-05         -1.6E-05	Total Population in study areas	31386	31386	31386	
bital change         -332.5 <td< td=""><td>% population in assessment age-group</td><td>100%</td><td>100%</td><td>16%</td></td<>	% population in assessment age-group	100%	100%	16%	
Propulation weighted XX (Ig/m):         -0.01059804         -0.01059804         -0.01059804           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk         0.999980         0.999955         0.999985           Attributable fraction (AF):         -2.0E-05         -4.5E-06         -1.2E-05           Increased number of cases in population:         -0.00276         -0.000565         -0.00072           Risk         -0.1040         10408         10408         10408           Total Population in study area:         10408         10408         10408           % population in assessment age-group:         100%         100%         1078           Baseline Incidence (per 100,000) (as per Table 4.4         524         40         1205           Baseline Incidence (per person)         0.00524         0.00002         0.00002           Relative Risk         0.999979         0.999953         0.999957           Attributable fraction (AF):         -2.1E-05         -4.5E-05         -1.3E-05           Increased number of cases in population:         -0.011         -0.0002         0.00002           Risk         -1.1E-0	Developing total change	-332.6	-332.63	-332.03	
Description         D00,000 (as per Table 4-2)         0.00224         0.00040         0.01205           Relative Risk         0.999980         0.999985         0.999986         0.999985         0.999986           Attributable fraction (AF):         -0.003276         -0.0003276         0.000022         Risk:         -1.0E-07         -1.8E-08         -1.5E-07           Indrivatual subrubs within LGA         0.00072         Risk:         -1.15E-07         -1.8E-08         -1.5E-07           Indrivatual subrubs within LGA         0.00076         1.00%         169%         -1.15E         -1.15E           Control (C) (as per Table 4-4)         5.24         40         -1.15E	Population weighted $\Delta X$ (µg/m <sup>-</sup> ): Receive Incidence (per 100,000) (ce per Toble 4.5)	-0.01059804	-0.01059804	-0.01059804	
Lasenie inductine (per person)         0.00047         0.001205           Relative Risk         0.099985         0.999985         0.999985         0.099985           Attributable fraction (AF):         -2.0E-05         -4.5E-05         -1.2E-05           Risk:         -1.0E-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         10408         10408         10408           Total Population in assessment age-group.         100%         100%         169%           Population in assessment age-group.         100%         100%         100%           Baseline Incidence (per 100,000) (as per Table 4.4)         524         -40         1205           Baseline Incidence (per 100,000) (as per Table 4.4)         524         -40         1205           Baseline Incidence (per 100,000) (as per Table 4.4)         524         -40         1205           Baseline Incidence (per person)         0.00524         0.00004         0.00120           Relative Risk:         0.999970         0.999963         0.999987           Increased number of cases in population.         -0.0011         -0.0002         -0.0002           Relative Risk:         0.1167         -1.9E-08         -1.5E-07           Total Population in study area:         20286	Baseline Incidence (per 100,000) (as per 1able 4-5)	0.00524	40	0.01209	
Attributable fraction (AF):         -2.02.00         -0.003276         -0.000256           Increased number of cases in population         -0.003276         -0.000256         -0.000726           Risk:         -1.0E-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         -0.000276         -0.000565         -0.000726           Mathematic Composition         -1.0E-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         -0.01068         -0.01068         -0.01068           % population in assessment age-group         100%         100%         1640         -1.056           Population in assessment age-group         -0.01110684         -0.01110684         -0.01110684         -0.01110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.0110684         -0.01020         -0.0004         0.01020         -0.0004         0.01020         -0.0004         -0.00120         Relative Risk         0.999975         0.999953         0.999956         -0.999953         0.999956         -0.999956         -0.999956         -0.99056         -0.99056         -0.99056         -0.99056         -0.99056         -0.99014107	Baseline incidence (per person) Relative Risk:	0.00524	0.00040	0.01209	
Increased number of cases in population:         -0.003276         -0.000565         -0.000726           Risk:         -1.0E-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         Botany	Attributable fraction (AF):	-2.0E-05	-4.5E-05	-1.2E-05	
Risk         1.0E-07         -1.8E-08         -1.5E-07           Individual subrubs within LGA         Botany	Increased number of cases in population	-0.003276	-0.000565	-0.000726	
Botany         Botany           Total Population in study area:         10408         10408           % population in sesessment age-group:         100%         100%         115.6           % population weighted Δx (µg/m):         -0.01110684         -0.01110684         -0.01110684           Baseline Incidence (µer person)         0.00524         0.00040         0.01205           Baseline Incidence (µer person)         0.00524         0.00040         0.01205           Relative Risk:         0.909997         0.909983         0.999987           0.00002         -0.0001         -0.0002         -0.0002           0.00002         -0.0002         -0.0002         -0.0002           0.00002         -0.0002         -0.0002         -0.0002           0.0002         -0.0002         -0.0002         -0.0002           0.0002         -0.0002         -0.0002         -0.0002           0.0002         -0.0002         -0.0002         -0.0002           0.0003         -0.01042098         -0.01042098         -0.01042098           0.00040         -0.0021         -0.0004         -0.0021           0.00040         -0.0021         -0.0004         -0.0021           Baseline Incidence (per person)         -0	Risk:	-1.0E-07	-1.8E-08	-1.5E-07	
Botany         Head           Total Population in study area:         10408         10408         10408           % population in assessment age-group.         100%         110%         1166           Population in assessment age-group.         0.01110684         -0.01110684         -0.01110684           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Baseline Incidence (per person)         0.00524         0.00040         0.01005           Relative Risk:         0.00149         0.00020         -0.0003           Relative Risk:         -1.1E-07         -1.9E-08         -1.5E-07           Mascot - Eastlakes         -         -         -1.0000         -0.0002           Colored Cases in population in study area:         20286         202144         -20114         -2114	Individual subrubs within LGA				
Total Population in study area:         10408         10408           % population in assessment age-group.         100%         100%           total change         -115.6         -115.6           Population weighted ∆x (µg/m):         -0.01110684         -0.01110684           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999973         0.999953         0.999987           Attributable fraction (AF):         -2.1E-05         -4.7E-05         -1.3E-05           Increased number of cases in population         -0.0011         -0.0002         -0.0002           Risk:         -1.1E-05         -1.7E-05         -1.5E-07           Mascot - Eastlakes         -1.1E-05         -1.4E-05         -1.5E-07           Mascot - Eastlakes         -1.1E-06         -1.0E-05         -1.4E-05         -1.4E-06           % population in study area:         20286         20286         20286         20286           % population in study area:         0.01042098         -0.01042098         -0.01042098         -0.01042098         -0.01042098         -0.01042098         -0.01042098         -0.01042098         -0.01042098	Botany	r			
% population in assessment age-group         100%         100%         16%           total change         -115.6         -115.6         -115.6           Population weighted Δx (µg/m <sup>3</sup> ):         -0.01110684         -0.01110684         -0.01110684           Baseline Incidence (per person)         0.00524         0.0004         0.00202           Relative Risk:         0.999979         0.999953         0.999987           Attributable fraction (AP):         -2.1E-05         -4.7E-06         -1.3E-05           Increased number of cases in population         -0.0011         -0.0002         -0.0002           Mascot         Eastlakes         -         -         -           Total Population in study area:         20286         20286         20286           % population weighted Δx (µg/m <sup>3</sup> ):         -0.01042098         -0.01042098         -0.01042098           Baseline Incidence (per person)         0.00524         0.00040         0.01202           Baseline Incidence (per per person)         0.00524         0.00040         0.00120           Relative Risk:         0.999980         0.999986         0.999986         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases in pop	Total Population in study area	10408	10408	10408	
total change         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -115.6         -1110684           Baseline Incidence (per person)         0.00124         0.00040         0.01202           Relative Risk:         0.999979         0.999953         0.999987           Attributable fraction (AF):         -2.1E-05         -4.7E-06         -1.3E-05           Increased number of cases in population         -0.0011         -0.0002         -0.0002           Risk:         -11.6         -1.6         -1.6         -1.6           Mascot - Eastlakes         -         -         -         -         -           Total Population in assessment age-group.         100%         100%         16%         -0.01042098         -0.0004         -0.0021         -0.0004         -0.0021 <td>% population in assessment age-group</td> <td>100%</td> <td>100%</td> <td>16%</td>	% population in assessment age-group	100%	100%	16%	
Population weighted Xx (µg/m):         -0.01110684         -0.01110684         -0.01110684           Baseline Incidence (per parson)         0.00524         0.00040         0.01205           Baseline Incidence (per parson)         0.00524         0.00040         0.01205           Relative Risk:         0.999979         0.999953         0.999987           Attributable fraction (AF):         2.1E-05         4.7E-05         1.13E-05           Increased number of cases in population         -0.0011         -0.0002         -0.0002           Risk:         -1.1E-07         -1.9E-08         -1.5E-07           Total Population in study area:         20286         20286         20286           % population in assessment age-group         100%         100%         16%           Macot - Eastlakes         -         -         -0.01042098         -0.01042098           Baseline Incidence (per 100.000) (as per Table 4.4)         524         40         1205           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999860         0.999986         0.999986         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases i	total change	-115.6	-115.6	-115.6	
Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999979         0.999953         0.999967           Attributable fraction (AF):         -2.1E-05         -4.7E-05         -1.3E-05           Increased number of cases in population:         -0.0011         -0.0002         -0.0002           Mascot - Eastlakes	Population weighted $\Delta x$ (µg/m <sup>°</sup> ):	-0.01110684	-0.01110684	-0.01110684	
Baseline incidence (per person)         0.00524         0.00040         0.0120s           Relative Risk:         0.999979         0.999953         0.999953         0.999953           Attributable fraction (AF):         -2.1E-05         -4.7E-05         -1.3E-05           Increased number of cases in population         -0.0011         -0.0002         -0.0003           Risk:         -1.1E-07         -1.9E-08         -1.5E-07           Mascot - Eastlakes         -         -         -           Total Population in study area:         20286         20286         20286           % population weighted Δx (µg/m):         -0.01042098         -0.01042098         -0.01042098           Baseline Incidence (per 100,000) (as per Table 4.4)         524         -0.0042008         0.010205           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999980         0.999986         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases in population:         -0.0021         -0.0004         -0.0026           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Total Population in study area:         631	Baseline Incidence (per 100,000) (as per 1 able 4.4)	524	40	1209	
Ketative Kisk.         0.939973         0.0002         0.0002         0.0002         0.0002         0.0006         0.0052         0.01042098         0.01020         0.000524         0.0004         0.00000         0	Baseline Incluence (per person)	0.00524	0.00040	0.01209	
Increased number of cases in population         -0.001         -0.0002         -0.0003           Risk:         -1.1E-07         -1.9E-08         -1.5E-07           Mascot - Eastlakes         -         -         -         -           Total Population in study area:         20286         20286         20286         20286           % population in assessment age-group:         100%         100%         16%           Mascot - Eastlakes         -	Attributable fraction (AF):	-2 1E-05	0.999953 -4 7E-05	0.999987 -1 3E-05	
Risk:         -1.1E-07         -1.9E-08         -1.5E-07           Mascot - Eastlakes         - <td>Increased number of cases in population</td> <td>-0.0011</td> <td>-0.0002</td> <td>-0.0003</td>	Increased number of cases in population	-0.0011	-0.0002	-0.0003	
Mascot - Eastlakes         1           Total Population in study area:         20286         20286         20286           % population in assessment age-group:         100%         100%         16%           Work         total change         -211.4         -211.4         -211.4           Population weighted Δx (µg/m <sup>3</sup> ):         -0.01042098         -0.01042098         -0.01042098           Baseline Incidence (per preson)         0.00524         0.00040         0.01202           Baseline Incidence (per person)         0.00524         0.00040         0.01202           Relative Risk:         0.999980         0.999956         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-07           Increased number of cases in population:         -0.0021         -0.0004         -0.0005           Total Population in study area:         631         631         631           % population in assessment age-group:         100%         100%         16%           % population in assessment age-group:         100%         100%         16%           % population weighted Δx (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per person)         0.00524         0.00040	Risk:	-1.1E-07	-1.9E-08	-1.5E-07	
Total Population in study area:         20286         20286         20286           % population in assessment age-group:         100%         100%         16%           total change         -211.4         -211.4         -211.4         -211.4           Population weighted Δx (µg/m <sup>3</sup> ):         -0.01042098         -0.01042098         -0.01042098           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1205           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999980         0.999956         0.999988           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases in population:         -0.0021         -0.0004         -0.0005           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Total Population in study area:         631         631         631           % population in assessment age-group:         100%         100%         16%           % population weighted Δx (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1205           Baseline Incidence (per per	Mascot - Eastlakes				
% population in assessment age-group:         100%         100%         16%           total change         -211.4         -211.4         -211.4         -211.4           Population weighted $\Delta x$ ( $\mu g/m^3$ ):         -0.01042098         -0.01099         -0.0004         -0.00091         -0.0004         -0.00091         -0.0004         -0.00091         -0.0004         -0.00919176         -0.0919176         -0.0919176         -0.0919176         -0.0919176         -0.0919176         -0.00919176         -0.00919176         -0.00919176         -0.00919176         -0.00919176         -0.00919176         -0.00919176         -0.00919176         -0.00919176         -0.0	Total Population in study areas	20286	20286	20286	
total change         -211.4         -211.4         -211.4           Population weighted $\Delta x$ (µg/m <sup>3</sup> )         -0.01042098         -0.01042098         -0.01042098           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1202           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999980         0.999986         0.999986           Ontreased number of cases in population         -0.0021         -0.0004         -0.0002           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Pagewood - Hillsdale - Daceyville         -         -         -           Total Population in study area:         631         631         631           % population weighted $\Delta x$ (µg/m <sup>3</sup> )         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per ton),000) (as per Table 4.4)         524         40         1202           Baseline Incidence (per person)         0.00524         0.0040         0.01202           Relative Risk:         0.999983         0.999961         0.999986           Outputton weighted $\Delta x$ (µg/m <sup>3</sup> )         -0.00919176         -0.00919176           Baseline Incidence (per person)         0.0.0524         0	% population in assessment age-group	100%	100%	16%	
Population weighted Δx (µg/m <sup>5</sup> ):         -0.01042098         -0.01042098           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1203           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         0.999980         0.999956         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases in population:         -0.0021         -0.0004         -0.0004           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Pagewood - Hillsdale - Daceyville         -         -           Total Population in study area:         631         631         631           % population in assessment age-group:         100%         100%         16%           Utal change         -5.8         -5.8         -5.8           Population weighted Δx (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999983         0.999961         0.999983           Increased number of cases in population         -0.0001         0.0000         0.0000 <tr< td=""><td>total change</td><td>-211.4</td><td>-211.4</td><td>-211.4</td></tr<>	total change	-211.4	-211.4	-211.4	
Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1200           Baseline Incidence (per person)         0.00524         0.00040         0.01206           Relative Risk:         0.999980         0.999956         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases in population:         -0.0021         -0.0004         -0.0000           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Pagewood - Hillsdale - Daceyville         -         -         -           Total Population in study area:         631         631         631           % population in assessment age-group:         100%         100%         16%           total change         -5.8         -5.8         -5.8           Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         0.999983         0.999961         0.999986           Matributable fraction (AF):         -1.7E-05         -3.9E-05         -1.1E-05           Increased number of cases in population         -0.0001         0.0000	Population weighted Δx (µg/m <sup>3</sup> ):	-0.01042098	-0.01042098	-0.01042098	
Baseline Incidence (per person)         0.00524         0.00040         0.01202           Relative Risk:         0.999986         0.999986         0.999986         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases in population         -0.0021         -0.0004         -0.0005           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Pagewood - Hillsdale - Daceyville	Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209	
Kelative Kisk         0.999980         0.999986         0.999986           Attributable fraction (AF):         -2.0E-05         -4.4E-05         -1.2E-05           Increased number of cases in population:         -0.0021         -0.0004         -0.0005           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Pagewood - Hillsdale - Daceyville         -         -         -           Total Population in study area:         631         631         631           % population in assessment age-group:         100%         100%         16%           total change         -5.8         -5.8         -5.8           Population weighted Δx (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1200           Baseline Incidence (per person)         0.00524         0.00040         0.01205           Relative Risk:         0.999983         0.999961         0.999983           Attributable fraction (AF):         -1.7E-05         -3.9E-05         -1.1E-05           Increased number of cases in population:         -0.0001         0.0000         0.00000           Risk:         -9.1E-08         -1.6E-08         -1.3E-07 </td <td>Baseline Incidence (per person)</td> <td>0.00524</td> <td>0.00040</td> <td>0.01209</td>	Baseline Incidence (per person)	0.00524	0.00040	0.01209	
Attributable inaction (Ar):         -2.0E-03         -4.4E-03         -1.2E-03           Increased number of cases in population:         -0.0021         -0.0004         -0.0005           Risk:         -1.0E-07         -1.8E-08         -1.4E-07           Pagewood - Hillsdale - Daceyville		0.999980	0.999956	0.999988	
Number of cases in population       0.0001       0.0001         Risk:       -1.0E-07       -1.8E-08       -1.4E-07         Pagewood - Hillsdale - Daceyville         Total Population in study area:       631 <t< td=""><td>Allibutable fraction (AF).</td><td>-2.0E-05</td><td>-4.4E-05</td><td colspan="2">-1.2E-05</td></t<>	Allibutable fraction (AF).	-2.0E-05	-4.4E-05	-1.2E-05	
Pagewood - Hillsdale - Daceyville         1002 01         1002 00         1112 01           Total Population in study area:         631         631         631         631           % population in assessment age-group:         100%         100%         16%           total change         -5.8         -5.8         -5.8           Population weighted Δx (µg/m³):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         0.999983         0.999961         0.999983           OLICE         Attributable fraction (AF):         -1.7E-05         -3.9E-05         -1.1E-07           Attributable fraction (AF):         -1.7E-05         -3.9E-05         -1.1E-07           Risk:         -9.1E-08         -1.6E-08         -1.3E-07           Sydney Airport         Model         Model         0.0000         0.0000           Risk:         -9.1E-08         -1.6E-08         -1.3E-07         1.6E/08         -1.3E-07           Model         Model         Attributable fraction (AF):         0.1000%         1.6%         0.16%	Risk:	-1.0E-07	-0.0004 -1.8E-08	-0.0003 -1 4F-07	
Total Population in study area:         631         631         631           % population in assessment age-group:         100%         100%         16%           total change         -5.8         -5.8         -5.8           Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per gerson)         0.00524         0.00040         0.01209           Relative Risk:         0.999983         0.999961         0.999983           Attributable fraction (AF):         -1.7E-05         -3.9E-05         -1.1E-05           Increased number of cases in population:         -0.0001         0.0000         0.0000           Risk:         -9.1E-08         -1.6E-08         -1.3E-07           Total Population in study area:         61         61         61           % population in assessment age-group:         100%         100%         16%           % population in assessment age-group:         100%         100%         16%           % population weighted $\Delta x$ (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524	Pagewood - Hillsdale - Daceyville				
% population in assessment age-group:         100%         100%         16%           total change         -5.8         -5.8         -5.8         -5.8           Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         0.999983         0.999961         0.999983           Attributable fraction (AF):         -1.7E-05         -3.9E-05         -1.1E-05           Increased number of cases in population:         -0.0001         0.0000         0.00000           Risk:         -9.1E-08         -1.6E-08         -1.3E-07           Sydney Airport         -         -         -           Total Population in study area:         61         61         61           % population in assessment age-group:         100%         100%         16%           Modulation weighted $\Delta x$ (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524	Total Population in study area:	631	631	631	
total change         -5.8         -5.8         -5.8           Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         0.999983         0.999961         0.999983           Other Control         0.0000         0.00000         0.00000           Relative Risk:         0.999983         0.999961         0.99983           Increased number of cases in population         -0.0001         0.0000         0.0000           Risk:         -9.1E-08         -1.6E-08         -1.3E-07           Sydney Airport         -         -         -         -           Total Population in study area:         61         61         61           % population in assessment age-group:         100%         100%         16%           Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.	% population in assessment age-group	100%	100%	16%	
Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         -0.00919176         -0.00919176         -0.00919176           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         0.999983         0.999961         0.999883           Attributable fraction (AF):         -1.7E-05         -3.9E-05         -1.1E-05           Increased number of cases in population:         -0.0001         0.0000         0.0000           Risk:         -9.1E-08         -1.6E-08         -1.3E-07           Sydney Airport         -         -         -           Total Population in study area:         61         61         61%           % population in assessment age-group:         100%         100%         16%           Word train Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06	total change	-5.8	-5.8	-5.8	
Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person) $0.00524$ $0.00040$ $0.01209$ Relative Risk: $0.999983$ $0.999961$ $0.999989$ Attributable fraction (AF): $-1.7E-05$ $-3.9E-05$ $-1.1E-05$ Increased number of cases in population: $-0.0001$ $0.0000$ $0.0000$ Risk: $-9.1E-08$ $-1.6E-08$ $-1.3E-07$ Sydney Airport $-1.6E-08$ $-1.3E-07$ Total Population in study area: $611$ $611$ $617$ % population in assessment age-group: $100\%$ $100\%$ $16\%$ Model change $0.18$ $0.18$ $0.18$ $0.120\%$ Baseline Incidence (per 100,000) (as per Table 4.4) $524$ $40$ $120\%$ Baseline Incidence (per person) $0.00524$ $0.00040$ $0.0120\%$ Relative Risk: $1.000006$ $1.000013$ $1.000003$ Attributable fraction (AF): $5.5E-06$ $1.3E-05$ $3.4E-06$ Increased number of cases in popula	Population weighted $\Delta x$ (µg/m <sup>3</sup> ):	-0.00919176	-0.00919176	-0.00919176	
Baseline Incidence (per person) $0.00524$ $0.00040$ $0.01209$ Relative Risk: $0.999983$ $0.999961$ $0.999883$ Attributable fraction (AF): $-1.7E-05$ $-3.9E-05$ $-1.1E-05$ Increased number of cases in population: $-0.0001$ $0.0000$ $0.0000$ Risk: $-9.1E-08$ $-1.6E-08$ $-1.3E-07$ Sydney Airport         Total Population in study area: $611$ $611$ $611$ % population in assessment age-group: $100\%$ $100\%$ $16\%$ Mathematical change $0.18$ $0.18$ $0.18$ $0.1209$ Baseline Incidence (per 100,000) (as per Table 4.4) $524$ $40$ $1209$ Baseline Incidence (per person) $0.00524$ $0.00040$ $0.01209$ Relative Risk: $1.000006$ $1.000013$ $1.000003$ Attributable fraction (AF): $5.5E-06$ $1.3E-05$ $3.4E-06$ Increased number of cases in population: $0.0000$ $0.00000$ $0.00003$ Attributable fraction (AF): $5.0E-00$ $1.4E-09$ <t< td=""><td>Baseline Incidence (per 100,000) (as per Table 4.4)</td><td>524</td><td>40</td><td>1209</td></t<>	Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209	
Relative Risk: $0.999983$ $0.999961$ $0.99988$ Attributable fraction (AF): $-1.7E-05$ $-3.9E-05$ $-1.1E-05$ Increased number of cases in population: $-0.0001$ $0.0000$ $0.0000$ Risk: $-9.1E-08$ $-1.6E-08$ $-1.3E-07$ Sydney Airport $-1.6E-08$ $-1.3E-07$ Total Population in study area: $61$ $61$ $61$ % population in assessment age-group: $100\%$ $100\%$ $16\%$ total change $0.18$ $0.18$ $0.18$ $0.1295082$ Baseline Incidence (per 100,000) (as per Table 4.4) $524$ $40$ $1209$ Baseline Incidence (per person) $0.00524$ $0.00040$ $0.01205$ Relative Risk: $1.000006$ $1.000013$ $1.000003$ Attributable fraction (AF): $5.5E-06$ $1.3E-05$ $3.4E-06$ Increased number of cases in population: $0.0000$ $0.00000$ $0.00003$	Baseline Incidence (per person)	0.00524	0.00040	0.01209	
Attributable fraction (AF): $-1.7E-05$ $-3.9E-05$ $-1.1E-05$ Increased number of cases in population: $-0.0001$ $0.0000$ $0.0000$ Risk: $-9.1E-08$ $-1.6E-08$ $-1.3E-07$ Sydney Airport	Relative Risk:	0.999983	0.999961	0.999989	
Increased number of cases in population         -0.0001         0.0000         0.0000           Risk:         -9.1E-08         -1.6E-08         -1.3E-07           Sydney Airport              Total Population in study area:         61         61         61           % population in assessment age-group:         100%         100%         16%           total change         0.18         0.18         0.18           Population weighted Δx (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.00000         0.00003	Attributable fraction (AF):	-1.7E-05	-3.9E-05	-1.1E-05	
Sydney Airport         10.100         11.0100         11.0100           Total Population in study area:         61         61         61           % population in assessment age-group:         100%         100%         16%           total change         0.18         0.18         0.18           Population weighted Δx (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.0000         0.0000		-0.0001	-1.6E-08	-1.3E-07	
Total Population in study area:         61         61         61           % population in assessment age-group:         100%         100%         16%           total change         0.18         0.18         0.18           Population weighted $\Delta x$ (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.00000         0.0000	Svdnev Airnord	-9.12-00	-1.02-08	-1.5Ľ-07	
% population in assessment age-group:         100%         100%         16%           total change         0.18         0.18         0.18         0.18           Population weighted Δx (µg/m³):         0.00295082         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.0000         0.0000	Total Population in study area	61	61	61	
total change         0.18         0.18         0.18           Population weighted Δx (µg/m³):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.00006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.0000         0.0000	% population in assessment age-group	100%	100%	16%	
Population weighted Δx (µg/m <sup>3</sup> ):         0.00295082         0.00295082         0.00295082           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.00000         0.0000	total change	0.18	0.18	0.18	
Baseline Incidence (per 100,000) (as per Table 4.4)         524         40         1209           Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.0000         0.0000	Population weighted $\Delta x (\mu g/m^3)$ :	0.00295082	0.00295082	0.00295082	
Baseline Incidence (per person)         0.00524         0.00040         0.01209           Relative Risk:         1.000006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.0000         0.0000	Baseline Incidence (per 100,000) (as per Table 4.4)	524	40	1209	
Relative Risk:         1.00006         1.000013         1.000003           Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.0000         0.0000           Bick:         2.0E-08         5.0E-00         4.4E-08	Baseline Incidence (per person)	0.00524	0.00040	0.01209	
Attributable fraction (AF):         5.5E-06         1.3E-05         3.4E-06           Increased number of cases in population:         0.0000         0.0000         0.0000           Bick:         2.0E-08         5.0E-00         4.4E-08	Relative Risk:	1.000006	1.000013	1.000003	
Increased number of cases in population: 0.0000 0.0000 0.0000	Attributable fraction (AF):	5.5E-06	1.3E-05	3.4E-06	
	Increased number of cases in population	0.0000	0.0000	0.0000	
	RISK.	2.9E-00	5.0⊑-09	4.1E-00	

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term		
Age Group:	anne IIA	appe IIA			
$\beta$ (change in offect per 1 µg/m <sup>3</sup> PM) (as per Table 6-16)	0.00188	0.00426	0.00115		
Kogarah - Rockdale I GA	0.00100	0.00120	0.00110		
Total Population in study area:	113547	113547	113547		
% population in assessment age-group:	100%	100%	15%		
total change	-553.3	-553.3	-553.3		
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00487287	-0.00487287	-0.00487287		
Baseline Incidence (per 100,000) (as per Table 4-5)	535	40	1209		
Baseline Incidence (per person)	0.00535	0.00040	0.01209		
Attributable fraction (AE):	0.999991	0.999979	0.999994		
Increased number of cases in population	-0.006	-0.0009	-0.0011		
Risk:	-4.9E-08	-8.3E-09	-6.8E-08		
Individual subrubs within LGA					
Arncliffe - Bardwell Park					
Total Population in study area:	21457	21457	21457		
% population in assessment age-group:	100%	100%	15%		
Deputation weighted Av (ug(m <sup>3</sup> ))	-97.4	-97.4	-97.4		
Baseline Incidence (per 100 000) (as per Table 4.4)	-0.00453931	-0.00453931	-0.00453931		
Baseline Incidence (per 100,000) (as per rable 4.4) Baseline Incidence (per person)	0.00535	0 00040	0.01209		
Relative Risk:	0.999991	0.999981	0.999995		
Attributable fraction (AF):	-8.5E-06	-1.9E-05	-5.2E-06		
Increased number of cases in population:	-0.0010	-0.0002	-0.00020		
Risk:	-4.6E-08	-7.7E-09	-6.3E-08		
Bexley Total Deputation in study areas	20002	20002	20002		
Population in assessment age-group	20002	20002	20002		
total change	16.9	16.9	16.9		
Population weighted $\Delta x (\mu g/m^3)$ :	0.00084492	0.00084492	0.00084492		
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209		
Baseline Incidence (per person)	0.00535	0.00040	0.01209		
Relative Risk:	1.000002	1.000004	1.000001		
Attributable fraction (AF):	1.6E-06	3.6E-06	9.7E-07		
Increased number of cases in population:	0.0002 8.5E-09	0.0000 1.4E-09	0.00003 1.2E-08		
Kingsgrove (South) - Bardwell Park	0.52-03	1.42-03	1.22-00		
Total Population in study area:	2879	2879	2879		
% population in assessment age-group:	100%	100%	15%		
total change	-3.5	-3.5	-3.5		
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00121570	-0.00121570	-0.00121570		
Baseline Incidence (per 100,000) (as per 1 able 4.4)	535	40	1209		
Baseline incidence (per person)	0.00535	0.00040	0.01209		
Attributable fraction (AF):	-2.3E-06	-5.2E-06	-1.4E-06		
Increased number of cases in population:	0.0000	-0.00001	-0.00001		
Risk:	-1.2E-08	-2.1E-09	-1.7E-08		
Kogarah					
Total Population in study area:	11323	11323	11323		
% population in assessment age-group:	100%	100%	15%		
Population weighted Ax (ug/m <sup>3</sup> ):	0.002967/1	0.002967/1	0.002967/1		
Baseline Incidence (per 100.000) (as per Table 4.4)	535	40	1209		
Baseline Incidence (per person)	0.00535	0.00040	0.01209		
Relative Risk:	1.000006	1.000013	1.000003		
Attributable fraction (AF):	5.6E-06	1.3E-05	3.4E-06		
Increased number of cases in population:	0.0003	0.00006	0.00007		
Risk: Kogarah Bay - Carlton - Allawah	3.0E-08	5.0E-09	4.1E-08		
Total Population in study area:	10923	10923	10923		
% population in assessment age-group:	10020	10020	15%		
total change	-22.1	-22.1	-22.1		
Population weighted Δx ( $\mu$ g/m <sup>3</sup> ):	-0.00202325	-0.00202325	-0.00202325		
Baseline Incidence (per 100,000) (as per Table 4.4)	535	40	1209		
Baseline Incidence (per person)	0.00535	0.00040	0.01209		
Relative Risk:	0.999996	0.999991	0.999998		
Attributable traction (AF):	-3.8E-06	-8.6E-06	-2.3E-06		
Risk:	-2.0E-08	-3.4E-09	-0.0004 -2.8E-08		
Monterey - Brighton-le-Sands - Kyeemagh		52 00	2.02 00		
Total Population in study area:	13915	13915	13915		
% population in assessment age-group:	100%	100%	15%		
total change	-100.4	-100.4	-100.4		
Population weighted Δx (µg/m <sup>2</sup> ):	-0.00721524	-0.00721524	-0.00721524		
	535	40	1209		

Health Endpoint	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16	0.00188	0.00426	0.00115
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	0.999986	0.999969	0.999992
Attributable fraction (AF)	-1.4E-05	-3.1E-05	-8.3E-06
Increased number of cases in population	-0.00101	-0.00017	-0.000204
Risk	-7.3E-08	-1.2E-08	-1.0E-07
Rockdale - Banksia	1		
Total Population in study area	: 19957	19957	19957
% population in assessment age-group	: 100%	100%	15%
total change	-76.4	-76.4	-76.4
Population weighted $\Delta x (\mu g/m^3)$	-0.00382823	-0.00382823	-0.00382823
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	0.999993	0.999984	0.999996
Attributable fraction (AF)	-7.2E-06	-1.6E-05	-4.4E-06
Increased number of cases in population	-0.00077	-0.00013	-0.000155
Risk	-3.8E-08	-6.5E-09	-5.3E-08
Sans Souci - Ramsgate	•		
Total Population in study area	13091	13091	13091
% population in assessment age-group	: 100%	100%	15%
total change	-304	-304	-304
Population weighted $\Delta x (\mu g/m^3)$	-0.02322206	-0.02322206	-0.02322206
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209
Baseline Incidence (per person	0.00535	0.00040	0.01209
Relative Risk	0.999956	0.999901	0.999973
Attributable fraction (AF)	-4.4E-05	-9.9E-05	-2.7E-05
Increased number of cases in population	-0.00305	-0.00052	-0.000617
Risk	-2.3E-07	-3.9E-08	-3.2E-07
	I		

Health Endpoint:	Mortality - All Causes, Short- term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions, Short-term		
Age Group:	All ages	All ages	1-14 years		
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-16	0.00188	0.00426	0.00115		
Hurstville LGA					
Total Population in study area	657	657	657		
% population in assessment age-group	: 100%	100%	15%		
total change	-0.5	-0.52	-0.52		
Population weighted $\Delta x (\mu g/m^3)$	-0.00079148	-0.00079148	-0.00079148		
Baseline Incidence (per 100,000) (as per Table 4-5	) 535	40	1209		
Baseline Incidence (per person	0.00535	0.00040	0.01209		
Relative Risk	0.999999	0.999997	0.999999		
Attributable fraction (AF)	-1.5E-06	-3.4E-06	-9.1E-07		
Increased number of cases in population	-0.000005	-0.000001	-0.000001		
Risk	-8.0E-09	-1.3E-09	-1.1E-08		
Individual subrubs within LGA					
Hurstville					
Total Population in study area	96	96	96		
% population in assessment age-group	: 100%	100%	15%		
total change	0.47	0.47	0.47		
Population weighted $\Delta x (\mu g/m^3)$	0.00489583	0.00489583	0.00489583		
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209		
Baseline Incidence (per person	0.00535	0.00040	0.01209		
Relative Risk	1.000009	1.000021	1.000006		
Attributable fraction (AF)	9.2E-06	2.1E-05	5.6E-06		
Increased number of cases in population	. 0.0000	0.0000	0.00000		
Risk	4.9E-08	8.3E-09	6.8E-08		
South Hurstville - Blakehurs					
Total Population in study area	561	561	561		
% population in assessment age-group	: 100%	100%	15%		
total change	-0.99	-0.99	-0.99		
Population weighted Δx (µg/m <sup>3</sup> )	-0.00176471	-0.00176471	-0.0017647		
Baseline Incidence (per 100,000) (as per Table 4.4	) 535	40	1209		
Baseline Incidence (per person	0.00535	0.00040	0.01209		
Relative Risk	0.999997	0.999992	0.999998		
Attributable fraction (AF)	-3.3E-06	-7.5E-06	-2.0E-06		
Increased number of cases in population	0.0000	0.0000	0.00000		
Risk	-1.8E-08	-3.0E-09	-2.5E-08		
Total population incidence - All Suburbs	-0.0098	-0.001670	-0.00197		

Annexure F – Risk calculations: Particulate matter

#### Quantification of Effects - PM<sub>2.5</sub> and PM<sub>10</sub> F6 Extension: 2026

		Air quality indicator	: PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>10</sub>	PM <sub>2.5</sub>	DPM				
		Endpoint:	Mortality - All	Hospitalisations -	Hospitalisations -	Mortality - All	Mortality - All	Mortality -	Mortality -	Mortality -	Morbidity - Asthma	Increased risk -
			Causes	Cardiovascular	Respiratory	Causes	Causes	Cardiopulmonary	Cardiovascular	Respiratory	ED Admissions	lung cancer
								. ,				
	E	ffect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term	Long-term	Short-Term	Short-Term	Short-Term	Based on WHO
Age Group				≥ 65 years	≥ 65 years	All ages	All ages	≥ 30 years	All ages	All ages	1-14 years	inhalation unit risk
	β (change in effect per 1 μg/m³) (as per Table 6-23				0.00041	0.0006	0.00094	0.013	0.00097	0.0019	0.00148	3.40E-05
Annual Baseline Incidence (as per Table 4-5												(ug/m3)-1
	1026	9235	3978	493	493	412	134.7	39.9	1209			
	0.01026	0.09235	0.03978	0.00493	0.00493	0.00412	0.001347	0.000399	0.01209			
												-
Sensitive Receptors	Change in Annual Average PM10 Concentration (µg/m <sup>3</sup> )	Change in Annual Average PM2.5 Concentration (µg/m <sup>3</sup> )	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk
Impacts from tunnel ventilation outlets												
Grid receptors: maximum regardless of landuse	0.68	0.44	3E-05	3E-05	7E-06	2E-06	2E-06	2E-05	6E-07	3E-07	8E-06	1E-05
Grid receptors: maximum residential	0.68	0.44	3E-05	3E-05	7E-06	2E-06	2E-06	2E-05	6E-07	3E-07	8E-06	1E-05
Grid receptors: maximum childcare	0.08	0.05	3E-06	4E-06	8E-07	2E-07	2E-07	3E-06	7E-08	4E-08	9E-07	2E-06
Grid receptors: maximum school	0.25	0.17	1E-05	1E-05	3E-06	7E-07	8E-07	9E-06	2E-07	1E-07	3E-06	6E-06
Grid receptors: maximum aged care	0.08	0.12	7E-06	9E-06	2E-06	2E-07	6E-07	6E-06	2E-07	9E-08	2E-06	4E-06
Only as a set one of a set of a set of the law of an a disert				0 0 00	05 00	1 1 1 1 1 1 1			1	0 0 0 0	05.00	1

Grid receptors: maximum school		0.25	0.17	1E-05	1E-05	3E-06	7E-07	8E-07	9E-06	2E-07	1E-07	3E-06	6E-06
Grid receptors: maximum aged care		0.08	0.12	7E-06	9E-06	2E-06	2E-07	6E-07	6E-06	2E-07	9E-08	2E-06	4E-06
Grid receptors: maximum hospital and medical		0.15	0.11	7E-06	8E-06	2E-06	4E-07	5E-07	6E-06	1E-07	8E-08	2E-06	4E-06
Grid receptors: commercial/industrial		0.52	0.35	2E-05	3E-05	6E-06	2E-06	2E-06	2E-05	5E-07	3E-07	6E-06	1E-05
Grid receptors: open space		0.15	0.12	7E-06	9E-06	2E-06	4E-07	5E-07	6E-06	2E-07	9E-08	2E-06	4E-06
Community Receptors													
St Finbar's Primary School	Primary School	0.0513	0.0365	2E-06	3E-06	6E-07	2E-07	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
St George Christian School Infants	Primary School	0.1146	0.0736	4E-06	5E-06	1E-06	3E-07	3E-07	4E-06	1E-07	6E-08	1E-06	3E-06
Ramsgate Public School	Primary School	0.0346	0.0815	5E-06	6E-06	1E-06	1E-07	4E-07	4E-06	1E-07	6E-08	1E-06	3E-06
Estia Health	Community Home	-0.0318	0.1663	1E-05	1E-05	3E-06	-9E-08	8E-07	9E-06	2E-07	1E-07	3E-06	6E-06
Wesley Hospital Kogarah	General Hospital	0.0239	0.0888	5E-06	7E-06	1E-06	7E-08	4E-07	5E-06	1E-07	7E-08	2E-06	3E-06
St George School	Special School	0.0971	0.1894	1E-05	1E-05	3E-06	3E-07	9E-07	1E-05	2E-07	1E-07	3E-06	6E-06
St George Hospital	General Hospital	0.0674	0.0402	2E-06	3E-06	7E-07	2E-07	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
Brighton-Le-Sands Public School	Primary School	0.1048	-0.0257	-2E-06	-2E-06	-4E-07	3E-07	-1E-07	-1E-06	-3E-08	-2E-08	-5E-07	-9E-07
Kogarah Public School	Primary School	0.0317	0.0619	4E-06	5E-06	1E-06	9E-08	3E-07	3E-06	8E-08	5E-08	1E-06	2E-06
St George Girls High School	High School	0.0261	0.0108	6E-07	8E-07	2E-07	8E-08	5E-08	6E-07	1E-08	8E-09	2E-07	4E-07
St Thomas More's Catholic School	Primary School	-0.1550	-0.0283	-2E-06	-2E-06	-5E-07	-5E-07	-1E-07	-2E-06	-4E-08	-2E-08	-5E-07	-1E-06
Jenny-Lyn Nursing Home	Community Home	-0.0939	0.0099	6E-07	7E-07	2E-07	-3E-07	5E-08	5E-07	1E-08	8E-09	2E-07	3E-07
Huntingdon Gardens Aged Care Facility	Community Home	0.0021	0.0070	4E-07	5E-07	1E-07	6E-09	3E-08	4E-07	9E-09	5E-09	1E-07	2E-07
Rockdale Public School	Primary School	-0.0607	0.0139	8E-07	1E-06	2E-07	-2E-07	6E-08	7E-07	2E-08	1E-08	2E-07	5E-07
Scalabrini Village Nursing Home-Bexley	Community Home	-0.1050	-0.0210	-1E-06	-2E-06	-3E-07	-3E-07	-1E-07	-1E-06	-3E-08	-2E-08	-4E-07	-7E-07
Rockdale Nursing Home	Community Home	-0.0223	0.0371	2E-06	3E-06	6E-07	-7E-08	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
Arncliffe Public School	Primary School	-0.2349	-0.1173	-7E-06	-9E-06	-2E-06	-7E-07	-5E-07	-6E-06	-2E-07	-9E-08	-2E-06	-4E-06
Athelstane Public School	Primary School	0.0299	0.0112	7E-07	8E-07	2E-07	9E-08	5E-08	6E-07	1E-08	9E-09	2E-07	4E-07
Al Zabra College	Combined Primary-			9E-07	1E-06	2E-07	1E-07	7E-08	8E-07	2E-08	1E-08	3E-07	5E-07
Ai Zania Goliege	Secondary School	0.0372	0.0150										
Cairsfoot School	Special School	-0.0634	-0.0213	-1E-06	-2E-06	-3E-07	-2E-07	-1E-07	-1E-06	-3E-08	-2E-08	-4E-07	-7E-07
Undercliffe Public School	Primary School	0.0420	-0.0410	-2E-06	-3E-06	-7E-07	1E-07	-2E-07	-2E-06	-5E-08	-3E-08	-7E-07	-1E-06
Ferncourt Public School	Primary School	-0.0240	-0.0123	-7E-07	-9E-07	-2E-07	-7E-08	-6E-08	-7E-07	-2E-08	-9E-09	-2E-07	-4E-07
Tempe High School	High School	0.0542	0.0030	2E-07	2E-07	5E-08	2E-07	1E-08	2E-07	4E-09	2E-09	5E-08	1E-07
St Peters Public School	Primary School	-0.0188	-0.0101	-6E-07	-7E-07	-2E-07	-6E-08	-5E-08	-5E-07	-1E-08	-8E-09	-2E-07	-3E-07
St Pius' Catholic Primary School	Primary School	-0.0358	-0.0265	-2E-06	-2E-06	-4E-07	-1E-07	-1E-07	-1E-06	-3E-08	-2E-08	-5E-07	-9E-07
Frobel Alexandria Early Learning Centre	Child Care Centre	0.0040	0.0179	1E-06	1E-06	3E-07	1E-08	8E-08	1E-06	2E-08	1E-08	3E-07	6E-07
Little Learning School - Alexandria	Child Care Centre	0.1950	0.1389	8E-06	1E-05	2E-06	6E-07	6E-07	7E-06	2E-07	1E-07	2E-06	5E-06
Active Kids Mascot	Child Care Centre	0.0595	-0.0353	-2E-06	-3E-06	-6E-07	2E-07	-2E-07	-2E-06	-5E-08	-3E-08	-6E-07	-1E-06
Mascot Public School	Primary School	-0.0364	-0.0084	-5E-07	-6E-07	-1E-07	-1E-07	-4E-08	-5E-07	-1E-08	-6E-09	-2E-07	-3E-07
Hippos Friends	Child Care Centre	0 1203	0.0652	4E-06	5E-06	1E-06	4E-07	3E-07	3E-06	9E-08	5E-08	1E-06	2E-06

#### Quantification of Effects - PM<sub>2.5</sub> and PM<sub>10</sub> F6 Extension: 2036

									1		
	Air quality i	dicator: PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>10</sub>	PM <sub>2.5</sub>	DPM				
	E	ndpoint: Mortality - All	Hospitalisations -	Hospitalisations -	Mortality - All	Mortality - All	Mortality -	Mortality -	Mortality -	Morbidity - Asthma	Increased risk -
		Causes	Cardiovascular	Respiratory	Causes	Causes	Cardiopulmonary	Cardiovascular	Respiratory	ED Admissions	lung cancer
Effect Exposure Duration			Short-term	Short-term	Short-Term	Short-Term	Long-term	Short-Term	Short-Term	Short-Term	Based on WHO
	e Group: ≥ 30 years	≥ 65 years	≥ 65 years	All ages	All ages	≥ 30 years	All ages	All ages	1-14 years	inhalation unit risk	
	ole 6-23) 0.0058	0.0008	0.00041	0.0006	0.00094	0.013	0.00097	0.0019	0.00148	3.40E-05	
	able 4-5)									(ug/m3)-1	
	100,000) 1026	9235	3978	493	493	412	134.7	39.9	1209		
	er year) 0.01026	0.09235	0.03978	0.00493	0.00493	0.00412	0.001347	0.000399	0.01209		
Sensitive Receptors	Change in Annual Change in A Average PM10 Average PI Concentration (µg/m <sup>3</sup> ) Concentration	nual 12.5 Risk (µg/m³)	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk
Impacts from tunnel ventilation outlets											
Grid receptors: maximum regardless of landuse	0.65 0.39	2E-05	3E-05	6E-06	2E-06	2E-06	2E-05	5E-07	3E-07	7E-06	1E-05
Grid receptors: maximum residential	0.65 0.39	2E-05	3E-05	6E-06	2E-06	2E-06	2E-05	5E-07	3E-07	7E-06	1E-05
Grid receptors: maximum childcare	0.14 0.04	2E-06	3E-06	6E-07	4E-07	2E-07	2E-06	5E-08	3E-08	6E-07	1E-06

Grid receptors: maximum childcare		0.14	0.04	2E-06	3E-06	6E-07	4E-07	2E-07	2E-06	5E-08	3E-08	6E-07	1E-06
Grid receptors: maximum school		0.21	0.23	1E-05	2E-05	4E-06	6E-07	1E-06	1E-05	3E-07	2E-07	4E-06	8E-06
Grid receptors: maximum aged care		0.09	0.08	5E-06	6E-06	1E-06	3E-07	4E-07	4E-06	1E-07	6E-08	1E-06	3E-06
Grid receptors: maximum hospital and medical		0.16	0.12	7E-06	9E-06	2E-06	5E-07	6E-07	6E-06	2E-07	9E-08	2E-06	4E-06
Grid receptors: commercial/industrial		0.52	0.30	2E-05	2E-05	5E-06	2E-06	1E-06	2E-05	4E-07	2E-07	5E-06	1E-05
Grid receptors: open space		0.20	0.15	9E-06	1E-05	2E-06	6E-07	7E-07	8E-06	2E-07	1E-07	3E-06	5E-06
Community Receptors													
St Finbar's Primary School	Primary School	0.0305	0.0082	5E-07	6E-07	1E-07	9E-08	4E-08	4E-07	1E-08	6E-09	1E-07	3E-07
St George Christian School Infants	Primary School	0.0639	-0.0012	-7E-08	-9E-08	-2E-08	2E-07	-6E-09	-7E-08	-2E-09	-9E-10	-2E-08	-4E-08
Ramsgate Public School	Primary School	0.1052	0.0563	3E-06	4E-06	9E-07	3E-07	3E-07	3E-06	7E-08	4E-08	1E-06	2E-06
Estia Health	Community Home	0.1837	0.1042	6E-06	8E-06	2E-06	5E-07	5E-07	6E-06	1E-07	8E-08	2E-06	4E-06
Wesley Hospital Kogarah	General Hospital	-0.0503	0.0338	2E-06	2E-06	6E-07	-1E-07	2E-07	2E-06	4E-08	3E-08	6E-07	1E-06
St George School	Special School	0.1520	0.1441	9E-06	1E-05	2E-06	4E-07	7E-07	8E-06	2E-07	1E-07	3E-06	5E-06
St George Hospital	General Hospital	0.1419	0.0366	2E-06	3E-06	6E-07	4E-07	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
Brighton-Le-Sands Public School	Primary School	0.1263	0.0012	7E-08	9E-08	2E-08	4E-07	5E-09	6E-08	2E-09	9E-10	2E-08	4E-08
Kogarah Public School	Primary School	0.0361	0.0636	4E-06	5E-06	1E-06	1E-07	3E-07	3E-06	8E-08	5E-08	1E-06	2E-06
St George Girls High School	High School	-0.0470	-0.0221	-1E-06	-2E-06	-4E-07	-1E-07	-1E-07	-1E-06	-3E-08	-2E-08	-4E-07	-8E-07
St Thomas More's Catholic School	Primary School	0.0025	-0.0266	-2E-06	-2E-06	-4E-07	8E-09	-1E-07	-1E-06	-3E-08	-2E-08	-5E-07	-9E-07
Jenny-Lyn Nursing Home	Community Home	-0.1511	0.0314	2E-06	2E-06	5E-07	-4E-07	1E-07	2E-06	4E-08	2E-08	6E-07	1E-06
Huntingdon Gardens Aged Care Facility	Community Home	-0.0323	-0.0250	-1E-06	-2E-06	-4E-07	-1E-07	-1E-07	-1E-06	-3E-08	-2E-08	-4E-07	-9E-07
Rockdale Public School	Primary School	0.0176	-0.0069	-4E-07	-5E-07	-1E-07	5E-08	-3E-08	-4E-07	-9E-09	-5E-09	-1E-07	-2E-07
Scalabrini Village Nursing Home-Bexley	Community Home	-0.0652	0.0355	2E-06	3E-06	6E-07	-2E-07	2E-07	2E-06	5E-08	3E-08	6E-07	1E-06
Rockdale Nursing Home	Community Home	0.0270	-0.0604	-4E-06	-4E-06	-1E-06	8E-08	-3E-07	-3E-06	-8E-08	-5E-08	-1E-06	-2E-06
Arncliffe Public School	Primary School	-0.1231	-0.0873	-5E-06	-6E-06	-1E-06	-4E-07	-4E-07	-5E-06	-1E-07	-7E-08	-2E-06	-3E-06
Athelstane Public School	Primary School	-0.0425	-0.0451	-3E-06	-3E-06	-7E-07	-1E-07	-2E-07	-2E-06	-6E-08	-3E-08	-8E-07	-2E-06
Al Zahra Callaga	Combined Primary-			-3E-06	-4E-06	-8E-07	-2E-07	-2E-07	-3E-06	-6E-08	-4E-08	-9E-07	-2E-06
Al Zallia College	Secondary School	-0.0510	-0.0478										
Cairsfoot School	Special School	-0.0744	-0.0160	-1E-06	-1E-06	-3E-07	-2E-07	-7E-08	-9E-07	-2E-08	-1E-08	-3E-07	-5E-07
Undercliffe Public School	Primary School	0.0493	0.0281	2E-06	2E-06	5E-07	1E-07	1E-07	2E-06	4E-08	2E-08	5E-07	1E-06
Ferncourt Public School	Primary School	-0.1243	0.0042	2E-07	3E-07	7E-08	-4E-07	2E-08	2E-07	5E-09	3E-09	7E-08	1E-07
Tempe High School	High School	-0.0631	0.0826	5E-06	6E-06	1E-06	-2E-07	4E-07	4E-06	1E-07	6E-08	1E-06	3E-06
St Peters Public School	Primary School	-0.0567	-0.0566	-3E-06	-4E-06	-9E-07	-2E-07	-3E-07	-3E-06	-7E-08	-4E-08	-1E-06	-2E-06
St Pius' Catholic Primary School	Primary School	-0.0364	0.0001	4E-09	5E-09	1E-09	-1E-07	3E-10	4E-09	9E-11	5E-11	1E-09	2E-09
Frobel Alexandria Early Learning Centre	Child Care Centre	0.1102	-0.0671	-4E-06	-5E-06	-1E-06	3E-07	-3E-07	-4E-06	-9E-08	-5E-08	-1E-06	-2E-06
Little Learning School - Alexandria	Child Care Centre	0.0051	0.0525	3E-06	4E-06	9E-07	2E-08	2E-07	3E-06	7E-08	4E-08	9E-07	2E-06
Active Kids Mascot	Child Care Centre	0.0318	-0.0328	-2E-06	-2E-06	-5E-07	9E-08	-2E-07	-2E-06	-4E-08	-2E-08	-6E-07	-1E-06
Mascot Public School	Primary School	-0.0578	-0.0579	-3E-06	-4E-06	-9E-07	-2E-07	-3E-07	-3E-06	-8E-08	-4E-08	-1E-06	-2E-06
Hippos Friends	Child Care Centre	-0.0764	-0.1715	-1E-05	-1E-05	-3E-06	-2E-07	-8E-07	-9E-06	-2E-07	-1E-07	-3E-06	-6E-06

### Quantification of Effects - PM<sub>2.5</sub> and PM<sub>10</sub> F6 Extension: 2036 Cumulative

	Air quali	ity indicator: PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>10</sub>	PM <sub>2.5</sub>	DPM				
		Endpoint: Mortali	ality - All Hospitalisa	ions - Hospitalisations -	Mortality - All	Mortality - All	Mortality -	Mortality -	Mortality -	Morbidity - Asthma	Increased risk -
		Causes	es Cardiovaso	ular Respiratory	Causes	Causes	Cardiopulmonary	Cardiovascular	Respiratory	ED Admissions	lung cancer
	Effect Exposu	re Duration: Long-te	term Short-term	Short-term	Short-Term	Short-Term	Long-term	Short-Term	Short-Term	Short-Term	Based on WHO
		Age Group: ≥ 30 ye	/ears ≥ 65 years	≥ 65 years	All ages	All ages	≥ 30 years	All ages	All ages	1-14 years	inhalation unit risk
	β (change in effect per 1 µg/m <sup>3</sup> ) (as per	Table 6-23) 0.0058	8 0.0008	0.00041	0.0006	0.00094	0.013	0.00097	0.0019	0.00148	3.40E-05
	Annual Baseline Incidence (as p	er Table 4-5)									(ug/m3)-1
	Annual baseline incidence (	per 100,000) 1026	9235	3978	493	493	412	134.7	39.9	1209	
	Baseline Incidence (per pers	on per year) 0.0102	26 0.09235	0.03978	0.00493	0.00493	0.00412	0.001347	0.000399	0.01209	
Sensitive Receptors	Change in Annual Change in Average PM10 Average Concentration (µg/m³) Concentrat	n Annual e PM2.5 ion (µq/m³)	Risk Ris	c Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk

Sensitive Receptors		Average PM10	Average PM2.5	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk
		Concentration (µg/m <sup>3</sup> )	Concentration (µg/m <sup>3</sup> )										
Impacts from tunnel ventilation outlets													
Grid receptors: maximum regardless of landuse		0.50	0.37	2E-05	3E-05	6E-06	1E-06	2E-06	2E-05	5E-07	3E-07	7E-06	1E-05
Grid receptors: maximum residential		0.50	0.37	2E-05	3E-05	6E-06	1E-06	2E-06	2E-05	5E-07	3E-07	7E-06	1E-05
Grid receptors: maximum childcare		0.14	0.08	4E-06	6E-06	1E-06	4E-07	3E-07	4E-06	1E-07	6E-08	1E-06	3E-06
Grid receptors: maximum school		0.11	0.10	6E-06	7E-06	2E-06	3E-07	5E-07	5E-06	1E-07	8E-08	2E-06	3E-06
Grid receptors: maximum aged care		0.09	0.07	4E-06	5E-06	1E-06	3E-07	3E-07	4E-06	9E-08	5E-08	1E-06	2E-06
Grid receptors: maximum hospital and medical		0.10	0.06	4E-06	5E-06	1E-06	3E-07	3E-07	3E-06	8E-08	5E-08	1E-06	2E-06
Grid receptors: commercial/industrial		0.46	0.23	1E-05	2E-05	4E-06	1E-06	1E-06	1E-05	3E-07	2E-07	4E-06	8E-06
Grid receptors: open space		0.25	0.11	6E-06	8E-06	2E-06	7E-07	5E-07	6E-06	1E-07	8E-08	2E-06	4E-06
Community Receptors													
St Finbar's Primary School	Primary School	-0.0745	-0.0123	-7E-07	-9E-07	-2E-07	-2E-07	-6E-08	-7E-07	-2E-08	-9E-09	-2E-07	-4E-07
St George Christian School Infants	Primary School	-0.2528	-0.1698	-1E-05	-1E-05	-3E-06	-7E-07	-8E-07	-9E-06	-2E-07	-1E-07	-3E-06	-6E-06
Ramsgate Public School	Primary School	-0.0181	0.0068	4E-07	5E-07	1E-07	-5E-08	3E-08	4E-07	9E-09	5E-09	1E-07	2E-07
Estia Health	Community Home	-0.2660	-0.1875	-1E-05	-1E-05	-3E-06	-8E-07	-9E-07	-1E-05	-2E-07	-1E-07	-3E-06	-6E-06
Wesley Hospital Kogarah	General Hospital	-0.0361	-0.0241	-1E-06	-2E-06	-4E-07	-1E-07	-1E-07	-1E-06	-3E-08	-2E-08	-4E-07	-8E-07
St George School	Special School	-0.0104	-0.0265	-2E-06	-2E-06	-4E-07	-3E-08	-1E-07	-1E-06	-3E-08	-2E-08	-5E-07	-9E-07
St George Hospital	General Hospital	0.0770	0.0486	3E-06	4E-06	8E-07	2E-07	2E-07	3E-06	6E-08	4E-08	9E-07	2E-06
Brighton-Le-Sands Public School	Primary School	0.1533	0.0297	2E-06	2E-06	5E-07	5E-07	1E-07	2E-06	4E-08	2E-08	5E-07	1E-06
Kogarah Public School	Primary School	-0.0106	0.0326	2E-06	2E-06	5E-07	-3E-08	2E-07	2E-06	4E-08	2E-08	6E-07	1E-06
St George Girls High School	High School	0.0421	-0.0184	-1E-06	-1E-06	-3E-07	1E-07	-9E-08	-1E-06	-2E-08	-1E-08	-3E-07	-6E-07
St Thomas More's Catholic School	Primary School	0.0456	-0.0225	-1E-06	-2E-06	-4E-07	1E-07	-1E-07	-1E-06	-3E-08	-2E-08	-4E-07	-8E-07
Jenny-Lyn Nursing Home	Community Home	-0.0495	-0.0318	-2E-06	-2E-06	-5E-07	-1E-07	-1E-07	-2E-06	-4E-08	-2E-08	-6E-07	-1E-06
Huntingdon Gardens Aged Care Facility	Community Home	-0.0929	-0.0206	-1E-06	-2E-06	-3E-07	-3E-07	-1E-07	-1E-06	-3E-08	-2E-08	-4E-07	-7E-07
Rockdale Public School	Primary School	0.0013	-0.0278	-2E-06	-2E-06	-5E-07	4E-09	-1E-07	-1E-06	-4E-08	-2E-08	-5E-07	-9E-07
Scalabrini Village Nursing Home-Bexley	Community Home	-0.0217	-0.0079	-5E-07	-6E-07	-1E-07	-6E-08	-4E-08	-4E-07	-1E-08	-6E-09	-1E-07	-3E-07
Rockdale Nursing Home	Community Home	0.0378	-0.0203	-1E-06	-2E-06	-3E-07	1E-07	-9E-08	-1E-06	-3E-08	-2E-08	-4E-07	-7E-07
Arncliffe Public School	Primary School	-0.1748	-0.0146	-9E-07	-1E-06	-2E-07	-5E-07	-7E-08	-8E-07	-2E-08	-1E-08	-3E-07	-5E-07
Athelstane Public School	Primary School	-0.0545	-0.0290	-2E-06	-2E-06	-5E-07	-2E-07	-1E-07	-2E-06	-4E-08	-2E-08	-5E-07	-1E-06
Al Zahra College	Combined Primary-		0.0101	-1E-06	-1E-06	-3E-07	-3E-07	-9E-08	-1E-06	-3E-08	-1E-08	-3E-07	-7E-07
Online (and Online)	Secondary School	-0.1053	-0.0194	05.00	05.00	45.07	05.07	45.07	45.00	45.00	05.00	FF 07	-
Carstoot School	Special School	-0.1001	-0.0274	-2E-06	-2E-06	-4E-07	-3E-07	-1E-07	-1E-06	-4E-08	-2E-08	-5E-07	-9E-07
Englished School	Primary School	0.0343	-0.0118	-7E-07	-9E-07	-2E-07	1E-07	-5E-08	-6E-07	-2E-08	-92-09	-2E-07	-4E-07
Ferncourt Public School	Primary School	-0.0768	0.0023	1E-07	2E-07	4E-08	-2E-07	1E-08	1E-07	3E-09	2E-09	4E-08	8E-08
St Potore Public School	Brimony School	-0.0674	0.0047	3E-07	3E-07	0E-00	-3E-07	2E-00	3E-07	0E-09	4E-09	0E-00	2E-07
St Peters Public School	Primary School	0.0028	0.0915	5E-06	7E-06	1E-06	4E-07	4E-07	5E-06	1E-07	7E-08	2E-06	3E-06
Schol Alexandria Early Learning Centre	Child Care Contro	0.0028	0.0319	200	200	3E-07	0E-09	10-07	200	40-08	2E-08	00-07	75.07
Little Learning Cehool Alexandria	Child Care Centre	-0.0230	-0.0200	-1E-06	-1E-06	-3E-07	-8E-U8	-9E-08	-1E-06	-3E-08	-2E-08	-4E-U/	-/E-U/
Little Learning School - Alexandria	Child Care Centre	0.1605	-0.0782	-3E-06	-0E-06	-1E-06	3E-07	-407	-4E-06	-1E-07	-0E-08	-1E-06	-3E-06
Active Nus Wascot Maccet Public School	Drimony School	0.0733	-0.1250	-/ E-06	-9E-06	-2E-06	2E-07	-0E-07	-/ E-06	-2E-07	-9E-08	-2E-06	-4E-06
Mascot Fubic SCHOOL	Child Core Centre	-0.0097	-0.1340	-8E-06	-1E-05	-2E-06	-2E-07	-0E-U/	-/E-06	-2E-07	-1E-07	-2E-U0	-5E-06
nippos riterios	Child Care Centre	-0.1631	-0.1425	-8E-U6	-1E-05	-2E-Ub	-5E-07	-/E-U/	-8E-06	-2E-07	-1E-07	-3E-Ub	-5E-06

#### Quantification of Effects - PM<sub>2.5</sub> and PM<sub>10</sub> F6 Extension: 2026 - Ventilation facilities only

Air quality indicator:	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM10	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	DPM
Endpoint:	Mortality - All Causes	Hospitalisations - Cardiovascular	Hospitalisations - Respiratory	Mortality - All Causes	Mortality - All Causes	Mortality - Cardiopulmonary	Mortality - Cardiovascular	Mortality - Respiratory	Morbidity - Asthma ED Admissions	Increased risk - lung cancer
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term	Long-term	Short-Term	Short-Term	Short-Term	Based on WHO
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	All ages	≥ 30 years	All ages	All ages	1-14 years	inhalation unit risk
β (change in effect per 1 μg/m³) (as per Table 6-23)	0.0058	0.0008	0.00041	0.0006	0.00094	0.013	0.00097	0.0019	0.00148	3.40E-05
Annual Baseline Incidence (as per Table 4-5)										(ug/m3)-1
Annual baseline incidence (per 100,000)	1026	9235	3978	493	493	412	134.7	39.9	1209	
Baseline Incidence (per person per year)	0.01026	0.09235	0.03978	0.00493	0.00493	0.00412	0.001347	0.000399	0.01209	

S		Change in Annual Average PM10 Concentration (µg/m <sup>3</sup> )	Change in Annual Average PM2.5 Concentration (µg/m <sup>3</sup> )	Risk	Risk	Ri
from tunnel ventilation outlets						
ceptors: maximum regardless of landuse		0.17	0.11	7E-06	8E-06	2E-06
ceptors: maximum residential		0.17	0.11	7E-06	8E-06	2E-06
eptors: maximum childcare		0.04	0.028	2E-06	2E-06	5E-07
eceptors: maximum school		0.082	0.059	4E-06	4E-06	1E-06
eceptors: maximum aged care		0.054	0.038	2E-06	3E-06	6E-07
ceptors: maximum hospital and medical		0.067	0.046	3E-06	3E-06	8E-07
eceptors: commercial/industrial		0.12	0.083	5E-06	6E-06	1E-06
eceptors: open space		0.073	0.049	3E-06	4E-06	8E-07
munity Receptors						
bar's Primary School	Primary School	0.0156	0.0077	5E-07	6E-07	1E-07
eorge Christian School Infants	Primary School	0.0133	0.0072	4E-07	5E-07	1E-07
gate Public School	Primary School	0.0150	0.0099	6E-07	7E-07	2E-07
Health	Community Home	0.0280	0.0224	1E-06	2E-06	4E-07
ey Hospital Kogarah	General Hospital	0.0252	0.0215	1E-06	2E-06	4E-07
eorge School	Special School	0.0515	0.0372	2E-06	3E-06	6E-07
eorge Hospital	General Hospital	0.0517	0.0377	2E-06	3E-06	6E-07
hton-Le-Sands Public School	Primary School	0.0800	0.0621	4E-06	5E-06	1E-06
arah Public School	Primary School	0.0453	0.0328	2E-06	2E-06	5E-07
George Girls High School	High School	0.0341	0.0226	1E-06	2E-06	4E-07
Thomas More's Catholic School	Primary School	0.0500	0.0310	2E-06	2E-06	5E-07
ny-I yn Nursing Home	Community Home	0.0230	0.0154	9E-07	1E-06	3E-07
tingdon Gardens Aged Care Facility	Community Home	0.0169	0.0132	8E-07	1E-06	2E-07
ckdale Public School	Primary School	0.0392	0.0256	2E-06	2E-06	4E-07
alabrini Village Nursing Home-Bexley	Community Home	0.0106	0.0085	5E-07	6E-07	1E-07
ckdale Nursing Home	Community Home	0.0204	0.0100	6E-07	7E-07	2E-07
ncliffe Public School	Primary School	0.0234	0.0161	1E-06	1E-06	3E-07
helstane Public School	Primary School	0.0103	0.0081	5E-07	6E-07	1E-07
Zahra College	Combined Primary Secondary School	- 0.0155	0.0054	3E-07	4E-07	9E-08
Cairsfoot School	Special School	0.0270	0.0171	1E-06	1E-06	3E-07
ndercliffe Public School	Primary School	0.0065	0.0040	2E-07	3E-07	7E-08
erncourt Public School	Primary School	0.0056	0.0056	3E-07	4E-07	9E-08
empe High School	High School	0.0018	-0.0001	-4F-09	-5E-09	-1E-09
Peters Public School	Primary School	-0.0318	-0.0230	-1E-06	-2E-06	-4E-07
Pius' Catholic Primary School	Primary School	0.0003	-0.0015	-9F-08	-1E-07	-3E-08
nbel Alexandria Farly Learning Centre	Child Care Centre	-0.0072	-0.0042	-2E-07	-3E-07	-7E-08
le Learning School - Alexandria	Child Care Centre	-0.0158	-0.0141	-2E-07	-1E-06	-2E-07
ive Kids Mascot	Child Care Centre	-0.0138	-0.0141	-1E-07	-1E-00	-3E-08
ascot Public School	Primary School	-0.0075	0.0015	9E-08	1E-07	2E-08
Noce Friende	Child Care Contro	0.0010	0.0013	9E-00	2E 07	2E-U0 7E 09
lippos r nenus	Child Care Centre	0.0005	0.0042	35-01	35-07	/ E-U0

### Quantification of Effects - PM<sub>2.5</sub> and PM<sub>10</sub> F6 Extension: 2036 - Ventilation facilities only

Air quality indicator:	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM10	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	DPM
Endpoint:	Mortality - All Causes	Hospitalisations - Cardiovascular	Hospitalisations - Respiratory	Mortality - All Causes	Mortality - All Causes	Mortality - Cardiopulmonary	Mortality - Cardiovascular	Mortality - Respiratory	Morbidity - Asthma ED Admissions	Increased risk - lung cancer
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term	Long-term	Short-Term	Short-Term	Short-Term	Based on WHO
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	All ages	≥ 30 years	All ages	All ages	1-14 years	inhalation unit risk
β (change in effect per 1 μg/m³) (as per Table 6-23)	0.0058	0.0008	0.00041	0.0006	0.00094	0.013	0.00097	0.0019	0.00148	3.40E-05
Annual Baseline Incidence (as per Table 4-5)										(ug/m3)-1
Annual baseline incidence (per 100,000)	1026	9235	3978	493	493	412	134.7	39.9	1209	
Baseline Incidence (per person per year)	0.01026	0.09235	0.03978	0.00493	0.00493	0.00412	0.001347	0.000399	0.01209	

ors		Change in Annual Average PM10 Concentration (µg/m <sup>3</sup> )	Change in Annual Average PM2.5 Concentration (µg/m <sup>3</sup> )	Risk	Risk	
ts from tunnel ventilation outlets						
eptors: maximum regardless of landuse		0.20	0.14	8E-06	1E-05	2E-06
ptors: maximum residential		0.20	0.14	8E-06	1E-05	2E-06
ptors: maximum childcare		0.051	0.038	2E-06	3E-06	6E-07
eptors: maximum school		0.10	0.075	4E-06	6E-06	1E-06
ceptors: maximum aged care		0.071	0.046	3E-06	3E-06	8E-07
ptors: maximum hospital and medical		0.08	0.055	3E-06	4E-06	9E-07
eceptors: commercial/industrial		0.13	0.097	6E-06	7E-06	2E-06
ceptors: open space		0.089	0.062	4E-06	5E-06	1E-06
imunity Receptors						
ibar's Primary School	Primary School	0.0162	0.0103	6E-07	8E-07	2E-07
orge Christian School Infants	Primary School	0.0189	0.0142	8E-07	1E-06	2E-07
sgate Public School	Primary School	0.0157	0.0175	1E-06	1E-06	3E-07
Health	Community Home	0.0342	0.0256	2E-06	2E-06	4E-07
ey Hospital Kogarah	General Hospital	0.0316	0.0314	2E-06	2E-06	5E-07
eorge School	Special School	0.0588	0.0430	3E-06	3E-06	7E-07
eorge Hospital	General Hospital	0.0620	0.0444	3E-06	3E-06	7E-07
hton-Le-Sands Public School	Primary School	0.0962	0.0714	4E-06	5E-06	1E-06
arah Public School	Primary School	0.0549	0.0418	2E-06	3E-06	7E-07
George Girls High School	High School	0.0390	0.0275	2E-06	2E-06	4E-07
Thomas More's Catholic School	Primary School	0.0502	0.0423	3E-06	3E-06	7E-07
ny-Lyn Nursing Home	Community Home	0.0228	0.0249	1E-06	2E-06	4E-07
ntingdon Gardens Aged Care Facility	Community Home	0.0261	0.0182	1E-06	1E-06	3E-07
ckdale Public School	Primary School	0.0489	0.0364	2E-06	3E-06	6E-07
alabrini Village Nursing Home-Bexley	Community Home	0.0161	0.0110	7E-07	8E-07	2E-07
ckdale Nursing Home	Community Home	0.0174	0.0143	8E-07	1E-06	2E-07
ncliffe Public School	Primary School	0.0280	0.0224	1E-06	2E-06	4E-07
nelstane Public School	Primary School	0.0107	0.0108	6E-07	8E-07	2E-07
Zahra College	Secondary School	0.0140	0.0108	6E-07	8E-07	2E-07
airsfoot School	Special School	0.0287	0.0276	2E-06	2E-06	5E-07
ndercliffe Public School	Primary School	0.0081	0.0055	3E-07	4E-07	9E-08
erncourt Public School	Primary School	0.0091	0.0084	5E-07	6E-07	1E-07
empe High School	High School	0.0081	0.0065	4E-07	5E-07	1E-07
Peters Public School	Primary School	-0.0474	-0.0261	-2E-06	-2E-06	-4E-07
Pius' Catholic Primary School	Primary School	-0.0043	-0.0036	-2E-07	-3E-07	-6E-08
obel Alexandria Early Learning Centre	Child Care Centre	-0.0086	-0.0066	-4E-07	-5E-07	-1E-07
le Learning School - Alexandria	Child Care Centre	-0.0225	-0.0200	-1E-06	-1E-06	-3E-07
ive Kids Mascot	Child Care Centre	-0.0070	-0.0019	-1E-07	-1E-07	-3E-08
scot Public School	Primary School	0.0033	0.0042	2E-07	3E-07	7E-08
oos Friends	Child Care Centre	0.0065	0.0099	6E-07	7E-07	2E-07

### Quantification of Effects - PM<sub>2.5</sub> and PM<sub>10</sub> F6 Extension: 2036 Cumulative - Ventilation facilities only

Air quality indicator:	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>10</sub>	PM <sub>2.5</sub>	DPM				
Endpoint:	Mortality - All	Hospitalisations -	Hospitalisations -	Mortality - All	Mortality - All	Mortality -	Mortality -	Mortality -	Morbidity - Asthma	Increased risk -
	Causes	Cardiovascular	Respiratory	Causes	Causes	Cardiopulmonary	Cardiovascular	Respiratory	ED Admissions	lung cancer
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term	Long-term	Short-Term	Short-Term	Short-Term	Based on WHO
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	All ages	≥ 30 years	All ages	All ages	1-14 years	inhalation unit risk
β (change in effect per 1 μg/m³) (as per Table 6-23)	0.0058	0.0008	0.00041	0.0006	0.00094	0.013	0.00097	0.0019	0.00148	3.40E-05
Annual Baseline Incidence (as per Table 4-5)										(ug/m3)-1
Annual baseline incidence (per 100,000)	1026	9235	3978	493	493	412	134.7	39.9	1209	
Baseline Incidence (per person per year)	0.01026	0.09235	0.03978	0.00493	0.00493	0.00412	0.001347	0.000399	0.01209	

Sensitive Receptors		Change in Annual Average PM10 Concentration (µg/m <sup>3</sup> )	Change in Annual Average PM2.5 Concentration (µg/m <sup>3</sup> )	Risk									
Impacts from tunnel ventilation outlets													
Grid receptors: maximum regardless of landuse		0.25	0.16	1E-05	1E-05	3E-06	7E-07	7E-07	9E-06	2E-07	1E-07	3E-06	5E-06
Grid receptors: maximum residential		0.25	0.16	1E-05	1E-05	3E-06	7E-07	7E-07	9E-06	2E-07	1E-07	3E-06	5E-06
Grid receptors: maximum childcare		0.08	0.051	3E-06	4E-06	8E-07	2E-07	2E-07	3E-06	7E-08	4E-08	9E-07	2E-06
Grid receptors: maximum school		0.14	0.096	6E-06	7E-06	2E-06	4E-07	4E-07	5E-06	1E-07	7E-08	2E-06	3E-06
Grid receptors: maximum aged care		0.096	0.066	4E-06	5E-06	1E-06	3E-07	3E-07	4E-06	9E-08	5E-08	1E-06	2E-06
Grid receptors: maximum hospital and medical		0.11	0.077	5E-06	6E-06	1E-06	3E-07	4E-07	4E-06	1E-07	6E-08	1E-06	3E-06
Grid receptors: commercial/industrial		0.17	0.12	7E-06	9E-06	2E-06	5E-07	6E-07	6E-06	2E-07	9E-08	2E-06	4E-06
Grid receptors: open space		0.12	0.076	5E-06	6E-06	1E-06	4E-07	4E-07	4E-06	1E-07	6E-08	1E-06	3E-06
Community Receptors													11
St Finbar's Primary School	Primary School	0.0175	0.0151	9E-07	1E-06	2E-07	5E-08	7E-08	8E-07	2E-08	1E-08	3E-07	5E-07
St George Christian School Infants	Primary School	0.0253	0.0192	1E-06	1E-06	3E-07	7E-08	9E-08	1E-06	3E-08	1E-08	3E-07	7E-07
Ramsgate Public School	Primary School	0.0236	0.0189	1E-06	1E-06	3E-07	7E-08	9E-08	1E-06	2E-08	1E-08	3E-07	6E-07
Estia Health	Community Home	0.0542	0.0368	2E-06	3E-06	6E-07	2E-07	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
Wesley Hospital Kogarah	General Hospital	0.0567	0.0374	2E-06	3E-06	6E-07	2E-07	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
St George School	Special School	0.0880	0.0572	3E-06	4E-06	9E-07	3E-07	3E-07	3E-06	7E-08	4E-08	1E-06	2E-06
St George Hospital	General Hospital	0.0869	0.0614	4E-06	5E-06	1E-06	3E-07	3E-07	3E-06	8E-08	5E-08	1E-06	2E-06
Brighton-Le-Sands Public School	Primary School	0.1303	0.0864	5E-06	6E-06	1E-06	4E-07	4E-07	5E-06	1E-07	7E-08	2E-06	3E-06
Kogarah Public School	Primary School	0.0714	0.0531	3E-06	4E-06	9E-07	2E-07	2E-07	3E-06	7E-08	4E-08	9E-07	2E-06
St George Girls High School	High School	0.0622	0.0405	2E-06	3E-06	7E-07	2E-07	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
St Thomas More's Catholic School	Primary School	0.0918	0.0614	4E-06	5E-06	1E-06	3E-07	3E-07	3E-06	8E-08	5E-08	1E-06	2E-06
Jenny-Lyn Nursing Home	Community Home	0.0476	0.0337	2E-06	2E-06	6E-07	1E-07	2E-07	2E-06	4E-08	3E-08	6E-07	1E-06
Huntingdon Gardens Aged Care Facility	Community Home	0.0378	0.0240	1E-06	2E-06	4E-07	1E-07	1E-07	1E-06	3E-08	2E-08	4E-07	8E-07
Rockdale Public School	Primary School	0.0686	0.0453	3E-06	3E-06	7E-07	2E-07	2E-07	2E-06	6E-08	3E-08	8E-07	2E-06
Scalabrini Village Nursing Home-Bexley	Community Home	0.0242	0.0177	1E-06	1E-06	3E-07	7E-08	8E-08	1E-06	2E-08	1E-08	3E-07	6E-07
Rockdale Nursing Home	Community Home	0.0295	0.0185	1E-06	1E-06	3E-07	9E-08	9E-08	1E-06	2E-08	1E-08	3E-07	6E-07
Arncliffe Public School	Primary School	0.0593	0.0360	2E-06	3E-06	6E-07	2E-07	2E-07	2E-06	5E-08	3E-08	6E-07	1E-06
Athelstane Public School	Primary School	0.0218	0.0123	7E-07	9E-07	2E-07	6E-08	6E-08	7E-07	2E-08	9E-09	2E-07	4E-07
Al Zahra College	Combined Primar Secondary School	y- 0.0289	0.0208	1E-06	2E-06	3E-07	9E-08	1E-07	1E-06	3E-08	2E-08	4E-07	7E-07
Cairsfoot School	Special School	0.0570	0.0378	2E-06	3E-06	6E-07	2E-07	2E-07	2E-06	5E-08	3E-08	7E-07	1E-06
Undercliffe Public School	Primary School	0.0147	0.0127	8E-07	9E-07	2E-07	4E-08	6E-08	7E-07	2E-08	1E-08	2E-07	4E-07
Ferncourt Public School	Primary School	0.0163	0.0126	7E-07	9E-07	2E-07	5E-08	6E-08	7E-07	2E-08	1E-08	2E-07	4E-07
Tempe High School	High School	0.0201	0.0099	6E-07	7E-07	2E-07	6E-08	5E-08	5E-07	1E-08	7E-09	2E-07	3E-07
St Peters Public School	Primary School	-0.0139	-0.0062	-4E-07	-5E-07	-1E-07	-4E-08	-3E-08	-3E-07	-8E-09	-5E-09	-1E-07	-2E-07
St Pius' Catholic Primary School	Primary School	0.0145	0.0054	3E-07	4E-07	9E-08	4E-08	3E-08	3E-07	7E-09	4E-09	1E-07	2E-07
Frobel Alexandria Early Learning Centre	Child Care Centre	0.0017	0.0017	1E-07	1E-07	3E-08	5E-09	8E-09	9E-08	2E-09	1E-09	3E-08	6E-08
Little Learning School - Alexandria	Child Care Centre	-0.0015	-0.0031	-2E-07	-2E-07	-5E-08	-4E-09	-1E-08	-2E-07	-4E-09	-2E-09	-6E-08	-1E-07
Active Kids Mascot	Child Care Centre	0.0050	0.0000	-8E-10	-1E-09	-2E-10	1E-08	-6E-11	-7E-10	-2E-11	-1E-11	-2E-10	-4E-10
Mascot Public School	Primary School	0.0045	0.0065	4E-07	5E-07	1E-07	1E-08	3E-08	3E-07	8E-09	5E-09	1E-07	2E-07
Hippos Friends	Child Care Centre	0.0120	0.0083	5E-07	6E-07	1E-07	4E-08	4E-08	4E-07	1E-08	6E-09	1E-07	3E-07

# Annexure G – Population incidence calculations: Particulate matter

## Assessment of Increased Incidence - PM<sub>2.5</sub> F6 Extension: 2026

		Primary Indicator	S		Se	condary Indicators	5	
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Strathfield - Burwood - Ashfield LGA								
Total Population in study area	20160	20160	20160	20160	20160	20160	20160	20160
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-102.5	-102.5	-102.5	-102.5	-102.5	-102.5	-102.5	-102.5
Population weighted Ax (ug/m <sup>3</sup> ):	-0.00508433	-0.00508433	-0.00508433	-0.00508433	-0.00508433	-0.00508433	-0.00508433	-0.00508433
Baseline Incidence (per 100.000) (as per Table 4-5)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (ner nerson)	0.01026	0.09235	0 03978	0.00335	0.00412	0 00099	0 00049	0.01209
Relative Risk:	0.999971	0.999996	0.999998	0.999995	0.999934	0.999995	0.999990	0.999992
Attributable fraction (AF)	-2.9E-05	-4.1E-06	-2.1E-06	-4.8E-06	-6.6E-05	-4.9E-06	-9.7E-06	-7.5E-06
Increased number of cases in population:	-0.0039	-0.00092	-0.00020	-0.00032	-0.0035	-0.000098	-0.000096	-0.00026
Risk	-3.0E-07	-3.8E-07	-8.3E-08	-1.6E-08	-2.7E-07	-4.9E-09	-4.8E-09	-9.1E-08
Individual subrubs within LGA								
Ashfield								
Total Population in study area	1512	1512	1512	1512	1512	1512	1512	1512
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-0.44	-0.44	-0.44	-0.44	-0.44	-0.44	-0.44	-0.44
Population weighted $\Delta x (ug/m^3)$	-0.00029101	-0.00029101	-0.00029101	-0.00029101	-0.00029101	-0.00029101	-0.00029101	-0.00029101
Baseline Incidence (per 100.000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0 00412	0 00099	0.00049	0.01209
Relative Risk:	0.999998	1.000000	1.000000	1.000000	0.999996	1.000000	0.999999	1.000000
Attributable fraction (AF)	-1.7E-06	-2.3E-07	-1.2E-07	-2.7E-07	-3.8E-06	-2.8E-07	-5.5E-07	-4.3E-07
Increased number of cases in population:	-0.000017	-0.0000040	-0.0000088	-0.0000014	-0.000015	-0.00000042	-0.00000041	-0.0000011
Risk	-1.7E-08	-2.1E-08	-4.7E-09	-9.2E-10	-1.6E-08	-2.8E-10	-2.7E-10	-5.2E-09
Canterbury (North) - Ashbury	r							
Total Population in study area	7179	7179	7179	7179	7179	7179	7179	7179
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-27.4	-27.4	-27.4	-27.4	-27.4	-27.4	-27.4	-27.4
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00381669	-0.00381669	-0.00381669	-0.00381669	-0.00381669	-0.00381669	-0.00381669	-0.00381669
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk	0.999978	0.999997	0.999998	0.999996	0.999950	0.999996	0.999993	0.999994
Attributable fraction (AF)	-2.2E-05	-3.1E-06	-1.6E-06	-3.6E-06	-5.0E-05	-3.7E-06	-7.3E-06	-5.6E-06
Increased number of cases in population:	-0.0010	-0.00025	-0.000055	-0.000086	-0.00094	-0.000026	-0.000026	-0.000069
Risk	-2.3E-07	-2.8E-07	-6.2E-08	-1.2E-08	-2.0E-07	-3.7E-09	-3.6E-09	-6.8E-08
Dulwich Hill - Lewisham	i							
Total Population in study area	11400	11400	11400	11400	11400	11400	11400	11400
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-74.6	-74.6	-74.6	-74.6	-74.6	-74.6	-74.6	-74.6
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00654386	-0.00654386	-0.00654386	-0.00654386	-0.00654386	-0.00654386	-0.00654386	-0.00654386
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk:	0.999962	0.999995	0.999997	0.999994	0.999915	0.999994	0.999988	0.999990
Attributable fraction (AF)	-3.8E-05	-5.2E-06	-2.7E-06	-6.2E-06	-8.5E-05	-6.3E-06	-1.2E-05	-9.7E-06
Increased number of cases in population:	-0.0028	-0.00067	-0.00015	-0.00023	-0.0025	-0.000071	-0.000070	-0.00019
Risk	-3.9E-07	-4.8E-07	-1.1E-07	-2.1E-08	-3.5E-07	-6.3E-09	-6.1E-09	-1.2E-07
Haberfield - Summer Hill								
I otal Population in study area	69	69	69	65	69	69	69	69
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
	-0.04	-0.04	-0.04	-0.04	-0.04	-0.04	-0.04	-0.04
Population weighted $\Delta x$ (µg/m <sup>*</sup> ):	-0.00057971	-0.00057971	-0.00057971	-0.00057971	-0.00057971	-0.00057971	-0.00057971	-0.00057971
baseline incidence (per 100,000) (as per 1 able 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk:	0.999997	1.000000	1.000000	0.9999999	0.999992	0.999999	0.999999	0.999999
Attributable traction (AF):	-3.4E-06	-4.bE-0/	-2.4E-07	-5.4E-07	-7.5E-06	-5.6E-07	-1.1E-06	-8.6E-07
Disk-	-0.00000152	-0.0000036	-0.00000080	-0.0000013	-0.00000137	-0.00000038	-0.000000038	-0.0000010
RISK	-3.4∟-00	-4.3∟-00	-5.52-09	-1.02-08	-3.12-00	-5.02-10	-3.42-10	-1.02-00
	1	1		1	1		1	

		Primary Indicator	s		Se	condary Indicators	5	
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Sydney Inner City LGA							1	
Total Population in study area	29695	29695	29695	29695	29695	29695	29695	29695
% population in assessment age-group:	58%	8%	8%	100%	58%	100%	100%	6%
total change	-10.62	-10.62	-10.62	-10.62	-10.62	-10.62	-10.62	-10.62
Population weighted $\Delta x (\mu q/m^3)$	-0.00035764	-0.00035764	-0 00035764	-0.00035764	-0 00035764	-0.00035764	-0.00035764	-0.00035764
Baseline Incidence (per 100.000) (as per Table 4-5)	1026	9235	3978	534.2	412.0	146.4	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00534	0 00412	0.00146	0 00049	0.01209
Relative Risk	0.999998	1 000000	1 000000	1 000000	0 999995	1 000000	0.999999	0.999999
Attributable fraction (AF)	-2 1F-06	-2 9E-07	-1.5E-07	-3 4E-07	-4 6F-06	-3.5E-07	-6.8E-07	-5.3E-07
Increased number of cases in population:	-0.00036	-0.000064	-0.000014	-0.000053	-0.00033	-0.000015	-0.000010	-0.000011
Bisk	-2 1E-08	-2 6E-08	-5.8E-09	-1 8E-09	-1.9E-08	-5 1E-10	-3 4E-10	-6 4E-09
Individual subrubs within LGA								
Erskinville - Alexandria								
Total Population in study area	11411	11411	11411	11411	11411	11411	11411	11411
% population in assessment age-group:	58%	8%	8%	100%	58%	100%	100%	6%
total change	62.2	62.2	62.2	62.2	62.2	62.2	62.2	62.2
Population weighted Δx (ug/m <sup>3</sup> ):	0.00545088	0.00545088	0.00545088	0.00545088	0.00545088	0.00545088	0.00545088	0.00545088
Baseline Incidence (per 100.000) (as per Table 4.4)	1026	9235	3978	534.2	412.0	146.4	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00534	0 00412	0.00146	0 00049	0.01209
Relative Risk:	1.000032	1.000004	1.000002	1.000005	1.000071	1.000005	1.000010	1.00008
Attributable fraction (AF)	3.2E-05	4.4E-06	2.2E-06	5.1E-06	7.1E-05	5.3E-06	1.0E-05	8.1E-06
Increased number of cases in population:	0.0021	0.00038	0.000083	0.00031	0.0019	0.000088	0.000058	0.000066
Risk	3.2E-07	4.0E-07	8.9E-08	2.7E-08	2.9E-07	7.7E-09	5.1E-09	9.8E-08
Newtown - Camperdown - Darlington								
Total Population in study area	5225	5225	5225	5225	5225	5225	5225	5225
% population in assessment age-group:	58%	8%	8%	100%	58%	100%	100%	6%
total change	48.6	48.6	48.6	48.6	48.6	48.6	48.6	48.6
Population weighted $\Delta x (\mu g/m^3)$ :	0.00930144	0.00930144	0.00930144	0.00930144	0.00930144	0.00930144	0.00930144	0.00930144
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	534.2	412.0	146.4	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00534	0.00412	0.00146	0.00049	0.01209
Relative Risk	1.000054	1.000007	1.000004	1.000009	1.000121	1.000009	1.000018	1.000014
Attributable fraction (AF)	5.4E-05	7.4E-06	3.8E-06	8.7E-06	1.2E-04	9.0E-06	1.8E-05	1.4E-05
Increased number of cases in population:	0.0017	0.00029	0.000065	0.00024	0.00150	0.000069	0.000046	0.000051
Risk	5.5E-07	6.9E-07	1.5E-07	4.7E-08	5.0E-07	1.3E-08	8.7E-09	1.7E-07
Waterloo - Beaconsfield								
Total Population in study area	13059	13059	13059	13059	13059	13059	13059	13059
% population in assessment age-group:	58%	8%	8%	100%	58%	100%	100%	6%
total change	-121.5	-121.5	-121.5	-121.5	-121.5	-121.5	-121.5	-121.5
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00930393	-0.00930393	-0.00930393	-0.00930393	-0.00930393	-0.00930393	-0.00930393	-0.00930393
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	534.2	412.0	146.4	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00534	0.00412	0.00146	0.00049	0.01209
Relative Risk	0.999946	0.999993	0.999996	0.999991	0.999879	0.999991	0.999982	0.999986
Attributable fraction (AF)	-5.4E-05	-7.4E-06	-3.8E-06	-8.7E-06	-1.2E-04	-9.0E-06	-1.8E-05	-1.4E-05
Increased number of cases in population:	-0.0042	-0.00074	-0.00016	-0.00061	-0.0037	-0.00017	-0.00011	-0.000128
Risk	-5.5E-07	-6.9E-07	-1.5E-07	-4.7E-08	-5.0E-07	-1.3E-08	-8.7E-09	-1.7E-07
		1			1	1		

		Primary Indicator	s		Se	condary Indicators	5	
Health Endpoint	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 µg/m <sup>3</sup> PM) (as per Table 6-23	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Marrickville - Sydenham - Petersham LGA								
Total Population in study area	: 35722	35722	35722	35722	35722	35722	35722	35722
% population in assessment age-group	64%	12%	12%	100%	64%	100%	100%	14%
total change	-331.8	-331.8	-331.8	-331.8	-331.8	-331.8	-331.8	-331.8
Population weighted $\Delta x (ug/m^3)$	-0.00928839	-0.00928839	-0.00928839	-0.00928839	-0.00928839	-0.00928839	-0.00928839	-0.00928839
Baseline Incidence (per 100.000) (as per Table 4-5	1026	9235	3978	508.0	412.0	138.9	49.4	1209.0
Baseline Incidence (per person	0.01026	0.09235	0.03978	0.00508	0 00412	0.00139	0 00049	0.01209
Relative Risk	0.999946	0.999993	0.999996	0.999991	0.999879	0.999991	0.999982	0.999986
Attributable fraction (AF)	-5.4E-05	-7.4E-06	-3.8E-06	-8.7E-06	-1.2E-04	-9.0E-06	-1.8E-05	-1.4E-05
Increased number of cases in population	-0.013	-0.0030	-0.00066	-0.0016	-0.011	-0.00045	-0.00031	-0.00084
Risk	-5.5E-07	-6.9E-07	-1.5E-07	-4.4E-08	-5.0E-07	-1.3E-08	-8.7E-09	-1.7E-07
Individual subrubs within LGA								
Marrickville								
Total Population in study area	25842	25842	25842	25842	25842	25842	25842	25842
% population in assessment age-group	64%	12%	12%	100%	64%	100%	100%	14%
total change	-101.5	-101.5	-101.5	-101.5	-101.5	-101.5	-101.5	-101.5
Population weighted Ax (ug/m <sup>3</sup> )	-0.00392771	-0.00392771	-0.00392771	-0.00392771	-0.00392771	-0.00392771	-0.00392771	-0.00392771
Baseline Incidence (per 100 000) (as per Table 4 4	1026	9235	3978	508.0	412.0	138.9	49.4	1209 (
Baseline Incidence (per recipiero) (de per rabie na	0.01026	0.00235	0.03978	0.00508	0.00/12	0.00130	0 00049	0.01200
Relative Risk	0.01020	0.03233	0.00070	0.00000	0.00412	0.00133	0.00043	0.01203
Attributable fraction (AF)	-2 3E-05	-3 1E-06	-1 6E-06	-3 7E-06	-5 1E-05	-3.8E-06	-7 5E-06	-5.8E-06
Increased number of cases in population	-0.0039	-0.00091	-0.00020	-0.00048	-0.0035	-0.00014	-0.00010	-0.00026
Risk	-2 3E-07	-2 9E-07	-6.4E-08	-1 9E-08	-2 1E-07	-5 3E-09	-3 7E-09	-7.0E-08
Petersham - Stanmore	2.02 01	2.02 01	0.42 00	1.52 00	2.12 07	0.02 00	0.7 2 00	1.02 00
Total Population in study area	2051	2051	2051	2051	2051	2051	2051	2051
% population in assessment age-group	64%	12%	12%	100%	64%	100%	100%	14%
total change	20.3	20.3	20.3	20.3	20.3	20.3	20.3	20.3
Population weighted Ax (ug/m <sup>3</sup> )	0.00080761	0.00080761	0.00080761	0.00080761	0.00080761	0.00080761	0.00080761	0.00080761
Baseline Incidence (per 100 000) (as per Table 4 4	1026	9235	3978	508.0	412.0	138.9	49.4	1209 (
Baseline Incidence (per response)	0.01026	0.09235	0.03978	0.00508	0.00412	0.00130	0.00049	0.01200
Relative Risk	1 000057	1 000008	1 000004	1 000009	1 000129	1 000133	1 000049	1 000015
Attributable fraction (AE)	5 7E-05	7 9E-06	4 1E-06	9 3E-06	1 3E-04	9.6E-06	1.000010	1.000010
Increased number of cases in population	0.00077	0.00018	0.000040	0.00010	0.00069	0.000027	0.000019	0.000051
Risk	5.9E-07	7.3E-07	1 6E-07	4 7E-08	5.3E-07	1.3E-08	9.3E-09	1 8E-07
Sydenham - Tempe - St Peters	0.02 01	1.02 01	1.02 01		0.02 0.	1.02 00	0.02 00	1102 01
Total Population in study area	7829	7829	7829	7829	7829	7829	7829	7829
% population in assessment age-group	64%	12%	12%	100%	64%	100%	100%	14%
total change	-250.6	-250.6	-250.6	-250.6	-250.6	-250.6	-250.6	-250.6
Population weighted Ax (ug/m <sup>3</sup> )	-0.03200920	-0.03200920	-0.03200920	-0.03200920	-0.03200920	-0.03200920	-0.03200920	-0.03200920
Baseline Incidence (per 100 000) (as per Table 4.4	1026	0.00200320	3078	508.0	412 N	138.0	49 A	1209.0
Baseline Incidence (per 100,000) (da per 14bie 4.4	0.01026	0.00225	0 02070	0.00509	0.00412	0.00120	0.00040	0.01200
Dasenne incluence (per person	0.01020	0.09233	0.03976	0.00506	0.00412	0.00139	0.00049	0.01205
Attributable fraction (AF)	-1 9F-04	-2 6E-05	-1 3E-05	-3 0E-05	-4 2 =- 04	-3 1E-05	-6 1E-05	-4 7E-04
Increased number of cases in population	-0.010	-2.00-00	-0.005	-0.02-03	-4.204	-0.12-03	-0.12-03	-9.00063
Risk	-1.9E-06	-2.4F-06	-5.2F-07	-1.5E-07	-1.7E-06	-4.3E-08	-3.0F-08	-5.7E-07
		22 00	0.22 07		2 00		0.02 00	0.12 01

Health Endpoint:       Mortality - All Causes, Long-term         Causes, Long-term       Causes, Long-term         β (change in effect per 1 µq/m <sup>3</sup> PM) (as per Table 6-23)       0.0058         Canterbury LGA       0.0058         Total Population in study area:       12562         % population in assessment age-group:       58%         total change       -3.55         Population e(per 100,000) (as per Table 4-5)       1026         Baseline Incidence (per person)       0.01028         Baseline Incidence (per person)       0.01026         Canterbury LGA       Relative Risk:       0.999998         Attributable fraction (AF):       -1.6E=06         Increased number of cases in population:       -0.00012         Risk:       -1.7E=08         Individual subrubs within LGA       1.6E=06         Canterbury (South) - Campsie       -         Total Population in study area:       149         % population in sesessment age-group:       58%         total change       1.24         Population weighted Δx (µg/m <sup>3</sup> ):       0.00832215         Baseline Incidence (per 100,000) (as per Table 4.4)       1026         Baseline Incidence (per 100,000) (as per Table 4.4)       1026         Baseline Incidence (per 100,000) (as per Table 4.4)<	Hospitalisations - Cardiovascular, Short-term ≥ 65 years 0.0008 12562 14% -3.55 -0.00028260 9235 0.09235 1.000036 -2.3E-07 -0.000036 -2.1E-08 149 149 144 0.00832215	Hospitalisations - Respiratory, Short-term ≥ 65 years 0.00041 12562 14% -3.55 -0.0028260 3978 0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09 -4.6E-09 -149 149 149	Mortality - All Causes, Short- term All ages 0.00094 	Mortality - Cardiopulmonary, Long-term ≥ 30 years 0.013 12562 58% -3.55 -0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 -149 58%	Mortality - Cardiovascular, Short-term All ages 0.00097 12562 100% -3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.0000039 -3.1E-10 -3.1E-10	Mortality - Respiratory, Short-term All ages 0.0019 12562 100% -3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 -2.7E-10	Morbidity - Asthma ED Admissions - Short-term 1-14 years 0.00148 12562 19% -3.55 -0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09
Age Group:         ≥ 30 years           β (change in effect per 1 µg/m³ PM) (as per Table 6-23)         0.0058           Canterbury LGA         0.0058           Total Population in study area:         12562           % population in assessment age-group:         58%           total change         -3.55           Population weighted Δx (µg/m³):         -0.00028260           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         Canterbury (South) - Campsie           Total Population in study area:         149           % population weighted Δx (µg/m³):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline I	Cardiovascular, Short-term 2 65 years 0.0008 12562 14% -3.55 -0.00028260 0.9235 1.000000 -2.35-07 -0.000036 -2.1E-08 149 149 144 0.00832215	Respiratory, Short-term ≥ 65 years 0.00041 12562 14% -3.55 -0.0028260 3978 0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09 149 149 14% 1.24%	Causes, Short- term	Cardiopulmonary, Long-term 2 30 years 0.013 12562 58% -3.55 -0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 449 58%	Cardiovascular, Short-term	Respiratory, Short-term All ages 0.0019 12562 100% -3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.000033 -2.7E-10 -2.7E-10	Asthma ED Admissions - Short-term 1-14 years 0.00148 12562 19% -3.55 -0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09
Age Group:         ≥ 30 years           β (change in effect per 1 µg/m³ PM) (as per Table 6-23)         0.0058           Canterbury LGA            Total Population in study area:         12562           % population in assessment age-group:         58%           total change         -3.55           Population weighted Δx (µg/m³):         -0.00028260           Baseline Incidence (per porson)         0.01026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.000120           Risk:         -1.7E-08           Individual subrubs within LGA         Canterbury (South) - Campsie           Total Population in study area:         149           % population in assessment age-group:         58%           124         Population weighted Δx (µg/m³):         0.00032215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Individual subrubs within LGA         0.00832215           Basel	Short-term           ≥ 65 years           0.0008           12562           14%           -3.55           -0.0028260           9235           0.09235           1.000000           -2.3E-07           -2.1E-08           149           1449           1.24           0.00832215	Short-term           ≥ 65 years           0.00041           12562           14%           -3.55           -0.00028260           3378           0.03978           1.000000           -1.2E-07           -0.0000080           -4.6E-09           149           149           149	term All ages 0.00094 12562 100% -3.55 -0.0028260 401.6 0.00402 1.000001 -2.7E-07 -0.000013 -1.1E-09 149 100% 124	Long-term ≥ 30 years 0.013 12562 58% -3.55 -0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 -149 58%	Short-term All ages 0.00097 12562 100% -3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.000039 -3.1E-10 -3.1E-10 -3.1E-10	Short-term All ages 0.0019 12562 100% -3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 -2.7E-10 149	Admissions - Short-term 1-14 years 0.00148 12562 19% -3.55 -0.00028260 1209.0 0.01209 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09 -0.00012 -5.1E-09 -0.00012
Age Group:         ≥ 30 years           β (change in effect per 1 µa/m³ PM) (as per Table 6-23)         0.0058           Canterbury LGA         0.0058           Total Population in study area:         12562           % population in assessment age-group:         58%           State Population weighted Δx (µg/m³):         -0.00028260           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Total Population in study area:         149           % population in assessment age-group:         58%           total change         1.24           Population weighted Δx (µg/m³):         0.000832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431	≥ 65 years 0.0008 12562 14% -3.55 -0.00028260 9235 1.000030 -2.3E-07 -0.00036 -2.1E-08 149 14% 1.24 0.00832215	≥ 65 years 0.00041 12562 14% -3.55 -0.00028260 3978 0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09 -4.6E-09 -149 149 14% -1.24%	All ages 0.00094 12562 100% -3.55 -0.0028260 401.6 0.00402 1.000000 -2.7E-07 -0.000013 -1.1E-09 149 100% 1 24	≥ 30 years 0.013 12562 58% -3.55 -0.00028260 412.0 0.00412 0.99996 -3.7E-06 -0.00011 -1.5E-08 -149 58%	All ages 0.00097 12562 100% -3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.000039 -3.1E-10 -3.1E-10 149	All ages 0.0019 12562 100% -3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 -2.7E-10	Short-term           1-14 years           0.00148           12562           19%           -3.55           -0.00028260           1209.0           0.01209           0.0010000           -4.2E-07           -0.000012           -5.1E-09
Age Group:         ≥ 30 years           β (change in effect per 1 µq/m³ PM) (as per Table 6-23)         0.0058           Canterbury LGA         Total Population in study area:         12562           % population in assessment age-group:         58%         total change         -3.55           Population experiment age-group:         58%         total change         -3.55           Population weighted Δx (µg/m³):         -0.00028260         Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per 100,000) (as per Table 4-5)         1026         Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012         Risk:         -1.7E-08           Individual subrubs within LGA         Canterbury (South) - Campsie         1.24           Total Population in study area:         149         % population in assessment age-group:         58%           total change         1.24         Population weighted Δx (µg/m³):         0.00832215         Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026         Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048         Relative Risk:         1.000048           Baseline Incidence (per person)	≥ 65 years 0.0008 12562 14% -3.55 -0.00028260 9235 0.09235 1.000006 -2.3E-07 -0.000036 -2.1E-08 149 149 144 0.00832215	≥ 65 years 0.00041 12562 14% -3.55 -0.00028260 0.3978 0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09 -4.6	All ages 0.00094 12562 100% -3.55 -0.00028260 401.6 0.00402 1.0000013 -2.7E-07 -0.000013 -1.1E-09 -1.1E-09 -1.40% 149 100% 1 24	≥ 30 years 0.013 12562 58% -3.55 -0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 -149 58%	All ages 0.00097 12562 100% -3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.0000039 -3.1E-10 -3.1E-10 149	All ages 0.0019 12562 100% -3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 -2.7E-10 149	1-14 years 0.00148 12562 19% -3.55 -0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09 -5.1E-09 -149
β (change in effect per 1 μg/m³ PM) (as per Table 6-23)         0.0058           Canterbury LGA           Total Population in study area.         12562           % population in assessment age-group:         58%.           total change         -3.55           Population weighted Δx (µg/m³):         -0.00028260           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         1026           Canterbury (South) - Campsie         1.24           Yoppulation in assessment age-group:         58%.           % population weighted Δx (µg/m³):         0.00032215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.010026           Relative Risk:         5.0E-07           Risk:         5.	0.0008 12562 14% -3.55 -0.00028260 0.9235 1.00000 -2.3E-07 -0.000036 -2.1E-08 149 14% 1.24 0.00832215	0.00041 12562 14% -3.55 -0.0028260 3978 0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09	0.00094 12562 100% -3.55 -0.00028260 401.6 0.00402 1.000001 -2.7E-07 -0.000013 -1.1E-09 -1.1E-09 -149 100% 1 24	0.013 12562 58% -3.55 -0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 -0.0001 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -1.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.0011 -0.5E-08 -0.5E-	0.00097 12562 100% -3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.0000039 -3.1E-10 -3.1E-10 149	0.0019 12562 100% -3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.000033 -2.7E-10 -2.7E-10 149	0.00148 12562 19% -3.55 -0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09 
Canterbury LGA           Total Population in study area:         12562           % population in assessment age-group:         58%,           total change         -3.55           Population weighted Ax (µg/m <sup>2</sup> );         -0.0028260           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF);         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         Canterbury (South) - Campsie           Canterbury (South) - Campsie         -           Total Population in study area:         149           % population in assessment age-group:         58%,           0.00032215         Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.00048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431	12562 14% -3.55 -0.00282800 9235 0.09235 1.00000 -2.3E-07 -0.00036 -2.1E-08 149 14% 1.24 0.00832215	12562 14% -3.55 -0.00028260 3978 1.000000 -1.2E-07 -0.0000080 -4.6E-09 -45.6E-09 -45.6E-09 -45.6E-09 -45.6E-09 -45.6E-09 -45.6	12562 100% -3.55 -0.00028260 401.6 0.00402 1.000000 -2.7E-07 -0.000013 -1.1E-09 149 100% 124	12562 58% -3.55 -0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08	12562 100% -3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.000039 -3.1E-10 -3.1E-10 149	12562 100% -3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 -2.7E-10	12562 19% -3.55 -0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.00012 -5.1E-09
Total Population in study area:         12562           % population in assessment age-group:         58%           total change         -3.55           Population weighted Δx (µg/m³):         -0.00028260           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.99998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         Canterbury (South) - Campsie           Total Population in study area:         149           % population weighted Δx (µg/m³):         0.000832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         Total Population in	12562 14% -3.55 -0.0028260 9235 1.000000 -2.3E-07 -0.000036 -2.1E-08 149 14% 1.24 0.00832215	12562 14% -3.55 -0.00028260 3978 1.000000 -1.2E-07 -0.0000800 -4.6E-09 -4.6E-09 -149 149 14%	12562 100% -3.55 -0.00028260 401.6 0.00402 1.000000 -2.7E-07 -0.000013 -1.1E-09 149 100% 124	12562 58% -3.55 -0.00028260 4112.0 0.0993996 -3.7E-06 -0.00011 -1.5E-08 -149 58%	12562 100% -3.55 -0.00028260 113.3 1.00000 -2.7E-07 -0.0000039 -3.1E-10 -3.1E-10	12562 100% -3.55 -0.00028260 49.4 0.999999 -5.4E-07 -0.0000033 -2.7E-10 149	12562 19% -3.55 -0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09
% population in assessment age-group:         58%           total change         -3.55           Population weighted Δx (µg/m <sup>3</sup> ):         -0.00028260           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per 100,000) (as per Table 4-5)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         Canterbury (South) - Campsie           Total Population in study area:         149           % population weighted Δx (µg/m <sup>3</sup> ):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Relative Risk:         1.00048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Mingsgrove (North) - Earlwood         Risk:         5.0E-07           Kingsgrove (North) - Earlwood         Total Population in study area:         12413           % population in assessment age-group:         58%         58%	14% -3.55 -0.00028260 9235 0.09235 1.000000 -2.3E-07 -0.000036 -2.1E-08 	14% -3.55 -0.00028260 3978 0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09 	100% -3.55 -0.00028260 401.6 0.00402 1.0000013 -2.7E-07 -0.000013 -1.1E-09 	58% -3.55 -0.00028260 0.000412 0.999996 -3.7E-06 -0.00011 -1.5E-08 	100% -3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.0000039 -3.1E-10	100% -3.55 -0.00028260 49.4 0.099999 -5.4E-07 -0.0000033 -2.7E-10 -2.7E-10	19% -3.55 -0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09
total change         -3.55           Population weighted Δx (µg/m³):         -0.00028260           Baseline Incidence (per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         1026           Canterbury (South) - Campsie         124           Total Population in study area:         149           % population weighted Δx (µg/m³):         0.00032215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.000043           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.000043           Attributable fraction (AF):         4.8E-05           Increased nu	3.55 -0.00028260 9235 0.09235 1.000000 -2.3E-07 -0.000036 -2.1E-08 149 14% 1.24 0.00832215	-3.55 -0.00028260 .0.3378 0.03378 1.000000 -1.2E-07 -0.000080 -4.6E-09 	-3.55 -0.00028260 401.6 0.00402 1.000001 -2.7E-07 -0.000013 -1.1E-09 	-3.55 -0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08	3.55 -0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.0000039 -3.1E-10 -3.1E-10 149	-3.55 -0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.000033 -2.7E-10 -2.7E-10	-3.55 -0.0028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09
Population weighted Δx (µg/m <sup>3</sup> ):         -0.00028260           Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         Sisk:           Canterbury (South) - Campsie         -           Total Population in study area:         149           % population in assessment age-group:         58%           total change         1.24           Population weighted Δx (µg/m <sup>5</sup> ):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Relative Risk:         1.00048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.000043           Attributable fraction in study area:         1.240           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         Risk:           Total Population in study area:         12413           % population in assessmen	-0.00028260 9235 0.09235 1.000000 -2.3E-07 -0.000036 -2.1E-08 149 14% 1.24 0.00832215	-0.00028260 3378 0.03978 1.000000 -1.2E-07 -0.0000080 -4.6E-09 -44.6E-00 -44.6E-09 -45.6E-09 -45.6E-09 -45.6E-09 -45.6E-09 -45.6E-09 -45.6E-09 -45	-0.00028260 401.6 0.00402 1.000000 -2.7E-07 -0.000013 -1.1E-09 149 100% 124	-0.00028260 412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 -149 58%	-0.00028260 113.3 0.00113 1.000000 -2.7E-07 -0.0000039 -3.1E-10 	-0.00028260 49.4 0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 149	-0.00028260 1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09
Baseline Incidence (per 100,000) (as per Table 4-5)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         0.99998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         Risk:           Canterbury (South) - Campsie         149           % population in study area:         149           % population weighted Δx (µg/m <sup>3</sup> ):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per scon)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         7014 Population in study area:           Total Population in study area:         12413           % population in assessment age-group:         58%           total change         4.48	9235 0.09235 1.000000 -2.3E-07 -0.000036 -2.1E-08 	3978 0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09 	401.6 0.00402 1.000000 -2.7E-07 -0.000013 -1.1E-09 	412.0 0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 	113.3 0.00113 1.00000 -2.7E-07 -0.0000039 -3.1E-10 	49.4 0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 	1209.0 0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09
Baseline Incidence (per person)         0.01026           Relative Risk:         0.999998           Attributable fraction (AF):         -1.6E-06           Increased number of cases in population:         -0.00012           Risk:         -1.7E-08           Individual subrubs within LGA         -0.0012           Canterbury (South) - Campsie         -1.7E-08           Total Population in study area:         149           % population in assessment age-group:         58%           total change         1.24           Population weighted Δx (µg/m <sup>3</sup> ):         0.000832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         -1021 Population in study area:           Total Population in study area:         12413           % population in assessment age-group:         58%           total change         4.8	0.09235 1.00000 -2.3E-07 -0.00036 -2.1E-08 	0.03978 1.000000 -1.2E-07 -0.000080 -4.6E-09 	0.00402 1.000000 -2.7E-07 -0.000013 -1.1E-09 	0.00412 0.999996 -3.7E-06 -0.00011 -1.5E-08 	0.00113 1.00000 -2.7E-07 -0.000039 -3.1E-10	0.00049 0.999999 -5.4E-07 -0.0000033 -2.7E-10 	0.01209 1.000000 -4.2E-07 -0.000012 -5.1E-09 
Relative Risk:       0.999998         Attributable fraction (AF):       -1.6E-06         Increased number of cases in population:       -0.00012         Risk:       -1.7E-08         Individual subrubs within LGA       Canterbury (South) - Campsie         Total Population in study area:       149         % population in assessment age-group:       58%         total change       1.24         Population weighted Ax (µg/m <sup>2</sup> ):       0.00832215         Baseline Incidence (per 100,000) (as per Table 4.4)       1026         Baseline Incidence (per 100,000) (as per Table 4.4)       1026         Baseline Incidence (per sperson)       0.01026         Relative Risk:       1.00048         Attributable fraction (AF):       4.8E-05         Increased number of cases in population:       0.0000431         Risk:       5.0E-07         Kingsgrove (North) - Earlwood       Total Population in study area:       12413         % population in assessment age-group:       58%       58%         total change       4.8       65%	1.000000 -2.3E-07 -0.000036 -2.1E-08 149 14% 1.24 0.00832215	1.00000 -1.2E-07 -0.000080 -4.6E-09 149 14% 1.24	1.000000 -2.7E-07 -0.000013 -1.1E-09 149 100% 1 24	0.999996 -3.7E-06 -0.00011 -1.5E-08 	1.000000 -2.7E-07 -0.0000039 -3.1E-10 149	0.999999 -5.4E-07 -0.0000033 -2.7E-10 149	1.000000 -4.2E-07 -0.000012 -5.1E-09
Attributable fraction (AF):       -1.6E-06         Increased number of cases in population:       -0.00012         Risk:       -1.7E-08         Individual subrubs within LGA       Canterbury (South) - Campsie         Total Population in study area:       149         % population in assessment age-group:       58%         total Population in study area:       149         % population in assessment age-group:       58%         total change       1.24         Population weighted $\Delta x$ (µg/m <sup>3</sup> ):       0.00832215         Baseline Incidence (per 100,000) (as per Table 4.4)       1026         Baseline Incidence (per groson)       0.01026         Relative Risk:       1.000048         Attributable fraction (AF):       4.8E-05         Increased number of cases in population:       0.0000431         Risk:       5.0E-07         Kingsgrove (North) - Earlwood       Total Population in study area:       12413         % population in assessment age-group:       58%         total change       4.8       65%	-2.3E-07 -0.000036 -2.1E-08 149 14% 1.24 0.00832215	-1.2E-07 -0.000080 -4.6E-09 149 14% 1.24	-2.7E-07 -0.000013 -1.1E-09 149 100% 1 24	-3.7E-06 -0.00011 -1.5E-08 149 58%	-2.7E-07 -0.0000039 -3.1E-10 149	-5.4E-07 -0.0000033 -2.7E-10 149	-4.2E-07 -0.000012 -5.1E-09
Increased number of cases in population: Risk: -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.7E-08 -1.24	-0.00036 -2.1E-08 149 14% 1.24 0.00832215	-0.000080 -4.6E-09 149 14% 1.24	-0.000013 -1.1E-09 149 100% 1 24	-0.00011 -1.5E-08 -149 58%	-0.0000039 -3.1E-10 	-0.0000033 -2.7E-10 	-0.000012 -5.1E-09
Risk:       -1.7E-08         Individual subrubs within LGA         Canterbury (South) - Campsie         Total Population in study area:       149         % population in assessment age-group:       58%         total change       1.24         Population weighted ∆x (µg/m³):       0.00832215         Baseline Incidence (per 100,000) (as per Table 4.4)       1026         Baseline Incidence (per person)       0.01026         Relative Risk:       1.000048         Attributable fraction (AF):       4.8E-05         Increased number of cases in population:       0.0000431         Risk:       5.0E-07         Kingsgrove (North) - Earlwood       Total Population in study area:       12413         % population in assessment age-group:       58%         total change       4.8	-2.1E-08 149 14% 1.24 0.00832215	-4.6E-09 149 14% 1.24	-1.1E-09 149 100% 1 24	-1.5E-08 149 58%	-3.1E-10 149	-2.7E-10 149	-5.1E-09
Individual subrubs within LGA Canterbury (South) - Campsie Total Population in study area: 149 % population in assessment age-group: 58% total change 1.24 Population weighted Δx (µg/m <sup>3</sup> ): 0.00832215 Baseline Incidence (per 100,000) (as per Table 4.4) 1026 Baseline Incidence (per preson) 0.01026 Relative Risk: 1.000048 Attributable fraction (AF): 4.8E-05 Increased number of cases in population: 0.0000431 Risk: 5.0E-07 Kingsgrove (North) - Earlwood Total Population in study area: 12413 % population in assessment age-group: 58% total change 4.8	149 14% 1.24 0.00832215	149 14% 1.24	149 100% 1 24	149 58%	149	149	149
Canterbury (South) - Campsie           Total Population in study area:         149           % population in assessment age-group:         58%           total change         1.24           Population weighted Δx (µg/m³):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         Total Population in study area:         12413           % population in assessment age-group:         58%         total change         4.8	149 14% 1.24 0.00832215	149 14% 1.24	149 100% 1 24	149 58%	149	149	149
Total Population in study area:         149           % population in assessment age-group:         58%           total change         1.24           Population weighted ∆x (µg/m³):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         Total Population in study area:         12413           % population in assessment age-group:         58%           total change         4.8	149 14% 1.24 0.00832215	149 14% 1.24	149 100% 1 24	149 58%	149	149	149
% population in assessment age-group:         58%, total change         1.24           Population weighted Δx (µg/m <sup>2</sup> ):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.00048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         12413           % population in study area:         12413           % population in assessment age-group:         58%, total change         4.8	14% 1.24 0.00832215	14% 1.24	100%	58%			145
total change         1.24           Population weighted Δx (µg/m³):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         Total Population in study area:           % population in assessment age-group:         58%           total change         -4.8	0.00832215	1.24	1 24		100%	100%	19%
Population weighted Δx (µg/m <sup>3</sup> ):         0.00832215           Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         1.000048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         Total Population in study area:           % population in assessment age-group:         58%           total change         -4.8	0.00832215	0.0000015	1.24	1.24	1.24	1.24	1.24
Baseline Incidence (per 100,000) (as per Table 4.4)         1026           Baseline Incidence (per person)         0.01026           Relative Risk:         10.00048           Attributable fraction (AF):         4.8E-05           Increased number of cases in population:         0.00043           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         12413           % population in assessment age-group:         58%           total change         -4.8		0.00832215	0.00832215	0.00832215	0.00832215	0.00832215	0.00832215
Baseline Incidence (per person)       0.01026         Relative Risk:       1.000048         Attributable fraction (AF):       4.8E-05         Increased number of cases in population:       0.0000431         Risk:       5.0E-07         Kingsgrove (North) - Earlwood       12413         % population in assessment age-group:       58%         total change       -4.8	9235	3978	401.6	412.0	113.3	49.4	1209.0
Relative Risk:       1.000048         Attributable fraction (AF):       4.8E-05         Increased number of cases in population:       0.0000431         Risk:       5.0E-07         Kingsgrove (North) - Earlwood       7         Total Population in study area:       12413         % population in assessment age-group:       58%         total change       -4.8	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209
Attributable fraction (AF): 4.8E-05 Increased number of cases in population: 0.0000431 Risk: 5.0E-07 Kingsgrove (North) - Earlwood Total Population in study area: 12413 % population in assessment age-group: 58% total change -4.8	1.000007	1.000003	1.000008	1.000108	1.000008	1.000016	1.000012
Increased number of cases in population:         0.0000431           Risk:         5.0E-07           Kingsgrove (North) - Earlwood         12413           Total Population in study area:         12413           % population in assessment age-group:         58%           total change         -4.8	6.7E-06	3.4E-06	7.8E-06	1.1E-04	8.1E-06	1.6E-05	1.2E-05
Risk:     5.0E-07       Kingsgrove (North) - Earlwood        Total Population in study area:     12413       % population in assessment age-group:     58%       total change     -4.8	0.0000127	0.0000028	0.0000047	0.0000388	0.0000014	0.0000012	0.0000043
Kingsgrove (North) - Earlwood Total Population in study area: 12413 % population in assessment age-group: 58% total change -4.8	6.1E-07	1.4E-07	3.1E-08	4.5E-07	9.1E-09	7.8E-09	1.5E-07
Total Population in study area: 12413 % population in assessment age-group: 58% total change -4.8							
% population in assessment age-group: 58% total change -4.8	12413	12413	12413	12413	12413	12413	12413
total change -4.8	14%	14%	100%	58%	100%	100%	19%
	-4.8	-4.8	-4.8	-4.8	-4.8	-4.8	-4.8
Population weighted Δx (µg/m <sup>3</sup> ): -0.00038669	-0.00038669	-0.00038669	-0.00038669	-0.00038669	-0.00038669	-0.00038669	-0.00038669
Baseline Incidence (per 100,000) (as per Table 4.4) 1026	9235	3978	401.6	412.0	113.3	49.4	1209.0
Baseline Incidence (per person) 0.01026	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209
Relative Risk: 0.999998	1.000000	1.000000	1.000000	0.999995	1.000000	0.999999	0.999999
Attributable fraction (AF): -2.2E-06	0.45.07	-1.6E-07	-3.6E-07	-5.0E-06	-3.8E-07	-7.3E-07	-5.7E-07
Increased number of cases in population: -0.00017	-3.1E-07	0 000011	-0.000018	-0.00015	-0.0000053	-0.0000045	-0.000016
KISK: -2.3E-08	-3.1E-07 -0.000049	-0.000011	1 55 00	245.00	4 25 40	-3.0E-10	-p.ar-0a
I	-3.1E-07 -0.000049 -2.9E-08	-0.000011 -6.3E-09	-1.5E-09	-2.1E-08	-4.2E-10		

		Primary Indicator	S		Se	Secondary Indicators		
Health Endpoint	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Botany LGA								
Total Population in study area	31386	31386	31386	31386	31386	31386	31386	31386
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-791.2	-791.2	-791.2	-791.2	-791.2	-791.2	-791.2	-791.2
Population weighted Ax (ug/m <sup>3</sup> )	-0.02520869	-0.02520869	-0.02520869	-0.02520869	-0.02520869	-0.02520869	-0.02520869	-0.02520869
Baseline Incidence (per 100 000) (as per Table 4-5	1026	9235	3978	501.7	412.0	133.6	49.4	1209 0
Baseline Incidence (per recipient) (de per rabie reci	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0 00049	0.01200
Relative Risk	0.999854	0.00200	0.00070	0.00002	0.999672	0.999976	0.00040	0.999963
Attributable fraction (AE)	-1.5E-04	-2 0E-05	-1 0E-05	-2 4F-05	-3.3E-04	-2 4E-05	-4 8E-05	-3 7E-05
Increased number of cases in population	-0.028	-0.0076	-0.0017	-0.0037	-0.025	-0.0010	-0.00074	-0.0022
Risk	-1.5E-06	-1.9E-06	-4.1E-07	-1.2E-07	-1.4E-06	-3.3E-08	-2.4E-08	-4.5E-07
Individual subrubs within LGA	1.02.00	1.02 00		1.22 01		0.02 00	2.12.00	
Botany	,							
Total Population in study area	· 10408	10408	10408	10408	10408	10408	10408	10408
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-245.3	-245.3	-245.3	-245.3	-245.3	-245.3	-245.3	-245.3
Population weighted Ax (ug/m <sup>3</sup> )	-0.023568/11	-0.023568/11	-0.023568/1	-0.023568/11	-0.023568/1	-0.023568/1	-0.023568/1	-0.023568/11
Baseline Incidence (per 100 000) (as per Table 4.4	1026	-0.02000041	-0.02330041	-0.0200041	-0.02330041	-0.02000041	-0.0200041	1209.0
Baseline Incidence (per 100,000) (as per 1able 4.4	0.01026	0.00225	0.02079	0.00502	912.0	0.00124	0.00040	0.01203
Baseline Incidence (per person	0.01020	0.09233	0.03978	0.00502	0.00412	0.00134	0.00049	0.01209
Attributable fraction (AF)	-1 4E-04	-1 9E-05	-9.7E-06	-2 2E-05	-3 1E-04	-2 3E-05	-4 5E-05	-3 5E-05
Increased number of cases in population	-0.0087	-1.92-03	-0.00052	-2.22-03	-0.0079	-2.32-03	-4.52-03	-0.0007
Piek	-0.0007	-0.0024	-0.00032	-0.0012	-0.0075	-0.00032	-0.00023	-4.2E-07
Mascot - Fastlakes	-1.42-00	-1.72-00	-3.02-07	-1.12-07	-1.52-00	-3.12-00	-2.2L-00	-4.22-07
Total Population in study area	20286	20286	20286	20286	20286	20286	20286	20286
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-552.1	-552.1	-552 1	-552.1	-552.1	-552.1	-552 1	-552 1
Bopulation weighted Ax (ug/m <sup>3</sup> )	0.02721591	0.02721591	0.02721591	0.02721591	0.02721591	0.02721591	0.02721591	0.02721591
Baseline Incidence (per 100 000) (as per Table 4.4	1026	-0.02721381	-0.02721301	-0.02721381	-0.02721301	133.6	-0.02721361	1209 (
Baseline Insidence (per 100,000) (as per 1able 4.4	0.01026	0.00235	0.03078	0.00503	0.00412	0.00134	0.00040	0.01200.0
Baseline Incidence (per person	0.01020	0.09233	0.03978	0.00302	0.00412	0.00134	0.00049	0.01208
Attributable fraction (AE)	-1 6E-04	-2 2E-05	-1 1E-05	-2 6E-05	-3 5E-04	-2 6E-05	-5 2E-05	-4 0E-05
Increased number of cases in population	-0.0196	-0.0053	-0.0012	-0.0026	-0.0177	-0.0007	-0.0005	-0.0016
Risk	-1 6E-06	-2 0E-06	-4 4F-07	-1.3E-07	-1.5E-06	-3.5E-08	-2 6E-08	-4 9E-07
Pagewood - Hillsdale - Dacevville	1.02.00	2.02.00		1.02 01	1.02 00	0.02 00	2.02.00	
Total Population in study area	631	631	631	631	631	631	631	631
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	9	9	9	9	9	9	9	g
Population weighted Ax (ug/m <sup>3</sup> )	0.01426307	0.01426307	0.01426307	0.01426307	0.01426307	0.01426307	0.01426307	0.01426307
Baseline Incidence (per 100 000) (as per Table 4 4	1026	9235	3978	501.7	412.0	133.6	49.4	1209.0
Baseline Incidence (per recipied) (de per rabie in r	0.01026	0.00235	0.03978	0.00502	0.00/12	0.00134	0.00049	0.01200
Relative Risk	1 000083	1 000011	1 000006	1 000013	1 000185	1 000014	1 000027	1 000021
Attributable fraction (AE)	8.3E-05	1 1E-05	5.8E-06	1.3E-05	1.000100 1.9F-04	1 4F-05	2 7E-05	2 1F-05
Increased number of cases in population	0.00032	0.00009	0.00019	0.000042	0.000288	0.000012	0.0000084	0.000025
Risk	8.5E-07	1.1E-06	2.3E-07	6.7E-08	7.6E-07	1.8E-08	1.3E-08	2.6E-07
Svdnev Airpor	t							
Total Population in study area	. 61	61	61	61	61	61	61	61
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-2.8	-2.8	-2.8	-2.8	-2.8	-2.8	-2.8	-2.8
Population weighted Ax (ug/m <sup>3</sup> )	-0.04590164	-0.04590164	-0.04590164	-0.04590164	-0.04590164	-0.04590164	-0.04590164	-0.04590164
Baseline Incidence (per 100.000) (as per Table 4.4	1026	9235	3978	501 7	412 0	133.6	49.4	1209 (
Baseline Incidence (per person	0.01026	0 00235	0 03078	0.00502	0.00/12	0.00134	0.00040	0.01200
Relative Rick	0.999734	0.09200	0.03978	0 999957	0.999403	0 999955	0.00049	0.01208
Attributable fraction (AF)	-2.7E-04	-3.7E-05	-1.9E-05	-4.3E-05	-6.0E-04	-4.5E-05	-8.7E-05	-6.8E-05
Increased number of cases in population	-0.000100	-0.000027	-0.0000059	-0.000013	-0.000090	-0.0000036	-0.0000026	-0.0000079
Risk	-2.7E-06	-3.4E-06	-7.5E-07	-2.2E-07	-2.5E-06	-5.9E-08	-4.3E-08	-8.2E-07
					,			
-	-	•	•	-	•	•	•	•

	Primary Indicators				•			
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
B (change in effect per 1 µg/m PM) (as per 1 able 6-23) Kogarah - Rockdale LGA	0.0038	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Total Population in study area:	113547	113547	113547	113547	113547	113547	113547	113547
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	414.5	414.5	414.5	414.5	414.5	414.5	414.5	414.5
Baseline Incidence (per 100.000) (as per Table 4-5)	0.00365047	0.00365047	0.00365047	0.00365047	0.00365047	0.00365047	0.00365047	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	1.000021	1.000003	1.000001	1.000003	1.000047	1.000004	1.000007	1.000005
Attributable fraction (AF):	2.1E-05	2.9E-06	1.5E-06	3.4E-06	4.7E-05	3.5E-06	6.9E-06	5.4E-06
Risk:	2.2E-07	2.7E-07	6.0E-08	1.4E-08	2.0E-07	3.9E-09	3.4E-09	6.5E-08
Individual subrubs within LGA								
Arncliffe - Bardwell Park	04.457	04457	04.457	04.457	04457	04.457	04.457	04457
% population in assessment age-group:	62%	21457	21457	21457	62%	21457	21457	21457
total change	-500.3	-500.3	-500.3	-500.3	-500.3	-500.3	-500.3	-500.3
Population weighted Δx (µg/m <sup>3</sup> ):	-0.02331640	-0.02331640	-0.02331640	-0.02331640	-0.02331640	-0.02331640	-0.02331640	-0.02331640
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	6 49.4	1209.0
Baseline Incidence (per person) Relative Risk:	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Attributable fraction (AF):	-1.4E-04	-1.9E-05	-9.6E-06	-2.2E-05	-3.0E-04	-2.3E-05	6 -4.4E-05	-3.5E-05
Increased number of cases in population:	-0.0183	-0.0057	-0.00125	-0.00186	-0.0165	-0.00054	-0.00047	-0.00131
Risk:	-1.4E-06	-1.7E-06	-3.8E-07	-8.7E-08	-1.2E-06	-2.5E-08	-2.2E-08	-4.2E-07
Total Population in study area:	20002	20002	20002	20002	20002	20002	20002	20002
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	41.6	41.6	41.6	41.6	41.6	41.6	41.6	41.6
Population weighted Δx (µg/m <sup>-</sup> ): Baseline Incidence (per 100 000) (as per Table 4 4)	0.00207979	0.00207979	0.00207979	0.00207979	0.00207979	0.00207979	0.00207979	0.00207979
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	1.000012	1.000002	1.000001	1.000002	1.000027	1.000002	1.000004	1.000003
Attributable fraction (AF):	1.2E-05	1.7E-06	8.5E-07	2.0E-06	2.7E-05	2.0E-06	6 4.0E-06	3.1E-06
Risk:	1.2E-07	1.5E-07	3.4E-08	7.7E-09	1.1E-07	0.000045 2.2E-09	2.0E-09	3.7E-08
Kingsgrove (South) - Bardwell Park								
Total Population in study area:	2879	2879	2879	2879	2879	2879	2879	2879
% population in assessment age-group: total change	62% -5.1	-5.1	-5.1	-5.1	62%	-5 1	-5.1	-5.1
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00177145	-0.00177145	-0.00177145	-0.00177145	-0.00177145	-0.00177145	-0.00177145	-0.00177145
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	6 49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Attributable fraction (AF):	0.999990 -1.0E-05	0.999999 -1 4F-06	-7.3E-07	0.999998 -1 7E-06	-2.3E-05	0.999998 -1 7E-06	-3 4F-06	-2 6E-06
Increased number of cases in population:	-0.00019	-0.000058	-0.000013	-0.000019	-0.00017	-0.0000055	-0.000048	-0.000013
Risk:	-1.1E-07	-1.3E-07	-2.9E-08	-6.6E-09	-9.5E-08	-1.9E-09	-1.7E-09	-3.2E-08
Kogarah Total Population in study area	11222	11222	11222	11222	11222	11222	11222	11222
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	789.3	789.3	789.3	789.3	789.3	789.3	8 789.3	789.3
Population weighted $\Delta x (\mu g/m^3)$ :	0.06970767	0.06970767	0.06970767	0.06970767	0.06970767	0.06970767	0.06970767	0.06970767
Baseline incidence (per 100,000) (as per 1able 4.4) Baseline Incidence (per person)	0.01026	9235	0.03978	0.00396	412.0	0.00111	0 00049	0.01209.0
Relative Risk:	1.000404	1.000056	1.000029	1.000066	1.000907	1.000068	1.000132	1.000103
Attributable fraction (AF):	4.0E-04	5.6E-05	2.9E-05	6.6E-05	9.1E-04	6.8E-05	5 1.3E-04	1.0E-04
Increased number of cases in population:	0.029 4 1E-06	0.0089 5.2E-06	0.0020	0.0029 2 6E-07	0.026 3.7E-06	0.00085 7.5E-08	0.00074 6.5E-08	0.0021 1.2E-06
Kogarah Bay - Carlton - Allawah	4.1E-00	0.2L-00	1.12-00	2.02-07	5.7E-00	7.52-00	0.52-00	1.22-00
Total Population in study area:	10923	10923	10923	10923	10923	10923	10923	10923
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
Population weighted Ax (ug/m <sup>3</sup> ):	0.02735512	0.02735512	290.0	0.02735512	0.02735512	290.0	0 02735512	0.02735512
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	5.02700012 6 49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	1.000159	1.000022	1.000011	1.000026	1.000356	1.000027	1.000052	1.000040
Attributable fraction (AF): Increased number of cases in population:	0.011	2.2E-05	0.00075	0.0011	3.6E-04 0.0098	0.00032	0.00028	4.0E-05
Risk:	1.6E-06	2.0E-06	4.5E-07	1.0E-07	1.5E-06	2.9E-08	3 2.6E-08	4.9E-07
Monterey - Brighton-le-Sands - Kyeemagh	10015	10015	10015	40045	10015	40045	10015	10015
l otal Population in study area: % population in assessment age-group;	13915	13915	13915	13915	13915	13915	13915	13915
total change	-208.4	-208.4	-208.4	-208.4	-208.4	-208.4	-208.4	-208.4
Population weighted $\Delta x (\mu g/m^3)$ :	-0.01497664	-0.01497664	-0.01497664	-0.01497664	-0.01497664	-0.01497664	-0.01497664	-0.01497664
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	6 49.4	1209.0
Baseline Incidence (per person) Relative Risk	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Attributable fraction (AF):	-8.7E-05	-1.2E-05	-6.1E-06	-1.4E-05	-1.9E-04	-1.5E-05	-2.8E-05	-2.2E-05
Increased number of cases in population:	-0.0076	-0.0024	-0.00052	-0.00078	-0.0069	-0.00022	-0.00020	-0.00054
Risk: Rockdale - Banksia	-8.9E-07	-1.1E-06	-2.4E-07	-5.6E-08	-8.0E-07	-1.6E-08	-1.4E-08	-2.7E-07
Total Population in study area:	19957	19957	19957	19957	19957	19957	19957	19957
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
	-283.1	-283.1	-283.1	-283.1	-283.1	-283.1	-283.1	-283.1
Baseline Incidence (per 100.000) (as per Table 4.4)	-0.01418550	-0.01418550	-0.01418550	-0.01418550	-0.01418550	-0.01418550	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	0.999918	0.999989	0.999994	0.999987	0.999816	0.999986	0.999973	0.999979
Attributable fraction (AF):	-8.2E-05	-1.1E-05	-5.8E-06	-1.3E-05	-1.8E-04	-1.4E-05	-2.7E-05	-2.1E-05
Risk:	-8.4E-07	- <u>1.0E-</u> 06	-2.3E-07	-5.3E-08	-7.6E-07	-1.5E-08	- <u>1.3E-</u> 08	-2.5E-07
Sans Souci - Ramsgate								
Total Population in study area:	13091	13091	13091	13091	13091	13091	13091	13091
total change	281.6	281.6	281.6	281.6	281.6	281.6	281.6	281.6

		Primary Indicator	s	Secondary Indicators					
Health Endpoint	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term	
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years	
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148	
Population weighted $\Delta x (\mu g/m^3)$	0.02151096	0.02151096	0.02151096	0.02151096	0.02151096	0.02151096	0.02151096	0.02151096	
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	49.4	1209.0	
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209	
Relative Risk	1.000125	1.000017	1.000009	1.000020	1.000280	1.000021	1.000041	1.000032	
Attributable fraction (AF)	1.2E-04	1.7E-05	8.8E-06	2.0E-05	2.8E-04	2.1E-05	4.1E-05	3.2E-05	
Increased number of cases in population	0.010	0.0032	0.00070	0.0010	0.0093	0.00030	0.00026	0.00074	
Risk	1.3E-06	1.6E-06	3.5E-07	8.0E-08	1.2E-06	2.3E-08	2.0E-08	3.8E-07	

	Primary Indicators			Secondary Indicators					
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term	
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years	
β (change in effect per 1 µg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148	
Hurstville LGA									
Total Population in study area	657	657	657	657	657	657	657	657	
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%	
total change	9.04	9.04	9.04	9.04	9.04	9.04	9.04	9.04	
Population weighted $\Delta x (\mu g/m^3)$ :	0.01375951	0.01375951	0.01375951	0.01375951	0.01375951	0.01375951	0.01375951	0.01375951	
Baseline Incidence (per 100,000) (as per Table 4-5)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0	
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0.00049	0.01209	
Relative Risk:	1.000080	1.000011	1.000006	1.000013	1.000179	1.000013	1.000026	1.000020	
Attributable fraction (AF)	8.0E-05	1.1E-05	5.6E-06	1.3E-05	1.8E-04	1.3E-05	2.6E-05	2.0E-05	
Increased number of cases in population:	0.00033	0.00010	0.000023	0.000039	0.00030	0.000011	0.0000085	0.000024	
Risk	8.2E-07	1.0E-06	2.2E-07	6.0E-08	7.4E-07	1.7E-08	1.3E-08	2.5E-07	
Individual subrubs within LGA									
Hurstville									
Total Population in study area	96	96	96	96	96	96	96	96	
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%	
total change	0.97	0.97	0.97	0.97	0.97	0.97	0.97	0.97	
Population weighted Δx (ug/m <sup>3</sup> ):	0.01010417	0.01010417	0.01010417	0.01010417	0.01010417	0.01010417	0.01010417	0.01010417	
Baseline Incidence (per 100.000) (as per Table 4.4)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0	
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0 00049	0.01209	
Relative Risk:	1.000059	1.000008	1.000004	1.000009	1.000131	1.000010	1.000019	1.000015	
Attributable fraction (AF)	5.9E-05	8.1E-06	4.1E-06	9.5E-06	1.3E-04	9.8E-06	1.9E-05	1.5E-05	
Increased number of cases in population:	0.000035	0.0000110	0.0000024	0.0000042	0.000032	0.0000012	0.00000091	0.0000025	
Risk	6.0E-07	7.5E-07	1.6E-07	4.4E-08	5.4E-07	1.2E-08	9.5E-09	1.8E-07	
South Hurstville - Blakehurst									
Total Population in study area	561	561	561	561	561	561	561	561	
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%	
total change	8.07	8.07	8.07	8.07	8.07	8.07	8.07	8.07	
Population weighted Δx (ug/m <sup>3</sup> ):	0.01438503	0.01438503	0.01438503	0.01438503	0.01438503	0.01438503	0.01438503	0.01438503	
Baseline Incidence (per 100.000) (as per Table 4.4)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0	
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0 00049	0.01209	
Relative Risk:	1.000083	1.000012	1.000006	1.000014	1.000187	1.000014	1.000027	1.000021	
Attributable fraction (AF)	8.3E-05	1.2E-05	5.9E-06	1.4E-05	1.9E-04	1.4E-05	2.7E-05	2.1E-05	
Increased number of cases in population:	0.00030	0.000091	0.000020	0.000035	0,00027	0.000010	0.0000076	0.000021	
Risk	8.6E-07	1.1E-06	2.3E-07	6.3E-08	7.7E-07	1.8E-08	1.4E-08	2.6E-07	
	0.02 01								
Total population incidence - All Suburbs	-0.030	-0.0068	-0.0015	-0.0041	-0.027	-0.0011	-0.00077	-0.0022	
## Assessment of Increased Incidence - PM<sub>2.5</sub> F6 Extension: 2036

	Primary Indicators Secondary Indicators					;		
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Strathfield - Burwood - Ashfield LGA				1				
Total Population in study area	20160	20160	20160	20160	20160	20160	20160	20160
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-45.3	-45.3	-45.3	-45.3	-45.3	-45.3	-45.3	-45.3
Population weighted Δx (ug/m <sup>3</sup> ):	-0.00224702	-0.00224702	-0.00224702	-0.00224702	-0.00224702	-0.00224702	-0.00224702	-0.00224702
Baseline Incidence (per 100,000) (as per Table 4-5)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk:	0.999987	0.999998	0.999999	0.999998	0.999971	0.999998	0.999996	0.999997
Attributable fraction (AF)	-1.3E-05	-1.8E-06	-9.2E-07	-2.1E-06	-2.9E-05	-2.2E-06	-4.3E-06	-3.3E-06
Increased number of cases in population:	-0.0017	-0.00041	-0.000090	-0.00014	-0.0015	-0.000043	-0.000043	-0.00011
Risk	-1.3E-07	-1.7E-07	-3.7E-08	-7.1E-09	-1.2E-07	-2.2E-09	-2.1E-09	-4.0E-08
Individual subrubs within LGA								
Ashfield								
Total Population in study area	1512	1512	1512	1512	1512	1512	1512	1512
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	6.64	6.64	6.64	6.64	6.64	6.64	6.64	6.64
Population weighted Δx (µg/m <sup>3</sup> ):	0.00439153	0.00439153	0.00439153	0.00439153	0.00439153	0.00439153	0.00439153	0.00439153
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk	1.000025	1.000004	1.000002	1.000004	1.000057	1.000004	1.00008	1.000006
Attributable fraction (AF)	2.5E-05	3.5E-06	1.8E-06	4.1E-06	5.7E-05	4.3E-06	8.3E-06	6.5E-06
Increased number of cases in population:	0.00025	0.000060	0.000013	0.000021	0.00023	0.000006	0.0000062	0.000017
Risk	2.6E-07	3.2E-07	7.2E-08	1.4E-08	2.4E-07	4.2E-09	4.1E-09	7.9E-08
Canterbury (North) - Ashbury	r							
Total Population in study area	7179	7179	7179	7179	7179	7179	7179	7179
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-29.6	-29.6	-29.6	-29.6	-29.6	-29.6	-29.6	-29.6
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00412314	-0.00412314	-0.00412314	-0.00412314	-0.00412314	-0.00412314	-0.00412314	-0.00412314
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk:	0.999976	0.999997	0.999998	0.999996	0.999946	0.999996	0.999992	0.999994
Attributable fraction (AF)	-2.4E-05	-3.3E-06	-1.7E-06	-3.9E-06	-5.4E-05	-4.0E-06	-7.8E-06	-6.1E-06
Increased number of cases in population:	-0.0011	-0.00027	-0.000059	-0.000093	-0.0010	-0.00003	-0.000028	-0.000075
Risk	-2.5E-07	-3.0E-07	-6.7E-08	-1.3E-08	-2.2E-07	-3.9E-09	-3.9E-09	-7.4E-08
Dulwich Hill - Lewisham								
Total Population in study area	11400	11400	11400	11400	11400	11400	11400	11400
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-22.1	-22.1	-22.1	-22.1	-22.1	-22.1	-22.1	-22.1
Population weighted $\Delta x (\mu g/m^2)$ :	-0.00193860	-0.00193860	-0.00193860	-0.00193860	-0.00193860	-0.00193860	-0.00193860	-0.00193860
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk:	0.999989	0.999998	0.999999	0.999998	0.999975	0.999998	0.999996	0.999997
Attributable fraction (AF)	-1.1E-05	-1.6E-06	-7.9E-07	-1.8E-06	-2.5E-05	-1.9E-06	-3.7E-06	-2.9E-06
Increased number of cases in population:	-0.00084	-0.00020	-0.000044	-0.000070	-0.00076	-0.000021	-0.000021	-0.000056
Haberfield - Summer Hill	-1.2E-07	-1.4E-07	-3.2E-00	-0.1E-09	-1.0E-07	-1.9E-09	-1.0E-09	-3.5E-00
Tatal Bapulation in study area	60	60	60	60	60	60	60	60
10tal Population in accessment age group	649	120/	129/	100%	649/	100%	100%	1/0/
/o population in assessment age-group. total change	-0.28	-0 28	-0.28	-0.28	-0.28	-0.28	-0.28	-0.28
Deputation weighted As (1-1/-3)	0.20	0.00405707	0.00405707	0.00405707	0.00405707	0.00405707	0.00405707	0.00405707
Population weighted ΔX (µg/m))	-0.00405797	-0.00405/9/	-0.00405/9/	-0.00405/9/	-0.00405797	-0.00405797	-0.00405797	-0.00405797
Daseline incluence (per 100,000) (as per Table 4.4)	1026	9230	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Attributable fraction (AE)	-2 /E 05	0.9999997	0.999998 -1 7E 06	-3 8E 06	0.999947	-3 0E UE	-7 7 = 06	-6 0= 06
Autiputable inaction (AF)	-2.4E-03	-3.2E-00	-1.7 =-00	-3.0E-00	-0.3E-00	-3.9E-00	-1.1 =-00	-0.0E-00
Rick	-2 4F-07	-0.0000023	-0.0000000	-0.0000009	-0.0000090 -2 2F-07	-0.0000003	-3.8F-00	-7 3E-09
	-2.46-07	-5.02-07	-0.02-00	-1.50-00	-2.22-07	-5.32=03	-5.02-03	-7.50-00
	1	1		1	1		1	1

		Primary Indicator	s	Secondary Indicators				
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Sydney Inner City LGA								
Total Population in study area	20605	20605	20605	20605	20605	20605	20605	20605
% population in assessment age-group:	58%	8%	8%	100%	58%	100%	100%	6%
total change	-258.6	-258.6	-258.6	-258.6	-258.6	-258.6	-258.6	-258.6
Bopulation weighted Av (ug/m <sup>3</sup> )	0.00970954	0.00970954	0.00970954	0.00970954	0.00970954	0.00970954	0.00970954	0.00970954
Population weighted 2X (pg/m).	-0.00670654	-0.00670634	-0.00670654	-0.00670654	-0.00670634	-0.00070634	-0.00670654	-0.00670654
Baseline Incidence (per 100,000) (as per Table 4-5)	1026	9235	3978	534.2	412.0	140.4	49.4	1209.0
	1						1	
	İ	1			İ		1	l
	1							
	1	1						
							1	
	1	1			1		1	
	1	I						

	Primary Indicators Secondary Indicators					6		
Health Endpoint	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 µg/m <sup>3</sup> PM) (as per Table 6-23	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Marrickville - Sydenham - Petersham LGA								
Total Population in study area	35722	35722	35722	35722	35722	35722	35722	35722
% population in assessment age-group	64%	12%	12%	100%	64%	100%	100%	14%
total change	-471.7	-471.7	-471.7	-471.7	-471.7	-471.7	-471.7	-471.7
Population weighted Δx (µg/m <sup>3</sup> )	-0.01320475	-0.01320475	-0.01320475	-0.01320475	-0.01320475	-0.01320475	-0.01320475	-0.01320475
Baseline Incidence (per 100.000) (as per Table 4-5)	1026	9235	3978	508.0	412.0	138.9	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00508	0.00412	0.00139	0.00049	0.01209
Relative Risk	0.999923	0.999989	0.999995	0.999988	0.999828	0.999987	0.999975	0.999980
Attributable fraction (AF)	-7.7E-05	-1.1E-05	-5.4E-06	-1.2E-05	-1.7E-04	-1.3E-05	-2.5E-05	-2.0E-05
Increased number of cases in population	-0.018	-0.0043	-0.00094	-0.0023	-0.016	-0.00064	-0.00044	-0.0012
Risk	-7.9E-07	-9.8E-07	-2.2E-07	-6.3E-08	-7.1E-07	-1.8E-08	-1.2E-08	-2.4E-07
Individual subrubs within LGA								
Marrickville								
Total Population in study area	25842	25842	25842	25842	25842	25842	25842	25842
% population in assessment age-group	64%	12%	12%	100%	64%	100%	100%	14%
total change	-161.5	-161.5	-161.5	-161.5	-161.5	-161.5	-161.5	-161 5
Population weighted Ax (ug/m <sup>3</sup> )	-0.00624952	-0.00624952	-0.00624952	-0.00624952	-0.00624952	-0.00624952	-0.00624952	-0.00624952
Baseline Incidence (per 100 000) (as per Table 4 4)	1026	-0.00024332	-0.00024332	-0.00024332	-0.00024332	-0.00024332	-0.00024332	1209 0
Baseline Incidence (per 100,000) (as per 1able 4.4)	0.01020	0.00235	0.03079	0.00508	912.0	0.00120	0.00040	0.01203.0
Baseline incluence (per person)	0.01020	0.09235	0.03970	0.00506	0.00412	0.00138	0.00049	0.01205
Attributable fraction (AE)	2 65 05	0.999993 5 0E 06	0.555557	0.999994 5 0E 06	0.555515	6 1 5 0 6	1 25 05	0.3555551
Increased number of cases in population	-3.0L-03	-5.02-00	-2.01-00	-5.92-00	-0.12-03	-0.12-00	0.00015	-9.21-00
Rick	-0.000	-0.0013	-0.00032	-3.0E-08	-0.000 -3 3E-07	-0.00022	-5.9E-00	-0.0004
Betersham - Stanmore	-5.72-07	-4.02-07	-1.02-07	-5.02-00	-0.52-07	-0.46-03	-5.52-03	-1.16-07
Total Population in study area	2051	2051	2051	2051	2051	2051	2051	2051
% population in assessment age-group	6/%	12031	12031	100%	2031	100%	100%	2031
total change	-16.7	-16.7	-16.7	-16.7	-16.7	-16.7	-16.7	-167
Bopulation weighted Av (ug/m <sup>3</sup> )	0.00914007	0.00914227	0.00914007	0.00914007	0.00914007	0.00914007	0.00914227	0.00914227
Baseline Incidence (per 100 000) (as per Table 4 4)	-0.00614237	-0.00014237	-0.00614237	-0.00614237	-0.00614237	-0.00014237	-0.00614237	-0.00614237
Baseline Incidence (per 100,000) (as per 1able 4.4	0.01020	0.00225	0.03078	0.00508	912.0	0.00120	0.00040	0.01203.0
Daseline incluence (per person	0.01020	0.09233	0.03970	0.00506	0.00412	0.00139	0.00049	0.01208
Attributable fraction (AE)	4 75 05	0.555555	2 2 5 06	7 75 06	1 1 5 04	7 05 06	1 55 05	1 25 06
Increased number of cases in population	-4.72-03	-0.32-00	0.00002	-7.72-00	-1.12-04	-7.92-00	0.00002	0.00004
Rick	-0.001	-0.0002	-0.00003	-3.9E-08	-0.001 -4.4E-07	-0.00002	-7.6E-09	-0.0000
Sydenham - Tempe - St Peters	-4.02-07	-0.0L-07	-1.50-07	-5.32-00	-4.42-07	-1.12-00	-7.02-03	-1.52-07
Total Population in study area	7920	7920	7920	7920	7920	7920	7920	7920
% population in assessment age-group	64%	12%	1023	1023	64%	100%	100%	1/023
total change	-203 5	-203 5	-203 5	-203.5	-203 5	-203 5	-203.5	-203 6
Deputation weighted As (/ <sup>3</sup> )	0.02740000	-293.3	-293.3	0.02740000	-293.3	-293.3	-233.3	-290.0
Population weighted ΔX (µg/m)	-0.03748882	-0.03/48882	-0.03/48882	-0.03/48882	-0.03748882	-0.03/48882	-0.03/48882	-0.03/48882
Daseline incidence (per 100,000) (as per 1able 4.4	1026	9235	3978	508.0	412.0	138.9	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00508	0.00412	0.00139	0.00049	0.01209
Relative Risk	0.999783	0.999970	0.999985	0.999965	0.999513	0.999964	0.999929	0.999945
Attributable fraction (AF)	-2.2E-04	-3.0E-05	-1.5E-05	-3.5E-05	-4.9E-04	-3.6E-05	-7.1E-05	-5.5E-05
increased number of cases in population	-0.011	-0.0026	-0.0006	-0.0014	-0.010	-0.0004	-0.00028	-0.00074
Risk	-2.2E-06	-2.8E-06	-0.1E-07	-1.8E-07	-2.0E-06	-5.1E-08	-3.5E-08	-0.7E-07
		1	1		1	1	1	1

Heath Endpoint   Morality - Markey, Long Grafitovacular, Stort-term   Hospitalison, Stort-term   Hospitalison, Stort-term   Hospitalison, Stort-term   Morality - Markey, Stort-term   Morality -		Primary Indicators				Secondary Indicators					
Cases, Long (arm)   Cardiovascular Nort-term   Respiratory, Short-term   Cases, Short (arm)   Cardioputmonal (arm)   Cardioputmonal Short-term   Short-term     6 (change in effect per 1 ug/m <sup>2</sup> PM (as per Table 5-2)   0.0058   0.0008   0.00041   0.0009   0.0019   0.00097   0.00097   0.00097   0.00097   0.00097   0.0019   0.00097   0.00113   0.0013   0.00113   0.0013   0.00197   0.00127   0.00013   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113 <td< th=""><th>Health Endpoint: Mortality</th><th>All Hospitalis</th><th>ations - H</th><th>- Iospitalisations</th><th>Mortality - All</th><th>Mortality -</th><th>Mortality -</th><th>Mortality -</th><th>Morbidity -</th></td<>	Health Endpoint: Mortality	All Hospitalis	ations - H	- Iospitalisations	Mortality - All	Mortality -	Mortality -	Mortality -	Morbidity -		
term   Short-term   term   Long-term   Nort-term   Short-term   All ages   ≥ 30 years   All ages   <	Causes,	ong- Cardiovas	cular, F	Respiratory,	Causes, Short-	Cardiopulmonary,	Cardiovascular,	Respiratory,	Asthma ED		
Age Group:   ≥ 65 years   All ages   ≥ 83 years   All ages   All ages     ß (change in effect per 1 µg/m <sup>2</sup> PM) (as per Table 6-23)   0.0058   0.0008   0.00041   0.00094   0.013   0.00097   0.0019     Canterbury LGA	term	Short-term	n 8	Short-term	term	Long-term	Short-term	Short-term	Admissions -		
Age Group:   ≥ 30 years   ≥ 65 years   All ages   ≥ 30 years   All ages   ≥ 30 years   All ages   0.00097   0.0019   0.00097   0.00097   0.00097   0.00097   0.00097   0.00097   0.00097   0.00097   0.00059973   -0.00569973   -0.00056   -0.00021   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113   0.00113						-			Short-term		
β (change in effect per 1 updm <sup>2</sup> PM) (as per Table 4-23)   0.0087   0.00081   0.00094   0.013   0.00097   0.0019     Canterbury LOS <th>Age Group: ≥ 30 y</th> <th>ars ≥65 ye</th> <th>ears</th> <th>≥ 65 years</th> <th>All ages</th> <th>≥ 30 years</th> <th>All ages</th> <th>All ages</th> <th>1-14 years</th>	Age Group: ≥ 30 y	ars ≥65 ye	ears	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years		
Canterbury L6A        Total Population in study area   12562 </th <th>e in effect per 1 µg/m<sup>3</sup> PM) (as per Table 6-23) 0.00</th> <th>3 0.000</th> <th>08</th> <th>0.00041</th> <th>0.00094</th> <th>0.013</th> <th>0.00097</th> <th>0.0019</th> <th>0.00148</th>	e in effect per 1 µg/m <sup>3</sup> PM) (as per Table 6-23) 0.00	3 0.000	08	0.00041	0.00094	0.013	0.00097	0.0019	0.00148		
Total Population in study area:   12562	Canterbury LGA										
% population in assessment age-group:   58%   14%   14%   100%   58%   100%   11     construction   Viola (hange)   -7.16	Total Population in study area:	2562	12562	12562	12562	12562	12562	12562	12562		
total change   -71.60   -71.6   -00569973   -000569973   -000569973   -000569973   -000569973   -000569973   -000569973   -000569973   -000569973   -000042   -000413   0.000   Risk   -0099996   -0099995   -0099995   -0099926   -0099926   -0099996   -00027   -000022   -000079   -00002   -00002   -00002   -00002   -000027   -0.00022   -0.000079   -0.00026   -0.00027   -0.00027   -0.00027   -0.00022   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029   -0.00029	% population in assessment age-group:	58%	14%	14%	100%	58%	100%	100%	19%		
Population weighted Xx (ug/m):   -0.00569973   -0.0056973   -0.0056973   -0.0056973   -0.0056973   -0.0056973   -0.00573   -0.00113   0.0007   Relative Risk   0.00113   0.0007   Relative Risk   0.00114   0.00027   0.00027   0.00027   -0.00027   <	total change	71.60	-71.6	-71.6	-71.6	-71.6	-71.6	-71.6	-71.6		
Baseline Incidence (per 100.000) (as per Table 4-5)   1026   9.25   3.978   401.6   412.0   113.3     Baseline Incidence (per person)   0.01026   0.09325   0.03978   0.04042   0.00142   0.01013   0.00     Relative Risk   0.999967   0.999995   0.999995   0.999996   0.999996   0.999996   0.999996   0.999996   0.999996   0.909996   0.90027   0.00022   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00028   0.00128   0.00228   0.00228   0.00228   0.004228   0.004228   0.004228   0.004228   0.004228   0.004228	Population weighted Δx (µg/m <sup>3</sup> ): -0.00	-0.00	569973	-0.00569973	-0.00569973	-0.00569973	-0.00569973	-0.00569973	-0.00569973		
Baseline Incidence (per person)   0.01026   0.03925   0.03978   0.04022   0.0412   0.00113   0.00     Relative Risk:   0.999967   0.999995   0.999995   0.999926<	seline Incidence (per 100,000) (as per Table 4-5)	1026	9235	3978	401.6	412.0	113.3	49.4	1209.0		
Relative Risk:   0.999967   0.999998   0.999998   0.999996   0.999926   0.999994   0.999994   0.99994   0.999946   0.90027   0.00027   0.00027   0.00027   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.0007   0.0007   0.0007   0.0017   0.0017   0.0017   0.0017   0.0017   0.0017   0.0017   0.0017   0.0017   0.00172   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819   0.00422819 <td>Baseline Incidence (per person)</td> <td>01026</td> <td>0.09235</td> <td>0.03978</td> <td>0.00402</td> <td>0.00412</td> <td>0.00113</td> <td>0.00049</td> <td>0.01209</td>	Baseline Incidence (per person)	01026	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209		
Attributable fraction (AF): -3.3E-05 -4.6E-06 -2.3E-06 -7.4E-06 -7.4E-06 -5.5E-06 -1.11   Increased number of cases in population: 0.0002 -0.00016 -0.00027 -0.00022 -0.00079 -0.0002   Individual subrubs within LGA -4.2E-07 -9.3E-08 -2.2E-08 -3.1E-07 -6.3E-09 -5.3E   Canterbury (South) - Campsie - - - - - - -   Modulation in study area 149 149 149 149 149 149 149 149 -	Relative Risk: 0.	99967 0	.999995	0.999998	0.999995	0.999926	0.999994	0.999989	0.999992		
Increased number of cases in population:   -0.0025   -0.00074   -0.00016   -0.00027   -0.0022   -0.000079   -0.000     Risk   -3.4E-07   -4.2E-07   -9.3E-08   -2.2E-08   -3.1E-07   -6.3E-09   -5.3I     Canterbury (South) - Campsie	Attributable fraction (AF):	3E-05 -	4.6E-06	-2.3E-06	-5.4E-06	-7.4E-05	-5.5E-06	-1.1E-05	-8.4E-06		
Risk:   -3.4E-07   -4.2E-07   -9.3E-08   -2.2E-08   -3.1E-07   -6.3E-09   -5.3E     Individual subrubs within LGA   Image: Cameroburg (South) - Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsie   Image: Campsi	Increased number of cases in population:	.0025 -	0.00074	-0.00016	-0.00027	-0.0022	-0.000079	-0.000067	-0.00025		
Individual subrubs within LGA   Image: Canterbury (South) - Campsie   Image: Canterbury (South) - Canterbu	Risk: -	4E-07 -	4.2E-07	-9.3E-08	-2.2E-08	-3.1E-07	-6.3E-09	-5.3E-09	-1.0E-07		
Canterbury (South) - Campsie   Image: Mark South and Sout	Individual subrubs within LGA										
Total Population in study area:   149   149   149   149   149   149     % population in assessment age-group:   58%   14%   14%   100%   58%   100%   11     % population in assessment age-group:   58%   14%   14%   100%   58%   100%   11     % population weighted Δx (µg/m <sup>3</sup> ):   0.00422819   0.00412   0.00113   0.000   0.0000   0.00001   0.0001   0.0001 <td>Canterbury (South) - Campsie</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td>	Canterbury (South) - Campsie										
$\begin{tabular}{ c c c c c c c c c c c c c c c c c c c$	Total Population in study area:	149	149	149	149	149	149	149	149		
total change   0.63   0.042   0.00422819   0.00422819   0.00422819   0.00422819   0.00422   0.00113   0.001   0.0001   0.0001   0.0001   0.0000   0.0001   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000 </td <td>% population in assessment age-group:</td> <td>58%</td> <td>14%</td> <td>14%</td> <td>100%</td> <td>58%</td> <td>100%</td> <td>100%</td> <td>19%</td>	% population in assessment age-group:	58%	14%	14%	100%	58%	100%	100%	19%		
Population weighted Ax (µg/m³):   0.00422819   0.00422   0.00413   0.00113   0.0013   0.00011   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.0000   0.00000   0.0000   0.0000   <	total change	0.63	0.63	0.63	0.63	0.63	0.63	0.63	0.63		
Baseline Incidence (per 100,000) (as per Table 4.4)   1026   9235   3378   401.6   412.0   113.3     Baseline Incidence (per person)   0.01026   0.09235   0.03978   0.00402   0.00412   0.00113   0.00     Relative Risk   1.000025   1.000003   1.000004   1.000055   1.000004   1.00004     Attributable fraction (AF):   2.5E-05   3.4E-06   1.7E-06   4.0E-06   5.5E-05   4.1E-06   8.00     Increased number of cases in population:   0.0000   0.00	Population weighted $\Delta x (\mu g/m^3)$ : 0.00	22819 0.00	422819	0.00422819	0.00422819	0.00422819	0.00422819	0.00422819	0.00422819		
Baseline Incidence (per person)   0.01026   0.09235   0.03978   0.00402   0.00412   0.00113   0.0001     Relative Risk:   1.000025   1.000002   1.000004   1.000055   1.000004   1.000055   1.000004   1.000055   1.000004   1.000005   1.000005   1.000004   1.000005   1.000004   1.000005   1.000004   1.000005   1.000004   1.000005   1.000004   1.000005   1.000004   0.0000   0.0000   0.0	seline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	401.6	412.0	113.3	49.4	1209.0		
Relative Risk:   1.000025   1.000003   1.000004   1.000055   1.000004   1.000055     Attributable fraction (AF):   2.5E-05   3.4E-06   1.7E-06   4.0E-06   5.5E-05   4.1E-06   8.00     Increased number of cases in population:   0.0000   0.0001   0.0005   0.0058   0.058	Baseline Incidence (per person)	01026	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209		
Attributable fraction (AF):   2.5E-05   3.4E-06   1.7E-06   4.0E-06   5.5E-05   4.1E-06   8.00     Increased number of cases in population:   0.00008   0.00581648	Relative Risk: 1.	00025 1	.000003	1.000002	1.000004	1.000055	1.000004	1.000008	1.000006		
Increased number of cases in population:   0.00000   0.00000   0.00000   0.00000   0.00000   0.00000   0.00000   0.00000   0.00000   0.00000   0.000000   0.000000   0.000000   0.000000   0.000000 <th0< td=""><td>Attributable fraction (AF):</td><td>5E-05</td><td>3.4E-06</td><td>1.7E-06</td><td>4.0E-06</td><td>5.5E-05</td><td>4.1E-06</td><td>8.0E-06</td><td>6.3E-06</td></th0<>	Attributable fraction (AF):	5E-05	3.4E-06	1.7E-06	4.0E-06	5.5E-05	4.1E-06	8.0E-06	6.3E-06		
Risk:   2.5E-07   3.1E-07   6.9E-08   1.6E-08   2.3E-07   4.6E-09   4.0E     Kingsgrove (North) - Earlwood   Image: Comparison of the study area   12413   1203   1203   1203	Increased number of cases in population:	.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000		
Kingsgrove (North) - Earlwood   Image: Constraint of the Constra	Risk:	5E-07	3.1E-07	6.9E-08	1.6E-08	2.3E-07	4.6E-09	4.0E-09	7.6E-08		
Total Population in study area:   12413	Kingsgrove (North) - Earlwood										
% population in assessment age-group:   58%   14%   14%   100%   58%   100%   110%     total change   -72.2   <	Total Population in study area:	2413	12413	12413	12413	12413	12413	12413	12413		
total change   -72.2	% population in assessment age-group:	58%	14%	14%	100%	58%	100%	100%	19%		
Population weighted Xx (µg/m <sup>2</sup> ):   -0.00581648	total change	-12.2	-12.2	-12.2	-12.2	-12.2	-12.2	-12.2	-12.2		
Baseline Incidence (per 100,000) (as per lable 4.4)   102b   9235   3978   401.6   412.0   113.3     Baseline Incidence (per person)   0.01026   0.09235   0.03978   0.00402   0.00412   0.00113   0.00     Relative Risk   0.999966   0.999995   0.999995   0.999995   0.999994   0.999944   0.99944   0.999444   0.99944   0.99944 <t< td=""><td>Population weighted Δx (µg/m<sup>3</sup>): -0.00</td><td>31648 -0.00</td><td>581648</td><td>-0.00581648</td><td>-0.00581648</td><td>-0.00581648</td><td>-0.00581648</td><td>-0.00581648</td><td>-0.00581648</td></t<>	Population weighted Δx (µg/m <sup>3</sup> ): -0.00	31648 -0.00	581648	-0.00581648	-0.00581648	-0.00581648	-0.00581648	-0.00581648	-0.00581648		
Baseline Incidence (per person)   0.01026   0.09235   0.03978   0.00402   0.00412   0.00113   0.00     Relative Risk:   0.999966   0.999998   0.999996   0.9999924   0.999999   0.9999924   0.999999   0.999994   0.999994   0.999994   0.999994   0.999996   0.999994   0.999996   0.9999924   0.9999996   0.9999924   0.999994   0.999996   0.999994   0.999996   0.999966   0.999966   0.999966   0.90007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007   0.00007	seline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	401.6	412.0	113.3	49.4	1209.0		
Relative Risk:   0.999995   0.999995   0.999995   0.999992   0.999994   0.99994   0.999994   0.999994   0.999994   0.999994   0.999994   0.9000	Baseline Incidence (per person) (	01026	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209		
Attroutable traction (AF):   -3.4E-US   -4.7E-US   -2.4E-US   -7.6E-US   -7.6E-US   -5.6E-US   -1.1T     Increased number of cases in population:   -0.0025   -0.0007   -0.0002   -0.0003   -0.0023   -0.000079   -0.000     Risk:   -3.5E-07   -4.3E-07   -9.5E-08   -2.2E-08   -3.1E-07   -6.4E-09   -5.5E	Relative Risk: 0.	19966 0	.9999995	0.999998	0.999995	0.999924	0.999994	0.999989	0.999991		
Increased number of cases in population:   -0.0023   -0.0007   -0.0002   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.00023   -0.0007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.00023   -0.00007   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003   -0.0003 </td <td>Attributable fraction (AF):</td> <td>4E-05 -</td> <td>4.7E-06</td> <td>-2.4E-06</td> <td>-5.5E-06</td> <td>-7.6E-05</td> <td>-5.6E-06</td> <td>-1.1E-05</td> <td>-8.6E-06</td>	Attributable fraction (AF):	4E-05 -	4.7E-06	-2.4E-06	-5.5E-06	-7.6E-05	-5.6E-06	-1.1E-05	-8.6E-06		
KISK: -3.3E-07 -4.3E-07 -9.5E-08 -2.2E-08 -3.1E-07 -6.4E-09 -5.5E	increased number of cases in population:	.0025	-0.0007	-0.0002	-0.0003	-0.0023	-0.000079	-0.000068	-0.00025		
	Risk: -	DE-U/ -	4.3E-07	-9.5E-08	-2.2E-08	-3.1E-07	-6.4E-09	-5.5E-09	-1.0E-07		
	I	I			l	l	I	l	1		

	Primary Indicators			Secondary Indicators				
Health Endpoint	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Botany LGA								
Total Population in study area	31386	31386	31386	31386	31386	31386	31386	31386
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-1369.7	-1369.7	-1369.7	-1369.7	-1369.7	-1369.7	-1369.7	-1369.7
Population weighted Ax (ug/m <sup>3</sup> )	-0.04364048	-0.04364048	-0.04364048	-0.04364048	-0.04364048	-0.04364048	-0.04364048	-0.04364048
Baseline Incidence (per 100 000) (as per Table 4-5	1026	9235	3978	501.7	412.0	133.6	49.4	1209 0
Baseline Incidence (per reson	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0 00049	0.01200
Buseline meddine (per person	0.999747	0.999965	0.00070	0.00002	0.00412	0.999958	0.00043	0.01200
Attributable fraction (AE)	-2 5E-04	-3.5E-05	-1 8E-05	-4 1E-05	-5 7E-04	-4 2E-05	-8.3E-05	-6.5E-05
Increased number of cases in population	-0.049	-0.013	-0.0029	-0.0065	-0.044	-0.0018	-0.0013	-0.0038
Risk	-2.6E-06	-3.2E-06	-7.1E-07	-2.1E-07	-2.3E-06	-5.7E-08	-4.1E-08	-7.8E-07
Individual subrubs within LG	2.02.00	0.22 00		2.12 01	2.02.00	0.12.00		1.02 01
Botany	,							
Total Population in study area	· 10408	10408	10408	10408	10408	10408	10408	10408
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-364 7	-364 7	-364 7	-364 7	-364 7	-364 7	-364 7	-364 7
Population weighted Ax (ug/m <sup>3</sup> )	-0.03504035	-0.0350/035	-0.03504035	-0.0350/035	-0.03504035	-0.03504035	-0.03504035	-0.03504035
Baseline Incidence (per 100 000) (as per Table 4.4	1026	-0.00004000	-0.03304033	-0.00004000	-0.03304033	-0.03304033	-0.03304033	-0.03304033
Baseline incidence (per 100,000) (as per 1able 4.4	0.01026	0.00235	0.03079	0.00502	912.0	0.00134	0.00040	0.01203.0
Baseline Incidence (per person	0.01020	0.09233	0.03976	0.00502	0.00412	0.00134	0.00049	0.01208
Attributable fraction (AF)	-2 0E-04	-2.8E-05	-1 4E-05	-3 3E-05	-4.6E-04	-3 4E-05	-6 7E-05	-5 2E-05
Increased number of cases in population	-0.0130	-2.0L-03	-1.4L-03	-0.0017	-4.02-04	-3.42-03	-0.72-03	-0.0010
Piek	-0.0130	-0.0000	-0.0000	-1.7E-07	-0.0117	-0.0003 -4.5E-08	-0.0003	-6.3E-07
Mascot - Fastlakes	-2.12-00	-2.0L-00	-3.7 2-07	-1.7 -07	-1.32-00	-4.52-00	-3.32-00	-0.52-07
Total Population in study area	20286	20286	20286	20286	20286	20286	20286	20286
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-996.5	-996.5	-996.5	-996.5	-996.5	-996.5	-996.5	-996.5
Benulation weighted Av (ug/m <sup>3</sup> )	0.04012255	0.04012255	0.04012255	0.04012255	0.04012255	0.04012255	0.04012255	0.04012255
Baseline Incidence (per 100 000) (as per Table 4.4	1026	-0.04912233	-0.04912233	-0.04912233	-0.04912233	-0.04912233	-0.04912233	1200 0
Daseline incidence (per 100,000) (as per 1able 4.4	0.01020	0.00005	0.00070	0.00500	412.0	0.00424	43.4	0.04000
Baseline Incidence (per person	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0.00049	0.01205
Attributable fraction (AF)	-2 8E-04	-3 9E-05	-2 0E-05	-4 6E-05	-6.4E-04	-4 8E-05	-9.3E-05	-7 3E-05
Increased number of cases in population	-2.02-04	-0.0096	-2.02-03	-4.02-03	-0.42-04	-4.02-03	-0.0009	-0.0028
Risk	-2.9E-06	-0.0030	-0.0021	-2.3E-07	-0.0313 -2.6E-06	-6.4E-08	-4.6E-08	-0.0020
Pagewood - Hillsdale - Dacevville	2.02 00	0.02 00	0.02 07	2.02 01	2.02 00	0.42 00	4.02 00	0.02 07
Total Population in study area	. 631	631	631	631	631	631	631	631
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-9.4	-9.4	-9.4	-9.4	-9.4	-9.4	-9.4	-94
Bopulation weighted Ax (ug/m <sup>3</sup> )	0.01490600	0.01490600	0.01490600	0.01490600	0.01490600	0.01490600	0.01490600	0.01490600
Baseline Incidence (per 100 000) (as per Table 4.4	1026	-0.01409099	-0.01409099	-0.01409099	-0.01409099	-0.01409099	-0.01409099	-0.01409093
Baseline Incidence (per 100,000) (as per 1able 4.4	0.01020	9233	0.03079	0.00502	412.0	0.00124	45.4	0.01209.0
Daseine incidence (per person	0.01026	0.09233	0.03978	0.00502	0.00412	0.00134	0.00049	0.01208
Attributable fraction (AE)	0.999914	0.999900	0.999994	0.999966	1 0E 04	0.999900	0.999972	0.999976
Increased number of cases in population	-0.02-03	-1.2L-03	-0.12-00	-1.4L-03	-1.92-04	-1.4L-03	-2.0L-03	-2.2L-00
Pick	-0.0003	1 1E 06	2 45 07	7.0E.09	-0.0003 8 0E 07	1 0E 09	1 4E 09	2.75.07
Sydney Airnor	-0.92-07	-1.12-00	-2.4L-07	-7.0L-00	-0.02-07	-1.92-00	-1.4L-00	-2.7 L-07
Total Population in study area	. 61	61	61	61	61	61	61	61
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	0.00	0.9	0.9	0.9	00/0	0.9	0.9	0.0
Population weighted Av (vg/m <sup>3</sup> )	0.01475410	0.01475440	0.01475440	0.01475410	0.01475440	0.01475410	0.01475410	0.01475410
Population weighted ΔX (µg/m) Baseline Incidence (por 100.000) (os por Table 4.4	0.01470410	0.014/0410	0.014/0410	501 7	0.01470410	0.01470410	0.014/0410	1200 0
	0.04000	9233	3976	0.00500	412.0	133.0	49.4	1209.0
Baseline incidence (per person	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0.00049	0.01209
Attributable freetien (AE)	0 80 000	1.000012		1.000014	1.000192	1 / 1 / 1 / 1	2 9 5 0 5	2 2 5 0
Autourable fraction (AF)	0.00-00	0.000006	0.000010	0.000042	0.000020	0.000012	2.0E-00	2.20
Dick	8 8E.07	1 1E 06	0.000019 2 /E 07	7 DE. 09	7 0E 07	1 0E 09	1 /E 09	2 6E 07
NISK	0.02-07	1.12-00	2.72-07	7.02=00	7.3L=07	1.32-00	1.42-00	2.02-07
1		1	I	•	1	I	1	1

		Primary Indicator	'e		Sa	condary Indicators	•	
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
B (change in effect per 1 µg/m PM) (as per 1 able 6-23) Kogarah - Rockdale LGA	0.0038	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Total Population in study area	113547	113547	113547	113547	113547	113547	113547	113547
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	142.6	142.6	142.6	142.6	142.6	142.6	6 142.6	142.6
Baseline Incidence (per 100.000) (as per Table 4-5)	0.00125587	0.00125587	0.00125587	0.00125587	412.0	0.00125587	0.00125587	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	1.000007	1.000001	1.000001	1.000001	1.000016	1.000001	1.000002	1.000002
Attributable fraction (AF):	7.3E-06	1.0E-06	5.1E-07	1.2E-06	1.6E-05	1.2E-06	6 2.4E-06	1.9E-06
Risk:	7.5E-08	9.3E-08	2.0E-08	4.7E-09	6.7E-08	1.3E-09	1.2E-09	2.2E-08
Individual subrubs within LGA								
Arncliffe - Bardwell Park	04.457	04457	04.457	04.457	04457	04.457	04.457	04457
% population in assessment age-group:	62%	21457	21457	21457	62%	21457	21457	21457
total change	-583.8	-583.8	-583.8	-583.8	-583.8	-583.8	-583.8	-583.8
Population weighted Δx (µg/m <sup>3</sup> ):	-0.02720790	-0.02720790	-0.02720790	-0.02720790	-0.02720790	-0.02720790	-0.02720790	-0.02720790
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	6 49.4	1209.0
Baseline Incidence (per person) Relative Risk:	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Attributable fraction (AF):	-1.6E-04	-2.2E-05	-1.1E-05	-2.6E-05	-3.5E-04	-2.6E-05	5 -5.2E-05	-4.0E-05
Increased number of cases in population:	-0.021	-0.0066	-0.0015	-0.0022	-0.019	-0.00063	-0.00055	-0.0015
Risk:	-1.6E-06	-2.0E-06	-4.4E-07	-1.0E-07	-1.5E-06	-2.9E-08	-2.6E-08	-4.9E-07
Total Population in study area:	20002	20002	20002	20002	20002	20002	20002	20002
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	117	117	117	117	117	117	117	117
Population weighted Δx ( $\mu$ g/m <sup>3</sup> ): Baseline Incidence (per 100 000) (as per Toble 4.4)	0.00584942	0.00584942	0.00584942	0.00584942	0.00584942	0.00584942	0.00584942	0.00584942
Baseline Incidence (per 100,000) (as per 1able 4.4)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	1.000034	1.000005	1.000002	1.000005	1.000076	1.000006	1.000011	1.000009
Attributable fraction (AF):	3.4E-05	4.7E-06	2.4E-06	5.5E-06	7.6E-05	5.7E-06	5 1.1E-05	8.7E-06
Increased number of cases in population: Risk:	0.0043 3.5E-07	4.3E-07	0.00029 9.5E-08	0.00044 2.2E-08	0.0039 3 1E-07	0.000125 6.3E-09	0.000110 5.5E-09	0.00031 1.0E-07
Kingsgrove (South) - Bardwell Park	0.02 01		0.02 00	2.22 00	0112 01	0.02 00	0.02.00	1102 01
Total Population in study areas	2879	2879	2879	2879	2879	2879	2879	2879
% population in assessment age-group: total change	62%	-17.7	-17.7	-17.7	62%	-17.7	100%	-17.7
Population weighted Ax (µg/m <sup>3</sup> ):	-0.00614797	-0.00614797	-0.00614797	-0.00614797	-0.00614797	-0.00614797	-0.00614797	-0.00614797
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	6 49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	0.999964	0.999995	0.999997	0.999994	0.999920	0.999994	0.999988	0.999991
Increased number of cases in population:	-0.00065	-4.9E-08	-0.000044	-0.000066	-0.00058	-0.000019	-0.000017	-0.000046
Risk:	-3.7E-07	-4.5E-07	-1.0E-07	-2.3E-08	-3.3E-07	-6.6E-09	-5.8E-09	-1.1E-07
Kogarah	11000	14000	44000	44000	44000	44000	44000	44000
% population in assessment age-group:	62%	11323	11323	11323	62%	11323	11323	11323
total change	861.8	861.8	861.8	861.8	861.8	861.8	8 861.8	861.8
Population weighted $\Delta x (\mu g/m^3)$ :	0.07611057	0.07611057	0.07611057	0.07611057	0.07611057	0.07611057	0.07611057	0.07611057
Baseline Incidence (per 100,000) (as per 1 able 4.4)	1026	9235	3978	396.0	412.0	110.5	49.4	1209.0
Relative Risk:	1.000442	1.000061	1.000031	1.000072	1.000990	1.000074	1.000145	1.000113
Attributable fraction (AF):	4.4E-04	6.1E-05	3.1E-05	7.2E-05	9.9E-04	7.4E-05	5 1.4E-04	1.1E-04
Increased number of cases in population:	0.032	0.0097	0.0022	0.0032 2.8E-07	0.028	0.00092 8.2E-08	2 0.00081	0.00225
Kogarah Bay - Carlton - Allawah	4.3L-00	3.0E-00	1.22-00	2.02-07	4.12-00	0.22-00	7.12-00	1.42-00
Total Population in study areas	10923	10923	10923	10923	10923	10923	10923	10923
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
iotal change Population weighted Av (un/m <sup>3</sup> ):	203.3	283.3	283.3	283.3	283.3	283.3	0 02503610	283.3 0.02503610
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	5 49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	1.000150	1.000021	1.000011	1.000024	1.000337	1.000025	1.000049	1.000038
Increased number of cases in population:	0.010	0.0032	0.00071	2.4E-05 0.0011	3.4E-04 0.0093	0.00030	0.00027	0.00074
Risk:	1.5E-06	1.9E-06	4.2E-07	9.7E-08	1.4E-06	2.8E-08	3 2.4E-08	4.6E-07
Monterey - Brighton-le-Sands - Kyeemagh	10015	10015	10015	40045	10015	40045	10015	10015
I otal Population in study area: % population in assessment age-group	13915	13915	13915	13915	13915	13915	13915	13915
total change	-243.1	-243.1	-243.1	-243.1	-243.1	-243.1	-243.1	-243.1
Population weighted Δx (µg/m <sup>3</sup> ):	-0.01747036	-0.01747036	-0.01747036	-0.01747036	-0.01747036	-0.01747036	-0.01747036	-0.01747036
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	6 49.4	1209.0
Baseline Incidence (per person) Relative Risk:	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Attributable fraction (AF):	<u>-1.0E-</u> 04	<u>-1.4E</u> -05	-7.2E-06	<u>-1.6E</u> -05	-2.3E-04	-1.7E-05	<u>-3.3E</u> -05	-2.6E-05
Increased number of cases in population:	-0.0089	-0.0027	-0.00061	-0.00090	-0.0080	-0.00026	-0.00023	-0.00064
Risk: Rockdale - Banksia	-1.0E-06	-1.3E-06	-2.8E-07	-6.5E-08	-9.4E-07	-1.9E-08	-1.6E-08	-3.1E-07
Total Population in study area:	19957	19957	19957	19957	19957	19957	19957	19957
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	-458.6	-458.6	-458.6	-458.6	-458.6	-458.6	-458.6	-458.6
Baseline Incidence (per 100.000) (as per Table 4.4)	-0.02297941	-0.02297941 9235	-0.02297941 3978	-0.02297941 396.0	-0.02297941 412.0	-0.02297941 110.5	-0.02297941	-0.02297941 1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	0.999867	0.999982	0.999991	0.999978	0.999701	0.999978	0.999956	0.999966
Attributable fraction (AF):	-1.3E-04	-1.8E-05	-9.4E-06	-2.2E-05	-3.0E-04	-2.2E-05	-4.4E-05	-3.4E-05
Risk:	-1.4E-06	- <u>1.7E-</u> 06	-3.7E-07	-8.6E-08	-1.2E-06	-2.5E-08	-2.2E-08	-4.1E-07
Sans Souci - Ramsgate								
Total Population in study areas	13091	13091	13091	13091	13091	13091	13091	13091
total change	183.6	183.6	183.6	183.6	183.6	183.6	5 183.6	183.6

		Primary Indicator	s	Secondary Indicators					
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term	
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years	
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148	
Population weighted Δx (µg/m <sup>3</sup> ):	0.01402490	0.01402490	0.01402490	0.01402490	0.01402490	0.01402490	0.01402490	0.01402490	
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	49.4	1209.0	
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209	
Relative Risk:	1.000081	1.000011	1.000006	1.000013	1.000182	1.000014	1.000027	1.000021	
Attributable fraction (AF):	8.1E-05	1.1E-05	5.8E-06	1.3E-05	1.8E-04	1.4E-05	2.7E-05	2.1E-05	
Increased number of cases in population:	0.0067	0.0021	0.00046	0.00068	0.0060	0.00020	0.00017	0.00048	
Risk:	8.3E-07	1.0E-06	2.3E-07	5.2E-08	7.5E-07	1.5E-08	1.3E-08	2.5E-07	
	l	l	l	l	l	l	l		

	Primary Indicators Secondary Indicators					;		
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 µg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Hurstville LGA								
Total Population in study area:	657	657	657	657	657	657	657	657
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	11.6	11.6	11.6	11.6	11.6	11.6	11.6	11.6
Population weighted $\Delta x (\mu g/m^3)$ :	0.01765601	0.01765601	0.01765601	0.01765601	0.01765601	0.01765601	0.01765601	0.01765601
Baseline Incidence (per 100,000) (as per Table 4-5)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0.00049	0.01209
Relative Risk:	1.000102	1.000014	1.000007	1.000017	1.000230	1.000017	1.000034	1.000026
Attributable fraction (AF):	1.0E-04	1.4E-05	7.2E-06	1.7E-05	2.3E-04	1.7E-05	3.4E-05	2.6E-05
Increased number of cases in population:	0.00042	0.00013	0.000029	0.000050	0.00038	0.000014	0.000011	0.000030
Risk:	1.1E-06	1.3E-06	2.9E-07	7.7E-08	9.5E-07	2.2E-08	1.7E-08	3.2E-07
Individual subrubs within LGA								
Hurstville								
Total Population in study areas	96	96	96	96	96	96	96	96
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	1.71	1.71	1.71	1.71	1.71	1.71	1.71	1.71
Population weighted Δx (ug/m <sup>3</sup> ):	0.01781250	0.01781250	0.01781250	0.01781250	0.01781250	0.01781250	0.01781250	0.01781250
Baseline Incidence (per 100.000) (as per Table 4.4)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0.00049	0.01209
Relative Risk:	1.000103	1.000014	1.000007	1.000017	1.000232	1.000017	1.000034	1.000026
Attributable fraction (AF):	1.0E-04	1.4E-05	7.3E-06	1.7E-05	2.3E-04	1.7E-05	3.4E-05	2.6E-05
Increased number of cases in population:	0.000063	0.000019	0.0000043	0.0000074	0.0000563	0.0000021	0.0000016	0.0000045
Risk:	1.1E-06	1.3E-06	2.9E-07	7.7E-08	9.5E-07	2.2E-08	1.7E-08	3.2E-07
South Hurstville - Blakehurst								
Total Population in study areas	561	561	561	561	561	561	561	561
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	9.94	9.94	9.94	9.94	9.94	9.94	9.94	9.94
Population weighted $\Delta x (\mu g/m^3)$ :	0.01771836	0.01771836	0.01771836	0.01771836	0.01771836	0.01771836	0.01771836	0.01771836
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0
Baseline Incidence (per person)	0 01026	0 09235	0.03978	0 00462	0 00412	0 00127	0 00049	0.01209
Relative Risk:	1.000103	1.000014	1.000007	1.000017	1.000230	1.000017	1.000034	1.000026
Attributable fraction (AF):	1.0E-04	1.4E-05	7.3E-06	1.7E-05	2.3E-04	1.7E-05	3.4E-05	2.6E-05
Increased number of cases in population:	0.00036	0.00011	0.000025	0.000043	0.00033	0.000012	0.000009	0.000026
Risk:	1.1E-06	1.3E-06	2.9E-07	7.7E-08	9.5E-07	2.2E-08	1.7E-08	3.2E-07
Total population incidence - All Suburbs	-0.074	-0.018	-0.0041	-0.010	-0.067	-0.0027	-0.0019	-0.0053

## Assessment of Increased Incidence - PM<sub>2.5</sub> F6 Extension: 2036 Cumulative

	Primary Indicators Secondary Indicators					5		
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	> 30 years	> 65 years	> 65 years	All ages	> 30 years	All ages	All ages	1-14 years
Q (abanna in affact non 4 un/m <sup>3</sup> DM) (as non Table C 22)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00149
p (change in effect per 1 µg/m PM) (as per 1 able 6-23) Strathfield - Burwood - Ashfield I GA	0.0000	0.0000	0.00041	0.00034	0.013	0.00037	0.0013	0.00146
Total Population in study area	20160	20160	20160	20160	20160	20160	20160	20160
% population in assessment age-group	6/%	12%	12%	100%	20100	20100	100%	20100
total change	-24 3	-24 3	-24 3	-24.3	-24.3	-24.3	-24.3	-24 3
Population weighted Ax (ug/m <sup>3</sup> ):	-0.00120536	-0.00120536	-0.00120536	-0.00120536	-0.00120536	-0.00120536	-0.00120536	-0.00120536
Baseline Incidence (per 100 000) (as per Table 4-5)	1026	9235	-0.00120330	-0.00120330	412 0	98.7	49.4	12030
Baseline Incidence (per reciper  0.01026	0.09235	0.03978	0.00335	0.00412	0 00099	0 00049	0.01209	
Relative Risk:	0.999993	0.999999	1.000000	0.999999	0.999984	0.999999	0.999998	0.999998
Attributable fraction (AF)	-7.0E-06	-9.6E-07	-4.9E-07	-1.1E-06	-1.6E-05	-1.2E-06	-2.3E-06	-1.8E-06
Increased number of cases in population:	-0.00092	-0.00022	-0.000048	-0.000077	-0.00083	-0.000023	-0.000023	-0.000061
Risk	-7.2E-08	-8.9E-08	-2.0E-08	-3.8E-09	-6.5E-08	-1.2E-09	-1.1E-09	-2.2E-08
Individual subrubs within LGA								
Ashfield								
Total Population in study area	: 1512	1512	1512	1512	1512	1512	1512	1512
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-0.49	-0.49	-0.49	-0.49	-0.49	-0.49	-0.49	-0.49
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00032407	-0.00032407	-0.00032407	-0.00032407	-0.00032407	-0.00032407	-0.00032407	-0.00032407
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk:	0.999998	1.000000	1.000000	1.000000	0.999996	1.000000	0.999999	1.000000
Attributable fraction (AF)	: -1.9E-06	-2.6E-07	-1.3E-07	-3.0E-07	-4.2E-06	-3.1E-07	-6.2E-07	-4.8E-07
Increased number of cases in population:	-0.000019	-0.0000044	-0.0000010	-0.0000015	-0.000017	-0.00000047	-0.0000046	-0.0000012
RISK:	-1.9E-08	-2.4E-08	-5.3E-09	-1.0E-09	-1.7E-08	-3.1E-10	-3.0E-10	-5.8E-09
Canterbury (North) - Ashbury	. 7170	7170	7170	7170	7170	7170	7170	7170
V population in assessment and group	6494	129/	129/	100%	64%	100%	100%	1/1/9
// population in assessment age-group.	-17.7	-17.7	-17.7	-17.7	-17.7	-17.7	-17.7	-17.7
Bopulation weighted Ax (ug/m <sup>3</sup> )	0.00246552	0.00246552	0.00246552	0.00246552	0.00246552	0.00246552	0.00246552	0.00246552
Baseline Incidence (per 100 000) (as per Table 4.4)	10240332	-0.00240332	-0.00240332	-0.00240332	-0.00240332	-0.00240332	-0.00240332	1209.0
Baseline Incidence (per 100,000) (as per 1able 4.4)	0.01026	0.09235	0.03978	0.00335	0.00/12	0.0009	0.00049	0.01209
Relative Risk	0.999986	0.03233	0.00070	0.999998	0.999968	0.999998	0.999995	0.999996
Attributable fraction (AF)	-1.4E-05	-2.0E-06	-1.0E-06	-2.3E-06	-3.2E-05	-2.4E-06	-4.7E-06	-3.6E-06
Increased number of cases in population:	-0.00067	-0.00016	-0.000035	-0.000056	-0.00060	-0.000017	-0.000017	-0.000045
Risk	-1.5E-07	-1.8E-07	-4.0E-08	-7.8E-09	-1.3E-07	-2.4E-09	-2.3E-09	-4.4E-08
Dulwich Hill - Lewisham								
Total Population in study area	: 11400	11400	11400	11400	11400	11400	11400	11400
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-6.52	-6.52	-6.52	-6.52	-6.52	-6.52	-6.52	-6.52
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00057193	-0.00057193	-0.00057193	-0.00057193	-0.00057193	-0.00057193	-0.00057193	-0.00057193
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	335.0	412.0	98.7	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00335	0.00412	0.00099	0.00049	0.01209
Relative Risk:	0.999997	1.000000	1.000000	0.999999	0.999993	0.999999	0.999999	0.999999
Attributable fraction (AF)	: -3.3E-06	-4.6E-07	-2.3E-07	-5.4E-07	-7.4E-06	-5.5E-07	-1.1E-06	-8.5E-07
Increased number of cases in population:	-0.00025	-0.00006	-0.000013	-0.000021	-0.00022	-0.000006	-0.000006	-0.000016
Risk: Haborfield - Summer Hill	-3.4E-08	-4.2E-08	-9.3E-09	-1.8E-09	-3.1E-08	-5.5E-10	-5.4E-10	-1.0E-08
Total Population in study area	. 60	60	60	60	60	60	60	60
% population in assessment age-group	64%	12%	12%	100%	6/0/	100%	100%	14%
total change	0.38	0.38	0.38	0.38	0.38	0.38	0.38	0.38
Population weighted Av (ug/m <sup>3</sup> ):	0.00550725	0.00550725	0.00550725	0.00550725	0.00550725	0.00550725	0.00550725	0.00550725
Baseline Incidence (per 100.000) (as per Table 4.4)	1026	9235	3978	335.0	412 0	98.7	49.4	1209 0
Baseline Incidence (per person)	0.01026	0 09235	0 03978	0 00335	0 00412	0 0009	0 00049	0.01209
Relative Risk:	1.000032	1.000004	1.000002	1.000005	1.000072	1.000005	1.000010	1.000008
Attributable fraction (AF):	: 3.2E-05	4.4E-06	2.3E-06	5.2E-06	7.2E-05	5.3E-06	1.0E-05	8.2E-06
Increased number of cases in population:	0.0000144	0.0000034	0.0000008	0.0000012	0.0000130	0.0000004	0.0000004	0.0000010
Risk	3.3E-07	4.1E-07	9.0E-08	1.7E-08	2.9E-07	5.3E-09	5.2E-09	9.9E-08

	Primary Indicators Secondary Indicators					5		
Health Endpoint	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Sydney Inner City LGA								
Total Population in study area	: 29695	29695	29695	29695	29695	29695	29695	29695
% population in assessment age-group	58%	8%	8%	100%	58%	100%	100%	6%
total change	-202.1	-202.1	-202.1	-202.1	-202.1	-202.1	-202.1	-202.1
Population weighted $\Delta x (ug/m^3)$	-0.00680586	-0.00680586	-0.00680586	-0.00680586	-0.00680586	-0.00680586	-0.00680586	-0.00680586
Baseline Incidence (per 100.000) (as per Table 4-5	1026	9235	3978	534.2	412.0	146.4	49.4	1209.0
Baseline Incidence (per person	0.01026	0.09235	0.03978	0.00534	0.00412	0.00146	0.00049	0.01209
Relative Risk	0.999961	0.999995	0.999997	0.999994	0.999912	0.999993	0.999987	0.999990
Attributable fraction (AF)	-3.9E-05	-5.4E-06	-2.8E-06	-6.4E-06	-8.8E-05	-6.6E-06	-1.3E-05	-1.0E-05
Increased number of cases in population	-0.0069	-0.0012	-0.00027	-0.0010	-0.0062	-0.00029	-0.00019	-0.00021
Risk	-4.1E-07	-5.0E-07	-1.1E-07	-3.4E-08	-3.6E-07	-9.7E-09	-6.4E-09	-1.2E-07
Individual subrubs within LGA								
Erskinville - Alexandria								
Total Population in study area	· 11411	11411	11411	11411	11411	11411	11411	11411
% population in assessment age-group	58%	8%	8%	100%	58%	100%	100%	6%
total change	-46.3	-46.3	-46.3	-46.3	-46.3	-46.3	-46.3	-46.3
Population weighted Ax (ug/m <sup>3</sup> )	-0.00405749	-0 00405749	-0 00405749	-0.00405749	-0 00405749	-0 00405749	-0 00405749	-0.00405749
Baseline Incidence (per 100 000) (as per Table 4 4	1026	9235	3978	534 2	412.0	146.4	49.4	1209 (
Baseline Incidence (per recipiero) (de per rabie na	0.01026	0.09235	0 03078	0.00534	0.00/12	0.00146	0.00049	0.01200
Balative Risk	0.01020	0.03233	0.00070	0.00004	0.00412	0.00140	0.00043	0.01203
Attributable fraction (AF)	-2 4E-05	-3 2E-06	-1 7E-06	-3.8E-06	-5 3E-05	-3 9E-06	-7 7E-06	-6 0E-06
Increased number of cases in population	-2.42-03	-0.00028	-0.000062	-0.00023	-0.0014	-0.00066	-0.000043	-0.00-40
Rick	-0.0010	-0.00020	-0.000002	-0.00023	-0.0014	-0.000000	-0.000043	-0.000048
Newtown - Camperdown - Darlington	-2.46-07	-3.02-07	-0.02-00	-2.02-00	-2.2L-01	-5.02-03	-3.02-03	-1.52-00
Total Population in study area	5225	5225	5225	5225	5225	5225	5225	5225
% population in assessment age-group	58%	8%	8%	100%	58%	100%	100%	6%
total change	35.1	35.1	35.1	35.1	35.1	35.1	35.1	35 1
Deputation weighted Av (ug/m <sup>3</sup> )	0.00671770	0.00671770	0.00671770	0.00671770	0.00671770	0.00671770	0.00671770	0.00671770
Baseline Incidence (per 100 000) (as per Table 4.4	1026	0.00071770	0.00071770	534.2	0.00071770	0.00071770	0.00071770	1209 (
Baseline Insidence (per 100,000) (de per 14bie 4.4	0.01026	0.00235	0.02078	0.00534	0.00412	0.00146	0.00040	0.01200.0
Baseline Incidence (per person	1.000020	1 000005	1 000003	1 000006	1 00097	1 000007	1 000049	1 000010
Attributable fraction (AF)	3 9E-05	5.4E-06	2.8E-06	6 3E-06	8.7E-05	6.5E-06	1.000013	9 9E-06
Increased number of cases in population	0.0012	0.00021	0.000047	0.0018	0.0108	0.00050	0.000033	0.000037
Risk	4 0E-07	5.0E-07	1 1E-07	3.4E-08	3.6E-07	9.5E-09	6 3E-09	1 2E-07
Waterloo - Beaconsfield	4.02-07	3.0L-01	1.12-07	J.4L-00	3.0L-07	3.5L-03	0.52-03	1.22-01
Total Population in study area	. 13059	13059	13059	13059	13059	13059	13059	13059
% population in assessment age-group	58%	8%	8%	100%	58%	100%	100%	6%
total change	-190.9	-190.9	-190.9	-190.9	-190.9	-190.9	-190.9	-190 9
Population weighted Ax (ug/m <sup>3</sup> )	0.01461927	0.01/61927	0.01461927	0.01461927	0.01/61927	0.01461927	0.01461927	0.01/61927
Population weighted ΔX (µg/m) Baseline Incidence (per 100.000) (as par Table 4.4	10.01401027	-0.01401027	-0.01401027	52/ 2	-0.01401027	1/6 /	-0.01401027	1200 (
Baseline incluence (per 100,000) (as per Table 4.4	0.01020	9235	0.02070	0.00524	412.0	140.4	49.4	1209.0
Baseline incidence (per person	0.01026	0.09235	0.03978	0.00034	0.00412	0.00146	0.00049	0.01205
Attributable fraction (AE)	0.999915	0.999988	0.999994	0.999986	0.999810	0.999986	0.999972	0.999978
Autouable fraction (AF)	-0.5E-05	-1.2E-00	-0.0E-06	-1.4E-00	-1.9E-04	-1.4E-00	-2.0E-00	-2.2E-0
Disk	-0.0005	-0.0012	-0.00020	-0.00090 -7 3E-08	-0.0059	-0.00027	-0.00018 -1.4E-08	-0.00020
1130	0.72-07	1.12-00	2.76-07	1.02-00	7.5E-07	2.12-00	1.42-00	2.02-07

		Primary Indicator	S	Secondary Indicators				
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Marrickville - Svdenham - Petersham LGA								
Total Population in study area	35722	35722	35722	35722	35722	35722	35722	35722
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-66.5	-66.5	-66.5	-66.5	-66.5	-66.5	-66.5	-66.5
Population weighted Ax (ug/m <sup>3</sup> ):	-0.00186160	-0.00186160	-0.00186160	-0.00186160	-0.00186160	-0.00186160	-0.00186160	-0.00186160
Baseline Incidence (per 100 000) (as per Table 4-5)	1026	9235	3978	508.0	412.0	138.9	49.4	1209.0
Baseline Incidence (per response)	0.01026	0.09235	0 03978	0.00508	0.00/12	0.00130	0 00049	0.01200
Relative Risk	0.01020	0.03233	0.00070	0.00000	0.00412	0.00133	0.00043	0.01203
Attributable fraction (AF)	-1 1E-05	-1 5E-06	-7 6E-07	-1 7E-06	-2.4E-05	-1 8E-06	-3 5E-06	-2 8E-06
Increased number of cases in population:	-0.0025	-0.00060	-0.00013	-0.00032	-2.42-03	-0.00090	-0.000062	-0.00017
Risk	-1 1E-07	-1 4E-07	-3 0E-08	-8 9E-09	-1.0E-07	-2 5E-09	-1 7E-09	-3 3E-08
Individual subrubs within LGA	-1.12-07	-1.42-07	-5.02-00	-0.32-03	-1.02-07	-2.5L-03	-1.7 -03	-3.32-00
Marrickville								
Total Population in study area	258/2	258/2	258/2	258/2	258/2	258/2	258/2	258/2
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	33.4	33.4	33.4	33.4	33.4	33.4	33.4	33.4
Deputation weighted Av (ug/m <sup>3</sup> )	0.00120247	0.00120247	0.00120247	0.00120247	0.00120247	0.00120247	0.00120247	0.00120247
Population weighted Δx (µg/m).	0.00129247	0.00129247	0.00129247	0.00129247	0.00129247	0.00129247	0.00129247	1200.0
Baseline incidence (per 100,000) (as per 1able 4.4)	0.01020	9233	3970	0.00500	412.0	130.9	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00508	0.00412	0.00139	0.00049	0.01209
Attribute la fraction (AE)	1.000007	1.000001	1.000001	1.000001	1.000017	1.000001	1.000002	1.000002
Attributable fraction (AF).	7.5E-06	1.0E-06	5.3E-07	1.2E-06	1.7E-05	1.3E-06	2.5E-06	1.9E-06
Increased number of cases in population:	0.0013	0.00030	0.000066	0.00016	0.0011	0.000045	0.000031	0.000084
Risk:	7.7E-08	9.5E-08	2.1E-08	6.2E-09	6.9E-08	1.7E-09	1.2E-09	2.3E-08
Tetel Deputation in study and	2054	0054	0054	0054	2054	0054	2054	0054
	2051	2051	2051	2051	2051	2051	2051	2051
% population in assessment age-group:	0.14	12%	12%	100%	04%	100%	100%	14%
	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14
Population weighted $\Delta x$ (µg/m)	0.00006826	0.00006826	0.00006826	0.00006826	0.00006826	0.00006826	0.00006826	0.00006826
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	508.0	412.0	138.9	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00508	0.00412	0.00139	0.00049	0.01209
Relative Risk:	1.000000	1.000000	1.000000	1.000000	1.000001	1.000000	1.000000	1.000000
Attributable fraction (AF):	4.0E-07	5.5E-08	2.8E-08	6.4E-08	8.9E-07	6.6E-08	1.3E-07	1.0E-07
Increased number of cases in population:	0.0000053	0.000013	0.000003	0.000007	0.000048	0.0000019	0.0000013	0.00000035
Risk:	4.1E-09	5.0E-09	1.1E-09	3.3E-10	3.7E-09	9.2E-11	6.4E-11	1.2E-09
Sydenham - Tempe - St Peters	7000	7000	7000	7000	7000	7000	7000	7000
	. 7829	/829	/829	1829	7829	7829	1829	7829
% population in assessment age-group:	64%	12%	12%	100%	64%	100%	100%	14%
total change	-100	-100	-100	-100	-100	-100	-100	-100
Population weighted $\Delta x (\mu g/m^2)$ :	-0.01277302	-0.01277302	-0.01277302	-0.01277302	-0.01277302	-0.01277302	-0.01277302	-0.01277302
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	508.0	412.0	138.9	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00508	0.00412	0.00139	0.00049	0.01209
Relative Risk:	0.999926	0.999990	0.999995	0.999988	0.999834	0.999988	0.999976	0.999981
Attributable fraction (AF)	-7.4E-05	-1.0E-05	-5.2E-06	-1.2E-05	-1.7E-04	-1.2E-05	-2.4E-05	-1.9E-05
Increased number of cases in population:	-0.0038	-0.00090	-0.00020	-0.00048	-0.0034	-0.00013	-0.000094	-0.00025
Risk	-7.6E-07	-9.4E-07	-2.1E-07	-6.1E-08	-6.8E-07	-1.7E-08	-1.2E-08	-2.3E-07
	1	1	1		1	1	1	1

		Primary Indicators Secondary Indicators						
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 µg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Canterbury LGA								
Total Population in study area	12562	12562	12562	12562	12562	12562	12562	12562
% population in assessment age-group:	58%	14%	14%	100%	58%	100%	100%	19%
total change	-17.20	-17.2	-17.2	-17.2	-17.2	-17.2	-17.2	-17.2
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00136921	-0.00136921	-0.00136921	-0.00136921	-0.00136921	-0.00136921	-0.00136921	-0.00136921
Baseline Incidence (per 100,000) (as per Table 4-5)	1026	9235	3978	401.6	412.0	113.3	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209
Relative Risk:	0.999992	0.999999	0.999999	0.999999	0.999982	0.999999	0.999997	0.999998
Attributable fraction (AF)	-7.9E-06	-1.1E-06	-5.6E-07	-1.3E-06	-1.8E-05	-1.3E-06	-2.6E-06	-2.0E-06
Increased number of cases in population:	-0.00060	-0.00018	-0.000039	-0.000065	-0.00054	-0.000019	-0.000016	-0.000059
Risk	-8.1E-08	-1.0E-07	-2.2E-08	-5.2E-09	-7.3E-08	-1.5E-09	-1.3E-09	-2.4E-08
Individual subrubs within LGA	<u> </u>							
Canterbury (South) - Campsie								
Total Population in study area	149	149	149	149	149	149	149	149
% population in assessment age-group:	58%	14%	14%	100%	58%	100%	100%	19%
total change	1.35	1.35	1.35	1.35	1.35	1.35	1.35	1.35
Population weighted Δx (µg/m <sup>3</sup> ):	0.00906040	0.00906040	0.00906040	0.00906040	0.00906040	0.00906040	0.00906040	0.00906040
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	401.6	412.0	113.3	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209
Relative Risk:	1.000053	1.000007	1.000004	1.000009	1.000118	1.000009	1.000017	1.000013
Attributable fraction (AF)	5.3E-05	7.2E-06	3.7E-06	8.5E-06	1.2E-04	8.8E-06	1.7E-05	1.3E-05
Increased number of cases in population:	0.000047	0.000014	0.0000031	0.0000051	0.0000422	0.0000015	0.0000013	0.0000046
Risk	5.4E-07	6.7E-07	1.5E-07	3.4E-08	4.9E-07	1.0E-08	8.5E-09	1.6E-07
Kingsgrove (North) - Earlwood								
Total Population in study area	12413	12413	12413	12413	12413	12413	12413	12413
% population in assessment age-group:	58%	14%	14%	100%	58%	100%	100%	19%
total change	-18.6	-18.6	-18.6	-18.6	-18.6	-18.6	-18.6	-18.6
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00149843	-0.00149843	-0.00149843	-0.00149843	-0.00149843	-0.00149843	-0.00149843	-0.00149843
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	401.6	412.0	113.3	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00402	0.00412	0.00113	0.00049	0.01209
Relative Risk	0.999991	0.999999	0.999999	0.999999	0.999981	0.999999	0.999997	0.999998
Attributable fraction (AF)	-8.7E-06	-1.2E-06	-6.1E-07	-1.4E-06	-1.9E-05	-1.5E-06	-2.8E-06	-2.2E-06
Increased number of cases in population:	-0.00065	-0.00019	-0.000042	-0.000070	-0.00058	-0.000020	-0.000017	-0.00006
Risk	-8.9E-08	-1.1E-07	-2.4E-08	-5.7E-09	-8.0E-08	-1.6E-09	-1.4E-09	-2.7E-08

		Primary Indicator	S		Se	condary Indicators		
Health Endpoint	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
ß (change in effect per 1 ug/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148
Botany I GA								
Total Population in study area	. 31386	31386	31386	31386	31386	31386	31386	31386
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-1078.6	-1078.6	-1078.6	-1078.6	-1078.6	-1078.6	-1078.6	-1078 6
Population weighted Ax (ug/m <sup>3</sup> )	-0.03436564	-0.03/3656/	-0.03/3656/	-0.03436564	-0.03436564	-0.03436564	-0.03436564	-0.03/3656/
Baseline Incidence (ner 100 000) (as per Table 4-5	1026	9235	3978	501.7	412.0	133.6	49.4	1209 0
Baseline Insidence (per 100,000) (as per 1able 4 of	0.01026	0.00225	0.02079	0.00502	0.00412	0.00124	0.00040	0.01200
Baseline incidence (per person	0.01020	0.03233	0.00370	0.00002	0.00412	0.00134	0.00043	0.01203
Attributable fraction (AF)	-2 0E-04	-2 7E-05	-1 4E-05	-3 2E-05	-4 5E-04	-3 3E-05	-6 5E-05	-5 1E-05
Increased number of cases in population	-0.038	-0.010	-0.0023	-0.0051	-4.52-04	-0.0014	-0.02-03	-0.0030
Risk	-2 0E-06	-2 5E-06	-5.6E-07	-1 6E-07	-1.8E-06	-4 5E-08	-3.2E-08	-6 1E-07
Individual subrubs within I GA	2.02 00	2.02 00	0.02 07	1.02 07	1.02 00	4.02 00	0.22 00	0.12 01
Botany	,							
Total Population in study area	10408	10408	10408	10408	10408	10408	10408	10408
% population in assessment age-group	60%	13%	13%	10400	60%	100%	100%	16%
total change	-370.8	-370.8	-370.8	-370.8	-370.8	-370.8	-370.8	-370.8
Benulation weighted Av (ug/m <sup>3</sup> )	0.02562644	0.02562644	0.02562644	0.02562644	0.02562644	0.03563644	0.02562644	0.02562644
Population weighted ΔX (μg/m)	-0.03502044	-0.03362644	-0.03562644	-0.03362644	-0.03562644	-0.03362644	-0.03502044	-0.03362644
Baseline Incidence (per 100,000) (as per 1able 4.4	0.01020	9233	3970	0.00502	412.0	133.0	49.4	1209.0
Baseline Incidence (per person	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0.00049	0.01205
Relative Risk	0.999793	0.999971	0.999985	0.999967	0.999537	0.999965	0.999932	0.999947
Allibulable fraction (AF)	-2.1E-04	-2.9E-03	-1.5E-05	-3.3E-03	-4.0E-04	-3.3E-03	-0.0E-03	-5.3E-00
Dick	2 15 06	-0.0030 2.6E.06	-0.00079	1 75 07	1.0E.06	-0.00048	-0.00033	-0.0010 6.4E.07
Mascot - Eastlakos	-2.12-00	-2.0L-00	-3.6L-07	-1.72-07	-1.92-00	-4.0L-00	-3.3L-00	-0.42-07
Total Population in study area	20296	20296	20296	20296	20296	20286	20296	20296
% population in assessment and group	. 20200	20200	20200	20200	20200	20200	20200	20200
// population in assessment age-group	-690.2	-600.2	-690.2	-600.2	-690.2	-600.2	-690.2	-690
Desulation weighted Au (weight	-030.2	0.00400040	0.02400240	-030.2	0.02400240	0.00400040	-030.2	-030.2
Population weighted ΔX (µg/m) Receive Incidence (per 100,000) (as per Table 4.4)	-0.03402346	-0.03402346	-0.03402346	-0.03402346	-0.03402346	-0.03402346	-0.03402346	-0.03402340
Baseline incidence (per 100,000) (as per 1able 4.4	1020	9233	3970	0.00500	412.0	133.0	45.4	1209.0
Baseline Incidence (per person	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0.00049	0.01209
Attributable fraction (AE)	0.999603	0.999973	0.999900	0.999900	0.999556	0.999907	0.999933	0.999950
Allibulable fraction (AF)	-2.0E-04	-2.7 E-03	-1.4E-03	-3.2E-03	-4.4E-04	-3.3E-03	-0.5E-05	-5.0E-00
Dick	2 05 06	-0.0000 2.5E.06	-0.0013 5.5E.07	-0.0033 1 6E 07	1.9E.06	-0.00089	-0.00003	6.15.07
Pagewood - Hillsdale - Dacevville	-2.0L-00	-2.3L-00	-5.52-07	-1.0L-07	-1.0L-00	-4.42-00	-5.2L-00	-0.12-07
Total Population in study area	631	631	631	631	631	631	631	631
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	-18.1	-18.1	-18.1	-18.1	-18.1	-18.1	-18.1	-18 1
Benulation weighted Av (ug/m <sup>3</sup> )	0.02969462	0.02969462	0.02969462	0.02969462	0.02969462	0.02969462	0.02969463	0.02969.462
Population weighted ΔX (μg/m)	-0.02000403	-0.02000403	-0.02000403	-0.02000403	-0.02000403	-0.02000403	-0.02000403	-0.02000403
Daseline Incidence (per 100,000) (as per 1able 4.4	0.01020	9233	0.02070	0.00500	412.0	0.00424	45.4	1209.0
Baseline Incidence (per person	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0.00049	0.01205
Attributable fraction (AE)	1 75 04	0.999977	0.999900	0.999973	0.999027	0.999972	0.999940	0.999950
Allibulable fraction (AF)	-1.7E-04	-2.3E-03	-1.2E-03	-2.7E-03	-3.7 E-04	-2.6E-03	-5.5E-05	-4.2E-0
Dick	1 75 06	-0.00017	-0.000038 4 7E 07	-0.000083	-0.00036	-0.000023 2 7E 09	2 7E 09	-0.000031
Sydney Airpor	-1.72-00	-2.1L-00	-4.72-07	-1.4L-07	-1.3L-00	-3.7 L-00	-2.7 L-00	-5.12-07
Total Population in study area	. 61	61	61	61	61	61	61	61
% population in assessment age-group	60%	13%	13%	100%	60%	100%	100%	16%
total change	0.59	0.59	0.59	0.59	00%	0.59	0.50	0.50
Bopulation weighted Av /ug/m <sup>3</sup>	0.0067242	0.0067242	0.0067242	0.0067242	0.0067343	0.0067242	0.0067243	0.0067243
Population weighted ΔX (µg/m))	0.00907213	0.00907213	0.00907213	0.00907213	0.00907213	0.00907213	0.00907213	1200 (
Daseline incluence (per 100,000) (as per 1able 4.4	1026	9235	39/8	0.00500	412.0	133.6	49.4	1209.0
Baseline Incidence (per person	0.01026	0.09235	0.03978	0.00502	0.00412	0.00134	0.00049	0.01209
	1.000056	1.000008	1.000004	1.000009	1.000126	1.000009	1.000018	1.000014
Attributable fraction (AF)	. 5.0E-05	7.7E-06	4.0E-06	9.1E-06	1.3E-04	9.4E-06	1.8E-05	1.4E-05
increased number of cases in population	0.000021	0.0000057	0.000013	0.000028	0.000019	0.000008	0.000006	0.000017
Risk	5.8⊑-07	7.1E-07	1.0E-07	4.0E-08	5.2E-07	1.3⊑-08	9.1E-09	1.7E-07
1	I	I	I	1	I	I	I	I

		Primary Indicator	¢		Sa	condary Indicators	•	
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	s Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	, Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years
β (change in effect per 1 μg/m <sup>-</sup> PM) (as per Table 6-23) Kogarah - Rockdale LGA	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00146
Total Population in study area:	113547	113547	113547	113547	113547	113547	113547	113547
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	-2028.4	-2028.4	-2028.4	-2028.4	-2028.4	-2028.4	-2028.4	-2028.4
Baseline Incidence (per 100.000) (as per Table 4-5)	-0.01786397	-0.01786397 9235	-0.01786397	-0.01786397 396.0	-0.01786397 412.0	-0.01786397	-0.01786397	-0.01786397 1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	0.999896	0.999986	0.999993	0.999983	0.999768	0.999983	0.999966	0.999974
Attributable fraction (AF):	-1.0E-04	-1.4E-05	-7.3E-06	-1.7E-05	-2.3E-04	-1.7E-05	-3.4E-05	-2.6E-05
Risk:	-1.1E-06	-0.023	-2.9E-07	-6.6E-08	-9.6E-07	-1.9E-08	-1.7E-08	-3.2E-07
Individual subrubs within LGA								
Arncliffe - Bardwell Park	04.457	04457	04 457	04.457	04457	04.457	04.457	04457
% population in assessment age-group;	21457	21457	21457	21457	62%	21457	21457	21457
total change	-352.7	-352.7	-352.7	-352.7	-352.7	-352.7	-352.7	-352.7
Population weighted Δx (µg/m <sup>3</sup> ):	-0.01643753	-0.01643753	-0.01643753	-0.01643753	-0.01643753	-0.01643753	-0.01643753	-0.01643753
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	49.4	1209.0
Baseline incidence (per person) Relative Risk:	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Attributable fraction (AF):	-9.5E-05	-1.3E-05	-6.7E-06	-1.5E-05	-2.1E-04	-1.6E-05	-3.1E-05	-2.4E-05
Increased number of cases in population:	-0.013	-0.0040	-0.0009	-0.0013	-0.012	-0.00038	-0.00033	-0.0009
Risk:	-9.8E-07	-1.2E-06	-2.7E-07	-6.1E-08	-8.8E-07	-1.8E-08	-1.5E-08	-2.9E-07
Total Population in study area:	20002	20002	20002	20002	20002	20002	20002	20002
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	63.3	63.3	63.3	63.3	63.3	63.3	63.3	63.3
Population weighted Δx (µg/m <sup>2</sup> ): Baseline Incidence (per 100 000) (as per Table 4 4)	0.00316468	0.00316468	0.00316468	0.00316468	0.00316468	0.00316468	0.00316468	0.00316468
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	1.000018	1.000003	1.000001	1.000003	1.000041	1.000003	1.000006	1.000005
Attributable fraction (AF):	1.8E-05	2.5E-06	1.3E-06	3.0E-06	4.1E-05	3.1E-06	6.0E-06	4.7E-06
Risk:	1.9E-07	2.3E-07	5.2E-08	1.2E-08	1.7E-07	0.000068 3.4E-09	3.0E-09	5.7E-08
Kingsgrove (South) - Bardwell Park								
Total Population in study area:	2879	2879	2879	2879	2879	2879	2879	2879
% population in assessment age-group: total change	-13.2	-13.2	-13.2	-13.2	-13.2	-13.2	-13.2	-13.2
Population weighted Δx (µg/m <sup>3</sup> ):	-0.00458493	-0.00458493	-0.00458493	-0.00458493	-0.00458493	-0.00458493	-0.00458493	-0.00458493
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	i 49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk: Attributable fraction (AF):	0.999973 -2 7E-05	0.999996 -3.7E-06	0.999998	0.999996 -4 3E-06	0.999940 -6.0E-05	0.999996 -4.4E-06	0.999991	0.999993 -6.8E-06
Increased number of cases in population:	-0.00048	-0.00015	-0.000033	-0.000049	-0.00043	-0.000014	-0.000012	-0.000034
Risk:	-2.7E-07	-3.4E-07	-7.5E-08	-1.7E-08	-2.5E-07	-4.9E-09	-4.3E-09	-8.2E-08
Kogarah Total Population in study area	11222	11222	11222	11222	11222	11222	11222	11222
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	124.3	124.3	124.3	124.3	124.3	124.3	124.3	124.3
Population weighted $\Delta x (\mu g/m^3)$ :	0.01097766	0.01097766	0.01097766	0.01097766	0.01097766	0.01097766	0.01097766	0.01097766
Baseline incidence (per 100,000) (as per 1able 4.4) Baseline Incidence (per person)	0.01026	9235	0.03978	0.00396	412.0	0.00111	0 00049	0.01209.0
Relative Risk:	1.000064	1.000009	1.000005	1.000010	1.000143	1.000011	1.000021	1.000016
Attributable fraction (AF):	6.4E-05	8.8E-06	4.5E-06	1.0E-05	1.4E-04	1.1E-05	2.1E-05	1.6E-05
Increased number of cases in population: Risk:	0.005 6.5E-07	8.1E-07	0.0003 1.8E-07	0.0005 4.1E-08	0.004 5.9E-07	0.00013 1.2E-08	1.0E-08	2.0E-07
Kogarah Bay - Carlton - Allawah	0.02 01	0112 01	1102 01			TIZE 00	1102 00	2.02 01
Total Population in study area:	10923	10923	10923	10923	10923	10923	10923	10923
% population in assessment age-group:	62%	-82.6	-82.6	100%	62%	100%	100%	-82.6
Population weighted Ax (µg/m <sup>3</sup> ):	-0.00756203	-0.00756203	-0.00756203	-0.00756203	-0.00756203	-0.00756203	-0.00756203	-0.00756203
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	0.999956	0.999994	0.999997	0.999993	0.999902	0.999993	0.999986	0.999989
Increased number of cases in population:		-0.0009	-0.00021	-0.0003	-0.0027	-0.00009	-0.00008	-0.00022
Risk:	-4.5E-07	-5.6E-07	-1.2E-07	-2.8E-08	-4.1E-07	-8.1E-09	-7.1E-09	-1.4E-07
Monterey - Brighton-le-Sands - Kyeemagh Total Population in study group	12015	12015	12015	12015	12015	12015	12015	12015
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
total change	-361.1	-361.1	-361.1	-361.1	-361.1	-361.1	-361.1	-361.1
Population weighted Δx (µg/m <sup>3</sup> ):	-0.02595041	-0.02595041	-0.02595041	-0.02595041	-0.02595041	-0.02595041	-0.02595041	-0.02595041
Baseline Incidence (per 100,000) (as per 1able 4.4)	1026	9235	3978	396.0	412.0	0.00111	49.4	1209.0
Relative Risk:	0.999849	0.999979	0.999989	0.999976	0.999663	0.999975	0.999951	0.999962
Attributable fraction (AF):	-1.5E-04	-2.1E-05	-1.1E-05	-2.4E-05	-3.4E-04	-2.5E-05	-4.9E-05	-3.8E-05
Increased number of cases in population:	-0.0132	-0.0041	-0.00090	-0.00134	-0.0119	-0.00039	-0.00034	-0.00094
Risk: Rockdale - Banksia	-1.3E-06	-1.9E-06	-4.2E-07	-3.72-08	-1.4E-06	-2.0E-08	-2.4E-08	-4.0E-07
Total Population in study area:	19957	19957	19957	19957	19957	19957	19957	19957
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%
Example total change	-284.1	-284.1	-284.1	-284.1	-284.1	-284.1	-284.1	-284.1
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	49.4	1209.0
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209
Relative Risk:	0.999917	0.999989	0.999994	0.999987	0.999815	0.999986	0.999973	0.999979
Attributable fraction (AF): Increased number of cases in population:	-8.3E-05 -0.010	-1.1E-05	-0.0007	-1.3E-05 -0.0011	-1.9E-04	-1.4E-05	-2.7E-05	-2.1E-05 -0.0007
Risk:	-8.5E-07	-1.1E-06	-2.3E-07	-5.3E-08	-7.6E-07	-1.5E-08	-1.3E-08	-2.5E-07
Sans Souci - Ramsgate	10001	10001	10001	10001	10001	10001	10001	10001
votal Population in study area: % population in assessment age-group	13091	13091	13091	13091	13091	13091	13091	13091
total change	-1122.3	-1122.3	-1122.3	-1122.3	-1122.3	-1122.3	-1122.3	-1122.3

		Primary Indicator	s	Secondary Indicators					
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term	
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years	
β (change in effect per 1 μg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148	
Population weighted Δx (µg/m <sup>3</sup> ):	-0.08573065	-0.08573065	-0.08573065	-0.08573065	-0.08573065	-0.08573065	-0.08573065	-0.08573065	
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	396.0	412.0	110.5	49.4	1209.0	
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00396	0.00412	0.00111	0.00049	0.01209	
Relative Risk:	0.999503	0.999931	0.999965	0.999919	0.998886	0.999917	0.999837	0.999873	
Attributable fraction (AF):	-5.0E-04	-6.9E-05	-3.5E-05	-8.1E-05	-1.1E-03	-8.3E-05	-1.6E-04	-1.3E-04	
Increased number of cases in population:	-0.0411	-0.0127	-0.00280	-0.00418	-0.0370	-0.00120	-0.00105	-0.00293	
Risk:	-5.1E-06	-6.3E-06	-1.4E-06	-3.2E-07	-4.6E-06	-9.2E-08	-8.0E-08	-1.5E-06	
			l		I	I			

		Primary Indicator	S	Secondary Indicators						
Health Endpoint:	Mortality - All Causes, Long- term	Hospitalisations - Cardiovascular, Short-term	Hospitalisations - Respiratory, Short-term	Mortality - All Causes, Short- term	Mortality - Cardiopulmonary, Long-term	Mortality - Cardiovascular, Short-term	Mortality - Respiratory, Short-term	Morbidity - Asthma ED Admissions - Short-term		
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years		
β (change in effect per 1 µg/m <sup>3</sup> PM) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148		
Hurstville LGA										
Total Population in study areas	657	657	657	657	657	657	657	657		
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%		
total change	-2.0	-1.97	-1.97	-1.97	-1.97	-1.97	-1.97	-1.97		
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00299848	-0.00299848	-0.00299848	-0.00299848	-0.00299848	-0.00299848	-0.00299848	-0.00299848		
Baseline Incidence (per 100,000) (as per Table 4-5)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0		
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0.00049	0.01209		
Relative Risk:	0.999983	0.999998	0.999999	0.999997	0.999961	0.999997	0.999994	0.999996		
Attributable fraction (AF):	-1.7E-05	-2.4E-06	-1.2E-06	-2.8E-06	-3.9E-05	-2.9E-06	-5.7E-06	-4.4E-06		
Increased number of cases in population:	-0.000072	-0.000022	-0.0000049	-0.000086	-0.000065	-0.0000024	-0.0000018	-0.0000051		
Risk:	-1.8E-07	-2.2E-07	-4.9E-08	-1.3E-08	-1.6E-07	-3.7E-09	-2.8E-09	-5.4E-08		
Individual subrubs within LGA										
Hurstville										
Total Population in study areas	96	96	96	96	96	96	96	96		
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%		
total change	1.78	1.78	1.78	1.78	1.78	1.78	1.78	1.78		
Population weighted $\Delta x (\mu g/m^3)$ :	0.01854167	0.01854167	0.01854167	0.01854167	0.01854167	0.01854167	0.01854167	0.01854167		
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0		
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0.00049	0.01209		
Relative Risk:	1.000108	1.000015	1.000008	1.000017	1.000241	1.000018	1.000035	1.000027		
Attributable fraction (AF):	1.1E-04	1.5E-05	7.6E-06	1.7E-05	2.4E-04	1.8E-05	3.5E-05	2.7E-05		
Increased number of cases in population:	0.000065	0.000020	0.0000044	0.0000077	0.0000586	0.0000022	0.0000017	0.0000047		
Risk:	1.1E-06	1.4E-06	3.0E-07	8.1E-08	9.9E-07	2.3E-08	1.7E-08	3.3E-07		
South Hurstville - Blakehurst										
Total Population in study areas	561	561	561	561	561	561	561	561		
% population in assessment age-group:	62%	15%	15%	100%	62%	100%	100%	15%		
total change	-3.8	-3.8	-3.8	-3.8	-3.8	-3.8	-3.8	-3.8		
Population weighted $\Delta x (\mu g/m^3)$ :	-0.00677362	-0.00677362	-0.00677362	-0.00677362	-0.00677362	-0.00677362	-0.00677362	-0.00677362		
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	3978	462.3	412.0	127.4	49.4	1209.0		
Baseline Incidence (per person)	0.01026	0.09235	0.03978	0.00462	0.00412	0.00127	0.00049	0.01209		
Relative Risk:	0.999961	0.999995	0.999997	0.999994	0.999912	0.999993	0.999987	0.999990		
Attributable fraction (AF):	-3.9E-05	-5.4E-06	-2.8E-06	-6.4E-06	-8.8E-05	-6.6E-06	-1.3E-05	-1.0E-05		
Increased number of cases in population:	-0.00014	-0.00004	-0.000009	-0.000017	-0.00013	-0.000005	-0.000004	-0.000010		
Risk:	-4.0E-07	-5.0E-07	-1.1E-07	-2.9E-08	-3.6E-07	-8.4E-09	-6.4E-09	-1.2E-07		
Total population incidence - All Suburbs	-0.12	-0.036	-0.0078	-0.014	-0.11	-0.0040	-0.0032	-0.0088		

## Quantification of Effects - PM<sub>2.5</sub> F6 Extension: 2036 Cumulative - Elevated receptors

Air quality indicator:	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	PM <sub>2.5</sub>	DPM
Endpoint:	Mortality - All	Hospitalisations -	Hospitalisations -	Mortality - All	Mortality -	Mortality -	Mortality -	Morbidity -	Increased risk -
	Causes	Cardiovascular	Respiratory	Causes	Cardiopulmonary	Cardiovascular	Respiratory	Asthma ED	lung cancer
								Admissions	
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Long-term	Short-Term	Short-Term	Short-Term	Based on WHO
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	≥ 30 years	All ages	All ages	1-14 years	inhalation unit risk
β (change in effect per 1 μg/m³) (as per Table 6-23)	0.0058	0.0008	0.00041	0.00094	0.013	0.00097	0.0019	0.00148	3.40E-05
Annual Baseline Incidence (as per Table 4-5)									(ug/m3)-1
Annual baseline incidence (per 100,000)	1026	9235	3978	493	412	134.7	39.9	1209	
Baseline Incidence (per person per year)	0.01026	0.09235	0.03978	0.00493	0.00412	0.001347	0.000399	0.01209	
							•		

Sensitive Receptors	Change in Annual Average PM2.5 Concentration (µg/m <sup>3</sup> )	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk	Risk
Impacts from tunnel ventilation outlets										
Grid receptors: maximum at 10m height	1.40	8E-05	1E-04	2E-05	6E-06	7E-05	2E-06	1E-06	3E-05	5E-05
Grid receptors: maximum at 20m height	0.23	1E-05	2E-05	4E-06	1E-06	1E-05	3E-07	2E-07	4E-06	8E-06



