Appendix H

Technical working paper:
Human health risk assessment

NorthConnex

Building for the future

Australian Government

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Technical working paper: Human Health Risk Assessment

Prepared for: Roads and Maritime Services

Document History and Status

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Limitations

Environmental Risk Sciences has prepared this technical working paper for the use of Roads and Maritime Services in accordance with the usual care and thoroughness of the consulting profession. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this technical working paper.

It is prepared in accordance with the scope of work and for the purpose outlined in the Section 1 of this technical working paper.

The methodology adopted and sources of information used are outlined in this technical working paper. Environmental Risk Sciences has made no independent verification of this information beyond the agreed scope of works and assumes no responsibility for any inaccuracies or omissions. No indications were found that information contained in the reports for use in this assessment was false.

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Table of Contents

Tables (in Text)

Figures (in Text)

Appendices

Glossary of Terms

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.
ANZECC	Australia and New Zealand Environment and Conservation Council
AQIA	Technical Working Paper: Air Quality (AECOM, 2014) for the NorthConnex project.
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.
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.
Chronic exposure	Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure].
COPD	Chronic Obstructive Pulmonary Disease
DECCW	Department of Environment, Climate Change and Water
Detection limit	The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.
DGRs	Director General Requirements
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.
EC	European Commission
EP&A Act	Environmental Planning and Assessment Act 1979
EPA	Environment Protection Authority
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. 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 end point (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 receiver population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.
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 Organisation (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 will have different names, such as investigation level, trigger value, ambient guideline etc.
HIA	Health Impact Assessment
HHRA	Human Health Risk Assessment
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].
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.
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
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
OEH	Office of Environment and Heritage
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA)
PAH	Polycyclic aromatic hydrocarbon
PM	Particulate matter
$PM_{2.5}$	Particulate matter of aerodynamic diameter 2.5 µm and less
PM_{10}	Particulate matter of aerodynamic diameter 10 µm 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).

Receiver population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something will cause injury or harm.
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]
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, summarizes, 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.
Toxicology	The study of the harmful effects of substances on humans or animals.
TSP	Total suspended particulate
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 will cause harm to people [also sometimes called a safety factor].
USEPA	United States Environmental Protection Agency
VOC	Volatile Organic Compound
WHO	World Health Organisation

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Executive Summary

Roads and Maritime Services is seeking approval under Part 5.1 of the *Environmental Planning and Assessment Act 1979* to construct and operate a tolled motorway linking the M1 Pacific Motorway at Wahroonga to the Hills M2 Motorway at the Pennant Hills Road interchange at Carlingford in northern Sydney (the project), which would consist of twin tunnels approximately nine kilometres in length that would generally follow the alignment of the existing Pennant Hills Road. The purpose of the project is to reduce congestion on Pennant Hills Road, particularly for heavy vehicle traffic. This technical working paper was prepared to assess potential risks to human health associated with key aspects of the project, namely local air quality impacts, noise and vibration.

A human health risk assessment is a way of deciding now, what the consequences (to health) of some future action (such as this project) may be. We try to learn from previous experience about impacts from road tunnels and their potential effects on people who live or work around them. We then use this information to predict the impacts of the project on community health.

In this case the technical working paper includes a detailed review of what impacts to air quality, noise and vibration may occur, who may be exposed to these impacts and whether there is potential for these impacts to result in adverse health effects within the local community. It is conducted in accordance with national guidance available from enHealth (enHealth 2001, 2012a) and it 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) 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 receivers 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:
    - review of the key pollutants (associated with vehicle emissions) to air that are predicted from the operation of the project;
    - identify guidelines that are based on protection of the health of all members of the population for exposure to these pollutants all day, every day;
    - compare the predicted impacts with the health based guidelines;
    - for particulate matter, the guidelines available do not adequately address all the potential health effects that may occur and hence a more detailed assessment has been undertaken;
    - a more detailed assessment of exposure to particulate matter has utilised robust (published) associations between exposure to increased concentrations of particulates (as $PM_{2.5}$ or PM_{10}) and specific health effects (or health endpoints). The assessment conducted has evaluated the impact of the project on these health endpoints within the local community;
    - The potential for adverse health impacts in the community has been assessed on the basis of a range of considerations (including the size of the population exposed, calculated annual risk from exposure and the increase in the number of cases [for a

specific health endpoint] that may occur in the community as a result of exposure and benefits of the project)

- assessment of noise and vibration impacts on health including:
  - review of the impacts that are predicted from the operation of the project;
  - identify guidelines that are based on the protection of the health and wellbeing (including sleep disturbance) during all phases of the project (construction and operations);
  - compare the predicted impacts with the health based guidelines. Where the health based guidelines cannot be met, consideration of the implementation of mitigation/management measures and whether these can be effectively implemented to ensure the identified impacts meet the health based guidelines.

Based on the assessment undertaken and presented in this technical working paper the following has been concluded:

- In relation to impacts to air quality, potential health impacts have been evaluated using appropriate health based guidelines (that are protective of public health), or, in the case of exposure to $PM_{2.5}$ and PM_{10}, a detailed assessment of the impact of the emissions on key community health indicators. All predicted concentrations of carbon monoxide, nitrogen dioxide, key individual volatile organic compounds and polycyclic aromatic hydrocarbons are below health based guidelines. For the assessment of potential impacts of $PM_{2.5}$ and PM_{10} from the operation of the tunnel, potential health impacts are low and essentially negligible in proximity to the ventilation outlets. Overall, taking a significant number of vehicles, in particular trucks off the existing road corridor along Pennant Hills Road, and managing emissions via the tunnel ventilation system, would lead to a net benefit to health within the community.
- In relation to noise and vibration, potential impacts during construction and operation have been considered. During construction potential impacts from noise and vibration on the local community can be managed and/or mitigated through the implementation of a range of measures. For construction noise and vibration, these management and mitigation measures (including the requirement for noise monitoring) are to be outlined in detail within the Construction Noise and Vibration Management Plan.
- During operation of the project a number of individual homes located adjacent to the northern interchange as well as the southern interchange and the Hills M2 Motorway integration works have been identified where noise impacts are in excess of the health based guidelines.. The recommended mitigation measures would ensure that the levels of road traffic noise experienced by residents would be reduced as low as feasible and reasonable. The requirements and the form of operational noise mitigation will be confirmed when assessed against the detailed road and tunnel designs. This would include consideration of the feasibility of noise barriers given potential engineering constraints, and the outcomes of consultation with the affected community.

Section 1. Introduction

1.1 Project overview

Roads and Maritime Services (Roads and Maritime) is seeking approval under Part 5.1 of the *Environmental Planning and Assessment Act 1979* (EP&A Act) for the construction and operation of a multi-lane tolled motorway linking the M1 Pacific Motorway at Wahroonga to the Hills M2 Motorway at the Pennant Hills Road interchange at West Pennant Hills in northern Sydney (the project) (refer to **Figure 1-1**).

Key features of the project would include:

- Twin motorway tunnels up to around nine kilometres in length with two lanes in each direction. The tunnels would be constructed with provision for a possible third lane in each direction if required in the future.
- A northern interchange with the M1 Pacific Motorway and Pennant Hills Road, including sections of tunnel for on-ramps and off-ramps, which also facilitate access to and from the Pacific Highway.
- A southern interchange with the Hills M2 Motorway and Pennant Hills Road, including sections of tunnel for on-ramps and off-ramps.
- Integration works with the Hills M2 Motorway including alterations to the eastbound carriageway to accommodate traffic leaving the Hills M2 Motorway to connect to the project travelling northbound, and the provision of a new westbound lane on the Hills M2 Motorway extending through to the Windsor Road off-ramp.
- Tie-in works with the M1 Pacific Motorway extending to the north of Edgeworth David Avenue.
- A motorway operations complex located near the southern interchange on the corner of Eaton Road and Pennant Hills Road that includes operation and maintenance facilities.
- Two tunnel support facilities, which incorporates emergency smoke extraction outlet points and substations along the main alignment.
- Ancillary facilities for motorway operation, such as electronic tolling facilities, signage, ventilation systems and fire and life safety systems including emergency evacuation infrastructure.
- Modifications to service utilities and associated works at surface roads near the two interchanges and operational ancillary facilities.
- Modifications to local roads, including widening of Eaton Road near the southern interchange and repositioning of the Hewitt Avenue cul-de-sac near the northern interchange.
- Ancillary temporary construction facilities and temporary works to facilitate the construction of the project.

Subject to the project obtaining planning approval, construction of the project is anticipated to commence in early 2015 and is expected to take around four years to complete.

1.2 Project location

The project would be located within The Hills, Hornsby and Ku-ring-gai local government areas about 20 kilometres north-west of the central business district of Sydney. The regional context of the project is shown in **Figure 1-2**.

1.3 Purpose of this report

The Director-General's environmental assessment requirements (DGRs) for the project were issued on 29 October 2013 and re-issued with amendments on 11 April 2014. The DGRs have informed the preparation of the environmental impact statement for the project.

The DGRs require an assessment of potential impacts on air quality during construction and operation of the project, and to include a human health risk assessment (HHRA). Specifically, the DGR states that the assessment should include but not be limited to:

> *An assessment of construction and operation activities that have the potential to impact on local and regional air quality. The assessment should provide an assessment of the risk associated with potential discharges of fugitive and point source emissions, and include:…..*
>
> *consideration of the requirements of Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazards (enHealth, 2012),…..*

This technical working paper presents a Human Health Risk Assessment (HHRA) associated with key aspects of the project, namely local air quality impacts, noise and vibration (as proposed in the design as outlined in **Section 2**).

Other aspects of the DGR relating to air quality have been addressed in technical working paper: air quality (AECOM, 2014).

In providing input into the DGRs, the Ministry of Health (NSW Health) had provided a letter to the then Department of Planning and Infrastructure, dated 4 October 2013, outlining a range of aspects to be considered in the HHRA, including an assessment of:

- Impacts to air during construction and operation (impacts to the surrounding community and in-tunnel exposures);
- Impacts associated with noise and vibration during construction and operation.

These matters have also been considered within this technical working paper.

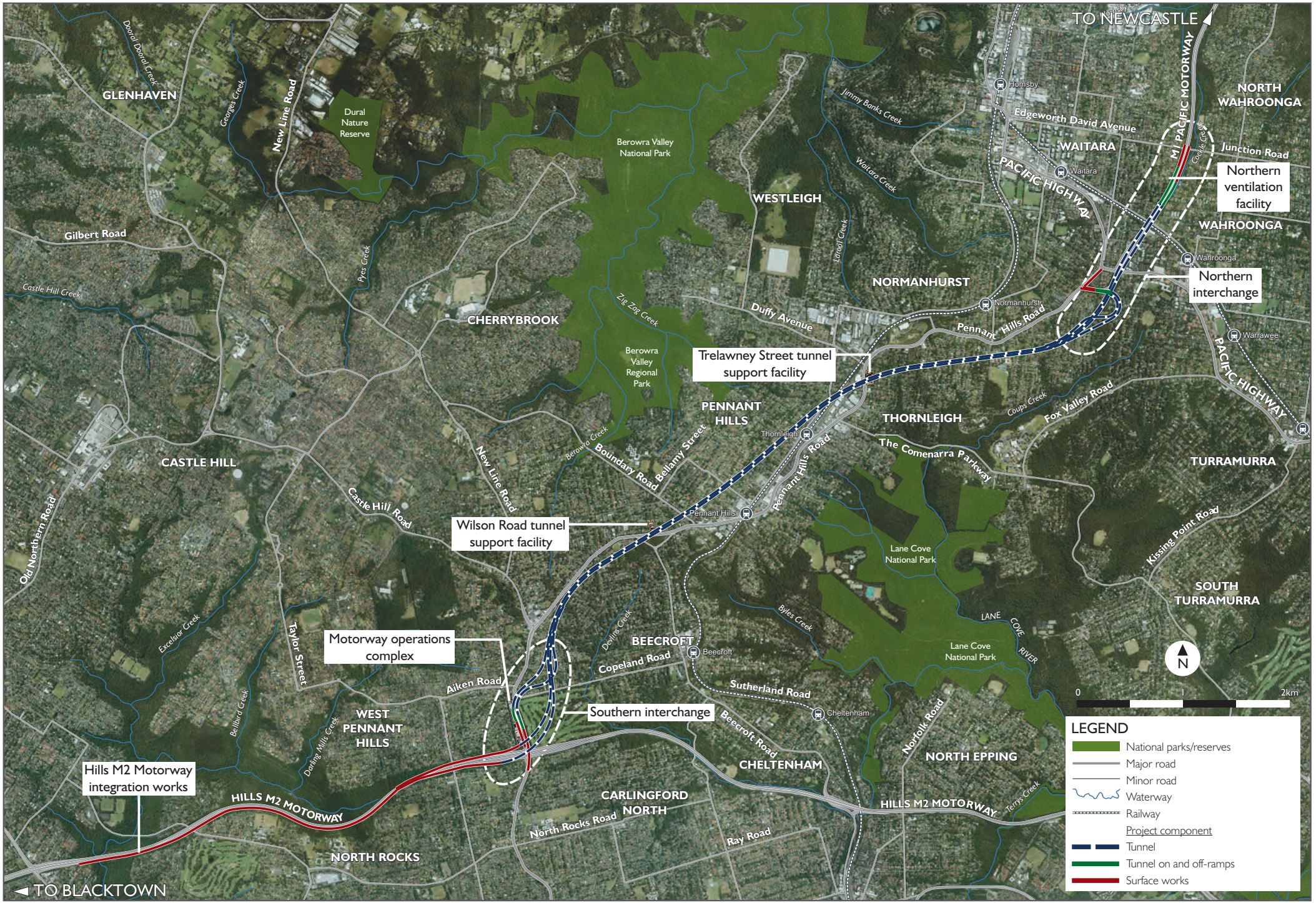

Figure 1-1 The project

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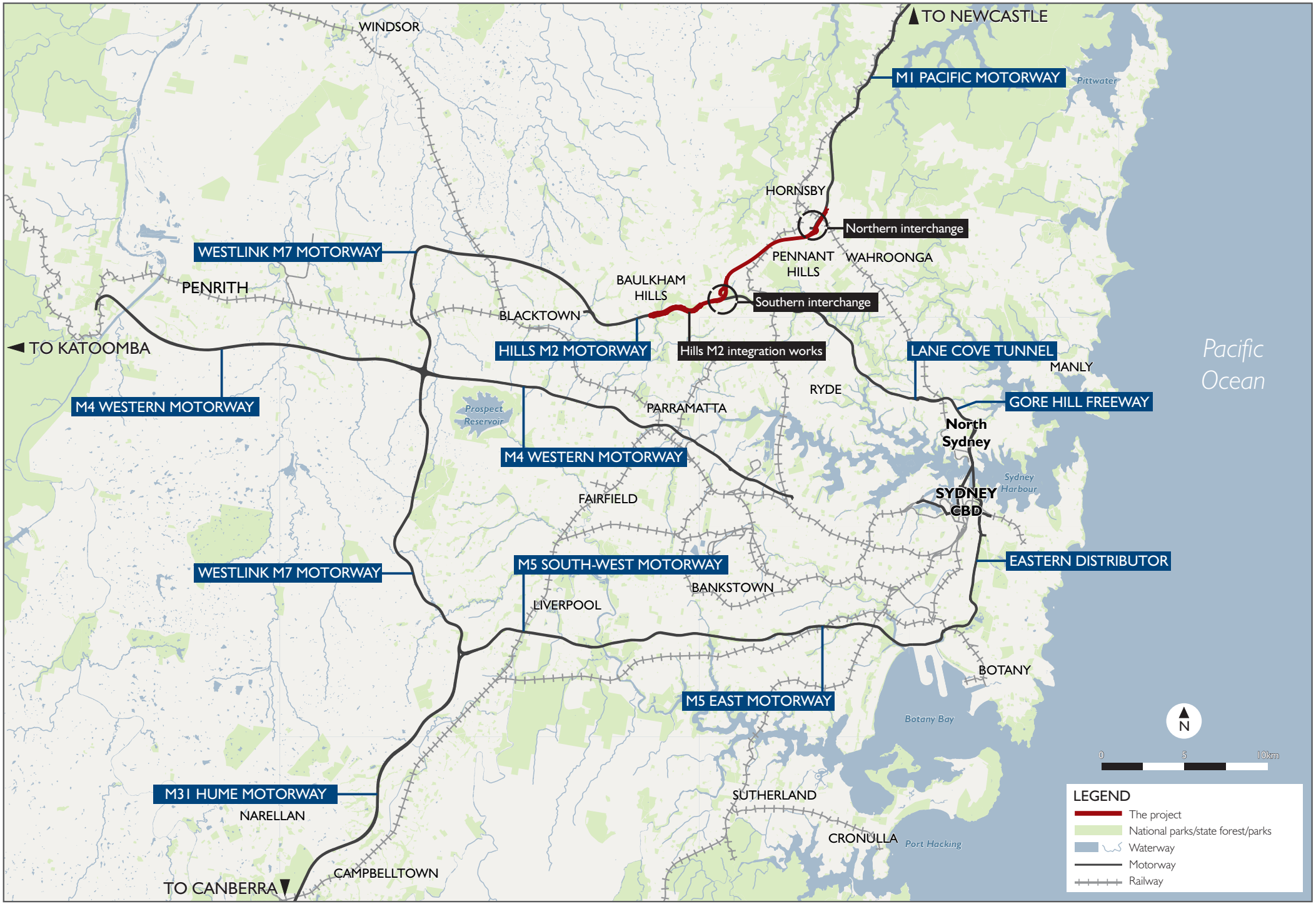

Figure 1-2 Regional context of the project

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1.4 Objectives

The overall objective of the HHRA presented in this technical working paper is to assess health risks associated with the following:

- Emissions to air and exposures in the local community (principally dust) during construction works (construction of the tunnel, interchanges, intersections and roadside infrastructure).
- Emissions to air (associated with vehicle emissions) and exposures of the local community to emissions from the ventilation facilities during the operation of the competed tunnels.
- Exposures that may occur in the tunnel (by users of the tunnel) during operation (normal operations and during breakdown situations).
- Noise and vibration, primarily during construction works.

The assessment presented has considered both short-term/acute and long-term/chronic risks to surrounding communities, based on outcomes presented in the technical working papers that have been completed as part of the environmental impact statement for air quality, noise and vibration.

1.5 Approach to Human Health Risk Assessment

1.5.1 What is a risk assessment?

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 in the environment and evaluation of projects with 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, tornados, etc, and exposures to environmental contaminants are examples of involuntary risks.

Defining risk

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 from a range of facilities (eg industrial or infrastructure) are usually assessed through qualitative and/or quantitative risk assessment techniques. In general, risk 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.

1.5.2 Overall approach

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

- EnHealth Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards: 2012 (enHealth 2012a);
- EnHealth Health Impact Assessment Guidelines: September 2001 (enHealth 2001);
- EnHealth Exposure Factors Guide, EnHealth Council, 2012 (enHealth 2012b);
- National Environment Protection Council (NEPC) Schedule B(8) Guideline on Community Consultation and Risk Communication, National Environment Protection (Assessment of Site Contamination) Measure, 1999 (NEPC 1999 amended 2013);
- NEPC National Environmental Protection (Air Toxics) Measure, Impact Statement for the National Environment Protection (Air Toxics) Measure, 2003 (NEPC 2003); and
- United States Environment Protection Agency (USEPA) 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 (USEPA 2009a).

More specifically in relation to the assessment of health impacts associated with exposure to particulates, guidelines available from the NEPC ((Burgers & Walsh 2002; NEPC 1998, 2002, 2003, 2009, 2010), World Health Organisation (Ostro 2004; WHO 2003, 2006b; 2006a, {Ostro, 2004 #861; 2013b) and the USEPA (USEPA 2005, 2009b) have been used as required.

The methodology used for the conduct of the HHRA presented in this reported has been presented to and discussed with NSW Health prior to the completion of this assessment.

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

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, 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 HHRA has considered a more detailed evaluation of exposures to particulate emissions within the surrounding community from the operation of the tunnel.

Exposure assessment

This involves the identification of populations located in the vicinity of the project who may be exposed to impacts from the project, in particular, the populations in areas adjacent to the southern and northern interchanges. The existing air and noise environments as well as the health of the existing population has been considered in relation to the key health endpoints, relevant to the assessment of exposures to particulate matter, that require further detailed consideration in this assessment. The assessment of potential particulate matter exposure has considered both short-term (acute) and chronic inhalation exposures relevant to the project.

Toxicity assessment

The objective of the toxicity assessment is to identify the adverse health effects and quantitative toxicity values or exposure-response relationships that are associated with the key pollutants 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 has been undertaken:

1. Identify the adverse health effects associated with exposure to particulate matter. Based on the available information, the most robust health end-points (effects or outcomes) for the assessment of inhalation exposure to particulate matter (assessed over different size fractions) have been identified. The most robust health end-points are where a relationship has been established between exposure to particulate matter and a specific health end-point (effect/outcome).

2. Identify the most relevant and robust exposure-response relationship for the quantitative assessment of exposure to particulate matter. The exposure-response relationships are derived from published peer reviewed sources and relate to the identified health end-points (effects/outcomes).

The health-endpoints and associated exposure-response relationships adopted for the assessment of particulate matter, particularly derived from combustion sources (such as petrol and diesel vehicles) have been agreed with NSW Health prior to the completion of this assessment.

For other air pollutants national guidelines based on the protection of health have been adopted.

Risk characterisation

Risks have been characterised using quantitative and qualitative assessment methods. The quantitative assessment of potential exposure to particulate emissions from the project combined with information on exposure (ie what additional concentrations of particulate matter would be present in the community as a result of the project) and the exposure-response relationships relevant for the health-endpoints (effect) has been used. This enables 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 unacceptable in the local population.

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

The overall approach is outlined in **Figure 1-3**.

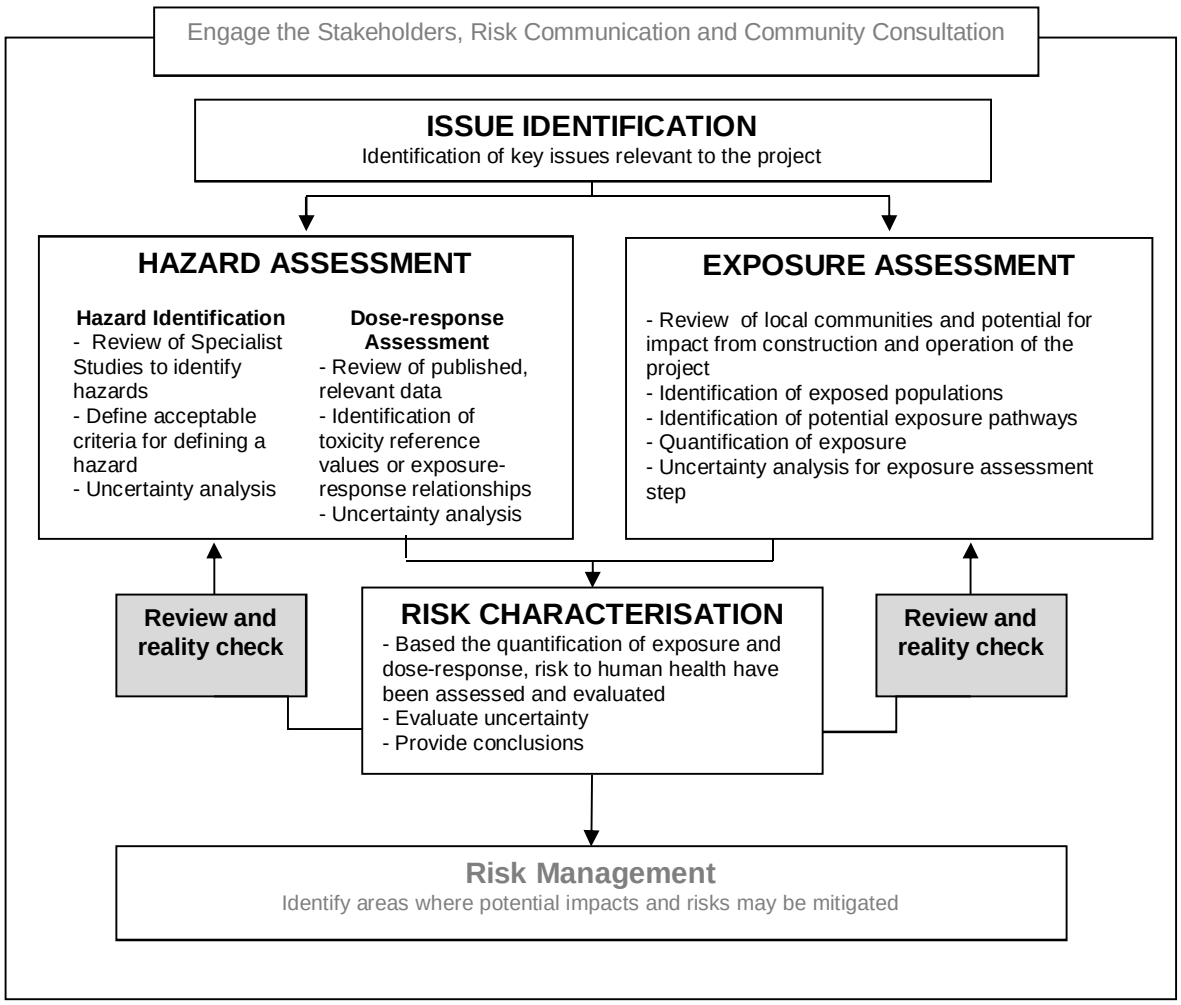

Figure 1-3 Overall human health risk assessment approach (modified from enHealth, 2012)

1.5.3 Features of the risk assessment

The HHRA has been carried out in accordance with international best practice and general principles and methodology accepted in Australia by groups such as 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:

- 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, ground level concentrations modelled in the local community and hence is expected to overestimate the actual risks.
- Conclusions can only be drawn with respect to emissions to air derived from the project as outlined in this technical working paper.
- Available statistics in relation to the existing health status of the existing community are presented in the technical working paper; however the HHRA 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, further studies are undertaken and more detailed and critical review of information is conducted.

This assessment does not address all the health impacts, both positive and negative, associated with the project. Rather the assessment presented in this technical working paper has focused on key impacts (negative impacts) to air quality and noise/vibration identified by NSW Health as requiring detailed consideration within the environmental impact statement. It is noted that the project is set to deliver a number of key improvements and these are further outlined in Chapter 11 of the environmental impact statement.

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Section 2. Project design

2.1 General

This section presents an overview of the project design being considered in this technical working paper. The details presented provide a summary of key aspects of the project that are discussed in detail within Chapter 5 of the environmental impact statement.

Key features of the project would include:

- Twin motorway tunnels up to around nine kilometres in length with two lanes in each direction. The tunnels would be constructed with provision for a possible third lane in each direction if required in the future.
- A northern interchange with the M1 Pacific Motorway and Pennant Hills Road, including sections of tunnel for on-ramps and off-ramps, which also facilitate access to and from the Pacific Highway.
- A southern interchange with the Hills M2 Motorway and Pennant Hills Road, including sections of tunnel for on-ramps and off-ramps.
- Integration works with the Hills M2 Motorway including alterations to the eastbound carriageway to accommodate traffic leaving the Hills M2 Motorway to connect to the project travelling northbound, and the provision of a new westbound lane on the Hills M2 Motorway extending through to the Windsor Road off-ramp.
- Tie-in works with the M1 Pacific Motorway extending to the north of Edgeworth David Avenue.
- A motorway operations complex located near the southern interchange on the corner of Eaton Road and Pennant Hills Road that includes operation and maintenance facilities.
- Two tunnel support facilities, which incorporates emergency smoke extraction outlet points and substations along the main alignment.
- Ancillary facilities for motorway operation, such as electronic tolling facilities, signage, ventilation systems and fire and life safety systems including emergency evacuation infrastructure.
- Modifications to service utilities and associated works at surface roads near the two interchanges and operational ancillary facilities.
- Modifications to local roads, including widening of Eaton Road near the southern interchange and repositioning of the Hewitt Avenue cul-de-sac near the northern interchange.
- Ancillary temporary construction facilities and temporary works to facilitate the construction of the project.

Construction activities would generally include:

- Enabling and temporary works, including construction power, water supply, site establishment, demolition works, property and utility adjustments and public transport modifications (if required).
- Construction of the road tunnels, interchanges, intersections and roadside infrastructure.
- Haulage of spoil generated during tunnelling and excavation activities.
- Fit-out of the road tunnels and support infrastructure, including ventilation and emergency response systems.
- Construction and fit-out of the motorway control centre and ancillary operations buildings.
- Realignment, modification or replacement of surface roads, bridges and/or underpasses.
- Environmental management and pollution control facilities for the project.

2.2 Interchanges

2.2.1 Southern interchange

The southern interchange would be located near the existing intersection of the Hills M2 Motorway and Pennant Hills Road at Carlingford (refer to **Figure 2-1**). The interchange would provide connections to and from the project with the Hills M2 Motorway and Pennant Hills Road.

To enable these new connections, surface road works along Pennant Hills Road immediately north of the Hills M2 Motorway would be required. Works along the Hills M2 Motorway for connection to the project tunnel portals would also be required.

Portals to the northbound on-ramp and southbound off-ramp along Pennant Hills Road would be located south of Eaton Road. The main alignment tunnel portals would emerge adjacent to the shoulders of the Hills M2 Motorway to the west of Pennant Hills Road providing an uninterrupted connection between the Hills M2 Motorway.

2.2.2 Northern interchange

The northern interchange would be located near the intersection of the M1 Pacific Motorway and Pennant Hills Road at Wahroonga (refer to **Figure 2-2**). The northern interchange would connect the project with the M1 Pacific Motorway and Pennant Hills Road to enable traffic to travel north, south or east. In addition to this, the northern interchange would provide connections for traffic on or from Pennant Hills Road and the Pacific Highway to continue travelling via these existing roads.

Portals to the southbound on-ramp and northbound off-ramp for Pennant Hills Road would be located to the east of Pennant Hills Road within the median of the Pennant Hills Road / M1 Pacific Motorway connector. This would require a widened section of road between these portals and Pennant Hills Road. This design approach has been adopted to minimise the need for permanent alterations to existing roadways and traffic arrangements.

The portals of the main alignment tunnels would emerge in the shoulders of the M1 Pacific Motorway to the north of Alexandria Parade in the vicinity of Bareena Avenue, Wahroonga.

Figure 2-1 Southern interchange operational layout

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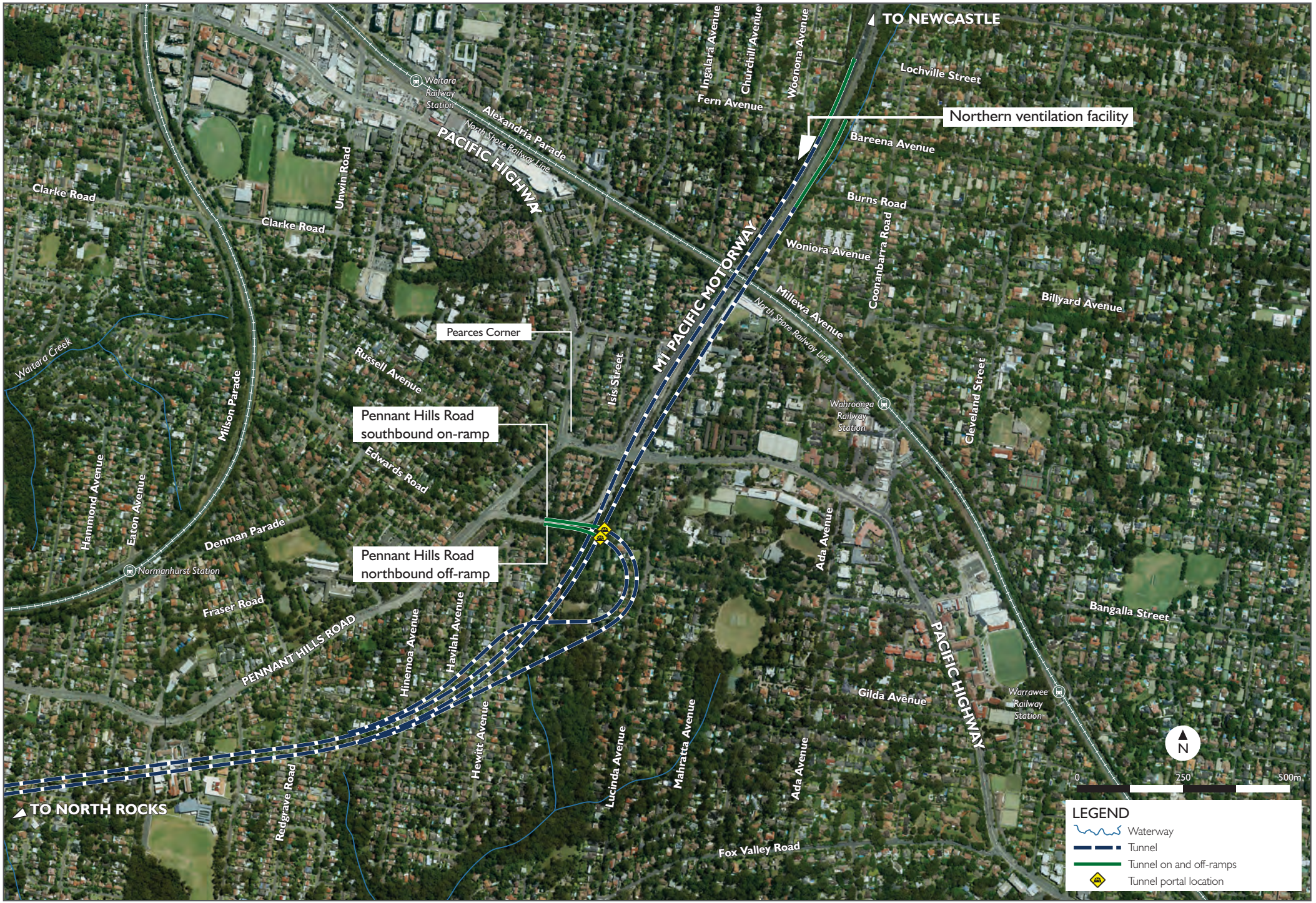

Figure 2-2 Northern interchange operational layout

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2.3 Ventilation system

Tunnel ventilation is proposed to be undertaken through the use of the following:

- During normal operation fresh air is drawn into the portals via a vehicle generated piston effect (ie the suction created behind a moving vehicle pulls air into and through the tunnel). Air in the tunnels would be pushed towards the main tunnel exit portals. Near the main tunnel exit portals air would be drawn upwards into the ventilation facilities and vented to atmosphere via the discharge points.
- Jet fans, mounted in pairs within the northbound and southbound tunnels, separated by a minimum of 90 metres. Jet fans would be located throughout the tunnel and would operate as required to maintain in tunnel air quality requirements.
- Ventilation facilities near the northern and southern main alignment portals (refer to **Figure 2-3**. Near the main tunnel exit portals air from the tunnel would be drawn into the ventilation facility where it would be discharged via a 15 metres high discharge point (when measured from adjoining land). Jet fans are used to draw air back in to the ventilation facility from the on and off-ramps.
- The ventilation system has been designed so there are no portal emissions (ie emissions from the tunnel exit portals directly to surrounding air). All air within the tunnel would be extracted from the tunnel and discharged to the atmosphere via the ventilation facilities.
- Two emergency smoke extraction facilities would be located on the corner of Wilson Road and Pennant Hills Road (southern) and on the corner of Trelawney Street and Pennant Hills Road (northern), refer to **Figure 2-3**. These facilities would be designed to extract smoke in the event of an emergency fire incident with a capacity of around 400 m^3/s. During low speed traffic conditions the emergency smoke extraction facilities could be used to provide additional fresh air into the tunnels.
- During low-speed traffic conditions there is the potential for additional fresh air to be supplied to the main tunnels via the reverse flow operation of the fans in the two tunnel support facilities.

The project has been designed so that all air from the project tunnels can be discharged via the two tunnel ventilation facilities.

The project does not currently propose portal emissions from the main alignment tunnels, however this approach may be considered in future and would be subject to appropriate assessment and approval. This would include a human health risk assessment.

2.4 Construction works

The majority of the construction footprint is located underground within the main alignment tunnels, however surface areas would be required to support tunnelling activities, and to construct the interchanges, tunnel portals, the Hills M2 Motorway integration, the M1 Pacific Motorway tie-in, the motorway operations complex, northern and southern ventilation buildings, tunnel support facilities and ancillary operations buildings and facilities. The surface construction footprint is presented in **Figure 2-4**.

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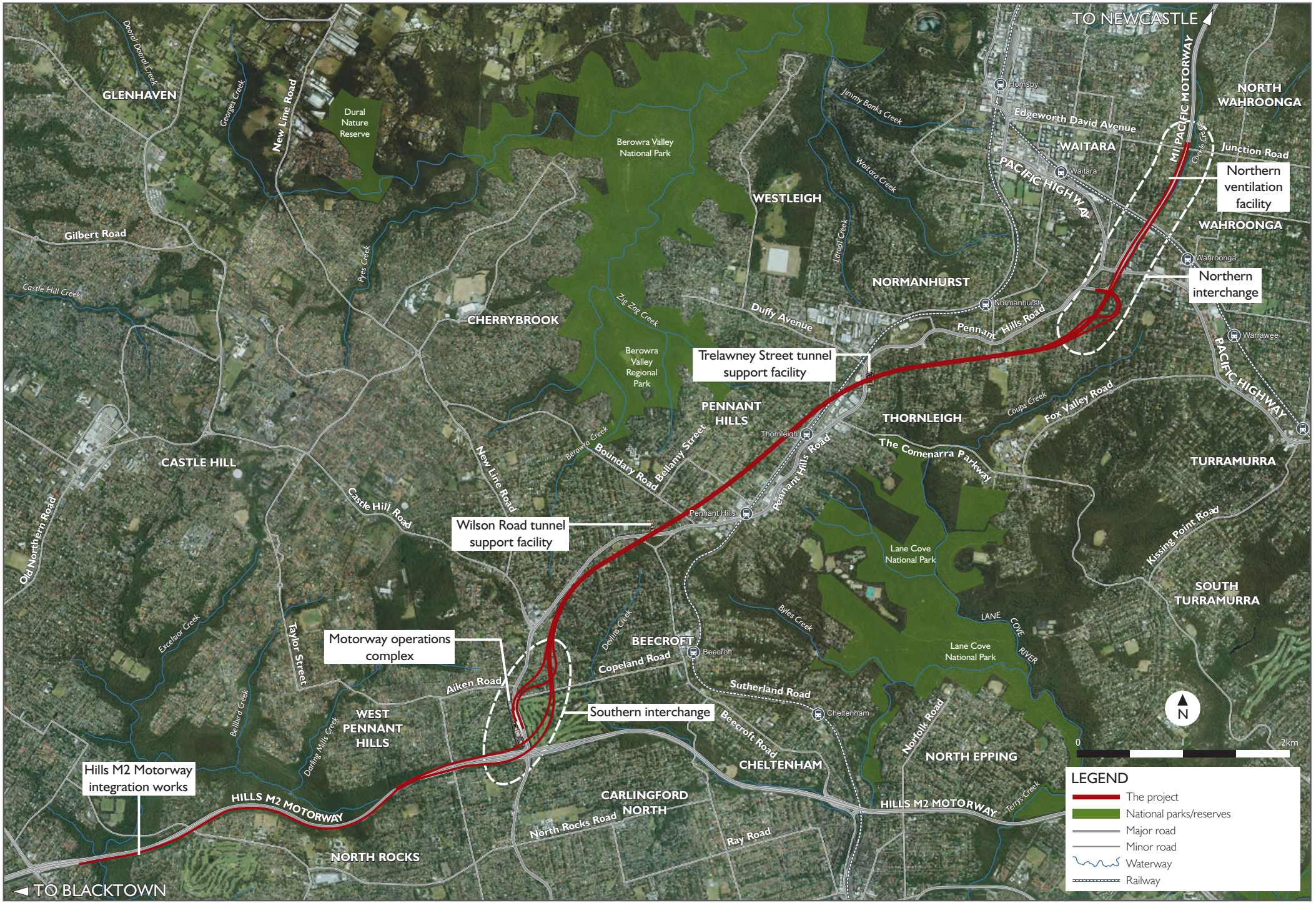

Figure 2-3 Ventilation and emergency smoke extraction facilities

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Figure 2-4 Overview of the construction footprint and ancillary facilities

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2.5 Benefits of the project

The project is set to deliver a number of key improvements that are outlined in Chapter 11 of the environmental impact statement. In summary the benefits of the project include:

- Providing the missing link in Sydney's motorway network and the National Land Transport Network between the Hills M2 Motorway and the M1 Pacific Motorway.
- Future travel time savings of up to 40 minutes compared to without the project.
- Bypassing of 21 sets of traffic lights.
- Improving the efficiencies of intrastate and interstate freight movements through travel time saving and reduced operating costs.
- Improving safety of motorists, cyclists and pedestrians on Pennant Hills Road through the reduction in heavy vehicles.
- Improving local amenity and connectivity for people living, working and traveling along Pennant Hills Road.
- Providing opportunities for future public transport improvements and the reinvigoration of the Pennant Hills Road corridor.

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Section 3. Community profile

3.1 General

This section provides an overview of the community potentially impacted by the project. The key focus of the assessment presented is the local community, however some aspects of the assessment require consideration of statistics that are derived from larger populations, such as those within the Northern Area Health District and the greater Sydney Area. Hence, where relevant, information related to both the local community and other areas within Sydney (and NSW) have been presented.

3.2 Surrounding area and population

The main alignment tunnel covers a distance of around nine kilometres from Carlingford in the south to Wahroonga in the north. The population considered in this assessment includes those who live along Pennant Hills Road (where a reduction in road use and vehicle emissions is expected as part of this project) as well as within the vicinity of the southern and northern interchanges (ie where the tunnel interfaces with the surface road network).

Southern interchange

The southern interchange and ventilation facility is located near the current intersection between Pennant Hills Road and the M2 Hills Motorway. This is located in West Pennant Hills. The suburbs (or partial suburbs) surrounding the southern interchange include:

- West Pennant Hills.
- Carlingford.
- Beecroft/Cheltenham.
- North Rocks.
- Epping.

These suburbs surrounding the interchange are predominantly low to medium/high density residential areas with some retail/commercial areas. There are a number of day care centres and schools located in the suburbs surrounding the interchange.

Northern interchange

The northern interchange is located near the current intersection of Pennant Hills Road and the M1 Pacific Highway. This is located in the central western portion of Wahroonga. The ventilation facility is located on the western side of the M1 Pacific Motorway near Woonona Avenue in Wahroonga. The suburbs (or parts of these suburbs) surrounding the northern interchange include:

- Wahroonga.
- North Wahroonga.
- Waitara.
- Hornsby.
- Normanhurst.

Pennant Hills Road Alignment (parts of these suburbs):

- Wahroonga.
- Normanhurst.
- Thornleigh.
- Pennant Hills.
- West Pennant Hills
- Beecroft.

The suburbs located adjacent to Pennant Hills Road and surrounding the southern and northern interchanges are predominantly low to medium/high density residential areas with some retail/commercial areas. There are a number of day care centres, schools, aged care and hospitals located in these suburbs.

Sensitive receivers

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 sensitive receivers, have been identified in the suburbs surrounding the southern and northern interchanges and evaluated. Sensitive receivers are 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.

The location of sensitive receivers within one to two kilometres of the southern and northern interchanges are shown on **Figure 3-1** and **Figure 3-2**, and listed in **Table 3-1** and **Table 3-2**. The receivers presented in **Tables 3-1 and 3-2** are not an exhaustive list and some receivers have been grouped together (where they are located close to each other).

Table 3-1 Location of sensitive receivers surrounding the southern interchange

No.	Sensitive receivers	Address
Child Care		
51	Bird House Early Learning Centre	4/6 Leigh Place West Pennant Hills
52	Shine Preschool	54 Dryden Ave Carlingford
53	Thinking Hats Early Learning Centre and Twinklestar Childcare	3 and 3A Welham St Beecroft
54	Beecroft Long Day & Early Learning Centre	23A Wongala Crescent, Beecroft
Aged Care		
55	Twilight Aged Care: Jamieson House	8 York St Beecroft
56	Southern Cross Nordby Village	15 Hill Road West Pennant Hills
57	Beecroft Nursing Home	134 Beecroft Rd, Beecroft
Schools		
58	Murray Farm Public School	Tracey Ave Carlingford
59	Beecroft Primary School	90-98 Beecroft Rd Beecroft
60	North Rocks Public School	359 North Rocks Rd North Rocks
61	St Gerards Primary School	543 North Rocks Rd Carlingford
62	Roselea Primary School	549 North Rocks Rd Carlingford
63	Carlingford High School	North Rocks Rd Carlingford
64	Colin Place Out of School Care	2 Colin Place Carlingford
65	Muirfield High School	9-13 Barclay Road, North Rocks
66	West Pennant Hills Public School	Church Street, West Pennant Hills
67	Arden Anglican School	50 Oxford Street Epping
Other		
68	Pennant Hills Golf Course	Burns Rd Beecroft/Carlingford
69	West Pennant Hills Community Church	41-43 Eaton Rd West Pennant Hills
70	Roselea Community Centre	647-671 Pennant Hills Rd Carlingford

Table 3-2 Location of sensitive receivers surrounding the northern interchange

No.	Sensitive receivers	Address
Child Care		
1	KU Wahroonga	23 Millewa Lane Wahroonga
2	Next Generation Child Care	30 Myra St Wahroonga
3	Bumble Bees Early Learning Centre	76 King Road Hornsby
4	Balamara Preschool	79 Edgeworth David Ave Waitara
5	Peter Rabbit Community Preschool	St Pauls Church Hall, Pearces Corner Wahroonga
6	Centacare Broken Bay Waitara Children's Services Long Daycare (Waitara Family Centre)	29 Yardley Ave Waitara
7	Wahroonga Long Day Care	37 Hewitt Ave Wahroonga
8	Normanhurst Child Care Centre	66 Denman Pde Normanhurst
9	Pymble Turramurra Kindergarten	21 Handley Ave, Turramurra
10	Wahroonga Beehive Pre-School	168 Eastern Rd, Wahroonga
11	Kids Academy Hornsby	36-38 Northcote Rd, Hornsby
12	Twinkle Tots Cottage	18 Wentworth Ave, Waitara
13	Explore & Develop Waitara Little Learning School Hornsby Bright Horizons Early Learning Centre	41 Balmoral Street, Hornsby 90 Balmoral Street, Hornsby 94 Balmoral Street, Hornsby
14	Little Learning School Wahroonga	89 Burdett Street, Wahroonga
Aged Care		
15	The Woniora Aged Care	9 Woniora Ave Wahroonga
16	Tallwoods Corner	1 Myra St Wahroonga
17	The Grange	2 McAuley Place Waitara
18	B'nai B'rith Retirement Village	3-9 Jubilee St Wahroonga
19	Bowden Brae Retirement Village	40-50 Pennant Hills Rd, Normanhurst
20	Greenwood Aged Care	9-17 Hinemoa Ave, Normanhurst
21	Wahroonga Nursing Home	31 Pacific Hwy, Wahroonga

No.	Sensitive receivers	Address
22	Netherby Aged Care Belvedere Aged Care Wahroonga Waldorf Apartments	17-19 Pacific Hwy, Wahroonga 9 Pacific Hwy, Hornsby 1 Woolcott Ave, Wahroonga
25	Thomas & Rosetta Aged Care Facility Redleaf Serviced Apartments/Aged Care	1634 Pacific Hwy, Wahroonga 1630 Pacific Hwy, Wahroonga
26	UPA of NSW Ltd (United Prodestant Association, aged care facility)	1614 Pacific Hwy, Wahroonga
Schools		
28	Waitara Public School	68 Edgeworth David Ave Wahroonga
29	Wahroonga Preparatory School	61 Coonanbarra Rd Wahroonga
30	Wahroonga Public School	71 Burns Road, Wahroonga
31	Hornsby Girls High School	Edgeworth David Ave Hornsby
32	Normanhurst Boys High School	Pennant Hills Rd Normanhurst
33	Normanhurst Public School	2/14 Normanhurst Rd Normanhurst
34	Abbotsleigh	1666 Pacific Highway Wahroonga
35	Abbotsleigh Junior School and Early Learning Centre	22 Woonona Ave Wahroonga
36	Knox Grammar	7 Woodville Ave Wahroonga
37	Knox Preparatory School	1-13 Billyard Ave, Wahroonga
38	Our Lady of the Rosary Primary School	23 Yardley Ave Waitara
39	St Lucys School	21 Cleveland Street Wahroonga
40	Prouille Catholic College	Cleveland Street, Wahroonga
41	Prouille Catholic Primary School	5 Water Street Wahroonga
42	St Leos	Woolcott Ave Wahroonga
43	Barker College	Pacific Highway, Hornsby
44	Warrawee Public School	1486 Pacific Hwy Warrawee
45	St Edmund's School for Blind and Visually Impaired	60 Burns Road, Wahroonga
46	Hornsby South Public School Clarke Road School	57-63 Clarke Road, Hornsby Clarke Road and Neutral Rd, Hornsby
48	Retaval School	100 Fox Valley Rd, Wahroonga
Other		
49	Hornsby Hospital (and childcare centre)	Palmerston Rd Hornsby
50	Neringah Hospital (hope Healthcare)	4 Neringah Ave Wahroonga

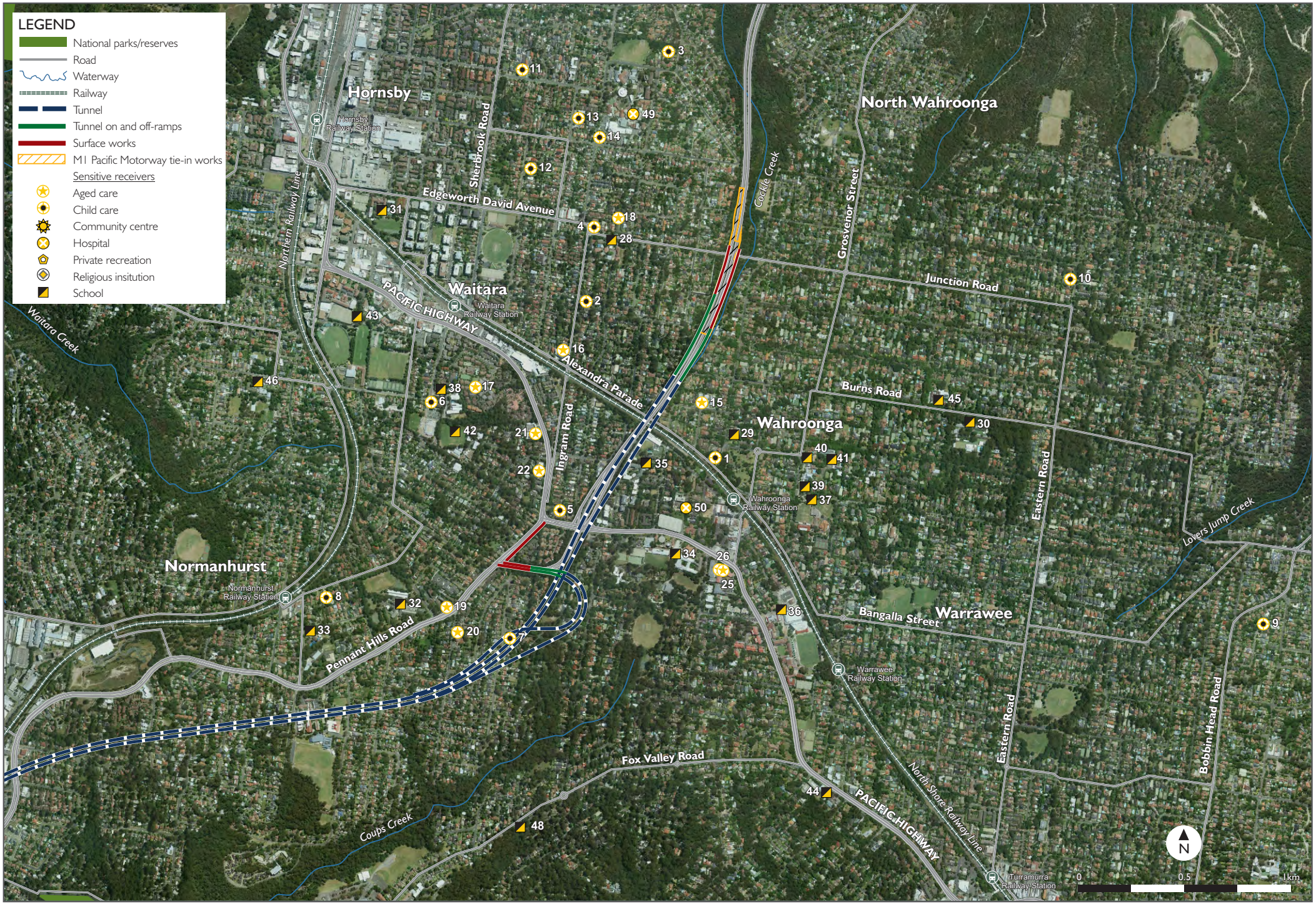

Figure 3-1 Location of sensitive receivers - northern interchange

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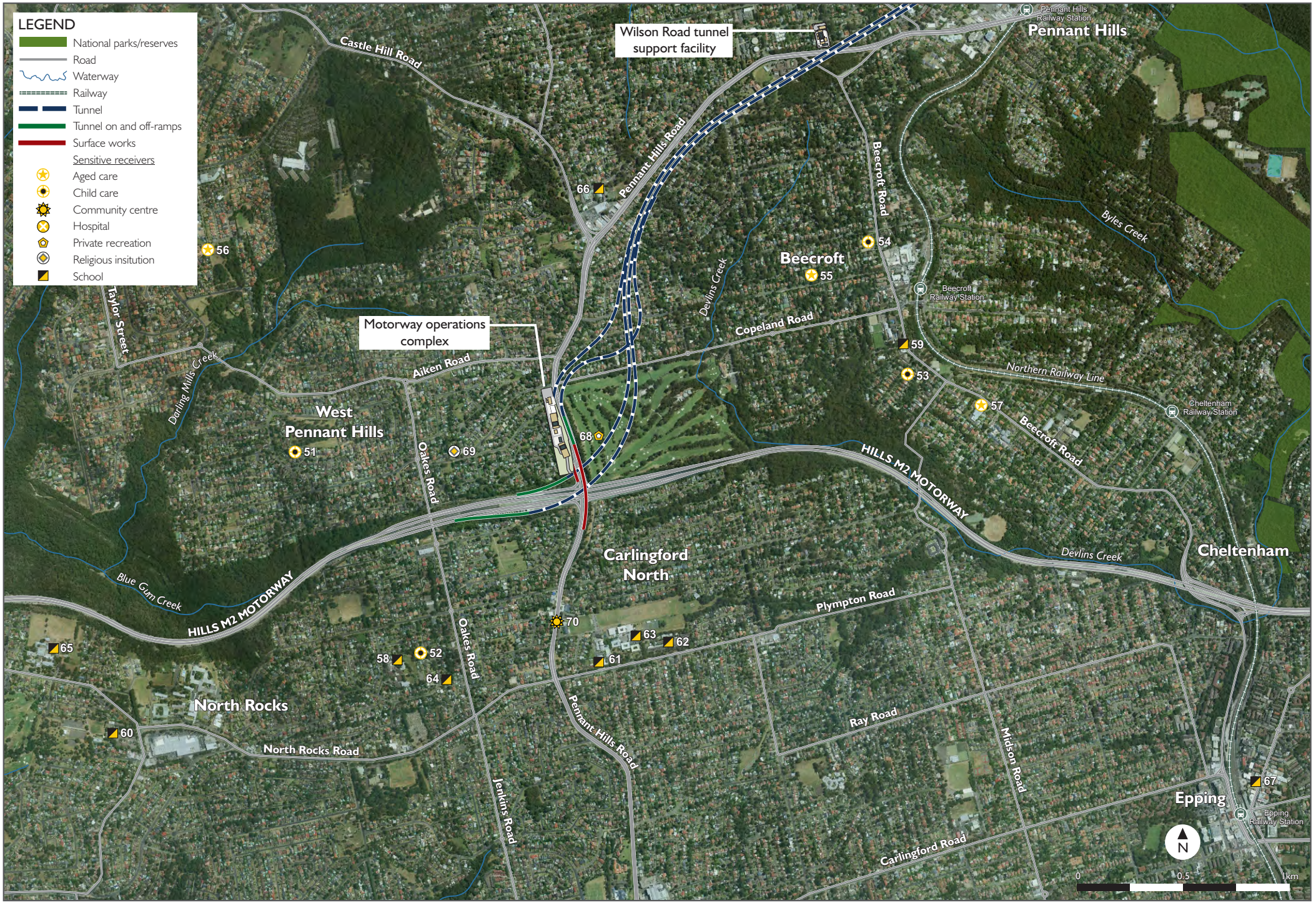

Figure 3-2 Location of sensitive receivers - southern interchange

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3.3 Population profile

The population within the areas surrounding the southern and northern interchanges comprise residents and workers as well as those attending schools, day-care and recreational areas within the surrounding suburbs. The composition of the populations located within one to two kilometres km of the northern and southern interchanges is expected to be generally consistent with population statistics for the larger individual suburbs. Population statistics for the suburbs (based on state suburb areas) surrounding the northern and southern interchanges are available from the Australian Bureau of Statistics for the census year 2011 and are summarised in **Figure 3-3** and the following graph. For the purpose of comparison the population statistics presented also include the statistics for the larger statistical areas of Hornsby South (which includes most of the suburbs of interest for this project), greater Sydney and the rest of the NSW (excluding greater Sydney).

Table 3-3 presents a summary of a selected range of demographic measures relevant to the population of interest with comparison to statistical local area of Hornsby South, greater Sydney and the rest of the NSW (excluding greater Sydney).

Table 3-3 Summary of population statistics

Location	Total Population		% Population by Key Age Groups				
	Male	Female	0-4	5-19	20-64	65+	30+
Southern interchange							
Carlingford	10594	10976	5.2	20	58.6	16.1	63
West Pennant Hills	7813	8154	5.1	21.3	61.2	12.4	61
Beecroft	4186	4650	4.7	21.8	54.9	18.5	63
North Rocks	3761	3864	6.5	19.9	57.4	16.2	64
Epping	9883	10344	4.8	18.9	63	13.3	60
Northern interchange							
Wahroonga	8001	8725	5.6	23	53.7	17.7	62
North Wahroonga	949	937	4.8	22.3	56.6	16.3	63
Warrawee	1440	1472	4.6	23.7	58.1	13.6	58
Waitara	2584	2786	7.8	14.3	62.9	14.9	64
Hornsby	9694	10169	7.2	15.7	65.5	11.6	62
Normanhurst	2410	2746	6.6	22.9	52	18.5	61
Additional Suburbs Along Pennant Hills Road							
Thornleigh	3976	4139	7.7	20.6	58.2	13.6	70
Pennant Hills	3443	3588	5.8	20	58.6	15.6	74
Larger Statistical Areas							
Hornsby South (Statistical Area)	43701	46404	6.2	19.4	59.6	14.7	62
Greater Sydney	2162221	2229453	6.8	18.7	61.7	12.9	60
Rest of NSW (excluding greater Sydney)	1239007	1273942	6.3	19.7	55.9	18	63

Ref: Australian Bureau of Statistics, Census Data 2011

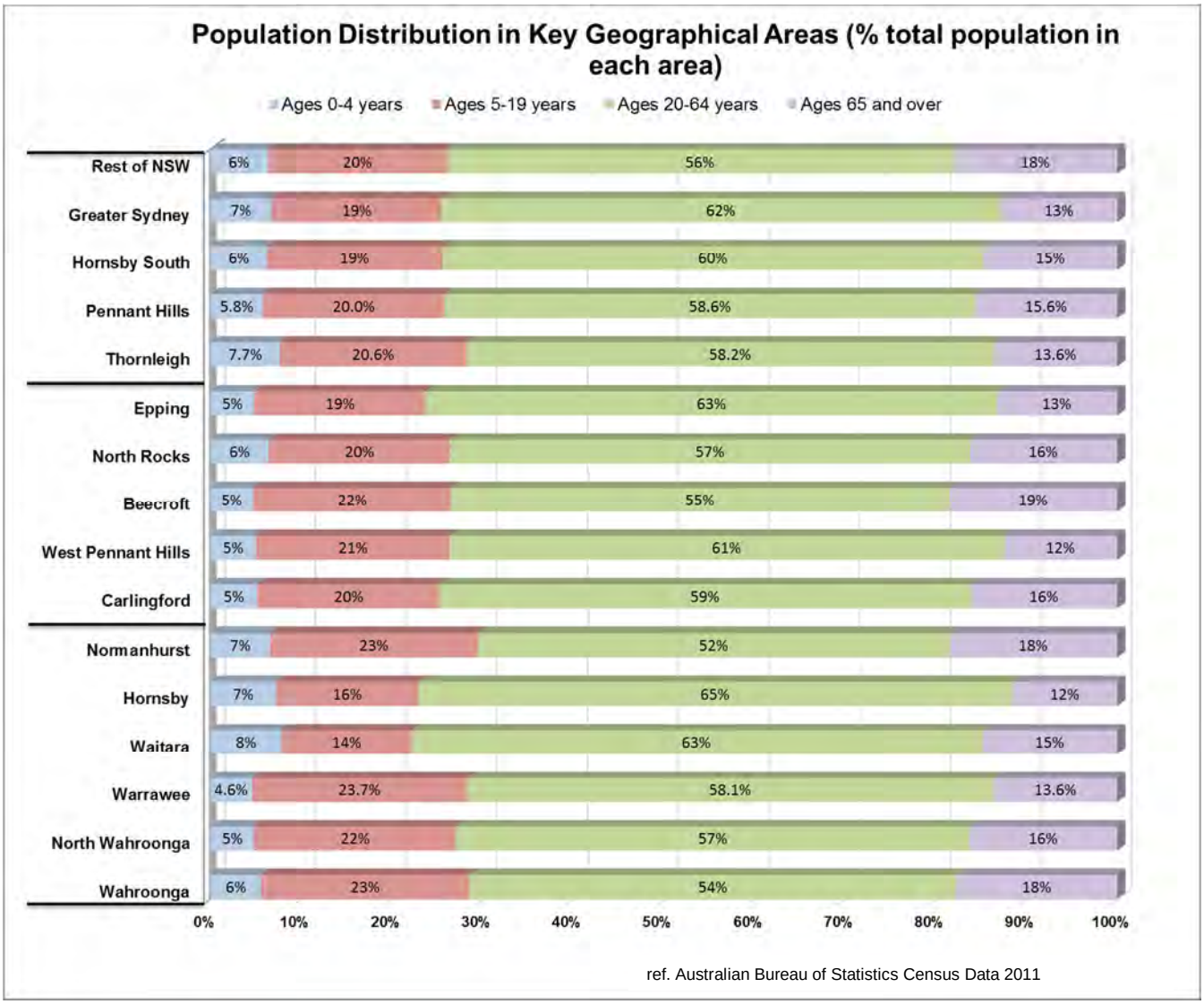


Figure 3-3 Population distribution

Based on this general population data, the suburbs surrounding the southern interchange are generally similar to Greater Sydney with the exception of Beecroft where there is a higher percentage of people aged 65 years and older. The suburbs surrounding the northern interchange are a little more variable with the suburbs of Wahroonga, North Wahroonga, Warrawee and Normanhurst indicating a slightly higher proportion of people aged 5-19 years and 65 years and older (with a corresponding lower proportion of people aged 20-64 years), and the suburbs of Waitara and Hornsby indicating a lower proportion of people aged 5-19 years when compared with the larger area of Greater Sydney. Hornsby South includes most of the suburbs of interest in this project and shows a relatively similar population distribution to that of Greater Sydney.

The suburbs of interest in the project are located in three different local government areas – Hornsby Shire Council, Hills Shire Council and Ku-ring-gai Council.

The estimated population growth for these areas are:

- 37 per cent in the Hills Shire local government area from 2014-2031[1]
- 9 per cent in Hornsby Shire local government area from 2011-2031[2]
- 12 per cent in Ku-ring-gai Shire local government area from 2011-2031[2]

Table 3-4 Selected demographics of population of interest

Location	Median age	Median household income ($/week)	Median mortgage repayment ($/month)	Median rent ($/week)	Average household size	Unemployment rate (%)
Southern Interchange						
Carlingford	40	1572	2200	410	2.9	5.5
West Pennant Hills	41	2449	2600	480	3.1	4.3
Beecroft	43	2523	2650	500	3.0	4.1
North Rocks	40	1891	2500	450	3.0	4.0
Epping	38	1683	2286	420	2.8	6.1
Northern Interchange						
Wahroonga	41	2381	3000	501	2.9	4.2
North Wahroonga	42	2519	3360	673	3.1	4.2
Warrawee	40	2658	3200	530	3.0	5.6
Waitara	34	1413	2167	420	2.3	7.7
Hornsby	35	1436	2167	380	2.5	5.7
Normanhurst	40	1775	2531	334	2.8	5.2
Additional Suburbs Along Pennant Hills Road						
Thornleigh	38	1964	2600	395	2.9	5.6
Pennant Hills	40	1842	2400	400	2.8	5.6
Larger Statistical Areas						
Hornsby South (Statistical Area)	38	1730	2383	400	2.8	5.2
Greater Sydney	36	1447	2167	351	2.7	5.7
Rest of NSW (excluding greater Sydney)	41	961	1560	220	2.4	6.1

Ref: Australian Bureau of Statistics, Census Data 2011

The social demographics of an area have some influence on the health of the existing population. As shown in **Table 3-4**, the population located in the vicinity of the northern and southern interchanges, and along Pennant Hills Road, generally has lower unemployment (with the exception of Waitara and Epping) with a higher income and also higher mortgage repayments and rental costs compared with Greater Sydney and the rest of NSW.

[1] http://forecast.id.com.au/the-hills/home

[2] http://www.nsforum.org.au/files/HACC-Misc/HACC-Planning-Framework/Northern%20Sydney%20Planning%20Framework%202008%20S3.pdf

3.4 Existing health of population

3.4.1 General

The assessment presented in this report has focused on key pollutants that are associated with construction and combustion sources (from vehicles), including particulate matter (namely $PM_{2.5}$ and PM_{10}). For these pollutants there are a large number of sources in the project area including other combustion sources (other than from the project), other local construction/earthworks and personal exposures (such as smoking) and risk taking behaviours that have the potential to affect the health of any population.

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, socio-economic status, social capital, 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.

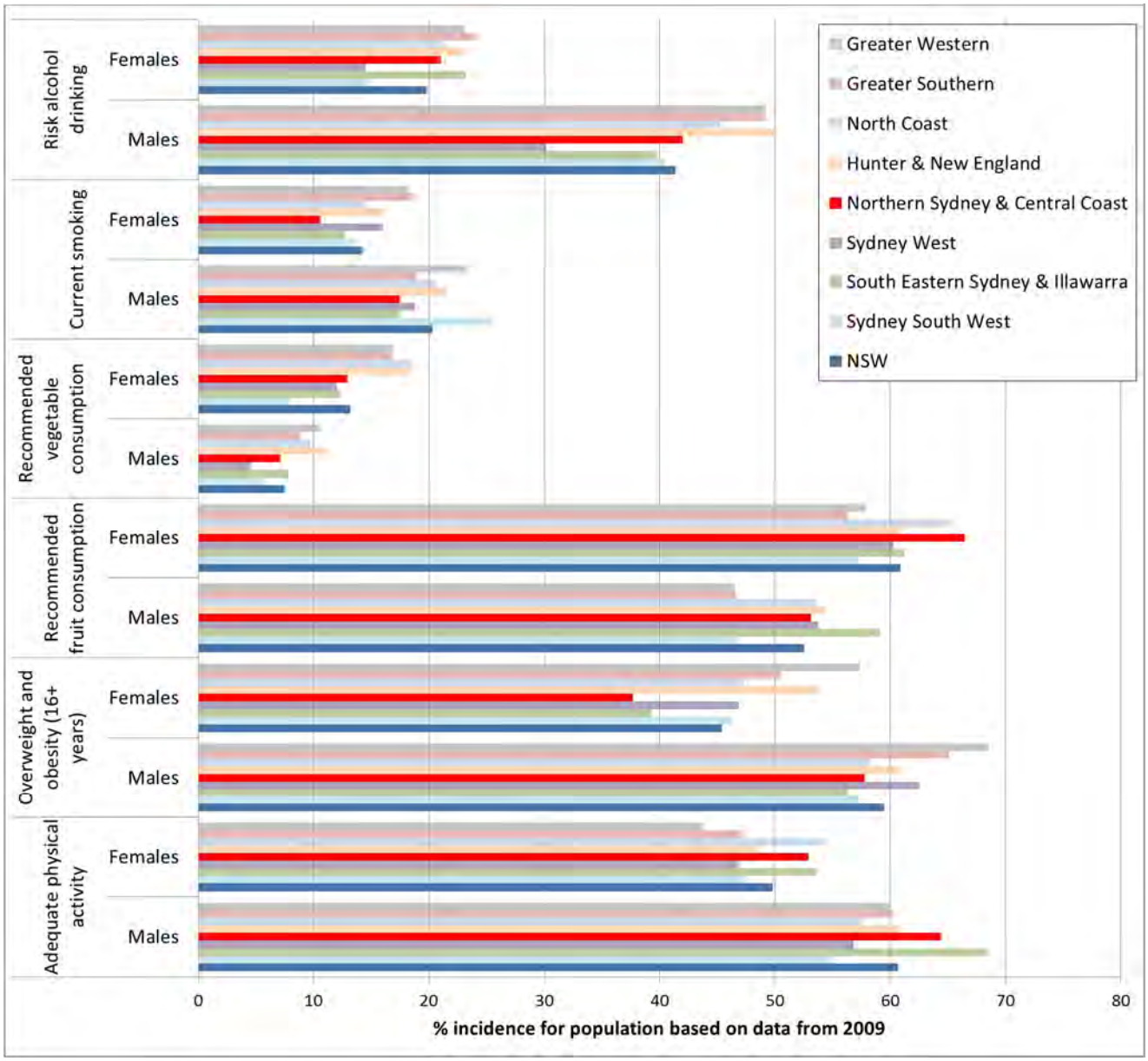
Most of the health indicators presented in this report are not available for each of the smaller suburbs/statistical areas surrounding the site, as outlined in **Section 3.1** to **Section 3.3**. Health indicators are only available from a mix of larger areas (that incorporate the study area) - the Northern Sydney Area Health Service and/or the combined area of Northern Sydney and the Central Coast. 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 in the vicinity of the northern and southern interchanges given the similarity of the demographics of these populations to Greater Sydney.

3.4.2 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) are available for large health population areas in Sydney and NSW. This includes risky alcohol drinking, smoking, consumption of fruit and vegetables, overweight and obesity and adequate physical activity. The study population is grouped in the larger population area of Northern Sydney and Central Coast. The incidence of these health-related behaviours in this area, compared with other health areas in NSW, and the state of NSW (based on data from 2009) is illustrated in Figure 3-4.

Review of this data generally indicates the population in the Northern Sydney and Central Coast area:

- Have similar rates of risky alcohol drinking, recommended consumption of vegetables and overweight and obesity compared with NSW.
- Have higher rates of recommended consumption of fruit and adequate physical activity compared with NSW.



Note: these health-related behaviours include those where the behaviour/factor may adversely affect health (e.g. alcohol drinking, smoking, being overweight and obesity) and others where the behaviour/factor may positively affect (enhance) health (e.g. adequate fruit and vegetable consumption and adequate physical activity)

Figure 3-4 Summary of incidence of health-related behaviours 2009 (source: NSW Health, 2010)

3.4.3 Health indicators

Figure 3-5 and **Figure 3-6** present a comparison of the rates of the key mortality indicators (all causes, potentially avoidable, cardiovascular disease, lung cancer and chronic obstructive pulmonary disease (COPD in the elderly 65+ years)) and hospitalisations (diabetes, cardiovascular disease, asthma (5-34 years) and COPD (65+ years)) reported in the larger Northern Sydney and Central Coast Area Health Service, with comparison to other NSW area health services (in urban and regional areas) as well as NSW as a whole.

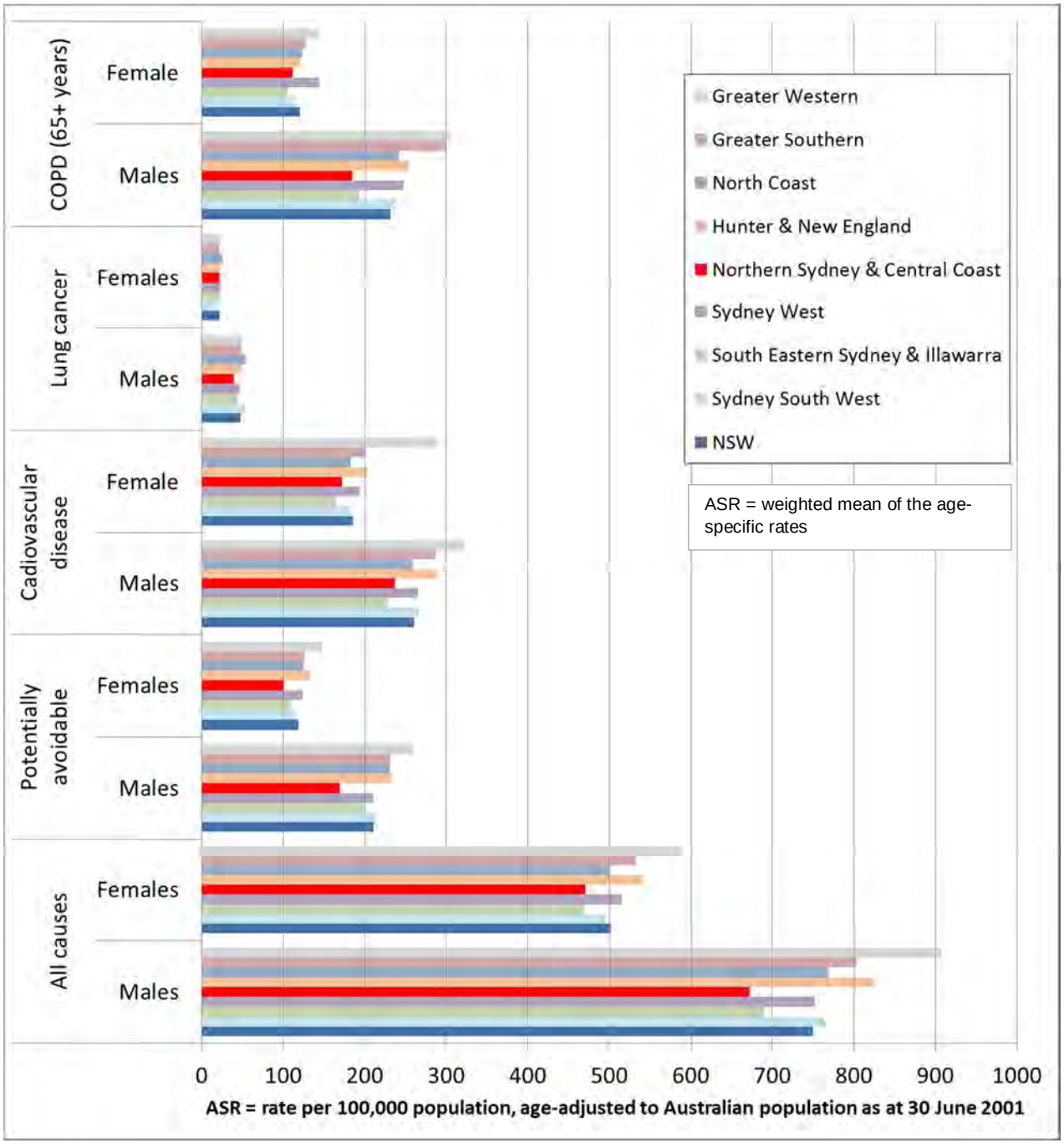


Figure 3-5 Summary of mortality data 2003 – 2007 (source: NSW Health, 2010)

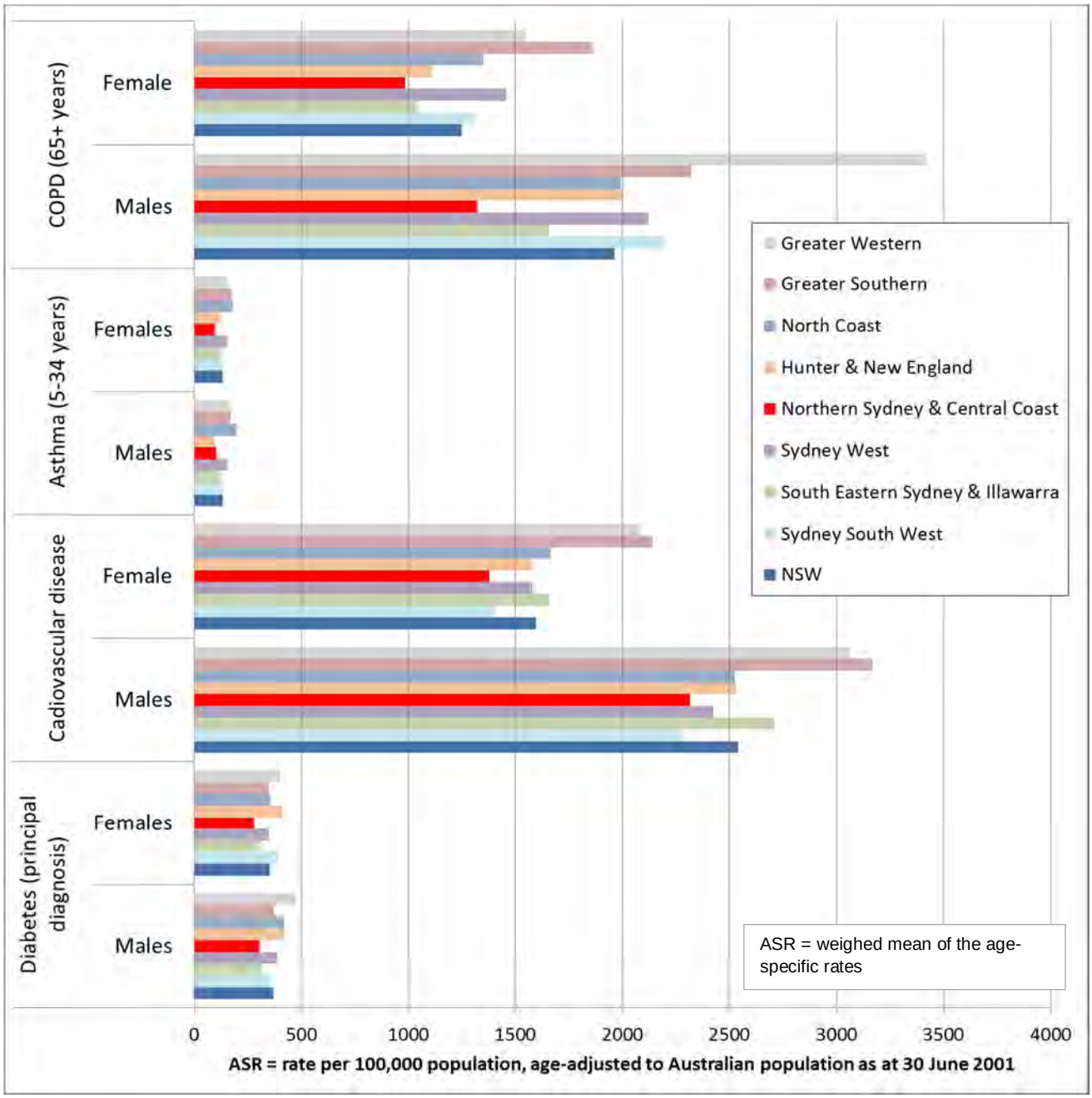


Figure 3-6 Summary of hospitalisation data 2008 – 2009 (source: NSW Health 2010)

In relation to some more specific health indicators **Table 3-5** presents the available data for the slightly smaller population areas defined under the Northern Sydney Area Health and for the Hornsby, Ku-ring-gai and the Hills local government areas (or GP health areas). These have been compared with available data for Sydney and NSW. The health indicators include those that are specifically relevant to the quantification of exposure to particulate matter presented in **Section 5.**

Table 3-5 Summary of key health indicators

Health Indicator	Data available for Population (rate per 100,000 population)					
	Hornsby Shire	Ku-ring-gai Shire	The Hills Shire	Northern Sydney Area Health	Greater Sydney	NSW
Mortality						
All causes – all ages*	--	--	--	496.6[1]	586.9[1]	670#[2]
All causes ≥30 years*	--	--	--	--	--	1087#[2]
Cardiopulmonary ≥30 years*	--	--	--	--	--	490#[2]
Cardiovascular – all ages*	--	--	--	--	--	164#[2]
Respiratory – all ages*	--	--	--	--	--	57#[2]
Hospital admissions						
Coronary heart disease	539.5[3]	462.7[3]	597.5[3]	442.3[4]	391.6[4]	608.7[4]
COPD >65 years	647.9[3]	558.1[3]	735.6[3]	745.2[4]	1194.2[4]	1470.4[4]
Cardiovascular disease						
All ages	--	--	--	1642.3[5]	1582.6[5]	1949.9[5]
>65 years*	--	--	--			23352#[3]
Respiratory Disease						
All ages	--	--	--	1520.1[5]	1530.3[5]	1770.2[5]
>65 years*	--	--	--	--	--	8807#[3]
Asthma						
Asthma hospitalisations (ages 5-34 years)	--	--	--	85.7[4]	105.1[4]	133.6[4]
Current asthma for ages 16 and over	--	--	--	12.1%[4]	7.8%[4]	11.3%[4]

* Health indicators directly relevant to the characterisation of potential impacts associated with exposure to particulate matter as presented in **Section 5**
# Data provided by NSW Health (upon written request) for the purpose of this assessment.
All other data has been obtained from Health Statistics New South Wales
1 - Data from 2006-2007
2 – Data for 2005-2007
3 - Data for 2009-2011
4 – Data for 2010-2011
5 – Data for 2011-2012
-- No data available

In relation to asthma, the **Figure 3-7** shows the general indicators reported for the larger population area of Northern Sydney and Central Coast compared with the data available for NSW (also refer to **Appendix A** for comparison with other area health services).

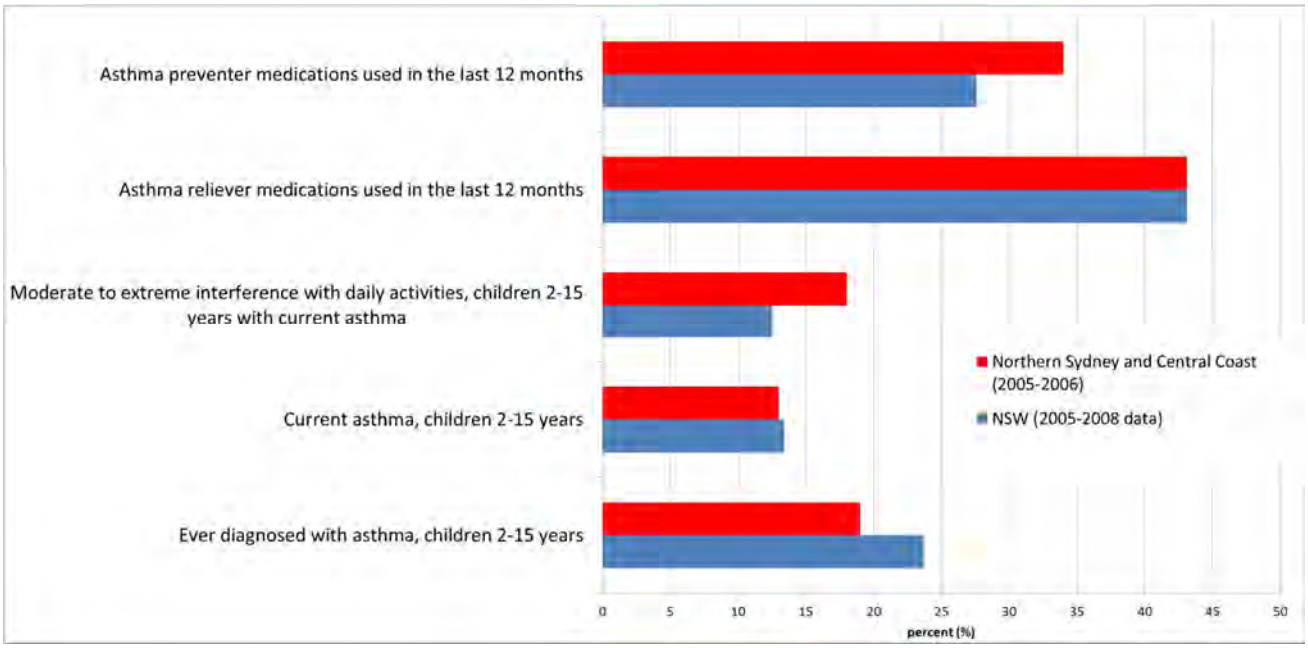


Figure 3-7 Summary of asthma prevalence and management (NSW and Northern Sydney/Central Coast)

Review of the available data generally indicates that for the population in the Northern Sydney area (including the Northern Sydney and Central Coast combined areas where relevant) the health statistics (including mortality rates and hospitalisation rates for most of these categories) are generally lower than compared with a number of other health areas and the whole of NSW.

For the assessment of potential health impacts from the project, where specific health statistics for the smaller population adjacent to the southern and northern interchanges 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 (ie over estimating existing health issues), summary of the existing health of the population of interest.

Uncertainties

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.

3.5 Existing environment

3.5.1 Existing air quality

The existing air quality in the study area is described in the technical working paper: air quality (AQIA) (AECOM, 2014). This technical working paper has used background air quality data collected by the Office of Environment and Heritage at Lindfield and Prospect, which are the closest stations to the project area. The Lindfield monitoring station is around 9.7 kilometres southeast of the southern ventilation outlet and the Prospect monitoring station is around 11 kilometres southwest of the southern ventilation outlet).

Air quality in the greater Sydney area is most significantly affected by bushfires (including hazard reduction burns) and dust storms with transport-related emissions identified as the largest source of human-related pollution. In general, NSW is considered to have good air quality in relation to international standards. Review of $PM_{2.5}$ and PM_{10} in many countries by the WHO[3] identified that concentrations reported in Australia low (amongst the lowest of all countries evaluated) compared with international levels.

Exceedances of the NEPC guidelines and advisory goals for particulate matter (PM) do occur in Sydney (as presented in the AQIA), primarily due to occasional bushfires, dust storms and hazard reduction burns rather than more every day conditions.

In relation to $PM_{2.5}$, review of the sources (emissions) that contribute to the measured $PM_{2.5}$ reported in the Sydney area by the NSW EPA (based on emissions inventory data – for the year 2008, published 2012[4]), as illustrated in **Figure 3-8**, indicates that the most significant sources are household activities (including residential wood heaters – with peak emissions in the winter months from wood-smoke). Emissions from road transport in the Sydney area contribute a consistent amount to the total $PM_{2.5}$ emissions (as would be expected as use of vehicles in Sydney is relatively constant throughout the year). As a percentage of the total emissions, road transport comprises a greater proportion of the total $PM_{2.5}$ emissions in summer compared with winter (where other sources are more dominant).

In relation to the project, five air quality monitoring stations where commissioned in locations along project corridor to supplement data collected by the Office of Environment and Heritage. This data has been collected since late 2013 and has been considered in the AQIA.

[3] WHO, Ambient (outdoors) air pollution in cities database 2014, available from http://www.who.int/phe/health_topics/outdoorair/databases/cities/en/

[4] http://www.epa.nsw.gov.au/woodsmoke/index.htm

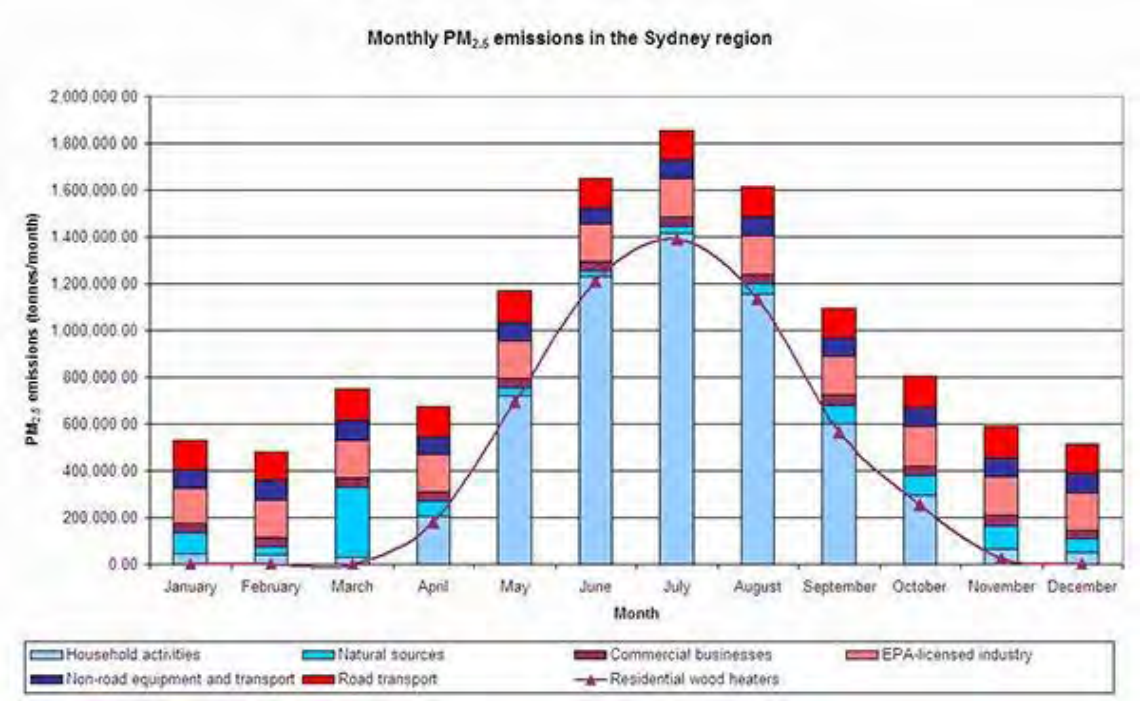


Figure 3-8 $PM_{2.5}$ emissions in Sydney – variability and contributions on monthly basis (2008, source: NSW EPA)

3.5.2 Existing noise environment

The existing noise environment in the study area (particularly adjacent to the Hills M2 Motorway east from Windsor Road, near the southern interchange, Pennant Hills Road, the northern interchange and the M1 Pacific Motorway) is described in the Noise and Vibration Technical Paper (AECOM 2014).

Existing noise in the study area is dominated by road traffic noise, primarily from the M1 Pacific Motorway, Pacific Highway, Pennant Hills Road and the Hills M2 Motorway. Noise in the study area is highly dependent on proximity to the existing roads.

Background noise monitoring (along with traffic counts) has been undertaken at 23 locations throughout the study area to determine the existing background noise levels. The background noise data is used to define appropriate construction noise management limits consistent with the NSW EPA Interim Construction Noise Guideline, and criteria to assess operational road noise or 'fixed' ancillary facilities such as the ventilation facilities (consistent with the NSW EPA Industrial Noise Policy). Background noise monitoring was also used in the assessment of operational traffic noise.

Background noise levels for the 23 locations in the study area were as follows:

- Day (7am to 6pm): rating background levels ranged from 41 to 59 dB(A) as $LA_{90,15}$
- Evening (6pm to 10pm): rating background levels range from 42 to 54 dB(A) as $LA_{90,15}$
- Night (10pm to 7am): rating background levels ranged from 30 to 45 dB(A) as $LA_{90,15}$

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Section 4. Review of air impacts

4.1 Air impact assessment

4.1.1 Summary

Emissions to air associated with the project have been evaluated in detail within the technical working paper: air quality (AECOM 2014) (AQIA). The AQIA has considered emissions to air that may occur during construction activities as well as during the operation of the project.

In relation to construction, emissions to air have been considered from the following sources:

- Construction traffic, plant and equipment where emissions to air are primarily derived from diesel powered vehicles and equipment, however some emissions are derived from motor vehicles.
- Bulk earthworks (underground vented at the surface via a tunnel ventilation system and aboveground) where emissions to air are associated with dust.

Impacts associated with the construction activities were evaluated in the AQIA along with a range of best practice mitigation measures. With the implementation of mitigation measures, the effects of the proposed works on local air quality and receivers are expected to be minimal and of short duration. Hence the focus of the more detailed (quantitative) evaluation of impacts to air quality has focused on the operation of the tunnel ventilation system.

Operational emissions have been estimated from petrol and diesel powered vehicles using the tunnel (in both directions) which are vented to atmosphere via the southern and northern interchange ventilation facilities, and increases in traffic volumes on approaches to the tunnel.

Emissions to air from the operation of the tunnel have been assessed using CALPUFF and CAL3QHCR models, meteorological data collected by the Office of Environment and Heritage (over 2009, 2010 and 2011) and terrain information relevant for the area. The modelling has considered impacts to sensitive receivers located close to the southern and northern interchanges extending (at increasingly reduced density of coverage as distance to the interchanges increase) around 20 kilometres in all directions. In addition a number of sensitive receivers have been included in the modelling for the purposes of this assessment as outlined in **Section 3.2**.

Emission factors for the pollutants of interest from the vehicles proposed to be using the tunnel have been obtained from published sources that include Australian-specific emissions based on the relevant vehicle fleet composition. These factors were used to estimate emissions in the years up to 2020 (taking into account improvements in vehicle emissions over time). The emission factors estimated in 2029 were conservatively assumed to be the same as those determined for 2020 (ie no further improvements in emissions technology assumed).

Vehicle emissions within the tunnel are discharged to air via the ventilation facilities located at the southern and northern interchanges. Specific details of the ventilations facilities (height and diameter, exit velocity and temperature) are presented in the AQIA. These emissions have been used to model air quality using the CALPUFF air dispersion model. In addition, emissions to air that occur on the road network proximal to the main tunnel portals (ie on the approaches) have been

modelled using the CAL3QHCR dispersion model. Predicted impacts from both these models have been summed to obtain the combined impact from the project for the scenarios evaluated. This approach is appropriate for the estimation of impacts associated with the project.

The AQIA has evaluated the key pollutants that are relevant to emissions to air during the operation of the project, which include:

- Particulate matter (PM) including size fractions PM_{10} and $PM_{2.5}$ which are of importance for the assessment of potential health impacts from combustion sources.
- Oxides of nitrogen (in particular NO_2).
- Carbon monoxide (CO).
- Volatile organic compounds (VOCs) as total VOCs.
- Polycyclic aromatic hydrocarbons (PAHs, as total PAHs) which are particularly associated with diesel emissions.

Background levels of key pollutants (particulate matter, carbon monoxide and nitrogen dioxide) levels have been determined from available data on existing air quality from monitoring stations located in Lindfield and Prospect. The background air quality data is relevant for the assessment of cumulative impacts from the project.

Predicted impacts at all gridded and sensitive receiver locations and the maximum predicted concentration, for the scenarios considered, have been provided for consideration in this assessment. The impacts have been presented as incremental impacts (ie the project only) and cumulative impacts (ie the project plus background air quality).

4.1.2 Assessment scenarios

The assessment of emissions to air from the project has been undertaken within the AQIA for a number of scenarios, as outlined below:

Without project (scenario 1)

- This scenario assessed the standard 'do nothing' scenario, which predicted future pollutant concentrations from the surface roads in the event that the project is not constructed, with impacts compared with those predicted with the tunnel operating. Emissions were assessed using the CAL3QHCR model and expected future traffic volumes for the existing road network for 2019 and 2029.
- The outcome of the assessment of this scenario presented within the AQIA identified that predicted roadside concentrations of particulate matter would go down (by between five per cent and 35 per cent) along the existing road corridor of Pennant Hills Road and near the southern interchange. Roadside concentrations of particulate matter near the northern interchange are more variable (due to existing low levels of particulate matter at some locations) with some concentrations predicted to be lower with the tunnel (44 per cent lower) and others slightly higher (14 per cent).
- Overall air quality along the road corridor considered was improved with the construction and operation of the tunnel.

- The assessment presented in this technical working paper has not specifically considered this scenario further in relation to health impacts. However the calculations undertaken for scenario 1 have been utilised in an assessment of the project as a whole (impacts form the ventilation stacks as well as decreases in impacts along the existing road corridor of Pennant Hills Road) as presented in **Section 5.3.6**.

With Project – Expected traffic volumes – 2019 (scenario 2a) and 2029 (scenario 2b):

- This scenario assessed the forecast hourly traffic volumes with variable emission concentrations based on hourly traffic flows, and volumetric flow rates scaled by the predicted traffic volumes. This was done for 2019 (scenario 2a, the proposed year of opening) and 2029 (scenario 2b, design year).
- This scenario is representative of likely traffic flows (and variability including peak hour traffic flows) and hence is considered to be representative of more likely emissions and potential exposures that may occur during normal/expected operations.
- These scenarios have been further evaluated in this technical working paper.

With project – Theoretical maximum peak hour capacity (design analysis A)

- This design analysis has been conducted to ensure that the project's ventilation system is adequately sized to cater for tunnel full of traffic. It assumes that during peak hours, the maximum number of vehicles that can fit into the tunnel (4,000 passenger car units per two lane main alignment tunnel adjusted for speed). This design analysis represents the physical limit of the main alignment tunnels and is based on forecast traffic volumes that are unlikely to eventuate due to a range of factors including traffic management measures, projected land use, employment, demographics and constraints on the surrounding surface road network.

  As this design analysis is not likely to occur (particularly as modelled in 2019) the impacts predicted for this scenario, and the potential for exposure, are considered to be unlikely. The impacts predicted have only been considered (and presented in **Appendix E**) as an indication of worst-case conditions.

With project – Forecast traffic volumes with maximum hourly emissions – 2019/2029 (design analysis B):

- This design analysis has been conducted to ensure that regardless of when the peak traffic period occurs or for how it lasts, the project's ventilation system would be able to meet applicable air quality criteria. This design analysis assumes that the project's ventilation outlets emit the maximum concentration of pollutants based on peak forecast traffic flows on a continuous basis. In reality, emissions concentrations would vary during the day depending on the number and type of vehicles using the tunnels at the time.

  The design analysis is not representative of emissions that may occur during normal or peak traffic flow conditions and is therefore not relevant for the further assessment of exposure and health impacts in the local community.

Breakdown traffic flow:

- Expected vehicle emissions in the tunnel during a credible worst-case breakdown situation were used to calculate the associated pollutant concentrations. This scenario has been addressed on a qualitative basis within the AQIA, where the following has been concluded:
    - Emission rates of carbon monoxide, oxides of nitrogen and particulates during a breakdown are generally lower than the 'with project – expected traffic flows (northern ventilation outlet) in 2029 (which was presented as the highest expected mass emission rates of the scenarios considered in the AQIA) (scenario 2b).
    - Because the mass emission rates for the breakdown scenario are comparable to, but no greater than, the 'with project – forecast traffic flows' scenario, and the breakdown scenario would occur over a relatively short period, it is expected that the breakdown scenario would also comply with applicable air quality criteria it is expected that the breakdown scenario would also comply with applicable air quality criteria

  On the basis of the above, no separate assessment of the breakdown scenario is presented in this report.

This technical working paper has focused on air quality impacts predicted during scenarios 2a (2019) and 2b (2029). Calculations relevant to design analysis A is presented in **Appendix E**.

The following sections provide an initial, or screening level review of the predicted impacts associated with these scenarios. This screening level assessment has focused on the maximum predicted impacts (incremental and cumulative as relevant) from the project to determine if a more detailed review of health impacts would be required.

Impacts in all other areas (including the sensitive receivers) are lower than these maximum predicted impacts/concentrations. Further assessment of the sensitive receivers has been undertaken in the detailed review of exposures to particulate matter emissions presented in **Section 5**.

4.1.3 Vehicle emissions

Petrol and diesel vehicles emit a range of air pollutants that are known to be associated with adverse health impacts. Common air pollutants emitted from these vehicles include:

- Petrol vehicles: nitrogen oxides, in particular nitrogen dioxide, carbon monoxide, fine particulates and volatile organic compounds. The key volatile organic compounds of concern from motor vehicle emissions include benzene, toluene and xylenes (BTX) which have been associated with a range of health effects that range from headaches to eye irritation and cancer (depending on the compound).
- Diesel vehicles: nitrogen oxides, in particular nitrogen dioxide, carbon monoxide, fine particulates, volatile organic compounds (in particular BTX and 1,3-butadiene) and aldehydes (formaldehyde and acetaldehyde); and polycyclic aromatic hydrocarbons (EA 2003). Polycyclic aromatic hydrocarbons are another group of compounds where the toxicity will vary depending on the presence of individual polycyclic aromatic hydrocarbons.

The assessment of emissions from vehicles requires consideration of key urban air pollutants (nitrogen oxides, carbon monoxide), the individual compounds likely to be present in the more general measures of volatile organic compounds (which include BTX, 1,3-butadiene and the aldehydes) and polycyclic aromatic hydrocarbons, and particulates. These are further discussed in the following sections.

4.2 Review of key air pollutants

4.2.1 Oxides of nitrogen

Nitrogen oxides (NOx) refer to a collection of highly reactive gases containing nitrogen and oxygen, most of which are colourless and odourless. Nitrogen oxide gases form when fuel is burnt. Motor vehicles, along with industrial, commercial and residential 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 of concern (WHO 2000a). Nitrogen dioxide is a colourless and tasteless gas with a sharp odour. 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 (Morgan et al. 1998). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of nitrogen dioxide (NEPC, 2010). The health effects associated with exposure to nitrogen dioxide depend on the duration or exposure as well as the concentration; hence guidelines have been developed in Australia (and internationally) that reflect both acute and chronic exposures.

Guidelines are available from the NSW EPA and NEPC (NEPC 2003) that are based on protection from adverse health effects following short-term (acute) and longer-term (chronic) exposure. Review of these guidelines by NEPC (2010) identified additional supporting studies 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 (2010[5], specifically listed to be protective of exposures to 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.

[5] Most recent review of the Primary National Ambient Air Quality Standards for Nitrogen Dioxide published by the USEPA in the Federal Register Volume 75, No. 26, 2010, available from: http://www.gpo.gov/fdsys/pkg/FR-2010-02-09/html/2010-1990.htm

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) 1-hour average concentration in air. The guideline of 246 µg/m^3 (or 120 ppbv) is based on a lowest observed adverse effect level (LOAEL) of 409 to 613 µg/m^3 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 (i.e. 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 4-1 presents a summary of the maximum (for all locations modelled over the years 2009-2011) predicted cumulative 1-hour average concentration of nitrogen dioxide for scenarios 2a (2019) and 2b (2029) relevant for expected emissions from the project.

Table 4-1 Review of potential acute health impacts – nitrogen dioxide (NO_2)

Location and scenario	Maximum 1-hour average concentration of NO_2 (µg/m^3)
Southern interchange	
- Scenario 2a (2019)	165
- Scenario 2b (2029)	167
Northern interchange	
- Scenario 2a (2019)	151
- Scenario 2b (2029)	159
Acute health based guideline	**246**

All the concentrations of nitrogen dioxide presented in the above table are well below the acute NEPC guideline of 246 µg/m^3. Hence there are no adverse health effects expected in relation to acute exposures to nitrogen dioxide in the local area surrounding the project. Hence no further detailed assessment of these exposures is warranted.

Assessment of chronic exposures:

The NEPC ambient air quality guideline for the assessment of chronic (long-term or lifetime) exposures to nitrogen dioxide relates to the maximum predicted total (cumulative) annual average concentration in air. The guideline of 62 µg/m^3 (or 30 ppbv) is based on a lowest observed adverse effect level (LOAEL) of the order of 40 – 80 ppbv (around 75-150 µg/m^3) 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 ppbv (38-75 µg/m^3) (NEPC 1998). On this basis the NEPC (and OEH) chronic guideline is protective of adverse health effects in all individuals, including sensitive individuals.

Table 4-2 presents a summary of the maximum (for all locations modelled over the years 2009-2011) predicted cumulative annual average concentration of nitrogen dioxide for scenarios 2a (2019) and 2b (2029) relevant for expected emissions from the project.

Table 4-2 Review of potential chronic health impacts – Nitrogen dioxide (NO_2)

Location and scenario	Maximum annual average concentration of NO_2 (µg/m^3)
Southern interchange	
- Scenario 2a (2019)	42.4
- Scenario 2b (2029)	42.8
Northern interchange	
- Scenario 2a (2019)	38.7
- Scenario 2b (2029)	39.9
Chronic health based guideline	**62**

All the concentrations of nitrogen dioxide presented in the above table are well below the chronic NEPC guideline of 62 µg/m^3. Hence there are no adverse health effects expected in relation to chronic exposures to nitrogen dioxide in the local area surrounding the project.

As the assessment of potential acute and chronic health impacts are addressed in the guidelines adopted (and considered above), and no predicted impacts exceed these guidelines, no further detailed assessment of these exposures is warranted.

4.2.2 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 (OEH) 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[6] 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[7], specifically listed to be protective of exposures by

[6] 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.

[7] 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: http://www.gpo.gov/fdsys/pkg/FR-2011-08-31/html/2011-21359.htm

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 LOAEL (lowest observed adverse effect level) and NOAELs (no observed adverse effect level) 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 ppmv (or 10 mg/m^3 or 10 000 µg/m^3) over an 8-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 Environment Protection Authority) guideline is protective of adverse health effects in all individuals, including sensitive individuals.

The Environment Protection Authority have also established a guideline for 15-minute average (100 mg/m^3) and 1-hour average (30 mg/m^3) concentrations of carbon monoxide in ambient air. These guidelines are based on criteria established by the WHO (WHO 2000b) 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 4-3 presents a summary of the maximum (for all locations modelled over the years 2009-2011) predicted cumulative 1-hour average and 8-hour average concentrations of carbon monoxide for scenarios 2a (2019) and 2b (2029) relevant for expected emissions from the project.

Table 4-3 Review of potential acute and chronic health impacts – Carbon monoxide (CO)

Location and scenario	Maximum 1-hour average concentration of CO (µg/m^3)	Maximum 8-hour average concentration of CO (µg/m^3)
Southern interchange		
- Scenario 2a (2019)	3695	2635
- Scenario 2b (2029)	3715	2660
Northern interchange		
- Scenario 2a (2019)	3712	2634
- Scenario 2b (2029)	3732	2656
Relevant health based guideline	**30 000**	**10 000**

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

As the assessment of potential acute and chronic health impacts are addressed in the guidelines adopted (and considered above), and no predicted impacts exceed these guidelines, no further detailed assessment of these exposures is warranted

4.3 Review of volatile organic compounds and polycyclic aromatic hydrocarbons

4.3.1 General

The AQIA has considered emissions of volatile organic compounds and polycyclic aromatic hydrocarbons to air from the project. Both volatile organic compounds and polycyclic aromatic hydrocarbons 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 volatile organic compounds and polycyclic aromatic hydrocarbons evaluated will 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 (i.e. identified and quantified as a percentage of the total volatile organic compounds or total polycyclic aromatic hydrocarbons) to ensure that potential impacts associated with exposure to these compounds can be adequately assessed.

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

Volatile organic compounds and polycyclic aromatic hydrocarbons from the project are associated with emissions from vehicles assumed to be using the tunnel (and approaches). The makeup of the volatile organic compounds and polycyclic aromatic hydrocarbons emissions would depend on the mix of vehicles considered as these pollutants will 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 proportion of passenger vehicles, light duty vehicles and heavy goods vehicles in 2013 has been considered in the AQIA as follows:

- Of the total vehicle fleet using the tunnel the proportion that will be heavy goods vehicles is estimated to be:
    - 2019: 27.8 per cent to 28.6 per cent (maximum assumed for calculations).
    - 2029: 24.5 per cent to 25.2 per cent (maximum assumed for calculations).
- The remaining vehicles using the tunnel comprise 83.4 per cent passenger vehicles and 16.6 per cent light duty vehicles.
- All the heavy goods vehicles are assumed to be diesel powered.
- Passenger vehicles are assumed to comprise 92.1 per cent petrol and 7.9 per cent diesel powered vehicles. Conservatively, none are assumed to be hybrid, electric or LPG (where emissions would be lower than from petrol or diesel vehicles).
- Light duty vehicles are assumed to comprise 50.1 per cent petrol and 49.9 per cent diesel powered vehicles.

4.3.2 Volatile organic compounds

Volatile organic compounds have been modelled in the AQIA based on emissions from all vehicles considered. The proportion of each of the individual volatile organic compounds that may be present in the air is then estimated based on the assumed composition of the vehicle fleet 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, heptanes etc) (EPA 2012). From a toxicity perspective the key volatile organic compounds 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 (DEH 2003; EPA 2012).

The proportion of each of the key volatile organic compounds 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 the AQIA (as summarised above). In relation to passenger vehicles it has been assumed that sixty per cent[8] of fuel used is E10. It is assumed that the composition of volatile organic compounds in vehicle emissions remains the same over time, and does not improve (lower) with improved vehicle emissions technology.

Table 4-4 presents a summary of volatile organic compounds speciation profile considered for the different vehicle types considered in the project as well as the weighted mass fraction for these volatile organic compounds considered for the project in 2019 and 2029.

Table 4-4 Volatile organic compounds speciation profile for vehicle emissions

VOC	Mass fraction (per cent VOC)					Mass Faction for Vehicle Fleet in Project (%VOC)	
	Passenger Vehicles		Light duty vehicles		Heavy goods vehicles	2019	2029
	No Ethanol	E10	Petrol	Diesel*	Diesel		
1,3-butadiene	1.27	1.2	1.27	0.4	0.4	0.91	1.0
acetaldehyde	0.46	1.3	0.46	3.81	3.81	2.1	1.6
benzene	4.96	4.54	4.96	1.07	1.07	3.3	3.8
formaldehyde	1.46	1.82	1.46	9.86	9.86	4.9	3.9
xylenes	7.6	7.22	7.6	0.38	0.38	4.6	5.5
toluene	9.18	8.79	9.18	0.47	0.47	5.6	6.7

Volatile organic compounds speciation from EPA (2012)
* speciation for diesel emissions also adopted for diesel passenger vehicles

[8] The value of 60 per cent of ethanol in total fuel volume sales was adopted as the target for petrol sold in NSW as outlined in the *Biofuels Act 2007*.

4.3.3 Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons have been considered in the AQIA as key pollutants that may be derived from diesel powered heavy goods vehicles. The presence of polycyclic aromatic hydrocarbons in diesel exhaust has been found to be more a function of the polycyclic aromatic hydrocarbon content of the fuel than of engine technology. For a given refinery and crude oil, diesel fuel polycyclic aromatic hydrocarbon 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 are 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 polycyclic aromatic hydrocarbons in diesel exhaust.

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

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

On the basis of this approach the speciation of individual polycyclic aromatic hydrocarbons (as per cent of total polycyclic aromatic hydrocarbons) has been calculated based on the data from DEH (2003). The data presented relates to emissions that occur during two traffic scenarios (termed segments):

- Segment 1 – congested urban traffic which comprises stop/start traffic flow. This data has been used to be representative of the worst-case situation of heavy congested traffic in the tunnel.
- Segment 4 – highway or freeway traffic which comprises moving traffic. This data is considered more representative of the continuous flow traffic expected in the tunnel.

Table 4-5 presents a summary of the polycyclic aromatic hydrocarbon speciation profile considered in this assessment for the above traffic conditions.

Table 4-5 Polycyclic aromatic hydrocarbon speciation profile for diesel vehicle emissions

Individual PAH	Fraction of total PAH emissions (% PAHs)	
	Congested traffic (worst-case) – Used to evaluate emissions for design analysis A (refer to Appendix E)	Highway/freeway (steady traffic flow) – Used to evaluate emissions for scenarios 2a and 2b
Non-carcinogenic PAHs		
Naphthalene	70	65.7
Acenaphthalene	4.9	5.4
Acenaphthene	2	1.4
Fluorene	5	6.9
Phenanthrene	3.4	13.7
Anthracene	0.49	1.1
Fluoranthene	0.45	0.8
Pyrene	0.71	1.4
Carcinogenic PAHs		
Benzo(a)pyrene TEQ	4.6	0.9

4.3.4 Review of health impacts

The predicted (incremental) concentration of individual volatile organic compounds and polycyclic aromatic hydrocarbons associated with the project (based on the speciation as outlined above) have been reviewed 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 1-hour maximum concentration) that may be associated with volatile organic compounds in the air;
- 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 polycyclic aromatic hydrocarbons in the air.

Table 4-6 and **Table 4-7** present a summary of the maximum predicted 1-hour or annual average concentration with comparison against acute (**Table 4-6**) and chronic (**Table 4-7**) 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 volatile organic compounds and polycyclic aromatic hydrocarbons considered. The total HI is a sum of the potential hazards associated with all the volatile organic compounds and polycyclic aromatic hydrocarbons together assuming the health effects are additive, and is evaluated as follows:

- A total HI ≤ 1 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 > 1 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 volatile organic compounds or polycyclic aromatic hydrocarbons 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 following evaluation is based on the maximum predicted (incremental) concentration in air for scenarios 2a (2019) and 2b (2029) as modelled in the AQIA.

Concentrations in other areas of the surrounding community would be lower and hence the tables present a worst-case evaluation only.

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Table 4-6 Evaluation of potential acute impacts in local area

Key VOC	Proportion of total VOCs (%)*		Health based acute guideline, and basis (µg/m^3)	Maximum predicted 1-hour average concentration from project** and calculated HI for each scenario and interchange							
				Scenario 2a (operational emissions - 2019)				Scenario 2b (operational emissions - 2029)			
				Northern interchange		Southern interchange		Northern interchange		Southern interchange	
	2019	2029		Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI
Total VOCs				**4.1**		**3.7**		**5.4**		**7.4**	
Benzene	3.3	3.8	**29^{A1} to 170^{T1}** (lower value adopted) A1: Acute guideline (1hr to 14 day exposure), based on immunological effects in mice. T1: Acute 1 hour health based guideline, based on depressed peripheral lymphocytes and depressed mitogen-induced blastogenesis (mice study)	0.13	0.0046	0.12	0.0042	0.20	0.0070	0.20	0.0070
Toluene	5.6	6.7	**4500^{T2}** Acute 1 hour health based guideline, based on eye and nose irritation, increased occurrence of headache and intoxication in human male volunteers	0.23	0.000051	0.21	0.000047	0.36	0.000080	0.36	0.000080
Xylenes	4.6	5.5	**2200^{T3}** Acute 1 hour health based guideline, based on mild respiratory effects and subjective symptoms of neurotoxicity in human volunteers	0.19	0.000086	0.17	0.000079	0.30	0.00013	0.30	0.00013
1,3-Butadiene	0.9	1.0	**660^{O1}** Acute 1 hour health based guideline, based on developmental effects	0.037	0.000056	0.034	0.000051	0.054	0.000082	0.054	0.000082
Formaldehyde	4.9	3.9	**15^{T4}** Acute 1 hour health based guideline, based on eye and nose irritation in human volunteers	0.20	0.013	0.18	0.012	0.21	0.014	0.206	0.014

Key VOC	Proportion of total VOCs (%)*		Health based acute guideline, and basis (µg/m^3)	Maximum predicted 1-hour average concentration from project** and calculated HI for each scenario and interchange							
				Scenario 2a (operational emissions - 2019)				Scenario 2b (operational emissions - 2029)			
				Northern interchange		Southern interchange		Northern interchange		Southern interchange	
	2019	2029		Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI
Acetaldehyde	2.1	1.6	**470**[O2] Acute 1 hour health based guideline, based on effects on sensory irritation, bronchoconstriction, eye redness and swelling	0.083	0.00018	0.076	0.00016	0.088	0.00019	0.087	0.00019
			Total HI		**0.018**		**0.017**		**0.021**		**0.021**

Notes:

* Percentage of each individual volatile organic compound is based on a weighted average of emissions from the range of vehicle types proposed to be used on the project in 2019 and 2029 (refer to discussion above table)

** Concentrations presented for the 1 hour average are the predicted incremental 99.9th percentile concentrations (as provided from the AQIA)

A1: Acute inhalation guideline (for exposures from 1 hour to 14 days) from review by ATSDR 2008 for benzene

T1: TCEQ 2007, Benzene, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor). This acute guideline is lower than that derived by the OEHHA (based on older studies)

T2: TCEQ 2008, Toluene, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor)

T3: TCEQ 2009, Xylenes, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor)

T4: TCEQ 2008, Formaldehyde, Development Support Document. Texas Commission of Environmental Quality, 1 hour average guideline value (include additional 3.3 fold safety factor). This guideline is noted to be lower than the acute guideline available from the WHO (2000a, 2010) of 100 µg/m^3 for formaldehyde

O1: OEHHA 2013, Acute (1 hour average) guideline derived by the California Office of Environmental Health Hazard Assessment. The guideline developed is lower than developed by TCEQ (2008) based on the same critical study

O2: OEHHA 2008, Acute (1 hour average) guideline derived by the California Office of Environmental Health Hazard Assessment

Table 4-7 Evaluation of potential chronic impacts in local area

Key VOC	Proportion of total VOCs* (%)		Health based chronic guideline and basis (µg/m^3)	Maximum predicted annual average concentration from project** and calculated HI for each scenario and interchange							
				Scenario 2a (operational emissions - 2019)				Scenario 2b (operational emissions - 2029)			
				Northern interchange		Southern interchange		Northern interchange		Southern interchange	
				Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI
	2019	2029	**Total VOCs**	**0.11**		**0.11**		**0.14**		**0.13**	
Benzene	3.3	3.8	**1.7**[W1] Benzene is classified as a known human carcinogen by IARC. Chronic guideline based on excess risk of leukaemia	0.0035	0.0020	0.0037	0.0022	0.0052	0.0030	0.0049	0.0029
Toluene	5.6	6.7	**5000**[U1] Chronic guideline based on neurological effects in an occupational study (converted to public health value using safety factors)	0.0059	$1.2X10^{-6}$	0.0063	$1.3X10^{-6}$	0.0092	$1.8X10^{-6}$	0.0087	$1.7X10^{-6}$
Xylenes	4.6	5.5	**220**[A1] Chronic guideline based on mild subjective respiratory and neurological symptoms in an occupational study (converted to public health value using safety factors)	0.0049	0.000022	0.0052	0.000023	0.0076	0.000034	0.0072	0.000033
1,3-Butadiene	0.9	1.0	**0.3**[U2] 1,3-Butadiene is classified by IARC as a probable human carcinogen. Chronic air guideline based on an excess risk of leukaemia	0.00095	0.0032	0.00101	0.0034	0.00138	0.0046	0.00131	0.0044
Formaldehyde	4.9	3.9	**3.3**[T1] Formaldehyde is classified by IARC as carcinogenic to humans. The guideline developed is based on the protection of all adverse effects including cancer and non-cancer (including short term effects)	0.0051	0.0015	0.0054	0.0016	0.0053	0.00160	0.0050	0.00152
Acetaldehyde	2.1	1.6	**9**[U3] Chronic guideline based on nasal effects (in a rat study) (converted to a public health value using safety factors)	0.0022	0.00024	0.0023	0.00025	0.0022	0.00025	0.0021	0.00024

Key VOC	Proportion of total VOCs* (%)	Health based chronic guideline and basis (µg/m^3)		Maximum predicted annual average concentration from project** and calculated HI for each scenario and interchange							
				Scenario 2a (operational emissions - 2019)				Scenario 2b (operational emissions - 2029)			
				Northern interchange		Southern interchange		Northern interchange		Southern interchange	
				Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI	Max Conc. (µg/m^3)	HI
		Total PAHs		**1.9X10^{-5}**		**2.1X10^{-5}**		**2.3X10^{-5}**		**2.1X10^{-5}**	
Naphthalene	65.7	**3**[U4] Chronic guideline based on nasal effects (in a mouse study) (converted to a public health value using safety factors)		1.3X10^{-5}	4.2X10^{-6}	1.4X10^{-5}	4.5X10^{-6}	1.5X10^{-5}	5.0X10^{-6}	1.4X10^{-5}	4.6X10^{-6}
Acenaphthylene	5.4	200[U5S]	Refer to notes for ref U5	1.0X10^{-6}	5.2X10^{-9}	1.1X10^{-6}	5.6X10^{-9}	1.2X10^{-6}	6.1X10^{-9}	1.1X10^{-6}	5.7X10^{-9}
Acenaphthene	1.4	200[U5]		2.7X10^{-7}	1.3X10^{-9}	2.9X10^{-7}	1.4X10^{-9}	3.2X10^{-7}	1.6X10^{-9}	2.9X10^{-7}	1.5X10^{-9}
Fluorene	6.9	140[U5]		1.3X10^{-6}	9.5X10^{-9}	1.4X10^{-6}	1.0X10^{-8}	1.6X10^{-6}	1.1X10^{-8}	1.4X10^{-6}	1.0X10^{-8}
Phenanthrene	13.7	140[U5S]		2.6X10^{-6}	1.9X10^{-8}	2.8X10^{-6}	2.0X10^{-8}	3.1X10^{-6}	2.2X10^{-8}	2.9X10^{-6}	2.1X10^{-8}
Anthracene	1.1	100[U5]		2.1X10^{-7}	2.1X10^{-9}	2.3X10^{-7}	2.3X10^{-9}	2.5X10^{-7}	2.5X10^{-9}	2.3X10^{-7}	2.3X10^{-9}
Fluoranthene	0.8	140[U5]		1.5X10^{-7}	1.1X10^{-9}	1.6X10^{-7}	1.2X10^{-9}	1.8X10^{-7}	1.3X10^{-9}	1.7X10^{-7}	1.2X10^{-9}
Pyrene	1.4	100[U5]		2.7X10^{-7}	2.7X10^{-9}	2.9X10^{-7}	2.9X10^{-9}	3.2X10^{-7}	3.2X10^{-9}	2.9X10^{-7}	2.9X10^{-9}
Benzo(a)pyrene TEQ	0.9	**0.00012**[W2] 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 value. The chronic guideline is based on protection from lung cancer for an occupational study		1.7X10^{-7}	0.00144	1.9X10^{-7}	0.00155	2.0X10^{-7}	0.0017	1.9X10^{-7}	0.0016
		Total HI (VOCs + PAHs)			**0.0085**		**0.0090**		**0.011**		**0.011**

Notes:

*	Percentage of each individual volatile organic compounds and polycyclic aromatic hydrocarbons is based on a weighted average of emissions from the range of vehicle types proposed to be used on the project in 2019 and 2029, and for normal traffic flow or congested traffic flow (refer to discussion above table)
**	Concentrations presented for the annual average are as provided from the AQIA
A	Polycyclic aromatic hydrocarbon speciation data for normal traffic conditions – utilised in the assessment of scenarios 2a and 2b
W1:	WHO 2000 Air Quality Guidelines, value for benzene is based on non-threshold carcinogenic effects (excess lifetime risk of leukaemia). Guideline value based on incremental cancer risk of $1x10^{-5}$, consistent with guidance provided by NEPM (1999 amended 2013) and enHealth (2012)
W2:	WHO 2010 Guidelines for Indoor Air Quality, value for BaP is based on non-threshold carcinogenic effects from occupational study of coke workers (lung cancer is critical effect). Guideline value based on incremental cancer risk of $1x10^{-5}$, consistent with guidance provided by NEPM (1999 amended 2013) and enHealth (2012)
T1:	TCEQ 2008, Formaldehyde, Development Support Document. Texas Commission of Environmental Quality. The air guideline 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 (2010)
A1:	ATSDR 2007, Toxicological Profile for Xylene, chronic inhalation guideline derived is the most current robust evaluation
U1:	USEPA evaluation for toluene (most recently reviewed in 2005). 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 (1999 amended 2013) health based guidelines
U2:	USEPA evaluation of 1,3-butadiene (most recently updated in 2002) with the chronic guideline adopted as the lower from the evaluation of non-threshold carcinogenic effects and non-cancer effects. This is the most conservative evaluation of this compound. A more recent review by TCEQ (2013) on the basis of the same critical studies as well as more current studies resulted in a higher chronic air guideline value.
U3:	USEPA evaluation of acetaldehyde (most recently updated in 1991). The guideline established is lower than more recent reviews undertaken by the WHO (2000) and the Californian OEHHA where less conservative evaluations are presented.
U4:	USEPA evaluation of naphthalene (most recently updated in 1998). The guideline established is and is consistent with the value used to derive the NEPM (1999 amended 2013) health based guidelines
U5:	Guideline available from the USEPA. Chronic guidelines for non-carcinogenic polycyclic aromatic hydrocarbons 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 protection of public health, including the use of safety factors) for use in this assessment. 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 70kg and inhalation of 20 m^3/day (as per (USEPA 2009a))
U5S:	No guideline available for individual polycyclic aromatic hydrocarbon, hence a surrogate compound has been used for the purpose of screening. The surrogate compound is a polycyclic aromatic hydrocarbon of similar structure and toxicity. In relation to the surrogates adopted in this evaluation, acenaphthene has been adopted as a surrogate for acenaphthylene, fluoranthene has been adopted as a surrogate for phenanthrene

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Review of the acute assessment presented in **Table 4-6** indicates that during expected operation of the tunnel (in 2019 and 2029) the maximum short-duration peak (1 hour average) concentrations of volatile organic compounds (assessed as the key individual volatile organic compounds and as a sum of all the individual volatile organic compounds) in air surrounding the northern and southern interchanges are well below the relevant acute health based guidelines. The maximum HI calculated for acute exposure to the volatile organic compounds is 0.021, well below the target HI of 1 (around 50 times lower than the target HI). On this basis no further detailed assessment of the peak emissions of volatile organic compounds from the project is warranted.

Review of the chronic assessment presented in **Table 4-7** indicates that during expected operation of the tunnel (in 2019 and 2029) the maximum long-term average (annual average) concentrations of volatile organic compounds and polycyclic aromatic hydrocarbons (assessed as the key individual volatile organic compound and polycyclic aromatic hydrocarbon compounds and as a sum of all the individual volatile organic compounds and polycyclic aromatic hydrocarbons) in air surrounding the northern and southern interchanges are well below the relevant long-term (chronic) health based guidelines. These are guidelines that are based on the protection of public health for inhalation exposures all day (24 hours), every day (365 days per year) for a lifetime (at least 70 years). The maximum HI calculated for exposure to the volatile organic compounds and polycyclic aromatic hydrocarbons is 0.011, well below the target HI of 1 (around 90 times lower than the target HI). On this basis no further detailed assessment of the emissions of individual volatile organic compounds and polycyclic aromatic hydrocarbons from the project is warranted.

4.4 Review of particulate matter

4.4.1 General

Particulate matter (PM) is a widespread air pollutant with a mixture of physical and chemical characteristics that vary by location (and source). Unlike many other pollutants, particulates comprise a broad class of diverse materials and substances, with varying morphological, chemical, physical and thermodynamic properties, with sizes that vary from <0.005 µm to >100 µm. Particulates 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[9] 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.

4.4.2 Particulate size and composition

The potential for particulate matter to result in adverse health effects is dependent on the size and composition of the particulate matter.

The size of particulates is important as it determines how far from an emission source the particulates may be present in air (with larger particulates settling out close to the source and smaller particles remaining airborne for greater distances) and also the potential for adverse effects to occur as a result of exposure.

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 particulates with an equivalent aerodynamic particle[10] size below 50 microns (µm) in diameter[11]. 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 (µm) and larger) are more of a nuisance as they will deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory system[12] and do not reach the lungs. Finer particles (smaller than 10 µm, termed “respirable”) tend to be transported further from the source and are of more concern with respect to human health as these particles can penetrate into the lungs. 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.

[9] 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 (i.e. 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.

[10] The term equivalent aerodynamic particle is used to reference the particle to a particle of spherical shape and particle of density 1 g/cm^3

[11] The size, diameter, of dust particles is measured in micrometers (microns, µm).

[12] 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.

- **PM_{10}, particulate matter below 10 µm in diameter, $PM_{2.5}$, particulate matter below 2.5 µm in diameter and PM_1, particulate matter below 0.1 µm in diameter (termed ultrafine particles):** 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[13] and lungs. Once in the lungs adverse health effects may result (OEHHA 2002). It is well accepted nationally and internationally that monitoring for PM_{10} is a good method of determining the community's exposure to potentially harmful dust (regardless of the source) and is most commonly measured in local and regional air quality monitoring programs. Smaller particulates such as $PM_{2.5}$ and PM_1, however, are of most significance with respect to evaluating health effects as a higher proportion of these particles penetrate deep into the lungs. Urban air, that has a significant contribution from combustion sources, tends to have a significant proportion of $PM_{2.5}$ and PM_1 in ambient air.

Evaluation of size alone as a single factor in determining the potential for particulate toxicity and 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 (<$PM_{2.5}$) and crustal materials (like soil) in the coarse mode (PM_{10} or larger). 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 (ozone, nitrogen dioxide, carbon monoxide and sulfur dioxide).

There is strong evidence to conclude (USEPA 2012; WHO 2003, 2013b) that fine particles (< 2.5 µm, $PM_{2.5}$) are more hazardous than larger ones (coarse particles), primarily on the basis of studies conducted in urban air environments where there is a higher proportion (as a percentage of all particulates) of fine particulates and other gaseous pollutants present from fuel combustion sources, as compared to particulates derived from crustal origins. Toxicological and controlled human exposure studies indicate that primary particles generated from fossil fuel combustion processes may be a significant contributor to adverse health outcomes with several physical, biological and chemical characteristics of particles found to elicit cardiopulmonary responses. Amongst the characteristics found to be contributing to toxicity in epidemiological and controlled exposure studies are high organic carbon content, metal content, presence of polycyclic aromatic hydrocarbons, presence of other organic components or endotoxins and both small (< 2.5 µm) and extremely small size (< 1 µm) (USEPA 2009b; WHO 2003, 2006a).

[13] 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.

A significant amount of research, primarily from large epidemiology studies, has been conducted on the health effects of particulates with causal effects relationships identified for exposure to $PM_{2.5}$ (acting alone or in conjunction with other pollutants) (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). The health effects identified from these studies has been specifically related to $PM_{2.5}$ or PM_{10} as these are the most commonly adopted robust and widespread measures of particulate matter available in urban air environments.

A recent study of potential health effects associated with exposure to fine and ultrafine particulates in a heavy polluted city in China[14] (Meng et al. 2013), where data were specifically collected to characterise many bands of fine, very fine and ultrafine particulates (not normally measured in ambient air), identified that fine and very fine particulates (PM_1, but more specifically the sizes 0.25-0.50 µm) were significantly associated with total and cardiovascular mortality, but not respiratory mortality. Effect estimates increased with decreasing particle size. This suggests PM_1 may be associated with more significant health effects (particularly in relation to cardiovascular effects). A number of other studies have also identified that exposure to fine and ultrafine particulates (measured as PM_1 or $PM_{0.1}$) are associated with more significant effects than the coarse particulates (NEPC 2010). However, it was not clear whether observed effects were due to particle size alone or to chemical characteristics, in that the ultrafine particles would have a relatively larger surface area per unit mass for potential adsorption of other chemicals than would the larger size particulates.

In urban air environments, where most of the epidemiology studies have been undertaken, PM_1 comprises a significant proportion of $PM_{2.5}$. Measurements indicate that the ratio of PM_1:$PM_{2.5}$ is around 0.8-0.9 in Europe (Gomišček et al. 2004) (showing results similar to other European urban areas) with data from Australia (Keywood et al. 1999) suggesting a ratio of around 0.72. Data from Italy (Giugliano et al. 2005) suggests that within tunnels the fraction of $PM_{2.5}$ that is also PM_1 is slightly higher than in open air areas, but consistent with that reported in Europe. As the primary source of both PM_1 and $PM_{2.5}$ in urban air are combustion (traffic) emissions, the ratio of PM_1:$PM_{2.5}$ has been observed to be relatively stable throughout the year within urban air environments. For this project (where vehicle emissions are being assessed, the ratio of PM_1:$PM_{2.5}$ is expected to remain stable. Hence the use of exposure response relationships established for $PM_{2.5}$ from large epidemiology studies conducted in urban air environments (such as Europe and the US, as adopted in this assessment), these relationships will have also accounted for the presence of PM_1 and the health effects associated with exposure to these fine particulates.

A more detailed review of epidemiology and air monitoring data in Europe determined that monitoring PM_1 would not significantly add to the information content of data obtained on $PM_{2.5}$ (Gomišček et al. 2004).

[14] Authors of the paper note that the level of particulate pollution, and the likely composition, in cities in developing countries such as China differ from developed countries where many of the health effect relationships for exposure to particulate matter have been identified.

In relation to ultrafine particles (particles that are ≤100 nm, or ≤0.1 µm in diameter) the current science has been recently evaluated (HEI 2013), where the following is noted in relation to exposure and health effects:

- The key source of ultrafine particulates is vehicle emissions.
- Assessing exposure to ultrafine particulates is more challenging as the concentrations are much more variable (spatially) than measures of $PM_{2.5}$ and concentrations of ultrafine particulates are not routinely measured in urban areas.
- 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 $PM_{2.5}$.
- Epidemiology studies conducted in relation to exposure to ultrafine particulates have shown inconsistent (but suggestive) evidence of adverse effects associated with short-term exposure.
- The current body of evidence does not support strong and consistent conclusions of independent effects of ultrafine particulates on human health.

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 $PM_{2.5}$ comprises a significant proportion of PM_1) 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 $PM_{2.5}$.

4.4.3 Health effects

Health effects that have been associated with exposure to PM_{10} and $PM_{2.5}$ relate to exposure over both the short term (hours or days where effects may occur on the same day or after a day or two) and long term (months or years) and include (Anderson et al. 2004; NEPC 2010; OEHHA 2002; USEPA 2009b; WHO 2003, 2013b):

- Respiratory and cardiovascular morbidity, such as aggravation of asthma, respiratory symptoms and an increase in hospital admissions.
- Mortality from all causes, and specifically cardiovascular and respiratory diseases and from lung cancer.

There is good evidence of the effects of short-term exposure to PM_{10} on respiratory health, but for mortality and cardiovascular effects the evidence of effects for PM_{10} exposure is weaker. For these health effects $PM_{2.5}$ (particles in the 2.5–10 µm range) is a stronger risk factor (particles in the 2.5–10 µm range).

In short-term studies (based on 24-hour particulate levels), groups with pre-existing respiratory, lung or heart disease, as well as elderly people were more susceptible to the morbidity and mortality effects of ambient particulate matter exposure (Esworthy 2013; WHO 2013b). In longer term studies it has been suggested that the socially disadvantaged and poorly educated populations respond more strongly in terms of mortality (Esworthy 2013; WHO 2003, 2013b).

Based on the available studies, there is no evidence of a safe level of exposure or a threshold below which no adverse health effects occur (NEPC 2010; WHO 2013b).

Additional discussion on health effects associated with exposure to $PM_{2.5}$ and PM_{10} is presented in **Section 5.1**, including quantitative associations (exposure-response relationships) between exposure and the most significant health effects.

At present, at the population level, there is not enough evidence to identify differences in the effects of particles with different chemical compositions or emanating from various sources (NEPC 2010; WHO 2013b). The evidence for the hazardous nature of combustion-related particulate matter (from both mobile and stationary sources that dominate urban air where most of the epidemiological studies are conducted) is more consistent than that for particulate matter from other sources, and dominate the epidemiological studies used to develop relationships between exposure and adverse health effects. This is the relevant source of particulate matter for this project.

Particulates that are derived from specific sources, such as diesel emissions, are known to comprise other compounds such as volatile organic compounds and polycyclic aromatic hydrocarbons that are known to also be associated with adverse health effects. The presence of these other compounds has been addressed separately however the presence of these (and likely other compounds) compounds and other co-pollutants (also derived from combustion sources) adds to the complexity of utilising data form urban air epidemiological studies for assessing health effects from particulate matter.

Recently, outdoor air pollution has been classified by the International Agency for Research on Cancer (IARC 2013) as carcinogenic (Group 1) to humans based on sufficient evidence that exposure to outdoor air pollution causes lung cancer. Particulate matter, a major component of outdoor air pollution, was evaluated separately and also classified as carcinogenic to humans (Group 1).

In 2012, IARC evaluated exhaust from diesel engines (consisting mostly of particulate matter) and classified these emissions as carcinogenic (Group 1) to humans.

4.4.4 Initial assessment of potential health issues from exposure to particulate matter

For many of the key health effects associated with exposures to PM_{10} and $PM_{2.5}$ the exposure-response relationship is linear (where there is no threshold below which no adverse effects have been identified) (NEPC 2010). This means that any exposure to particulate matter has the potential to be associated with an effect. Guidelines have been established in Australia (and internationally) to determine a level at which cumulative exposure (ie exposure to particulates from all sources) are likely to minimise the potential for adverse impacts in a population. The available guidelines are discussed and further considered below.

However as there is no threshold for adverse effects it is also important that any incremental exposure to particulate matter derived from the project is also assessed. The more detailed evaluation of incremental impacts associated with the project is presented in **Section 5**.

Guidelines

Air quality goals for PM_{10}, and advisory goal for $PM_{2.5}$, have been established by NEPC (NEPC 2002, 2003) that are based on the protection of human health and well-being. The goals apply 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 addition, the assessment of impacts from any development requires consideration of air quality goals/guidelines that are outlined in the Environment Protection Authority's "Approved Methods for the Modelling and Assessment of Air Pollutants in NSW" (DEC 2005a). The guidelines are primarily derived from the NEPC, with the exception of an annual average PM_{10} guideline which is derived from older goals adopted by the Environment Protection Authority (EPA 1998). The air quality goals relate to total particulate matter burden in the air and not just the particulate matter from the project, hence use of these criteria requires consideration of background levels of particulate matter and other local sources. Similar to the NEPC criteria, these guidelines do not apply to localised "hot-spot" areas such as locations near industry, busy roads or mining. However, in the absence of alternative measures, Environment Protection Authority does apply these criteria to assess the potential for impacts to arise at such locations, particularly for new projects.

Table 4-8 presents a summary of the current NEPC and Environment Protection Authority's air quality goals and guidelines for particulate matter. These guidelines are for cumulative impacts and should also be considered in conjunction with incremental impact calculations presented in **Section 5**.

Table 4-8 Air quality goals for particulates

Pollutant	Averaging period	Criteria	Reference
PM_{10}	24-hour	50 µg/m^3 Maximum of 5 days exceedance per year	(DEC 2005a; NEPC 2003)
	Annual	30 µg/m^3	(DEC 2005a)
$PM_{2.5}$	24-hour	25 µg/m^3	Advisory goal [15] (NEPC 2003)
	Annual	8 µg/m^3	

[15] The $PM_{2.5}$ criteria established by the National Environment Protection Council are advisory goals. The goals have been derived on the basis of available health based information that relates exposure to $PM_{2.5}$ to adverse health effects. However, as $PM_{2.5}$ had not been routinely monitored in the community at the time when the criteria were being considered, existing urban (and regional) levels were not known, and the ability to meet the advisory goals could not be determined in individual states. Hence these criteria were not established as standards as defined in the National Environment Protection Council Act 1994. The relevance of any exceedance of these goals will be fully assessed once a sufficient database of monitoring data is available. They are, however, goals that are based on the protection of population health.

In relation to the current NEPC PM_{10} guideline, the following is noted (NEPC 1998, 2010):

- 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_{10} 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 imposition of a criterion of 50 $\mu g/m^3$ 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 acceptable number of exceedances per year is not based on an assessment of health, rather it is based on review of existing air quality in urban areas and identifying a number of exceedances that are consistent with these existing areas.
- The assessment undertaken considered exposures and issues relevant to urban air environments that are expected to also be managed through the PM_{10} guideline. These issues included emissions from vehicles and wood heaters.
- Review of the air goals in 2010 did not identify that there was a need to revise the PM_{10} guideline.

A similar approach has been adopted by NEPC (Burgers & Walsh 2002; NEPC 2002) in relation to the derivation of the $PM_{2.5}$ air quality goals, with specific studies related to $PM_{2.5}$ and mortality and morbidity indicators considered.

Table 4-9 presents a comparison of the NEPC guidelines with those established (following more recent reviews) by the WHO (WHO 2005a), the EU and the USEPA (2012). The goals 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.

The air quality guidelines 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 the AQIA for this project includes a number of days that have been affected by occasional dust storms and bushfires. These extreme events result in exceedance of the NEPM guidelines (particularly in 2009). Hence, review of the 24-hour average, and the annual average, cumulative concentration is complex as it involves evaluating the incremental impact of the project on a background data set that includes these events. Detailed review of the 24-hour average and annual average concentrations associated with the operation of the project are presented in the AQIA. The review concluded that emissions from the project do not predict any additional exceedances of the NEPM criteria.

Table 4-9 Comparison of particulate matter air quality goals

Pollutant	Averaging period	Criteria/Guidelines/Goals			
		NEPC and NSW OEH	WHO (2005)	EU #	USEPA (2012)
PM_{10}	24-hour	**50 µg/m^3** Maximum of 5 days exceedance per year	**50 µg/m^3**	**50 µg/m^3** as limit value with 35 exceedances permitted each year	**150 µg/m^3** (not to be exceeded more than once per year on average over 3 years)
	Annual	**30 µg/m^3**	**20* µg/m^3**	**40 µg/m^3** as limit value	NA
$PM_{2.5}$	24-hour	**25 µg/m^3** (goal)	**25 µg/m^3**	NA	**35 µg/m^3** (98th percentile, averaged over 3 years)
	Annual	**8 µg/m^3** (goal)	**10* µg/m^3**	**25 µg/m^3** as target value from 2010 and limit value from 2015. **20 µg/m^3** as a 3 year average (average exposure indicator) from 2015 with requirements for ongoing percentage reduction and target of **18 µg/m^3** as 3 year average by 2020	**12 µg/m^3** (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% confidence in response to $PM_{2.5}$ in the ACS study (Pope et al. 2002). The use of $PM_{2.5}$ guideline is preferred (WHO 2005a).

Incremental Impacts of particulate matter

As there is no safe level for particulate matter in ambient air, the incremental impact of $PM_{2.5}$ and PM_{10} emissions to air from the project have been evaluated in more detail, as presented in **Section 5**.

The predicted incremental concentrations of $PM_{2.5}$ and PM_{10} are very low with:

- the maximum 24-hour average $PM_{2.5}$ incremental impact = 1.3-2 µg/m^3
- the maximum 24-hour average PM_{10} incremental impact = 1.4-2.1 µg/m^3
- the maximum annual average $PM_{2.5}$ incremental impact = 0.11-0.13 µg/m^3
- the maximum annual average PM_{10} incremental impact = 0.11-0.13 µg/m^3

To provide some context to the level of $PM_{2.5}$ predicted from the project, the maximum predicted 24 hour average $PM_{2.5}$ concentration has been compared with published (measured) levels of $PM_{2.5}$ in air during a range of common daily activities. This comparison is illustrated in

Figure 4-1.

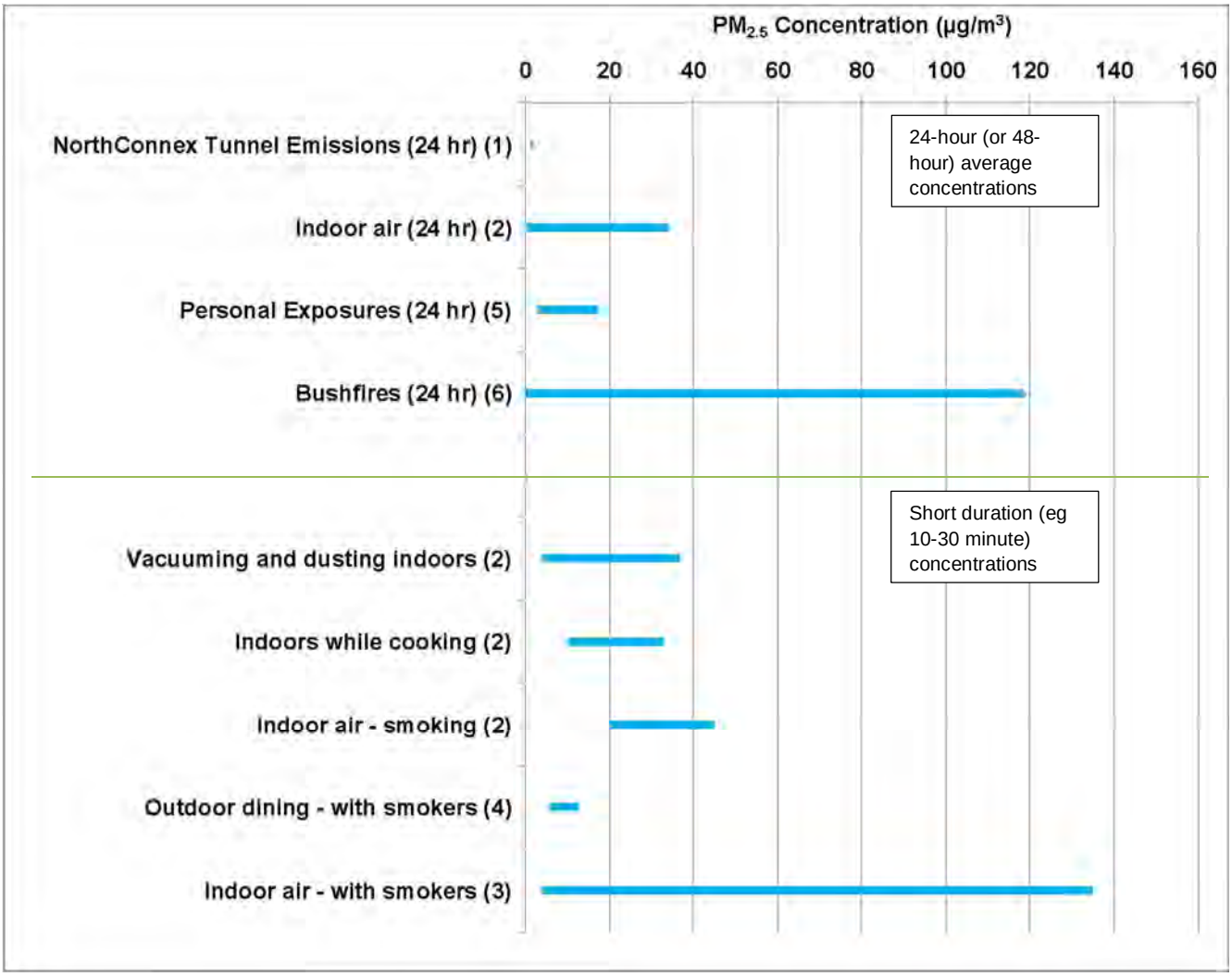

Figure 4-1 Comparison of incremental (above background) PM2.5 concentrations from range of events and activities

Notes for Figure 4-1:

1 – Maximum predicted incremental $PM_{2.5}$ impacts for project (from either northern or southern interchanges) for scenarios 2a or 2b.

2 – Data for range of indoor activities for homes in Brisbane (Morawska, Moore & Ristovski 2004). Range for 24 hour average concentrations is similar to but lower than reported in other studies in Australia (CAWCR 2010). The peak $PM_{2.5}$ concentrations in the kitchen during cooking have been reported to be significantly higher than present in the graph above, with levels up to 745 **µg/m^3** (He et al. 2004). The range reported for cooking activities in Australia are similar to the range reported in other countries (Abdullahi, Delgado-Saborit & Harrison 2013).

3 – Data for $PM_{2.5}$ levels in indoor venues in Western Australia (Stafford, Daube & Franklin 2010).

4 – Data for $PM_{2.5}$ in 69 outdoor dining areas in Melbourne (Cameron et al. 2010).

5 – Personal exposures throughout a day that include cooking, cleaning, burning of candles and other activities undertaken throughout the day (increment presented is the 25th to 75th percentile above the median background) (Sorensen et al. 2005).

6 – Data for 24 hour measurements of $PM_{2.5}$ that include bushfire events in Sydney (Burgers & Walsh 2002). Significantly higher peak concentrations of $PM_{2.5}$ (>500 µg/m^3) are often reported when bushfires are present (CSIRO 2008).

Section 5. Detailed assessment of exposure to particulate matter

5.1 Summary of adverse 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, Moore & Ristovski 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 shown with $PM_{2.5}$ (USEPA 2012).

There are a number of other 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 human 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.

The detailed assessment of potential health effects associated with exposure to emissions associated with the project has focused on health effects and exposure-response relationships[16] that are robust and relate to $PM_{2.5}$, being the more important particulate fraction size relevant for emissions from combustion sources. These health effects (or endpoints) have been identified and agreed with NSW Health and include the following:

- Primary health endpoints:
  - Long-term exposure to $PM_{2.5}$ on all-cause mortality (≥ 30 years of age).
  - Short-term exposure on the rate of hospitalisation with cardiovascular and respiratory disease (≥ 65 years of age).
- Secondary health endpoints (to supplement the primary assessment):
  - Long-term exposure to $PM_{2.5}$ on cardiopulmonary mortality (≥ 30 years of age).
  - Short-term exposure to $PM_{2.5}$ on mortality (all causes, cardiovascular and respiratory, all ages).
  - Short-term exposure to PM_{10} on mortality (all causes and all ages).

5.2 Exposure-response relationships

5.2.1 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 (refer to **Section 5.1**). 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

[16] An exposure-response relationship is a quantitative relationship between an exposure concentration of particulate matter in air (what is inhaled) and the health effect evaluated.

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 particulate matter, no threshold has been identified. Non-threshold exposure-response relationships have been identified for the primary and secondary health endpoints considered in this assessment.

A range of exposure-response relationships are available from the many studies that have been undertaken and published. Review of the available studies has been undertaken in Australia for the purpose of developing the NEPC Air Quality Guidelines (Burgers & Walsh 2002; NEPC 2002, 2010), where a range of health endpoints and exposure-response relationships were identified and evaluated. Similar exposure-response relationships have been considered in the development and review of air guidelines established by the WHO (WHO 2005a) and the USEPA (USEPA 2012). These organisations have identified which of the available relationships that have been identified are the most robust.

The exposure-response relationships adopted in this assessment have been identified on the basis of the studies considered in the development of the NEPC Air Quality Guidelines as well as updated supporting studies published in the literature.

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[17]. 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):

$$RR = exp[\beta(X-X_0)] \quad \text{…Equation 1}$$

Where:

$X-X_0$ = the change in particulate matter concentration to which the population is exposed ($\mu g/m^3$)

β = 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 $\mu g/m^3$ increase in particulate matter exposure.

[17] 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 $PM_{2.5}$ identified that the log-linear relationship calculated slightly higher relative risks compared with the linear relationship within the range 10-30 $\mu g/m^3$,(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 $PM_{2.5}$ that are well below 10 $\mu g/m^3$ and hence use of the linear relationship is expected to provide a more conservative estimate of relative risk.

Based on this equation, where the published studies have derived relative risk values that are associated with a 10 µg/m^3 increase in particulate matter exposure (as presented in **Table 5-**1), the β coefficient can be calculated using the following equation:

$$\beta = \frac{\ln(RR)}{10}$$ … Equation 2

Where:

RR = relative risk for the relevant health endpoint as published and listed in **Table 5-1** (µg/m^3)

10 = increase in particulate matter concentration associated with the RR (all the RR presented in **Table 5-1** are associated with a 10 µg/m^3 increase in particulate matter exposure).

Table 5-1 presents a summary of the health endpoints considered in this assessment, the relevant health impact functions (from the referenced published studies) and the associated β value relevant to the calculation of a relative risk.

The health impact functions presented in this table have been discussed and agreed with NSW Health as the most current and appropriate for the quantification of potential health effects for the health endpoints considered in this assessment.

Table 5-1 Adopted health impact functions and exposure-responses relationships

Health endpoint	Exposure period	Age group	Published relative risk [95% confidence interval] per 10 µg/m^3	Adopted β coefficient (as %) for 1 µg/m^3 increase in PM	Reference
Primary assessment health endpoints					
PM2.5: 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).
PM2.5: 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)
PM2.5: 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 assessment health endpoints					
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)
PM2.5: 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)
PM2.5: Cardiopulmonary 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).
PM2.5: 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)
PM2.5: 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)

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

5.2.2 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.

Data from the USEPA (USEPA 2002) 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 (100 per cent of the HGVs and 49.9 per cent of the LDVs). 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 light-headedness, 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 2002) 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 will also be met if the $PM_{2.5}$ guideline was met. Review of exposure to $PM_{2.5}$ has been assessed separately in relation to the current ambient air guidelines (refer to **Section 4.4.4**) where cumulative impacts of $PM_{2.5}$ for the project have been found to comply with the NEPC $PM_{2.5}$ advisory goal. Hence non-carcinogenic effects associated with exposure to diesel particulate matter are not considered to be of concern.

Review of exposures to diesel particulate matter (USEPA 2002) 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 of lung cancer.

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 2002) 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 1.6×10^{-5} to 7.1×10^{-5} per µg/m^3 (mean value of 3.4×10^{-5} per µg/m^3). This would suggest that an increase in lifetime exposure to diesel particulate matter between 0.14 and 0.625 µg/m^3 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 3.0×10^{-4} per µg/m^3 diesel particulate matter (OEHHA 1998). This was derived from data on exposed workers and based on evidence that suggested unit risks between 1.5×10^{-4} and 15×10^{-4} per µg/m^3. This would suggest that an increase in lifetime exposure to diesel particulate matter of 0.033 µg/m^3 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 3.4×10^{-5} per µg/m^3) 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 the AQIA; rather diesel particulate matter is part of the $PM_{2.5}$ assessment. For the purpose of this assessment it has been conservatively assumed that 100 per cent of the incremental $PM_{2.5}$ (from the project only) is derived from diesel sources. This is conservative as not all the vehicles using the tunnel (and emitting $PM_{2.5}$) 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).

5.3 Particulate impact assessment

5.3.1 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)[18] where the exposure-response relationships (presented in **Section 5.2**) have been directly considered on the basis of the approach outlined below.

The calculation of changes in health endpoints associated with exposure to 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 (as provided by the AQIA);
- Estimates of the number of people exposed to particulate matter at a given location (ie population data, refer to **Section 3.3**);
- Baseline incidence of the key health endpoints that are relevant to the population exposed (refer to **Section 3.4**); and
- Exposure-response relationships expressed as a percentage change in health endpoint per µg/m^3 change in particulate matter exposure (refer to **Section 5.2**), 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:

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 particulate matter considered as per Equation 1) as:

$$AF = \frac{RR-1}{RR} \quad \text{… Equation 3}$$

[18] 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 µg/m^3 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).