

Environment
Effects Statement

Technical Report J

Human health



North East Link Project North East Link Environment Effects Statement

Technical report J – Health impact assessment

Prepared for North East Link

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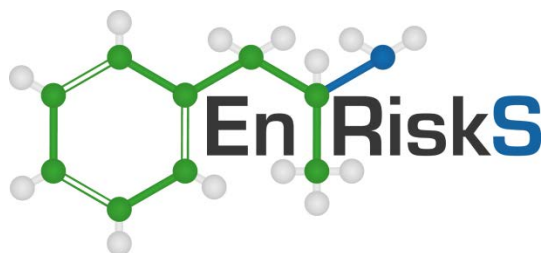


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Executive summary

This technical report is an attachment to the North East Link Environment Effects Statement (EES). It has been used to inform the EES required for the project, and defines the Environmental Performance Requirements (EPRs) necessary to meet the EES objectives.

Overview

North East Link ('the project') is a proposed new freeway standard road connection that would complete the missing link in Melbourne's ring road, giving the city a fully completed orbital connection for the first time. North East Link would connect the M80 Ring Road (otherwise known as the Metropolitan Ring Road) to the Eastern Freeway, and include works along the Eastern Freeway from near Hoddle Street to Springvale Road.

The Major Transport Infrastructure Authority (MTIA) is the proponent for North East Link. The MTIA is an administrative office within the Victorian Department of Transport with responsibility for overseeing major transport projects.

North East Link Project (NELP) is an organisation within MTIA that is responsible for developing and delivering North East Link. NELP is responsible for developing the reference project and coordinating development of the technical reports, engaging and informing stakeholders and the wider community, obtaining key planning and environmental approvals and coordinating procurement for construction and operation.

On 2 February 2018, the Minister for Planning declared North East Link to be 'public works' under Section 3(1) of the *Environment Effects Act 1978*, which was published in the *Victorian Government Gazette* on 6 February 2018 (No. S 38 Tuesday 6 February 2018). This declaration triggered the requirement for the preparation of an EES to inform the Minister's assessment of the project and the subsequent determinations of other decision-makers.

The EES was developed in consultation with the community and stakeholders and in parallel with the reference project development. The reference project has been assessed in this EES. The EES allows stakeholders to understand the likely environmental impacts of North East Link and how they are proposed to be managed.

Environmental Risk Sciences Pty Ltd (enRiskS) was commissioned to undertake a health impact assessment for the purpose of the EES.

Health context

The scoping requirements for the EES issued by the Minister for Planning set out the specific environmental matters to be investigated and documented in the project's EES, which informs that scope of the EES technical studies. The scoping requirements include a set of evaluation objectives. These objectives identify the desired outcomes to be achieved in managing the potential impacts of constructing and operating the project.



The evaluation objective relevant to human health is as follows:

- **Health, amenity and environmental quality** – To minimise adverse air quality, noise and vibration effects on the health and amenity of nearby residents, local communities and road users during both construction and operation of the project.

The evaluation objective for social elements is also relevant to human health, with relevant sections identified as:

- **Social, business, land use, public safety and infrastructure** – To manage effects of the project on land use and the social fabric of the community with regard to wellbeing, community cohesion, business functionality and access to goods, services and facilities.

The overarching objective of the health impact assessment is to evaluate how the project may benefit or impact upon the health and wellbeing of the local community, and to facilitate more health-conscious planning and development. More specifically, the assessment has addressed:

- Changes in air quality within the local community associated with emissions from the tunnel ventilation structures
- Changes in air quality associated with changes in emissions from major surface roads
- Exposure to vehicle occupants to emissions present within the tunnels, during operation
- Changes in noise and vibration within the community
- Social changes related to the project.

Based on the assessment undertaken and presented in this report the following has been concluded:

Health impacts associated with changes in air quality

The assessment of health impacts from changes in air quality associated with the project has identified:

- **Construction:** During construction, there is the potential for some impacts to occur but these impacts can be mitigated/minimised with implementation of EPRs AQ1, CL1 and EMF2. Where fully implemented, this should not result in significant or measurable impacts on community health, although the residual risk is considered to be low.
- **Operations:** During the operation of the project the assessment of health impacts has concluded:
 - Volatile organic compounds (VOCs) evaluated using health-based guidelines – potential exposures to VOCs are below all relevant health-based guidelines and so health impacts are considered negligible.
 - Carcinogenic VOCs, polycyclic aromatic hydrocarbons (PAHs) and diesel particulate matter (DPM) – potential exposures to carcinogenic VOCs, PAHs and DPM result in incremental carcinogenic risks that are considered to be low and acceptable.



- Carbon monoxide – potential exposures to carbon monoxide are below all relevant health-based guidelines and are considered negligible.
- Nitrogen dioxide:
 - Cumulative impacts associated with nitrogen dioxide emissions from the project are not considered to be of concern in relation to community health.
 - Population health impacts from tunnel ventilation structures and the redistribution of traffic on surface roads are considered to be low and acceptable.
 - Localised maximum increases in health risk from emissions to air from the tunnel ventilation structures and the redistribution of traffic on surface roads are not considered to be elevated.
- Particulates:
 - Population health impacts from tunnel ventilation structures and the redistribution of traffic on surface roads are considered to be low and acceptable.
 - Localised maximum increases in health risk from emissions to air from the tunnel ventilation structures and the redistribution of traffic on surface roads are not considered to be elevated.
- **In-tunnel air quality:** In-tunnel air guidelines for carbon monoxide and nitrogen dioxide would be adequately protective of the health of users of North East Link. Short-duration exposures to higher levels of particulates should be minimised through providing advice to motorists to keep windows closed and switch ventilation to recirculation as is currently being done in New South Wales.

Emissions to air during operations would be managed through EPRs AQ3 to AQ5.

Health impacts associated with changes in noise and vibration

The assessment undertaken in relation to health impacts that may occur due to changes in noise and vibration resulting from the project has concluded:

- **Construction:** Noise and vibration impacts identified during construction works are to be managed through the implementation EPRs NV3 to NV6, NV8 to NV12 which includes a Construction Noise and Vibration Management Plan (CNVMP). Specific mitigation and management measures required to manage noise and vibration impacts in the community during construction are presented in EES Technical report C – Surface noise and vibration. Where these measures are implemented the potential for noise impacts to result in significant health impacts in the community is low.

However, it is expected that some individuals within the community may find construction noise annoying at times, even with mitigation. The management of noise impacts during construction needs to include a notification and complaints system, as outlined in EPR SC2, where these issues can be identified and addressed.

- **Operations:** There are some areas where an increase in noise has been predicted. Additional noise mitigation has been identified for some areas to minimise the impact of changes (particularly increases) in project-related noise. Where these additional noise mitigation



measures are implemented, changes in noise levels associated with the project are not expected to result in health impacts within the community that would be measurable.

Noise impacts during operation of the project would be managed with implementation of EPRs NV1 and NV13, with monitoring undertaken in accordance with EPRs NV2 and NV7.

Health impacts associated with changes in social aspects

The assessment undertaken in relation to health impacts that may occur due to changes in a range of social aspects/areas due to the project has concluded:

- **Construction:** Construction works have the potential to impact community health, principally through increases in stress and anxiety related to key activities and changes associated with the project. In particular, these impacts may occur because of changes in traffic movements on local roads, increased traffic delays in some areas and increased safety risks on roadways associated with construction. In addition, construction activities would result in temporary changes to some pedestrian and cyclist access. These impacts could be managed with implementation of EPRs T2 to T5.

The project would involve the acquisition of a number of residential and business properties. The acquisition of any property can increase stress and anxiety and so these impacts would need to be managed so they do not adversely affect these residents, businesses or existing jobs.

During construction, some existing open space areas would be temporarily occupied (potentially for the duration of construction) and unavailable for community use. Some existing open space (including some sporting facilities) would need to be permanently acquired for the project. While the presence of construction sites and activities would affect visual amenity, cause some inconvenience and a temporary or permanent loss of access to some of the existing open space areas and recreational/sporting facilities, discussions and arrangements with relevant sporting clubs would identify alternative facilities where practical. In relation to open space and passive recreational use of these areas, alternative green space areas are within walking distance. Changes in green space and access to sporting and recreational facilities during construction would be managed with implementation of EPRs SC1 to SC4 and AR1 to AR3.

Changes in visual amenity may affect the community use of some recreational areas. These changes are temporary and there are other recreational areas available in the vicinity of the project. The project would include a range of re-vegetation and landscaping activities at the completion of construction, which would restore many of the open space areas impacted during construction.

As a result, the potential for significant impacts on the health of the local community during construction is considered low.

- **Operation:** Operation of the project provides opportunities for some health benefits associated with faster and less variable travel times, economic and employment benefits, improvements to pedestrian and cyclist access and safety, improvements to public transport and the remediation of contamination in some areas.

Structure of the EES

Summary Report

EES main report

- | | | |
|----------------------------------|----------------------------------|--|
| 1. Introduction | 11. Surface noise and vibration | 21. Ground movement |
| 2. Project rationale | 12. Tunnel vibration | 22. Groundwater |
| 3. Legislative framework | 13. Land use planning | 23. Contamination and soil |
| 4. EES assessment framework | 14. Business | 24. Surface water |
| 5. Communications and engagement | 15. Arboriculture | 25. Ecology |
| 6. Project development | 16. Landscape and visual | 26. Greenhouse gas |
| 7. Urban design | 17. Social | 27. Environmental management framework |
| 8. Project description | 18. Human health | 28. Conclusion |
| 9. Traffic and transport | 19. Historical heritage | |
| 10. Air quality | 20. Aboriginal cultural heritage | |

Technical reports

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|--------------------------------|---------------------------------|---------------------------|
| A. Traffic and transport | G. Arboriculture | M. Ground movement |
| B. Air quality | H. Landscape and visual | N. Groundwater |
| C. Surface noise and vibration | I. Social | O. Contamination and soil |
| D. Tunnel vibration | J. Human health | P. Surface water |
| E. Land use planning | K. Historical heritage | Q. Ecology |
| F. Business | L. Aboriginal cultural heritage | R. Greenhouse gas |

Attachments

- | | | |
|----------------------------|-------------------------------------|--------------------------------|
| I. Sustainability approach | IV. Stakeholder consultation report | VI. Works Approval Application |
| II. Urban design strategy | V. Draft Planning Scheme Amendment | |
| III. Risk report | | |

EES Map Book

Glossary of terms and abbreviations

Term	Meaning
A weighted decibels (dB(A))	The A weighting is a frequency filter applied to measured noise levels to represent how the human ear hears sounds. Adjustments are applied between 10Hz and 20 kHz. When an overall sound level is A-weighted it is expressed in units of dB(A) or dBA.
Acute or short-term exposure	Contact with a substance that occurs only once or for a short period of time, typically an hour or less, but may be 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.
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.
Carcinogen	A substance that causes cancer.
Chronic or long-term exposure	Contact with a substance that occurs repeatedly over a long time, with the United States Environmental Protection Agency (USEPA) indicating defining this as exposures that occur for more than approximately 10% of a lifetime, Exposures that occur for less than 10 per cent of a lifespan are considered sub-chronic.
Co-exposure	Exposure to more than one pollutant or stressor (such as noise) by a population
Combined	In the context of the health impact assessment, combined refers to the sum of exposures from different project impacts: such as impacts on health from emissions to air from the tunnel ventilation structures plus impacts on health from changes in air impacts from surface roads; or impacts on health from changes in air quality plus impacts on health from changes in noise.
Cumulative	Total exposure, used in the health impact assessment to refer to exposures that include the background plus project, or to multiple different sources from the project
Decibel (dB)	A logarithmic scale is used to describe the level of sound, referenced to a standard level. It is widely accepted that a 3dB change in traffic noise levels (of the same character) is barely, if at all detectable; whereas a change of 5 dB is clearly noticeable. A 10 dB increase is typically considered to sound twice as loud (noting a change of -10 dB would typically sound half as loud).
Department of Transport	The Victorian Department of Transport is responsible for delivering the government's transport infrastructure agenda. It was formed on 1 January 2019 when the former Victorian Department of Economic Development, Jobs, Transport and Resources transitioned into the Department of Transport and the Department of Jobs, Precincts and Regions.
Detection limit	The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.
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 milligrams (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 gets into the body through the eyes, skin, stomach, intestines, or lungs.
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].

Term	Meaning
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	A guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the New South Wales 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)). The guideline value 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, or the environment. Dependent on the source, guidelines have different names, such as investigation level, trigger value, ambient guideline etc.
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].
L ₁₀	The sound pressure level exceeded for 10% of the measurement period. The A-weighted form is denoted 'L _{A10} '.
L _{A10(18h)}	The L _{A10(18-hour)} noise level refers to the noise level exceeded for 10 per cent of the time during an 18-hour period (from 6am to midnight). This noise descriptor is calculated using the arithmetic average of the L _{A10} noise levels for each hour from 6am to midnight.
L _{den}	The average noise level over the day, evening and night (a 24-hour period).
L _{eq}	Equivalent continuous sound level. The constant sound level which, when occurring over the same period of time, would result in the receptor experiencing the same amount of sound energy. The A-weighted form is denoted 'L _{Aeq} '.
L _{night}	The average noise level over the night-time period, typically between 11pm or midnight and 6am.
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.
Major Transport Infrastructure Authority	The Major Transport Infrastructure Authority (MTIA) is the proponent for North East Link. The MTIA is an administrative office within the Victorian Department of Transport with responsibility for overseeing major transport projects.
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
Morbidity	A diseased condition or state or the incidence or prevalence of disease in a population
Mortality	Death, which may occur as a result of a range of reasons or diseases
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.
North East Link Project	North East Link Project is an organisation within MTIA that is responsible for developing and delivering North East Link. NELP was formerly known as the North East Link Authority prior to 1 January 2019. NELP is responsible for developing the reference project and coordinating development of the technical reports, engaging and informing stakeholders and the wider community, obtaining key planning and environmental approvals and coordinating procurement for construction and operation.

Term	Meaning
Not measurable	The term 'no measurable' or 'not measurable' is used in this health impact assessment when referring to changes in air quality, noise or health outcomes in a population. For air quality and noise, a change that would be not be measurable is one where the estimated change in the concentration of the pollutant in ambient air, or noise, is so small that it could not be measured (within the error of the analytical method/measurement equipment). For health outcomes, it refers to exposures that are below a threshold so there are no health effects, or to changes in the number of people that may be affected (an increase or decrease in deaths or hospitalisations) that is within the error/variability of the statistical measures (is not measurable).
Point of exposure	The place where someone comes 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 would cause injury or harm.
Route of exposure	The way people come into contact with a hazardous substance. The 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 is 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.
Uncertainty factor	Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure would cause harm to people (also sometimes called a safety factor).

Abbreviation	Term
AAQ	Ambient air quality
AQ	Air quality
ANZECC	Australia and New Zealand Environment and Conservation Council
ATSDR	Agency for Toxic Substances and Disease Register
BTEX	Benzene, toluene, ethylbenzene and total xylenes
CALD	Cultural and linguistic diversity
CEMP	Construction Environment Management Plan
CNVMP	Construction Noise and Vibration Management Plan
CO	Carbon monoxide
COPD	Chronic Obstructive Pulmonary Disease
DECCW	Department of Environment, Climate Change and Water
DHHS	Department of Health and Human Services
DPM	Diesel particulate matter
EC	European Commission
EES	Environment Effects Statement
EPA Victoria	Environment Protection Authority Victoria
EPR	Environmental Performance Requirement
HIA	Health Impact Assessment
HHRA	Human Health Risk Assessment
IARC	International Agency for Research on Cancer
LGA	Local Government Area
LOR	Limit of Reporting
NELP	North East Link Project
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NO ₂	Nitrogen dioxide
NPI	National Pollutant Inventory
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA)
PAH	Polycyclic aromatic hydrocarbon
PIARC	Permanent International Association of Road Congresses
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
TBM	Tunnel boring machine
TCEQ	Texas Commission on Environmental Quality



Abbreviation	Term
TRG	Technical Reference Group
TRV	Toxicity reference value
TSP	Total suspended particulate
USEPA	United States Environmental Protection Agency
VOC	Volatile Organic Compound
WHO	World Health Organization



1. Introduction

1.1 Purpose of this report

North East Link ('the project') is a proposed new freeway-standard road connection that would complete the missing link in Melbourne's ring road, giving the city a fully completed orbital connection for the first time. North East Link would connect the M80 Ring Road (otherwise known as the Metropolitan Ring Road) to the Eastern Freeway, and include works along the Eastern Freeway from near Hoddle Street to Springvale Road.

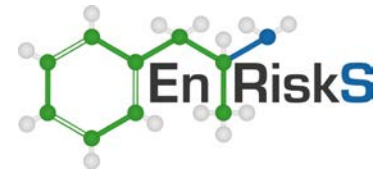
The Major Transport Infrastructure Authority (MTIA) is the proponent for North East Link. The MTIA is an administrative office within the Victorian Department of Transport with responsibility for overseeing major transport projects.

North East Link Project (NELP) is an organisation within MTIA that is responsible for developing and delivering North East Link. NELP is responsible for developing the reference project and coordinating development of the technical reports, engaging and informing stakeholders and the wider community, obtaining key planning and environmental approvals and coordinating procurement for construction and operation.

On 2 February 2018, the Minister declared the works proposed for North East Link as Public Works and issued a decision confirming that an Environment Effects Statement (EES) is required for the project due to the potential for significant environmental effects.

Similarly, the project was referred to the Australian Government's Department of the Environment and Energy on 17 January 2018. On 13 April 2018 the project was declared a 'controlled action', requiring assessment and approval under the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act). Separate to this EES, a Public Environment Report (PER) is required to be prepared to satisfy the EPBC Act requirements, and assess the impacts of the project on Commonwealth land and matters of national environmental significance (MNES).

Environmental Risk Sciences Pty Ltd (enRiskS) was commissioned to undertake a health impact assessment to inform the EES. In keeping with best practice, and as per the direction of the *Public Health and Wellbeing Act 2008*, the assessment has been integrated within the EES process to further improve transparency, consider local circumstance and relative sensitivity, and to facilitate more health-conscious project planning and development.



1.2 Why understanding health impacts are important

The assessment of health impacts associated with the proposed project is an important aspect of the EES.

North East Link is set in a highly urbanised area that includes long-established and diverse neighbourhoods and communities, shopping and commercial centres, industrial areas, parks and reserves, and community and recreational facilities. The project extends from Greensborough through to Bulleen, with Eastern Freeway upgrade extending the project to Nunawading and Donvale.

The operation of the project has the potential to result in changes to a range of issues considered to be of importance to community health and wellbeing. The health impact assessment seeks to understand the positive and negative changes associated with the project, and identify ways to enhance benefits and minimise negative impacts.

2. EES scoping requirements

2.1 EES evaluation objectives

The scoping requirements for the EES, released by the Minister for Planning, set out the specific environmental matters to be investigated and documented in the project's EES, which informs the scope of the EES technical studies. The scoping requirements include a set of evaluation objectives. These objectives identify the desired outcomes to be achieved in managing the potential impacts of constructing and operating the project.

The evaluation objective relevant to human health is:

- **Health, amenity and environmental quality** – To minimise adverse air quality, noise and vibration effects on the health and amenity of nearby residents, local communities and road users during both construction and operation of the project.

The evaluation objective for social elements is also relevant to human health, with relevant sections identified as:

- **Social, business, land use, public safety and infrastructure** – To manage effects of the project on land use and the social fabric of the community with regard to wellbeing, community cohesion, business functionality and access to goods, services and facilities.

2.2 EES scoping requirements

The aspects from the EES scoping requirements relevant to health impact assessment evaluation objectives are shown in **Table 2.1** as well as the location where these items have been addressed in this report

Table 2.1: Scoping requirements relevant to health impact assessment

Aspect	Scoping requirement	Section addressed
Key issues	Potential for impact on health of sensitive receptors due to exposure to vehicle emissions (noise and air)	Section 8 – Air quality Section 9 – Noise and vibration
Priorities for characterising the existing environment	Identify residences (including sites that are the subject of current planning permit applications or planning scheme amendments), urban developments (where development proposals are identified in the planning scheme or form part of a seriously entertained planning proposal) and land uses (schools, hospitals, outdoor recreation sites, etc) that require a particular focus on protecting the beneficial uses of the air and noise environment relating to human health and wellbeing, local amenity and aesthetic enjoyment	Section 8 – Air quality Section 9 – Noise and vibration
	Characterise the existing health status of the population in the vicinity of the project	Section 6 – Community profile

Aspect	Scoping requirement	Section addressed
Assessment of likely effects	Analyse risk of project emissions exceeding the relevant SEPP standards for surface roads and Schedule A Design Criteria for the tunnel ventilation system describing sources in isolation and in addition to background levels of air pollution and assessing their cumulative impact on air quality and human health	Section 8 – Air quality Technical report B –Air quality
	Evaluate any changes to air quality and noise conditions for nearby residents and local communities that the project would deliver, particularly through redistribution or management of heavy vehicle traffic or altered road and traffic conditions and the implications of these for human health and amenity	Section 8 – Air quality Section 9 – Noise and vibration
Approach to manage performance	Describe the environmental performance requirements to set air quality, traffic noise and vibration outcomes that the project must achieve.	Section 8 – Air quality Section 9 – Noise and vibration

2.3 Linkages to other reports

This report relies on or informs the technical assessments as indicated in **Table 2.2**. The health impact assessment has drawn on information provided in these reports and, in some areas, provides a summary of key (and relevant) aspects. All details relevant to the underlying assumptions, methodology and interpretation of impacts relevant to these specialist areas are presented in the individual reports. Where more detail than provided in the health impact assessment is required, the reader is directed to the relevant EES technical report.

Table 2.2: Linkages to other technical reports

Specialist report	Relevance to health impact assessment
Technical report A – Traffic and transport	Provides an assessment of the project's effects on the transport network within the report study area. Information related to changes in traffic volumes, routes, travel times, road safety and pedestrian and cyclist safety.
Technical report B – Air quality	Provides an assessment of the project's effects on local air quality within the report study area. Findings from the report have informed the assessment of health impacts from changes in air quality.
Technical report C – Surface noise and vibration	Provides an assessment of the project's potential noise and vibration impacts on sensitive receptors within the report study area. Findings from the report have informed the assessment of health impacts from changes in noise and vibration (from surface works).
Technical report D – Tunnel vibration	Provides an assessment of the project's potential vibration impacts specifically related to tunnelling activities during construction only. Findings from the report have informed the impacts on the community from tunnel vibration.
Technical report F – Business	Provides an assessment of the project's impact on businesses Findings from the report have informed the assessment of health issues related to impacts on businesses in the project area.
Technical report G – Arboriculture	Provides an assessment of the project's impact on the landscape. Findings have informed the assessment of impacts on green space, in particular the loss of tree and canopy cover.

Specialist report	Relevance to health impact assessment
Technical report H – Landscape and visual	Provides an assessment of the visual impact of the project's design sensitive receptors within the report study area. Findings from the report have informed the impact assessment on amenity and character, and community infrastructure facilities.
Technical report I – Social	Provides an assessment of the project's impact on social cohesion and amenity. Findings have informed the assessment of impacts on amenity and social changes within the community.
Technical report O – Contamination and soil	Provides an assessment of contamination and soil issues relevant to the construction of the project. Findings have informed the assessment of potential impacts of contamination on the community during construction.

2.4 Study objectives

The overarching objective of the health impact assessment is to evaluate how the project may benefit or impact the health and wellbeing of the local community, and to facilitate more health-conscious planning and development.

Key to this is understanding changes in traffic, air quality, noise and a range of social aspects that may be affected, either positively or negatively, by the project and their distribution within the community.

The methodology and scope of the health impact assessment are further described in **Section 5.2**.

The health impact assessment is attached as a technical report to the EES, with the key elements summarised in Chapter 18 of the EES.

3. Project description

3.1 Overview

The North East Link alignment and its key elements assessed in the Environment Effects Statement (EES) include:

- **M80 Ring Road to the northern portal** – from the M80 Ring Road at Plenty Road, and the Greensborough Bypass at Plenty River Drive, North East Link would extend to the northern portal near Blamey Road utilising a mixture of above, below and at surface road sections. This would include new road interchanges at the M80 Ring Road and Grimshaw Street.
- **Northern portal to southern portal** – from the northern portal the road would transition into twin tunnels that would connect to Lower Plenty Road via a new interchange, before travelling under residential areas, Banyule Flats and the Yarra River to a new interchange at Manningham Road. The tunnels would then continue to the southern portal located south of the Veneto Club.
- **Eastern Freeway** – from around Hoddle Street in the west through to Springvale Road in the east, modifications to the Eastern Freeway would include widening to accommodate future traffic volumes and new dedicated bus lanes for the Doncaster Busway. There would also be a new interchange at Bulleen Road to connect North East Link to the Eastern Freeway.

These areas are illustrated in **Figure 3.1**.

The project would also include improvements to existing bus services from Doncaster Road to Hoddle Street through the Doncaster Busway as well as pedestrian connections and the bicycle network with connected walking and walking paths from the M80 Ring Road to the Eastern Freeway.

For a detailed description of the project, refer to EES Chapter 8 – Project description.

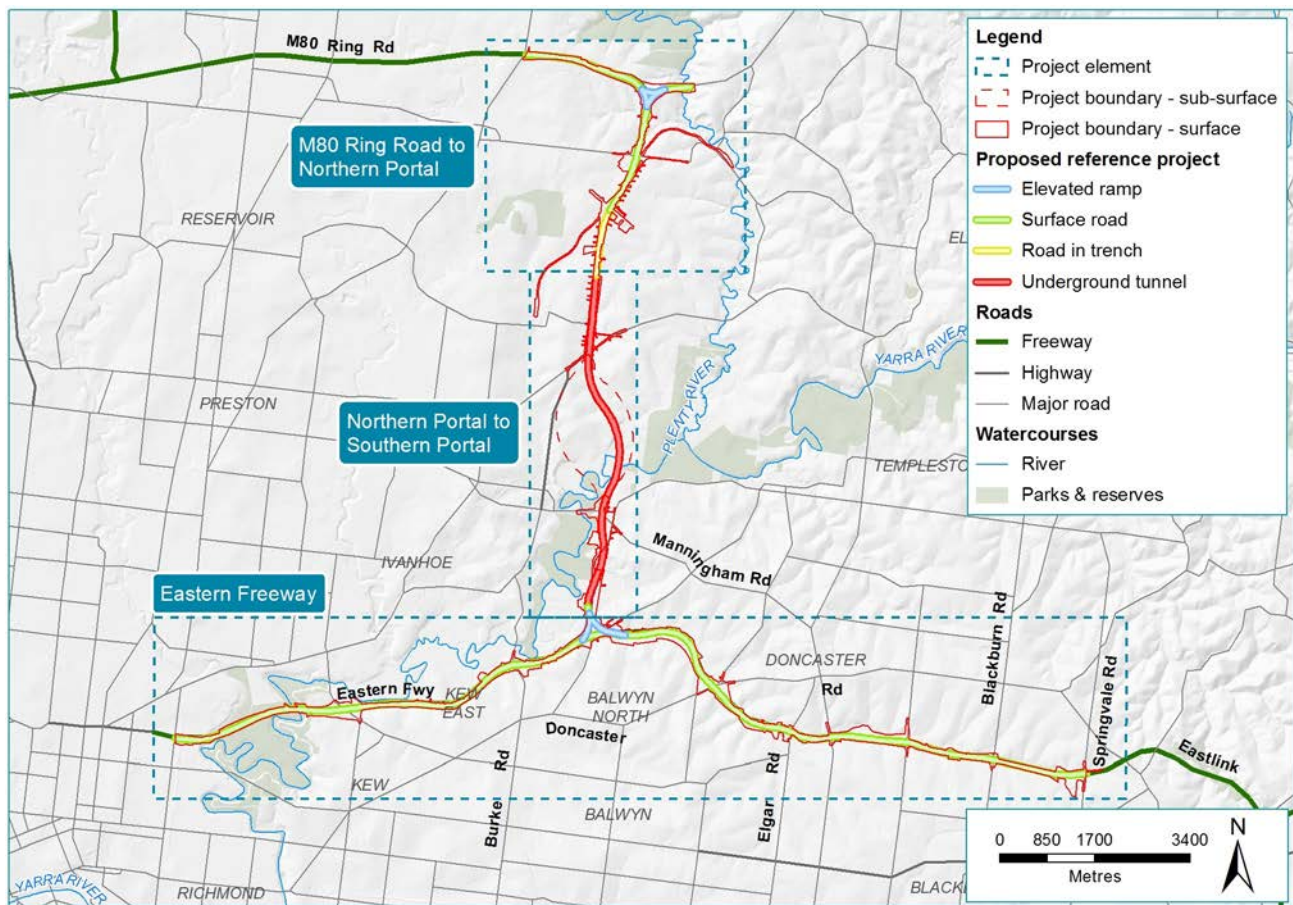
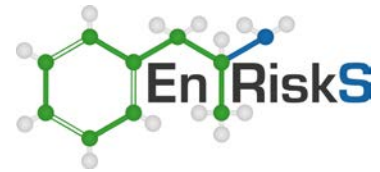


Figure 3.1: Overview of North East Link

3.2 Construction

Key construction activities for North East Link would include:

- General earthworks including topsoil removal, clearing and grubbing vegetation
- Relocation, adjustment or installation of new utility services
- Construction of retaining walls and diaphragm walls including piling
- Ground treatment to stabilise soils
- Tunnel portal and drive shaft construction
- Storage and removal of spoil
- Construction of cross passages, ventilation structures and access shafts
- Installation of drainage and water quality treatment facilities



- Installation of a Freeway Management System
- Tunnel construction using tunnel boring machines (TBMs), mining and cut and cover techniques
- Installation of noise walls
 - Restoration of surface areas.

3.3 Operation

Following construction of North East Link, the key operation phase activities would include:

- Operation and maintenance of new road infrastructure
- Operation and maintenance of Freeway Management System
- Operation of North East Link motorway control centre
- Operation and maintenance of the tunnel ventilation system
- Operation and maintenance of water treatment facilities
- Operation and maintenance of the motorways power supply (substations)
- Maintenance of landscaping and Water Sensitive Urban Design (WSUD) features.

4. Legislation and policy

4.1 Legislation, policy and guidelines

Numerous legislative, policy and guidance documents were found to be relevant to this health impact assessment and are discussed further in this report. Further details on these is provided in Sections 4.1 to 4.5.

The applicable legislative and policy context represents a significant influence on the transport and land use planning elements of the project. In relation to the conduct of a health impact assessment, the following legislation, policies and guidelines are relevant and are required to be considered. The national guidance documents were used as the basis of the design of this health impact assessment. Discussion as to how legislation, policy and guidelines specific to air quality, noise and vibration or social aspects is provided in each of relevant section of this impact assessment report.

The legislation and guidance listed in this section are current at the time of completion of the EES. Any changes to legislation and guidance that occur post completion of the EES, that is relevant to the project, would be expected to be considered and addressed at that time.

4.2 Australian Government

Table 4.1 summarises the policy and guideline documents relating to the conduct of health impact assessments in Australia, with some providing more specific guidance in health impacts associated with air quality. These documents were considered in the preparation of the health impact assessment for the project.

Table 4.1: Relevant legislation, policy and guidelines for addressing health impacts – Australian Government

Legislation, policy or guideline	Overview
<i>National Environment Protection Council Act 1994</i>	This Act relates to the establishment and operation of the National Environment Protection Council (NEPC), to meet the objectives that: - people enjoy the benefits or equivalent protection from air, water or soil pollution and from noise, wherever they live in Australia and - decisions of the business community are not distorted, and markets are not fragmented, by variations between participating jurisdictions in relation to the adoption or implementation of major environment protection measures The Act provides for the NEPC to make, and vary or revoke, National Environment Protection Measures (NEPM), and assess the implementation of the Measures.
enHealth Health Impact Assessment Guidelines (enHealth 2017)	The enHealth guidelines aim to promote and enhance the incorporation of health impact assessments into environmental and planning impact assessment generally, thereby improving the consideration of health issues. The document provides an introduction to the health impact assessment process, the different types of assessments that can be undertaken, the principles that may need to be addressed in an assessment, the roles of those involved in an assessment and general information on the preparation of a health impact assessment.

Legislation, policy or guideline	Overview
Health Impact Assessment: A Practical Guide (Harris 2007)	This document provides a more practical overview of the health impact assessment process in Australia. The document outlines the key phases and steps involved in conducting an assessment, the key concepts, the different levels of assessment that can be undertaken within a health impact assessment and approaches that can be considered in the conduct of a health impact assessment.
enHealth Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a)	This enHealth document provides an outline of the national approach adopted for the assessment of environmental health risks. While risk assessment is part of the health impact assessment process, the conduct of such an assessment typically focuses on key elements within the health impact assessment, where a more detailed assessment of exposure, toxicity and health risk is required, and can be undertaken. The enHealth guidance provides the Australian framework and approach for the conduct of such assessments.
enHealth Australian Exposure Factors Guide (enHealth 2012b)	This is a compendium to the enHealth (2012a) guideline. The document provides a review of quantitative exposure factors that may be used in the conduct of a quantitative health risk assessment.
NEPC National Environment Protection (Ambient Air Quality) Measure (2016)	This guidance is implemented under Section 14 of the <i>National Environment Protection Council Act 1994</i> and provides the desired environmental outcomes and protection standards and goals for ambient air quality in Australia. Protocols for the sampling and reporting of ambient air pollution within the guideline are also presented. The guideline sets standards and goals for carbon monoxide, nitrogen dioxide, photochemical oxidants, sulfur dioxide, lead and particulates as PM ₁₀ and PM _{2.5} . The 2016 update came into force in August 2016 and has been considered in the health impact assessment for the project, along with supporting documents.
NEPC National Environmental Protection (Air Toxics) Measure (NEPC 2004)	This guidance is implemented under Section 14 of the <i>National Environment Protection Council Act 1994</i> and provides the desired environmental outcomes, protection protocols, sampling methods, and monitoring investigation levels for benzene, benzo(a)pyrene, formaldehyde, toluene and xylenes in ambient air in Australia.
National Environment Protection Council (NEPC) Schedule B8 Guideline on Community Consultation and Risk Communication, National Environment Protection (Assessment of Site Contamination) Measure (NEPC 1999 amended 2013a)	The guideline provides an approach to effective community engagement and risk communication in relation to the assessment of environmental health risk issues.
Air Quality in and Around Traffic Tunnels (NHMRC 2008)	This document reviews data and information relevant to characterising and evaluating air quality in and around road tunnels, and the factors associated with poor air quality within tunnels. While the review is dated, it summarises key issues relevant to the assessment of air quality in and around tunnels.

4.3 Victorian Government

Table 4.2 summarises relevant Victorian Government legislation and guidance that broadly relate to the protection of health and wellbeing.

The *Public Health and Wellbeing Act 2008* (Vic) has provisions that can be used to require a health impact assessment for a project. The Act does not provide any reference or details as to the conduct of the assessment. This is why national guidance (as discussed in **Section 4.2**) was used to develop the overall methodology of the health impact assessment for the project.

Table 4.2: Relevant legislation, policy and guidelines for addressing health impacts – Victorian Government

Legislation, policy or guideline	Overview
<i>Public Health and Wellbeing Act 2008</i>	Under Part 5, Division 3, the Minister may require the conduct of a health impact assessment of the public health and wellbeing impact of a matter. The timing for completion of the assessment may be determined in such a direction. No such direction has been issued from the Minister in relation to North East Link. The Act does not specify any details relating to the completion of a health impact assessment.
<i>Environment Protection Act 1970</i> (EP Act) <i>Environment Protection Act 2017</i> (the 2017 Act)	This Act provides the legal framework for the protection of the environment in Victoria. The 2017 Act is part of legislative reform to the EP Act and came into effect on 1 July 2018. The 2017 Act must be read as if it forms part of the 1970 EP Act. It relates to noise and emissions to air, water and land. It states that health and wellbeing, local amenity and aesthetic considerations are important beneficial uses and values of the environment. The Act also states that the objective of the EPA is to protect human health and the environment by reducing the harmful effects of pollution and waste. The Act outlines penalties for pollution of air, water or land. It also addresses objectionable noise and powers to address noise complaints. This Act does not specify any details in relation to the completion of a health impact assessment.
State Environmental Protection Policy (Ambient Air Quality), No. S19 Gazette 9/2/1999 (SEPP)	This policy adopts the NEPM (Ambient Air quality) measure (outlined in Section 3.1) and outlines all ambient air quality objectives relevant to Victoria. Health and wellbeing are identified as a beneficial use to be protected throughout Victoria. The policy outlines objectives and goals for air quality.
State Environment Protection Policy (Air Quality Management), No. S240, Gazette 21/12/2001	This policy sits alongside the SEPP (Ambient Air Quality) and outlines the management measures for air quality in Victoria. The policy provides design ground level concentrations for a range of air quality pollutants grouped as Class 1, Class 2 and Class 3 indicators. These guidelines are considered in EES Technical report B – Air quality.
Variation to the State Environment Protection Policy (Ambient Air Guideline), No. G30, Gazette 28/06/2016	This is a variation to the SEPP, that adopts the revision to the NEPM (Ambient Air Quality) published in 2016.
Environment Protection (Vehicle Emissions) Regulations 2013	These regulations aim to minimise the impacts of air and noise emissions from motor vehicles and the release of petrol vapours related to the production of petrol. They prescribe vehicle emission and noise standards.
Vic Roads 2005, Traffic Noise Reduction Policy*	This is the current policy related to road traffic noise. The policy outlines measures that can be implemented to reduce road traffic noise and noise abatement measures.

Legislation, policy or guideline	Overview
Vic Roads 2007, Noise Guidelines – Construction and Maintenance Works*	The Noise Guidelines provide guidance for construction and maintenance projects to minimise and manage noise impacts. They relate to all VicRoads projects.
State Environment Protection Policy (Control of Noise from Commerce, Industry and Trade) No. N-1, No. S31, Gazette 15/6/1989 and variations	This SEPP provides guidance about the requirements regarding noise for industrial facilities.

* Does not apply to North East Link roads

4.4 Local government

Table 4.3 lists local government policies and guidelines relevant to key aspects associated with the health and wellbeing of local communities. They do not provide specific guidance on the conduct of a health impact assessment.

Table 4.3: Relevant policy and guidelines for addressing health impacts – local government

Guideline	Overview
Council plans: - Banyule 2017-2021 - Boroondara 2017-2021 - Manningham 2017-2021 - Nillumbik Draft 2017-2021 - Whitehorse 2017-2021 - Whittlesea 2017-2021 - Yarra 2017-2021	These plans relate to planning, development, allocation of resources and provision of services to the local community. Banyule: Vision is for a green, sustainable and vibrant place for a healthy, connected and inclusive community. Objectives include strong, healthy and inclusive communities to support and promote health and wellbeing. Boroondara: Vision is for a vibrant and inclusive city that meets the needs of its communities. Plans and strategic objectives include community facilities and services, parks and green spaces, the environment, and getting around Boroondara. Manningham: Healthy community, including a healthy, resilient and safe community; and a connected and inclusive community are key goals of the plan. Nillumbik: Goal of the plan is for healthy and safe communities. Whitehorse: The vision for the plan includes health, with health and wellbeing being a key value. Whittlesea: Priority and goal of the plan includes health and wellbeing, specifically to build a healthy and sustainable community that has a sense of wellbeing, inclusion and belonging. Yarra: A key objective of the plan is for a healthy Yarra, community health, safety and wellbeing are a focus. Incorporates the updated Health Plan for the area, to meet requirements under the Victorian <i>Public Health and Wellbeing Act 2008</i>. All plans reference the relevant Health and Wellbeing Plans.
Health & Wellbeing Plans: - Banyule Council Plan – 2017-2021 - Boroondara Community Plan 2017-21 - Manningham Healthy City Strategy 2017-2021	These plans are developed to meet requirements under the Victorian <i>Public Health and Wellbeing Act 2008</i>. These documents provide strategic plans to establish priorities that promote health, wellbeing and safety in the local community. These plans inform the various Council Plans. The Boroondara Public Health and Wellbeing Plan 2013-2017 is also incorporated in the Boroondara Community Plan 2017-27.

Guideline	Overview
• Nillumbik Health and Wellbeing Plan 2017-2021 • Whitehorse Health and Wellbeing Plan 2017-2021 • Whittlesea Health and Wellbeing Partnership Plan 2017-2021 • Yarra Health Plan 2013-2017	
Planning schemes for all relevant LGAs	Local planning schemes are required under the <i>Planning & Environment Act 1987</i>. These plans require planning to address health, wellbeing and safety, with reference to the need for healthy neighbourhoods to foster healthy and active living and community wellbeing. These plans also address urban and regional growth, outline environmental values, economic development and transport. More specific visions include the requirement to foster community spirit, social health and wellbeing (Banyule); maintain healthy and liveable communities and neighbourhoods (Boroondara); encourage healthy, safe and resilient communities (Nillumbik); support a healthy, vibrant, inclusive and diverse community (Whitehorse).

References for guidelines:

Banyule: <https://www.banyule.vic.gov.au/Council/Council-Plan>; <https://www.banyule.vic.gov.au/Services/Public-Health-for-Residents/Public-Health-for-Residents>; <http://planning-schemes.delwp.vic.gov.au/schemes/banyule>

Boroondara: <https://www.boroondara.vic.gov.au/about-council/council-administration/council-plan-and-budget/council-plan>; <https://www.boroondara.vic.gov.au/about-council/council-administration/policies-plans-and-strategies/reports/boroondara-community-plan/our-health-priorities>; <http://planning-schemes.delwp.vic.gov.au/schemes/boroondara>

Manningham: <https://www.manningham.vic.gov.au/council-plan>; <https://www.manningham.vic.gov.au/healthy-city-strategy>; <https://www.manningham.vic.gov.au/community-wellbeing>; <http://planning-schemes.delwp.vic.gov.au/schemes/manningham>

Nillumbik: <http://www.nillumbik.vic.gov.au/Planning-matters>; <http://www.nillumbik.vic.gov.au/People-and-family/Health/Health-and-Wellbeing>; <http://planning-schemes.delwp.vic.gov.au/schemes/nillumbik>

Whitehorse: <http://www.whitehorse.vic.gov.au/CouncilPlan.html>; <http://www.whitehorse.vic.gov.au/Health-and-Wellbeing-Plan.html>; <http://planning-schemes.delwp.vic.gov.au/schemes/whitehorse>

Whittlesea: <https://www.whittlesea.vic.gov.au/about-us/news-publications/plans-strategies-and-policies/council-plan-shaping-our-future/>; <https://www.whittlesea.vic.gov.au/about-us/news-publications/plans-strategies-and-policies/health-and-wellbeing-partnership-plan/>; <http://planning-schemes.delwp.vic.gov.au/schemes/whittlesea>

Yarra: <https://www.yarracity.vic.gov.au/about-us/council-information/council-plan>; <https://www.yarracity.vic.gov.au/about-us/council-information/council-plan/public-health-plan>; <http://planningschemes.dpcd.vic.gov.au/schemes/yarra>

4.5 Other standards and guidelines

Additional specific technical guidelines relevant to the more detailed assessment of health impacts associated with changes in air quality, noise and social stressors (that is, changes in stress from changes in the social environment) from international agencies such as the World Health Organisation and US EPA are referred to where relevant. These references are included in the health impact assessment as they relate to specific details and assessment methods in the assessment. References to these documents are included in each section of this report where they are relevant.

5. Methodology

5.1 Overview

This section describes the method that was used to assess the potential impacts of North East Link. A risk-based approach was applied to prioritise the key issues for assessment and inform measures to avoid, minimise and offset potential effects. **Figure 5.1** shows an overview of the assessment method.

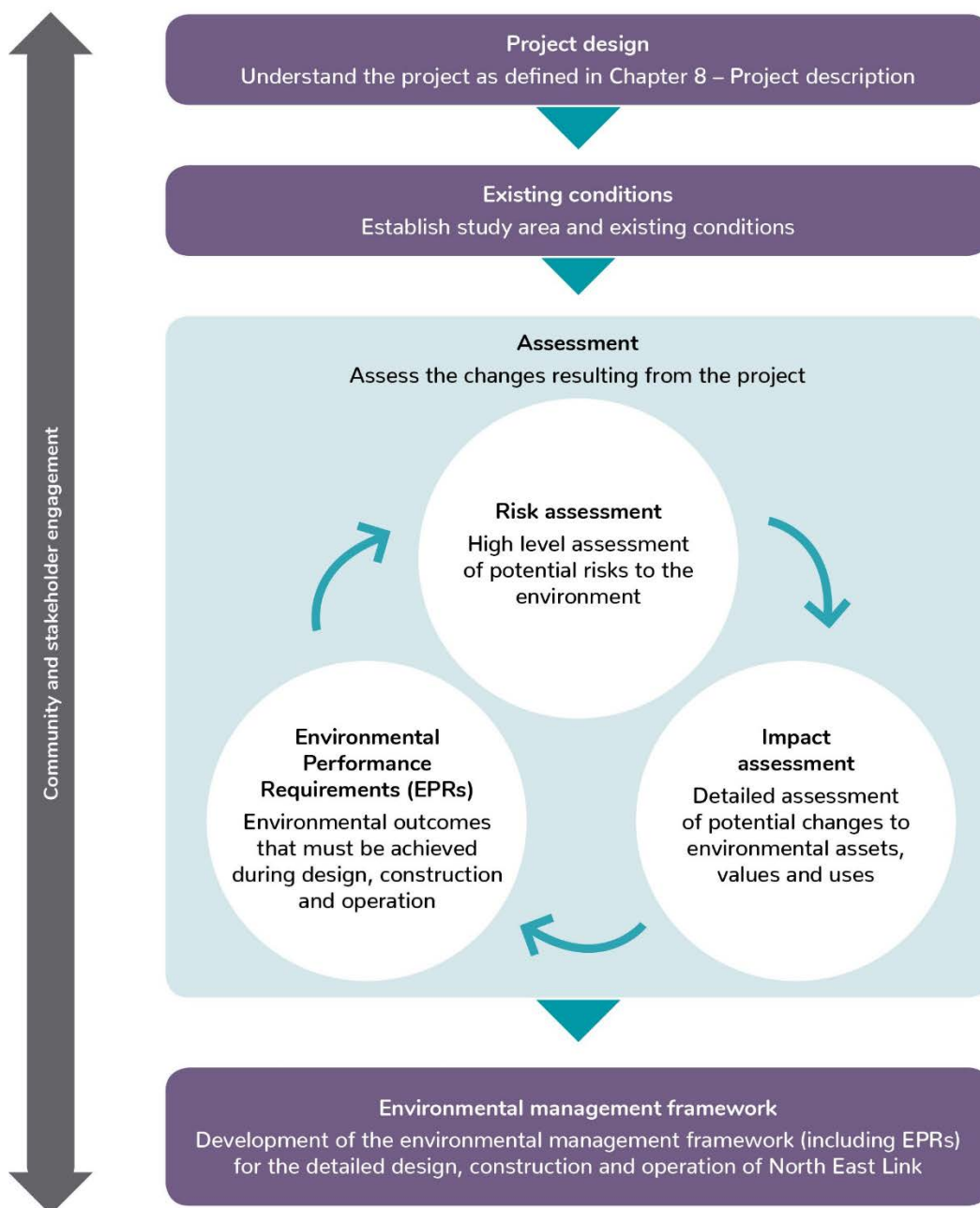


Figure 5.1: Overview of assessment method



In following this overall approach, this report presents two types of risk assessment for the project, which are outlined in this assessment:

1. Methodology adopted to undertake the technical evaluation of health impacts related to the project. This methodology (presented in **Section 5.2**) is specific to the conduct of a health impact assessment and follows the guidance as outlined in **Section 4**.
2. Project-wide risk assessment, where the key aspects of the health impact assessment have been evaluated within the broader project risk assessment to identify and summarise measures required to mitigate key issues. The methodology adopted for this aspect is outlined in **Section 5.3**.

It is noted there is some overlap in terminology used in the conduct of the health impact assessment and the broader risk assessment. To minimise mixing of the approaches adopted and terminology used, the outcomes relevant to each of these assessments, the structure of this report differs from the standard structure presented in other EES technical reports.

The following sections outline the method adopted for the health impact assessment.

5.2 Methodology and scope for health impact assessment

5.2.1 General

The health impact assessment was undertaken as a desk-top assessment. The term desk-top assessment is used to identify that the assessment has not involved the collection of any additional data over and above that which would be provided from project-specific EES technical studies, community consultation and statistics on the existing population. Rather, the assessment has been conducted using existing information with additional detail obtained via literature review only.

The impact assessment was undertaken in accordance with the guidelines outlined in **Section 4** and involved quantitative and qualitative evaluations. Definitions of some of the key terms that are used in the assessment are presented in Appendix A. The guidelines outlined in **Section 4** provide general guidance on the conduct of a health impact assessment. More specific details associated with how the assessment was undertaken are presented in **Sections 8, 9 and 10**.

More specifically, the scope of the health impact assessment was determined to meet the relevant scoping requirements of the EES (refer to **Section 2.2**), with specific details on the way in which the assessment was undertaken consistent with health impact assessments undertaken for similar major roadway projects in Australia, and discussions with Victoria's Environment Protection Authority (EPA Victoria) and Department of Health and Human Services (DHHS).

The focus of the health impact assessment was to assess the benefits and/or impacts to the local community and users of the project.



The impact assessment investigated:

- Changes in air quality within the local community associated with emissions from the tunnel ventilation structures
- Changes in air quality associated with changes in emissions from major surface roads
- Exposure of vehicle occupants to emissions present within the tunnels, during operation
- Changes in noise and vibration within the community
- Health implications of social changes related to the project.

These areas are directly affected by changes in traffic movements in the community and so traffic changes and impacts in the local community are also addressed.

The health impact assessment has not addressed occupational exposures during the construction or operation of the project. Occupational health and safety aspects of the project would be managed separately under current occupational health and safety regulations and guidelines as outlined and enforced by WorkSafe Victoria.

The health impact assessment drew directly upon other specific technical studies undertaken for the EES such as traffic, air quality, noise and social impacts. Wider aspects addressed in the EES (such as broad community benefits) and specific technical studies (such as land contamination) that address issues related to community health were also evaluated in the health impact assessment where relevant.

The health impact assessment has drawn on information provided in the relevant specialist reports and in some areas the assessment summarises key (and relevant) aspects. All details relevant to the underlying assumptions, methodology and interpretation of impacts relevant to these specialist areas are presented in the individual reports. Where more detail than provided in the health impact assessment is required, the reader is directed to the relevant specialist report.

The health impact assessment also addressed:

- The assessment of risk to facilitate stakeholder understanding of identified health issues.
The health impact assessment provides a consolidated overview of health-related risks relevant to the project.
- The development of measures to minimise impacts on human health and to inform the development of human health performance requirements (where possible).

The following sections outline the method adopted for the health impact assessment.

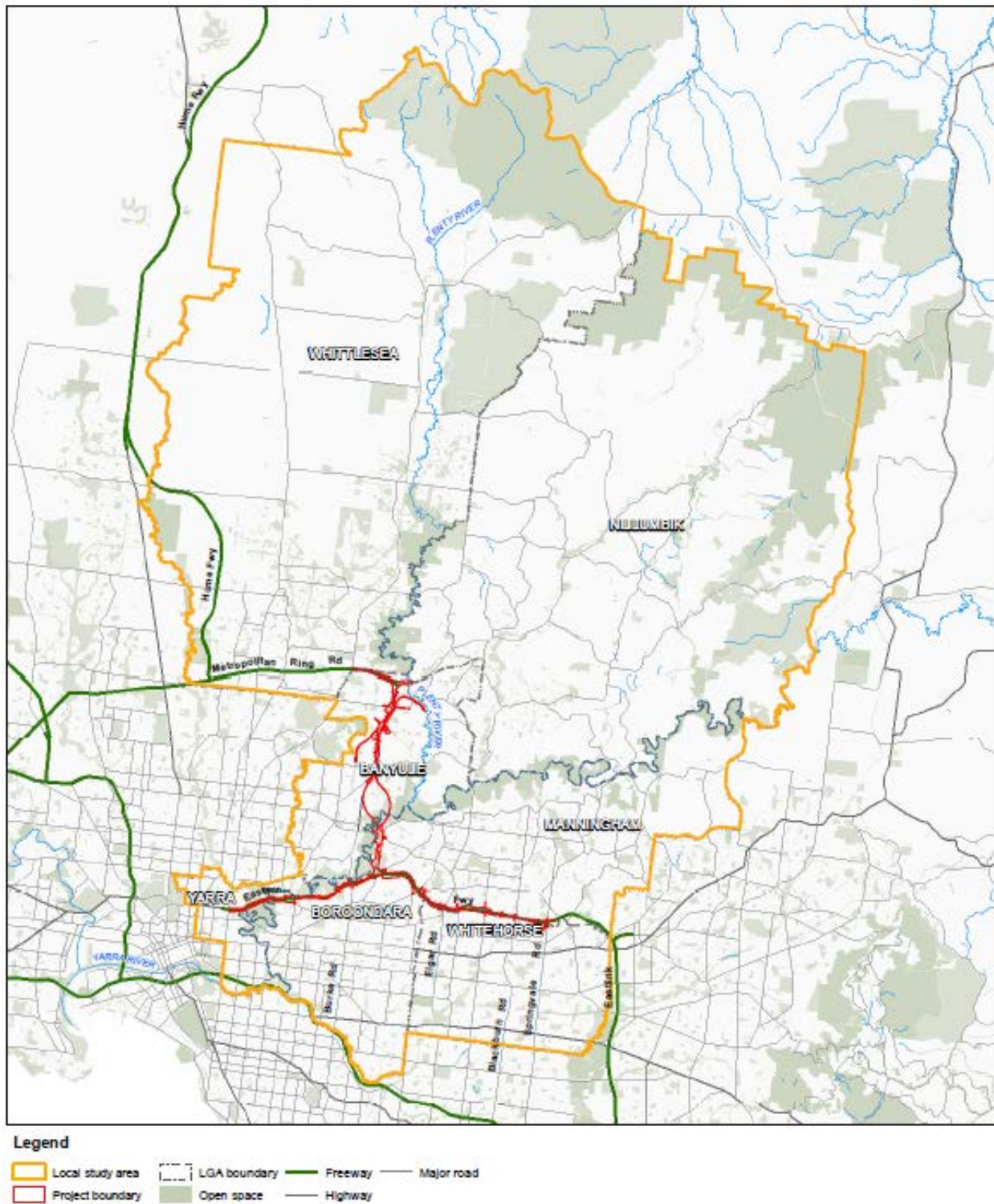


Figure 5.2: Local study area and project

5.2.2 Study area

The study area referred to in this report addresses the project corridor and surrounding areas, as assessed within the various key technical studies. More generally the health impact assessment has considered impacts in areas that extend beyond the immediate surrounding of the project, where possible. As a result, the assessment has generally considered:

- The regional study area of the Melbourne metropolitan area which includes the north-eastern suburbs. This area has been considered more broadly as health and wellbeing impacts may extend to this wider area;
- The local study area which includes the local government areas (LGAs) and suburbs intersected by the project as illustrated in **Figure 5.2**. These areas may be directly and indirectly affected by the project activities especially during construction and would experience positive and negative impacts.

It is noted that for the assessment of some of the more key impacts related to the project, the study area is specific to the technical studies relied on in this report. These areas are further defined as:

- Study area relevant to assessment of impacts to air quality are addressed in **Section 8.4**
- Study area relevant to the assessment of impacts from noise are addressed in **Section 9.4**.

5.2.3 Key issues addressed in health impact assessment

Air quality

Assessment of health impacts from changes in air quality associated with the project is presented in **Section 8** along with a more detailed discussion of the methodology adopted. In summary, the approach adopted in the assessment has addressed:

- The assessment of potential cumulative acute and chronic health impacts from changes in air quality particularly from nitrogen dioxide and carbon monoxide. This assessment has considered current NEPM guidance to evaluate potential health impacts and is presented in **Sections 8.6 and 8.7**.
- The assessment of potential incremental and cumulative acute and chronic health impacts from changes in air quality particularly from volatile organic compounds, polycyclic aromatic hydrocarbons and diesel particulate matter. This assessment has utilised current and appropriate health-based criteria for acute and chronic exposures and characterise risks in accordance with enHealth Guidelines, including a cancer risk estimate and is presented in **Section 8.6**.
- The evaluation of the potential cumulative and incremental health impacts from changes in air quality impacts associated with nitrogen dioxide and particulates such as PM_{2.5} and PM₁₀. The assessment has utilised current and appropriate health-based criteria for acute and chronic exposures on the basis of the World Health Organisation approach. This is presented in **Sections 8.8 and 8.9**.



These assessments focused on the operational phases of the project, and evaluated exposures within the tunnels and the local community to changes in air quality associated with changes in traffic composition and movements, and from tunnel ventilation structures.

Construction impacts have been addressed on the basis of a qualitative assessment, where potential impacts and the identification of relevant management measures to minimise impacts (including nuisance¹ dust) were evaluated.

The assessment of health impacts associated with changes in air quality has relied on EES Technical report B – Air quality.

Noise and vibration

The assessment of health impacts from changes in noise associated with the project is presented in **Section 9** along with a more detailed discussion of the methodology adopted. In summary, the approach adopted in the assessment has addressed:

- Qualitatively evaluate potential impacts of changes in noise and vibration on the local community
- Quantitatively assess potential impacts on the health of the local community due to changes in noise based on exposure response relationships developed in international studies relevant to characterising health impacts from noise, including annoyance and sleep disturbance which are discussed in **Section 9.5**.

The assessment considered health impacts in line with existing road traffic noise reduction policies in Victoria as well as current health information and assessment guidelines available from key organisations such as the World Health Organisation. The noise impact assessment considered changes in traffic composition and movements in the local areas resulting from the project.

The assessment of noise and vibration addressed the construction (utilising a qualitative assessment) and operation (using a quantitative assessment) of the project, and evaluated changes in noise and vibration within the local community.

The assessment of health impacts associated with changes in noise and vibration has relied on EES Technical report C –Surface noise and vibration.

¹ Nuisance, as considered in this report relates to: nuisance dust which is dust particles that are too large to penetrate into the lungs (and result in adverse health effects) but will settle out on various surfaces and may create a visible dust layer or require cleaning; nuisance odours which are odours that are noticeable and may be considered offensive. Health effects associated with exposure to chemicals that are the cause of the odours are assessed separately.

Social

The assessment of health impacts from changes in the social and community environment associated with the project is presented in **Section 10** along with a more detailed discussion of the methodology adopted. In summary, the approach adopted in the assessment has addressed:

- Qualitatively assess the social characteristics which have potential to affect community health (positive and negative impacts). This assessment has considered changes in air quality, noise, traffic composition and movements, pedestrian and cyclist access and safety, changes in recreational uses of the local area, changes in the connectivity (or displacement) of the community and changes in the urban environment. The assessment has drawn on published studies relating to health impacts of social changes and the social impact assessment.

The social assessment has focused on the construction and operation of the project, and evaluated social changes related to the project with potential to affect the local community.

5.3 Risk assessment

An environmental risk assessment has been completed to identify environmental risks associated with construction and operation of North East Link. The risk-based approach is shown in **Figure 5.3** and integral to the EES as required by section 3.1 of the Scoping Requirements and the *Ministerial guidelines for assessment of the environmental effects under the Environment Effects Act 1978*.

Specifically the EES risk assessment aimed to:

- Systematically identify the interactions between project elements and activities and assets, values and uses
- Focus the impact assessment and enable differentiation of significant and high risks and impacts from lower risks and impacts
- Inform development of the reference project to avoid, mitigate and manage environmental impacts
- Inform development of EPRs that set the minimum outcomes necessary to avoid, mitigate or manage environmental impacts and reduce environmental risks during delivery of the project.

This section presents an overview of the EES risk assessment process. EES Attachment III Environmental risk report describes each step in the risk assessment process in more detail and contains a consolidated risk register.

This technical report describes the risks associated with the project on [technical discipline].

Wherever risks relating to this study are referred to, the terminology 'risk XX01' is used.

Wherever EPRs relating to this study are referred to, the terminology 'EPR XX1' is used. The risk assessment completed for this study is provided as **Appendix I**.

5.3.1 Risk assessment process

The risk assessment process adopted for North East Link is consistent with AS/NZS ISO 31000:2009 Risk Management Process. The following tasks were undertaken to identify, analyse and evaluate risks:

- Use existing conditions and identify applicable legislation and policy to establish the context for the risk assessment
- Develop likelihood and consequence criteria and a risk matrix
- Consider construction and operational activities in the context of existing conditions to determine risk pathways
- Identify standard controls and requirements (Environmental Performance Requirements (EPRs)) to mitigate identified risks
- Assign likelihood and consequence ratings for each risk to determine risk ratings considering design, proposed activities and standard EPRs.

While there are clear steps in the risk process, it does not follow a linear progression and requires multiple iterations of risk ratings, pathways and EPRs as the technical assessments progress. Demonstrating this evolution, a set of initial and residual risk ratings and EPRs are produced for all technical reports. **Figure 5.3** shows this process.

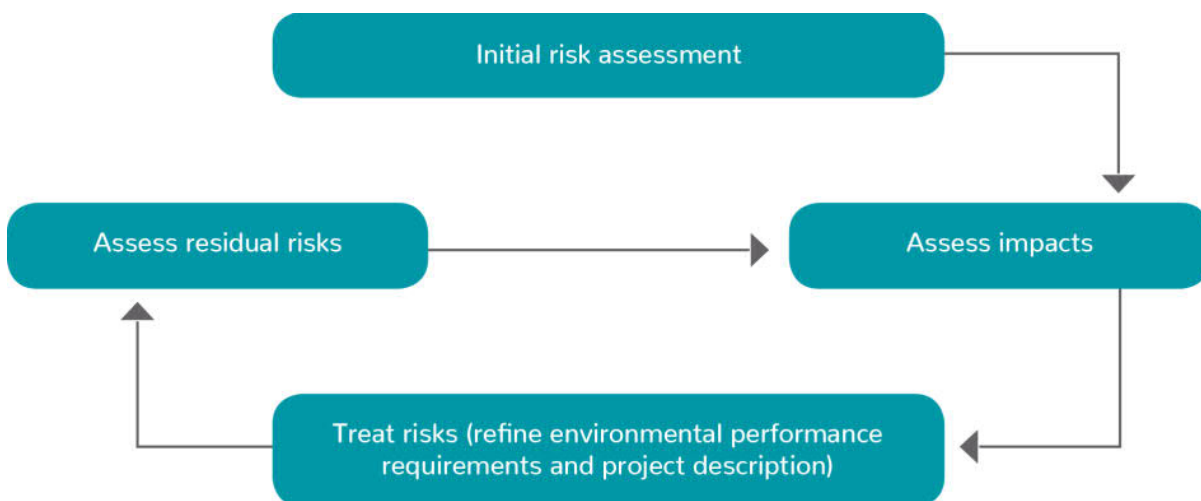


Figure 5.3: Risk analysis process

Rating risk

Risk ratings were assessed by considering the consequence and likelihood of an event occurring. In assessing the consequence, the extent, severity and duration of the risks were considered. These are discussed below.

Assigning the consequences of risks

'Consequence' refers to the maximum credible outcome of an event affecting an asset, value or use. Consequence criteria as presented in Chapter 4 – EES assessment framework, were developed for the North East Link EES to enable a consistent assessment of consequence across the range of potential environmental effects. Consequence criteria were assigned based on the maximum credible consequence of the risk pathway occurring. Where there was uncertainty or incomplete information, a conservative assessment was made on the basis of the maximum credible consequence.

Consequence criteria have been developed to consider the following characteristics:

- Extent of impact
- Severity of impact
- Duration of threat.

Severity has been assigned a greater weighting than extent and duration as this is considered the most important characteristic.

Each risk pathway was assigned a value for each of the three characteristics, which were added together to provide an overall consequence rating.

Further detail on the consequence criteria are provided in Chapter 4 – EES assessment framework.

Assigning the likelihood of risks

'Likelihood' refers to the chance of an event happening and the maximum credible consequence occurring from that event. The likelihood criteria are presented in **Table 5.1**.

Table 5.1: Likelihood of an event occurring

Planned	The event is certain to occur
Almost certain	The event is almost certain to occur one or more times a year
Likely	The event is likely to occur several times within a five-year timeframe
Possible	The event may occur once within a five-year timeframe
Unlikely	The event may occur under unusual circumstances but is not expected (ie once within a 20-year timeframe)
Rare	The event is very unlikely to occur but may occur in exceptional circumstances (ie once within a 100-year timeframe)

Risk matrix and risk rating

Risk levels were assessed using the matrix presented in **Table 5.2**.

Table 5.2: Risk matrix

Likelihood	Consequence				
	Negligible	Minor	Moderate	Major	Severe
Rare	Very low	Very low	Low	Medium	Medium
Unlikely	Very low	Low	Low	Medium	High
Possible	Low	Low	Medium	High	High
Likely	Low	Medium	Medium	High	Very high
Almost certain	Low	Medium	High	Very high	Very high
Planned	Planned (negligible consequence)	Planned (minor consequence)	Planned (moderate consequence)	Planned (major consequence)	Planned (severe consequence)

Planned events

North East Link would result in some planned events, being events with outcomes that are certain to occur (ie planned impacts such as land acquisition), as distinct from risk events where the chance of the event occurring and its consequence is uncertain. Although planned events are not risks, these were still documented in the risk register as part of Attachment III – Risk report for completeness and assigned a consequence level in order to enable issues requiring further assessment or treatment to be prioritised.

These planned events were assessed further through the impact assessment process.

Risk evaluation and treatment

The risk assessment process was used as a screening tool to prioritise potential impacts and the subsequent level of assessment undertaken as part of the impact assessment. For example, an issue that was given a risk level of medium or above, or was identified as a planned event with a consequence of minor or above, would go through a more thorough impact assessment process than a low risk.

Where initial risk ratings were found to be ‘medium’ or higher, or were planned events with a consequence of ‘minor’ or higher, options for additional or modified EPRs or design changes were considered where practicable. It should be noted that the consequence ratings presented in the risk register are solely based on the consequence criteria presented in Attachment III – Risk report. Further analysis and evaluation of the impacts potentially arising from both risks and planned events and information on how these would be managed is provided in **Sections 8, 9 and 10**.

5.4 Limitations and considerations

There are certain features of health impact assessment methodology important to acknowledge in the development of any assessment. These relate to the limitations of the methodology and the constraints applied within the health impact assessment to ensure a focus on aspects that can be influenced as part of the project. These are summarised below (also refer to **Section 11** for discussion of uncertainties):

- A health impact assessment is a systematic tool used to review key aspects of a specific project that may affect the health of the local community. The assessment includes qualitative and quantitative assessment methods.
- Where quantitative assessment methods are presented, a health impact assessment is typically based on a conservative estimate of impacts in the local community and thus is expected to overestimate the risks for all members of the community.
- A health impact assessment involves a number of aspects where a qualitative assessment is required to be undertaken. Where this is undertaken, it provides a general indication of potential benefits or impacts only.
- The community evaluated in a health impact assessment is limited by the extent of the studies undertaken in informing an EES. It is not possible to evaluate impacts on community health outside these areas.
- A health impact assessment relies on data provided from other studies prepared for an EES (as listed for this project in **Table 7.1**). The conclusions of this health impact assessment, therefore, depends on the assumptions and calculations undertaken to generate the data from these other studies utilised in this assessment.
- Conclusions can only be drawn with respect to impacts related to a project as outlined in an EES. Other health issues, not related to the project, that may be of significance to the local community are not addressed in the health impact assessment.
- The health impact assessment for this project did not address occupational health for construction workers.
- The health impact assessment reflects the current state of knowledge regarding the potential health effects of identified chemicals and pollutants for this project. 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.

5.5 Stakeholder engagement

Stakeholders and the community were consulted to support the preparation of the EES and to inform the development of the project and understanding of its potential impacts. **Table 5.3** lists specific engagement activities that have occurred in relation to this health impact assessment with more general engagement activities occurring at all stages of the project. Feedback received during community consultation sessions is summarised in **Section 5.6**.

Table 5.3: Stakeholder engagement undertaken for health impact assessment

Activity	When	Matters discussed	Outcome
Meeting with EPA Victoria and DHHS	22 October 2018	Approach to the health impact assessment, methodology for the quantitative assessment of air quality impacts and acceptability of risk	Discussion on the assessment of broader community risks vs localised risks, and the application or consideration of acceptable levels of risk for different types of risk calculations.
	20 November 2018	Key outcomes of the health impact assessment	Involved the presentation and discussion on the outcomes of the health impact assessment available to 20 November 2018

5.6 Community feedback

In addition to consultation undertaken with specific stakeholders, consultation has been ongoing with the community throughout the design development and the EES process. Feedback relevant to the health impacts assessment is summarised in **Table 5.4**, along with where and how we have addressed those topics in this report.

Table 5.4: Community consultation feedback addressed by the health impact assessment

Issues raised during community consultation	How it's been addressed
Increased noise and decreased air quality during operations will negatively affect the health of residents living along the project alignment.	These aspects have been considered in detail in the health impact assessment for all residents along the project alignment. Impacts associated with changes in air quality within the community are addressed in Section 8 and considered changes in air quality at receptors that included child care centres and aged care facilities. In addition, the maximum changes have been evaluated assuming exposure may occur to residents who include young children and the elderly. Impacts associated with changes in noise within the community have are addressed in Section 9. The assessment has evaluated how the changes in noise may impact on community health and affect levels of annoyance and sleep disturbance.
Decreased air quality and the health impacts on residents with asthma or other respiratory illnesses living along the project alignment.	The health impact assessment has included assessment of health impacts that includes respiratory effects including asthma in Section 8 . The health impact assessment has addressed these issues for all residents along the project alignment including exposure to young children and the elderly.



5.7 Peer review

This assessment has been independently peer reviewed by Dr Brian G. Priestly of Priestly Toxicology Consulting. The peer reviewer reviewed and provided feedback on drafts of this report, as well as the methodology, approach, assumptions and assessment criteria applied to the assessment. The peer reviewer's methodology is set out in his peer review report, which also included addressing whether there were any additional matters which should be considered as part of the impact assessment in order to address the EES scoping requirements, 'public works' Order or to otherwise adequately assess the likely impacts of the project relevant to this assessment or the management of those impacts. The peer reviewer also considered whether there were any gaps or matters in this assessment which they disagreed with. The final peer review report is attached as Appendix J of this report.

6. Community profile

6.1 General

This section summarises the demographics and existing health of the community potentially impacted by the project. While the key focus of the assessment was the local community surrounding the project, some aspects of the assessment required consideration of statistics derived from larger populations, such as those within larger local government areas (LGAs), the Melbourne metropolitan area as well as Victoria. Where relevant, information related to the local community as well as other areas within Melbourne (and Victoria) is presented.

North East Link would pass through an existing urbanised landscape in the area to the north-east of the Melbourne city area, which features established and diverse residential neighbourhoods and industrial areas, with shopping and commercial centres, parks and reserves, and community and recreation facilities.

North East Link would be located within the following metropolitan regions: Inner Metro Region, Inner South East Region, Northern Region, and Eastern Region. These regions comprise the following municipalities (with the LGAs more specifically evaluated in this assessment highlighted in bold):

- Inner Metro Region – LGAs of Melbourne, Port Phillip, **Yarra**
- Inner South East Region – LGAs of Stonnington, Bayside, **Boroondara**, Glen Eira
- Northern Region – LGAs of **Banyule**, **Whittlesea**, **Nillumbik**, Hume, Moreland, Darebin, Mitchell (part)
- Eastern Region – LGAs of **Manningham**, **Whitehorse**, Knox, Yarra Ranges, Maroondah, Monash.

North East Link would traverse regions ranging from the densely populated and highly urbanised Inner Metro and Inner South East regions, to the low density residential and recreational and natural areas of the outer parts of the Northern and Eastern regions.

When considering potential health impacts within any community, the health impact assessment considers the whole population as well as specific sensitive or vulnerable groups within the population. These communities and their related sensitive or vulnerable groups are:

- Community groups:
 - Residents
 - Recreational users (such as cyclists and users of recreational open space)
 - Commercial and industrial (such as businesses within the project area that may be directly impacted by property acquisitions)

■ Sensitive and vulnerable groups within the community groups:

- Young children (in particular children under the age of five years, but also including children up to 14 years)
- Older populations (>65 years of age)
- Disabled and those with pre-existing medical conditions
- Disadvantaged (socio-economically disadvantaged).

These receptors may reside or access any areas within the community. The air quality impact assessment has considered changes in air quality across a large grid, 10 by 15 kilometres, with varying levels of grid resolution. The assessment also considered properties located adjacent to key roadways where changes in traffic are anticipated, and so air quality. The grid and areas assessed adjacent to roadways include all the sensitive receptors as detailed above.

The assessment of noise impacts considered a range of residential and other sensitive community receptors within a corridor adjacent to the proposed project. This included the sensitive receptors as detailed above.

6.2 Surrounding area and population

North East Link would involve surface road changes and a new road and tunnels. These infrastructure changes would extend from the M80 Ring Road in the north to the Eastern Freeway in the south, as shown in **Figure 6.1**. The population in the suburbs directly affected by the project, as well as some adjacent suburbs that may also be affected by the project were considered in the health impact assessment. LGAs and suburbs included in the assessment were:

- Whittlesea, including the suburbs of Bundoora West, Bundoora North, Mill Park South, Thomastown and Lalor
- Nillumbik, including the suburb of Greensborough
- Banyule, including the suburbs of Bundoora, Greensborough, Watsonia North, Watsonia, Macleod, Yallambie, Rosanna, Viewbank, Heidelberg, Eaglemont, Ivanhoe East
- Manningham, including the suburbs of Bulleen, Doncaster, Doncaster East, Donvale, Nunawading
- Boroondara, including the suburbs of Balwyn North, Kew East and Kew
- Whitehorse, including the suburbs of Mont Albert North, Box Hill North, Blackburn North, Nunawading
- Yarra, including the suburbs of Alphington, Fairfield, Clifton Hill and Abbotsford.



Much of the study area is residential. In the southern part of the project corridor are a number of public conservation and recreation zones as shown in **Figure 6.2**. The study area also includes a number of areas noted to be of environmental significance and where vegetation protection through planning overlays is in place, as shown in **Figure 6.3**.

These suburbs also have an existing network of cycle paths and access routes shown in **Figure 6.4** as well as a variety of recreational areas.

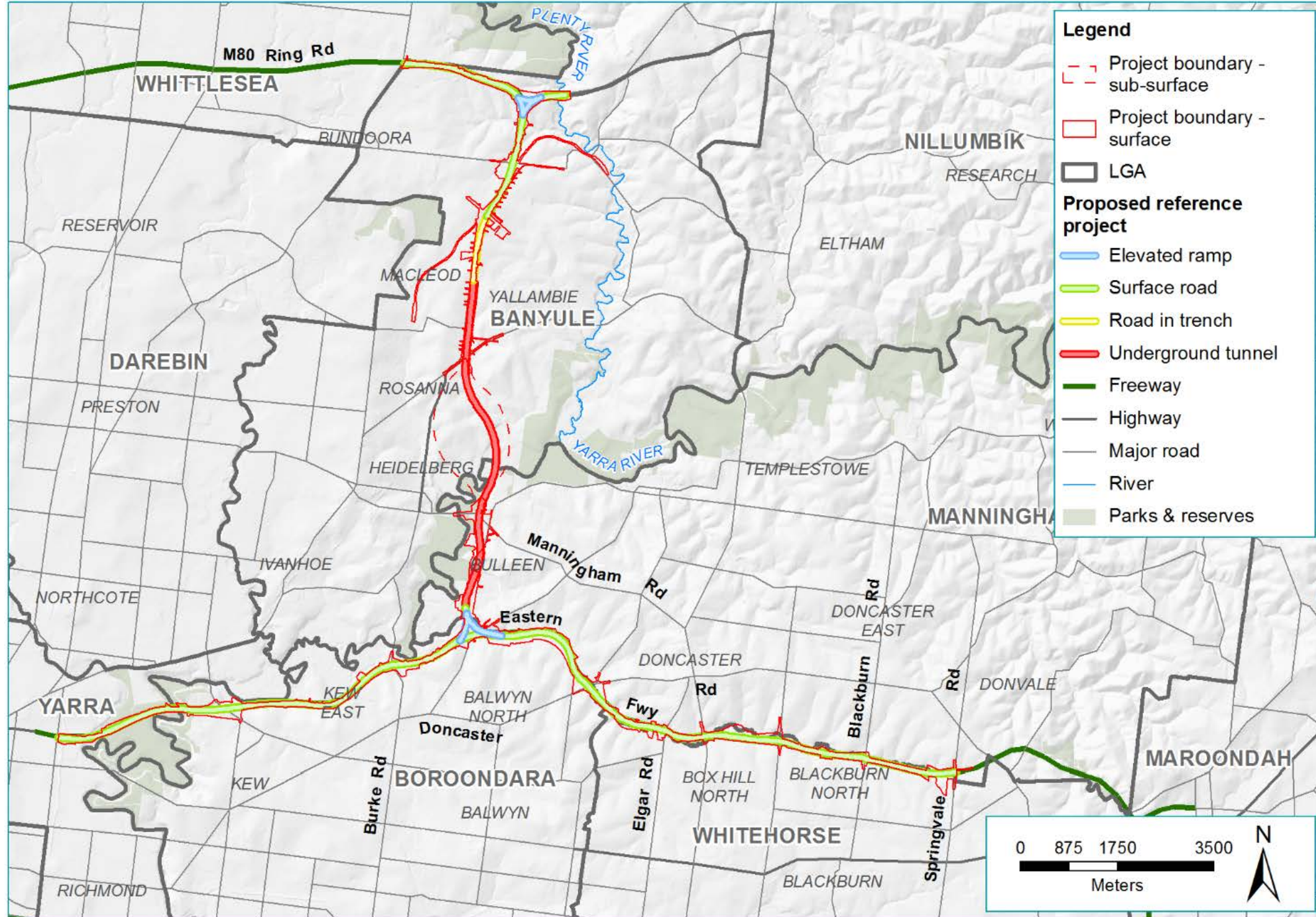


Figure 6.1: North East Link - LGAs

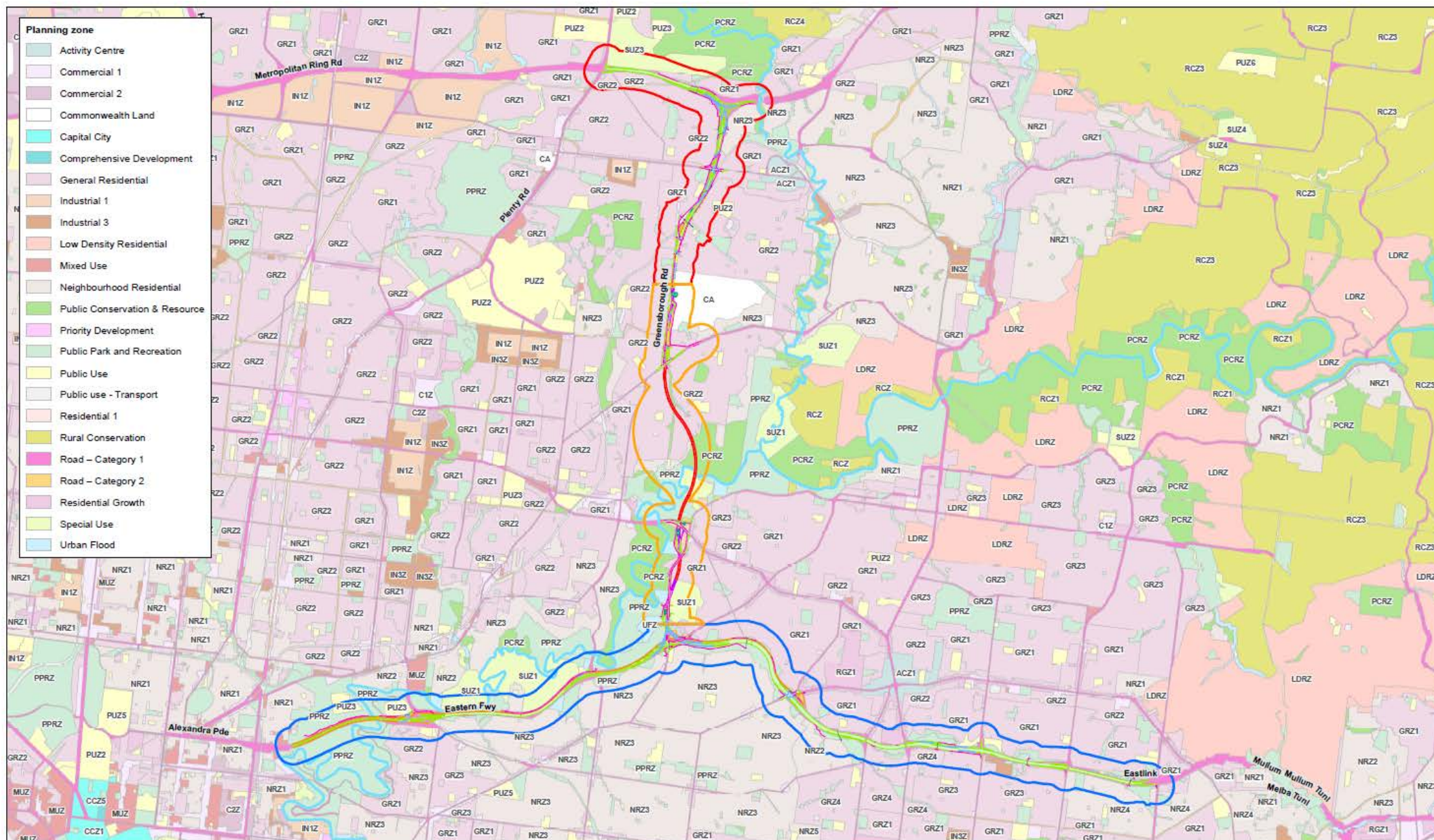
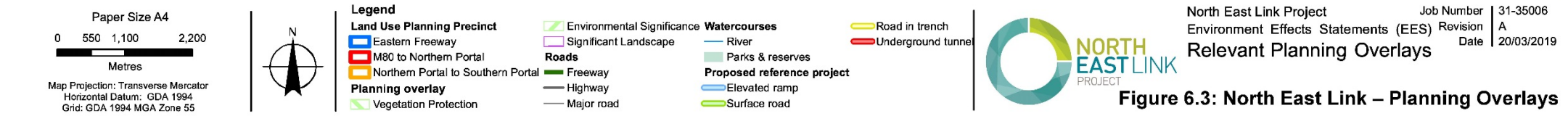
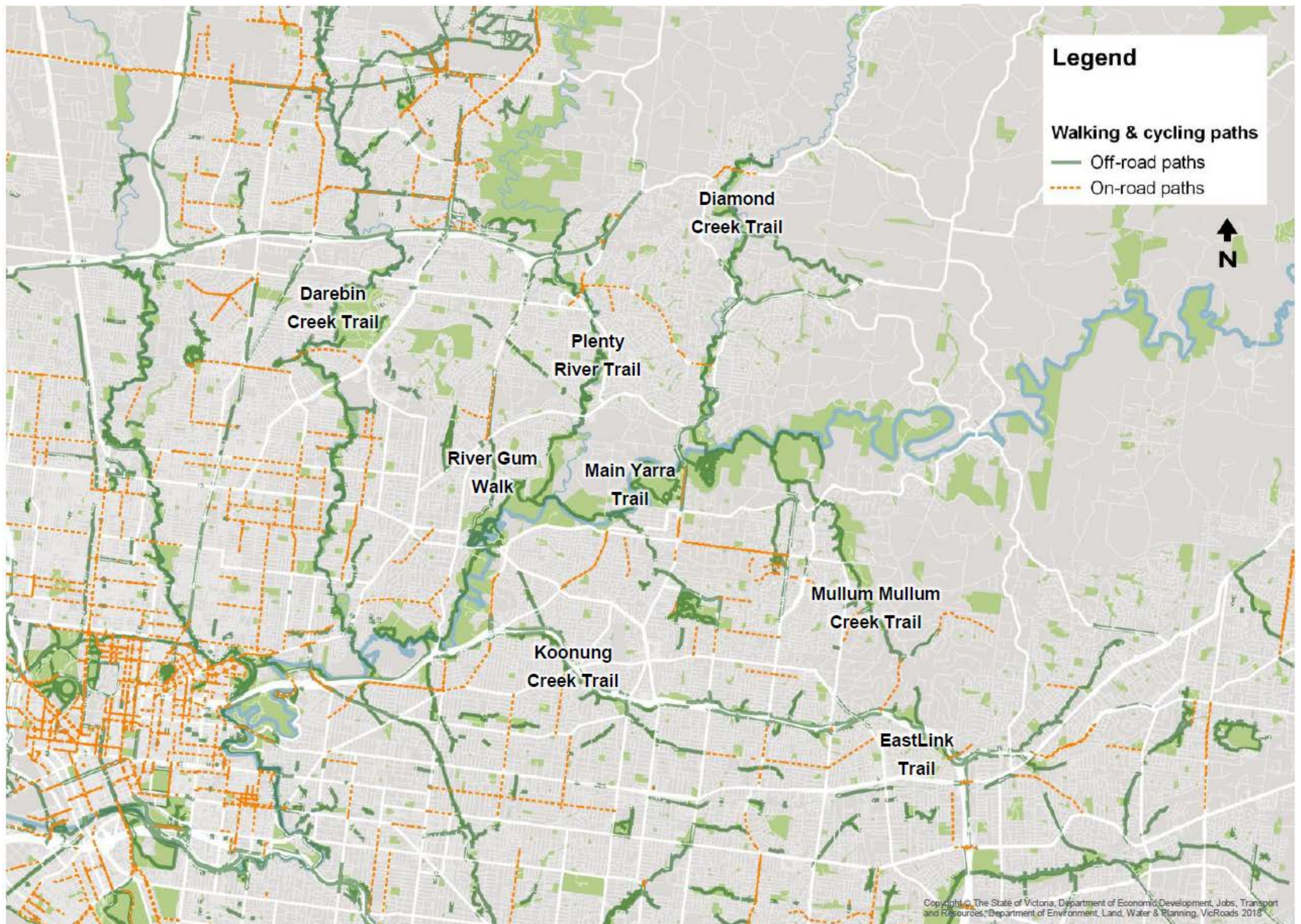


Figure 6.2: North East Link – Land use



Figure

North East Link Project	Job Number	31-35006
Environment Effects Statements (EES)	Revision	A
Relevant Planning Overlay	Date	20/03/2019



Ref: GNE/18/HIAR001/Rev 7

Figure 6.4: North East Link – Existing walking and bicycle networks

6.3 Population profile

Population statistics for suburbs and LGAs within the study area are available from the Australian Bureau of Statistics for the census year 2016 and are summarised in **Table 6.1** and shown in **Figure 6.5**. The composition of the populations located adjacent to the proposed project is expected to be generally consistent with population statistics for the individual suburbs. For the purpose of comparison, the population statistics presented also include the statistics for the larger statistical areas that include the LGAs as well as the Greater Melbourne Metropolitan area (as defined by the ABS which also encompasses the LGAs and suburbs relevant to this project) and Victoria.

It is noted the population profile presented in this section is general (at a suburb and LGA level). More specific detail that may be of interest within these areas is discussed in EES Technical report I – Social.

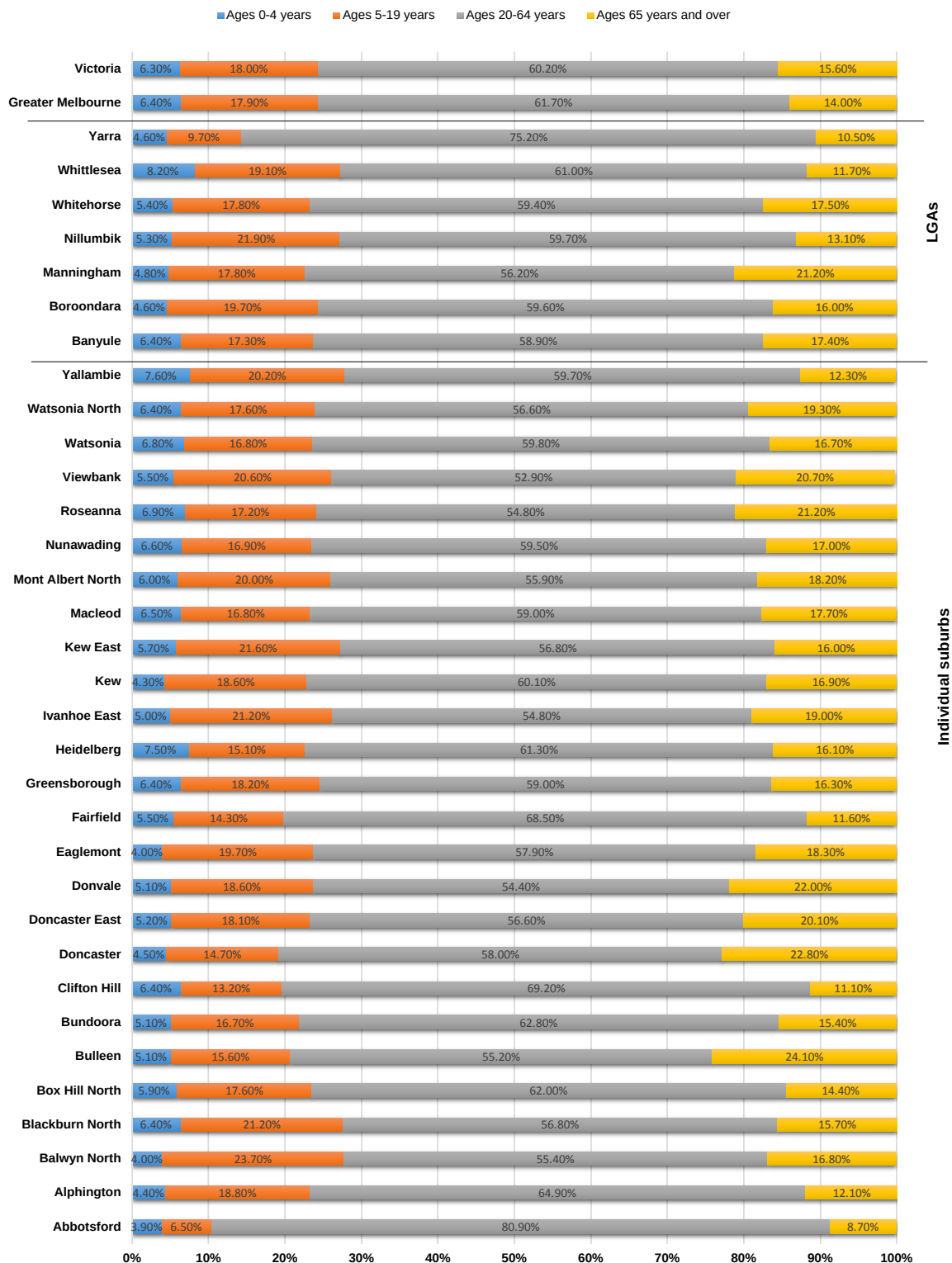
Table 6.1: Population statistics

Location	Population		% Population by key age groups					
	Male	Female	0–4	5–19	20–64	65+*	1–14*	30+*
Suburbs								
Abbotsford	4111	4081	3.9	6.5	80.9	8.7	6.8	59.5
Alphington	2410	2668	4.4	18.8	64.9	12.1	15.6	61.7
Balwyn North	9923	10481	4.0	23.7	55.4	16.8	18.0	60.4
Blackburn North	3611	3829	6.4	21.2	56.8	15.7	19.5	61.6
Box Hill North	5761	6114	5.9	17.6	62.0	14.4	16.3	60.7
Bulleen	5328	5548	5.1	15.6	55.2	24.1	14.4	67.9
Bundoora	14197	14460	5.1	16.7	62.8	15.4	13.6	55.9
Clifton Hill	3064	3273	6.4	13.2	69.2	11.1	14.9	64.3
Doncaster	10070	10881	4.5	14.7	58.0	22.8	12.4	65.5
Doncaster East	13664	14697	5.2	18.1	56.6	20.1	15.8	64.9
Donvale	5891	6455	5.1	18.6	54.4	22.0	16.3	65.8
Eaglemont	1897	1973	4.0	19.7	57.9	18.3	16.1	64.2
Fairfield	3207	3353	5.5	14.3	68.5	11.6	13.8	62.4
Greensborough	10126	10689	6.4	18.2	59.0	16.3	17.8	64.0
Heidelberg	3020	3207	7.5	15.1	61.3	16.1	16.3	62.8
Ivanhoe East	1880	1934	5.0	21.2	54.8	19.0	18.4	63.4
Kew	11747	12859	4.3	18.6	60.1	16.9	15.3	61.7
Kew East	3282	3354	5.7	21.6	56.8	16.0	19.3	61.0
Macleod	4688	5077	6.5	16.8	59.0	17.7	17.0	64.3
Mont Albert North	2589	2910	6.0	20.0	55.9	18.2	18.1	64.3
Nunawading	5752	6126	6.6	16.9	59.5	17.0	17.1	63.4
Roseanna	4012	4485	6.9	17.2	54.8	21.2	17.7	65.5
Viewbank	3432	3482	5.5	20.6	52.9	20.7	19.6	65.1

Location	Population		% Population by key age groups					
	Male	Female	0–4	5–19	20–64	65+*	1–14*	30+*
Watsonia	2553	2662	6.8	16.8	59.8	16.7	17.0	64.7
Watsonia North	1880	1933	6.4	17.6	56.6	19.3	16.8	64.8
Yallambie	2132	1981	7.6	20.2	59.7	12.3	19.4	57.5
Larger local statistical areas (Local government areas – includes state suburbs)								
Banyule	59222	62644	6.4	17.3	58.9	17.4	16.8	63.8
Boroondara	80145	87085	4.6	19.7	59.6	16.0	16.4	60.4
Manningham	56306	59953	4.8	17.8	56.2	21.2	15.3	65.5
Nillumbik	30266	31007	5.3	21.9	59.7	13.1	18.8	61.7
Whitehorse	78051	84032	5.4	17.8	59.4	17.5	15.9	61.5
Whittlesea	97541	99952	8.2	19.1	61.0	11.7	19.8	57.9
Yarra	42039	44619	4.6	9.7	75.2	10.5	10.1	61.3
Statistical areas of Melbourne and Victoria								
Greater Melbourne Metropolitan area	2199597	2285616	6.4	17.9	61.7	14.0	17.0	60.2
Victoria	2908077	3018549	6.3	18.0	60.2	15.6	17.0	61.3

Ref: Australian Bureau of Statistics, Census Data 2016

* Age groups specifically relevant to the characterisation of risk in the health impact assessment



Ref.: Australian Bureau of Statistics Census Data 2016

Figure 6.5: Population distribution in suburbs and LGAs relevant to project (% population in each area)



Based on this general population data, the suburbs relevant to North East Link are variable but broadly similar to that of greater Melbourne and Victoria. The exception is the suburbs in the City of Yarra (Alphington, Fairfield, Clifton Hill and Abbotsford), which had a notably higher proportion of people aged 20 to 64 years. For most suburbs the age structure of the population in the project area for North East Link is reflective of their respective larger LGAs.

Table 6.2 summarises the expected population growth in the LGAs considered in the health impact assessment.

Table 6.2: Projected population growth

Aspect	Banyule LGA ¹	Boroondara LGA ²	Manningham LGA ³	Nillumbik LGA ⁴	Whitehorse LGA ⁵	Whittlesea LGA ⁶	Yarra LGA ⁷
Resident population in 2016 from ABS	121,865	167,231	116,255	61,273	162,078	197,491	86,657
Predicted population in 2041	147,098	217,676	149,274	70,390	207,424 (for 2036)	382,896	157,607
Percentage population increase	15%	23%	21%	10%	22%	84%	82%
Most significant suburbs of population increase	Heidelberg Heidelberg Heights Ivanhoe	Hawthorn East Camberwell Hawthorn Deepdene	Doncaster Hill Doncaster Balance	Eltham (Central) Plenty – Yarrambat	Box Hill Burwood East Blackburn Burwood	Donnybrook Wollert	NA
Predicted changes in age distribution in suburbs relevant to project (proportion of local population)	Bundoora: predominately stable Greensborough: Slight decrease in very young persons and increase in seniors and elderly Watsonia North: decrease in young and increase in elderly Watsonia: Slight decrease in very young and slight increase in seniors and elderly. Macleod: predominately stable Yallambie: decrease in young and increase in elderly Rosanna: Slight decrease in young and slight increase in old. Viewbank: predominately stable	Balwyn North: slight increase in young and old with slight decrease in middle age Kew East: Decrease in young and increase in old Kew: slight decrease in middle age and slight increase in elderly	Bulleen: slight increase in empty nests and decrease in elderly Doncaster: N/A Doncaster East: decrease in young, increase in old. Donvale: predominately stable Nunawading: N/A	Greensborough: Decrease in young and middle age and increase in old	Mont Albert North: slight decrease in young and slight increase in old Box Hill North: slight decrease in young and slight increase in old Blackburn North: predominately stable Nunawading: predominately stable	NA	NA

Aspect	Banyule LGA ¹	Boroondara LGA ²	Manningham LGA ³	Nillumbik LGA ⁴	Whitehorse LGA ⁵	Whittlesea LGA ⁶	Yarra LGA ⁷
	Heidelberg: Increase in middle ages, slight decrease in young and old Eaglemont: variable through the age structure Ivanhoe East: decrease in the young and increase in the old						

Notes for Table 6.2:

- 1 <https://forecast.id.com.au/banyule/population-summary?WebID=10&AgeTypeKey=3> and <https://forecast.id.com.au/banyule/population-age-structure?WebID=10&AgeTypeKey=3>
- 2 <https://forecast.id.com.au/boroondara/population-summary> and <https://forecast.id.com.au/boroondara/population-age-structure?WebID=10&AgeTypeKey=3>
- 3 <https://forecast.id.com.au/manningham/population-summary> and <https://forecast.id.com.au/manningham/population-age-structure?AgeTypeKey=2>
- 4 <https://forecast.id.com.au/nillumbik/population-summary> and <https://forecast.id.com.au/nillumbik/population-age-structure>
- 5 <https://forecast.id.com.au/whitehorse/population-summary> and <https://forecast.id.com.au/whitehorse/population-age-structure>
- 6 <https://forecast.id.com.au/whittlesea/population-summary?AgeTypeKey=3> and <https://forecast.id.com.au/whittlesea/population-age-structure?AgeTypeKey=3>
- 7 <https://forecast.id.com.au/yarra/home>

The statistics above indicate that significant population growth is predicted in the Yarra and Whittlesea LGAs, with a lower rate of growth predicted in the other LGAs. These larger growth predictions include changes associated with urban renewal projects planned in these areas. These predicted changes indicate there is the potential for the population to increase over time with a disproportionate increase in more sensitive and vulnerable populations (such as young children and the elderly) in some areas.

Table 6.3 summarises a selected range of demographic measures relevant to the population of interest with comparison against the larger population areas. This includes the Index of Relative Socio-economic Disadvantage, which is an index that summarises a range of information about the economic and social conditions of people and households in an area. The index uses 10 deciles (ranging from 1 to 10, with each decile representing 10 per cent of the index range), with a low score or decile indicating a relatively greater disadvantage (for example, many households with low income, many people with no qualifications) and a high score indicating a general relative lack of disadvantage.

Table 6.3: 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 (%)	Index of Relative Socio-economic Disadvantage (Decile)*
Suburbs							
Abbotsford	32	2001	2142	426	2.1	5.3	9
Alphington	38	2123	2037	370	2.5	4.8	10
Balwyn North	41	1996	2500	480	3.0	6.1	10
Blackburn North	38	1528	2000	391	2.8	6.3	8
Box Hill North	37	1423	2000	385	2.7	7.2	7
Bulleen	45	1501	2000	400	2.7	6.2	8
Bundoora	34	1337	1733	346	2.8	9.4	6
Clifton Hill	36	2272	2383	475	2.4	3.7	10
Doncaster	42	1363	1993	416	2.5	7.6	7
Doncaster East	41	1504	2000	420	2.8	7.0	8
Donvale	44	1783	2100	395	2.8	4.5	10
Eaglemont	44	2348	2500	396	2.7	5.1	10
Fairfield	35	1710	2052	329	2.3	5.6	9
Greensborough	39	1724	1950	350	2.7	4.7	9
Heidelberg	37	1753	2167	365	2.4	5.2	9
Ivanhoe East	43	2495	2600	420	2.8	4.9	10
Kew	39	2206	2513	438	2.6	5.1	10
Kew East	40	2149	2500	430	2.8	4.8	10
Macleod	40	1627	2000	346	2.5	6.2	9

Location	Median age	Median household income (\$/week)	Median mortgage repayment (\$/month)	Median rent (\$/week)	Average household size	Unemployment rate (%)	Index of Relative Socio-economic Disadvantage (Decile)*
Mont Albert North	42	1908	2167	428	2.7	5.8	10
Nunawading	38	1510	1950	350	2.6	5.5	8
Roseanna	41	1720	2048	360	2.6	5.8	9
Viewbank	42	1746	2000	400	2.8	5.1	9
Watsonia	38	1448	1733	331	2.4	5.1	6
Watsonia North	40	1694	1950	356	2.8	4.6	8
Yallambie	35	1871	1950	320	2.9	4.3	10
Larger local statistical areas (Local government areas – includes state suburbs)							
Banyule	39	1655	1950	350	2.6	5.5	10
Boroondara	38	2083	2500	406	2.6	5.6	10
Manningham	43	1642	2050	415	2.8	6.1	10
Nillumbik	41	2098	2000	369	3.0	4.3	10
Whitehorse	38	1507	2000	376	2.6	7.0	10
Whittlesea	34	1444	1798	331	3.0	7.2	7
Yarra	33	1958	2167	421	2.1	5.3	9
Statistical areas of Melbourne and Victoria							
Greater Melbourne	36	1542	1800	350	2.7	6.8	--
Victoria	37	1419	1728	325	2.6	6.6	--

Source: Australian Bureau of Statistics, Census Data 2016

* Decile ranges from 1 which is most disadvantaged to 10 which is the least disadvantaged

The social demographics of an area have some influence on health and are important when considering equity aspects of a project such as North East Link.

All suburbs are in a position of relative social advantage, many lying at the highest decile of 10 (see **Table 6.3** above) in the ABS index. Bundoora and Watsonia were the lowest ranking of the identified suburbs with a value of 6.

It is also noted that EES Technical report I – Social has also considered culturally and linguistically diverse (CALD) groups within each LGA. Whittlesea and Yarra LGAs have a significant proportion of people from CALD backgrounds, while Nillumbik LGAs has relatively few people from CALD backgrounds. Banyule and Manningham LGAs are increasingly diverse in terms of CALD groups.

6.4 Existing health of population

6.4.1 General

When considering the health of a local community there are a large number of factors to consider. Community health 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. While it is possible to review existing health statistics for the areas surrounding the project and compare them with the greater Melbourne area or Victoria, it is not possible or appropriate to be able to identify a causal source, particularly individual or localised sources.

Information relevant to the health of populations in Victoria is available from various state and Australian government agencies including Victoria's Department of Health and Human Services (DHHS), the Australian Government's Department of Health, the Australian Institute of Health and Welfare and the Australian Commission on Safety and Quality in Health Care. This data relates to populations grouped by local government area or a metropolitan area. These data sets are not available for individual suburbs. In addition, not all the health data that may need to be considered for a health impact assessment is available for all these areas. The data that can be considered in a health impact assessment depends on the availability of data relevant to the populations in the areas to be evaluated.

The assessment presented in this report focused on key pollutants associated with construction and combustion sources (from vehicles), including particulate matter (namely PM_{2.5} and PM₁₀). 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 and earthworks, and personal exposures (such as smoking) and risk-taking behaviours that have potential to affect the health of any population.

6.4.2 Health-related behaviours

Information in relation to health-related behaviours linked to poorer health status and chronic disease including cardiovascular and respiratory diseases, cancer and other conditions that account for much of the burden of morbidity and mortality in later life is available for larger populations within LGAs or larger regions in metropolitan Melbourne. This data is regularly collected (on an annual basis) by the DHHS Intelligence Unit and reported within the Victorian Population Health Survey. The most comprehensive published survey is from 2014 (Department of Health and Human Services 2016b). This provides information rates of smoking, alcohol drinking, adequate physical activity, adequate intakes of fruit and vegetables, prevalence of overweight and obesity in the population and prevalence of high or very high levels of psychological distress.

Figure 6.6 and **Figure 6.7** show a comparison of relevant LGAs and Victoria in relation to these factors. These graphs summarise the rates of these behaviours in the LGAs relevant to the project in comparison to the state average.

The health-related behaviours presented are those with potential to adversely affect the health of the population.

Review of the data relevant to the LGAs of Banyule, Boroondara, Manningham, Nillumbik, Whitehorse, Whittlesea and Yarra indicate that for most of the health behaviours evaluated, the prevalence is statistically similar to that reported for Victoria. For some health behaviours there is a statistically significant difference between those found in the LGA when compared with Victoria. This includes:

- Lower prevalence of smoking in Whitehorse
- Lower prevalence of short-term risk from alcohol related injury in Whittlesea and higher prevalence in Nillumbik and Yarra
- Lower prevalence of long-term risks from alcohol related harm in Whittlesea and higher in Boroondara and Nillumbik
- Lower prevalence of non-compliance with fruit and vegetable intake in Nillumbik
- Lower prevalence of sufficient physical activity in Whittlesea and higher prevalence in Boroondara
- Lower prevalence of overweight in Whitehorse and Yarra
- Lower prevalence of obesity in Yarra
- Lower prevalence of high or very high psychological distress and fair or poor self-reported health in Nillumbik.

The data indicates the population in some of the LGAs of interest undertake health-related behaviours that are significantly more adverse than those relevant to the Victorian population. Those behaviours related to alcohol consumption and physical activity.

6.4.3 Health indicators

The Victorian Population Health Survey 2014 (Department of Health and Human Services 2016a) provides a summary of the rate of selected chronic diseases within the Victorian population, with data for individual LGAs compared against the rate for Victoria. Chronic diseases² considered generally relevant to the conduct of a health impact assessment for transport infrastructure projects include heart disease, stroke and cancer. In addition, data on asthma are also relevant, with the latest asthma prevalence data in available in the Victorian Population Health Survey 2011–2012 (Department of Health 2014). **Section 6.4.4** provides further discussion about asthma.

² Many different illness and health conditions can be classified under the broad heading of chronic disease. Typically, chronic diseases are long-lasting, and have persistent effects. Chronic diseases can range from mild conditions, such as short-sightedness, dental decay and minor hearing loss, to debilitating arthritis and low back pain, and to life-threatening heart disease and cancers. These conditions may never be cured completely, so there is generally a need for long-term management. Once present, chronic diseases often persist throughout life, although they are not always the cause of death (refer to the Australian Government Department of Health for further details on chronic diseases).



Figure 6.7 summarises data relevant to the prevalence of selected chronic diseases as reported in the 2014 and 2011–2012 surveys for the LGAs considered in the health impact assessment for the project, with comparison against Victoria. These data indicate that some of the LGAs have a statistically significant difference in the prevalence of heart disease, hypertension, cancer, asthma in adults and depression or anxiety. While all but one of the differences are lower prevalence, the LGA of Whittlesea has a higher prevalence of heart disease than reported for the Victorian population.

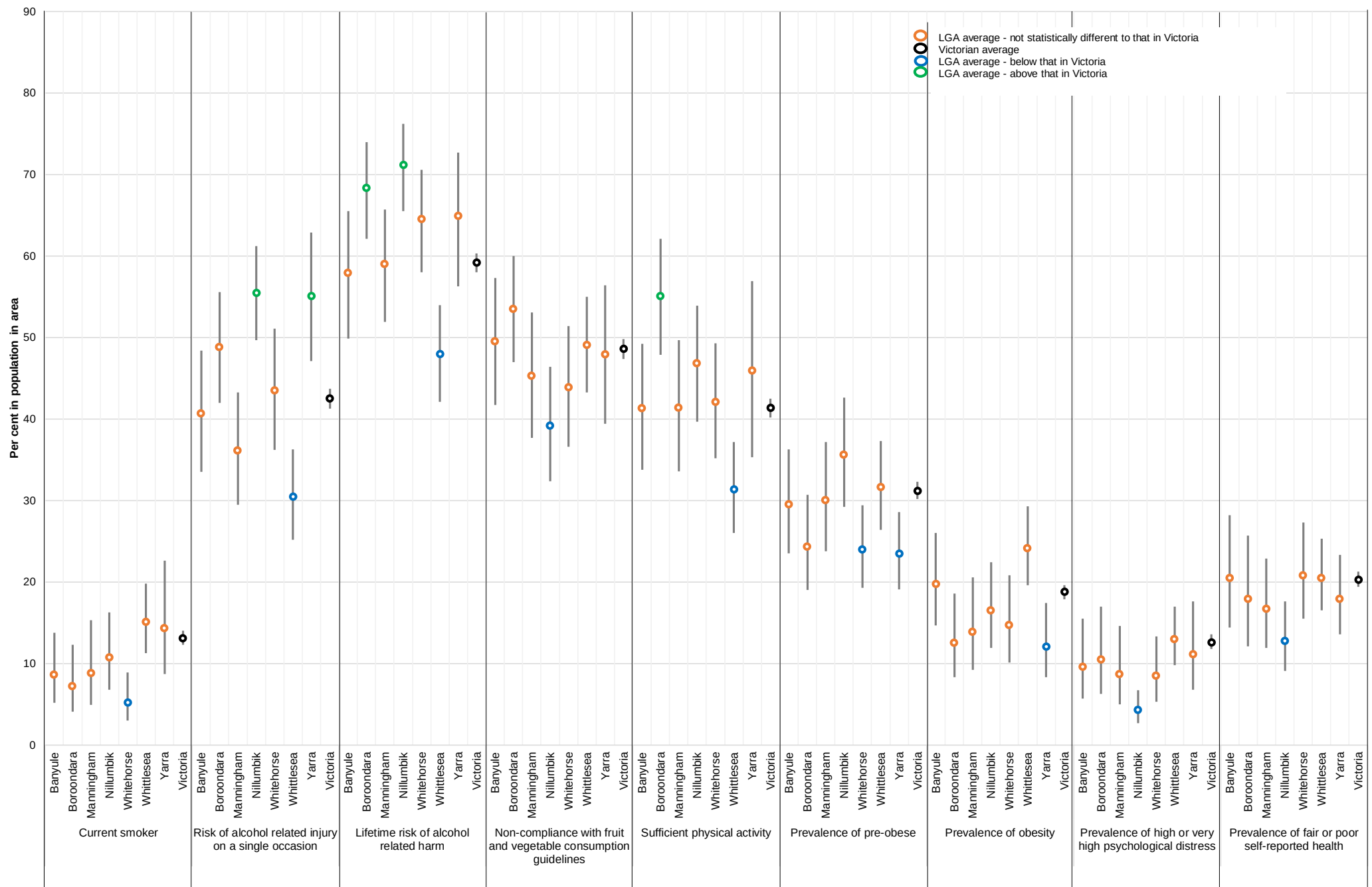


Figure 6.6: Summary of health-related behaviours in LGAs of interest compared with Victoria (2014) (average and 95% confidence interval)

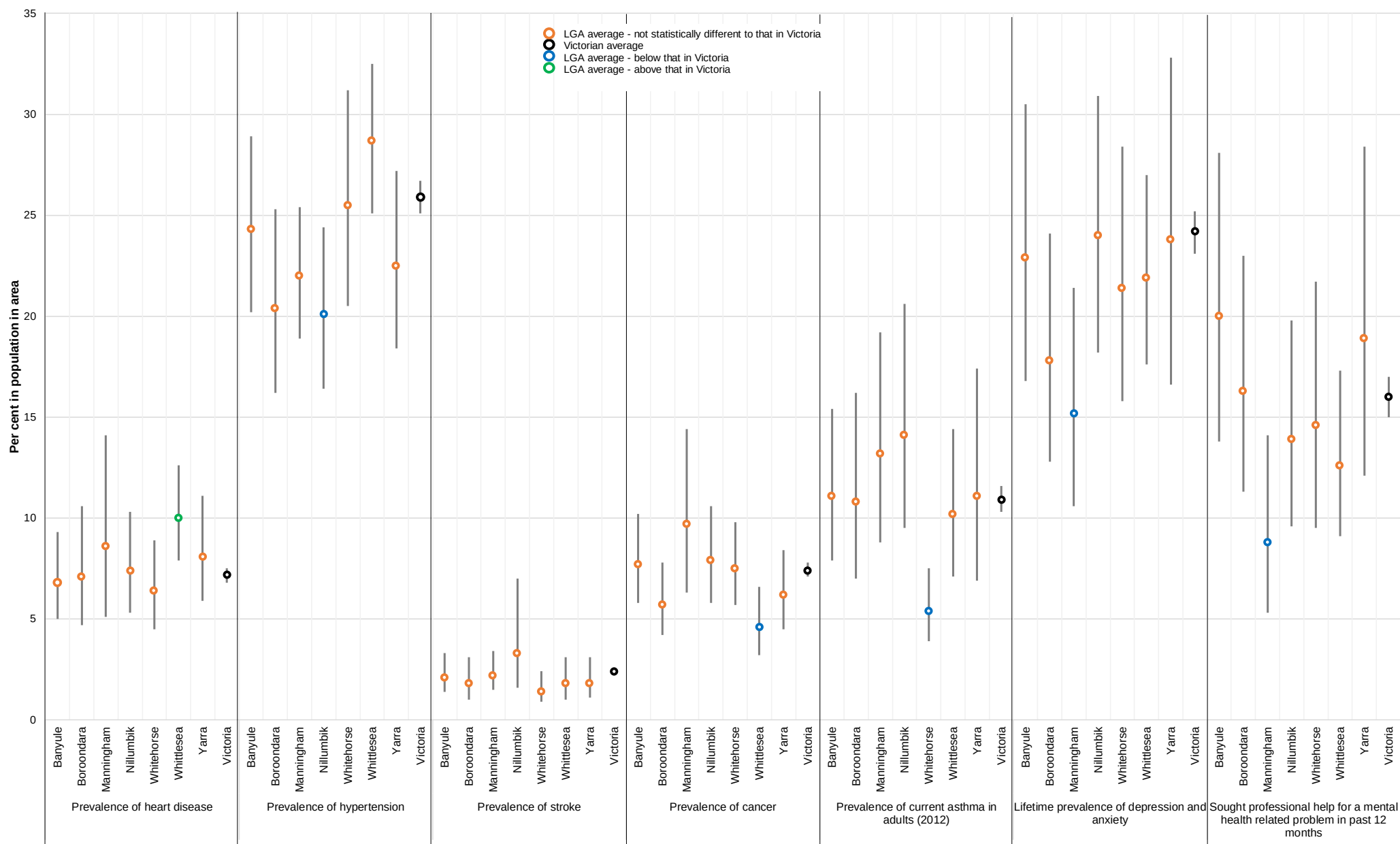


Figure 6.7: Summary of selected chronic diseases in LGAs of interest compared with Victoria (2014) (average and 95% confidence interval)

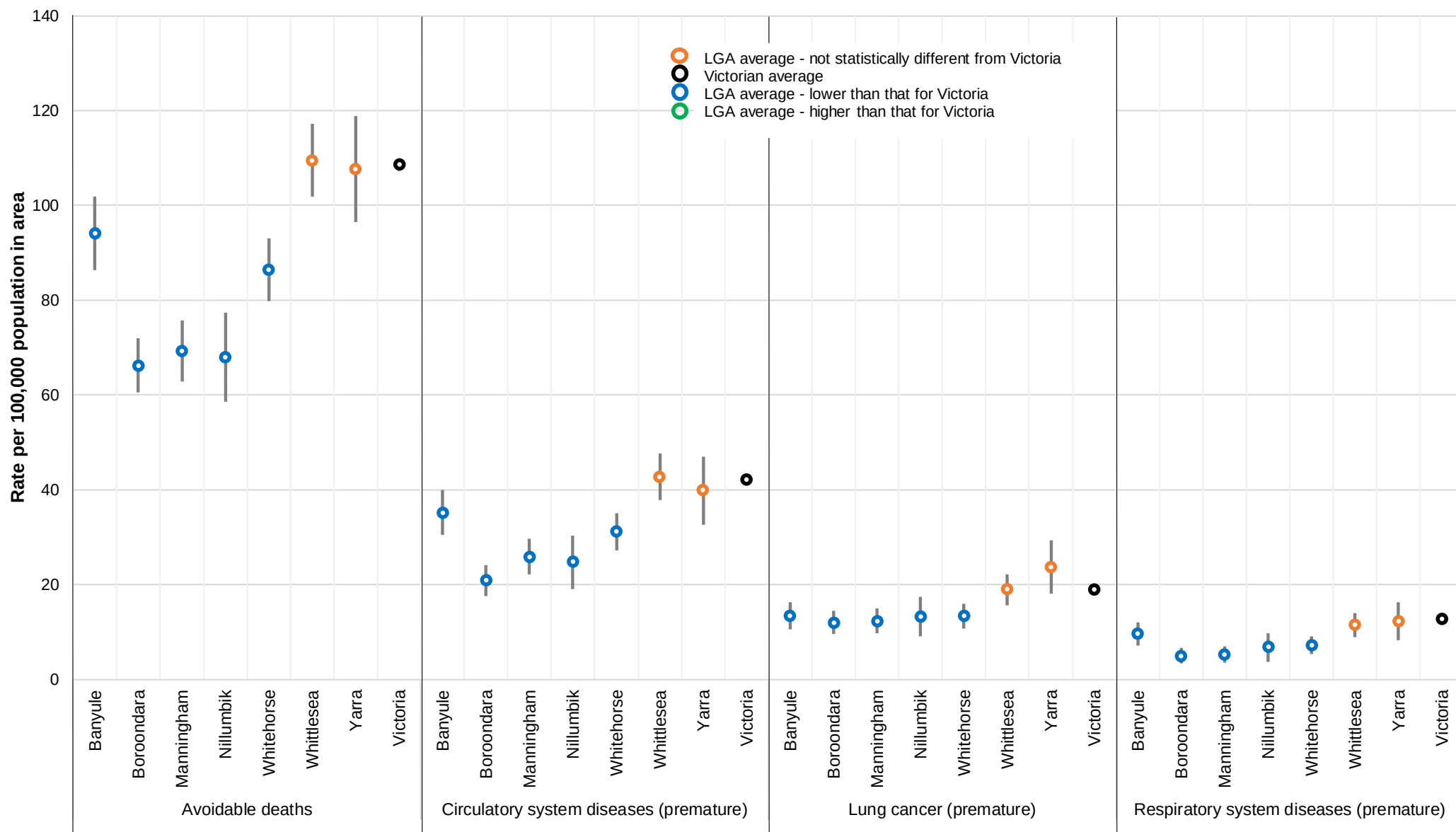


Figure 6.8: Summary of selected avoidable and premature mortality for ages 0–74 years in LGAs of interest compared with Victoria (2010–2014)
(average annual rate [age standardised] as average and 95% confidence interval)

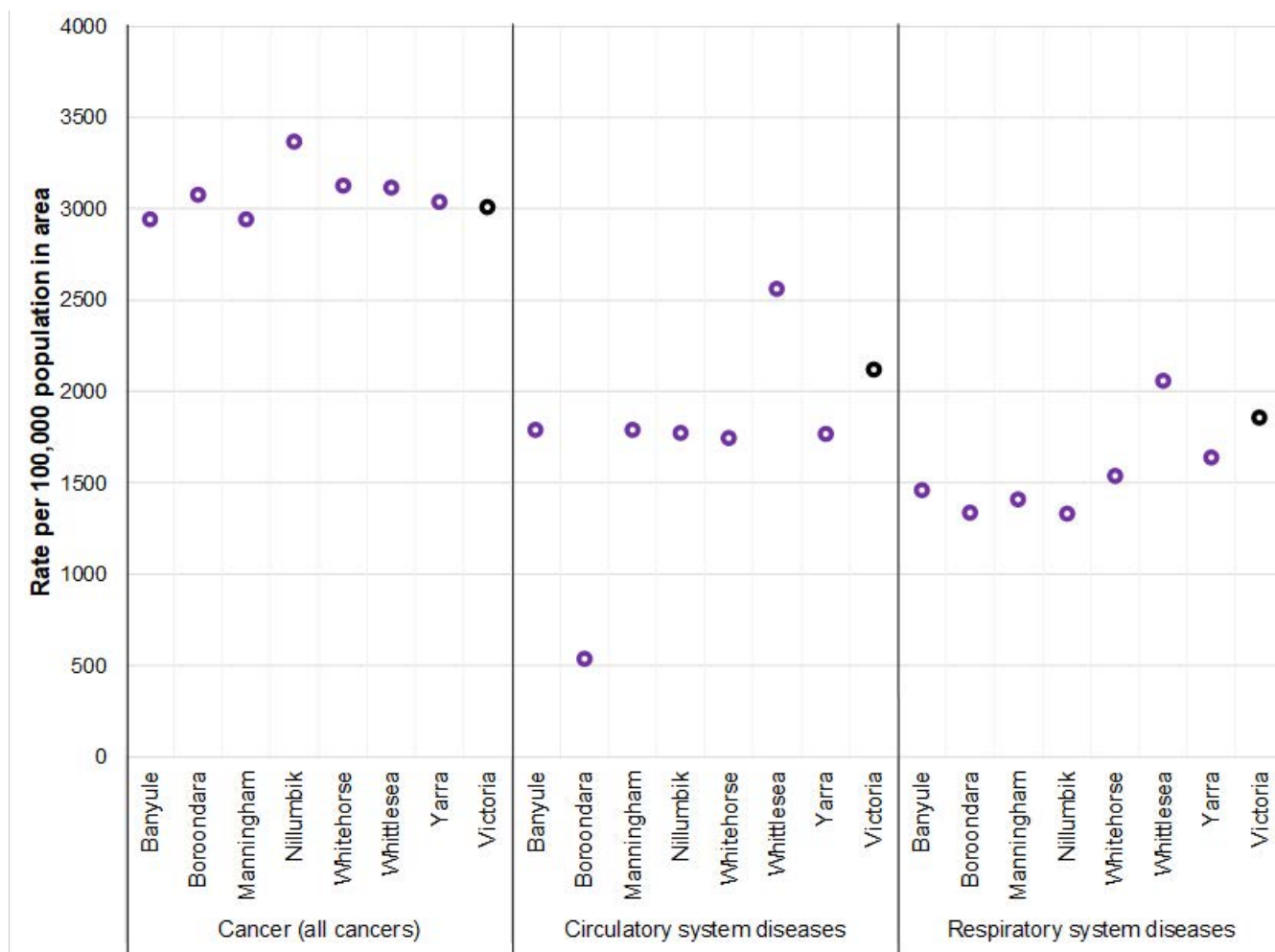


Figure 6.9: Summary of selected hospitalisation rates in LGAs of interest compared with Victoria [age standardised] (20014/15)

More specific data relevant to mortality and hospitalisations in the LGAs and Victoria considered in the health impact assessment is available for data reported for 2010–2014³. **Figure 6.8** and **Figure 6.9** above summarise the reported rates of selected mortality (as premature mortality for ages 0–74 years) and hospitalisation rates for selected diseases in these LGAs compared against that reported for Victoria.

A review of the available data in relation to mortality, the prevalence of disease and hospitalisation rates for the LGAs evaluated indicates that:

- For Banyule, Boroondara, Manningham, Nillumbik and Whitehorse the prevalence of premature deaths (avoidable, circulatory, respiratory and lung cancer) is significantly lower than that reported for Victoria.
- With the exception of Whittlesea, the rate of circulatory system and respiratory system diseases are lower in the identified LGAs than for Victoria as a whole.
- The rate of hospital admissions for cancer in the identified LGAs is similar to those reports for Victoria as a whole, with the exception of Nillumbik.

More specific health indicators expected to be considered in the health impact assessment were obtained from DHHS and other published sources. These data sets relate to specific health indicators for mortality and hospitalisations relevant to impacts that may be related to exposures to air pollution from combustion sources and from noise. The data relates to different age groups where relationships have been established between exposure and changes in health outcomes.

Table 6.4 presents the available data for the LGAs evaluated in this assessment, the Melbourne Metropolitan Area and Victoria.

Section 6.4 and **Table 6.5** provide further discussion and data relevant to asthma. It is noted that data is not available for all the areas listed in the table.

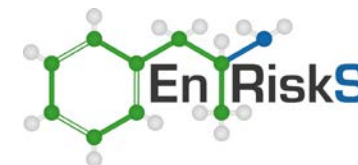
A review of **Table 6.4** indicates the rate of mortality (for all indicators listed in the table) are lower in the Nillumbik, Whittlesea and Yarra LGAs compared with Banyule, Boroondara, Manningham and Whitehorse LGAs as well as Melbourne and Victoria. This may be in part due to the low proportion of people ages 65 years and older in these LGAs compared with the other LGAs as well as Melbourne and Victoria.

The rate of antidepressant medication prescriptions is an indicator that can be used to review changes in stress and anxiety levels within a community. While these data were not directly used in the health impact assessment, to evaluate specific impacts, the data is relevant to assist in ongoing monitoring of potential indicators of changes that increase or decrease stress and anxiety in the community. In relation to the rate of medication prescriptions for antidepressants presented in **Table 6.4**, the rate for the LGAs evaluated are lower than for Victoria, with the exception of Nillumbik and Whitehorse LGAs.

³ Social Health Atlas of Australia: Victoria Local Government Areas, published 2018, <http://phidu.torrens.edu.au/social-health-atlases/maps#social-health-atlases-of-australia-local-government-areas>

Table 6.4: Summary of key health indicators considered in the health impact assessment

Health indicator	Data available for population (rate per 100,000 population*)								
	Banyule LGA	Boroondara LGA	Manningham LGA	Nillumbik LGA	Whitehorse LGA	Whittlesea LGA	Yarra LGA	Greater Melbourne Metropolitan Area*	Victoria
Mortality									
All causes – all ages	634 ¹	617 ¹	650 ¹	362 ¹	653 ¹	422 ¹	373 ¹	530 ¹	599 ¹
All causes (non-trauma) ≥30 years	--	--	--	--	--	--	--	949 ²	--
All causes ≥30 years	1000 ¹	1027 ¹	995 ¹	597 ¹	1061 ¹	724 ¹	610 ¹	880 ¹ , 984 ²	980 ¹
Cardiopulmonary ≥30 years	5.6 ¹	4.5 ¹	2.6 ¹	1.6 ¹	4.8 ¹	2.2 ¹	2.3 ¹	3.8 ¹ , 3.75 ²	4.3 ¹
Cardiovascular – all ages	138 ¹	141 ¹	143 ¹	80 ¹	135 ¹	82 ¹	79 ¹	113 ¹ , 184 ²	131 ¹
Respiratory – all ages	59 ¹	54 ¹	57 ¹	27 ¹	69 ¹	42 ¹	34 ¹	51 ¹ , 49 ²	59 ¹
Morbidity									
Cardiovascular disease hospitalisations									
All ages	2127 ⁶	1870 ⁶	2437 ⁶	1539 ⁶	2338 ⁶	2008 ⁶	1296 ⁶	2010 ⁶	2212 ⁶
>65 years	8643 ⁶	8634 ⁶	8858 ⁶	6825 ⁶	10079 ⁶	10939 ⁶	7684 ⁶	9616 ⁶	9617 ⁶
Respiratory disease hospitalisations									
All ages	1561 ⁶	1360 ⁶	1504 ⁶	1093 ⁶	1667 ⁶	1724 ⁶	1193 ⁶	1615 ⁶	1753 ⁶
>65 years	3964 ⁶	3944 ⁶	3828 ⁶	2515 ⁶	5068 ⁶	5257 ⁶	4106 ⁶	4720 ⁶	1615 ⁶
Stroke hospitalisations	--	--	--	--	--	--	--	--	132 ⁵
Ischemic heart disease hospitalisations	--	--	--	--	--	--	--	--	537 ⁵
Prevalence of hypertension ≥18 years	21960 ³	19276 ³	20045 ³	23781 ³	23036 ³	20840 ³	27800 ³	22872 ³	22881 ³
Mental health									
Number of prescriptions for antidepressant medication per 100,000 people									
17 years and under	7495 ⁴	7166 ⁴	5785 – 8446 ⁴	9596 ⁴	8482 – 8932 ⁴	4327 ⁴	6191 ⁴	--	7789 ⁴
18 to 64 years	96189 ⁴	77202 ⁴	66913 – 84930 ⁴	101513 ⁴	78778 – 87802 ⁴	83225 ⁴	88414 ⁴	--	99,774 ⁴
65 years and over	179481 ⁴	161183 ⁴	157576 – 167902 ⁴	202132 ⁴	165468 – 176872 ⁴	182557 ⁴	174144 ⁴	--	194,225 ⁴



Notes for Table 6.4:

Annual rate where relevant to 1 year of data, or an average annual rate is presented where relevant to more than 1 year of data

- 1 Data provided by AIHW for the years 2012-2016. The values present the average crude rate reported over the 5 years from 2012-2016.
- 2 Data for the Greater Melbourne Metropolitan area for 2010 based on hospital statistics and population data (both for 2010) used in review of exposure and risks to inform recommendations for updating the National Environment Protection Measure (NEPM) Ambient Air Quality (AAQ) (Golder 2013)
- 3 Calculated crude rate – data relevant to the years 2014-2015 from the Social Health Atlas of Australia, Victoria (as published April 2018)
- 4 Age standardised rate (as data is only being used for comparison between areas) – data provided in the Australian Atlas of Healthcare Variation for the years 2013-2014 <https://www.safetyandquality.gov.au/atlas/atlas-2015/atlas-2015-downloads/>
- 5 No data on hospitalisations accessible for the study area, so data available for hospitalisations for coronary heart disease (ischemic heart disease) and stroke (as a principal diagnosis) in Major cities 2012-2013 has been adopted, as an age-standardised rate (Australian Institute of Health and Welfare 2014)
- 6 Data provided by DHHS for the years 2012/13 – 2016/17. The values presented relate to the average hospitalisations reported over the 5 years and annualised rate with 2016 census population data.
- No data available

Values in **bold** are those adopted in the quantitative assessment of health impacts

6.4.4 Incidence of asthma

Impacts of changes in air quality associated with vehicle emissions on asthma in the community are encompassed within the broader evaluation of respiratory effects. However, asthma is commonly identified as a key health impact of concern in relation to vehicle emissions. The health impact assessment therefore also included a more specific evaluation of changes in air quality related to the project on asthma.

The impact of air pollution on asthma has been the subject of a review by the Australian Institute of Health and Welfare (AIHW, 2010). This review makes it clear there are multiple contributors to the exacerbation of asthma in any individual, as illustrated in **Figure 6.10**, so that isolating any one single factor is very difficult.

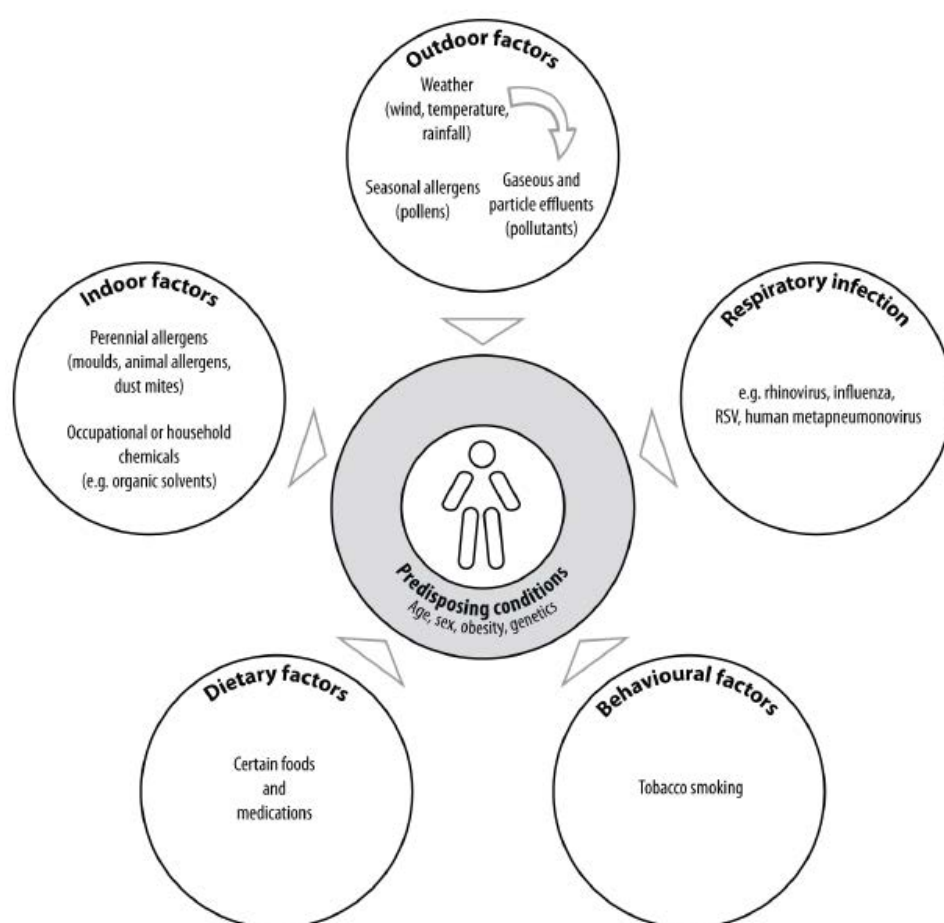


Figure 6.10: Asthma risk factors (AIHW 2010)

For children other factors include (AIHW, 2009):

- Prenatal and postnatal factors such as maternal smoking, mode of delivery, prematurity, multiple births and breastfeeding
- Early childhood exposures such as bronchitis, reduced physical activity, siblings, child care attendance and pet ownership.

In children, asthma is the most common long-term medical condition with the prevalence higher amongst boys than girls. It is estimated that 20.8 per cent of Australian children aged 0–15 years have ever been diagnosed with asthma, while 11.3 per cent of children in the same age group have a current diagnosis. The rate of current asthma in Melbourne, including the Eastern Melbourne Area (that includes the LGAs relevant to this project), presented in **Table 6.5** is consistent with the national rate. No more specific data is currently available on the asthma rates in the specific LGAs.

Table 6.5 sets out data relevant to the rate of asthma in children, asthma hospitalisations, rate of medication prescriptions issued to manage asthma and children with asthma management plans.

Hospitalisation and medication data related to asthma are presented in **Table 6.5**:

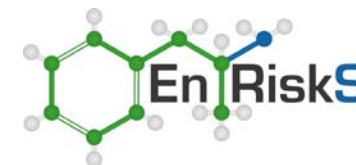
- The rate of asthma hospitalisations for children and adolescents aged 3–19 years reported in the LGAs evaluated are lower than reported for Victoria.
- The rate of asthma hospitalisations for children aged 0–8 years is lower than the state average in the LGAs evaluated with the exception of Yarra LGA.
- The rate of asthma hospitalisations for adolescents aged 10–17 years is lower than the state average in the LGAs evaluated with the exception of Whittlesea and Yarra LGAs.
- The rate of asthma medication prescriptions issued for children and adolescents aged 3–19 years in the LGAs evaluated is below the state average with the exception of Yarra LGA.
- The rate of hospital admissions for adults aged 20–44 years in the LGAs evaluated is lower than the state average with the exception of Whittlesea LGA.
- The rate of asthma medication prescriptions issued for adults aged 20–44 years in the LGAs evaluated is below the state average with the exception of Nillumbik LGA.

In relation to asthma prevalence, the available data indicates for children aged 5–6 years and at school entry, the rate of current asthma in the evaluated LGAs is lower or similar to the Victorian average. This is also the case for adolescents aged 12–17 years with the exception of Nillumbik and Whitehorse LGAs, which are slightly higher.

The data on asthma prevalence in each LGA relates to data from throughout the LGA, or from all schools located in the LGAs. No data is available that enables more detailed analysis of asthma prevalence in areas closer to existing major roadways.

Table 6.5: Summary of asthma indicators to be considered in health impact assessment

Health indicator	Data available for population (rate per 100,000 population*)								
	Banyule LGA	Boroondara LGA	Manningham LGA	Nillumbik LGA	Whitehorse LGA	Whittlesea LGA	Yarra LGA	Greater Melbourne Metropolitan Area*	Victoria
Asthma hospitalisations, including related respiratory admissions (3–19 years)	231 ²	192 ²	179 – 210 ²	190 ²	201 – 213 ²	236 ²	306 ²	--	310 ²
Asthma hospitalisations (0–8 years)	710 ⁵	608 ⁵	506 ⁵	470 ⁵	776 ⁵	688 ⁵	878 ⁵	--	778 ⁵
Asthma hospitalisations (10–17 years)	70 ⁵	52.5 ⁵	84 ⁵	90 ⁵	100 ⁵	118 ⁵	114 ⁵	--	110 ⁵
Asthma medication prescriptions (3 to 19 years)	22,234 ²	17,223 ²	18,131 – 19,036 ²	19,299 ²	20,067 – 23,047 ²	21,209 ²	25,095 ²	--	23,810 ²
Asthma hospitalisations (20–44 years)	73 ²	51 ²	58 ²	48 ²	55 – 65 ²	95 ²	74 ²	--	87 ²
Asthma medication prescriptions (20 –44 years)	18,551 ²	15,850 ²	15,528 – 19,437 ²	20,284 ²	14,826 – 18,255 ²	16,019 ²	15,415 ²		19,496 ²
Asthma emergency department hospitalisations (1–14 years)	1206¹	705¹	757¹	857¹	797¹	1027¹	1491¹	1095 ¹	1110 ¹
Asthma prevalence (current) for children									
All ages	General: 11.5% in Inner East Melbourne Area, 12.3% in Outer East Melbourne Area, 10.1% in North East Melbourne Area ³							10.9% ³	11.3% ³
School entry (5-6 years)	10.5% ⁶	9.8% ⁶	11.5% ⁶	12.0% ⁶	9.8% ⁶	12.1% ⁶	11.8% ⁶	11.3% ⁶	11.8% ⁶
12–17 years	11.7% ⁴	11.2% ⁴	9.3% ⁴	14.8% ⁴	13.0% ⁴	9.7% ⁴	6.9% ⁴	--	11.6% ⁴
Proportion of asthmatic children with current written asthma plan									
All ages	General: 67.5% in Inner East Melbourne Area, 60.9% in Outer East Melbourne Area, 72.6% in North East Melbourne Area ³							67.0% ³	67.2% ³
School entry (5-6 years)	67.2% ⁶	62.6% ⁶	60.7% ⁶	70.0% ⁶	56.8% ⁶	55.1% ⁶	54.6% ⁶	58.2% ⁶	57.8% ⁶
12–17 years	40.5% ⁴	40.3% ⁴	40.7% ⁴	32.4% ⁴	28.5% ⁴	18.8% ⁴	46.9% ⁴		32.0% ⁴



Notes for Table 6.5:

- * Annual rate where relevant to 1 year of data, or an average annual rate is presented where relevant to more than 1 year of data
- 1 Data for provided by the DHHS upon request from enRiskS, data presented is the average number of presentations for the 5 years from 2012/13 to 2016/17 and annualised rate with 2016 census population data.
- 2 Data provided in the Australian Atlas of Healthcare Variation for the years 2010-2011 to 2012-2013 <https://www.safetyandquality.gov.au/atlas/atlas-2015/atlas-2015-downloads/>
- 3 Data available from the Victoria Child and Adolescent Monitoring System (VCAMS) for 2013
- 4 Data available for 2008 from the City of Banyule, City of Boroondara, City of Manningham, City of Nillumbik, City of Whitehorse, City of Whittlesea and City of Yarra Early Childhood Profiles (2010) and data for 2009 from the City of Banyule, City of Boroondara, City of Manningham, City of Nillumbik, City of Whitehorse, City of Whittlesea and City of Yarra Adolescent Community Profile (2010)
- 5 Data available for 2004-05 to 2008-09 from the City of Banyule, City of Boroondara, City of Manningham, City of Nillumbik, City of Whitehorse, City of Whittlesea and City of Yarra Early Childhood Profiles (2010) and data for 2005-06 to 2009-10 from the City of Banyule, City of Boroondara, City of Manningham, City of Nillumbik, City of Whitehorse, City of Whittlesea and City of Yarra Adolescent Community Profile (2010) (average of the yearly rates calculated)
- 6 Data available from School Entrant Health Questionnaire, 2016 <https://www.education.vic.gov.au/about/research/Pages/reportdatahealth.aspx>
- No data available
- BOLD** Values in bold are those adopted in the quantitative assessment of health impacts

6.5 Overview of existing community and health

The overall demography and health of the broader community is generally consistent with or better than the Melbourne Metropolitan area and Victorian population. However, at a local level there are existing community concerns relating to air quality and noise impacts on the health of residents and school children in areas located close to existing major roadways.

It is expected that given the general health of this community, at a broad scale, the health of the local community may not be particularly sensitive to changes associated with the project that may cover a period of less than a year to a number of years (such as would occur during construction). However, there may be health benefits from the long-term redistribution of transport, and transport related emissions related to the operation of the project. This is further evaluated in this report.

7. EES risk assessment

A risk assessment of project activities was performed in accordance with the methodology described in **Section 5.3**. The risk assessment has been used as a screening tool to prioritise the focus of the impact assessments and development of Environmental Performance Requirements (EPRs). The risk pathways link project activities (causes) to their potential effects on the environmental assets, values or uses that are considered in more detail in the impact assessment. Risks were assessed for the construction and operation of the project.

The identified risks and associated residual risk ratings are listed in **Table 7.1**. The likelihood and consequence ratings determined during the risk assessment process and the adopted EPRs are presented in **Appendix I**.

Table 7.1: Health impact assessment EES risks

Risk ID	Potential threat and effect on the environment	Risk rating
Construction		
Risk HE01	Noise and vibration emissions from construction activities affecting the health of the local community	Low
Risk HE02	Dust generated and emissions to air from construction equipment and construction activities affecting the health of local community	Low
Risk HE03	Inappropriate handling, stockpiling, transport and treatment/disposal of contaminated soil resulting in exposure and potential health impacts to the local community	Low
Risk HE05	Changes within the community such as the altered access or connectivity to/between recreational areas, community facilities, commercial premises and active transport infrastructure resulting in potential implications to public safety and wellbeing of individuals during construction	Low
Risk HE06	Changes within the community from the temporary use of some green space areas for construction resulting in impacts on the health and wellbeing of the community and permanent loss of some green space during operations.	Low
Operation		
Risk HE07	Noise generated by the redistribution of traffic due to operation causes an increase in noise potentially affecting the health and amenity of the local community. In particular these impacts may increase annoyance affecting cognitive function in schools and workplaces, or increase sleep disturbance causing an increase in hypertension and increased risks of cardiovascular morbidity or premature mortality	Medium
Risk HE08	Tunnel operation leading to higher in car pollutant concentrations and exposures by occupants using the tunnel	Low
Risk HE09	Redistribution of traffic on surface roads during operation changes ambient pollutant levels (particulate matter, nitrogen dioxide, carbon monoxide, volatile organic compounds and polycyclic aromatic hydrocarbons) and emissions from tunnel ventilation structures potentially impacting on the health of the surrounding the community. Health effects may include respiratory and cardiovascular effects morbidity or premature mortality	Medium
Risk HE10	Changes within the community, such as altered access or connectivity to/between recreational areas and community facilities and active transport infrastructure may have implications to public safety and the wellbeing of individuals from the operation of the project	Low
Risk HE11	Changes within the community from the permanent loss of green space and tree canopy areas resulting in impacts on the health and wellbeing of the community.	Planned

8. Assessment of air quality impacts on health

8.1 Approach

This section assesses the potential for changes in air quality due to the project and how these changes might impact health within the community. This assessment has drawn on information provided in EES Technical report B – Air quality and in some areas summarises key (and relevant) aspects. All details relevant to the underlying assumptions, methodology and interpretation of impacts relevant to changes in air quality are provided within EES Technical report B – Air quality. Where more detail than provided in the health impact assessment is required, the reader is directed to the EES Technical report B – Air quality.

The characterisation of health impacts from changes in air quality due to the project is complex.

This section provides an overview of the key aspects of the air quality impact assessment and an assessment of potential health impacts associated with the predicted changes in air quality in the local community. The assessment includes:

- Information on existing traffic conditions (sourced EES Technical report B – Air quality), presented in **Section 8.2**
- Information on existing air quality (EES Technical report B – Air quality), presented in **Section 8.3**
- Summary of air quality impact assessment (EES Technical report B – Air quality), presented in **Section 8.4**
- Assessment of construction impacts on health, presented in **Section 8.5**
- Detailed assessment of the individual identified air quality parameters (exposure and potential impacts), presented in **Sections 8.6 to 8.9**
- Discussion of health issues in relation to in tunnel air quality, presented in **Section 8.10**.

The air quality impact assessment evaluated incremental changes in the relevant air quality parameters (that is, changes in concentrations due to the project alone) and cumulative (that is, background plus project) changes, which are those from the project added to the background air quality in the project area. Both the incremental and cumulative changes, relevant to the operational phase of the project, were used for the health impact assessment to assess potential impacts to health.

The assessment of health impacts associated with the operation of the project involves the quantification of health risks and impacts.

The quantification of health impacts from changes in air quality requires the use of a few different approaches to address the range of air pollutants relevant to this project:

- **Use of health-based air guidelines:** For air pollutants where there is a threshold for acute and chronic effects (that is, a level below which there are no health impacts), published health-based guidelines have been identified and used in this assessment. The assessment of health impacts has focused on the maximum impacted locations and compared the predicted concentration of these air pollutants in air (from the project as well as other urban sources) with the air guideline. Where the exposure concentration is less than the air guideline there is no risk. This approach applies to a number of air toxics (discussed further in **Section 8.6**) as well as carbon monoxide (discussed further in **Section 8.7**).
- **Calculation of an incremental lifetime cancer risk:** For air pollutants that are considered to be genotoxic carcinogens, there is no threshold. The approach therefore adopted for the assessment of these chemicals is to calculate an incremental lifetime cancer risk, utilising published non-threshold inhalation toxicity reference values (or unit risk values) and an estimation of the maximum increase in air concentration (or exposure) within the community. This results in the calculation of an incremental carcinogenic risk and utilises commonly used risk assessment methods as outlined by enHealth (enHealth, 2012a).

As the methodology adopted for the assessment of an incremental carcinogenic risk is commonly used in risk assessments, there are a range of existing guidance where acceptable risk levels have been determined for population-wide exposures (relevant to establishing drinking water guidelines]).

For this assessment, acceptable risks are those where the incremental carcinogenic risk is $\leq 1 \times 10^{-6}$

While this has been applied to the maximum impact from the project (that is, localised area) the acceptable risk level is consistent with that adopted for population-wide assessments.

This approach applies to the assessment of community exposure to benzene, 1,3-butadiene, carcinogenic polycyclic aromatic hydrocarbons and diesel particulate matter (as discussed further in **Section 8.6**).

- **Calculation of impacts, risks and health burden, of changes in nitrogen dioxide and particulate matter:** The data available on health impacts from exposure to nitrogen dioxide and particulate matter, particularly within urban air environments, comes from large population or epidemiological studies. These studies enable relationships between exposure and various health effects (specifically mortality [that is, a shortening of life-span] and morbidity effects). These concentration-response or exposure-response relationships are developed based on large population exposures and are utilised in the assessment of population health, and for establishing ambient (population-wide) air guidelines. These relationships are not developed for the assessment of specific sources or localised impacts, as is the case for the assessment of impacts from the project.

North East Link would involve the construction of new roadway infrastructure that would redistribute traffic within the community, rather than create a new source of traffic. As a result, vehicle and truck emissions within the broader community remain much the same which makes the conduct of community or larger population-wide assessments of health impacts difficult as the overall health impact is expected to reflect the small change in total vehicle movements. However, as traffic is more locally redistributed it is important to also evaluate the potential significance of this redistribution, particularly localised increases in exposure. While this may only affect a small number of households, increases in risk associated with these maximum changes also need to be considered. This assessment has therefore considered community health impacts, to inform the assessment of the overall health burden of the project, as well as localised health impacts, to inform management decisions in relation to the magnitude of localised impacts.

Community/population health impacts have been assessed on the basis of the overall change in population risk (within the relevant LGAs) and health incidence (change in the number of cases). There is very limited guidance available in relation to acceptability of community risks associated with changes in airshed concentrations of nitrogen dioxide and particulate matter (refer to **Appendix D** for further discussion). However for the purpose of this assessment, guidance available from the NEPC relevant to the assessment of population exposures to air pollutants (including nitrogen dioxide and particulate matter) indicates the estimated risk from population exposures should not exceed 1 additional case per 100,000 of the population per year (NEPC, 2011). The following has therefore been adopted:

For this assessment, acceptable population risk for nitrogen dioxide or particulate matter is $\leq 1 \times 10^{-5}$

Localised health impacts have also been calculated to assess the potential significance of maximum increases in nitrogen dioxide and particulate matter due to the localised redistribution of traffic. As this is a localised impact it is not possible to calculate an increased population incidence and the calculation of risk relates to a maximum localised risk, not a population risk. Due to the limitations of applying the exposure-response functions to localised impacts, these localised risks are considered to only be semi-quantitative. There is no guidance available for the assessment of localised risks for changes in nitrogen dioxide or particulate matter.

Appendix D provides additional discussion in relation to determining various risk levels. Based on the discussion provided in **Appendix D**, and consideration of the need to determine an action level for the management of localised impacts, a risk management level that is equal to the level at which risks are considered unacceptable has been adopted in this assessment as follows:

For this assessment, the risk management level for localised risk $\geq 1 \times 10^{-4}$

Calculated population risks and localised risks for changes in nitrogen dioxide and particulate matter are presented in **Section 8.8** and **Section 8.9**.

The assessment of health impacts from changes in air quality has utilised outputs from the air quality modelling that are presented in EES Technical report B – Air quality. In addition, the health impact assessment has also utilised predicted annual average data for all receptors considered in the air quality modelling. These additional data have been provided from the air quality modelling for the health impact assessment.

8.2 Existing traffic

Melbourne's north-east currently relies on a relatively sparse arterial road network, which lacks the grid-based resilience found in the eastern and inner suburbs. Trips heavily rely on a limited number of arterial roads, such as Bell Street for east-west movements and Rosanna Road and Fitzsimons Lane for north-south movements.

The Yarra River cuts diagonally through the study area and its presence is a barrier to north-south movements throughout Melbourne's north-east. This means the north-eastern arterial road network is generally congested in peak periods, with poor travel time reliability and network resilience.

The area primarily includes residential dwellings, with some commercial and retail clusters located around major activity centres. Large industrial precincts are also located in Preston, Heidelberg West, Reservoir, Somerton and Epping which generates demand for truck movements to and from these regions. Further to the north-east, the urban growth boundary has been administered to protect parklands along the Yarra River and to promote rural conservation. Residential zoning along Melbourne's north-eastern fringe has already expanded to the edges of the Urban Growth Boundary, and so further development and densification in these areas is anticipated to be limited.

Household travel surveys indicate that private vehicles are the dominant mode of transport in the north-east, responsible for 84 per cent of all trips. Public transport mode share accounts for approximately 14 per cent of travel demand, while walking and cycling trips make up approximately 2 per cent.

Freight demand through the study area is underpinned by a concentration of industrial precincts in the south-east and north, which generates movements through the north-east. Local demand is also generated by retail centres and industrial hubs such as Northland and Heidelberg West. Freight in Melbourne's north-east is primarily distributed via heavy vehicles using the local road network. Truck curfews are currently enforced across several arterials in the north-east to reduce truck traffic through the area at night. The curfews initially restricted vehicles in excess of 4.5 tonnes from access to the area at night; however, adjustments have now been made to limit trucks in excess of 16.5 tonnes from the area between 10 pm and 6 am.

Changes in traffic movements associated with the project directly affect air quality and noise within the local community.

8.3 Existing air quality

The existing air quality for the project area is described in EES Technical report B – Air quality.

Regional air quality is influenced by a range of industrial and non-industrial emission sources, including emissions from road traffic. Existing air quality in the vicinity of the proposed North East Link is typical for this urban context. Emissions sources contributing to the local airshed include:

- Traffic using the road network, including the M80 Ring Road and Eastern Freeway
- Industrial and food manufacturing industries
- Domestic fuel combustion (gas, liquid and solid)
- Residential activities (such as lawn mowing and barbecues)
- Paved and unpaved roads
- Windblown dust
- Burning (fuel reduction, regeneration, agricultural, wildfires).

Regional air quality in the local area has been characterised on the basis of available data from the EPA Victoria air quality monitoring station located in Alphington. Air quality as reported at the EPA Victoria Alphington air quality monitoring station was generally considered to be good. It is noted that at times there were exceedances of the air quality objectives for PM_{2.5} and PM₁₀ in the data collected from period 2013 to 2017, principally due to bushfires, planned burns and domestic wood heaters (in cold calm conditions).

Air toxics data has been collected from a number of locations adjacent to major roadways. These data have been considered in the identification of representative background or existing concentrations within the community.

8.4 Overview of air quality assessment

8.4.1 General

The assessment of air quality impacts associated with the project is presented in EES Technical report B – Air quality. The assessment evaluated changes in air quality in the local community due to emissions from the tunnel ventilation structures, and changes in emissions from traffic on major roadways in the project area.

8.4.2 Construction

Construction-related emissions for large road and tunnel projects are complex due to the range, type and number of activities, the geographical extent over which these activities occur and the intensity and duration. As a result, the assessment of air quality impacts (refer EES Technical report B – Air quality) associated with construction activities was undertaken on a qualitative basis.

Potential air quality impacts associated with project construction primarily relate to:

- Dust emissions from construction activities, which may include wind-generated dust from disturbed soil or dust associated with a range of surface works. Dust generated during construction has the potential to pose:
 - A nuisance, where the larger particles settle out and deposit on surfaces in the community
 - A potential health issue, where particles that are small enough enter the respiratory system.
- Odorous emissions due to asphalt sealing of constructed roads – while these impacts are more of a nuisance and amenity issue, it is noted that some individuals find odours offensive and may feel unwell when exposed to these odours.
- Emissions from diesel-fuelled construction vehicles and earth moving machinery – emissions from this equipment are related to fuel combustion, which have the potential to impact health.

8.4.3 Operations – tunnels

Emissions to air from the North East Link tunnel ventilation structures were modelled using an air dispersion model (AERMOD) approved for use by EPA Victoria. The modelling calculated changes in air quality over a grid, comprising two inner grids each 2.5 kilometres by 2.5 kilometres (6.25 square kilometres) centred around the northern and southern ventilation structures with a 25-metre grid resolution, and an outer larger grid of 10 by 15 kilometres (150 square kilometres) with a 100-metre resolution. The modelled grids are shown in **Figure 8.1**.

The modelling also included the local topography, the presence of buildings near the ventilation structures and the local meteorology. The specific design features of the tunnel ventilation structures were considered, specifically the height and diameter of the structures, their exit discharge velocity, temperature and concentrations (from vehicle emissions within the tunnels).

In addition, impacts of nitrogen dioxide and particulate matter associated with emissions from the ventilation facilities have been predicted at a number of selected, representative, sensitive receptors. The sensitive receptors considered in this assessment relate to child care, schools, aged care and hospital/medical facilities in the study area. Additional representative sensitive receptors considered are listed in **Table 8.1**.

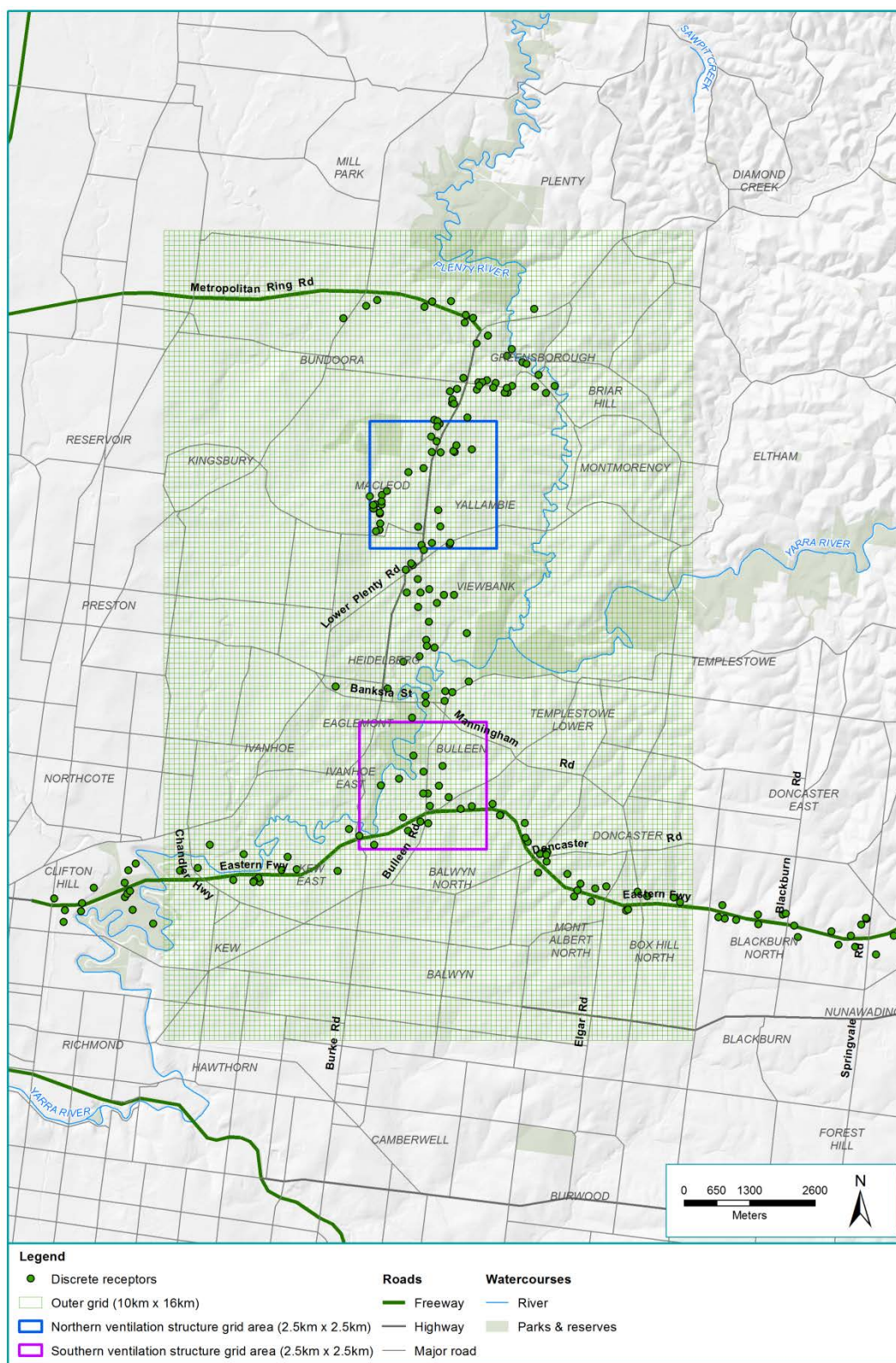


Figure 8.1: Air modelling domain (and receptor grid) and sensitive receptors (from Golder, 2019)

Table 8.1: Additional sensitive receptors (for assessment of nitrogen dioxide and particulate impacts from ventilation structures)

Sensitive receptor	Type of receptor
Northpark Private Hospital	Hospital
Greensborough Preschool	Child care
Abacus Child Care Centre	Child care
Kalparrin Early Intervention Program child care	Child care
Diaverum Diamond Valley Dialysis Clinic	Hospital
Greensborough Maternal and Child Health	Hospital
St Mary's School	School
Greensborough College	School
Greensborough Primary School	School
Watsonia Occasional Child Care Centre	Child care
MS Society Retirement Village	Aged care
Concord School	School
Watsonia Primary School	School
Greensborough Road Early Learning and Kinder	Child care
Macleod Preschool	Child care
Baptcare Strathalan Macleod	Aged care
Regis Macleod	Aged care
Macleod Maternal and Child Health	Hospital
IDV (including disability accommodation)	Care facility
Goodstart Early Learning	Child care
Assisi Centre Aged Care	Aged care
Japara Rosanna Views Nursing Home	Aged care
St Martin of Tours Catholic Primary School	School
Banyule Primary School	School
Viewbank College	School
Austin Hospital Precinct	Hospital
Creative Play Early Learning Centre	Child care
Kalker Montessori Centre	Child care
Marcellin College	School
Wonderland Childcare and Kinder	Child care
Applewood Residence	Aged care
Birrilee Primary School	School
Japara Millward Nursing home	Aged care
Heatherwood School	School
Belle Vue Primary School	School

Sensitive receptor	Type of receptor
Royal Talbot Rehabilitation Centre	Medical
Presbyterian Theological College	School
Warekila Preschool	Child care
Thomas Embling Hospital	Hospital
Melbourne Polytechnic	School
Petit Early Learning Journey	Child care

The mix and magnitude of emissions to air from the ventilation structures depends on the mix and volume of traffic using the tunnels, as well as the tunnel design (including gradients within the tunnel). The mix of vehicles included consideration of passenger vehicles (petrol, diesel, hybrid and electric), light commercial vehicles and heavy commercial vehicles. The mix and volume of these vehicles would vary throughout the day. The assessment evaluated hourly emissions from the ventilation structures for the following traffic scenarios:

- Scenario A – Normal operation (3 lanes) projected traffic volume and fleet mix for 2026
- Scenario B – Normal operation (3 lanes) projected traffic volume and fleet mix for 2036.

In addition, a sensitivity analysis was undertaken that considered the tunnels operating at maximum capacity (that is, all 3 lanes of each tunnel operating at maximum capacity for 24 hours for 365 days of the year), emissions occurring at in-tunnel air quality limits (in relation to carbon monoxide and nitrogen dioxide) and an increase in diesel to petrol-fuelled car ratios.

8.4.4 Operation – surface roads

Changes in air quality due to changes in traffic on key roads in the local community were evaluated. The assessment considered changes in traffic volumes and vehicle emissions on a number of local roads as illustrated in **Figure 8.2**.

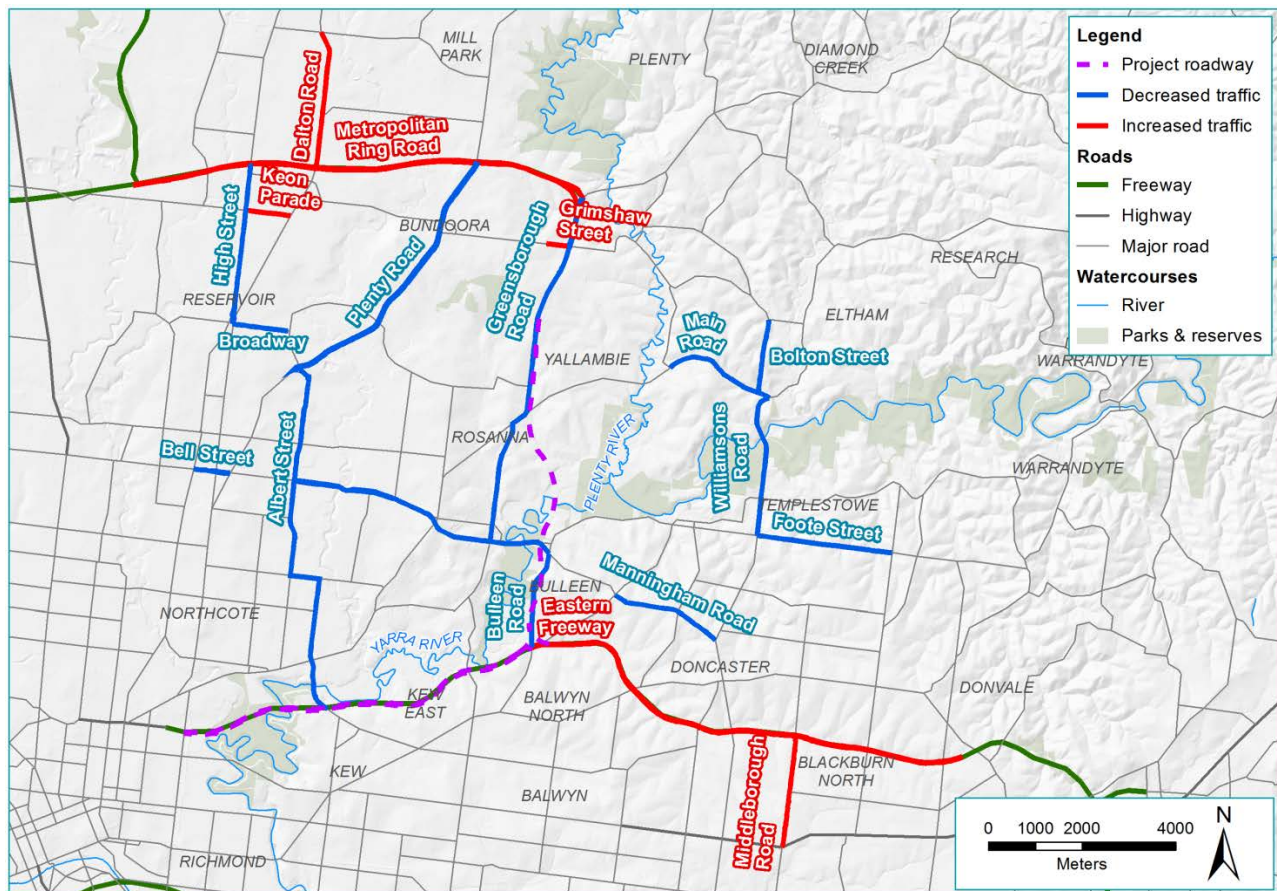


Figure 8.2: Key local roads evaluated in air quality assessment (from Golder, 2019)

The modelling of changes in surface road emissions was undertaken using the AERMOD air dispersion model. The use of this model for assessing impacts from surface road emissions was evaluated by comparing the results to the previous validated model CALRoads and found to be suitable for predicting impacts adjacent to roadways.

The assessment considered existing traffic information (volumes, traffic composition and speed limits) and predicted changes in traffic volumes for the years 2026 and 2036.

Changes in air quality were evaluated at 2,600 sensitive receptor locations located adjacent to the road way at 50 metre intervals along both sides of the road for residences, schools and childcare centres, and at 200 metre intervals if the property was public open space. The receptors modelled are shown in **Figure 8.3**.

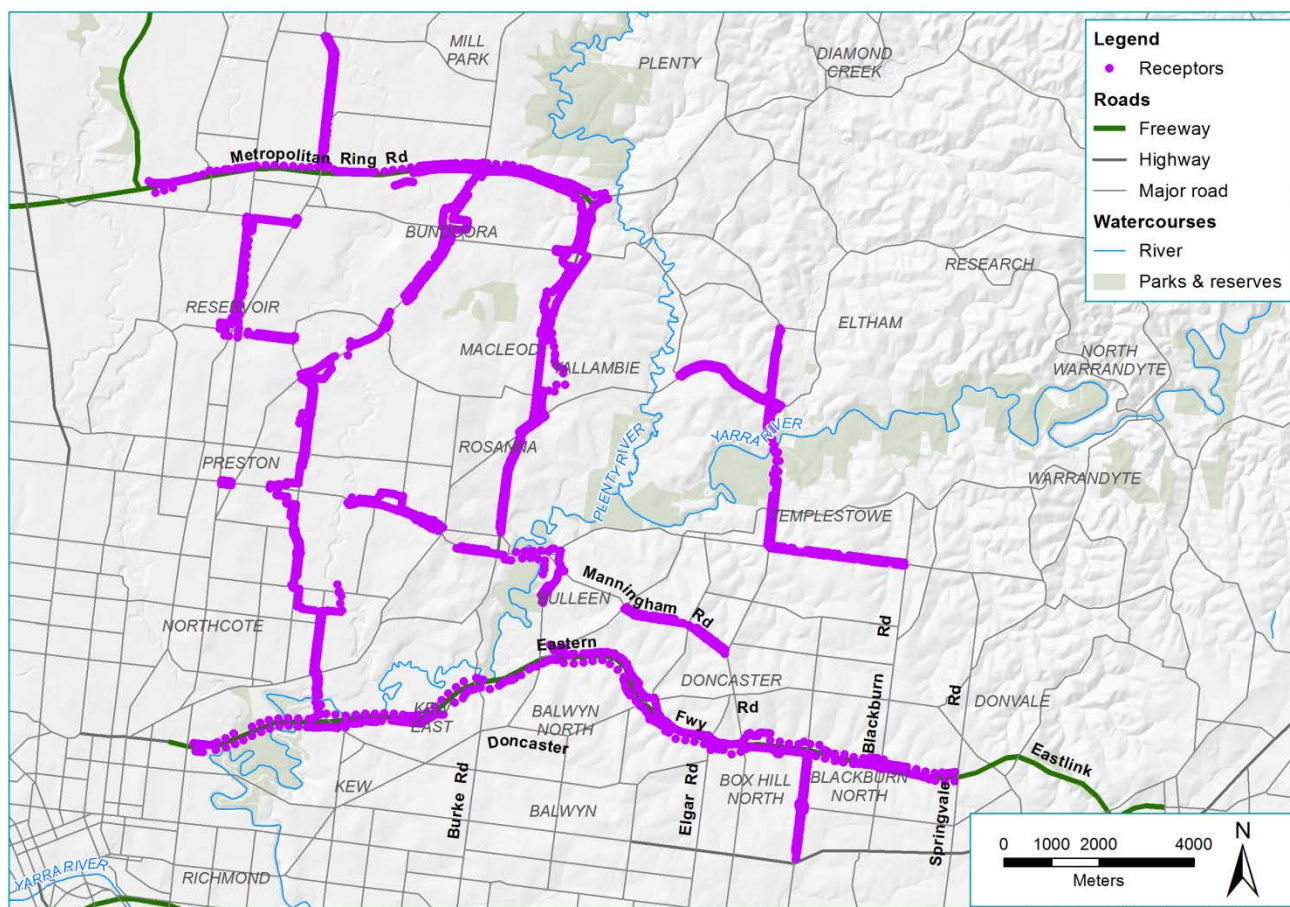


Figure 8.3: Roadside receptors considered in air quality assessment (from Golder, 2019)

8.4.5 Vehicle emissions

When quantifying emissions to air from vehicles, the mix of vehicles that may be present on the roads needs to be considered along with emissions factors that are used to quantify emissions from the various different vehicles.

Fleet volumes and mix:

For 2026, the projected traffic volumes and fleet mix for 2026 were considered. For 2036, the projected traffic volumes and fleet mix for 2036 were considered. The traffic volumes and fleet mix were based on traffic modelling presented in EES Technical report A – Traffic and transport.

Emissions

Vehicle emission factors have been considered for two scenarios: **a conservative scenario (Scenarios A1 and B1), and a more realistic scenario (Scenarios A2 and B2).**

All emissions were calculated using adjusted COPERT Australia vehicle emission factors for the 2010 Victorian fleet and projected diurnal weekday traffic conditions.

For the conservative emissions scenario, the emission factors were based on the 2010 COPERT factors adjusted by World Road Association (PIARC) factors that account for the future year 2020 and road gradient.

For the more realistic emissions scenario, the 2010 COPERT factors adjusted by the relative factors for the Brisbane vehicle fleet in 2010 and 2025 and PIARC road gradient factors.

These emissions estimates (for the conservative and realistic scenarios) remain conservative as:

- Vehicle emission factors used in the air quality impact assessment are considered conservatively high because there is a general trend towards lower emission vehicles (older technology vehicles being replaced over time with newer, improved technology vehicles), with expected improvements in vehicle technology beyond 2020 (conservative emissions scenario) and 2025 (realistic emissions scenario) not accounted for.
- Hybrid and electric vehicles were not considered in the fleet mix. The percentages of these lower emission and zero emission vehicles in the Victorian vehicle fleet are expected to increase significantly in future years.
- Noise walls were not considered to have any effect on pollutant concentrations downwind. There is a significant body of evidence to suggest that noise walls reduce pollutant concentrations immediately downwind of roadways at the most impacted sensitive receptors.

8.4.6 Outcome of air quality impact assessment

Construction impacts

The air quality impact assessment for the project (refer to EES Technical report B – Air quality) evaluated construction impacts using a qualitative approach. The main impacts identified related to the generation of airborne dust, including deposited dust, total suspended particulates and the finer dust fractions of PM₁₀ and PM_{2.5}.

Emissions to air from project construction vehicles and plant equipment are considered minor as they would be intermittent over the duration of construction and spread over a significant area.

Odours may also be generated during some activities, in particular works involving contaminated soil or acid sulfate soil. Odours may be detected close to the source of these materials.

The impacts on air quality arising from construction activities were evaluated as being intermittent, localised and likely to occur over a relatively short and defined period. The implementation of appropriate EPRs would minimise the impacts (including health impacts) on nearby sensitive receptors and the receiving environment (refer to EES Technical report B – Air quality).

These EPRs include:

- EMF2: Environmental Strategy and Management Plans, specifically the Construction Environmental Management Plan (CEMPs) based on EPA Victoria Publication 480, Best Practice Environmental Management: Environmental Guidelines for Major Construction Sites (EPA 1996), developed in consultation with relevant councils, VicRoads, Melbourne Water, EPA Victoria and other authorities as required.
- AQ1: Dust and Air Quality Management and Monitoring Plan. This plan would set out how the project would control emissions of smoke, dust, fumes and other pollution to air, and would also describe the dust management and monitoring system and the mitigation measures and include monitoring requirements for sensitive receptors. Additional mitigation and management measures are outlined in EES Technical report B – Air quality for incorporation into the Dust and Air Quality Management and Monitoring Plan.

It is relevant that health authorities are also consulted for the development of the Dust and Air Quality Monitoring Plan so the measures adequately address potential health concerns.

- CL1: Soil Management Plan. This plan would identify measures to manage spoil and ensure these materials are appropriately managed, monitored and disposed. The plan would include a Remediation Management Plan to address contaminated soil and groundwater encountered during the course of the project.
- Environmental compliance, where the CEMP would be independently reviewed, approved and audited for compliance.

- SC2: Communications and Community Engagement Plan which includes the management of complaints.

Tunnel ventilation structures

The assessment of changes in ground level concentrations associated with emissions from the ventilation structures determined general compliance with the State Environment Protection Policy (AQM). Non-compliances were identified for PM₁₀ (Scenario B1 and B2: 2036) and PM_{2.5} (all scenarios). These non-compliances are due to existing air quality, with the incremental impact associated with the tunnel ventilation structures not considered to provide a significant increase.

Where the sensitivity scenarios are considered (that is, the tunnel is operating at maximum capacity at all times, emissions are always at the in-tunnel air quality limits or there is an increase in the diesel to petrol vehicle ratio), there is relatively little change in the maximum predicted impacts from the ventilation structures.

For more detail refer to EES Technical report B – Air quality.

Surface roads

The assessment of air quality associated with changes in traffic on key local surface roads identified:

- Improvement in air quality adjacent to 15 roads assessed: Albert Street, Banksia Street, Bell Street, Bolton Street, Broadway, Fitzsimons Lane, Grange Road, High Street, Lower Plenty Road, Main Road, Plenty Road, Reynolds Road, Rosanna Road, Station Street and Williamsons Road
- For Manningham Road there would be a mix of changes, with decreases in some pollutant concentrations, but short-term (hourly average) nitrogen dioxide increases of up to 15 per cent
- Increased traffic volumes and thus impacts on air quality adjacent to eight roads: Bulleen Road, Dalton Road, Eastern Freeway, Grimshaw Street, Keon Parade, M80 Ring Road, Middleborough Road and Greensborough Road (North East Link). The maximum increases would generally occur near intersections where contributions from several sources impact on one receptor. The largest increase would occur along the North East Link alignment between Yallambie Road and the M80 Ring Road interchange.

For more detail refer to EES Technical report B – Air quality.

Combined impacts

An assessment of combined impacts associated with emissions from the project's tunnel ventilation structures and changes in surface road traffic was also undertaken.

The combined impacts were assessed at two receptors chosen from the surface road receptors and the discrete receptors used for ventilation structure modelling. The receptors were chosen based on their proximity to the project's surface roads and a ventilation structure such that they would likely

be impacted significantly by both. The chosen receptors in the north and south of the project area corresponded to a surface roads assessment receptor, 450 metres north of the northern portal on Watsonia Street, and land likely to form public open space following completion of the project located approximately 150 metres to the south-east of the southern ventilation structure.

The assessment of changes in air quality identified non-compliances for PM₁₀ (annual average) and PM_{2.5} (24-hour and annual average). These non-compliances primarily relate to background levels, however the combined impact of the project, specifically emissions from surface roads results in an increased number of exceedances of the AEPP (AAQ) objectives, at the maximum impacted locations.

The predicted combined impacts are dominated by surface roads. The contribution from the tunnel ventilation structures is very low. The assessment of health impacts from surface road emissions is therefore expected to be representative of combined exposures within the community.

For more detail refer to EES Technical report B – Air quality.

8.5 Assessment of construction health impacts

If construction impacts are not mitigated or managed, there are a range of potential impacts on community health. Certain air emissions, such as fine particulate matter, can affect the health of residents. While nuisance issues such as the deposition of larger dust (that is, greater than PM₁₀) do not directly impact on health, the deposition of enough dust to be a nuisance can increase stress and anxiety, with the community perceiving the presence of significant amounts of dust may be affecting their health. Odours can also pose a nuisance, with some also considered to be noxious which can make the community feel unwell.

Air quality during construction would be monitored and managed through the implementation of a wide range of planning, engineering and operational controls. This would meet the requirements of EPA Victoria Publication No. 480 – Guidelines for Major Construction Sites. This requires the management of air quality so that impacts to the environment and the health of residents are minimised or eliminated. These are the same guidelines that are required to be followed for any major construction project in Victoria.

Where the EPRs outlined in **Section 8.4.6** above are implemented, impacts on health are expected to be minimised in the community, as is the case with the management of all major construction projects. No further detailed assessment of air quality impacts on health during construction was undertaken.

8.6 Assessment of operational health impacts – air toxics

The air quality impact assessment for the project (refer to EES Technical report B – Air quality) considered emissions of air toxics, specifically BTEX, 1,3-butadiene, formaldehyde and polycyclic aromatic hydrocarbons (PAHs) (as benzo(a)pyrene equivalents) to air from the project.

Most of the volatile organic compounds (VOCs) emitted from vehicles comprise a range of hydrocarbons of low toxicity (such as methane, ethylene, ethane, butenes, butanes, pentenes, pentanes and heptanes) (EPA, 2012). From a toxicity perspective, the key VOCs considered for the

vehicle emissions are BTEX, 1,3-butadiene and formaldehyde (consistent with those identified and targeted in studies conducted in Australia on vehicle emissions (DEH, 2003; EPA, 2012)).

The emission rate of these VOCs is based on the traffic mix assumed for the project and emission rates relevant to the Australian vehicle fleet.

PAHs are predominantly derived from diesel exhausts, with the composition and concentrations dependant on the fuel and type of vehicle. The emission rate of PAHs from vehicles related to the project is based on the traffic mix relevant to the project, and the Australian vehicle fleet using Australian fuel. For this assessment only the conservative emissions estimates for the years 2026 and 2036 have been considered.

In relation to the toxicity of PAHs, this differs significantly for the different individual PAHs that may be present. However, it is common to evaluate PAHs as a group where the PAHs are summed together using toxicity equivalents. Toxicity equivalents are factors that relate the toxicity of an individual PAH to the most well understood and studied PAH, benzo(a)pyrene (BaP). This enables PAHs to then be assessed as a BaP toxicity equivalent concentration using the toxicity and health guidelines relevant to BaP. The assessment of PAHs was thus undertaken on the basis of a BaP toxicity equivalent concentration and using health guidelines for BaP.

In addition to the assessment of potential exposures to PAHs, this assessment has also considered exposure to diesel particulate matter (DPM). DPM includes PAHs, however DPM has been classified as a carcinogen by IARC and it is relevant to also assess exposures to total DPM as well as the sub-set of PAHs.

The assessment of inhalation exposures associated with VOCs, PAHs and DPM has considered:

- Health-based air guidelines and inhalation toxicity reference values (TRVs) for carcinogenic compounds have been selected on the basis of guidance provided by enHealth (enHealth 2012a). It is noted there is no one individual agency/organisation that provides the most robust and current guidelines and TRVs for the compounds considered in this assessment, as the relevant agencies/organisations do not necessarily review all the chemicals and do not update assessments on a regular basis. As a result, the guidelines and TRVs adopted in this assessment come from a number of different sources. The guidelines and TRVs adopted are based on consideration of the available information and reviews provided by relevant key organisations that undertake detailed evaluations of toxicity and determine quantitative values for the assessment of inhalation exposures. This information has been evaluated to determine the most appropriate value that can be used to quantify acute and chronic inhalation exposures. This requires consideration of the hazards identified and the mechanisms for action particularly in relation to the assessment of carcinogenic effects, transparency of the review (that is, is all the information presented and the derivation of the guideline transparent), robustness of the evaluation (that is, critical review and evaluation of all available and relevant studies), currency of the evaluation (including whether more recent key studies were considered) and the application of uncertainty factors.

- For VOCs, PAHs and DPM which are considered genotoxic carcinogens (consistent with guidance provided by enHealth (enHealth, 2012a)) an incremental lifetime carcinogenic risk has been calculated. For the VOCs and PAHs evaluated in this assessment a carcinogenic risk calculation has been adopted for the assessment of maximum potential (incremental) increase in benzene, 1,3-butadiene and PAHs assessed as a benzo(a)pyrene toxicity equivalent (TEQ). In addition, carcinogenic risks associated with exposure to DPM has been assessed. DPM has not been specifically modelled or assessed in the air modelling of vehicle emissions.

For the purpose of this assessment it has been assumed that 80 per cent of PM_{2.5} is DPM (refer to **Appendix C**). The assessment undertaken has adopted the calculation methodology outlined in **Appendix C**, adopting the inhalation unit risk values presented in **Table 8.3**, and assuming the maximum impacts occur at a residential home where individuals are at home 24 hours per day, 365 days of the year and they live at the same house for 35 years (enHealth, 2012b).

- For other VOCs, where the health effects are associated with a threshold (that is, a level below which there are no effects), the maximum predicted concentration of individual VOCs (background plus the change due to the project) associated with the project have been compared against published peer-reviewed health-based guidelines 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 inhalation exposures from all sources and reflect duration of exposure where:
 - Acute guidelines are based on exposures that may occur for a short period of time (typically between one hour or up to 14 days). These guidelines are available to assess peak exposures (based on the modelled one-hour maximum concentration) that may be associated with VOCs in the air and are presented in **Table 8.2**.
 - 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 VOCs in the air and are presented in **Table 8.3**. Use of these values assumes the maximum impact occurs at a residential home where individuals are at home 24 hours per day for 365 days of the year.

Table 8.2: Adopted acute inhalation guidelines based on protection of public health

Compound assessed	Acute health-based guideline ($\mu\text{g}/\text{m}^3$)	Basis
Benzene	580	Acute 1-hour health-based guideline, based on depressed peripheral lymphocytes from the Texas Commission on Environmental Quality (TCEQ) evaluation (TCEQ, 2013a).
Toluene	15,000	Acute 1-hour health-based guideline, based on eye and nose irritation, increased occurrence of headache and intoxication in human male volunteers from TCEQ evaluation (TCEQ, 2013d).
Ethylbenzene	22,000	Acute inhalation guideline, relevant to exposures up to 14 days, based on auditory threshold changes in rats, with conversion to a value relevant to humans from the Agency for Toxic Substances and Disease Registry (ATSDR, 2010). This is more conservative than the acute 1 hour health-based guideline of 86000 $\mu\text{g}/\text{m}^3$ (based on the same health effect in rats) available from TCEQ (TCEQ, 2010).
Xylenes	7,400	Acute 1-hour health-based guideline, based on mild respiratory effects and subjective symptoms of neurotoxicity in human volunteers from TCEQ evaluation (TCEQ, 2013c).
1,3-Butadiene	660	Acute 1-hour health-based guideline, based on developmental effects derived by the California Office of Environmental Health Hazard Assessment (OEHHA, 2013). The guideline developed is lower than developed by TCEQ (TCEQ, 2007) based on the same critical study.
Formaldehyde	100	Acute health-based guideline, based on changes in blink eye response in human volunteers (WHO, 2000b, 2010).

Table 8.3: Adopted chronic guidelines and carcinogenic unit risk values based on protection of public health

Compound assessed	Chronic health-based guideline	Basis
Threshold guidelines		
Benzene	30 $\mu\text{g}/\text{m}^3$	The most significant chronic health effect associated with exposure to benzene is the increased risk of cancer, specifically leukaemia, which is assessed separately (below). The assessment of other health effects (other than cancer) has been undertaken using a chronic guideline derived by the United States Environmental Protection Agency (USEPA) (2002b) based on haematological effects in an occupational inhalation study (converted to public health value using safety factors). This is the most current evaluation of effects associated with chronic inhalation exposure to benzene and is consistent with the value used to derive the NEPM (NEPC, 1999 amended 2013c) health-based guidelines.
Toluene	5,000 $\mu\text{g}/\text{m}^3$	Chronic guideline derived by the USEPA (2005b) based on neurological effects in an occupational study (converted to public health value using safety factors). This is the most current evaluation of effects associated with chronic inhalation exposure to toluene and is consistent with the value used to derive the NEPM (NEPC, 1999 amended 2013c) health-based guidelines.
Ethylbenzene	260 $\mu\text{g}/\text{m}^3$	Chronic guideline derived by ATSDR (ATSDR, 2010) based on nephropathy in rats in an inhalation study, with conversion to a value relevant to humans. This is the most current evaluation of effects associated with chronic inhalation exposure to ethylbenzene.

Compound assessed	Chronic health-based guideline	Basis
Xylenes	220 µg/m ³	Chronic guideline derived by ATSDR (ATSDR, 2007) based on mild subjective respiratory and neurological symptoms in an occupational study (converted to public health value using safety factors).
Formaldehyde	100 µg/m ³	Formaldehyde is classified by IARC as carcinogenic to humans. The guideline developed by the WHO (WHO 2000b, 2010) is considered to be protective of short and long-term exposures, for non-carcinogenic and carcinogenic health effects. Some lower guidelines are available from the US but these are based on approaches to the assessment of carcinogenic effects inconsistent with that adopted by enHealth (enHealth, 2012a) and the WHO (WHO, 2010).
Carcinogenic inhalation unit risk values adopted for carcinogenic risk calculation		
Benzene	6x10 ⁻⁶ (µg/m ³) ⁻¹	Benzene is classified as a known human carcinogen by the International Agency for Research on Cancer (IARC). Inhalation unit risk value is from the WHO (WHO, 2000b, 2010) and is based on excess risk of leukaemia from epidemiological studies.
1,3-Butadiene	5x10 ⁻⁷ (µg/m ³) ⁻¹	1,3-Butadiene is classified as a known human carcinogen by the International Agency for Research on Cancer (IARC). Inhalation unit risk values are available from a number of agencies, including the WHO, USEPA and TCEQ. The most current evaluation has been undertaken by TCEQ (TCEQ, 2013b). This has considered the same studies as the WHO and USEPA, but included more recent studies and more relevant dose-response modelling.
Benzo(a)pyrene TEQ	0.087 (µg/m ³) ⁻¹	BaP is classified by IARC as a known human carcinogen, which relates to BaP as well as all the other carcinogenic PAHs assessed as a BaP toxicity equivalent (TEQ) value. Inhalation unit risk value is from the WHO (WHO, 2010) and is based on protection from lung cancer for an occupational study associated with coke oven emissions. It is noted that carcinogenic risks associated with lung cancer from diesel particulate matter (which is dominated by the presence of carcinogenic PAHs) is also assessed separately.
Diesel particulate matter	3.4x10 ⁻⁵ (µg/m ³) ⁻¹	DPM is classified by IARC as a known human carcinogen. Inhalation unit risk values are available from California (OEHHA, 1998) as well as the WHO (WHO, 1996), with the assessment provided by the WHO considered the more robust. The WHO value, adopted in this assessment is based on data from four different studies where lung cancer was the endpoint.

Table 8.4 and **Table 8.5** present a summary of the maximum predicted 1-hour or annual average concentrations of VOCs assessed by comparison against acute and chronic health-based guidelines (developed using a threshold approach). Calculations associated with the analysis of the situation where the tunnels were operating at maximum capacity are only relevant to the assessment of short-term acute exposures and are therefore not presented for chronic exposures. The tables also present a Hazard Index (HI) which is the ratio of the maximum predicted concentration to the guideline (that is, maximum concentration/guideline). Each individual HI is added up to obtain a total HI for all the threshold VOCs considered. The total HI is a sum of the potential hazards associated with all the threshold VOCs together assuming the health effects are additive, and is evaluated as follows (enHealth, 2012a):

- A total HI less than or equal to one means that all the maximum predicted concentrations are below the health-based guidelines and there are no additive health impacts of concern

- A total HI greater than one means the predicted concentrations (for at least one individual compound) are above the health-based guidelines, or there are at least a few individual VOCs 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.

Table 8.6 summarises calculated incremental lifetime carcinogenic risk associated with chronic exposure to the maximum predicted annual average concentrations of benzene, 1,3-butadiene and carcinogenic PAHs as benzo(a)pyrene TEQ. The calculated carcinogenic risk for these compounds has been summed for benzene, 1,3-butadiene and carcinogenic PAHs, in accordance with enHealth guidance (enHealth, 2012a). The calculated carcinogenic risk for DPM has not been summed as this assessment includes particulate bound chemicals. Summing DPM with the other carcinogenic compounds would result in significant double counting of risks. Incremental carcinogenic risks have been assessed against the criteria discussed in **Section 8.1** (and **Appendix D**).

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

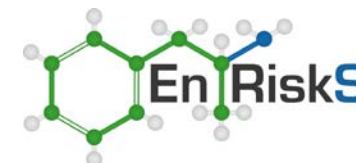


Table 8.4: Assessment of acute exposures to VOCs – maximum impacts in the community

Health based acute guideline - refer to Table 8.1 (µg/m³)	Maximum 1-hr average concentrations predicted - cumulative* (µg/m³)																		Calculated Hazard Index						
	Benzene			Toluene			Ethylbenzene			Xylenes			1,3-Butadiene			Formaldehyde			Benzene	Toluene	Ethylbenzene	Xylenes	1,3-Butadiene	Formaldehyde	Total HI
	580			15000			22000			7400			660			100									
	Project	Background	Total	Project	Background	Total	Project	Background	Total	Project	Background	Total	Project	Background	Total	Project	Background	Total							
Impacts from tunnel ventilation outlets (maximum from all receptors)																									
2026 Conservative: Scenario A	3.1	18.4	21.5	6.3	108	114	2.3	12	14.3	6.1	51	57.1	0.66	1.1	1.76	1.3	12.3	13.6	0.037	0.0076	0.0007	0.0077	0.003	0.14	0.2
2036 Conservative: Scenario B	3.1	18.4	21.5	6.2	108	114	2.3	12	14.3	6.1	51	57.1	0.67	1.1	1.77	1.3	12.3	13.6	0.037	0.0076	0.0007	0.0077	0.003	0.14	0.2
Maximum traffic scenario	5.5	18.4	23.9	11	108	119	3.9	12	15.9	10	51	61	1.5	1.1	2.6	3.1	12.3	15.4	0.04	0.008	0.0007	0.008	0.004	0.15	0.2

* Health based guidelines relate to exposures from all sources, hence the total exposure (background + project) has been evaluated

Background air quality based on Table 33 in the Air Quality assessment report (utilising a factor of 2.5 to convert a 24 hour average to 1 hour average)

Table 8.5: Assessment of chronic exposures to VOCs – maximum impacts in the community

Health based chronic guideline - refer to Table 8.2 (µg/m³)	Maximum annual average concentrations predicted - cumulative* (µg/m³)															Calculated Hazard Index					
	Benzene			Toluene			Ethylbenzene			Xylenes			Formaldehyde			Benzene	Toluene	Ethylbenzene	Xylenes	Formaldehyde	Total HI
	30			5000			260			220			100								
	Project	Background	Total	Project	Background	Total	Project	Background	Total	Project	Background	Total	Project	Background	Total						
Impacts from tunnel ventilation outlets (maximum from all receptors)																					
2026 Conservative: Scenario A	0.047	2.56	2.607	0.095	22.6	22.695	0.035	2.2	2.235	0.093	6.98	7.073	0.022	2.7	2.722	0.087	0.0045	0.0086	0.032	0.027	0.16
2036 Conservative: Scenario B	0.055	2.56	2.615	0.11	22.6	22.71	0.041	2.2	2.241	0.11	6.98	7.09	0.026	2.7	2.726	0.087	0.0045	0.0086	0.032	0.027	0.16

* Health based guidelines relate to exposures from all sources, hence the total exposure (background + project) has been evaluated

Background air quality based on Table 33 in the Air Quality assessment report

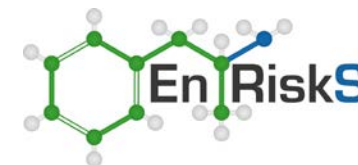


Table 8.6: Assessment of incremental lifetime carcinogenic risk – maximum impacts in community

	Maximum annual average concentrations predicted (ΔC) - incremental from project* ($\mu\text{g}/\text{m}^3$)				Calculated Incremental Cancer Risk**				
	Benzene	1,3-Butadiene	Benzo(a) pyrene TEQ	DPM	Benzene	1,3- Butadiene	Benzo(a) pyrene TEQ	Total risk	DPM
Inhalation unit risk (IUR) - refer to Table 8.2 ($\mu\text{g}/\text{m}^3$) ⁻¹	6.00E-06	5.00E-07	0.087	3.40E-05					
Impacts from tunnel ventilation outlets (maximum from all receptors)									
2026 Conservative: Scenario A	0.047	0.011	1.00E-05	0.059	1E-07	3E-09	4E-07	6E-07	1E-06
2036 Conservative: Scenario B	0.055	0.013	1.00E-05	0.070	2E-07	3E-09	4E-07	6E-07	1E-06

* Calculation of cancer risk relates to an incremental risk from the project

** Calculated as $\Delta C \times \text{IUR} \times 35 \text{ year exposure} / 70 \text{ year averaging time}$ for carcinogens

For the assessment of acute exposures to VOCs (**Table 8.4**), the calculated HI associated with exposure to the maximum concentrations predicted is less than one for all the project scenarios. On this basis, there are no acute risk issues in the local community associated with the project.

For the assessment of chronic exposures to VOCs (**Table 8.5**), the calculated HI associated with exposure to the maximum concentrations predicted is less than one for all the project scenarios. The calculated lifetime cancer risks associated with the maximum change in benzene, 1,3-butadiene and PAHs as benzo(a)pyrene TEQ are less than 1×10^{-6} in relation to all impacts associated with emissions from the tunnel ventilation structures. In addition, the calculated lifetime cancer risks associated with exposure to DPM is equal to 1×10^{-6} . It is noted that where the more realistic emissions estimates are considered in the assessment of DPM, the risk is reduced to 6×10^{-7} . On this basis, the calculated carcinogenic risks are considered acceptable.

There are thus no chronic health risk issues of concern in the local community associated with air toxics or DPM from the project.

On the basis of the assessment undertaken, there are no acute or chronic health risk issues in the local community associated with air toxics or DPM from the project.

8.7 Assessment of health impacts – 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, an 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 from the EPA Victoria (as environmental quality objectives) (EPA Victoria 1999 as varied to 2016) and NEPC (as standards) (NEPC 2016) that are based on the protection of adverse health effects associated with carbon monoxide. The air standards currently available from NEPC are consistent with health-based guidelines currently available from the WHO (WHO, 2005, 2010) and the United States Environmental Protection Agency (USEPA) (2011⁴, specifically listed to be protective of exposures by sensitive populations including asthmatics, children and the elderly). On this basis, the current NEPC standards are considered appropriate for the assessment of potential health impacts associated with the project. It is noted the EPA Victoria State Environment Protection Policy (Air Quality Management) has also adopted a 1-hour guideline available, which is consistent with guidance from the WHO.

⁴ 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>

The NEPC ambient air quality standard for the assessment of exposures to carbon monoxide has considered the lowest observed adverse effect level (LOAEL) and the no observed adverse effect level (NOAEL) associated with a range of health effects in healthy adults, with people with ischemic heart disease and with foetal effects.

In relation to these data, a level of carbon monoxide of nine parts per million (ppm) by volume (or 10 milligrams per cubic metre or 10,000 micrograms per cubic metre) over an 8-hour period was considered to provide protection (for acute and chronic health effects) for most members of the population (NEPC 2016). An additional 1.5-fold uncertainty factor to protect more susceptible groups in the population was included. On this basis, the NEPC (and the EPA Victoria) standard is protective of adverse health effects in all individuals, including sensitive individuals.

The 1-hour criteria of 30 mg/m³ (WHO, 2000d) is consistent with the more recent update from the WHO (WHO, 2010) and the value adopted by EPA Victoria (EPA, Victoria 2001).

Table 8.7 summarises the maximum predicted cumulative (that is, project plus background) 1-hour average and 8-hour average concentrations of carbon monoxide for the assessment years 2026 and 2036, in relation to emissions to air from the ventilation structures for the tunnels. This assessment has considered impacts related to the conservative emissions estimates only.

Table 8.7: Review of potential acute and chronic health impacts – carbon monoxide (CO)

Scenario	Maximum 1-hour average concentration of CO (mg/m³)		Maximum 8-hour average concentration of CO (mg/m³)	
Impacts from tunnel ventilation structures				
	Background	Background plus project	Background	Background plus project
2026: Scenario A1 – all receptors	2.1	2.2	0.43	0.48
2036: Scenario B1 – all receptors	2.1	2.2	0.43	0.48
Maximum capacity – all receptors	2.1	2.3	NA	NA
Relevant health-based standard/ guideline	30		10	

NA – it is not applicable or relevant to assess chronic exposures for the maximum emissions scenario

All the concentrations of carbon monoxide presented in **Table 8.7** are below the relevant health-based standards/guidelines listed at the base of the table.

North East Link would not change the existing health outcomes that relate to exposures in the community to carbon monoxide, either adversely or beneficially. The changes due to the project would not be significant. No adverse health effects are expected in relation to exposures (acute and chronic) to carbon monoxide in the local area surrounding the project.

Carbon monoxide levels are also considered in **Section 8.9** where in-tunnel air quality is discussed, where the potential for health risks due to carbon monoxide may be more relevant.

8.8 Assessment of health impacts – nitrogen dioxide

8.8.1 Approach

Nitrogen oxides (NO_x) 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 (such as gas heating or cooking) combustion sources, are primary producers of nitrogen oxides. The main source of nitrogen oxides in urban areas is from on-road vehicles.

In terms of health effects, nitrogen dioxide is the only oxide of nitrogen that is of concern (WHO, 2000c). 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 (WHO, 2013b). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of nitrogen dioxide (Morgan et al., 2013; NEPC, 2010). The health effects associated with exposure to nitrogen dioxide depend on the duration of exposure as well as the concentration.

Guidelines are available from EPA Victoria (as environmental quality objectives) (EPA Victoria, 1999 as varied to 2016) and NEPC (as standards) (NEPC, 2016) which indicate acceptable concentrations of nitrogen dioxide. These guidelines are based on protection from adverse health effects following short-term (acute) and longer-term (chronic) exposure for all members of the population including sensitive populations like asthmatics, children and the elderly.

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

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

- Whether the assessment of potential health effects associated with exposure to different levels of nitrogen dioxide can be undertaken on the basis of existing guidelines, or whether specific risk calculations are required to be undertaken. The current guidelines in Australia for the assessment of nitrogen dioxide in air relate to cumulative (total) exposures, and adopt criteria that are considered to be protective of short and long-term exposures. It is therefore relevant these guidelines be considered in this assessment.
- In addition, the current standards relate to regional air quality, not localised sources and so use of these standards for the assessment of localised exposures is of limited value.

For these situations, it is relevant to also evaluate the impact on community health of the change in nitrogen dioxide concentration in the local community using appropriate risk calculations. For the conduct of risk assessments in relation to exposure to nitrogen dioxide, the WHO (WHO, 2013b) identified the strongest evidence of health effects related to respiratory hospitalisations and to a lesser extent mortality (associated with short-term exposures) and recommend these health endpoints should be considered in any core assessment of health impacts associated with exposure.

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

8.8.2 Assessment of cumulative exposures

EPA Victoria and 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 micrograms per cubic metre (or 120 parts per billion by volume) is based on a LOAEL of 409–613 micrograms per cubic metre derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of two to protect susceptible people (that is, asthmatic children) was applied to the LOAEL (NEPC, 1998). On this basis, the NEPC (and EPA Victoria) acute guideline is protective of adverse health effects in all individuals, including sensitive individuals.

EPA Victoria environmental quality objectives and the NEPC ambient air quality standard for the assessment of chronic (long-term) exposures to nitrogen dioxide relates to the maximum predicted total (cumulative) annual average concentration in air. The standard of 62 micrograms per cubic metre (or 30 ppbv) is based on a LOAEL of the order of 40–80 parts per billion by volume (around 75–150 micrograms per cubic metre). This relates to the early and middle childhood years when exposure can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent 'colds', a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school.

An uncertainty factor of two was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 parts per billion by volume (38–75 micrograms per cubic metre) (NEPC, 1998). On this basis, the NEPC (and EPA Victoria) standard is protective of adverse health effects in all individuals, including sensitive individuals.

Table 8.8 summarises the maximum predicted cumulative 1-hour average and annual average concentrations of nitrogen dioxide for the conservative as well as realistic emissions scenarios (noting the realistic emissions scenario has only been presented for 2036).

The maximum annual average concentration is the annual average concentration at the maximally affected grid location or individual sensitive receptor. It is noted the maximum change in nitrogen dioxide concentrations is lower under the realistic emissions scenario, when compared with the conservative scenario.

Table 8.8: Review of potential acute and chronic health impacts – nitrogen dioxide (NO₂)

Scenario	Maximum 1-hour average concentration of NO ₂ (µg/m ³)	Maximum annual average concentration of NO ₂ (µg/m ³)		
Impacts from tunnels ventilation structures				
Maximum from all receptors	Background	Background plus project	Background	Background plus project
2026 Conservative: Scenario A1	94	123	19	19.6
2036 Conservative: Scenario B1	94	129	19	19.7
Conservative: Maximum capacity	94	169	NA	NA
2036 Realistic: Scenario B2	94	105	19	19.2
Impacts from changes on surface roads (excluding background*)				
Maximum from all receptors	No project	With project	No project	With project
2026 Conservative	32	69	4.6	8.5
2036 Conservative	37	80	4.9	9.7
2036 Realistic	NA	NA	4.9	8.1
Relevant health-based standard	246		62	

NA – it is not relevant to assess chronic risks for the maximum scenario

* Data as provided for the surface roads. Where cumulative (that is, background plus emissions from surface roads) are required to be considered, a conservative approach would be to add the background (94 µg/m³ for 1-hour averages and 19 µg/m³ for annual averages) to the no project and project estimates. This would result in some double counting of road emissions as the existing background included existing road emissions which are also counted in the no project and project calculations.

For normal operations in 2026 and 2036, and for the conservative and realistic emissions scenarios, all the concentrations of nitrogen dioxide presented in **Table 8.8** are below the acute and chronic NEPC standards.

Changes that occur due to the redistribution of traffic on surface roads results a number of areas where the project results in a reduction in nitrogen dioxide exposures, and others where there is an increase in nitrogen dioxide exposures. The maximum changes in 1-hour average nitrogen dioxide concentrations (for the conservative emissions scenario) at receptors adjacent to each of the key roadways assessed are illustrated in **Figure 8.4**.

To further address potential risks to human health that may be associated with population exposures and localised changes in nitrogen dioxide that relate to the project, incremental risk calculations have been undertaken and are presented in **Section 8.8.3**.

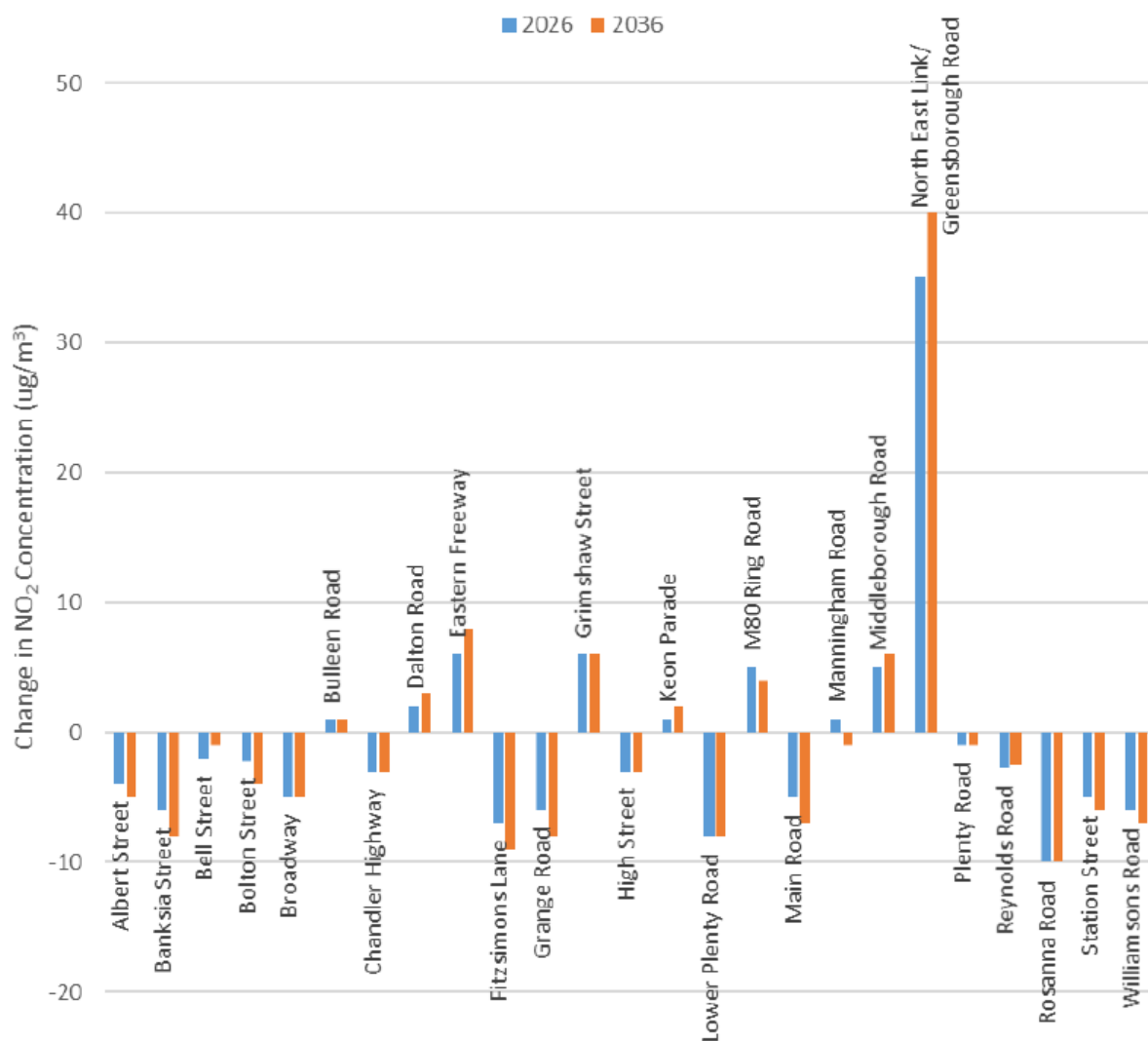


Figure 8.4: Maximum change in 1-hour average nitrogen dioxide concentration for key surface roads based on conservative emissions estimates in 2026 and 2036 (from Golder, 2019)

8.8.3 Assessment of incremental exposures

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

Table 8.9 summarises the health endpoints considered in this assessment, the β coefficient relevant to the calculation of a relative risk (refer to **Appendix C** for details on the calculation of a β coefficient from published studies). The coefficients adopted for the assessment of impacts on mortality and asthma emergency department admissions are derived from the detailed assessment undertaken for the current review of health impacts of air pollution undertaken by NEPC (Golder, 2013) and are considered to be robust.

Table 8.9: Adopted exposure-response relationships for assessment of changes in nitrogen dioxide concentrations

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

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

As discussed in **Section 8.1**, the assessment of health impacts associated with nitrogen dioxide has considered population impacts as well as localised impacts.

Population health impacts

Table 8.10a and **8.10.b** present the calculated population health risks and incidence (that is, increase in the number of cases) relevant to the assessment of population health impacts associated with emissions from the ventilation structures, as well as emissions from the redistribution of vehicles on key surface roads within the project area. The tables present the calculated risks relevant to the consideration of the conservative and the realistic emissions estimates.

The change in incidence across the population for each health indicator relevant to changes in nitrogen dioxide exposures in the local community (for the population exposed) has been calculated on the basis:

- The relative risk has been calculated for a population weighted annual average incremental increase in concentrations. The population weighted average has been calculated on the basis of the smallest statistical division provided by the Australian Bureau of Statistics within a suburb (that is, mesh blocks – which are small blocks that cover an area of approximately 30 to 60 urban residences). For each mesh block in a LGA the average incremental increase in concentration has been calculated and multiplied by the population living in the mesh block (data available from the ABS for the 2016 census year). The weighted average has been calculated by summing these calculations for each mesh block in a LGA and dividing by the total population in the area evaluated (that is, in all the mesh blocks in the LGA evaluated)
- A change in the number of cases associated with the change in nitrogen dioxide impact evaluated in the population within the study area has been calculated (refer to **Appendix C** for details on the methodology). The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered (refer to **Table 6.4** and **Table 6.5**) and the population (for the relevant age groups) present in the suburb (refer to **Table 6.1**).

Calculations relevant to the characterisation of risks associated with changes in nitrogen dioxide concentrations in the community are presented in **Appendix F**.

Figure 8.5 presents a graphical representation of calculated population risks relevant to the assessment of mortality (all causes, all ages).

Review of **Tables 8.10.a and 8.10.b** (and **Figure 8.5**) indicates:

- The calculated population risks where more realistic emissions estimates are considered are lower than presented for the conservative emissions scenario
- All calculated population risks relevant to the assessment of nitrogen dioxide impacts from the ventilation structures and changes from the redistribution of traffic on surface roads, for the conservative and the realistic emissions scenarios, are less than 1×10^{-5}
- All calculated changes in population incidence are considered to be negligible
- Based on the discussion provided in **Section 8.1**, these population health impacts are considered to be acceptable.

Localised health impacts

Table 8.11 presents the change in risk associated with the maximum localised change in nitrogen dioxide concentrations due to emissions from the ventilation structures as well as the redistribution of traffic on surface roads. The tables present the calculated risks relevant to the consideration of the conservative as well as realistic emissions estimates.

As discussed in **Section 8.1**, the assessment of localised health risks has been undertaken to assist in evaluating the significance of the maximum impacts and inform the need for risk management. As a result, the maximum calculated risks are presented along with localised risks calculated for specific sensitive receptors, and these should be considered semi-quantitative only. Risks for all other areas within the project area would be lower than presented in **Table 8.11**.

Calculations relevant to the characterisation of risks associated with localised changes in nitrogen dioxide concentrations are presented in **Appendix F**.

Figure 8.6 presents a graphical representation of calculated localised changes in risks relevant to the assessment of mortality (all causes, all ages).

Table 8.10.a: Population health risk from changes in nitrogen dioxide

Scenario and area	Change in population risk relevant to the key health endpoints								
	Mortality: All causes (all ages)			Mortality: Respiratory (all ages)			Asthma ED Admissions (1–14 years)		
	Conservative 2026	Conservative 2036	Realistic 2036	Conservative 2026	Conservative 2036	Realistic 2036	Conservative 2026	Conservative 2036	Realistic 2036
Impacts from tunnel ventilation structures									
Banyule LGA	1E-06	2E-06	5E-07	3E-07	4E-07	1E-07	2E-06	2E-06	7E-07
Boroondara LGA	6E-07	7E-07	2E-07	1E-07	2E-07	5E-08	8E-07	9E-07	3E-07
Manningham LGA	9E-07	1E-06	3E-07	2E-07	2E-07	8E-08	1E-06	1E-06	5E-07
Nillumbik LGA	6E-07	7E-07	2E-07	1E-07	2E-07	5E-08	8E-07	9E-07	3E-07
Whitehorse LGA	2E-07	3E-07	1E-07	6E-08	7E-08	2E-08	3E-07	4E-07	1E-07
Whittlesea LGA	6E-07	7E-07	2E-07	1E-07	2E-07	6E-08	8E-07	1E-06	3E-07
Yarra LGA	6E-07	7E-07	2E-07	1E-07	2E-07	5E-08	8E-07	1E-06	3E-07
Impacts from redistribution of traffic on surface roads									
Project area	4E-06	4E-06	1E-06	1E-06	1E-06	4E-07	6E-06	6E-06	2E-06
Risk criteria (refer to Section 8.1)	Acceptable risk $\leq 1E-05$ (1×10^{-5})								

Table 8.10.b: Population health incidence from changes in nitrogen dioxide

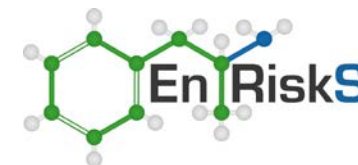
Scenario and area	Change in population incidence relevant to the key health endpoints (persons)								
	Mortality: All causes (all ages)			Mortality: Respiratory (all ages)			Asthma ED Admissions (1–14 years)		
	Conservative 2026	Conservative 2036	Realistic 2036	Conservative 2026	Conservative 2036	Realistic 2036	Conservative 2026	Conservative 2036	Realistic 2036
Impacts from tunnel ventilation structures									
Banyule LGA	0.14	0.17	0.056	0.029	0.034	0.011	0.030	0.035	0.012
Boroondara LGA	0.037	0.043	0.014	0.0077	0.0091	0.0030	0.0079	0.0093	0.0031
Manningham LGA	0.037	0.044	0.015	0.0018	0.0021	0.00069	0.0069	0.0082	0.0027
Nillumbik LGA	0.0043	0.0050	0.0017	0.00022	0.00025	0.000083	0.0010	0.0012	0.00040
Whitehorse LGA	0.0052	0.0061	0.0020	0.00026	0.00031	0.00010	0.0011	0.0013	0.00041
Whittlesea LGA ¹	0.017	0.020	0.0067	0.00091	0.0011	0.00036	0.0046	0.0055	0.0018
Yarra LGA ²	0.0052	0.0061	0.0020	0.00026	0.00031	0.00010	0.00067	0.00079	0.00026
Total – all LGAs	0.25	0.30	0.10	0.040	0.047	0.015	0.052	0.062	0.020
Impacts from redistribution of traffic on surface roads*									
Project area	0.16	0.18	0.060	0.0082	0.0090	0.0030	0.034	0.037	0.012

1 = Includes population within sub-set of Darebin North; 2 = Includes population within sub-set of Darebin South

* Calculated assuming every receptor evaluated roadside is residential, there 6 households per modelled receptor and the number of people per household is the same as the average for the LGAs evaluated in the project area

Table 8.11: Review of localised health impacts from maximum changes in nitrogen dioxide concentrations

Scenario and receptor	Maximum change in individual risk from short-term exposure to nitrogen dioxide for the following health endpoints and scenarios								
	Mortality: All causes (all ages)			Mortality: Respiratory (all ages)			Asthma ED Admissions (1–14 years)		
	Conservative 2026	Conservative 2036	Realistic 2036	Conservative 2026	Conservative 2036	Realistic 2036	Conservative 2026	Conservative 2036	Realistic 2036
Localised impacts from tunnels ventilation structures									
Maximum – all receptors	6E-06	7E-06	3E-06	1E-06	2E-06	6E-07	8E-06	9E-06	4E-06
Maximum child care	4E-06	5E-06	2E-06	1E-06	1E-06	4E-07	6E-06	7E-06	2E-06
Maximum schools	4E-06	5E-06	2E-06	1E-06	1E-06	4E-07	6E-06	7E-06	2E-06
Maximum aged care	6E-06	7E-06	2E-06	1E-06	2E-06	5E-07	8E-06	9E-06	3E-06
Maximum hospital/medical	4E-06	4E-06	1E-06	9E-07	1E-06	4E-07	5E-06	6E-06	2E-06
Localised impacts from changes in surface roads									
Albert Street (Bell St to Murray Rd)	-3E-07	-4E-07	-1E-07	-7E-08	-9E-08	-3E-08	-4E-07	-5E-07	-2E-07
Banksia Street (Albert St to North East Link)	1E-05	1E-05	5E-06	3E-06	4E-06	1E-06	2E-05	2E-05	7E-06
Bell Street (Upper Heidelberg Rd to Oriol Rd)	-4E-07	-5E-07	-2E-07	-1E-07	-1E-07	-4E-08	-6E-07	-7E-07	-2E-07
Bell Street (Plenty Rd to High St)	-7E-07	-1E-06	-4E-07	-2E-07	-3E-07	-1E-07	-1E-06	-2E-06	-6E-07
Boulton Street (Bridge Rd to Main Rd)	-2E-06	-2E-06	-8E-07	-4E-07	-6E-07	-2E-07	-2E-06	-3E-06	-1E-06
Broadway (High St to Bolderwood Pde)	-2E-06	-1E-06	-5E-07	-4E-07	-4E-07	-1E-07	-2E-06	-2E-06	-7E-07
Dalton Road (M80 Ring Rd and Childs Rd)	3E-06	4E-06	1E-06	8E-07	1E-06	3E-07	5E-06	6E-06	2E-06
Eastern Freeway (Springvale Road to Middleborough Road)	2E-05	2E-05	7E-07	5E-06	5E-06	2E-07	3E-05	3E-05	1E-06
Eastern Freeway (Middleborough Road to Elgar Road)	3E-05	4E-05	1E-05	8E-06	9E-06	3E-06	5E-05	5E-05	2E-05
Eastern Freeway (Elgar Road to Bulleen Road)	7E-05	8E-05	3E-05	2E-05	2E-05	6E-06	1E-04	1E-04	4E-05
Eastern Freeway (Bulleen Road to Hoddle Street)	2E-05	2E-05	7E-06	4E-06	5E-06	2E-06	3E-05	3E-05	1E-05
Fitzsimons Land (Foote St and Main Rd)	-1E-06	-2E-06	-6E-07	-3E-07	-5E-07	-1E-07	-2E-06	-3E-06	-9E-07
Grange Road (Darebin Rd to Heidelberg Rd)	-4E-07	-4E-07	-1E-07	-9E-08	-1E-07	-4E-08	-5E-07	-6E-07	-2E-07
Grimshaw Street (Watsonia Road to M80 Ring Road)	5E-05	5E-05	2E-05	1E-05	1E-05	4E-06	7E-05	8E-05	2E-05
High Street (Broadway and M80 Ring Road)	3E-07	2E-07	3E-08	7E-08	4E-08	8E-09	4E-07	3E-07	5E-08
Keon Parade (High St and Dalton Pde)	2E-06	2E-06	8E-07	4E-07	6E-07	2E-07	3E-06	3E-06	1E-06
Lower Plenty Road (Rosanna Rd to Greensborough Rd)	-3E-06	-3E-06	-1E-06	-7E-07	-8E-07	-3E-07	-4E-06	-4E-06	-1E-06
M80 Ring Road (M80 Ring Road Interchange)	5E-05	6E-05	2E-05	1E-05	2E-05	5E-06	8E-05	9E-05	3E-05
M80 Ring Road (M80 Ring Road to Plenty Road)	4E-05	4E-05	1E-05	9E-06	1E-05	3E-06	6E-05	6E-05	2E-05
M80 Ring Road (Plenty Road to Hume Freeway)	1E-05	1E-05	4E-06	3E-06	3E-06	1E-06	2E-05	2E-05	6E-06
Main Road (Parra Rd to Fitzsimons Lane)	-9E-07	-2E-06	-5E-07	-2E-07	-4E-07	-1E-07	-1E-06	-2E-06	-7E-07
Manningham Road (Thompsons Rd and Williamsons Rd)	-2E-07	-4E-07	-1E-07	-5E-08	-9E-08	-3E-08	-3E-07	-5E-07	-2E-07
Middleborough Road (Whitehorse Rd to the Eastern Freeway)	8E-06	9E-06	3E-06	2E-06	2E-06	7E-07	1E-05	1E-05	4E-06



Scenario and receptor	Maximum change in individual risk from short-term exposure to nitrogen dioxide for the following health endpoints and scenarios								
North East Link (North East Link Lower Plenty interchange)	5E-06	6E-06	2E-06	1E-06	1E-06	4E-07	7E-06	8E-06	3E-06
North East Link (Lower Plenty Road to Grimshaw St)	9E-05	1E-04	3E-05	2E-05	3E-05	8E-06	1E-04	1E-04	5E-05
Plenty Road (Albert St to M80 Ring Road)	1E-05	1E-05	4E-06	2E-06	3E-06	9E-07	1E-05	2E-05	5E-06
Reynolds Road (Blackburn Rd and Fitzsimons Lane)	-8E-07	-9E-07	-3E-07	-2E-07	-2E-07	-7E-08	-1E-06	-1E-06	-4E-07
Rosanna Rd (Bell Street Rd and Lower Plenty Rd)	8E-07	9E-07	3E-07	2E-07	2E-07	7E-08	1E-06	1E-06	4E-07
Station Street (Bell Street Rd and Darebin Rd)	-3E-07	-4E-07	-1E-07	-7E-08	-9E-08	-3E-08	-4E-07	-5E-07	-2E-07
Williamsons Road (Foote St and Warrandyte Rd)	-3E-06	-3E-06	-1E-06	-6E-07	-8E-07	-3E-07	-4E-06	-4E-06	-1E-06
Risk management criteria (refer to Section 8.1)	≥ 1E-04 (1 x 10⁻⁴)								

1E-6 = Change in localised air quality and so risk is positive – this means the localised air quality has been impacted as a result of the project.

-1E-06 = Change in localised air quality and so risk is negative – this means the localised air quality has improved and the maximum localised risks are reduced (lower) as a result of the project

1E-04 = Change in localised air quality and so risk is equal to or exceeds the risk management guideline adopted for the assessment of localised impacts.

Risk criteria adopted, below which population risks are acceptable (refer to **Section 8.1**)

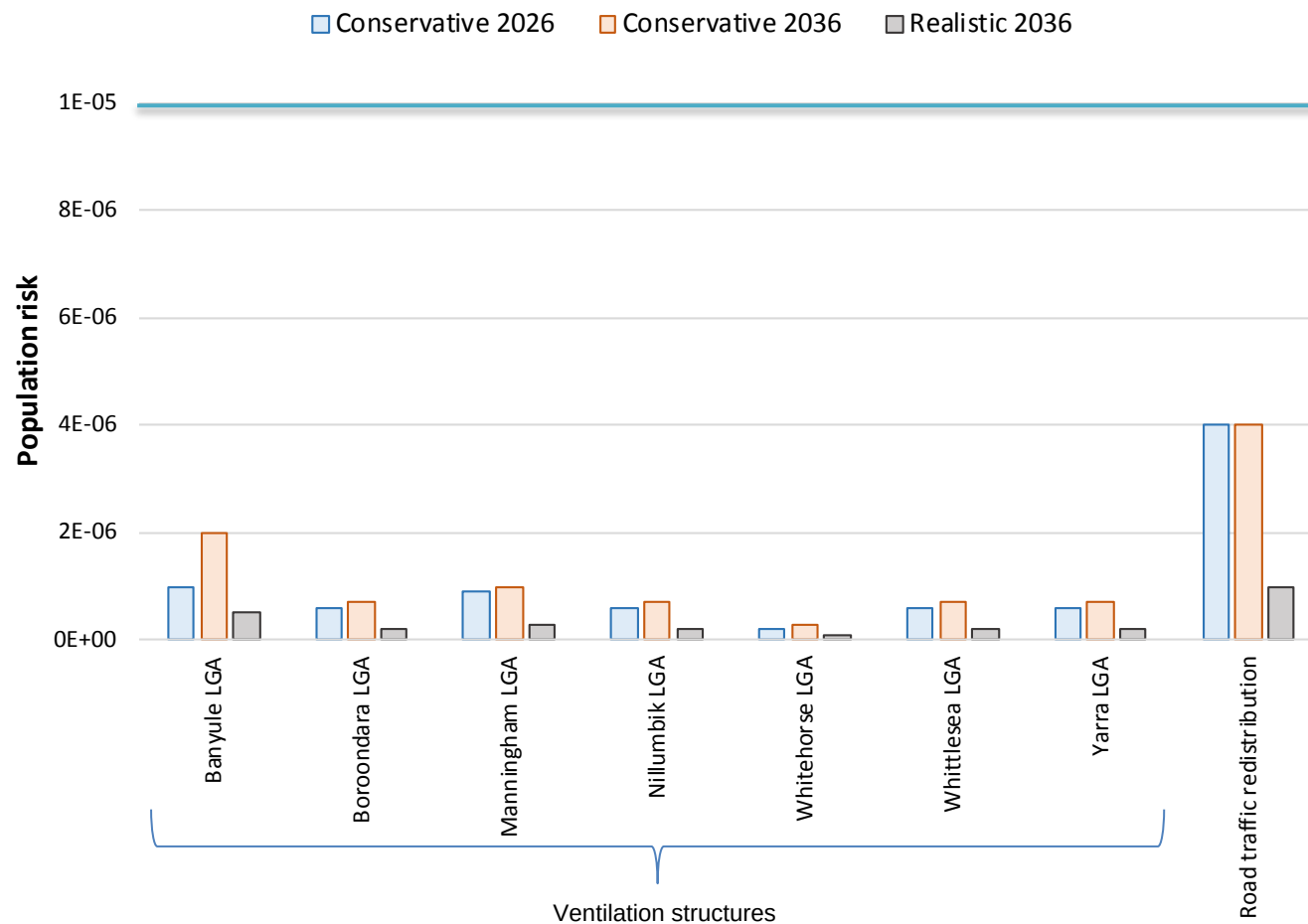
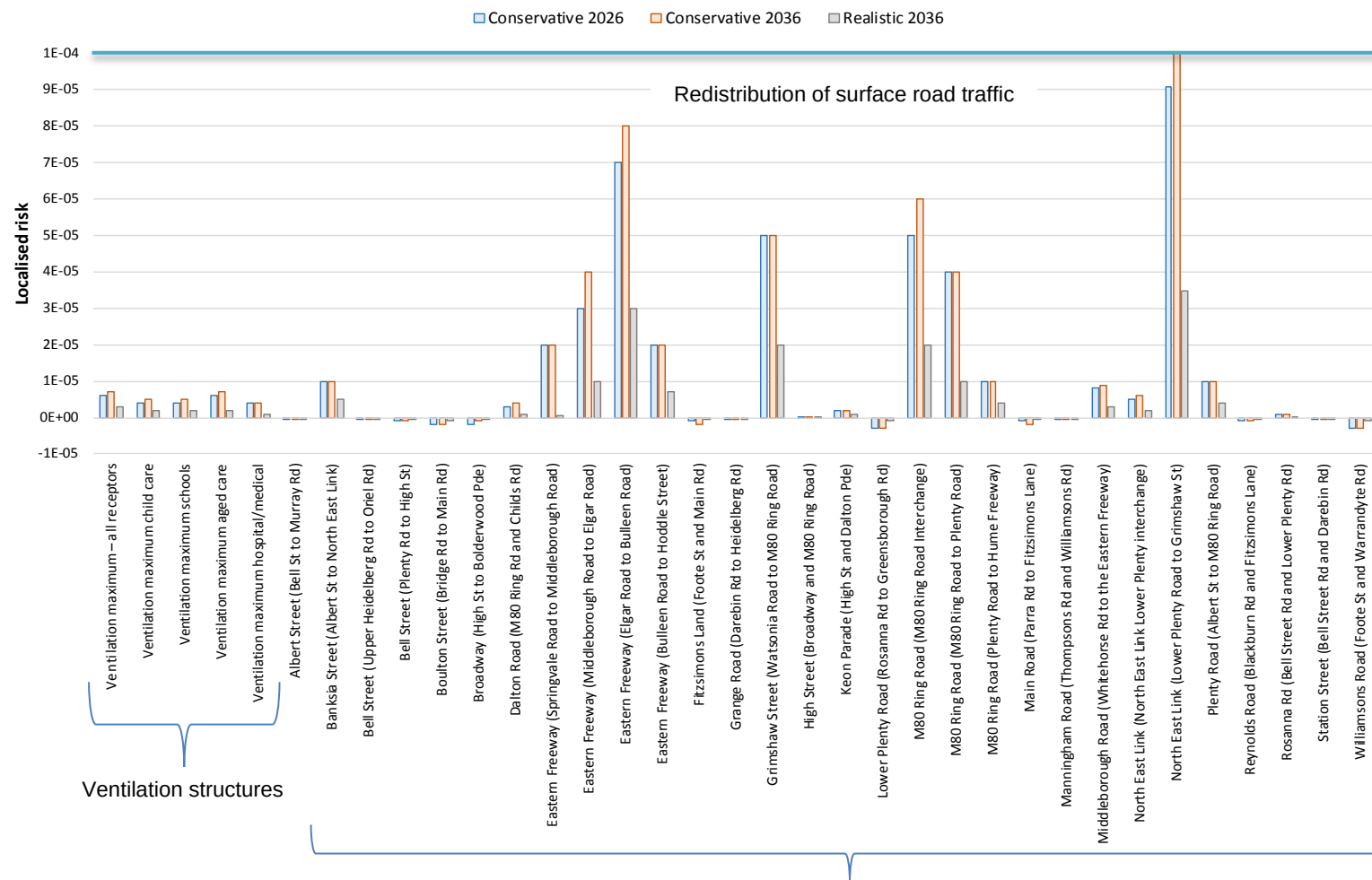


Figure 8.5: Population health risk from changes in nitrogen dioxide: All cause mortality (all ages)



Management criteria adopted for localised changes in risk (refer to Section 8.1)

Figure 8.6: Localised changes in health risk from changes in nitrogen dioxide: All cause mortality (all ages)

A review of **Table 8.11** and **Figure 8.6** above indicates:

- Localised impacts due to the redistribution of surface road traffic result in changes in risk that are greater than from the emissions from the ventilation structures.
- Calculated changes in risk based on the more realistic emissions scenario are lower than those calculated for the conservative emissions scenario.
- Localised impacts from the operation of the tunnel ventilation structures result in maximum risks that are below 1×10^{-5} and are not considered to require any further consideration of risk management (that is, below the risk management criteria of 1×10^{-4}).
- Localised impacts from the redistribution of surface road traffic results in:
 - A number of key surface roadways where risks are negative, which means localised air quality is improved and localised health impacts are lower
 - Conservative emissions estimates:
 - There are some key surface roadways that indicate an increase in the maximum localised risk up to an equal to the risk management level of 1×10^{-4}
 - Calculated risks are equal to the management level adjacent to the Eastern Freeway (Edgar Road to Bulleen Road) related to asthma emergency department admissions (children aged 1-14 years in 2026 and 2036)
 - Calculated risks are equal to the management level adjacent to the section of North East Link from Lower Plenty Road to Grimshaw Street, related to mortality (all cause and all ages in 2036) and asthma emergency department admissions (children aged 1-14 years in 2026 and 2036)
 - These calculated risks are considered to be conservative and an overestimate of actual exposures that may occur as a result of the project, so a more realistic assessment of potential exposures has also been undertaken and discussed below.
 - Realistic emissions estimates:
 - All calculated changes in risk are below the adopted risk management level of 1×10^{-4}
 - As this assessment relates to a more realistic assessment of the impact of vehicle emissions relevant to 2026 and 2036, no risk management measures need to be further considered in relation to localised changes in nitrogen dioxide.

Overall, calculated population risks (for all health endpoints considered) associated with changes in nitrogen dioxide levels in the community from North East Link are considered acceptable. The impact of the changes in nitrogen dioxide concentrations on the health of the population (as a population incidence as presented in **Table 8.11**) is very low and would not be measurable within the community.

Assessment of localised impacts within the community has not identified any areas that require further risk management due to emissions from the proposed tunnel ventilation structures or the redistribution of traffic on surface roads.

8.9 Assessment of health impacts – particulates

8.9.1 Particle size

Particulate matter is a widespread air pollutant with a mixture of physical and chemical characteristics that vary by location (and source). Unlike many other pollutants, particulates comprise a broad class of diverse materials and substances, with varying morphological, chemical, physical and thermodynamic properties, with sizes that vary from less than 0.005 microns to greater than 100 microns. 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 studies⁵ have reported significant positive associations between particulate air pollution and adverse health outcomes, particularly mortality as well as a range of adverse cardiovascular and respiratory effects.

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

⁵ 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 (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.

- Total suspended particulates (TSP): This refers to all particulates with an equivalent aerodynamic particle⁶ size below approximately 50 microns in diameter⁷. It is a fairly gross indicator of the presence of dust with a wide range of sizes. Larger particles (termed 'inspirable', comprise particles around 10 microns and larger) are more of a nuisance as they would deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory system⁸ and do not reach the lungs. Finer particles (smaller than 10 microns, 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 (see following point). Not all of the dust characterised as total suspended particulates is thus 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.
- PM₁₀ (particulate matter below 10 microns in diameter, μm), PM_{2.5} (particulate matter below 2.5 μm in diameter) and PM₁ (particulate matter below one μm in diameter, often termed very fine particles) and ultrafines (particulate matter below 0.1 μm in diameter), as illustrated in **Figure 8.7**. 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 tract⁹ and lungs. Once in the lungs, adverse health effects may result (OEHHA, 2002).

⁶ The term equivalent aerodynamic particle is used to reference the particle to a particle of spherical shape and particle of density one gram per cubic metre.

⁷ The size, diameter, of dust particles is measured in micrometers (microns).

⁸ 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.

⁹ 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.

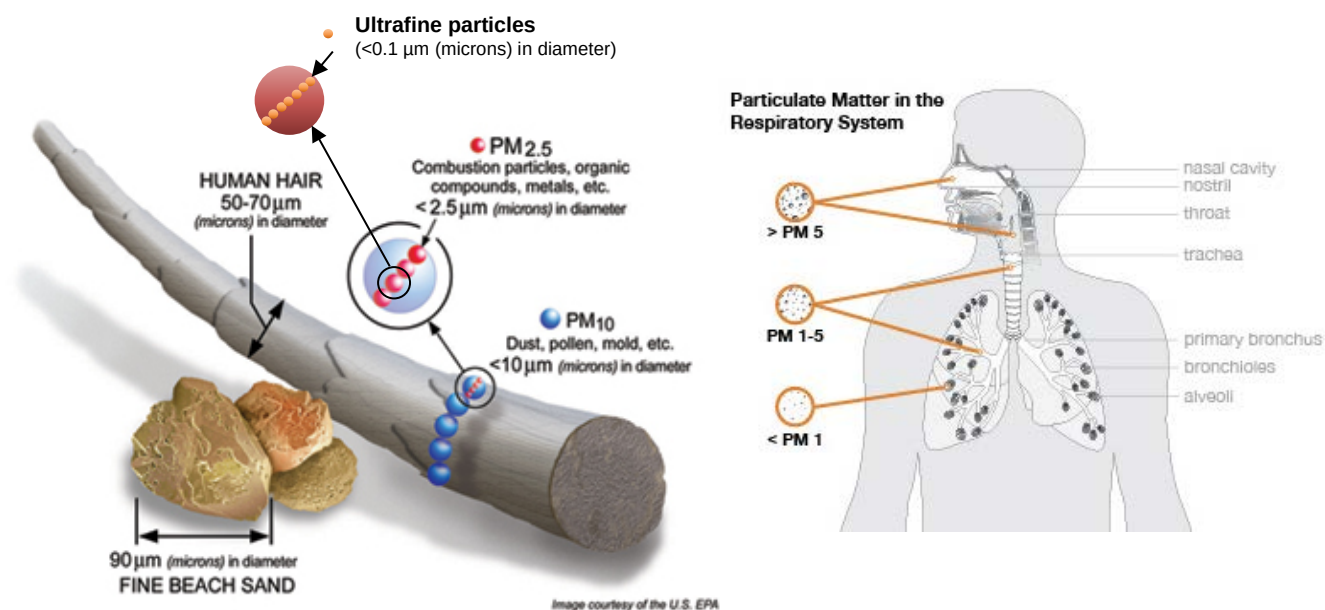


Figure 8.7: Illustrative representation of particle sizes and penetration into the lungs

Evaluation of size alone as a single factor in determining the potential for particulate toxicity is difficult since the potential health effects are not independent of chemical composition. There are certain particulate size fractions that tend to contain certain chemical components. Metals are commonly found attached to fine particulates (less than $PM_{2.5}$) while crustal materials (like soil) are usually larger and are present as 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 (such as nitrogen dioxide and carbon monoxide). This results in what is referred to as co-exposure and is an issue that has to be accounted for when evaluating studies that come from studying health effects in large populations exposed to pollution from many sources (as is the case in urban air).

Where co-exposure is accounted for the available science supports that exposure to fine particulate matter (less than $2.5\text{ }\mu\text{m}$, $PM_{2.5}$) is associated (and shown to be causal in some cases) with health impacts in the community (USEPA, 2012). A more limited body of evidence suggests an association between exposure to larger particles, PM_{10} and adverse health effects (USEPA, 2009a, 2018; WHO, 2003).

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

In relation to measuring particulate matter in urban air, the following should be noted:

- The measurement of particulate matter in urban air most commonly reports PM₁₀. This is the concentration of particulate matter less than or equal to 10 µm in diameter (and includes the smaller fractions of PM_{2.5} and very fine particles). The measurement techniques for PM₁₀ are well established and provide stable, robust, verifiable data that is considered to be consistently reported across all countries. This means this data on PM₁₀ collected in different parts of a city, in different parts of a country and by different countries can be compared against each other. This is the key reason why many of the epidemiological studies have looked at associations between PM₁₀ and various health effects.
- The measurement of PM_{2.5} is becoming more common in urban environments. This is the concentration of particulate matter less than or equal to 2.5 µm in diameter (and includes the smaller fractions of very fine particles and ultrafines). The measurement techniques used for PM_{2.5} are less well established resulting in data that varies depending on the type of equipment used and how it is set-up and maintained. Due to either a lack of monitoring data or the inconsistency of monitoring data some epidemiology studies have assessed associations between PM_{2.5} and health effects by using PM₁₀ data and assuming that a certain percentage of PM₁₀ comprises PM_{2.5}. Some studies have directly used measurements of PM_{2.5} in urban air. Even where these measurement issues are considered, the studies still clearly show strong relationships between changes in PM_{2.5} concentrations and health effects.
- The measurement of very fine and ultrafine particles is difficult (using equipment that is less robust/stable and provides variable data) and has not been undertaken in most urban air environments. As a result, there are no robust epidemiological studies that relate changes in ultrafine particle levels and health effects that can be used in a risk assessment. There is sufficient data available to confirm that motor vehicles are a key source of ultrafine particles. Available studies in animals and humans have identified a range of adverse health effects associated with exposure to ultrafine particulates, however the studies do not show that short-term exposure to ultrafine particulates have effects that are significantly different from those associated with exposure to PM_{2.5} (HEI, 2013).

When assessing health impacts from fine particulates, the robust associations of effects (that are based on large epidemiology studies primarily from the US and Europe) have been determined on the basis of PM_{2.5}, as PM_{2.5} which is what is commonly measured in urban air. No robust associations (that can be used in a quantitative assessment) are available for PM₁ and the current science is inconclusive in relation to ultrafine particulates. The associations developed for PM_{2.5} would include a significant contribution from PM₁ (as PM_{2.5} comprises a significant proportion of PM₁) and so health effects observed for PM₁ would be captured in the studies that have been conducted on the basis of PM_{2.5}. It is important 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. The further assessment of exposure to fine particulate matter has thus focused on particulates reported/evaluated as PM_{2.5}.

8.9.2 Health effects

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

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

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

In relation to morbidity effects, this refers to a wide range of health indicators used to define illness that have been associated with (or caused by) exposure to particulate matter. In relation to exposure to particulate matter, effects are primarily related to the respiratory and cardiovascular system and include (Morawska et al., 2004; USEPA, 2009a, 2018):

- 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.

The most recent review of the available studies (USEPA, 2018) have also indicated that effects on the nervous system and carcinogenic effects are likely to have a causal relationship with long-term exposures to $PM_{2.5}$. IARC (2013) has classified particulate matter as carcinogenic to humans based on data relevant to lung cancer.

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, 2009a).

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). While similar relationships have also been determined for PM_{10} , the supporting studies do not show relationships as clear as shown with $PM_{2.5}$ (USEPA, 2012).

There are a number of studies that have been undertaken where other health effects have been evaluated. These studies have a large degree of uncertainty or a limited examination of the relationship and are generally only considered to be suggestive or inadequate (in some cases) of an association with exposure to $PM_{2.5}$ (USEPA, 2018). This includes long-term exposures and metabolic effects, male and female reproduction and fertility, pregnancy and birth outcomes; and short-term exposures and nervous system effects (USEPA, 2018).

In relation to the key health endpoints relevant to evaluating exposures to $PM_{2.5}$, there are some associated health measures or endpoints where the exposure-response relationships are not as strong or robust as those for the key health endpoints and are considered to be a subset of the key health endpoints. This includes 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, 2011b; Ostro, 2004; WHO, 2006b).

8.9.3 Approach to the assessment of particulate exposures

In relation to the assessment of exposures to particulate matter, there is sufficient evidence to demonstrate an association between exposure to $PM_{2.5}$ (and to a lesser extent PM_{10}) and effects on health that are causal.

The available evidence does not suggest a threshold below which health effects do not occur. Accordingly, there are likely to be health effects associated with background levels of $PM_{2.5}$ and PM_{10} , even where the concentrations are below the current guidelines. Standards and goals are currently available for the assessment of $PM_{2.5}$ and PM_{10} in Victoria and Australia (NEPC, 2016). These standards and goals are not based on a defined level of risk that has been determined to be acceptable, rather they are based on balancing the potential risks due to background and urban sources to lower impacts on health in a practical way.

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

This assessment has therefore been undertaken to consider cumulative exposure impacts (refer to **Section 8.9.4**) as well as incremental exposure impacts associated with changes in PM_{2.5} and PM₁₀ concentrations that are associated with the project (refer to **Section 8.9.5**). Incremental changes are those due to the project alone while cumulative changes are those where background air quality in addition to those due to the project alone are considered.

8.9.4 Assessment of cumulative exposures

The assessment of cumulative exposures to PM_{2.5} and PM₁₀ is based on a comparison of the cumulative concentrations predicted with the current air quality standards and goals presented in the National Environment Protection Council (NEPC) (Ambient Air Quality) Measure (NEPM) (NEPC, 2016). The requirements of the NEPM are adopted in the State Environment Protection Policy (Ambient Air Quality) (SEPP), with the most recent variation gazetted on 28 July 2016 (No. G30 of the *Victorian Government Gazette*) (EPA Victoria, 1999 as varied to 2016). These standards and goals are total concentrations in ambient air, within the community, that are based on the most current science in relation to health effects. It is noted the SEPP (Air Quality Management) includes additional criteria for the assessment and management of impacts from specific sources of PM_{2.5} and PM₁₀. Assessment of compliance against the SEPP (Air Quality Management) is included in EES Technical report B – Air quality. The most current standards and goals, based on the protection of community health presented by the NEPC, have been further considered in this health impact assessment report.

In relation to the current NEPM PM₁₀ standard, the following is noted (NEPC, 1998, 2010, 2014, 2016):

- The standard was derived through a review of appropriate health studies by a technical review panel of the NEPC where short-term exposure-response relationships for PM₁₀ 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 standard.
- On the basis of the available data for key air sheds in Australia, the criterion of 50 micrograms per cubic metre 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 standard is not based on any acceptable level of risk.
- The assessment undertaken considered exposures and issues relevant to urban air environments that are expected to also be managed through the PM₁₀ standard. These issues included emissions from vehicles and wood heaters.

A similar approach has been adopted by NEPC (Burgers & Walsh, 2002; NEPC, 2002, 2014) in relation to the derivation of the PM_{2.5} air quality standards, with specific studies related to PM_{2.5} and mortality and morbidity indicators considered. Goals for lower PM_{2.5} standards to be met by 2025 are also outlined by NEPC (NEPC, 2016).

Table 8.12 presents a comparison of the current NEPC standards and goals with those established by the WHO (WHO, 2005), the European Union and the United States Environmental Protection Agency (USEPA) (2012). The 2025 goals established by the NEPM for PM_{2.5} (and adopted in this assessment) are similar to but slightly more conservative (health protective) than those provided by the WHO, European Union and the USEPA. The NEPM PM₁₀ guidelines are also similar to those established by the WHO and EU, but the guidelines are significantly lower than the 24-hour average guideline available from the USEPA.

Table 8.12: Comparison of particulate matter air quality goals

Pollutant	Averaging period	Criteria/guidelines/goals			
		NEPC and EPA Victoria (SEPP – Ambient Air Quality)	WHO (2005)	EU #	USEPA (2012)
PM ₁₀	24-hour	50 µg/m ³	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	25 µg/m ³	20* µg/m ³	40 µg/m ³ as limit value	NA
PM _{2.5}	24-hour	25 µg/m ³ 20 µg/m ³ (goal for 2025)	25 µg/m ³	NA	35 µg/m ³ (98th percentile, averaged over 3 years)
	Annual	8 µg/m ³ 7 µg/m ³ (goal for 2025)	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 European Union Air Quality Standards available from <http://ec.europa.eu/environment/air/quality/standards.htm>

* The WHO Air Quality guidelines are based on the lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95 per cent confidence in response to PM_{2.5} in the ACS study (Pope, CA, 3rd et al. 2002). The use of a PM_{2.5} guideline is preferred by the WHO (WHO 2005).

The air quality standards and goals for PM_{2.5} and PM₁₀ relate to total concentrations in the air (from all sources including the project). The background air quality data used in this project is outlined in EES Technical report B – Air quality. The background data includes a contribution of PM that is derived from vehicles that utilise the existing road network, but is not a background for properties adjacent to existing major roadways. Use of this background data would result in some double counting of the contribution of vehicle emissions to air quality in the local area, as the project has assumed emissions from vehicles using the project (or changes in surface road vehicles) are in addition to those currently using roads in the local area. This is a conservative approach.

Table 8.13 summarises the maximum 24-hour average and annual average concentrations of PM_{2.5} and PM₁₀ relevant to the assessment of emissions in 2026 and 2036, based on conservative as well as realistic emissions estimates. The maximum annual average concentration is the annual average concentration at the maximally affected grid location or individual sensitive receptor.

Table 8.13: Review of cumulative PM concentrations

Location and scenario	Maximum 24-hour average concentration (µg/m ³)				Maximum annual average concentration (µg/m ³)			
	PM _{2.5}		PM ₁₀		PM _{2.5}		PM ₁₀	
	Background	Background + tunnel vent	Background	Background + tunnel vent	Background	Background + tunnel vent	Background	Background + tunnel vent
Impacts from tunnels ventilation structures – maximum from all receptors								
2026 Conservative: Scenario A1	30	30.6	30	30.8	8.9	8.97	15	15.1
2036 Conservative: Scenario B1	30	30.7	30	30.9	8.9	8.99	15	15.1
2036 Realistic: Scenario B2	30	30.4	30	30.6	8.9	8.94	15	15.1
Impacts from changes in surface road traffic (excluding background*) – maximum from all receptors								
	No project	With Project	No project	With Project	No project	With Project	No project	With Project
2026 Conservative	1.7	4.0	2.2	4.9	0.8	1.5	1.0	1.9
2036 Conservative	1.9	4.5	2.5	5.4	0.9	1.7	1.1	2.0
2036 Realistic	1.9	NA	2.5	NA	0.9	1.5	1.1	NA
Standards and goals	25 (20 as goal for 2025)		50		8 (7 as goal by 2025)		25	

* Data as provided for the surface roads. Where cumulative (that is, background plus emissions from surface roads) are required to be considered, a conservative approach would be to add the background (30 µg/m³ for 24-hour averages and 8.9 µg/m³ for annual averages) to the no project and project estimates. This would result in some double counting of road emissions as the existing background included existing road emissions which are also counted in the no project and project calculations.

A review of **Table 8.13** indicates:

- The maximum total/cumulative concentrations of PM_{2.5} from the project are above the relevant standard and goal for the 24-hour and annual average, regardless of the project. This is due to existing levels (that is, background levels) of PM_{2.5} in the urban environment.
- The maximum cumulative 24-hour average and annual average concentrations of PM₁₀ from the project are below the relevant standard.
- The contribution of emissions from the tunnel ventilation structures is minor and does not significantly change existing air quality in the area, regardless of whether the conservative or realistic emissions estimates are considered. Larger changes in PM_{2.5} and PM₁₀ relate to localised impacts near surface roads.

Changes that occur due to the redistribution of traffic on surface roads results a number of areas where the project results in a reduction in PM_{2.5} and PM₁₀ exposures, and others where there is an increase in PM_{2.5} and PM₁₀ exposures. The maximum changes in 24-hour average PM_{2.5} concentrations at receptors adjacent to each of the key roadways assessed are illustrated in **Figure 8.8**. The same pattern of increases and decreases in impacts are relevant to PM₁₀.

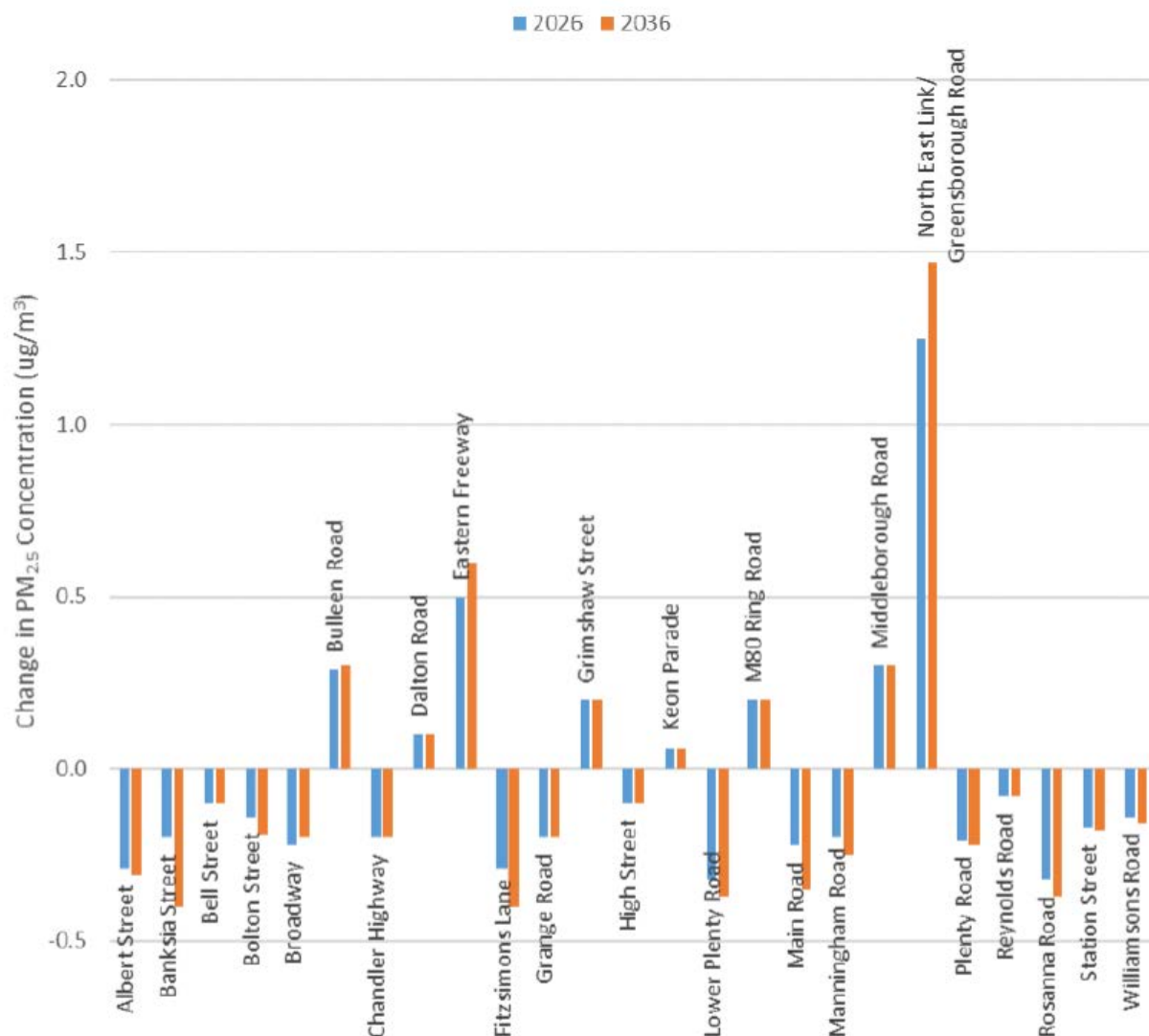


Figure 8.8: Maximum change in 24-hour average PM_{2.5} concentration based on conservative emissions estimates adjacent to key roadways in 2026 and 2036 (from Golder, 2019)

To further address potential risks to human health that may be associated with population exposures and localised changes in PM_{2.5} and PM₁₀ that relate to the project, incremental risk calculations have been undertaken and are presented in **Section 8.9.5**.

8.9.5 Changes in air quality – incremental exposures

Methodology for assessment of PM_{2.5} and PM₁₀

A detailed assessment of potential health effects associated with exposure to changes in air quality as a result of the project has been undertaken. As no threshold has been determined for exposure to PM_{2.5} or PM₁₀ the assessment of impacts on health has utilised robust, published, quantitative relationships (exposure-response relationships) that relate a change in PM_{2.5} or PM₁₀ concentration with a change in a health indicator. **Appendix C** presents an overview of the methodology adopted for using exposure-response relationships for the assessment of health impacts in a community.

For the assessment of potential exposures to changes in particulate matter, the assessment focused on health effects and exposure-response relationships that are robust and relate to PM_{2.5}, being the more important particulate fraction size relevant for emissions from combustion sources. Assessment of PM₁₀ has also been included. Refer to **Appendix E** for further discussion on the selection of these health endpoints, in the context of the current literature in relation to health effects from exposure to particulates.

The specific health effects (or endpoints) evaluated in this assessment include:

■ Primary health endpoints:

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

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

- Short-term exposure to PM₁₀ and changes in all-cause mortality (all ages)
- Long-term exposure to PM_{2.5} and changes in cardiopulmonary mortality (equal or greater than 30 years of age)
- Short-term exposure to PM_{2.5} and changes in cardiovascular and respiratory mortality (all ages)
- Short-term exposure to PM_{2.5} and changes in emergency department admissions for asthma in children aged 1–14 years.

Table 8.14 summarises the health endpoints considered in this assessment, the relevant health impact functions (from the referenced published studies) and the associated β coefficient relevant to the calculation of a relative risk (refer to **Appendix B** for details on the calculation of a β coefficient from published studies).

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

Table 8.14: 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					
PM _{2.5} : 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)
PM _{2.5} : 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, 2012; Bell et al., 2008)
PM _{2.5} : 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, 2012; Bell, et al., 2008)
Secondary assessment health endpoints					
PM ₁₀ : Mortality, all causes	Short-term	All ages*	1.006 [1.004-1.008]	0.0006 (0.06)	Based on analysis of data from European studies from 33 cities and includes panel studies of symptomatic children (asthmatics, chronic respiratory conditions) (Anderson et al., 2004)
PM _{2.5} : 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)
PM _{2.5} : Cardio-pulmonary mortality	Long-term	≥30yrs	1.14 [1.11-1.17]	0.013 (1.3)	Relationship derived for all follow-up time periods to the year 2000 (for approx. 500,000 participants in the US) with adjustment for seven ecologic (neighbourhood level) covariates (Krewski et al., 2009)
PM _{2.5} : 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)
PM _{2.5} : Asthma (emergency department admissions)	Short-term	1-14 years	--	0.00148 (0.148)	Relationship established from review conducted on Australian children (Sydney) for the period 1997 to 2001 (Jalaludin et al., 2008)
PM _{2.5} : 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

The assessment of health impacts for a population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro, 2004) (also outlined in **Appendix B**) where the exposure-response relationships (presented in **Table 8.13**) have been directly considered.

As discussed in **Section 8.1**, the assessment of health impacts associated with particulate matter has considered population impacts as well as localised impacts.

Population health impacts

Tables 8.15.a, 8.15.b, 8.16.a, 8.16.b, 8.17.a and 8.17.b present the calculated population health risks and incidence (that is, increase in the number of cases) relevant to the assessment of population health impacts associated with emissions of PM_{2.5} from the ventilation structures, as well as emissions of PM_{2.5} from the redistribution of vehicles on key surface roads within the project area (**Tables 8.15.a, 8.15.b, 8.16.a and 8.16.b** for the conservative emissions in 2026 and 2036 and **Tables 8.17.a and 8.17.b** for realistic emissions).

It is noted that only a population risk is presented for the redistribution of emissions on surface roads as the potential population that may be exposed next to these roadways is not known and would be too small for the calculation of population incidence to be meaningful.

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

- The relative risk has been calculated for a population weighted annual average incremental increase in concentrations. The population weighted average has been calculated on the basis of the smallest statistical division provided by the Australian Bureau of Statistics within a suburb (that is, mesh blocks – which are small blocks that cover an area of approximately 30 to 60 urban residences). For each mesh block in a LGA the average incremental increase in concentration has been calculated and multiplied by the population living in the mesh block (data available from the ABS for the 2016 census year). The weighted average has been calculated by summing these calculations for each mesh block in a LGA and dividing by the total population in the area evaluated (that is, in all the mesh blocks in the LGA evaluated).
- A change in the number of cases associated with the change in PM_{2.5} impact evaluated in the population within the study area has been calculated (refer to **Appendix B** for details on the methodology). The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered (refer to **Table 6.4** and **Table 6.5**) and the population (for the relevant age groups) present in the suburb (refer to **Table 6.1**).

Calculations relevant to the characterisation of population health risks and incidence associated with changes in PM_{2.5} concentrations in the community are presented in **Appendix G**.

In relation to mortality (all cause) **Figure 8.9** presents the calculated population risks relevant to the project for the conservative as well as realistic emissions scenarios.

Table 8.15.a: Population health risk from changes in PM_{2.5} concentrations: 2026 Conservative emissions

Scenario and receptor	Change in population risk from exposure to PM _{2.5} for the following health endpoints							
	Primary health indicators			Secondary health indicators				
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : Cardiovascular hospitalisations (≥65 years)	PM _{2.5} : Respiratory hospitalisations (≥65 years)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: Cardiopulmonary (ages 30+)	PM _{2.5} : Mortality: Cardiovascular (all ages)	PM _{2.5} : Mortality: Respiratory (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Impacts from tunnel ventilation structures								
Banyule LGA	8E-07	9E-07	2E-07	8E-08	1E-08	2E-08	1E-08	2E-07
Boroondara LGA	3E-07	4E-07	9E-08	3E-08	3E-09	8E-09	6E-09	6E-08
Manningham LGA	5E-07	6E-07	1E-07	5E-08	3E-09	1E-08	9E-09	1E-07
Nillumbik LGA	2E-07	3E-07	5E-08	2E-08	1E-09	4E-09	3E-09	7E-08
Whitehorse LGA	2E-07	2E-07	5E-08	2E-08	2E-09	3E-09	3E-09	4E-08
Whittlesea LGA ¹	2E-07	4E-07	1E-07	2E-08	1E-09	4E-09	4E-09	7E-08
Yarra LGA ²	2E-07	4E-07	1E-07	2E-08	2E-09	5E-09	4E-09	1E-07
Impacts from redistribution of traffic on surface roads								
Project area	2E-06	3E-06	8E-07	2E-07	3E-08	5E-08	5E-08	8E-07
Risk criteria (refer to Section 8.1)	Acceptable risk ≤ 1E-05 (1 x 10⁻⁵)							

1 = Includes population within sub-set of Darebin North

2 = Includes population within sub-set of Darebin South

Table 8.15.b: Calculated population incidence associated with changes in PM_{2.5} concentrations: 2026 Conservative emissions

Scenario and receptor	Change in population incidence from exposure to PM _{2.5} for the following health endpoints (persons)							
	Primary health indicators			Secondary health indicators				
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : Cardiovascular hospitalisations (≥65 years)	PM _{2.5} : Respiratory hospitalisations (≥65 years)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: Cardiopulmonary (ages 30+)	PM _{2.5} : Mortality: Cardiovascular (all ages)	PM _{2.5} : Mortality: Respiratory (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Impacts from tunnel ventilation structures								
Banyule LGA	0.058	0.019	0.0044	0.0094	0.00073	0.0021	0.0018	0.0047
Boroondara LGA	0.016	0.0048	0.0011	0.0025	0.00015	0.00060	0.00045	0.00074
Manningham LGA	0.015	0.0062	0.0014	0.0025	0.000091	0.00057	0.00044	0.00070
Nillumbik LGA	0.0023	0.00060	0.00013	0.00039	0.000013	0.000089	0.000069	0.00014
Whitehorse LGA	0.0022	0.00076	0.00017	0.00038	0.000013	0.000085	0.000067	0.00011
Whittlesea LGA ¹	0.0084	0.0021	0.00046	0.0015	0.000049	0.00035	0.00027	0.00056
Yarra LGA ²	0.0021	0.00044	0.00010	0.00036	0.000012	0.000082	0.000064	0.000067
Total	0.10	0.030	0.0069	0.017	0.0011	0.0039	0.0031	0.0070
Impacts from redistribution of traffic on surface roads*								
Project area evaluated	0.061	0.019	0.0041	0.010	0.00036	0.0024	0.0019	0.0031

1 = Includes population within sub-set of Darebin North; 2 = Includes population within sub-set of Darebin South

* Calculated assuming every receptor evaluated roadside is residential, there are 6 households per receptor and the number of people per household is the same as the average for the LGAs evaluated in the project area

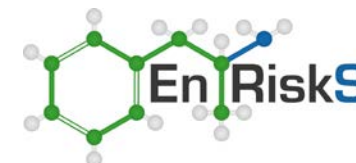


Table 8.16.a: Population health risk from changes in PM_{2.5} concentrations: 2036 Conservative emissions

Scenario and receptor	Change in population risk from exposure to PM _{2.5} for the following health endpoints							
	Primary health indicators			Secondary health indicators				
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : Cardiovascular hospitalisations (≥65 years)	PM _{2.5} : Respiratory hospitalisations (≥65 years)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: Cardiopulmonary (ages 30+)	PM _{2.5} : Mortality: Cardiovascular (all ages)	PM _{2.5} : Mortality: Respiratory (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Impacts from tunnel ventilation structures								
Banyule LGA	9E-07	1E-06	3E-07	1E-07	1E-08	2E-08	2E-08	3E-07
Boroondara LGA	4E-07	5E-07	1E-07	4E-08	4E-09	9E-09	7E-09	7E-08
Manningham LGA	6E-07	8E-07	2E-07	7E-08	4E-09	2E-08	1E-08	1E-07
Nillumbik LGA	2E-07	4E-07	7E-08	2E-08	1E-09	5E-09	3E-09	8E-08
Whitehorse LGA	2E-07	2E-07	6E-08	2E-08	2E-09	4E-09	4E-09	4E-08
Whittlesea LGA ¹	3E-07	6E-07	1E-07	3E-08	2E-09	5E-09	6E-09	1E-07
Yarra LGA ²	2E-07	4E-07	1E-07	2E-08	2E-09	5E-09	5E-09	2E-07
Impacts from redistribution of traffic on surface roads								
Project area	3E-06	4E-06	9E-07	3E-07	3E-08	6E-08	5E-08	9E-07
Risk criteria (refer to Section 8.1)	Acceptable risk ≤ 1E-05 (1 x 10⁻⁵)							

1 = Includes population within sub-set of Darebin North

2 = Includes population within sub-set of Darebin South

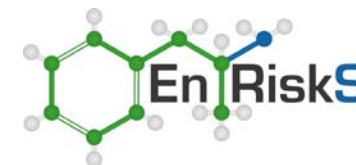


Table 8.16.b: Calculated population incidence associated with changes in PM_{2.5} concentrations: 2036 Conservative emissions

Scenario and receptor	Change in population incidence from exposure to PM _{2.5} for the following health endpoints (persons)							
	Primary health indicators			Secondary health indicators				
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : Cardiovascular hospitalisations (≥65 years)	PM _{2.5} : Respiratory hospitalisations (≥65 years)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: Cardiopulmonary (ages 30+)	PM _{2.5} : Mortality: Cardiovascular (all ages)	PM _{2.5} : Mortality: Respiratory (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Impacts from tunnel ventilation structures								
Banyule LGA	0.067	0.022	0.0051	0.011	0.00084	0.0024	0.0020	0.0054
Boroondara LGA	0.018	0.0056	0.0013	0.0030	0.00018	0.00070	0.00052	0.00087
Manningham LGA	0.018	0.0072	0.0016	0.0029	0.00011	0.00066	0.00052	0.00082
Nillumbik LGA	0.0020	0.00052	0.00012	0.00034	0.000012	0.000078	0.000061	0.00012
Whitehorse LGA	0.0025	0.00087	0.00019	0.00043	0.000015	0.00010	0.000076	0.00012
Whittlesea LGA ¹	0.0079	0.0020	0.00043	0.0014	0.000046	0.00033	0.00026	0.00052
Yarra LGA ²	0.0025	0.00052	0.00011	0.00043	0.000014	0.00010	0.000075	0.000079
Total	0.12	0.035	0.0080	0.019	0.0012	0.0044	0.0035	0.0080
Impacts from redistribution of traffic on surface roads*								
Project area evaluated	0.016	0.0047	0.0010	0.0027	0.000091	0.00060	0.00047	0.00079

1 = Includes population within sub-set of Darebin North; 2 = Includes population within sub-set of Darebin South

* Calculated assuming every receptor evaluated roadside is residential, there are 6 households per receptor and the number of people per household is the same as the average for the LGAs evaluated in the project area

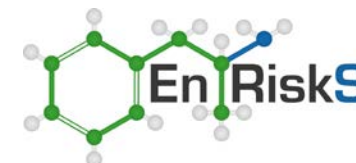


Table 8.17.a: Population health risk from changes in PM_{2.5} concentrations: 2036 Realistic emissions

Scenario and receptor	Change in population risk from exposure to PM _{2.5} for the following health endpoints							
	Primary health indicators			Secondary health indicators				
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : Cardiovascular hospitalisations (≥65 years)	PM _{2.5} : Respiratory hospitalisations (≥65 years)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: Cardiopulmonary (ages 30+)	PM _{2.5} : Mortality: Cardiovascular (all ages)	PM _{2.5} : Mortality: Respiratory (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Impacts from tunnel ventilation structures								
Banyule LGA	5E-07	6E-07	1E-07	5E-08	6E-09	1E-08	9E-09	1E-07
Boroondara LGA	2E-07	2E-07	5E-08	2E-08	2E-09	5E-09	3E-09	3E-08
Manningham LGA	3E-07	4E-07	8E-08	3E-08	2E-09	7E-09	6E-09	6E-08
Nillumbik LGA	1E-07	2E-07	4E-08	1E-08	7E-10	3E-09	2E-09	4E-08
Whitehorse LGA	9E-08	1E-07	3E-08	9E-09	9E-10	2E-09	2E-09	2E-08
Whittlesea LGA ¹	1E-07	3E-07	8E-08	1E-08	1E-09	3E-09	3E-09	5E-08
Yarra LGA ²	1E-07	2E-07	6E-08	1E-08	1E-09	3E-09	2E-09	8E-08
Impacts from redistribution of traffic on surface roads								
Project area	2E-06	2E-06	5E-07	2E-07	2E-08	3E-08	3E-08	6E-07
Risk criteria (refer to Section 8.1)	Acceptable risk ≤ 1E-05 (1 x 10⁻⁵)							

1 = Includes population within sub-set of Darebin North

2 = Includes population within sub-set of Darebin South

Table 8.17.b: Calculated population incidence associated with changes in PM_{2.5} concentrations: 2036 Realistic emissions

Scenario and receptor	Change in population incidence from exposure to PM _{2.5} for the following health endpoints (persons)							
	Primary health indicators			Secondary health indicators				
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : Cardiovascular hospitalisations (≥65 years)	PM _{2.5} : Respiratory hospitalisations (≥65 years)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: Cardiopulmonary (ages 30+)	PM _{2.5} : Mortality: Cardiovascular (all ages)	PM _{2.5} : Mortality: Respiratory (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Impacts from tunnel ventilation structures								
Banyule LGA	0.034	0.011	0.0026	0.0055	0.00043	0.0012	0.0010	0.0028
Boroondara LGA	0.0092	0.0028	0.00066	0.0015	0.000090	0.00035	0.00026	0.00044
Manningham LGA	0.0091	0.0036	0.00080	0.0015	0.000053	0.00033	0.00026	0.00041
Nillumbik LGA	0.0010	0.00027	0.000060	0.00018	0.0000061	0.000041	0.000032	0.000062
Whitehorse LGA	0.0012	0.00043	0.000096	0.00021	0.0000073	0.000049	0.000038	0.000062
Whittlesea LGA ¹	0.0040	0.00099	0.00022	0.00073	0.000023	0.00017	0.00013	0.00026
Yarra LGA ²	0.0012	0.00026	0.000058	0.00021	0.0000073	0.000049	0.000038	0.000040
Total	0.060	0.0176	0.0041	0.0098	0.00062	0.0022	0.0018	0.0041
Impacts from redistribution of traffic on surface roads*								
Project area evaluated	0.040	0.012	0.0027	0.0069	0.00024	0.0016	0.0012	0.0021

1 = Includes population within sub-set of Darebin North; 2 = Includes population within sub-set of Darebin South

* Calculated assuming every receptor evaluated roadside is residential, there are 6 households per receptor and the number of people per household is the same as the average for the LGAs evaluated in the project area

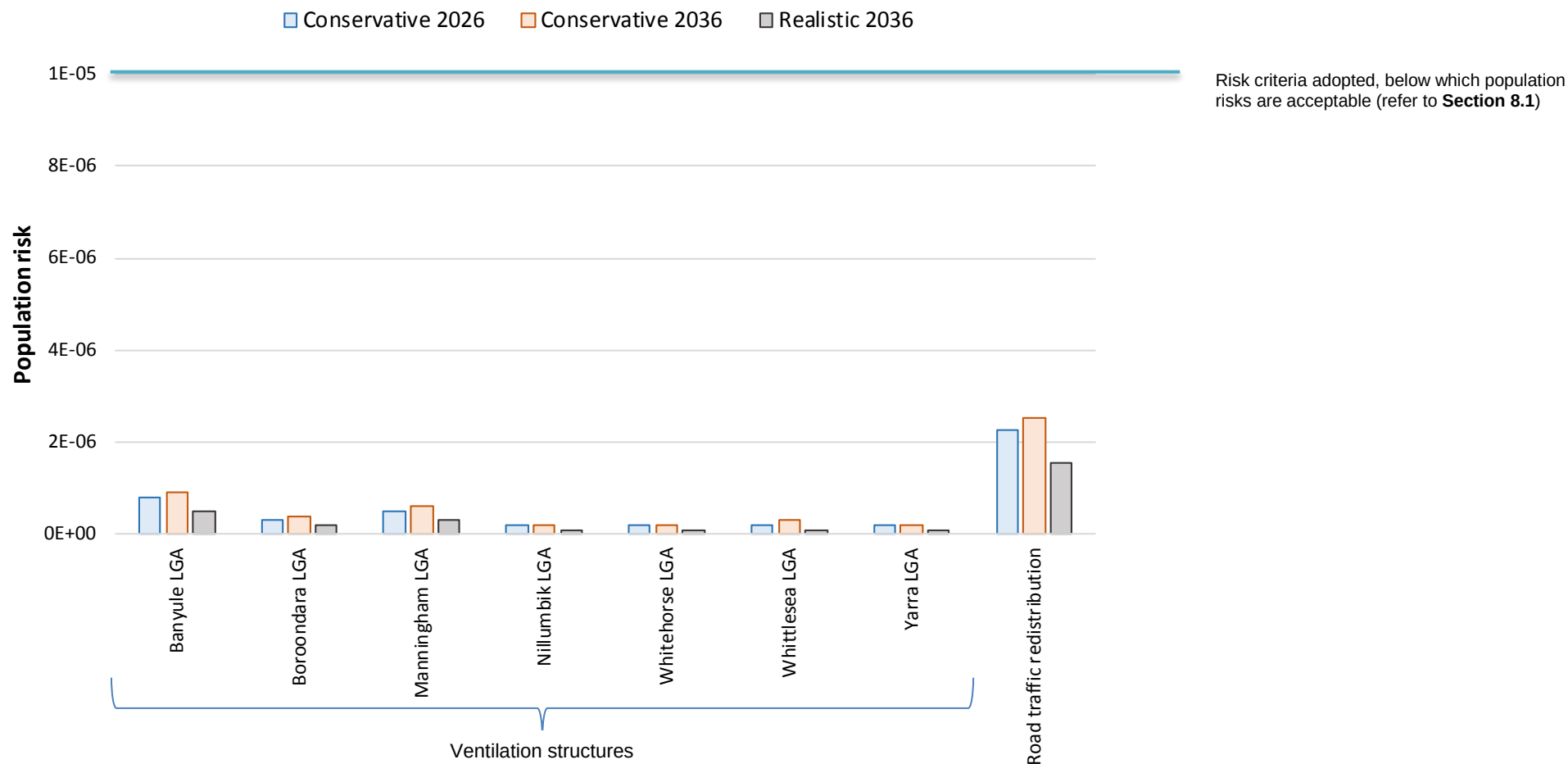


Figure 8.9: Population health risk from changes in PM_{2.5}: All cause mortality (30 years and older)

It is noted the assessment of potential health impacts from exposure to PM_{2.5} considers a number of health endpoints that are similar to those evaluated for changes in exposure to nitrogen dioxide. The health impacts assessed are not additive, as the relationships they are derived from epidemiological studies that include co-exposures to particulates and nitrogen dioxide and the data from which have been analysed in detail to isolate the relationships for the two classes of pollutants (consistent with the approach adopted by NEPC (Golder, 2013), also refer to **Section 11.5**).

Review of **Tables 8.15.a, 8.15.b, 8.16.a, 8.16.b, 8.17.a and 8.17.b** indicates:

- For the ventilation structures, risks calculated for the conservative emissions scenarios are higher than those calculated for the realistic scenario. Where the redistribution of traffic on surface roads is considered the conservative scenario and realistic scenarios are similar (noting a lower level of risk is calculated for the 2036 conservative scenario).
- All calculated population risks relevant to the assessment of PM_{2.5} impacts from the ventilation structures and changes from the redistribution of traffic on surface roads, where the conservative and the realistic emissions scenarios are considered, are less than 1×10^{-5} ;
- All calculated changes in population incidence are considered to be negligible and would not be measurable within the community;
- Based on the discussion provided in **Section 8.1**, these population health impacts are considered to be acceptable.

Localised health impacts

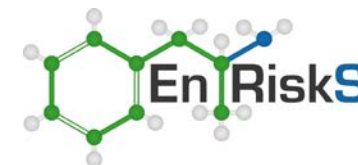
Tables 8.18.a, 8.18.b and 8.18.c presents the change in risk associated with the maximum localised change in PM_{2.5} and PM₁₀ concentrations due to emissions from the ventilation structures as well as the change in risk associated with the maximum change in PM_{2.5} from the redistribution of traffic on surface roads, for the conservative and realistic emissions scenarios. As discussed in **Section 8.1** the assessment of localised health risks has been undertaken to assist in evaluating the significance of the maximum impacts and inform the need for risk management. As a result, the maximum calculated risks are presented along with localised risks calculated for specific sensitive receptors, and these should be considered semi-quantitative only. Risks for all other areas within the project area would be lower than presented in **Tables 8.18.a, 8.18.b and 8.18.c**.

Calculations relevant to the characterisation of risks associated with localised changes in PM_{2.5} and PM₁₀ concentrations are presented in **Appendix G**.

Figure 8.10 presents the change in risk calculated as a result of the maximum localised change in PM_{2.5} for mortality (all causes).

Table 8.18.a: Review of localised health impacts from maximum changes in PM_{2.5} and PM₁₀ concentrations associated with project operations: 2026 Conservative emissions

Scenario and receptor	Maximum change in individual risk from exposure to localised changes in PM _{2.5} and PM ₁₀ for the following health endpoints and scenarios								
	Primary health indicators			Secondary health indicators					
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : CV hosp. (≥65 years)	PM _{2.5} : Resp. hosp. (≥65 years)	PM ₁₀ : Mortality: All causes (all ages)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: CP (ages 30+)	PM _{2.5} : Mortality: CV (all ages)	PM _{2.5} : Mortality: Resp. (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Localised impacts from tunnel ventilation structures									
Maximum – all receptors	5E-06	6E-06	2E-06	3E-07	5E-07	5E-08	1E-07	1E-07	2E-06
Maximum child care	3E-06	4E-06	9E-07	2E-07	3E-07	3E-08	6E-08	6E-08	9E-07
Maximum schools	3E-06	4E-06	1E-06	2E-07	3E-07	3E-08	6E-08	6E-08	1E-06
Maximum aged care	3E-06	5E-06	1E-06	3E-07	3E-07	4E-08	8E-08	7E-08	1E-06
Maximum hospital/medical	2E-06	3E-06	8E-07	2E-07	2E-07	3E-08	5E-08	5E-08	8E-07
Localised impacts from redistribution of surface road traffic									
Albert Street (Bell St to Murray Rd)	-3E-07	-4E-07	-1E-07		-3E-08	-4E-09	-7E-09	-6E-09	-1E-07
Banksia Street (Albert St to North East Link)	1E-05	2E-05	5E-06		1E-06	2E-07	3E-07	3E-07	5E-06
Bell Street (Upper Heidelberg Rd to Oriel Rd)	-4E-07	-6E-07	-1E-07		-4E-08	-5E-09	-9E-09	-9E-09	-1E-07
Bell Street (Plenty Rd to High St)	-6E-07	-9E-07	-2E-07		-6E-08	-7E-09	-1E-08	-1E-08	-2E-07
Boulton Street (Bridge Rd to Main Rd)	-2E-06	-3E-06	-7E-07		-2E-07	-2E-08	-5E-08	-4E-08	-7E-07
Broadway (High St to Bolderwood Pde)	-1E-06	-2E-06	-5E-07		-1E-07	-2E-08	-3E-08	-3E-08	-5E-07
Dalton Road (M80 Ring Rd and Childs Rd)	3E-06	4E-06	1E-06		3E-07	3E-08	7E-08	6E-08	1E-06
Eastern Freeway (Springvale Road to Middleborough Road)	1E-05	2E-05	5E-06		1E-06	2E-07	3E-07	3E-07	5E-06
Eastern Freeway (Middleborough Road to Elgar Road)	3E-05	4E-05	9E-06		3E-06	3E-07	6E-07	6E-07	9E-06
Eastern Freeway (Elgar Road to Bulleen Road)	6E-05	9E-05	2E-05		6E-06	8E-07	1E-06	1E-06	2E-05
Eastern Freeway (Bulleen Road to Hoddle Street)	2E-05	2E-05	6E-06		2E-06	2E-07	4E-07	3E-07	6E-06
Fitzsimons Land (Foote St and Main Rd)	-1E-06	-2E-06	-5E-07		-1E-07	-2E-08	-3E-08	-3E-08	-5E-07
Grange Road (Darebin Rd to Heidelberg Rd)	-4E-07	-6E-07	-2E-07		-4E-08	-5E-09	-1E-08	-9E-09	-2E-07
Grimshaw Street (Watsonia Road to M80 Ring Road)	3E-05	5E-05	1E-05		3E-06	4E-07	8E-07	7E-07	1E-05
High Street (Broadway and M80 Ring Road)	2E-07	3E-07	6E-08		2E-08	2E-09	4E-09	4E-09	7E-08
Keon Parade (High St and Dalton Pde)	2E-06	3E-06	6E-07		2E-07	2E-08	4E-08	4E-08	6E-07
Lower Plenty Road (Rosanna Rd to Greensborough Rd)	-3E-06	-4E-06	-1E-06		-3E-07	-3E-08	-6E-08	-6E-08	-1E-06
M80 Ring Road (M80 Ring Road Interchange)	4E-05	5E-05	1E-05		4E-06	4E-07	8E-07	8E-07	1E-05
M80 Ring Road (M80 Ring Road to Plenty Road)	3E-05	4E-05	1E-05		3E-06	3E-07	6E-07	6E-07	1E-05



Scenario and receptor	Maximum change in individual risk from exposure to localised changes in PM _{2.5} and PM ₁₀ for the following health endpoints and scenarios								
M80 Ring Road (Plenty Road to Hume Freeway)	7E-06	1E-05	3E-06		7E-07	9E-08	2E-07	2E-07	3E-06
Main Road (Parra Rd to Fitzsimons Lane)	-1E-06	-2E-06	-4E-07		-1E-07	-1E-08	-3E-08	-3E-08	-4E-07
Manningham Road (Thompsons Rd and Williamsons Rd)	-4E-07	-6E-07	-2E-07		-4E-08	-5E-09	-1E-08	-9E-09	-2E-07
Middleborough Road (Whitehorse Rd to the Eastern Freeway)	7E-06	1E-05	2E-06		7E-07	8E-08	2E-07	1E-07	2E-06
North East Link (North East Link Lower Plenty interchange)	6E-06	8E-06	2E-06		6E-07	7E-08	1E-07	1E-07	2E-06
North East Link (Lower Plenty Road to Grimshaw St)	5E-05	7E-05	2E-05		5E-06	6E-07	1E-06	1E-06	2E-05
Plenty Road (Albert St to M80 Ring Road)	6E-06	8E-06	2E-06		6E-07	7E-08	1E-07	1E-07	2E-06
Reynolds Road (Blackburn Rd and Fitzsimons Lane)	-9E-07	-1E-06	-3E-07		-9E-08	-1E-08	-2E-08	-2E-08	-3E-07
Rosanna Rd (Bell Street Rd and Lower Plenty Rd)	6E-07	9E-07	2E-07		6E-08	7E-09	1E-08	1E-08	2E-07
Station Street (Bell Street Rd and Darebin Rd)	-3E-07	-4E-07	-1E-07		-3E-08	-4E-09	-7E-09	-7E-09	-1E-07
Williamsons Road (Foote St and Warrandyte Rd)	-2E-06	-4E-06	-9E-07		-2E-07	-3E-08	-6E-08	-5E-08	-9E-07

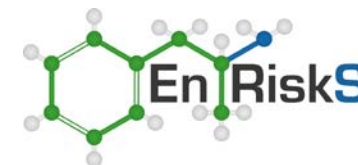
Risk management criteria (refer to Section 8.1)	≥ 1E-04 (1 x 10⁻⁴)
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Change in air quality and so risk is negative – this means that air quality has improved, and risks are reduced (lower) as a result of the project. Where not shaded, this means the risk is positive which means that air quality has been impacted as a result of the project.

CV = cardiovascular
 CP = cardiopulmonary
 Resp. = respiratory

Table 8.18.b: Review of localised health impacts from maximum changes in PM_{2.5} and PM₁₀ concentrations associated with project operations: 2036 Conservative emissions

Scenario and receptor	Maximum change in individual risk from exposure to localised changes in PM _{2.5} and PM ₁₀ for the following health endpoints and scenarios								
	Primary health indicators			Secondary health indicators					
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : CV hosp. (≥65 years)	PM _{2.5} : Resp. hosp. (≥65 years)	PM ₁₀ : Mortality: All causes (all ages)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: CP (ages 30+)	PM _{2.5} : Mortality: CV (all ages)	PM _{2.5} : Mortality: Resp. (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Localised impacts from tunnel ventilation structures									
Maximum – all receptors	5E-06	8E-06	2E-06	4E-07	5E-07	6E-08	1E-07	1E-07	2E-06
Maximum child care	3E-06	4E-06	1E-06	2E-07	3E-07	4E-08	7E-08	7E-08	1E-06
Maximum schools	3E-06	5E-06	1E-06	2E-07	3E-07	4E-08	7E-08	7E-08	1E-06
Maximum aged care	4E-06	6E-06	1E-06	3E-07	4E-07	5E-08	9E-08	9E-08	1E-06
Maximum hospital/medical	3E-06	4E-06	9E-07	2E-07	3E-07	3E-08	6E-08	6E-08	1E-06
Localised impacts from redistribution of surface road traffic									
Albert Street (Bell St to Murray Rd)	-5E-07	-7E-07	-2E-07		-5E-08	-6E-09	-1E-08	-1E-08	-2E-07
Banksia Street (Albert St to North East Link)	1E-05	2E-05	5E-06		1E-06	2E-07	3E-07	3E-07	5E-06
Bell Street (Upper Heidelberg Rd to Oriel Rd)	-7E-07	-1E-06	-3E-07		-7E-08	-9E-09	-2E-08	-2E-08	-3E-07
Bell Street (Plenty Rd to High St)	-2E-06	-2E-06	-6E-07		-2E-07	-2E-08	-4E-08	-4E-08	-6E-07
Boulton Street (Bridge Rd to Main Rd)	-3E-06	-5E-06	-1E-06		-3E-07	-4E-08	-7E-08	-7E-08	-1E-06
Broadway (High St to Bolderwood Pde)	-2E-06	-2E-06	-5E-07		-2E-07	-2E-08	-3E-08	-3E-08	-6E-07
Dalton Road (M80 Ring Rd and Childs Rd)	-1E-06	-2E-06	-4E-07		-1E-07	-1E-08	-3E-08	-3E-08	-4E-07
Eastern Freeway (Springvale Road to Middleborough Road)	1E-05	2E-05	4E-06		1E-06	1E-07	3E-07	3E-07	4E-06
Eastern Freeway (Middleborough Road to Elgar Road)	2E-05	3E-05	8E-06		2E-06	3E-07	5E-07	5E-07	8E-06
Eastern Freeway (Elgar Road to Bulleen Road)	6E-05	9E-05	2E-05		6E-06	7E-07	1E-06	1E-06	2E-05
Eastern Freeway (Bulleen Road to Hoddle Street)	1E-05	2E-05	5E-06		1E-06	2E-07	3E-07	3E-07	5E-06
Fitzsimons Land (Foote St and Main Rd)	-2E-06	-4E-06	-9E-07		-2E-07	-3E-08	-6E-08	-5E-08	-9E-07
Grange Road (Darebin Rd to Heidelberg Rd)	-7E-07	-1E-06	-3E-07		-7E-08	-9E-09	-2E-08	-2E-08	-3E-07
Grimshaw Street (Watsonia Road to M80 Ring Road)	3E-05	5E-05	1E-05		3E-06	4E-07	7E-07	7E-07	1E-05
High Street (Broadway and M80 Ring Road)	-5E-07	-7E-07	-2E-07		-5E-08	-6E-09	-1E-08	-1E-08	-2E-07
Keon Parade (High St and Dalton Pde)	-6E-08	-9E-08	-2E-08		-6E-09	-7E-10	-1E-09	-1E-09	-2E-08
Lower Plenty Road (Rosanna Rd to Greensborough Rd)	-4E-06	-5E-06	-1E-06		-4E-07	-4E-08	-8E-08	-8E-08	-1E-06
M80 Ring Road (M80 Ring Road Interchange)	4E-05	5E-05	1E-05		4E-06	4E-07	8E-07	8E-07	1E-05
M80 Ring Road (M80 Ring Road to Plenty Road)	3E-05	4E-05	9E-06		3E-06	3E-07	6E-07	6E-07	9E-06



Scenario and receptor	Maximum change in individual risk from exposure to localised changes in PM _{2.5} and PM ₁₀ for the following health endpoints and scenarios								
M80 Ring Road (Plenty Road to Hume Freeway)	5E-06	7E-06	2E-06		5E-07	6E-08	1E-07	1E-07	2E-06
Main Road (Parra Rd to Fitzsimons Lane)	-2E-06	-3E-06	-7E-07		-2E-07	-2E-08	-5E-08	-4E-08	-7E-07
Manningham Road (Thompsons Rd and Williamsons Rd)	-1E-06	-1E-06	-4E-07		-1E-07	-1E-08	-2E-08	-2E-08	-4E-07
Middleborough Road (Whitehorse Rd to the Eastern Freeway)	5E-06	8E-06	2E-06		5E-07	6E-08	1E-07	1E-07	2E-06
North East Link (North East Link Lower Plenty interchange)	5E-06	7E-06	2E-06		5E-07	6E-08	1E-07	1E-07	2E-06
North East Link (Lower Plenty Road to Grimshaw St)	5E-05	7E-05	2E-05		5E-06	6E-07	1E-06	1E-06	2E-05
Plenty Road (Albert St to M80 Ring Road)	3E-06	4E-06	1E-06		3E-07	4E-08	7E-08	7E-08	1E-06
Reynolds Road (Blackburn Rd and Fitzsimons Lane)	-2E-06	-3E-06	-7E-07		-2E-07	-2E-08	-5E-08	-4E-08	-7E-07
Rosanna Rd (Bell Street Rd and Lower Plenty Rd)	5E-07	7E-07	2E-07		5E-08	6E-09	1E-08	1E-08	2E-07
Station Street (Bell Street Rd and Darebin Rd)	-5E-07	-7E-07	-2E-07		-5E-08	-6E-09	-1E-08	-1E-08	-2E-07
Williamsons Road (Foote St and Warrandyte Rd)	-4E-06	-5E-06	-1E-06		-4E-07	-4E-08	-8E-08	-8E-08	-1E-06

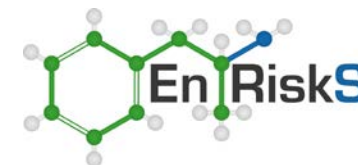
Risk management criteria (refer to Section 8.1)	≥ 1E-04 (1 x 10⁻⁴)
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Change in air quality and so risk is negative – this means that air quality has improved, and risks are reduced (lower) as a result of the project. Where not shaded, this means the risk is positive which means that air quality has been impacted as a result of the project.

CV = cardiovascular
 CP = cardiopulmonary
 Resp. = respiratory

Table 8.18.c: Review of localised health impacts from maximum changes in PM_{2.5} and PM₁₀ concentrations associated with project operations: 2036 Realistic emissions

Scenario and receptor	Maximum change in individual risk from exposure to localised changes in PM _{2.5} and PM ₁₀ for the following health endpoints and scenarios								
	Primary health indicators			Secondary health indicators					
	PM _{2.5} : Mortality: All causes (ages 30+)	PM _{2.5} : CV hosp. (≥65 years)	PM _{2.5} : Resp. hosp. (≥65 years)	PM ₁₀ : Mortality: All causes (all ages)	PM _{2.5} : Mortality: All causes (all ages)	PM _{2.5} : Mortality: CP (ages 30+)	PM _{2.5} : Mortality: CV (all ages)	PM _{2.5} : Mortality: Resp. (all ages)	PM _{2.5} : Asthma ED admissions (5-14 years)
Localised impacts from tunnel ventilation structures									
Maximum – all receptors	2E-06	3E-06	8E-07	2E-07	2E-07	3E-08	5E-08	5E-08	9E-07
Maximum child care	2E-06	2E-06	6E-07	2E-07	2E-07	2E-08	4E-08	3E-08	6E-07
Maximum schools	2E-06	2E-06	6E-07	2E-07	2E-07	2E-08	4E-08	3E-08	6E-07
Maximum aged care	2E-06	3E-06	7E-07	2E-07	2E-07	2E-08	5E-08	4E-08	8E-07
Maximum hospital/medical	1E-06	2E-06	5E-07	1E-07	1E-07	2E-08	3E-08	3E-08	5E-07
Localised impacts from redistribution of surface road traffic									
Albert Street (Bell St to Murray Rd)	-1E-07	-2E-07	-5E-08		-1E-08	-2E-09	-3E-09	-3E-09	-5E-08
Banksia Street (Albert St to North East Link)	9E-06	1E-05	3E-06		9E-07	1E-07	2E-07	2E-07	3E-06
Bell Street (Upper Heidelberg Rd to Oriel Rd)	-2E-07	-2E-07	-6E-08		-2E-08	-2E-09	-4E-09	-4E-09	-6E-08
Bell Street (Plenty Rd to High St)	-3E-07	-5E-07	-1E-07		-3E-08	-4E-09	-7E-09	-7E-09	-1E-07
Boulton Street (Bridge Rd to Main Rd)	-2E-06	-3E-06	-6E-07		-2E-07	-2E-08	-4E-08	-4E-08	-6E-07
Broadway (High St to Bolderwood Pde)	-5E-07	-6E-07	-2E-07		-5E-08	-5E-09	-1E-08	-1E-08	-2E-07
Dalton Road (M80 Ring Rd and Childs Rd)	1E-06	2E-06	4E-07		1E-07	1E-08	3E-08	3E-08	4E-07
Eastern Freeway (Springvale Road to Middleborough Road)	1E-05	1E-05	4E-06		1E-06	1E-07	2E-07	2E-07	4E-06
Eastern Freeway (Middleborough Road to Elgar Road)	2E-05	3E-05	6E-06		2E-06	2E-07	4E-07	4E-07	7E-06
Eastern Freeway (Elgar Road to Bulleen Road)	4E-05	6E-05	1E-05		4E-06	5E-07	9E-07	8E-07	1E-05
Eastern Freeway (Bulleen Road to Hoddle Street)	1E-05	2E-05	4E-06		1E-06	1E-07	3E-07	2E-07	4E-06
Fitzsimons Land (Foote St and Main Rd)	-1E-06	-2E-06	-4E-07		-1E-07	-1E-08	-3E-08	-3E-08	-4E-07
Grange Road (Darebin Rd to Heidelberg Rd)	-2E-07	-3E-07	-7E-08		-2E-08	-2E-09	-4E-09	-4E-09	-7E-08
Grimshaw Street (Watsonia Road to M80 Ring Road)	2E-05	3E-05	8E-06		2E-06	3E-07	5E-07	5E-07	8E-06
High Street (Broadway and M80 Ring Road)	2E-07	3E-07	8E-08		2E-08	3E-09	5E-09	5E-09	8E-08
Keon Parade (High St and Dalton Pde)	1E-06	2E-06	5E-07		1E-07	2E-08	3E-08	3E-08	5E-07
Lower Plenty Road (Rosanna Rd to Greensborough Rd)	-2E-06	-2E-06	-6E-07		-2E-07	-2E-08	-4E-08	-3E-08	-6E-07
M80 Ring Road (M80 Ring Road Interchange)	2E-05	3E-05	8E-06		2E-06	3E-07	5E-07	5E-07	8E-06
M80 Ring Road (M80 Ring Road to Plenty Road)	2E-05	3E-05	6E-06		2E-06	2E-07	4E-07	4E-07	6E-06



Scenario and receptor	Maximum change in individual risk from exposure to localised changes in PM _{2.5} and PM ₁₀ for the following health endpoints and scenarios								
M80 Ring Road (Plenty Road to Hume Freeway)	3E-06	5E-06	1E-06		3E-07	4E-08	7E-08	7E-08	1E-06
Main Road (Parra Rd to Fitzsimons Lane)	-8E-07	-1E-06	-3E-07		-8E-08	-9E-09	-2E-08	-2E-08	-3E-07
Manningham Road (Thompsons Rd and Williamsons Rd)	-3E-07	-4E-07	-1E-07		-3E-08	-3E-09	-7E-09	-6E-09	-1E-07
Middleborough Road (Whitehorse Rd to the Eastern Freeway)	5E-06	7E-06	2E-06		5E-07	6E-08	1E-07	1E-07	2E-06
North East Link (North East Link Lower Plenty interchange)	4E-06	6E-06	2E-06		4E-07	5E-08	1E-07	9E-08	2E-06
North East Link (Lower Plenty Road to Grimshaw St)	3E-05	4E-05	1E-05		3E-06	4E-07	7E-07	7E-07	1E-05
Plenty Road (Albert St to M80 Ring Road)	2E-06	4E-06	9E-07		2E-07	3E-08	6E-08	5E-08	9E-07
Reynolds Road (Blackburn Rd and Fitzsimons Lane)	-7E-07	-1E-06	-2E-07		-7E-08	-8E-09	-2E-08	-1E-08	-2E-07
Rosanna Rd (Bell Street Rd and Lower Plenty Rd)	4E-07	5E-07	1E-07		4E-08	4E-09	8E-09	8E-09	1E-07
Station Street (Bell Street Rd and Darebin Rd)	-1E-07	-2E-07	-5E-08		-1E-08	-2E-09	-3E-09	-3E-09	-5E-08
Williamsons Road (Foote St and Warrandyte Rd)	-1E-06	-2E-06	-5E-07		-1E-07	-2E-08	-3E-08	-3E-08	-5E-07

Risk management criteria (refer to Section 8.1)	≥ 1E-04 (1 x 10⁻⁴)
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Change in air quality and so risk is negative – this means that air quality has improved, and risks are reduced (lower) as a result of the project. Where not shaded, this means the risk is positive which means that air quality has been impacted as a result of the project.

CV = cardiovascular
 CP = cardiopulmonary
 Resp. = respiratory

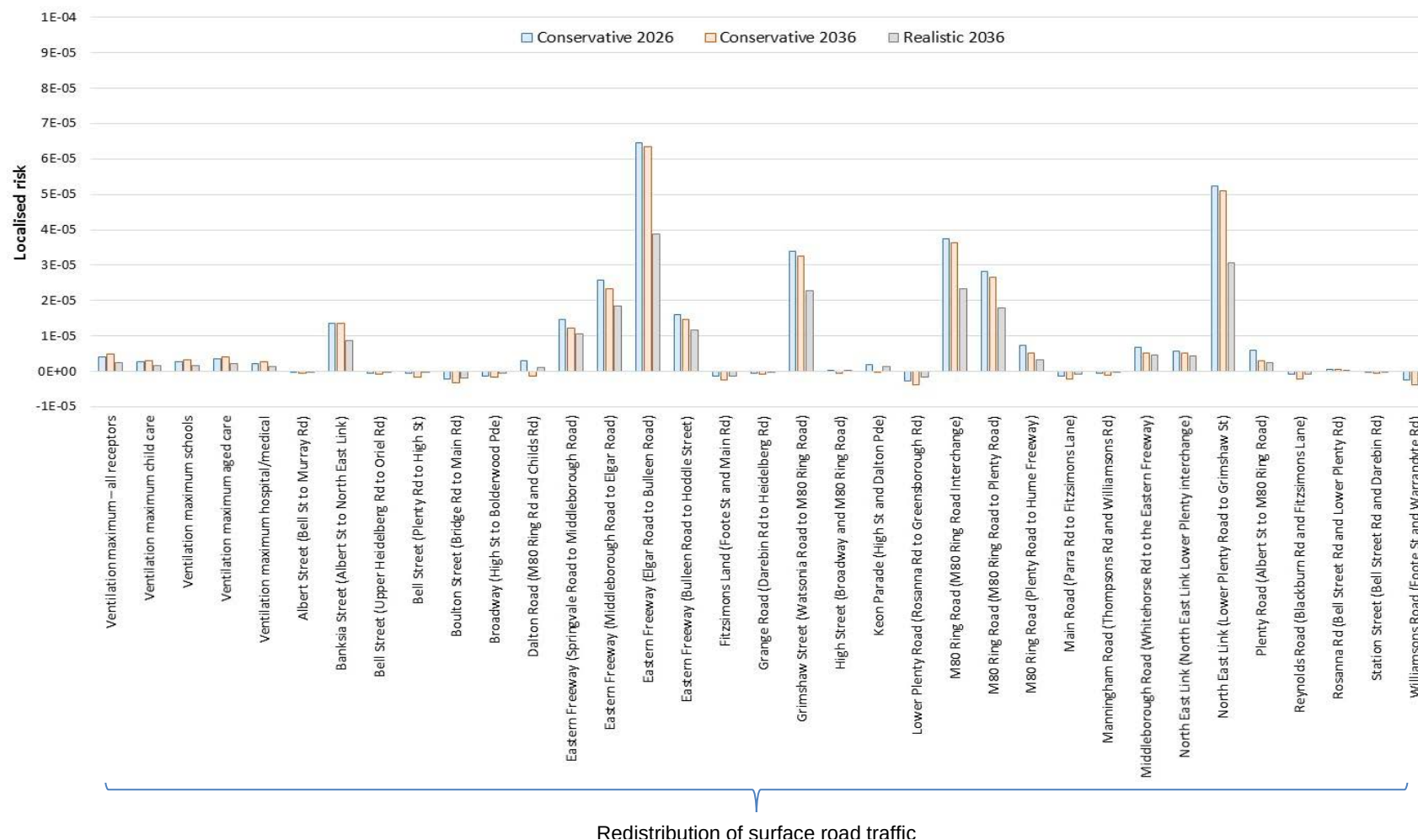


Figure 8.10: Localised changes in health risk from changes in PM_{2.5}: All cause mortality (ages 30 years and over)

Review of **Tables 8.18.a, 8.18.b and 8.18.c** (and **Figure 8.10**) indicates:

- Localised impacts due to the redistribution of surface road traffic result in changes in risk that are greater than from the emissions from the ventilation structures.
- Calculated changes in risk based on the more realistic emissions scenario are lower than those calculated for the conservative emissions scenario.
- Localised impacts from the operation of the tunnel ventilation structures, for the conservative and the realistic emissions scenarios, result in maximum risks that are below 1×10^{-5} and are not considered to require any further consideration of risk management (that is, below the risk management level of 1×10^{-4}).
- Localised impacts from the redistribution of surface road traffic, for the conservative as well as realistic emissions scenarios, are below the adopted risk management level of 1×10^{-4} .
- On the basis of the above assessment no risk management measures need to be considered in relation to localised changes in particulate matter.

Overall, calculated population risks (for all health endpoints considered) associated with changes in particulate matter, specifically $PM_{2.5}$, levels in the community from North East Link are considered acceptable. The impact of the changes in $PM_{2.5}$ concentrations on the health of the population (as a population incidence) is very low and would not be measurable within the community.

Assessment of localised impacts of $PM_{2.5}$ and PM_{10} within the community has not identified any areas that require further risk management due to emissions from the proposed tunnel ventilation structures.

The redistribution of traffic on surface roads would result in localised health risks from changes in $PM_{2.5}$ concentrations that range from negative risks implying some improvement in air quality and health, to increased health risks that imply impacts to local air quality and health. All calculated localised risks are below the risk management level and do not require the consideration of any risk management measures.

8.10 Assessment of in-tunnel air quality

Exposures that may occur within the tunnels depend on the concentration of pollutants in the tunnels (which would vary depending on the time of day and location within a tunnel, with higher concentrations expected towards the end of each tunnel compared with the entrance)) and the time spent in the tunnel.

The amount of time spent in a tunnel is expected to be limited. The length of the tunnel is approximately six kilometres, which would take approximately 4.5 minutes to travel at 80 km/hour. At peak travel times the time spent in the tunnel may double to approximately nine minutes.

Carbon monoxide and nitrogen dioxide

The tunnel ventilation system would be designed to control in-tunnel air quality so it meets the guidelines listed in **Table 8.19** for carbon monoxide and nitrogen dioxide, as outlined in EES Technical report B – Air quality.

Table 8.19: In-tunnel air quality guidelines

Parameter	Value
Carbon monoxide – 15-minute average	50 ppm (57 mg/m ³)
Nitrogen dioxide – 15-minute average	0.5 ppm (0.9 mg/m ³)

In relation to carbon monoxide, the adopted in-tunnel air quality limit is lower than the available health-based guidelines for exposure from the WHO (WHO, 2010). The in-tunnel air quality limits are therefore considered to be adequately protective of the health of people who use the tunnels in relation to carbon monoxide exposures.

Short-term exposure to nitrogen dioxide has been shown to cause respiratory health effects and is suspected of causing other health impacts such as cardiovascular effects (USEPA, 2016b). The concentration at which these impacts occur was subject to a review in 2015 (Jalaludin, 2015). This review, which has been used to develop the New South Wales NO₂ in-tunnel guideline, evaluated available studies in relation to health effects from in-tunnel and short-term exposures to nitrogen dioxide. The review evaluated studies associated with exposures that occur for less than 30 minutes as well as those with exposures of more than 60 minutes.

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

- There were no effects identified in relation to lung function for individuals exposed to nitrogen dioxide between 0.12 and 0.5 ppm
- The results for inflammatory markers (physiological measures that indicate the respiratory system or other systems in the body are dealing with inflammation) are mixed
- An effect of exposure to nitrogen dioxide and airway responsiveness was identified in individuals with asthma

- There is no clear evidence of a dose-response relationship for exposure and airway responsiveness for nitrogen dioxide levels at or below 0.5 ppm
- The effects observed for airway responsiveness may be transient. There is no clear evidence that repeated exposure to nitrogen dioxide leads to cumulative effects.

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

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

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

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

More recently, another review (enRiskS, 2018) was undertaken to consider NO₂ exposures of up to 60 minutes. This review supported the conclusions of the Jalaludin report, even for exposures of NO₂ up to 60 minutes. It found that for NO₂ exposures 0.5 ppm or less, the strongest evidence for effects were seen on airways responsiveness, and generally in asthmatics. These effects, if detected were small and not defined to be clinically relevant.

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

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

Table 8.20 summarises the available guidelines for the assessment of short duration exposures to nitrogen dioxide within tunnels.

Table 8.20: Summary of nitrogen dioxide guidelines for in-tunnel exposures

Jurisdiction/Project	Guideline/Criteria/Limit	Averaging period	Nature of guideline (tunnel design or compliance)
New South Wales (ACTAQ 2016)	0.5 ppm tunnel average	15 minutes	Design and compliance
NorthConnex and WestConnex	0.5 ppm tunnel average	15 minutes	Design and compliance
Brisbane City Council/Clem 7 and LegacyWay tunnels	1 ppm tunnel average	NA	Design
PIARC	1 ppm tunnel average	Not to be exceeded 2% of the time	Design
New Zealand	1 ppm	15 minutes	Design
Belgium	0.5 ppm tunnel average	<20 minutes	Design
France	0.4 ppm tunnel average	15 minutes	Design
Norway	0.75 ppm at midpoint in tunnel 1.5 ppm at end of tunnel	15 minutes	Design and compliance
Hong Kong	1 ppm	5 minutes	Design

The guideline to be adopted for the North East Link tunnels is consistent with other in-tunnel guidelines and, based on the available information on potential health effects, is considered to be adequately protective of the health of tunnel users.

Visibility (particulates)

Another important consideration for tunnel ventilation design is visibility. Consideration of visibility criteria in the design of the tunnel ventilation system is required due to the need for visibility levels that exceed the minimum vehicle stopping distance at the design speed. Visibility is reduced by the scattering and absorption of light by PM suspended in the air. The amount of light scattering or absorption is dependent upon:

- Particle composition (dark particles, such as soot, are particularly effective at reducing visibility by scattering/reflecting light)
- Particle diameter (particles need to be larger than around 0.4 μm to scatter light and reduce visibility)
- Particle density.

Particles causing a loss of visibility also have an effect on human health, and so monitoring visibility also provides the potential for an alternative assessment of the air quality and health risk within a tunnel. However, such an assessment is limited by the short duration of exposure in tunnels compared with the longer exposure times (24 hours and one year) for which the health effects of ambient exposure to particles have been established. Moreover, there is no safe minimum threshold for particles, and so visibility cannot reliably be used as a criterion for health risk (NHMRC, 2008). Visibility limits within the tunnel have thus not been further evaluated in regard to long-term health outcomes.

In relation to potential health effects associated with exposure to particulates within the tunnels the following can be noted:

- The exposure-response relationships for particulate matter that have been established on the basis of adverse health effects from short-term exposures relate to changes in the health effects associated with variability in 24-hour average concentrations of PM_{2.5} in urban air. They do not relate to much shorter variations in PM_{2.5} exposure that may occur within a 24-hour period, where there may be exposures over a few minutes to higher levels of PM_{2.5}. No guidelines are currently available for assessing potential health effects that may occur due to exposures to particulates that may occur for minutes (or even an hour).
- A recent review (WHO, 2013b) of available studies in relation to short-duration (less than 24-hour) exposures to particulates indicates that:
 - Epidemiological and clinical studies have demonstrated that sub-daily exposures to elevated levels of particulate matter can lead to adverse physiological changes in the respiratory and cardiovascular system, in particular exacerbation of existing disease. This is generally consistent with the outcome of studies reviewed and considered by the USEPA (2009a)
 - The studies available do not cover a range of exposure concentrations, nor do they adequately address other variables such as co-pollutants (gases) or repeated short-duration exposures.
 - The studies have not determined if a 1-hour exposure would lead to a different response than a similar dose spread over 24-hours, or if an exposure-response can be determined.
 - Exposures that occur during the use of various transportation methods (such as in-vehicles) have been found to contribute to and affect 24-hour personal exposures.

No guidelines are thus currently available to evaluate health effects of very short-duration exposures to particulates. However, it is noted that keeping windows closed and switching ventilation to recirculation has been shown to reduce particulate exposures inside the vehicle by up to 80 per cent (NSW Health, 2003). Adopting these measures, as is done in New South Wales, would minimise exposures to motorists within the tunnels.

Overall, the proposed in-tunnel air quality limits for carbon monoxide and nitrogen dioxide are considered adequately protective of the health of people who use the North East Link tunnels. In relation to exposures to particulates in the tunnels, there are no guidelines currently available to evaluate health effects of very short-duration exposures to particulates. However, it is noted that keeping windows closed and switching ventilation to recirculation has been shown to reduce particulate exposures inside the vehicle by up to 80 per cent (NSW Health 2003). Adopting these measures would, as a precautionary measure, minimise exposures to motorists within the tunnels.

8.11 Summary of air quality impacts on health

The assessment of health impacts associated with changes in air quality associated with North East Link has concluded:

■ Construction:

- While there is the potential for some health impacts during construction, these can be mitigated or minimised with the implementation of EPRs, specifically EPRs EMF2, AQ1, CL1 and SC2. If fully implemented this should mean no significant or measurable impacts on community health, with residual risks estimated to be low.

■ Operation:

- VOCs evaluated using health-based guideline – potential exposures to VOCs are below all relevant health-based guidelines and so health impacts are considered negligible.
- Carcinogenic VOCs, PAHs and DPM – potential exposures to carcinogenic VOCs, PAHs and DPM result in incremental carcinogenic risks that are considered to be low and acceptable.
- Carbon monoxide – potential exposures to carbon monoxide are below all relevant health-based guidelines and are considered negligible.
- Nitrogen dioxide:
 - Cumulative impacts associated with nitrogen dioxide emissions from the project are not considered to be of concern in relation to community health.
 - Population health impacts from the tunnel ventilation structures and the redistribution of traffic on surface roads are considered to be low and acceptable.
 - Localised maximum increases in health risk from emissions to air from the tunnel ventilation structures and from the redistribution of traffic on surface roads are not considered to be elevated.

□ Particulates:

- Population health impacts from the tunnel ventilation structures and the redistribution of traffic on surface roads are considered to be low and acceptable.
- Localised maximum increases in health risk from emissions to air from the tunnel ventilation structures and the redistribution of traffic on surface roads are not considered to be elevated.

■ **In-tunnel air quality:**

- In-tunnel air guidelines for carbon monoxide and nitrogen dioxide would be adequately protective of the health of North East Link users. Short-duration exposures to higher levels of particulates should be, as a precautionary measure, minimised by providing advice to motorists to keep windows closed and switch ventilation to recirculation as is currently done in New South Wales.

9. Assessment of noise and vibration impacts on health

9.1 Approach

This section assesses the potential for changes in noise and vibration from North East Link and how these changes might impact health within the community.

This assessment has drawn on information provided in EES Technical report C – Surface noise and vibration and EES Technical report D – Tunnel vibration, and in some areas summarises key (and relevant) aspects. All details relevant to the underlying assumptions, methodology and interpretation of impacts relevant to changes in noise and vibration are provided within these technical reports. Where more detail than provided in the health impact assessment is required, the reader is directed to the individual technical reports.

The characterisation of health impacts from changes in noise due to the project is complex.

This section presents an overview of the key aspects of the noise and vibration assessment and an assessment of potential health impacts associated with the predicted changes in noise and vibration in the local community. The assessment includes:

- Information on the existing noise environment (sourced from EES Technical report C –Surface noise and vibration), presented in **Section 9.2**
- Overview of the noise assessment criteria adopted (sourced from EES Technical report C – Surface noise and vibration), presented in **Section 9.3**
- Summary of noise and vibration assessments relevant to construction and operations (sourced from EES Technical report C – Surface noise and Vibration EES Technical report D Tunnel vibration), presented in **Section 9.4**
- Detailed assessment of the impact of changes in noise on community health (exposure and potential impacts), presented in **Section 9.5**.

The assessment of health impacts associated with the operation of the project involves the quantification of health risks and impacts.

The quantification of health impacts from changes in noise requires has been undertaken on the basis of:

- **Calculation of impacts, risks and health burden, of changes in noise:** The data available on health impacts from exposure to noise, including noise from road traffic, comes from large population or epidemiological studies. These studies enable relationships between noise exposures and various health effects (specifically mortality—a shortening of life-span—and morbidity effects). These exposure-response relationships are developed based on large population exposures and are utilised in the assessment of population health, and for establishing community noise guidance. These relationships are not developed for the

assessment of specific sources or localised impacts, as is the case for the assessment of impacts from North East Link.

North East Link would involve constructing new roadway infrastructure that would cause the redistribution of traffic within the community. The ventilation structures would also be a source of noise. Vehicle and truck movements on the roads within the broader community would remain much the same, which makes the conduct of community or larger population-wide assessments of health impacts difficult as the overall health impact is expected to reflect the small change in total vehicle movements. However, as traffic is more locally redistributed it is important to also evaluate the potential significance of this redistribution, particularly localised increases in noise levels. While this may only affect a small number of households, increases in risk associated with these maximum changes also need to be considered. In addition, localised changes in noise from the ventilation structures has been considered. This assessment has therefore considered community health impacts to inform the assessment of the overall health burden of the project, as well as localised health impacts (from changes on local surface roads and the noise of the ventilation structures) to inform management decisions relating to the magnitude of localised impacts.

Community/population health impacts have been assessed on the basis of the overall change in population risk (for the population evaluated close to the project and associated key surface roads) and health incidence (change in the number of cases). There is very limited guidance available in relation to acceptability of community risks associated with changes in noise (refer to **Appendix D** for further discussion). However for the purpose of this health impact assessment, guidance available from the National Environment Protection Council (NEPC, 2011) has been adopted. It is noted that while this guidance was developed for the assessment of health impacts from changes in air quality, in the absence of any other guidance more specific to noise, the same risk level has been adopted for the assessment of noise. The following has therefore been adopted:

For this assessment, acceptable population risk for changes in noise is $\leq 1 \times 10^{-5}$

Localised health impacts have also been calculated to assess the potential significance of maximum increases in noise due to localised redistribution of traffic. As this is a localised impact it is not possible to calculate an increased population incidence and the calculation of risk relates to a maximum localised risk, not a population risk. Due to the limitations of applying the exposure-response functions to localised impacts, these localised risks are considered to only be semi-quantitative. There is no guidance available for the assessment of localised risks for changes in noise. **Appendix D** provides additional discussion in relation to determining various risk levels. Based on the discussion provided in **Appendix D**, and consideration of the need to determine an action level for the management of localised impacts, a risk management level that is equal to the level at which risks are considered unacceptable has been adopted in this assessment as follows:

For this assessment, the risk management level for localised risk $\geq 1 \times 10^{-4}$

Calculated population risks and localised risks for changes in noise are presented in **Section 9.5.3**.

Calculation of the proportion of population that is annoyed or experiences sleep disturbance:

The assessment of changes in annoyance and sleep disturbance requires some consideration of what level of change may be of concern in terms of health, and complaints. The assessment presented has focused on the key measures of highly annoyed and highly sleep disturbed (WHO, 2018). **Appendix D** provides further discussion about determining where changes in these measures within a population may be of significance. Based on the discussion presented, the following has been adopted in this assessment:

For this assessment, acceptable change in population highly annoyed $\leq 5\%$

For this assessment, acceptable change in population highly sleep disturbed $\leq 3\%$

There are no guidance available for the assessment of localised changes in noise, so the above criteria for highly annoyed and highly sleep disturbed have also been adopted for the assessment of localised impacts.

9.2 Existing noise environment

The existing noise environment in the project area is described in detail in EES Technical report C – Surface noise and vibration.

In summary, the existing noise environment is dominated by noise from existing major roadways, freeways and local roads (in particular Greensborough Road, the M80 Ring Road, Bulleen Road and the Eastern Freeway), with noise levels varying depending on the proximity to these areas and the effectiveness of existing noise walls.

Maximum noise events are typically caused by heavy vehicle movements and the use of heavy vehicle engine brakes as well as motorbikes. This is typical of major road corridors.

Other key noise sources include occasional high-level aircraft movements and railway movements on the Hurstbridge rail line, as well as other local activities.

Background noise levels are important for informing the noise assessment on the existing noise in the community, and how the additional noise sources associated with construction or changes in traffic without and with the project may impact the community.

9.3 Noise assessment criteria

The noise assessment criteria relevant to North East Link are outlined in EES Technical report C – Surface noise and vibration. Specifically, these criteria include:

- **Construction:** EPA Victoria publication 480 – Guidelines for Major Construction Sites, EPA Victoria Publication 1254 – Noise Control Guidelines, and Australian Standard 2436 2010 – Guide to Noise Control on Construction, Maintenance and Demolition Sites. The assessment of construction noise impacts has also considered the Interim Construction Noise Guidelines from the Department of Environment and Climate Change in New South Wales (adopted as management levels rather than criteria). Guidelines relevant to the assessment of vibration (including blasting) impacts are derived from the New South Wales Assessing Vibration:

Technical Guide, Australian standards (AS2187.2) and International standards (DIN 4150 (1999/2016)). These guidelines relate to cosmetic and structural damage and human discomfort.

- **Operation:** For fixed structures, noise has been assessed in accordance with Victoria EPA State Environment Protection Policy (Control of Noise from Commerce, Industry and Trade No 1 – SEPP-N1). For the assessment of traffic noise relevant to all other receptors, project-specific noise criteria have been utilised (at the building façade) as follows:

- Category A: $L_{A10(18\text{hour})} = 63$ dBA (Category A relates to dwellings, aged care homes, hospitals, motels, caravan parks and other buildings with residential uses)
- Category B: $L_{A10(12\text{hour})} = 63$ dBA (Category B relates to schools, kindergartens, libraries, places of worship and other noise sensitive community uses).

The assessment of operational noise has also considered the WHO noise guidelines (WHO, 2009, 2018).

9.4 Overview of noise and vibration assessment

9.4.1 General

The assessment of noise was undertaken within a number of noise precincts: Precincts 1 to 5. The figures on the next pages show the location of each noise precinct, along with the sensitive receivers considered.

The assessment considered changes in noise associated with construction and operation of the project at a number of sensitive receivers within these precincts. These sensitive receivers include residential areas, community buildings (that include child care centres and kindergartens), outdoor recreation and public open spaces and heritage buildings.

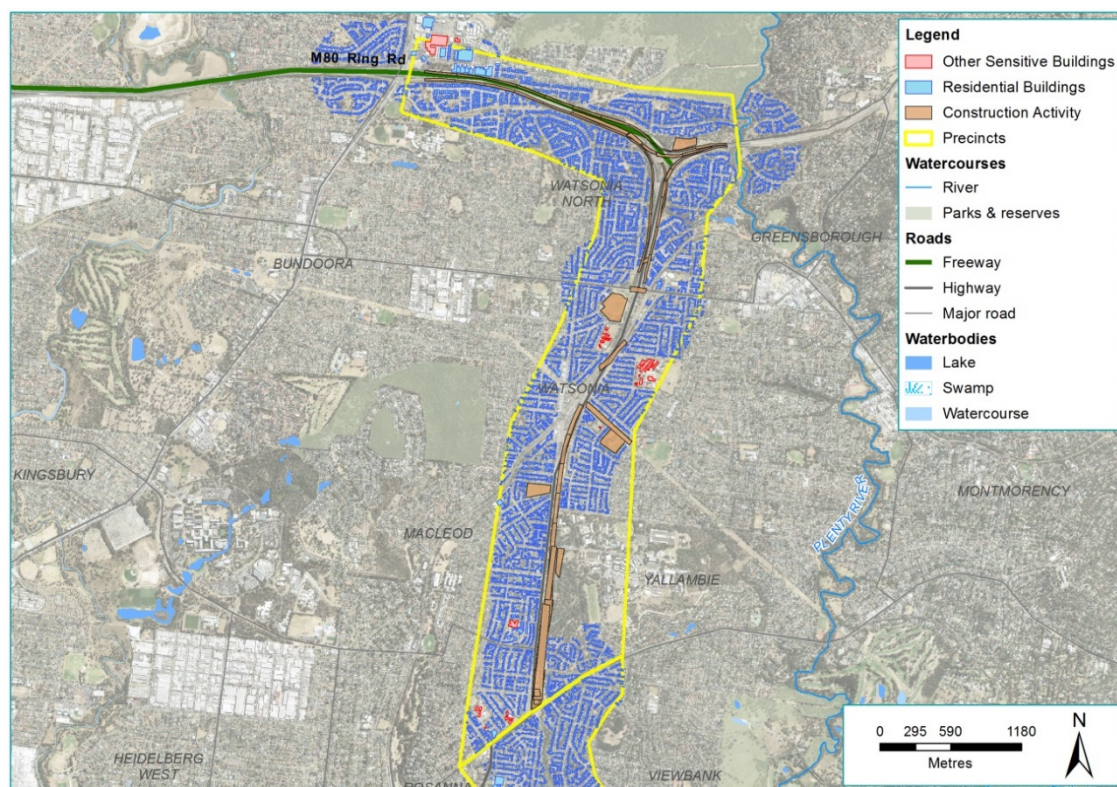


Figure 9.1 a: Precinct 1 (North)

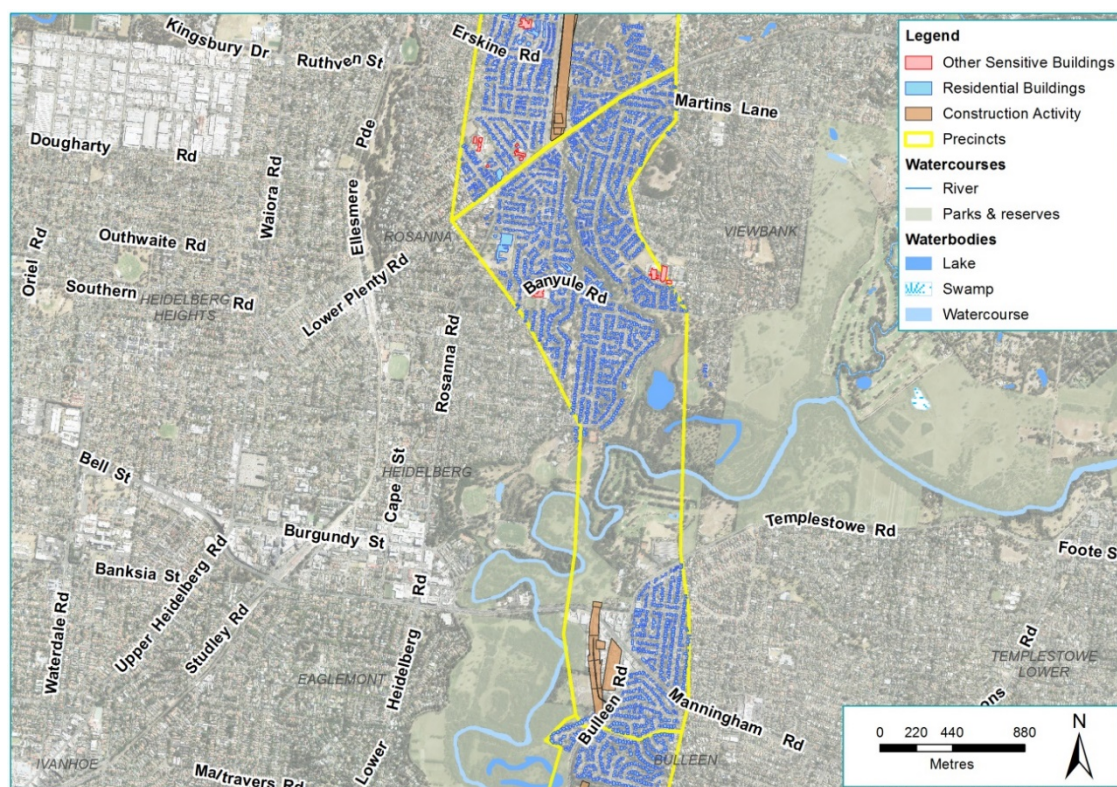


Figure 9.1 b: Precinct 2 (Tunnel portals and tunnelled section)

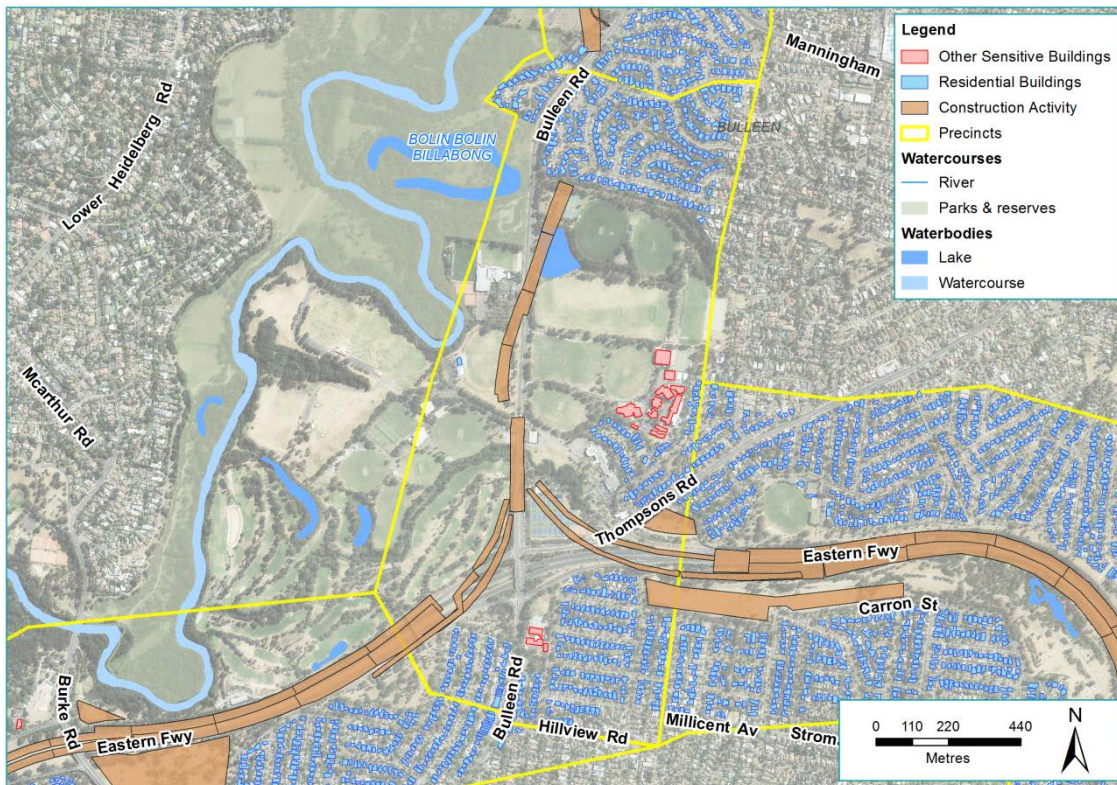


Figure 9.1 c: Precinct 3 (Manningham Road interchange to Eastern Freeway interchange)

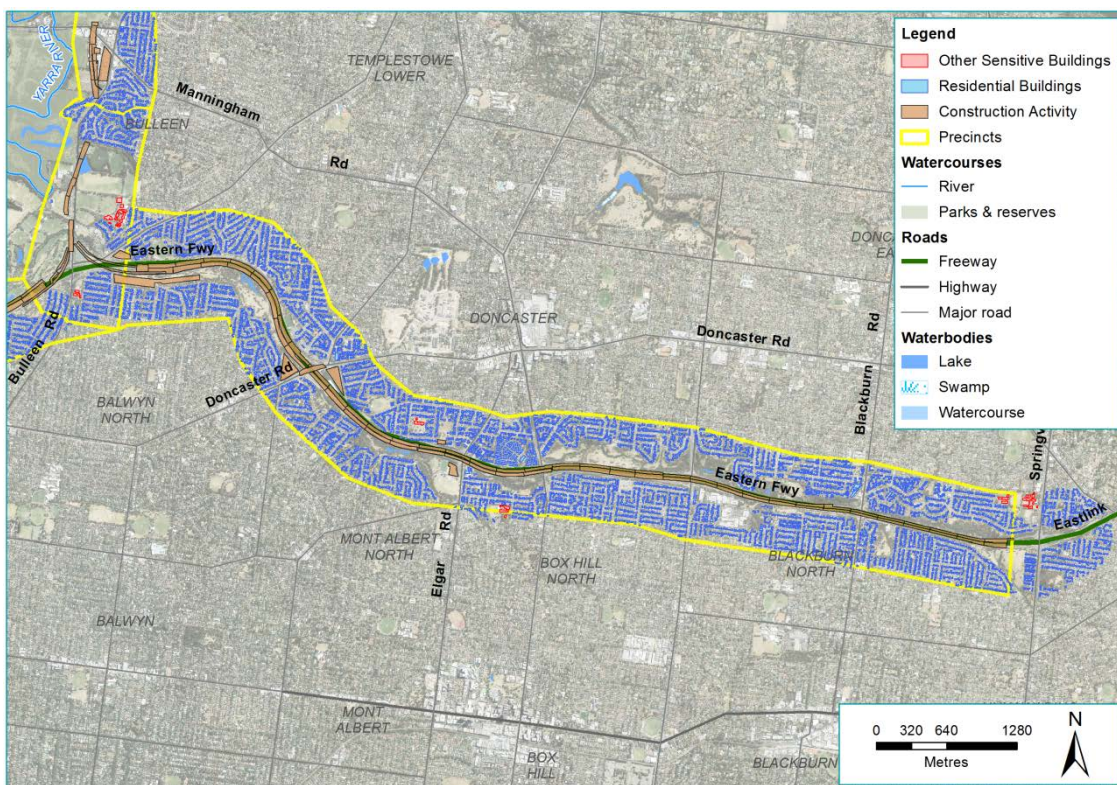


Figure 9.1 d: Precinct 4 (Eastern Freeway upgrades – East)

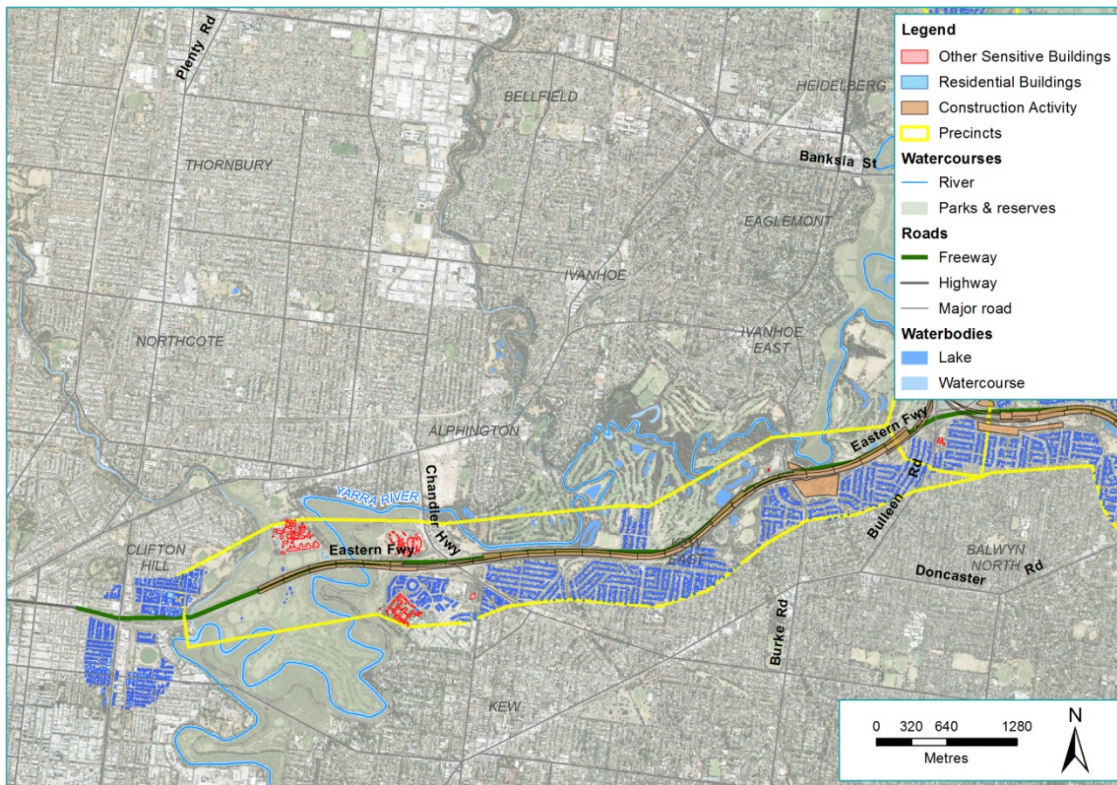


Figure 9.1 e: Precinct 4 (Eastern Freeway upgrades – West)

9.4.2 Construction noise

Surface works

The assessment considered noise generated from a range of equipment and activities during the construction phase of works as detailed in EES Technical report C – Surface noise and vibration.

The majority of construction activities (including most surface activities) are proposed to be undertaken from 7 am to 6 pm Monday to Friday and 7 am to 1 pm on Saturday, with no work on Sunday or public holidays. Some out-of-hours works may be required, such as those that require continuous work or which otherwise would pose an unacceptable risk to life or property or a major traffic hazard. With the exception of emergency works, activities would not take place outside standard hours without prior discussion with and/or notification of local residents, businesses and EPA Victoria, where required.

The assessment of construction noise has considered the range of activities and equipment likely to be required, the location of these works, the times these may be undertaken/use and the duration of these activities.

The noise impact assessment determined that some construction activities have the potential to cause substantial impacts which include:

- The excavation works for the trenched roadway, north of the northern portal
- During the replacement of existing noise walls along the Eastern Freeway, when it would not always be possible to erect the new walls before existing walls were dismantled. This would mean that some residential areas would not be shielded from traffic noise until the new walls are erected. It is the intent that new noise walls are installed before existing walls are dismantled. A specific EPR exists to reinforce the importance of this. At this stage, it is not possible to identify the locations where this would not be possible, as this will be determined during the detailed design phase. Residents would be notified and consulted during this process. However, when it is not possible to erect new walls before existing walls were dismantled there would be a noticeable increase in ambient traffic noise.

Careful mitigation strategies would be required to minimise impacts for construction of the trenched roadway and the replacement of existing noise walls. Potential mitigation measures are identified in EES Technical report C – Surface noise and vibration. These measures include: physical mitigation of plant, equipment and operations; limits to the hours of operation (for some operations); and community consultation. If mitigation was unable to alleviate the impacts, other measures such as temporary relocation could be considered.

The noise impacts associated with the movement of trucks related to the project's construction are assessed in EES Technical report C – Surface noise and vibration. The assessment determined that while the trucks may be identifiable on the roads, they should not significantly increase noise levels. The highest increase from spoil haulage is predicted to be only 0.9 dBA over the average of the day and 0.6 dBA over the average of the night. Impacts from haul trucks are best minimised through management practices, with a range of measures identified for consideration in EES Technical report C – Surface noise and vibration.

Tunnelling

Noise impacts associated with tunnel construction activities are assessed in EES Technical report D Tunnel vibration.

The assessment concluded the construction of the main tunnels, cross passages and portal dive structures would produce perceptible but generally acceptable and manageable levels of regenerated noise. Where impacts would be potentially unacceptable, mitigation measures are available and include adjustments to the scale of the works, real time monitoring and community consultation. Where measures could not reduce regenerated noise levels to acceptable values, other measures such as temporary relocation, vibration isolation devices or noise amelioration measures may be required.

9.4.3 Vibration

Surface works

Potential vibration impacts from surface works are addressed in EES Technical Report C – Surface noise and vibration. Construction activities with the most significant potential for vibration impacts would be from vibratory rollers, rock breakers and driven piling. Potential impacts relating to property damage and human comfort identified a number of properties that may be within the minimum working distance relevant to these impacts. Based on the surface noise assessment, receivers adjacent to the construction areas would likely at times perceive vibration impacts during construction works. This is expected to be primarily due to works associated with vibratory rollers, rock breakers, vibratory and other high vibration plant items.

In practice, vibration impacts from most construction activities would be intermittent during the project's construction. The required locations for vibration-intensive equipment should be reviewed during the detailed design phase, when finalised information relating to the works is available.

The potential impacts from vibration would be considered in the site-specific Construction Noise and Vibration Management Plan (CNVMP). In all cases, it is anticipated that vibration impacts would be able to be controlled to avoid cosmetic damage to any structures.

Measurements of existing ambient vibration levels should be undertaken at receivers identified as having vibration-sensitive equipment or scientific equipment during the detailed design phase.

Tunnelling

Vibration impacts associated with tunnel construction activities are assessed in EES Technical report D – Tunnel vibration.

Construction would likely include activities and equipment that could produce vibration and regenerated noise levels, which are elevated when compared with the existing background environment. Principal vibration-inducing activities are either continuous, or at least semi-continuous, as characterised by a tunnel boring machine (TBM), a road header or an excavator with a hydraulic hammer attachment. While the tunnel vibration assessment has not considered drilling and blasting excavation methods, it has identified EPRs to address impacts of blasting, should it occur.

Construction of the main tunnels, cross passages and portal dive structures would produce perceptible but generally acceptable and manageable levels of vibration. Where impacts were potentially unacceptable, mitigation measures are available and include adjustments to the scale of the works, real time monitoring and community consultation. Where measures could not reduce vibration to acceptable values, other measures such as temporary relocation, vibration isolation devices or noise amelioration measures may be required, as outlined in the relevant EPRs.

9.4.4 Management of noise impacts during construction

Where noise and vibration impacts are below the adopted guidelines (refer to discussion above and the specific details in the relevant technical reports) the potential for health impacts is low. Where there is the potential for noise or vibration to exceed the adopted guidelines, management of these impacts has been identified.

Noise and vibration impacts identified during construction works would be managed with the implementation of EPRs NV3 to NV6, and NV8 to NV12), which include:

- EPR NV3: Management of noise and vibration impacts to sensitive receptors
- EPR NV4: Construction Noise and Vibration Management Plan (CNVMP)
- EPRs NV8 to NV12: Construction vibration targets for amenity and structures (including blasting should it occur).

Where these measures are implemented, the potential for noise impacts to cause significant health impacts in the community is low.

However, it is expected that some individuals within the community may find construction noise discernible and annoying at times, even with mitigation. The management of noise impacts during construction would include a complaints system, as outlined in EPR SC2, where such issues could be identified and addressed.

On the basis of the above, no further detailed assessment of construction noise impacts on health has been undertaken.

9.4.5 Operation

An assessment of operational noise impacts of the reference project was undertaken, which included proposed noise mitigation measures, and the traffic noise objectives adopted for the project. The project traffic noise objectives are outlined in EES Technical report C – Surface noise and vibration.

Operational road traffic noise levels were calculated using SoundPLAN v8.0 software, which implements the Calculation of Road Traffic Noise (CoRTN) algorithm. The UK Department of Transport devised the CoRTN algorithm and with suitable corrections, this method has been shown to give accurate predictions of road traffic noise under Australian conditions. The assessment of noise from operational traffic noise has been undertaken for the following scenarios:

- 2017 existing conditions (existing road traffic volumes)
- 2026 year of opening – with the project (with the proposed road alignment and noise wall design and changes to major other roadways)
- 2036 with the project (10 years after opening with the proposed road alignment, traffic changes on major arterial roads and noise wall design)

- 2036 without the project (based on existing road alignments and noise wall designs, with 2036 traffic predictions).

The focus of the operational noise assessment relates to predicted noise impacts at ground level receivers. It is noted that potential noise impacts under World Health Organisation guidelines (2018) have also been considered on upper floors where multi-level buildings are located.

New or improved noise walls have been included into the future design where the existing noise walls are being upgraded, or there are exceedances of the criteria. The noise wall design considered may be modified during the project's detailed design.

The use of quieter road surface pavements have also been modelled for the project's major roads (main carriageways only).

The assessment has also considered maximum noise levels as concern about sleep disturbance due to individual traffic noise events have been raised by the community.

The modelling of noise impacts considers topographical data for the facilities and surrounding areas, layout and design of the project and the location of sensitive receivers in the vicinity of the project. The modelling also takes into account noise reflections.

It should be noted that strategies are currently being implemented to reduce road traffic noise across the state road network. This may reduce the number of maximum noise levels events over the longer term.

These strategies include local council requirements to include noise mitigation in new dwellings, metropolitan plans to increase the use of public transport, state-wide plans for upgrades of major transport routes.

In addition, state-wide strategies for sharing freight with rail modes are expected to reduce noise from heavy vehicle freight on roads in many areas with a corresponding reduction in high noise levels from road traffic.

Table 9.1 summarises the key outcomes of the operational noise assessment for the five noise precincts considered, as well as for the fixed facilities associated with the tunnels. When comparing the 2036 scenarios 'with project' against the 'no project':

- There is a small overall decrease in median traffic road noise levels at residences within the project corridor
- For the wider road network there is predicted traffic noise on local roads is predicted to reduce. Some local roads show a slight increase in traffic noise, with the greatest increase being 1.4 dBA. In terms of the longer-term assessment of noise, it is widely acknowledged that changes between 2 and 3 dBA are considered to cause minimal impacts and are generally unnoticeable. The largest reductions in noise correspond to those sections of roads currently used by commuters to link between the M80 Ring Road and the Eastern Freeway. The change in noise levels across the wider road network is illustrated in **Figure 9.2**.

On the basis of the operational noise assessment undertaken, 159 properties have been initially been identified as possibly requiring at-property treatment. Of these properties, approximately 46 per cent are just over the operational noise assessment criterion (within 1dBA).

Table 9.1: Summary of operational noise impacts

Precinct	Noise impacts – parklands and community buildings	Noise impacts – Residents
Precinct 1	All noise levels in compliance with relevant noise criteria	At the southern portion of Greensborough Road, North East Link would be completely covered so residents would experience substantial noise reductions of up to 6 dBA. Noise increases may be up to 4 dBA; however, to meet the noise criteria adopted for the project there may be up to 11 properties that qualify for additional mitigation. The final number would be determined during the project's detailed design stage.
Precinct 2	NA	North East Link would be in tunnels in this precinct. An increase in traffic noise less than 2 dBA is predicted at the entry/exit ramps at the Manningham Road interchange, Noise levels associated with surface roads would generally be reduced by 1.5 to 2.5 dBA.
Precinct 3	Predicted noise levels relevant to community buildings in this precinct comply with relevant noise criteria. There would be potential increases in noise up to 5 dBA at Carey Grammar Oval, Marcellin College Ovals and Trinity Grammar School Sporting Complex. The design of flood walls that act as noise walls is not finalised, but when considered these would likely reduce noise impacts so they were not noticeable.	Residents would experience noise reductions of up to 5 dBA at a number of areas in this precinct. The new interchange would mean noticeable increases to some buildings in the surrounding area. Noise increases may be up to 2 dBA; however, to meet the noise criteria adopted for the project there may be up to 13 properties that qualify for additional mitigation. The final number would be determined during the project's detailed design stage.
Precinct 4	Predicted noise impacts at community buildings in this precinct comply with the relevant noise criteria. New noise walls at some locations along the project's alignment would reduce noise levels to sections of parkland along the Eastern Freeway, including at locations where noise walls are currently not provided. This is expected to reduce noise by at least 3 dBA at: Frank Sedgman Reserve, Manningham Park Reserve, and the western portion of the Eastern Freeway Linear Reserve. Additionally, some noise walls along the Eastern Freeway would be replaced with higher walls. Some parks would not benefit from new or replaced noise walls and in these areas, the noise levels may increase by 1 to 4 dBA (with higher increases closer to the roadway). These increases would be noticeable enough to cause any loss of amenity to users of the various parks.	Noise at residential properties would vary in this precinct, depending on the height of noise walls, the height of the property relative to the roadway and whether there is a break in the noise wall (to accommodate other structures such as overpasses and cycling paths). Noise would reduce by up to 7 dBA at a number of properties. However, at approximately 128 properties residual noise impacts may remain, at-property noise mitigation may be required. Additional noise mitigation requirements would be determined at the project's detailed design phase.

Precinct	Noise impacts – parklands and community buildings	Noise impacts – Residents
Precinct 5	The recreational spaces of Musca Street Reserve, Hays Paddock and the park adjacent to Columbia Street in this precinct would all benefit from the new higher noise walls required for the adjoining residential areas. Noise levels within the parkland between east of Dights Falls Reserve and the Yarra Boulevard area, including the H. H. Oley and E.O. McCutchans Ovals and Fairlea Oval West may experience a small increase in noise of less than 3 dBA but the levels would comply with the overall noise goals relevant to the project.	Noise would reduce by up to 7 dBA at a number of properties in this precinct. However, residual noise impacts may remain at seven properties and at-property noise mitigation may be required. The final number would be determined during the project's detailed design stage.
Fixed facilities for tunnel	Noise modelling identified that noise mitigation would be required on several plant items to achieve compliance with the relevant external noise criteria. The mitigation measures include the use of acoustic attenuators/silencers on the intake and discharge of the in-tunnel jet fans and the design of the ventilation system.	

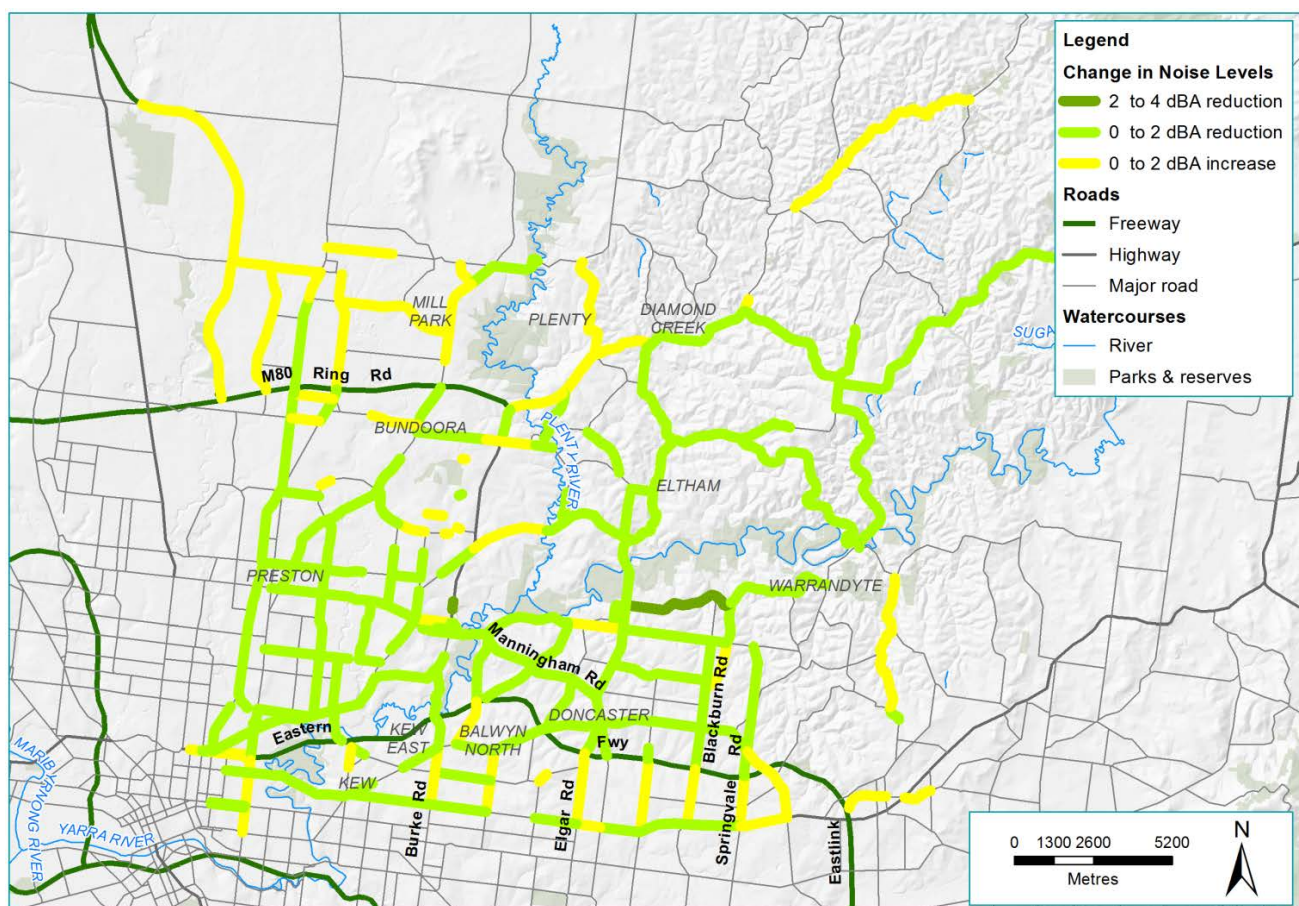


Figure 9.2: Noise changes on wider road network (2036: with project compared with no project)

Maximum noise events are most commonly related to the use of engine breaks. EES Technical report C – Surface noise and vibration has indicated the following in relation to engine noise.

Engine brakes are one type of secondary braking device fitted to a heavy vehicle intended to assist in slowing a heavy vehicle, rather than stop it. Engine brakes are predominantly used on the downhill sections of road to slow down, but could occasionally be used on flat or up-hill sections if the truck needed to slow or stop quickly.

Engine brakes are frequently reported as being a source of noise complaints from people, due to the character of the noise rather than the absolute level of the noise. There are no Australian Design Rules associated with the levels of noise that can be emitted from their use, although regulators in Australia have attempted to address the noise issue through measures including Offensive Noise provisions in the regulations. These measures have tended to be ineffective and so the associated noise emissions are largely unregulated. VicRoads does not provide any requirements to provide noise mitigation to control maximum noise levels. Rather, an assessment of the maximum noise levels can be used to assist the planning and design of new roads and ramps.

Along the majority of the Eastern Freeway (between Plenty Road and the Greensborough Bypass), the vertical alignment would not substantially change (though the road would be widened). It is therefore considered unlikely the number of engine braking events would increase, aside from the increase in the overall number of trucks on the roadway (commensurate with the increase in capacity). As the project would allow the traffic travelling on the main corridor to flow at a more consistent speed, this would be expected to lead to less reliance on braking and consequently reduce the use of engine brakes.

Residents living along the approaches or near the signalised intersections at the corner of the Greensborough Bypass and the M80 Ring Road, and the Greensborough Bypass and Grimshaw Street should benefit from the elimination of the traffic lights to enable more free-flowing traffic. This should lead to a decrease in the need to use engine brakes.

Overall noise from individual truck movements (including engine brakes) is a driver-related issue and signage would be used to assist in minimising impacts.

9.5 Health impacts relevant to noise

9.5.1 General

The assessment of noise impacts summarised in **Section 9.3** evaluated noise impacts against regulatory noise criteria relevant to the project. Not all these criteria specifically link to the protection of health and so a more detailed assessment of noise impacts on health has been undertaken. The assessment of noise impacts focused on the impacts of changes in the noise environment on community health.

Environmental noise has been identified (I-INCE, 2011; WHO, 2011, 2018) as a growing concern in urban areas because it has negative effects on quality of life and wellbeing and has the potential for causing harmful physiological health effects. With increasingly urbanised societies, impacts of noise on communities have the potential to increase over time.

Deciding on the most effective noise management options in a specific situation is not just a matter of defining noise control actions to achieve the lowest noise levels or meeting arbitrarily chosen criteria for exposure to noise. The goal should be designed to achieve the best available compromise between the benefits to society of reduced exposure to community noise versus the costs and technical feasibility of achieving the desired exposure levels given the project. On the one hand, there are the rights of the community to enjoy an acceptably quiet and healthy environment. On the other hand, there are the needs of the society for new or upgraded facilities, industries, roads, and recreation opportunities, all of which typically produce more community noise (I-INCE, 2011; WHO, 2011, 2018).

Sound is a natural phenomenon that only becomes noise when it has some undesirable effect on people or animals. Unlike chemical pollution, noise energy does not accumulate either in the body or in the environment, but it can have short-term and long-term adverse effects on people. These health effects include (WHO, 1999, 2011, 2018):

- Sleep disturbance (sleep fragmentation that can affect psychomotor performance, memory consolidation, creativity, risk-taking behaviour and risk of accidents)
- Annoyance
- Cardiovascular health
- Hearing impairment and tinnitus
- Cognitive impairment (effects on reading and oral comprehension, short and long-term memory deficits, attention deficit).

Other effects for which evidence of health impacts exists, and are considered to be important, but for which the evidence is weaker, include:

- Effects on quality of life, wellbeing and mental health (usually in the form of exacerbation of existing issues for vulnerable populations rather than direct effects)
- Adverse birth outcomes (pre-term delivery, low birth weight and congenital abnormalities)
- Metabolic outcomes (type 2 diabetes and obesity).

Within a community, the severity of the health effects of exposure to noise and the number of people who may be affected are schematically illustrated in **Figure 9.3**.

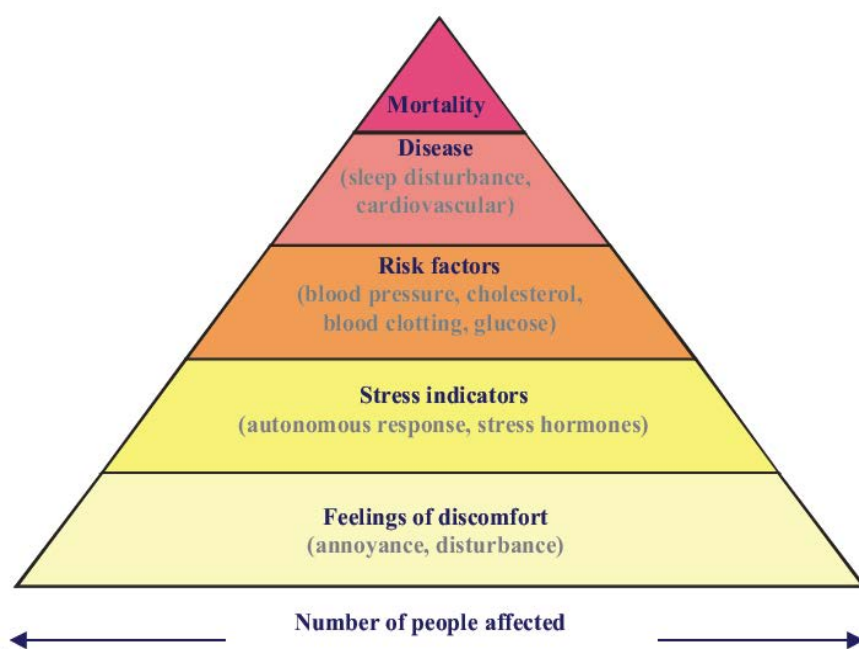


Figure 9.3: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO, 2011)

Often, annoyance is the major consideration because it reflects the community's dislike of noise and their concerns about the full range of potential negative effects, and it affects the greatest number of people in the population (I-INCE, 2011; WHO, 2011, 2018).

There are many possible reasons for noise annoyance in different situations. Noise can interfere with speech communication or other desired activities. Noise can contribute to sleep disturbance which has the potential to lead to other long-term health effects. Sometimes noise is just perceived as being inappropriate in a particular setting without there being any objectively measurable effect at all. In this respect, the context in which sound becomes noise can be more important than the sound level itself (I-INCE, 2011; WHO, 2011, 2018).

Different individuals have different sensitivities to types of noise and this reflects differences in expectations and attitudes more than it reflects any differences in underlying auditory physiology. A noise level that is perceived as reasonable by one person in one context (such as in their kitchen when preparing a meal) may be considered completely unacceptable by that same person in another context (such as in their bedroom when they are trying to sleep). In this case, the annoyance relates partly to the intrusion from the noise. Similarly, a noise level considered to be completely unacceptable by one person may be of little consequence to another, even if they are in the same room. In this case, the annoyance depends almost entirely on the personal preferences, lifestyles and attitudes of the listeners concerned (I-INCE, 2011; WHO, 2011, 2018).

Perceptible vibration (such as from construction activities) also has the potential to cause annoyance or sleep disturbance, causing adverse health outcomes in the same way as airborne noise. However, the health evidence available relates to occupational exposures or the use of vibration in medical treatments. No data is available to evaluate health effects associated with community exposures to perceptible vibrations (I-INCE, 2011; WHO, 2011, 2018).

It is against this background that an assessment of potential noise impacts of the project on health was undertaken.

9.5.2 Health impacts from road traffic noise

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

A number of large international studies have specifically evaluated health impacts associated with exposure to road traffic noise. Where exposure to road traffic noise is associated with, or can be shown to be causal, adverse health effects an exposure-response relationship is often established. The main health effects that have been studied in these types of investigations in relation to road traffic noise are annoyance, sleep disturbance, cardiovascular disease, stroke and memory/concentration (cognitive) effects. The most recent review of noise and impacts on health, presented by the WHO (WHO, 2018) included a detailed review of the available literature, including impacts specifically related to road noise.

Cardiovascular effects

Cardiovascular diseases are the class of diseases that involve the heart or blood vessels (arteries and veins). These diseases can be separated by end target organ and health outcomes. Strokes reflecting cerebrovascular events and ischaemic heart disease (IHD) or coronary heart disease (CHD) are the most common representation of cardiovascular disease.

High-quality epidemiological evidence on cardiovascular and metabolic effects of environmental noise indicates that exposure to road traffic noise increases the risk of IHD.

A link between noise and hypertension is relatively well established in the relevant literature. While there is not a consensus on the precise causal link between the two, there are a number of credible hypotheses. A leading hypothesis is that exposure to noise could lead to triggering of the nervous system (autonomic) and endocrine system which may lead to increases in blood pressure, changes in heart rate, and the release of stress hormones. Depending on the level of exposure to excess noise, the duration of the exposure and certain attributes of the person exposed, this can cause an imbalance in the person's normal state (including blood pressure and heart rate), which may make a person hypertensive (consistently increased blood pressure) which can then lead to other cardiovascular diseases (DEFRA, 2014). This hypothesis is illustrated in **Figure 9.4**.

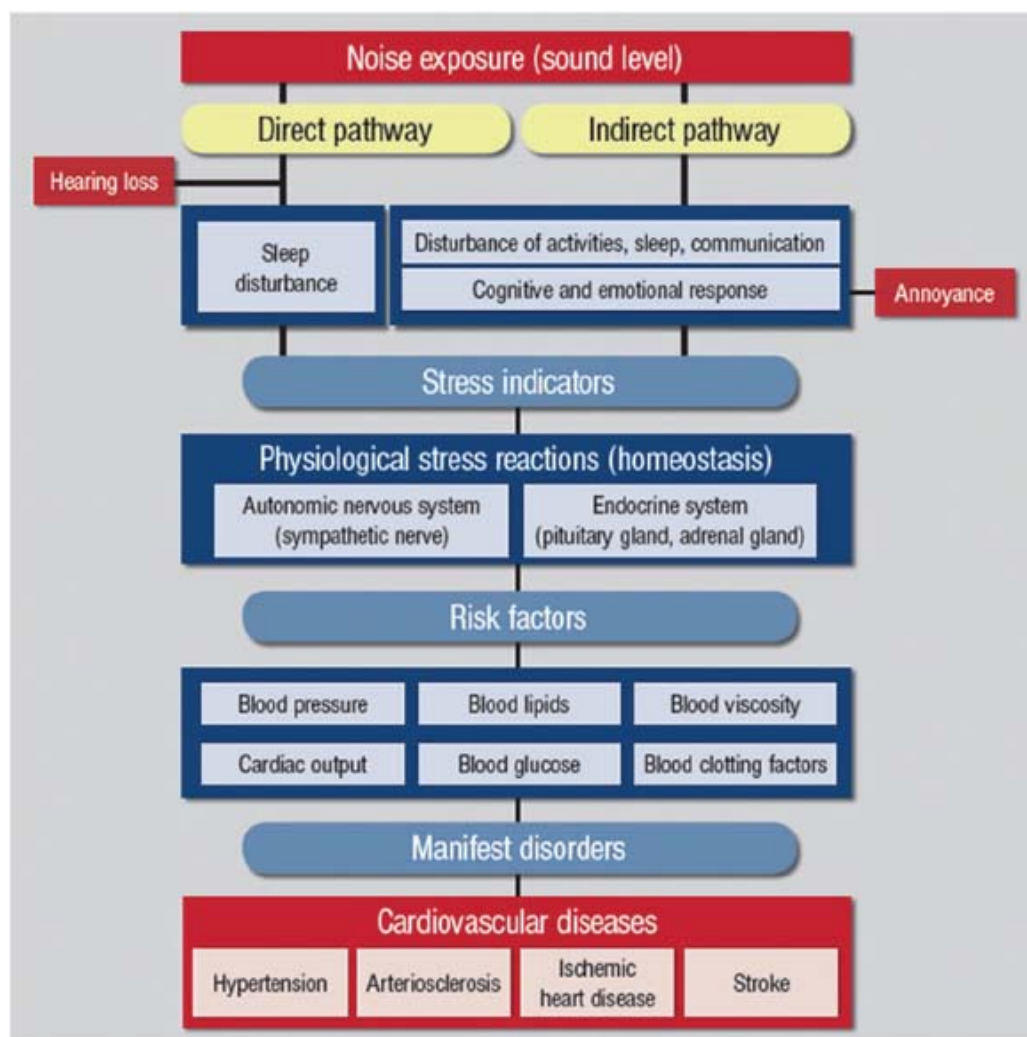


Figure 9.4: Noise reaction model/hypothesis (Babisch, 2014)

The available studies regarding road traffic noise and cardiovascular disease risk largely involve meta-analysis (statistical analysis that combines the results of multiple scientific studies). A number of studies have been published by Babisch (Babisch, 2002, 2006, 2008, 2014; van Kempen & Babisch, 2012) and others (WHO, 2018) have provided the basis for a number of exposure-response relationships adopted for the assessment of cardiovascular health effects associated with road-traffic noise.

In relation to hypertension the most relevant recent study (van Kempen & Babisch, 2012) involved analysis of 27 studies between 1970 and 2010, where a relationship between road traffic noise and hypertension was determined. This relates to the incidence of hypertension in the population and has been adopted by the European Commission for the assessment of health impacts of road noise in Europe (EEA, 2014). Review by the WHO (2018) considered the available studies on the incidence of hypertension and road noise provided evidence that was rated very low quality. The relationship recommended by the WHO relates to a non-statistically significant outcome in relation to hypertension. On this basis the relationship as adopted by the European Commission (EEA, 2014) has been used in this assessment.

For the assessment of IHD, the WHO (2018) has undertaken a meta-analysis of three cohort studies and four case-control studies that investigated a relationship between road noise and the incidence of IHD. The meta-analysis involved 67,224 participants (from 7,033 cases). The relationship established by the WHO, which is specific to road noise, has been adopted in this assessment. The relationship established was considered to be based on high quality evidence.

Review of the incidence of stroke and road noise by the WHO (2018) determined the available cohort studies and cross-sectional studies showed mixed outcomes, with the evidence rated very low to moderate quality. In relation to the risk of stroke from exposure to noise, there are limited meta-analysis type studies available and the studies available combine the risks from noise from road and air transport. A more specific study that just investigated the link between road traffic noise and cardiovascular disease/mortality has been undertaken in London (Halonen et al., 2015). This was a large epidemiological study that identified statistically significant associations between road traffic noise (as modelled to residential dwellings) and hospital admissions for stroke and all-cause mortality. The relationships identified related to exposure to day and evening noise as $L_{Aeq,16h}$. The study corrected for confounders such as $PM_{2.5}$ and NO_2 exposures and has been considered suitable for use in this assessment. The relative risk identified for hospital admissions for stroke is equivalent to that identified from a meta-analysis of air and road noise (Houthuijs et al., 2014).

The relative risks adopted in this assessment to evaluate the above health effects, associated with exposure to road traffic noise, are presented in **Table 9.2**.

Annoyance and sleep disturbance

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

Annoyance

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

Annoyance levels can be reliably measured by means of an ISO 15666 defined questionnaire, which has enabled the identification of relationships between annoyance and noise sources. The European Commission (EC, 2002) reviewed the available data and provided recommendations on relationships that define the percentage of persons annoyed (%A) and the percentage of persons highly annoyed (%HA) to total levels of noise reported as L_{DEN} (that is, average noise levels during the day, evening and night). These relationships were established for exposure to aircraft noise, road traffic noise and rail traffic noise, and have been adopted by the UK and European Environment Agency (DEFRA, 2014; EEA, 2010, 2014). These relationships have also been reviewed by the WHO (WHO, 2018), where the key outcome of %HA was considered most appropriate for determining actions and outcomes in relation to road noise. This health impact assessment has therefore focused on %HA, adopting a more recently developed relationship from a systematic review of the available studies relevant to road noise (Guski et al., 2017). The relationship adopted in this assessment is more relevant to flat landscapes (that is, with alpine and Asian studies excluded, which include significant terrain features).

The relationship recommended for the assessment of the percentage of individuals annoyed (as %HA) from road noise is presented in **Table 9.2**.

Sleep disturbance

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

Studies are available that have evaluated awakening by noise, increased mortality (that is, an increase in body movements during sleep), self-reported chronic sleep disturbances and medication use (EC, 2004). The most easily measurable outcome indicator is self-reported sleep disturbance, where there are a number of epidemiological studies available. From these studies the WHO (WHO,

2009, 2011, 2018) identified an exposure response relationship that relates to the percentage of persons sleep disturbed (%SD) and highly sleep disturbed (%HSD) to total levels of noise reported as L_{night} (that is, average noise levels during night, which is an 8-hour time period, as measured outdoors). The relationship adopted relates to the assessment of road-traffic noise, with other relationships for air and rail traffic noise. These relationships have been adopted by the WHO (2009, 2011), UK and European Environment Agency (DEFRA, 2014; EEA, 2010, 2014). Review by the WHO (2018) considered the key outcome of %HSD was considered most appropriate for determining actions and outcomes in relation to road noise. This health impact assessment has thus focused on %HSD.

The relationship recommended for the assessment of the percentage of individuals who are highly sleep disturbed from road noise is presented in **Table 9.2**.

Cognitive effects

There is evidence for effects of noise on cognitive performance in children such as lower reading performance (WHO, 2011). A major study was undertaken in the European Union (RANCH) and this study was reviewed in WHO (2011). The study found an exposure response relationship between noise and cognitive performance in children for aircraft noise but the relationship between performance and noise for road traffic was much less clear (Stansfeld et al., 2005a; Stansfeld et al., 2005b; WHO, 2011, 2018). The WHO (2011) used the aircraft noise relationships to assess the impact of noise on children's cognitive performance. For North East Link, it was not considered appropriate to use the relationships based on the impacts of aircraft noise. The same study showed that road traffic alone did not show an association between road traffic noise and adverse changes in children's cognitive functions studied (reading comprehension, episodic memory, working memory, prospective memory or sustained attention), nor with sustained attention, self-reported health, or mental health.

Exposure-response relationships

Table 9.2 summarises the exposure-response relationships considered relevant for the assessment of noise impacts of North East Link. The assessment of health effects associated with exposure to noise have been identified to occur (or increase above background) above some threshold. The lower threshold for effects (that is, the level above which the relationship is applied) is also thus presented in the table.

It is noted the exposure-response relationships presented relate to long-term average noise levels (an annual average noise level). By using these relationships to evaluate predicted noise levels that relate to maximum noise impacts, the assessment would provide a conservative evaluation.

Table 9.2: Exposure-response relationships for assessment of noise impacts on health

Health effect	Noise measure*	Threshold or range (dB)	Exposure-response relationship (per 10 dB increase in noise [95% confidence interval], unless other relationship presented)	Reference
Ischaemic heart disease: hospitalisations	L _{den}	53	RR = 1.08 [1.01-1.15]	(WHO 2018)
Hypertension (incidence)	L _{den}	47	RR# = 1.07 [1.02-1.12]	(van Kempen & Babisch 2012)
Stroke: hospital admissions	L _{Aeq,16h}	55-60 >60	RR = 1.04 [1.02-1.07] RR = 1.05 [1.02-1.09]	(Halonen et al. 2015)
Mortality: all causes	L _{Aeq,16h}	55-60 >60	RR = 1.03 [1.01-1.05] RR = 1.04 [1.00-1.07]	
Annoyance	L _{den}	42	%HA = 116.4304 – 4.7342 x L _{den} + 0.0497 x L _{den} ²	(Guski et al. 2017)
Sleep disturbance	L _{night}	Threshold for health effects ranges from 32 to 60 Who (2018) threshold for %HSD = 43	%HSD = 20.8 - 1.05L _{night} + 0.01486(L _{night}) ²	(EEA 2010; WHO 2009, 2011)

RR = relative risk

* Noise measure relates to noise levels measured over different time periods (as noted) outdoors (for all health effects evaluated)

HA = highly annoyed; HSD = highly sleep disturbed

It is noted that noise impacts can also occur because of individual noise events, such as engine braking or loud exhausts. The noise measures adopted above for the assessment of the health effects of noise relate to an average/equivalent sound level over different daily time periods, which would include individual noise events. Direct noise measurement is the preferred approach for evaluating annoyance and other health effects related to noise (NSW DECCW, 2011). Individual noise events are of most significance to the assessment of sleep disturbance. The available research indicates that one or two individual noise events per night, with a maximum indoor noise level of 65-70 dB(A) are not likely to affect health and wellbeing (NSW DECCW, 2011). However, it is noted that in relation to the assessment of future road noise, it is not possible to model individual noise events as these relate to individual vehicles or trucks and individual driving behaviour that cannot be predicted. This health impact assessment has therefore addressed health effects based on the relationships identified and outlined in **Table 9.2**.

9.5.3 Assessment of noise impacts from the project

The assessment of health impacts for a population exposed to changes in noise associated with North East Link was undertaken utilising the methodology outlined in **Appendix B** where the exposure-response relationships (presented in **Table 9.2**) have been directly considered.

As discussed in **Section 9.1**, the assessment has considered population health impacts as well as localised health impacts.

Health risks have been calculated based on:

- Estimates of the changes in noise exposure levels associated with the project (without project or base case compared to with project) for the year 2036 for receptors in all noise precincts (as provided in EES Technical report C – Surface noise and vibration) have been assessed on the basis of local government areas (LGAs). The changes in noise relate to impacts at the noise receptors evaluated in each noise precinct from the proposed project alignment and major arterial road traffic flows, and the proposed noise wall design. Changes in noise adjacent to other key roadways in the project area have been assessed only on the basis of the maximum impact associated with the maximum predicted increase in noise. The assessment of population risk has considered the average change in noise at these receptors, while the assessment of localised risk has evaluated the change in noise at every individual receptor.
- Baseline incidence of the key health endpoints that are relevant to the population exposed, namely hypertension, ischaemic heart disease, hospital admissions for stroke and mortality (all causes) (refer to **Table 6.4**).
- Exposure-response relationships expressed relevant to a 10 dB(A) change in noise level (refer to **Table 9.2**). These are only relevant to apply when the total predicted noise level (project plus existing noise levels) is above the thresholds as outlined in **Table 9.2**. No calculations are undertaken for total predicted noise levels below this threshold, as no health effects occur.

The change in incidence of each health indicator relevant to changes in noise exposures in the local community (for the population exposed) has been calculated based on:

- For each of the receptors evaluated the change in noise level has been determined, along with the number of residential properties at the receptor location
- For each property identified, the average number of people per household in the relevant LGA has been used to estimate the population who may be exposed to the change in noise level
- Once the relevant change in noise has been determined, the change in the incidence of each health endpoint, namely the incidence of hypertension, ischaemic heart disease, hospital admissions for stroke and mortality (all causes) has been calculated using the methodology outlined in **Appendix B**
- The total change in incidence for all areas assessed is the sum of the calculated incidence for each receptor in a LGA and then for all the receptors in the community evaluated as a whole.

The following noise measures were provided as outputs from the noise modelling completed by SLR (2018), which are relevant for the assessment of health impacts:

- $L_{A10,18\text{-hour}}$ – this has been taken to be representative of $L_{Aeq,16\text{-hour}}$ (note the calculations undertaken utilising this metric utilises the change in $L_{Aeq,16\text{-hour}}$, which will always be equal to the change in $L_{A10,18\text{-hour}}$)
- L_{den}
- $L_{Aeq\text{ night}}$

Assessment of annoyance and sleep disturbance does not use an exposure response relationship that relates to a 10 dB(A) change in noise exposure. These effects are calculated as a percentage of the population affected, and this is based on the use of an equation that incorporates the total noise exposure at the receptors. As the areas evaluated include a number of areas currently affected by noise, the assessment of annoyance and sleep disturbance has focused on changes associated with the project. This has involved calculating the percentage of people annoyed and sleep disturbed in the base case (no project) with the project and determining the change associated with the project.

Appendix H presents the calculations undertaken to estimate population and localised health impacts from changes in noise for each health endpoint outlined in **Table 9.2**.

The above measures relate to maximum noise predictions. The relationships presented in **Table 9.2** relate to annual average noise levels, which would be lower than the maximum levels evaluated in this assessment. The outcomes of the noise impact assessment on health is thus expected to be conservative.

The calculations undertaken have considered potential changes in noise at the ground floor as well as for upper floors, where relevant to the receptors being evaluated.

Appendix H also presents figures that show the location of noise increases in each of the noise catchment areas evaluated. These figures illustrate the locations where noise impacts are greatest.

Population health impacts

Table 9.3 presents the calculated population health impacts, as the change in risk and health incidence (number of people) from changes in noise within the LGAs, and whole project area. The table presents health impacts calculated for all areas evaluated from the project as well as the redistribution of traffic on key surface roads, where no further noise mitigation measures are implemented.



Table 9.3: Population health impacts associated with changes in noise – 2036

Area/Scenario	Change in risk for key health endpoints Maximum			Change in population incidence for key health endpoints (persons)			
	Mortality: all causes (all ages)	Hospitalisations: Stroke (all ages)	Hospitalisations: Ischaemic heart disease	Mortality: all causes (all ages)	Hospitalisations: Stroke (all ages)	Hospitalisations: Ischaemic heart disease	Incidence of hypertension
Banyule LGA	-6E-06	-2E-06	-2E-05	-0.12	-0.03	-0.2	-13
Boroondara LGA	-8E-06	-2E-06	-3E-05	-0.12	-0.03	-0.2	-11
Darebin LGA	-3E-06	-1E-06	-1E-05	-0.006	-0.002	-0.02	-0.3
Manningham LGA	-6E-07	-2E-07	-2E-06	-0.01	-0.003	-0.02	-2
Whitehorse LGA	2E-06	6E-07	6E-06	0.02	0.005	0.04	2
Yarra LGA	-7E-06	-3E-06	-4E-05	-0.02	-0.01	-0.06	-7
Total impact in community	-3E-06	-1E-06	-1E-05	-0.2	-0.07	-0.4	-31

-1x10⁻⁶ = negative change in individual risk which means there is a reduction in risk, or potential for improvement in health. Positive values relate to impacts on health from the project

-0.0002 = negative change in population incidence which means the total change in incidence of the health endpoint in the community evaluated is a reduction (that is, health improvement). Positive values are related to impacts on health from the project

Table 9.2 presents the calculated change in population levels of noise annoyance and sleep disturbance. The changes in the proportion of the population that is highly annoyed and/or highly sleep disturbed have been evaluated based on the project criteria outlined in **Section 9.1** and discussed in further detail in **Appendix D**.

Table 9.4: Changes in population annoyance and sleep disturbance – 2036

Location/Scenario	Health endpoint – Change in percentage of population affected (persons)	
	Highly annoyed	Highly sleep disturbed
Banyule LGA	-1.0%	-0.6%
Boroondara LGA	-0.9%	-0.6%
Darebin LGA	-0.4%	-0.2%
Manningham LGA	-0.05%	-0.02%
Whitehorse LGA	0.1%	0.1%
Yarra LGA	-2%	-1%
Total impact in community	-0.6%	-0.4%

Review of **Table 9.3** and **Table 9.4** indicates:

- The population risks and health incidence values are negative for all LGAs except the City of Whitehorse. This means that throughout all receptors in these LGAs, there is an overall reduction in noise, which means potential for improved health outcomes. It is noted that while health benefits within the community have been calculated, the size of the benefit is generally small and unlikely to be measurable within the community.
- Within the City of Whitehorse, there is a small increase in population risks and health incidence. The calculated population risks are below the criteria adopted for acceptable population risks and the calculated increase in population incidence is small and would not be measurable within the community.
- Hypertension is a relatively common condition in the population, affecting 17.9 to 25.9 per cent of the population in the LGAs evaluated, with the incidence greater in older age groups. The change in the population incidence of hypertension is a decrease of 31 cases annually (that is, potential health improvement). While this outcome is of potential benefit to the community, the decrease calculated is relatively small compared with the baseline.
- With the population evaluated, there is an overall small decrease in the percentage who may be highly annoyed and/or highly sleep disturbed. This indicates that at a population level the project offers some health benefit to the community, albeit a small benefit.

Localised health impacts

As the project would involve the redistribution of traffic, this could have localised changes in noise and health impacts. Health impacts from localised changes in noise due to the project have been assessed to assist in evaluating the significance of the maximum impacts and inform the need for risk management. As a result, the maximum calculated risks are presented along with localised risks calculated for specific locations, and these should be considered semi-quantitative only.

Assessment of changes in localised noise impacts identified a maximum increase in health risk of 1.9×10^{-4} for mortality (all causes and all ages) and 3.1×10^{-4} for ischaemic heart disease hospitalisations (all ages). These maximum increases in localised risk relate to predicted noise levels on the ground floor. Lower levels of risk relate to predicted noise impacts on the first and upper floors.

Further review of these maximum localised health impacts determined that all predicted risks that were at or exceeded the adopted risk management level of 1×10^{-4} were properties already identified as requiring additional at-property noise treatment to reduce noise exposures in EES Technical report C – Surface noise and vibration. This relates to 159 individual properties. Application of these, already identified noise mitigation measures would adequately address the noise impacts identified for ground floor areas. As noted in EES Technical report C – Surface noise and vibration, where impacts are considered for the upper floors, the number of properties that exceed the noise criteria (WHO noise guidelines) increases to 160. It is relevant that noise impacts relevant to the design phase of the project are fully considered to identify all properties that required at-property treatments.

Further assessment has been undertaken in relation to the maximum localised health impact from changes in noise, after all properties already identified for at-property noise treatment are considered to already have noise mitigation measures applied. Where this is undertaken, **Table 9.5** presents the maximum change in localised health risk and **Table 9.6** presents the maximum change in highly annoyed and highly sleep disturbed in each of the LGAs evaluated.

Table 9.5: Localised (maximum) health risks associated with changes in noise – 2036

Location/area	Change in risk for key health endpoints – maximum		
	Mortality: all causes (all ages)	Hospitalisations: Stroke (all ages)	Hospitalisations: Ischaemic heart disease
Banyule LGA	1E-04	3E-05	1E-04
Boroondara LGA	7E-05	2E-05	1E-04
Darebin LGA	3E-05	1E-05	1E-04
Manningham LGA	1E-04	3E-05	1E-04
Whitehorse LGA	7E-05	2E-06	1E-04
Yarra LGA	1E-05	5E-06	3E-05
Surface roads – redistribution	4E-05	9E-06	6E-05

1E-04 = Change in localised noise and so risk is equal to or exceeds the risk management guideline adopted for the assessment of localised impacts.

Table 9.6: Changes in localised levels of annoyance and sleep disturbance – 2036

Location/area	Health endpoint – maximum change in percentage of population affected (persons)	
	Highly annoyed	Highly sleep disturbed
Banyule LGA	5%	2%
Boroondara LGA	3%	2%
Darebin LGA	2%	1%
Manningham LGA	5%	2%
Whitehorse LGA	3%	2%
Yarra LGA	0.9%	0.6%
Surface roads – redistribution	1.1%	0.8%

A review of **Table 9.5** and **Table 9.6** indicates:

- The maximum localised risk from the maximum increase in noise in all areas evaluated, including adjacent to local key surface roads, are less than or equal to the adopted risk management level.
- The maximum change in the localised proportion of the population that is highly annoyed or highly sleep disturbed are less than or equal to the criteria adopted for determining acceptable changes in these measures. The maximum predicted changes are therefore considered to be acceptable and are not expected to be noticeable.
- It is expected the maximum changes in noise are conservative and provide an over estimate of the maximum increase in noise. This is because the noise model is conservative in that it includes a 'safety factor' and does not include attenuation provided by property boundary fencing, or non-habitable buildings on a property (such as garden shed). **Section 11.2** further discusses other aspects of the assessment of noise health impacts that are conservative. During the project's detailed design, it is reasonable to expect that with more detailed modelling and possible modifications to the road design, the noise predictions would be reviewed and updated, and it is likely the maximum localised impacts (taking all mitigation measures into account) will be lower than considered in this assessment.
- On the basis of the above, localised noise impacts are not considered to be of significance in relation to health.

Overall, calculated population risks and population incidence (for all health endpoints considered) associated with changes in noise from the project are negative indicating some potential health benefit to the community from an overall reduction in community noise. These changes, while indicative of some health benefit are small would not be measurable in the community.

While there are some localised areas where increases in noise levels have been identified, additional noise mitigation has already been identified for these properties within the EES Technical report C *Surface works noise and vibration impact assessment*. Where these additional noise mitigation measures are considered, the maximum localised changes in noise associated with the project are not considered to be of significance in relation to impacts on community health.

Regardless of the outcomes outlined above, changes in noise levels in the community due to the project are expected to comply with the noise limits outlined in EPR NV1 and NV13, with monitoring undertaken in accordance with EPR NV2 and NV7.

9.6 Overview of health impacts of noise

The assessment undertaken in relation to health impacts that may occur due to changes in noise and vibration resulting from the project has concluded:

- **Construction:** Noise and vibration impacts identified during construction works are to be managed through the implementation of a Construction Noise and Vibration Management Plan (CNVMP) as outlined in EPR NV4. Specific mitigation and management measures required to manage noise and vibration impacts in the community during construction are presented in EES Technical report C *Surface noise and vibration*. Where these measures are implemented the potential for noise impacts to result in significant health impacts in the community is low (with residual risk considered to be medium).

However, it is expected, that some individuals within the community may find construction noise annoying at times, even with mitigation. The management of noise impacts during construction would need to include a complaints system, as outlined in EPR SC2, where such issues can be identified and addressed.

- **Operations:** Overall, changes in noise levels associated with the project, where the mitigation measures as outlined in EPR NV1 and NV13 are adopted, are not expected to result in health impacts within the community that would be measurable.

It is noted that for most areas changes in traffic would reduce noise exposure levels to residents adjacent to key local roads. In these areas, the lower levels of noise may be of some benefit to health. In some other areas there are expected to be localised increases in noise. Where mitigated these localised increases in noise are not considered to be of concern to health.

10. Assessment of social impacts on health

10.1 General

The World Health Organisation (WHO) defines health as ‘a (dynamic) state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity’. The assessment of health should thus include the traditional and medical definition that focuses on illness and disease as well as the more-broad social definition that includes the general health and wellbeing of a population.

The assessment of changes in air quality and noise on the health of the local community (presented in **Sections 8 and 9**) addressed key aspects that have the potential to directly affect health.

However, a range of other impacts are associated with the project that can affect the health and wellbeing of the community in a more indirect way. In addition, changes within a community that may be associated with the project may be differentially distributed. This may affect population groups that may be advantaged or disadvantaged based on age, gender, socioeconomic status, geographic location, cultural background, aboriginality, and current health status and existing disability. This aspect relates to the equity of the impacts in the local community.

This section more specifically evaluates changes in the community that have the potential to indirectly affect the health and wellbeing of the community. In addition, this section provides a review of whether there are any impacts likely to be more significant in any section of the community, and if these areas may result in inequitable impacts on the health of the population.

The evaluation presented in this section provides a qualitative evaluation of potential health impacts on the community.

This assessment has drawn on information provided in a number of EES Technical reports (as referenced) and provides a summary of key (and relevant) aspects for some aspects. All details relevant to the underlying assumptions, methodology and interpretation of impacts are provided within these technical reports. Where more detail than provided in the health impact assessment is required, the reader is directed to the individual technical reports.

10.2 Changes in traffic

10.2.1 General

Changes in traffic and transport due to the project are evaluated in detail in EES Technical report A – Traffic and transport. That report provides an overview of the transport changes with the potential to benefit or impact community health.

Without the project, traffic volumes in the north-east are predicted to increase, particularly along arterial and local roads. Traffic movements in the area would continue to be dominated by private vehicles, which currently utilise a limited number of arterial roads for movements north-south. Current movements through the study area are influenced by the relatively sparse arterial road network in the north-east and the presence of the Yarra River, which only has five river crossings. This results in traffic congestion particularly during peak periods, with poor time reliability and

network resilience, and delays to the bus network particularly the Doncaster Area Rapid Transport (DART) bus service. Truck volumes through the north-east currently on arterial roads are also expected to increase.

Once completed and operating in 2036, North East Link would significantly redistribute medium and longer cross city trips away from local and arterial roads. The greatest reductions in traffic are expected on the parallel routes of Rosanna Road and Greensborough Road, as well as the five existing crossings of the Yarra River. Travel times are expected to improve, with savings up to 35 minutes between the M80 Ring Road and the Eastern Freeway. Some traffic increases are expected along some feeder routes.

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

Other impacts related to changes in traffic, such as changes in noise and air quality, have been evaluated separately.

10.2.2 Public safety

North East Link is forecast to reduce the total number of crashes across the north-east, despite an overall projected increase in vehicle kilometres travelled. The project would provide 135 lane-kilometres of additional freeway including North East Link and the widening of the Eastern Freeway and M80 Ring Road. This would generally divert traffic away from arterial roads onto the freeway network, with a net decrease in vehicle kilometres travelled on non-freeways. Overall non-freeway crashes are forecast to decrease. It is also expected the proposed collector-distributor lane arrangements would separate movements and further reduce crashes.

Proposed additions to and upgrades of shared use paths would reduce the need for cyclists to mix with general traffic and improve linkages to the existing network of trails and paths. New shared use paths along Greensborough Road would eliminate the need for cyclists to complete their trip on-road. Separating cyclists from general traffic would reduce the likelihood of incidents along the corridor and would improve the overall amenity and comfort of these trips. Frequent east-west crossings across the project would also make walking trips across Greensborough Road easier (shown on **Figure 10.1**).

The project would also generally reduce traffic and truck volumes in the north-east, providing a 'lower stress' environment for pedestrians and cyclists.

During the project's construction, the contractor would be required to develop Traffic Management Plans in consultation with relevant road authorities for works in all locations that affect pedestrians and cyclists and public transport or road users. This is outlined in ERP T2 – Transport Management Plan(s). These plans identify how safe and efficient passage through or around construction sites

would be provided, including details on proposed traffic control devices. The plans would demonstrate how they would minimise impact and maintain traffic flow on the surrounding road network. This would include details for all road users, including pedestrians, cyclists and bus users, not just general traffic.

During the operation of the tunnel, emergency planning (and response procedures) will be in place as discussed in the Project Description (Chapter 8 of the EES).

10.2.3 Public transport

Public transport is important for the whole community in terms of its contribution to a liveable neighbourhood. Access to public transport is important, particularly for people who cannot or are unable to drive (such as the elderly and those with disabilities). Lack of good access to public transport for these individuals can increase feelings of isolation, helplessness and dependence.

North East Link would include upgrades to public transport (as outlined in EES Technical report A – Traffic and transport), which offers some benefits to public transport in the local area, including:

- The Doncaster Busway project providing better bus access, reducing travel times (up to 30 per cent) and increasing the frequency of services
- Reduced travel times on all bus and tram services (up to 10 per cent) in the north-east, due to less congestion in the area.

It is expected these reduced travel times will increase patronage of these services, particularly on the Doncaster Busway.

During construction, existing public transport routes would be maintained where possible with suitable alternatives identified where needed. In addition, there may be some impacts on car parking at Watsonia railway station and the Doncaster Park and Ride during construction. Where car park numbers would be impacted, suitable alternatives would need to be identified to maintain the overall number of spaces. This may mean additional time walking from alternative parking areas to the station. A Traffic Management Plan would be developed to manage these impacts.

Pedestrian and cyclist access

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

North East Link would add new or upgraded walking or cycling connections within the local area. These include:

- The Greensborough Road path, which would complete an entirely off-road cycling corridor between the M80 Ring Road and the Eastern Freeway. This includes providing a connection to the Eastern freeway with a new north-south shared use path along Bulleen Road. The proposed upgrades are illustrated in **Figures 10.1 and 10.2**.

- New and upgraded shared use paths along the Eastern Freeway, including delivery of the North East Bicycle Corridor, which includes (as illustrated in **Figure 10.3**):
 - Connection to the Main Trail near Chandler Highway at the eastern end
 - Connection back to the Main Yarra Trail at the Merri Creek crossing near Roseneath Street
 - A new path under Chandler Highway to the Eastern Freeway.

These projects have been developed to minimise the likelihood of severance along the corridor. New shared use crossings are provided at multiple locations along the corridor to improve east-west connectivity. These new connections link to existing shared use paths improving access to the Watsonia and Heidelberg town centres, as well as the community facilities along Bulleen Road. North East Link would be in tunnels between Lower Plenty Road and Bulleen Road, preventing any severance throughout the Banyule Flats precinct.

The proposed improvements to shared use access and travel have the potential to improve health through the increased access to and use of safe routes which would reduce accidents.

Construction impacts on existing shared use routes would be minimised by providing suitable alternatives, with advance notice of changes. Alternative routes should maintain pedestrian and cyclist safety to prevent an increase in accidents or a perception of unsafe travel routes that discourage use and the health benefits of these activities. These aspects can be effectively managed through implementation of EPRs T2-T5.

Health and emergency services

North East Link (when operational) would include upgrades to emergency lanes and stopping bays on the outer carriageways to enable access for emergency services and maintenance vehicles. Emergency response plans and incident management procedures would be implemented from the new operations centre utilising the Freeway Management System infrastructure such as the Lane Use Management System. This would enable ease of access for emergency services and continued safe operation to other users of the freeway.

Construction works are not expected to affect community access to existing health and emergency services.

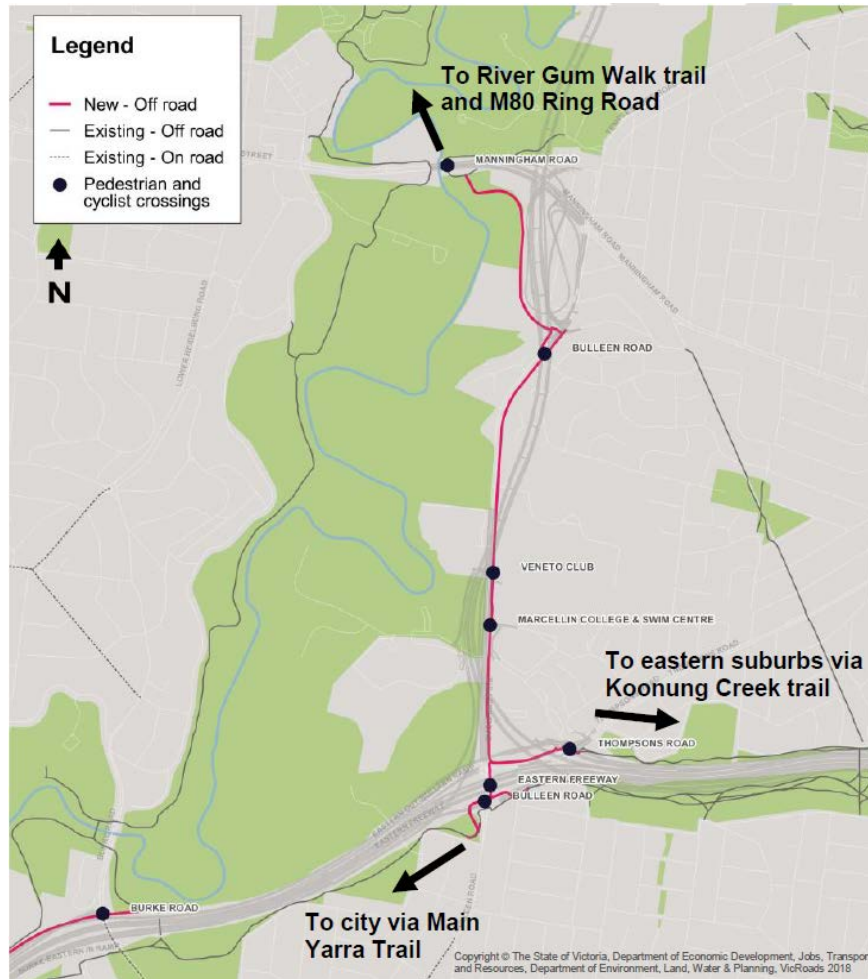


Figure 10.1: Greensborough Road Corridor – walking and cycling upgrades

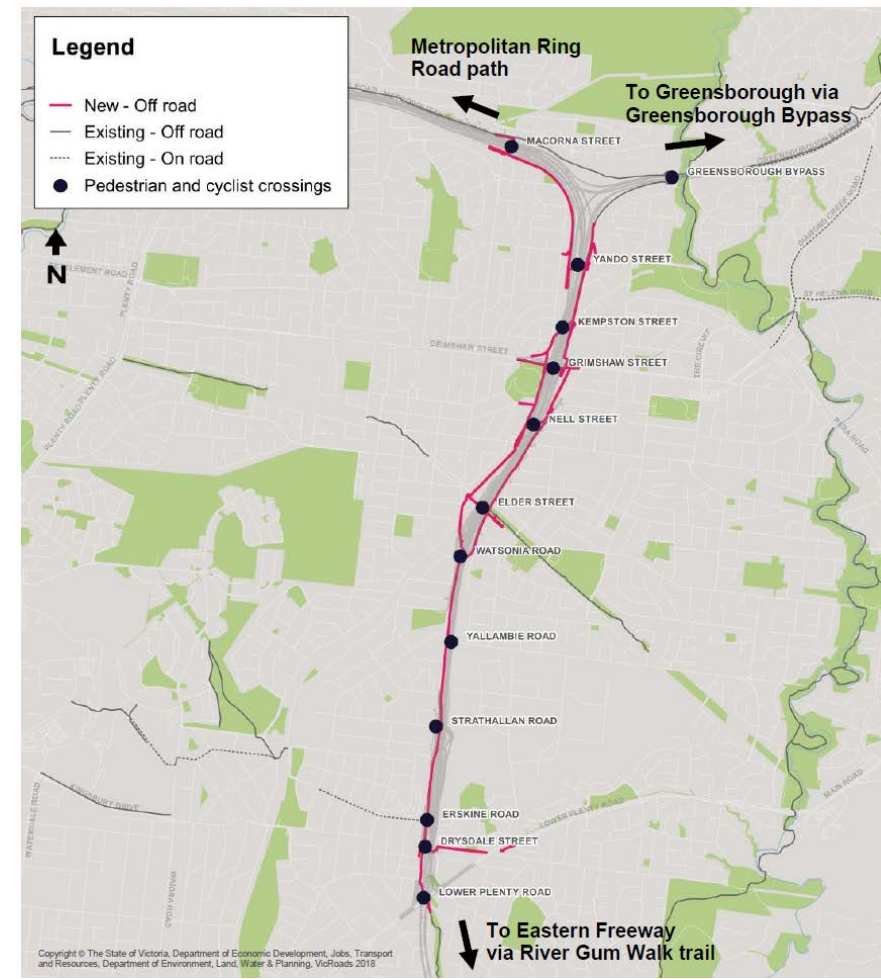


Figure 10.2: Bulleen Road shared use path

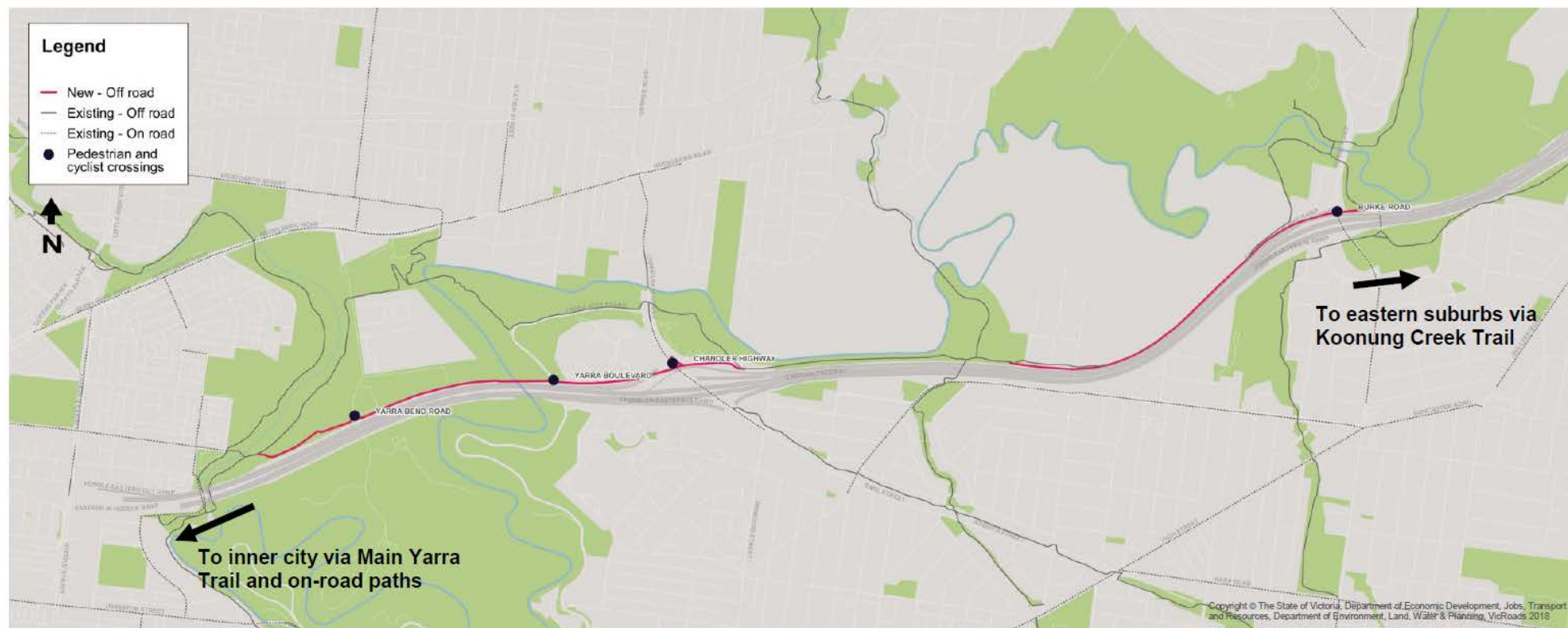


Figure 10.3: Proposed North East Bicycle Corridor alignment

10.3 Contamination

The presence of land and groundwater contamination has the potential to expose residents and workers to a range of chemicals which (depending on the concentration present) may have the potential to adversely affect health. The removal of contamination from some areas associated with the project's construction would be of some benefit to local community health.

A detailed assessment of the project's impact on land and water contamination is provided in EES Technical report O – Contamination and soil. The assessment includes consideration of the relevant State Environment Protection Policies (SEPPs):

- SEPP Prevention and management of contamination of land, No. S95 Gazette 4/6/2002
- SEPP Groundwaters of Victoria, No. S160 Gazette 17/12/1997 (SEPP).

A preliminary soil, groundwater and landfill gas investigation was undertaken to assist in understanding the potential for contamination to be present along the project alignment. No broad scale soil contamination was identified, although some contamination has been identified, and is expected to be potentially present due to historical activities in various areas. More specifically, the preliminary investigations identified contamination in soil/fill materials at Bulleen Oval. Groundwater impacts identified in some areas included elevated metals and the detection of per- and polyfluoroalkyl substances (PFAS) in the Bulleen area.

It is noted that contaminants encountered during this study are typical of those found on many other sites throughout Victoria.

Any contamination that may be present (currently known or identified during construction activities) would require management so that excavated materials were appropriately classified and disposed, and groundwater was not further impacted and was appropriately managed to prevent its discharge to an environmentally-sensitive receptor. These measures are outlined in the project EPRs:

- EPR CL1, Spoil Management Plan (Construction) and (Operation)
- EPR CL2, Minimise impacts from disturbance of acid sulfate soil
- EPR CL3, Minimise odour impacts
- EPR CL4, Minimise risks from vapour and ground gas intrusion
- EPR CL5, Management of chemicals, fuels and hazardous chemicals.

Implementation of these EPRs would also prevent the generation of dust from contaminated soil and the movement of these materials to various sensitive receptors.

The potential for contaminant migration to groundwater or other waterways (via groundwater extraction and/or discharge) during the project's construction has been assessed in EES Technical report N – Groundwater. That assessment identified appropriate management measures to minimise or prevent contaminant migration, including EPRs:

- EPR GW2, Monitor groundwater
- EPR GW3, Minimise changes to groundwater levels through tunnel drainage design and construction methods
- EPR GW4, Groundwater Management Plan.

No health impacts are expected to be associated with the management of contamination with the implementation of these EPRs.

10.4 Property acquisition

The acquisition and relocation of households and businesses due to property acquisition (particularly where individuals feel they have no say in the acquisition) can disrupt social networks and affect health and wellbeing due to raised stress and anxiety levels. This includes increased stress and anxiety during the process of negotiating reasonable compensation. The purchase of and moving into a house can be one of the most significant events in a person's life. A house and workplace are central to the daily routines of people, with the location of the premises influencing how they may travel to/from work or study, the social infrastructure and businesses they visit and the people they interact with. The displacement of businesses has the potential to impact on local employment opportunities. Further discussion on stress and anxiety is presented in **Section 10.10**.

North East Link would involve the acquisition of 68 residential properties, requiring the relocation of households. In addition, the project would affect approximately 100 businesses. Further detail on the impact of acquisitions on local businesses is provided within EES Technical report F – Business.

Residential property acquisition would be limited to the M80 Ring Road to northern portal and the northern portal to southern portal precincts, within the suburbs of Macleod, Yallambie, Greensborough, Watsonia and Bulleen.

Impacts associated with property acquisition would be managed through EPR SC1 which includes:

- Design and construct the project to reduce disruption to residences and community infrastructure facilities from direct acquisition or temporary occupation of land, as far as is practicable. Where residential land is to be permanently acquired:
 - Use a case-management approach for project interactions with affected landowners and occupants
 - Endeavour to reach agreement on the terms for possession of the land
 - Consider the relative vulnerability and special needs of landowners and occupants.

In addition, it is possible the decisions required for compensation and relocation, along with potential loss of social ties, disruption to lifestyle and demand on time may increase stress and anxiety levels in some individuals.

10.5 Visual changes

Visual amenity can be described as the pleasantness of the view or outlook of an identified receptor or group of receptors (such as at residences or for recreational users). Visual amenity is an important part of an area's identity and offers a wide variety of benefits to the community in terms of quality of life, wellbeing and economic activity. For some individuals, changes in visual amenity can increase stress and anxiety levels and may affect the use of outdoor spaces. However, these impacts are typically short in duration as most people adapt to changes in the visual landscape, particularly within an already urbanised area. It is noted that revegetation may take longer to be restored, but visual changes of revegetation are not considered to impact community wellbeing. Most changes in visual impacts are not expected to have a significant impact on community health.

During construction, visual amenity throughout the project area has the potential to be affected by factors such as the removal of established vegetation, the installation of construction hoardings/temporary noise walls and/or the visual appearance of construction sites. In some areas, the acoustic sheds, hoardings and barriers to manage noise impacts during construction would be large and may cause overshadowing (refer to EES Technical report E – Land use planning).

Once operating, the project would change local visual amenity due to the presence of new and amended infrastructure (including new roadways, ventilation structures, substations, bridges and drainage channels), landscaping and urban design features. The installation of noise walls may cause some overshadowing.

Due to these changes, some residents may experience reduced enjoyment and sense of pride in their properties, particularly their backyards or outdoor spaces and some may feel partial loss of privacy due to views from the shared overpasses into their properties. Over time, people may adapt to these and other changes to the landscape.

Overall, visual amenity would be enhanced where possible along the project as detailed in EES Technical report H – Landscape and visual.

10.6 Green space

Green space within urban areas includes green corridors (paths, rivers and canals), grassland, parks and gardens, outdoor sporting facilities, playing fields and children play areas. At a fundamental level there are links between human health/wellbeing and nature/biodiversity including within the urban setting (Brown & Grant, 2005; EC, 2011a; WHO, 2015).

Epidemiological studies have been undertaken that show a positive relationship between green space and health and wellbeing (de Vries et al., 2003; Health Scotland, 2008; Kendal et al., 2016; Maas et al., 2006; Mitchell & Popham, 2007). The outcomes of these international studies depend on the quality of the available green space. They showed that green space areas in low socioeconomic areas often had poor facilities, more graffiti, vacant/boarded up buildings and were less safe. The studies concluded these spaces had few health benefits.

The health benefits of green space in urban areas include (Health Scotland, 2008; Kendal et al., 2016; Lee & Maheswaran, 2011; Rozek et al., 2018):

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

Green space areas in urban areas may also present some hazards, such as attracting anti-social behaviours (particularly in isolated areas), providing areas for drug or sexual activity and unintentional injuries from sports or use of playground equipment. It has also been found that individuals from ethnic or minority groups and those with disabilities are less frequent users of green spaces areas (Friedrich et al., 2009; Lee & Maheswaran, 2011).

It is noted the detailed review of health benefits of urban green space areas undertaken by Lee (Lee & Maheswaran, 2011) determined only weak evidence for links between physical, mental health and wellbeing and urban green space. However, many of the studies are limited and confounded by other factors which affects the ability to draw conclusions.

More recent reviews (that include a number of Australian studies) (Dickinson, 2018; Rozek et al., 2018) conclude that access to high-quality public open space encourages people to be physically active and supports good mental and physical health. This is particularly evident where there is good access (that is, within walking distance and even up to five kilometres) to green public space particularly where the open space is large and includes desired amenities, safe or perceived safe walking neighbourhoods with good access and connections to green space, the green space area is considered safe, aesthetically pleasing, includes desired amenities (such as playgrounds, picnic tables, skate parks barbeques and toilets) and is well maintained.

The specific design and existing quality of green space that may be available in the local area has not been assessed in this report, only the changes that may occur as a result of the project. Therefore, while the following discussion outlines changes to green space related to the project, being able to draw clear conclusions on how these changes may affect health and wellbeing is

difficult and complex. Changes in green space may result in changes in stress and anxiety (refer to **Section 10.10** for further discussion).

North East Link would mean some changes to green space within the community, which are discussed below.

During construction, some existing open space and recreation areas would be acquired or temporarily occupied. Locations where more than 90 per cent of the open space area or park would be affected by construction activities include AK Lines Reserve, Borlase Reserve, Frensham Sec Reserve, Gabonia Avenue Reserve, Koonung Creek Reserve, Leonis Avenue Reserve, Stanton Street Reserve, Trist Street Reserve, Maugie Street Reserve, Watsonia Road Reserve, Watsonia Station Carpark Reserve, Winsor Reserve and a number of unnamed reserves, particularly to the west of Watsonia railway station, around Bulleen Road (southern extent of Bulleen Road, western side in the area of Ilma Court) and around the Eastern Freeway. In addition, it is estimated that approximately 25,000 planted trees are at risk of removal during construction (as discussed in EES Technical report G – Arboriculture).

Once the project was complete, a number of open space or park areas would be changed. The Watsonia Station Carpark Reserve is proposed to be essentially fully acquired, with a portion (greater than 10 per cent) of some unnamed reserves (behind the Boroondara Tennis Centre, bordered by Dan Murphies and the Bulleen Swim Centre; and south of Doncaster Road, between Hender Street to the east and the Eastern Freeway to the west), Stanton Street reserve and Watsonia Reserve. The long-term loss of trees and canopy cover during the project's construction could be mitigated with replanting once construction was complete. This would include implementation of a Tree Canopy Replacement Program (EPR AR3).

The project's full and partial temporary occupation and permanent acquisition requirements would mainly be clustered around the southern extent of Bulleen Road and the area surrounding the Eastern Freeway. This area is an active recreational precinct, which includes the sports fields provided by Carey Grammar, Trinity Grammar School, and Marcellin College as well as at Bulleen Park, the Bulleen Swim Centre, Boroondara Tennis Centre, the Veneto Club and the Freeway Public Golf Course. In addition to these facilities, a number of other facilities are used for recreational sports that would be impacted by temporary occupation. These facilities are dispersed across the project area, including at Gabonia Avenue Reserve, Winsor Reserve, AK Lines Reserve and Elgar Park.

Impacts on sporting, recreational and other facilities in these areas would be managed through EPR SC4 which requires that:

- Where recreation facilities are displaced by the construction or operation of the project, work in collaboration with facility operators, local councils and relevant State authorities to identify relocation opportunities with the objective of accommodating displaced facilities and maintaining the continuity of those recreational activities, where practicable.

- Where construction or operation activities directly impact community infrastructure facilities such as schools, child care centres, and aged care centres, consultation must occur with facility operators, owners and user groups of the facilities to understand and, where appropriate, implement any practical measures that can be taken to avoid or minimise impacts.

It is not generally expected that availability of sporting facilities and clubs would be reduced by the project's land requirements. Depending on where the facilities are relocated, it is possible if located further away from their existing location that some users from the immediate surrounding area may be deterred from travelling the additional distance to access them.

However, it was noted through the social impact assessment consultation process with managers/operators and users of the facilities that people generally travel a fair distance to use these facilities and that most would continue to use the facilities at their new location.

The reference project would require the acquisition of Bulleen Swim Centre. The function the facility provides would cease. The displacement of this business may inconvenience community members and other users of the facility, who would have to access similar services elsewhere. This may impact community health where swim activities are not continued due to the loss of this swim centre.

Where a full or partial but larger percentage of area of open spaces would be subject to temporary occupation or land acquisition, it is highly likely the neighbouring community would experience reduced availability of open spaces for active or passive recreation over the short to medium term. This could potentially reduce the opportunity for access to these areas for various activities. For example, this may occur in areas near Borlase Reserve, AK Lines Reserve, Boronia Grove Reserve, the Eastern Freeway Linear Reserve, Frensham Sec Reserve, Gray Street Reserve, Jack Otoole Reserve, the Koonung Creek Linear Park, Koonung Creek Reserve and Koonung Reserve. Some open spaces used for active and passive recreation may be permanently acquired, preventing access and use of these facilities into the future. However, alternative avenues would continue to be available to pursue an active lifestyle, access open space and interact with community in the general surroundings in the municipal areas of Manningham, Boroondara and Banyule.

Some open space or parks would be affected by partial acquisition or temporary occupation. Use of the remaining spaces within these areas may no longer be attractive or desirable in terms of recreational use. In the short term, this may affect active lifestyle access and use of these areas but it is expected that people would adapt to these changes and/or find alternatives.

Overall, the project's construction sites and activities would affect visual amenity, cause some long-term loss of trees and canopy cover, and mean inconvenience and a temporary loss of access to some existing passive and active green space and recreational areas. Some active recreational areas such as the Bulleen Swim Centre would be permanently lost. However, there would be no loss of existing community sporting facilities, and alternative recreational areas would remain accessible in the local areas. Changes in green space and access to community sporting and recreational facilities during the project's construction and operation would be managed through EPRs SC2 to SC4 and EPRs AR1 to AR3.

As a result, the potential for significant impacts on the health of the local community is considered low.

It is recognised that vulnerable groups are also users of community infrastructure and changes to access to green space and recreational facilities may reduce their physical activity and overall health and wellbeing.

10.7 Changes in community access and connectivity

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

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

Community severance effects often occur during major transportation projects (during construction and operation) due to detours in the local road network ('rat running'), changes to active and public transport routes, increases or decreases in traffic movements on connector roads, and property acquisitions (residential or commercial) which change social ties and connections. These changes may contribute to feelings of community severance and disconnection.

The project's construction would involve the temporary disruption of some pedestrian and cycling routes, and the permanent or temporary acquisition of open space or other recreational areas. This reduced connectivity may deter people from participating in community activities or active transport, potentially reducing the connection to an environment and feeling of community cohesion.

These impacts would be reduced through implementation of EPRs SC1 to SC4.

Where these EPRs are implemented, the potential for changes in the community to adversely impact health and wellbeing is considered low.

10.8 Equity issues

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

- Elderly (which is considered to be those over 65 years in this assessment)
- Individuals with pre-existing health problems
- Infants and young children
- Individuals with disabilities
- Individuals who live in areas of higher air or noise pollution levels.

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

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

The individual suburbs and local government areas (LGAs) within the study area all rate in the upper deciles (deciles 6 to 10) of the Index of Relative Socio-economic Disadvantage (refer to **Table 6.3**). This indicates these LGAs are not generally considered to be socio-economically disadvantaged. The lowest ranking of 6 relates to the suburbs of Watsonia and Bundoora, and the City of Whittlesea (located to the north-west of the main project corridor).

It is noted that housing prices are lower on main roadways in many urban areas. The median house prices in the study area are variable, but in most areas are consistent with the Melbourne average. Some public housing is located in the study area, where the following should be noted:

- The City of Banyule has a higher concentration of public housing (13 per cent compared with the Greater Melbourne average of 8 per cent), particularly in the south-western suburbs. These are not located close to the project.
- Other key LGAs have public housing similar to Greater Melbourne.

Where air and noise impacts are considered together, the following can be noted:

- In relation to emissions to air from the proposed tunnel ventilation structures, these would not significantly contribute to air pollution in the study area, and health impacts related to these emissions are considered acceptable and not measurable within the community. This conclusion does not change where noise impacts are also considered.

- The greatest contribution to changes in air quality in the study area would be from the redistribution of traffic on surface roads. Residents and other sensitive areas located adjacent to these surface roads have the potential to be more significantly impacted from changes in traffic and air quality. They may also be affected by changes in traffic noise.
- The approach adopted to manage traffic noise within the community once the project is operating is predicted to reduce overall traffic noise and generate some benefit to community health. At a community/population level, the combined benefits of lower levels of traffic noise, and potential impacts from vehicle emissions to air would not result in population risks any higher than already present.

Further assessment of localised air and noise impacts has identified:

- The greatest increase in air quality would occur adjacent to the North East Link surface road from Lower Plenty Road to Grimshaw Street, particularly the eastern side of Greensborough Highway between Yallambie Road and Watsonia Road intersection approximately 50 to 100 metres north of Yallambie Road.

Properties in this area are predicted to experience reduced traffic noise (generating some health benefit), or a small increase in noise. Where a small increase in noise would occur, the predicted noise levels in this area would be below the threshold for mortality and cardiovascular effects and so no health impacts from changes in noise in this area are expected.

Therefore, in the most impacted location, from an air quality perspective, combined impacts from air and noise at these locations would not result in localised health impacts higher than already present.

- In relation to noise, some properties located in the suburbs of Doncaster and Box Hill have already been identified as potentially requiring additional noise mitigation (at-property treatments). Where these mitigation measures were adopted, noise would not be considered to be a significant localised health impact. However, other properties adjacent to the Eastern freeway would experience a small increase in noise, but it is predicted the adopted noise criteria would still be achieved. These areas are also predicted to experience an increase in air pollution due to the increase in traffic along the freeway. While it is not appropriate to simply add the calculated localised health impacts from air and noise at these locations, this review has identified that properties adjacent to the Eastern freeway may experience impacts from changes in air quality and noise. No localised health impacts that are considered of significance (and which have not already been identified for at-property treatment to manage noise impacts) have been identified in these areas. The combined impacts on health are therefore also not expected to be significant.

A number of project benefits also need to be considered in relation to areas located adjacent to the Eastern Freeway. These benefits include those generated by the proposed public transport improvements (from the Doncaster Busway), reduced travel times and an improved active transport network. These improvements would be of greater benefit to people living closer to the freeway and associated services and networks, generating possible health benefits.

Overall, there are no areas identified that may result in any significant inequitable distribution of health impacts within the community.

10.9 Economic aspects

Economic aspects associated with the project are detailed in the North East Link business case¹⁰. This assessment identifies a number of significant economic benefits the project would generate. Many of these are associated with business and freight users, with significant productivity gains across the economy. Productivity improvements generated by North East Link are expected to increase Victoria's Gross State Productivity by about \$12.5 billion and increase the Gross Regional Product for the north-east by \$7.5 billion through to 2046.

More specifically, the economic benefits of the project would include:

- \$250 million in economic value each year from better business connectivity
- \$427 million of economic value each year from better freight connectivity
- \$590 million increase in productivity from business clustering
- 2 per cent increase in connectivity between manufacturers and suppliers
- More line haul freight carried on Higher Productivity Freight Vehicles (HPFVs) between the north and south-east
- \$148 million reduction in freight vehicle operating costs per year
- Businesses in the north-east would have access to 62,000 more workers
- Attraction of 5,500 jobs to businesses in the north-east
- Workers in the north-east would have access to 56,000 more job opportunities
- Additional 10,300 jobs during construction
- Support an additional 3,400 jobs in Victoria each year during operation
- Attract 9,700 more people to live in the north-east.

The North East Link business case (2018) sets out cost benefits associated with changes in household connectivity and improved safety, where the following is indicated:

- \$324 million in economic value each year from better household connectivity
- \$42 million in economic value each year from improved safety and amenity.

¹⁰ <https://northeastlink.vic.gov.au/project/businesscase>

Impacts of the project on businesses are presented in EES Technical report F – Business. That impact assessment considered the potential impacts on business assets, business value and business operations due to the construction and operation of North East Link.

The project's permanent land acquisition and temporary occupation requirements would impact approximately 100 businesses (three would be temporarily occupied, with the remainder being fully or partially acquired). Land acquisition impacts would be concentrated in the project's northern portal to southern portal precinct, where approximately 94 per cent (rounded) of the impacted business are located.

EPRs have been developed to mitigate and manage impacts of the project and these would form an approval requirement as well as part of contractual obligations for the project.

Likely pre-construction impacts include reduced business investment and expansion and challenges with increased staff turnover and staff retention. Uncertainty affecting businesses before the project's constructed started would likely continue (although decrease) until this time. These impacts would be managed through EPRs B1 and B2.

During construction, land acquisition would likely have the most significant impacts on businesses across the study area, followed by changes to access and amenity impacts associated with physical construction activities and laydown sites. Businesses that are interdependent for successful operations are more likely to be impacted by land acquisition (affecting their own or other businesses) and consequential business displacement during the project's construction.

Construction impacts for businesses would be managed through EPRs that include the requirement that access and amenity impacts on businesses are minimised (EPR B4) and that any damage or impacts to third-party property and infrastructure is minimised and remedied if it happens (EPR B3). As the relocation of some utilities would be required, it is a performance measurement that utility assets are protected and that impacts are minimised (EPR B5).

To encourage good communication between businesses and those responsible for construction, it is proposed that business liaison groups were established to facilitate business involvement in the project and ensure timely reporting of changes to traffic or parking and update them on construction activities (EPR B6).

Upon completion of the project, most impacts on businesses would likely be positive. Access to and through the study area is most commonly identified as an expected improvement. Improved connectivity, the diversion of trucks from local and arterial roads, improved amenity and improved pedestrian and cyclist connectivity are also anticipated benefits for businesses (staff and customers). The economic benefits of these are listed above.

Overall, the project represents an opportunity to generate significant benefits for businesses, including improved access, reduced travel times and enhanced transport network safety in alignment with government policies and strategic objectives. However, there would be significant impacts to businesses displaced by acquisition, particularly with the loss of the Bulleen Industrial Precinct, and these impacts would be required to be effectively managed through the project EPRs.

In terms of community health, where the impacts identified are appropriately managed and mitigated through with implementation of the EPRs, North East Link has the potential to positively impact community health. Many of the benefits relate to reduced stress and anxiety (refer to **Section 10.10**) as well as the health benefits of more employment opportunities.

Unemployment has a significant impact on physical and mental health and increases overall mortality rates, including: mortality from cardiovascular disease and suicide; poorer general health; poorer physical health (including increased rates of cardiovascular disease, lung cancer and susceptibility to respiratory infections); poorer mental health and psychological wellbeing; somatic complaints; long-standing illness; disability; and higher rates of medical consultation, medication consumption and hospital admission. For young people, unemployment leads to a range of psychological problems including depression, anxiety and low self-esteem (Royal Australasian College of Physicians, 2014).

Employment offers a range of health benefits including (Royal Australasian College of Physicians, 2014, 2015):

- Work improves general health and wellbeing including self-esteem, self-rated health, self-satisfaction, physical health and financial concerns
- Work is an effective way of reducing poverty and social exclusion
- Reduces psychological distress and minor psychiatric morbidity
- Leads to lower morbidity rates
- Improves physical functioning and mental health in older people
- improvements in mental health, in particular decreased risk of depression.

This is particularly relevant to more accessible employment and reduced congestion and travel times, which can reduce stress and anxiety.

10.10 Stress and anxiety issues

The area of mental health, and the interconnections between physical health and mental health, is highly complex both at a community level as well as on an individual level. As a result of this complexity, this assessment has focused on and provided a qualitative assessment of the key aspects of mental health that may be impacted by the project, namely changes in stress and anxiety.

A number of changes within the community (discussed in **Sections 10.2 to 10.9**) have the potential to affect stress and anxiety levels. Some changes may reduce stress and anxiety while others may increase them.

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

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

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

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

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

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

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

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

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

More generally, it must be noted that urbanisation, or increased urbanisation, regardless of specific projects has been found to affect stress levels and mental health (Srivastava, 2009). These impacts are greater where there is urbanisation without improvements in infrastructure to improve equitable access to employment and social areas/communities (Srivastava, 2009).

The role of acute or long-term environmental stress on the health of any community, in general and for specific project(s) including North East Link cannot be quantified. There are a wide range of complex factors that influence health and wellbeing, specifically mental health. It is not possible to determine any specific outcomes that may occur as a result of a specific project, or number of projects. However, it is noted that within any urban environment there would be a wide range of stressors present from infrastructure projects as well as other urban developments that may or may not contribute to the health effects outlined above.

It is noted that North East Link aims to improve infrastructure, connections and access within the urban environment. It also aims to reduce traffic and congestion on local roads. On a broader scale, the project, while requiring long-term management to minimise construction impacts, may assist in reducing stress and associated physiological and mental health impacts within the urban environment once operational.

11. Uncertainties

11.1 General

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

11.2 Exposure concentrations and noise levels

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

Traffic modelling

Assessment of impacts of the project on air and noise has relied on the modelling of traffic changes (refer to EES Technical report A – Traffic and transport). The traffic modelling incorporated inputs provided by the Victorian Government and a wide range of assumptions, with the aim of providing a realistic assessment of traffic changes in the project area. The model has been calibrated and validated based on existing data from 2016.

The traffic model has also been evaluated for changes in some of the key inputs including:

- Changes in land use reflecting 2031 and 2041 conditions, including population growth – the traffic forecasts were found to be most sensitive to these changes
- Changes in tolls, where increases or decreases of 20 per cent were considered – these changes were found to have minimal impact on arterial roads (for cars and trucks)
- Consideration of the Outer Metropolitan Ring (OMR) Road, including E6 project – this resulted in a significant decrease in traffic volumes for north-south arterial roads in the outer north
- North-east truck curfews, extending these to 24-hours – this resulted in a general decrease in truck volumes on curfew roads but an increase of 5 per cent in truck volumes on North East Link
- Alternative design layout for the Manningham Road interchange – which had a negligible impact on traffic volumes.

Air quality

The air quality impact assessment (refer to EES Technical report B – Air quality) incorporates information on traffic volumes and composition from the traffic model and other information on the design of the project. The air quality assessment was conducted, as far as possible, with the intention of providing ‘accurate’ or ‘realistic’ estimates of pollutant emissions and concentrations. The estimation of air concentrations within the community utilises air dispersion models approved by EPA Victoria as suitable for providing estimates of air quality from the tunnel ventilation structures and surface road traffic. The modelling incorporates information on the local area such as terrain, meteorology and measured existing air quality. A number of conservative assumptions and approaches have been adopted in the assessment of air quality impacts, which include:

- Five years of meteorology were considered so the year of meteorology corresponding to the greatest predicted impacts could be selected for the assessment.
- Background pollutant concentrations for the modelled years of 2026 and 2036 were assumed to remain at levels recorded for 2013 to 2017.
- EPA Victoria predicts a significant reduction in CO and NO₂ concentrations over the next 20 years with cleaner exhaust emissions from petrol, diesel and LPG engines and improvements in national motor vehicle emission standards – and a significant reduction in particle emissions (PM_{2.5}) from diesel vehicle engines is also expected by 2030.
- Concentrations of these pollutants in 2026 and particularly in 2036 would therefore be lower than those used as background levels in the air quality impact assessment.
- The adopted background concentrations for PM₁₀, PM_{2.5}, CO and NO₂ include exceptional events (as defined by EPA Victoria) such as bushfires, controlled burns and dust storms. During these periods, concentrations of particulate matter (PM₁₀ and PM_{2.5}) can reach extremely high levels. Inclusion of data during these periods as representative background concentrations for the project is highly conservative, contributing a significant proportion of the overall impact (background plus predicted).
- Vehicle emission factors for 2026 and 2036 were assumed to remain at levels predicted for 2020 or 2025. Emission factors used for this assessment are considered conservatively high because there is a general trend towards lower emission vehicles (older technology vehicles being replaced over time with newer, improved technology vehicles) and expected improvements in vehicle technology beyond 2020 and 2025, which are not accounted for in the air quality impact assessment.
- Hybrid and electric vehicles were not considered in the fleet mix. The share of these lower emission and zero emission vehicles is expected to increase in future years.
- For passenger car vehicles, 15 per cent diesel and 85 per cent petrol fuelled cars were assumed, which is higher than the Australian Bureau of Statistics motor vehicle census for 2017 which indicates the Australian passenger car fleet consisted of approximately 13 per cent diesel

vehicles. This assumption means higher particulate matter (PM₁₀ and PM_{2.5}) and NO₂ emissions from car vehicles for the 2026 and 2036 model scenarios.

- Surface roads were modelled at grade with the exception of the North East Link in trench.
- Motor vehicle emissions included tailpipe emissions and brake and tyre wear.
- Roads and ramps were correctly located relative to each other, so the combined impacts are appropriately assessed (road geometry was estimated from aerial photographs and design drawings).
- To obtain reasonable concentration predictions at the selected receptors, it is sufficient to ensure they are the correct distance from the modelled road (the location of modelled roads may become imprecise at some locations, as they are represented as straight lines whereas in reality, they may be slightly curved).
- All days in the modelled year (2016) were conservatively assumed to be a weekday for the purpose of assessing annual averages.
- There has been no consideration of the effects that existing or remaining vegetation or noise walls may have on the predicted pollutant concentrations, particularly at locations adjacent to key surface roads. The available information indicates that noise walls can affect pollutant concentrations downwind of the walls, in the near field.

Overall, the approach adopted for modelling changes in air quality is considered to have provided conservative estimates of exposure concentrations.

The air quality impact assessment (refer to EES Technical report B – Air quality) also considered the changes that some of the key assumptions (namely the air emissions from the tunnel ventilation structures and changes in vehicle composition) may make to the modelled air concentrations. The modelling was found to be relatively insensitive to uncertainty in these assumptions.

Noise assessment

The noise impact assessment (refer to EES Technical report C – Surface noise and vibration) incorporates information on traffic volumes and composition from the traffic model and other information on the design of the project. The modelling also incorporates measured background noise levels and a range of inputs and assumptions in relation to noise generated from the project.

For the assessment of construction noise, it has been assumed that all plant/equipment for each scenario is operating at the same time. This is unlikely to occur and would have overestimated construction noise impacts.

The model used in the assessment was validated based on existing information and traffic information for 2017. The modelling undertaken was used to determine a correction factor of 1.9 dBA to address overprediction of noise. The degree of overprediction the model observed is noted to be generally consistent with experience on previous projects.

The noise modelling undertaken for 2036 has assumed that future day-night profile of traffic is similar to the existing, as measured, pattern.

The characterisation of health effects associated with changes in noise has been undertaken using the maximum changes in noise during any one day. The noise exposure-response relationships adopted in this assessment relate to annual average changes in noise. The use of the daily maximum change in noise is expected to overestimate health impacts derived from noise (in particular localised impacts).

11.3 Approach to the assessment of risk for particulates

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

Overall, the epidemiological and toxicological data for the assessment presented in this report is based on current and robust information for the assessment of risks to human health associated with the potential exposure to particulate matter from combustion sources.

Exposure-response functions

The choice of exposure-response functions for the quantification of potential health impacts is important. For mortality health endpoints, many of the exposure-mortality functions have been replicated throughout the world. While many of these have shown consistent outcomes, the calculated relative risk estimates for these studies do vary. This is illustrated by **Figures 11.1 to 11.3** that show the variability in the relative risk estimates calculated in published studies for the US (and Canadian) population that are relevant to the primary health endpoints considered in this assessment (USEPA, 2012). A similar variability is observed where additional studies from Europe, Asia and Australia/New Zealand are considered.

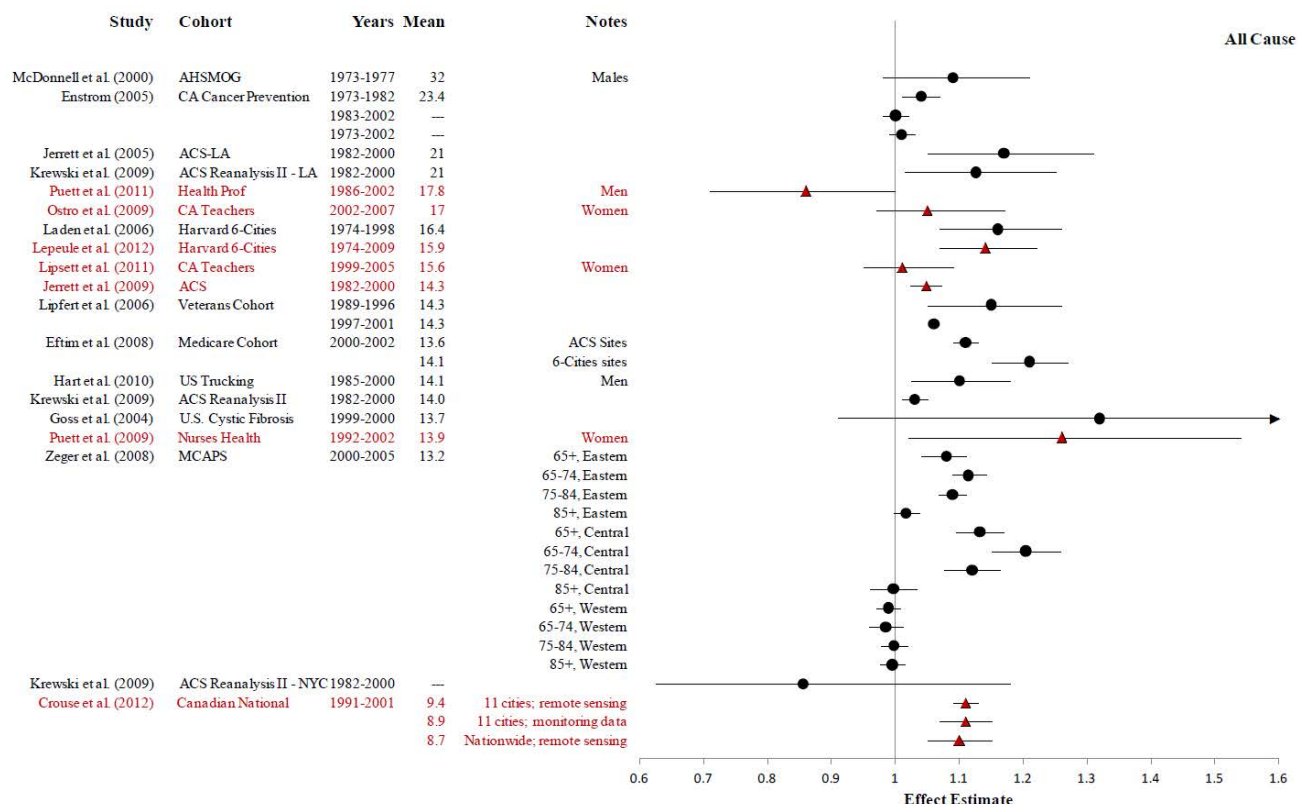


Figure 11.1: All-cause mortality relative risk estimates for long-term exposure to PM_{2.5} (USEPA, 2012, note studies in red are those completed since 2009)

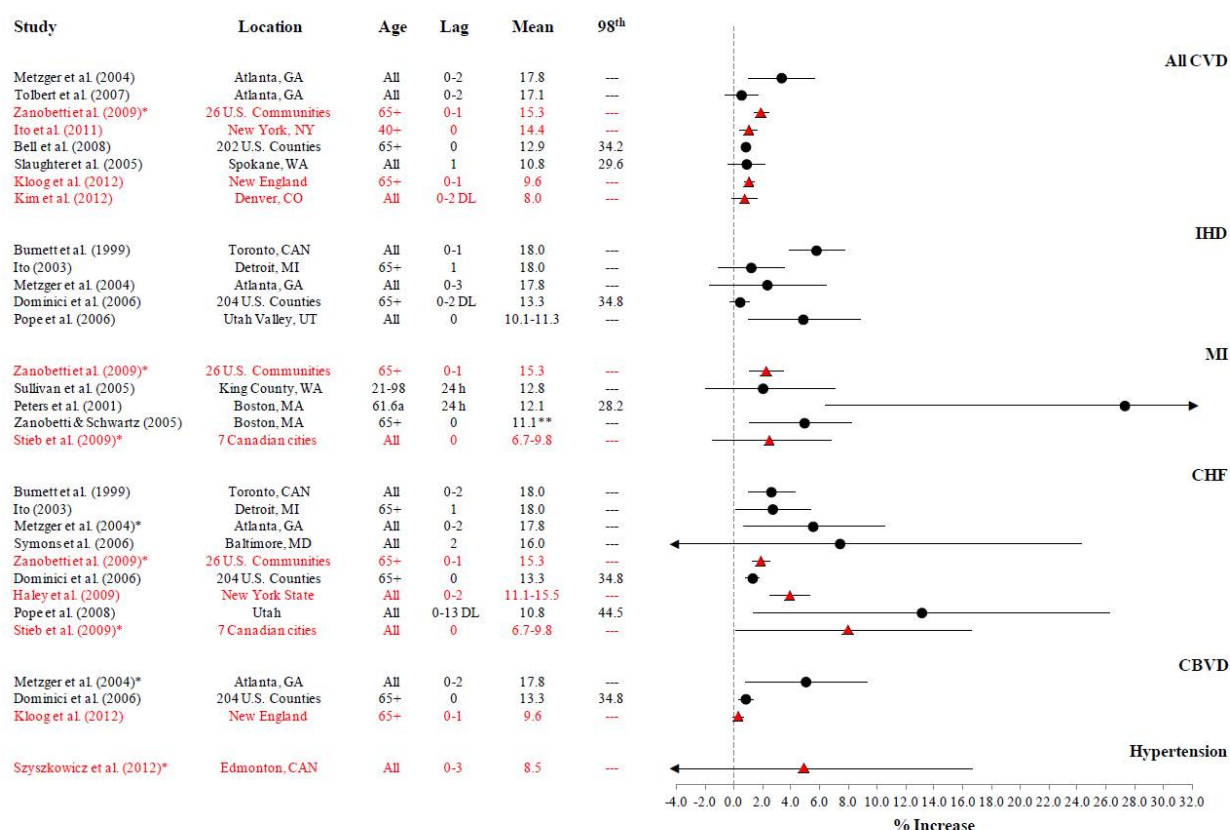


Figure 11.2: Per cent increase in cardiovascular-related hospital admissions for a 10 microgram per cubic metre increase in short-term (24-hour average) exposure to PM_{2.5} (USEPA, 2012, note studies in red are those completed since 2009)

(note: CVD = cardiovascular disease; IHD = ischemic heart disease; MI = myocardial infarction; CHF = congestive heart failure; CBVD = cerebrovascular disease)

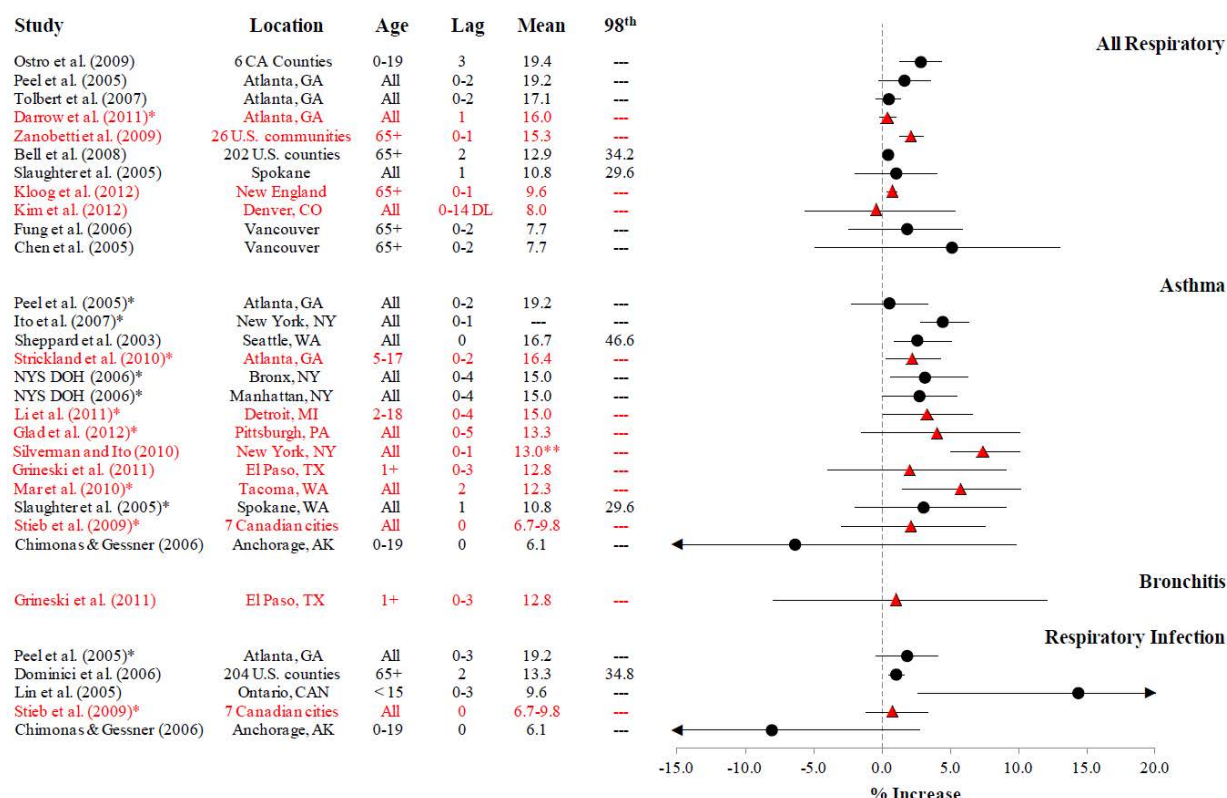


Figure 11.3: Per cent increase in respiratory-related hospital admissions for a 10 micrograms per cubic metre increase in short-term (24-hour average) exposure to PM_{2.5} (USEPA, 2012, note studies in red are those completed since 2009)

These figures illustrate the variability inherent in the studies used to estimate exposure-response functions. The variability is expected to reflect the local and regional variability in the characteristics of particulate matter to which the population is exposed.

Based on the available data, and the detailed reviews undertaken by organisations such as the USEPA (2010, 2012) and the WHO (WHO, 2003, 2006a, 2006b) and NEPC (NEPC, 2016), the adopted exposure-response estimates are considered to be current, robust and relevant to the characterisation of impacts from PM_{2.5}.

Shape of exposure-response function

The shape of the exposure-response function and whether there is a threshold for some of the effects endpoints remains an uncertainty. Reviews of the currently available data (that includes studies that show effects at low concentrations) have not shown evidence of a threshold. However, as these conclusions are based on epidemiological studies, discerning the characteristics of the particulates responsible for these effects and the observed shape of the dose-response relationship is complex. For example, it is not possible to determine if the observed no threshold response is

relevant to exposure to particulates from all sources, or whether it relates to particulates from combustion sources only.

Most studies have demonstrated a linear relationship between relative risk and ambient concentration. However, for long-term exposure-related mortality, a log-linear relationship is more plausible and should be considered where there is potential for exposure to very high concentrations of pollution. In this health impact assessment, the impact considered is a localised impact with low level incremental increases in concentration. At low levels the assumption of a linear relationship is considered appropriate.

Diesel particulate matter evaluation

The assessment of exposure to diesel particulate matter has assumed that 80 per cent of the PM_{2.5} associated with the project is derived from diesel sources. This is considered to be a conservative assumption.

The health hazard conclusions associated with exposure to diesel particulate matter are based on studies that are dominated by exhaust emissions from diesel engines built prior to the mid-1990s. With current engine use including some new and many older engines (engines typically stay in service for a long time), the health hazard conclusions are generally likely to be applicable to engines currently in use.

However as new and cleaner diesel engines and different diesel fuels replace a substantial number of existing engines, the general applicability of the health hazard conclusions may require further evaluation. The NEPC (NEPC, 2009) has established a program to reduce diesel emissions from the Australian heavy vehicle fleet. This is expected to lower the potential for all diesel emissions over time.

An increase in the number of vehicle kilometres travelled (more than estimated in the traffic modelling) may limit the benefits of cleaner diesel vehicles.

11.4 Approach to the assessment of risk for noise

The association between exposure to noise and adverse health effects is well documented and a number of robust studies are available to characterise these effects. A number of relationships adopted in this assessment come from research where data from a number of studies have been combined. The available studies used to determine these relationships often utilise different measures of noise levels (differing between covering average day and evening or day evening and night) and different methods for measuring the disease end-points. This results in the use of some conservative assumptions when combining these data.

Many of the available studies relate to health effects in males, or include populations that are predominantly male. The reported outcomes of these studies have been assumed to equally apply to females.

11.5 Co-pollutants and co-exposures

For the assessment of nitrogen dioxide, particulates and noise, the exposure-response relationships used in this health impact assessment are based on large epidemiology studies where exposures have occurred in urban areas. These exposures do not relate to only single pollutant or exposures (noise) but a mix of them, as well as others including occupational and smoking. While many of the studies have endeavoured to correct for exposures to other pollutants and exposures, no study can fully correct for these and there would always be some level of influence from other exposures on the relationships adopted.

In relation to air quality, many of the pollutants evaluated come from a common source (such as fuel combustion) so the use of only particulate matter (or nitrogen dioxide) as an index for the mix of pollutants that is in urban air at the time of exposure is reasonable but conservative.

In relation to the assessment of cardiovascular effects from road traffic noise, these effects are also associated with (and occur together with) increased exposures to vehicle emissions, specifically particulate exposures.

For this reason, it is important the health risks and incidence evaluations presented for exposure to nitrogen dioxide, particulates and noise should not be added together as these effects are not necessarily additive, due to the relationships already including co-exposures to all these aspects (and others).

11.6 Selected health outcomes

The assessment of risk has utilised exposure-response functions and relative risk values that relate to the more significant health endpoints where the most significant and robust positive associations have been identified. The approach does not include all possible subsets of effects that have been considered in various published studies. However, the assessment undertaken has considered the health endpoints/outcomes that incorporate many of the subsets, and has utilised the most current and robust relationships.

11.7 Exposure time / duration

The assessment of potential exposure and risk to changes in air quality and noise levels associated with the project has assumed that all areas evaluated are residential and people may be at home for 24 hours of the day for 365 days of the year, for a lifetime. This is a conservative assumption to ensure that all members of the public are adequately addressed in the assessment of health impacts, including the elderly and those with disabilities who may not leave home very often. As a result, the quantification of risk and health incidence is expected to be an overestimation.

11.8 Changing population size and demographics

The assessment presented has utilised information on the size of the population and distribution of the population in relevant ages from the ABS Census data from 2016. As discussed in **Section 6.3**, the population in the study area is projected to increase significantly by 2041. In addition, a number of the local government areas (LGAs) are expecting a significant increase in the proportion of the population aged 65+ years.

The increase in population size and distribution does not affect the calculation of an individual risk. The key aspect that does affect this calculation is the baseline incidence of the health effects within the population. Based on statistics from Victoria's Department of Health and Human Services (DHHS), the baseline incidence of most of the health effects evaluated in this assessment have been relatively stable or decreasing over time (with improvements in health care). The prevalence of hypertension in the community is noted to be slowly increasing over time, although this change is not sufficient to change the outcome of the health impact assessment, even where the population distribution changes. Changes in the population over time are not expected to increase the calculated individual risk.

For the calculation of the change in incidence in the community, the size and distribution of the population is important. The incidence numbers calculated for the project are low and unmeasurable, and even if the population were doubled the incidence of the key health effects would remain low and unmeasurable within the community.

11.9 Baseline incidence for asthma

Some concern has been raised in the community that the baseline incidence of asthma reported in the statistics for the LGAs may not reflect more localised suburbs, or part suburbs, where the incidence of asthma was perceived to be higher.

The calculated individual risks relevant to asthma presented in this health impact assessment have been further evaluated assuming the baseline incidence reported for all the relevant LGAs is double. Where this is assumed, the calculated risk increases but remains well below the unacceptable risk levels of 10^{-5} and 10^{-4} .

This change in baseline incidence for asthma does not change the conclusions presented in this health impact assessment.

11.10 Application of exposure-response functions to small populations

The exposure-response functions have been developed on the basis of epidemiological studies from large urban populations where associations have been determined between health effects (health endpoints) and changes in ambient (regional) pollutant levels (noise, particulates or NO_2). Typically, these exposure response functions are applied to large populations for the purpose of establishing/reviewing air guidelines or reviewing potential impacts of regional air quality issues on large populations.

When applied to small populations (less than larger urban centres such as the whole of Greater Melbourne) the uncertainty increases. They do not relate to specific local sources (which occur within a regional airshed) or daily variability in exposure that may occur because of various different activities that may occur in any one day.

11.11 Overall evaluation of uncertainty

Overall, the assessment of health impacts presented in this report has incorporated a range of assumptions and models that would have resulted in an overestimation of impacts. The most significant factors that result in the assessment providing conservative outcomes are:

- Modelling of air quality impacts – this has included a range of conservative assumptions about the type of vehicles and the emissions to air that may come from these vehicles over time. The assessment has also utilised a model to predict ground level concentrations (concentrations in the community) that are expected to be conservative.
- Modelling of noise impacts – this has been undertaken using a model that provides estimates of changes in noise levels that are expected to be conservative. In addition, the assessment of health impacts has utilised the maximum daily change in noise in the community, rather than the change in annual average noise levels (which the noise exposure – response (health effects) relationships are based on). This would have overestimated the noise impacts in the community by around 3 dB(A) (potentially more).
- Community exposures – there are a number of assumptions adopted in the characterisation of exposure that would have overestimated exposure:
 - It is assumed the maximum changes in localised air quality, regardless of where this may occur (such as an industrial area, in a roadway, open space area or residential area), affects a resident
 - All exposures to changes in air quality and noise that occur, in all areas, assume that all residents are at home all day, every day for a lifetime, and that changes in outdoor air pollution are mirrored indoors.

These assumptions are expected to overestimate exposures and risks in the community.

- Exposure-response – the relationships utilised in this health impact assessment are based on the most current, robust studies that relate to health effects from exposure to changes in nitrogen dioxide, particulates and noise. The relationships adopted come from large epidemiology studies that include a number of co-pollutants (that is, exposure occurs to a wide range of factors, not just the pollutant being evaluated) and confounding factors that can result in more conservative relationships being developed. In addition, it is assumed the relationships adopted are linear and apply to small changes in air quality or noise, at levels that would not be measurable with air monitoring or noise monitoring equipment.

12. Summary of outcomes of health impact assessment

This section summarises the health impacts and benefits identified in relation to the project.

Table 12.1 summarises the health impacts and benefits for the overall project. The risk pathways and EPRs identified to mitigate potential impacts are provided in **Appendix I Risk pathways**.

Table 12.1: Summary of health impact assessment

Health aspect/ issue	Health impacts identified	Health benefits identified
Air quality		
Construction	Potential for some impacts to occur during construction, although these would be minimised with implementation of EPRs AQ1, CL1 and SC2.	None
Operations	The project would result in some redistribution of traffic on surface roads, which would redistribute air emissions. For the community/population within the study area, health impacts from the tunnel ventilation structures and changes on surface roads are considered to be low and not measurable within the community. Localised health impacts from the tunnel ventilation structures and the redistribution of surface road traffic are considered to be low and not measurable within the community. Emissions to air during operations would be managed with implementation of EPRs AQ3 to AQ5.	The redistribution of traffic on surface roads would mean lower volumes of traffic and truck movements and lower levels of exposure to air emissions for residents adjacent to some roadways.
In-tunnel exposures	In-tunnel air guidelines for carbon monoxide and nitrogen dioxide would be adequately protective of the health of tunnel users. Short-duration exposures to higher levels of particulates should be minimised through providing advice to motorists to keep windows closed and switch ventilation to recirculation.	None
Vibration		
Construction	Some vibration impacts identified would require management as outlined in EPRs NV3 to NV6, NV8 to NV12. Where these impacts were appropriately managed, no impacts on human health are expected within the community.	None
Operation	No operational vibration impacts identified.	None
Noise		
Construction	Noise impacts identified during construction works would be managed with implementation of EPRs NV3 to NV6, NV8 to NV12. Where these measures were implemented the potential for noise impacts to result in significant health impacts in the community is low (with residual risks estimated to be medium). However, it is expected that some individuals within the community may find construction noise annoying at times, even with mitigation. The management of noise impacts during construction needs to include a notification and complaints system, as per EPR SC2.	None

Health aspect/ issue	Health impacts identified	Health benefits identified
Operation	There are some localised areas where an increase in noise has been predicted. In some areas additional noise mitigation has been identified to minimise the impact of changes in project-related noise. Where these additional noise mitigation measures were implemented, changes in noise levels associated with the project are not expected to result in health impacts within the community that would be measurable. Noise impacts during operation of the project would be managed with implementation of EPRs NV1 and NV13, with monitoring undertaken in accordance with EPRs NV2 and NV7.	The project-specific noise targets adopted for the project would result in an overall reduction in community exposure to noise. In many locations the project would reduce noise levels which may be of some benefit to health.
Social aspects		
Economic	Some impacts on business have been identified during construction due to acquisitions and access issues. These impacts would need to be managed to avoid adverse impacts on businesses or existing jobs.	Construction of the project would provide the opportunity for increased employment. Once the project was complete, a number of significant economic benefits would be generated for the north-east of Melbourne as well as Victoria. In addition, a range of benefits have been identified for businesses in the region with productivity and efficiency gains, improved access and potential for people to live closer to and be more accessible to places of employment. Increased employment has a range of significant health benefits. In addition, reductions in stress and anxiety levels due to improved business and home connectivity also generate health benefits.
Traffic	Some increase in traffic congestion during construction may increase stress and anxiety levels.	Once constructed, reduced travel times and less variable travel times would result in lower levels of stress and the potential for additional time to be used for social or physical exercise.
Safety	Construction works may cause additional congestion and increased the risk of accidents on some roads.	The project is expected to reduce crashes and improve pedestrian and cyclist safety through upgrades to the active transport network.
Public transport	Existing public transport routes would be maintained and any impacts managed with a Traffic Management Plan during construction to minimise disruption.	The project would include upgrades to the public transport network that would decrease travel times and boost patronage of these services.

Health aspect/ issue	Health impacts identified	Health benefits identified
Pedestrian and cyclist access	During construction, impacts on existing walking and cycling routes would be minimised with the provision of suitable alternatives. It is recommended that alternative routes maintain pedestrian and cyclist safety to prevent any increase in accidents or a perception of unsafe travel routes, discouraging and impacting the health benefits of these activities.	The proposed improvements to pedestrian and cyclist access and travel have the potential to generate health improvements with improved connectivity and the increased access to and use of safe walking and cycling routes and reduced pedestrian and cyclist accidents.
Health and emergency services	No changes to the access or availability of these services as a result of the project.	NA
Contamination	The remediation of contaminated land could affect the community during construction if not appropriately managed. The management of soil and groundwater during construction with implementation of EPRs CL1 to CL5, and GW2 to GW4, would prevent the local community from being exposed to chemicals associated with areas of contamination, during construction and post construction. No health impacts would be associated with the management of these materials.	Remediation of some contaminated land would reduce the level of pollutants at these locations and any future exposures by the community that may have occurred.
Green space	The project is not expected significantly impact community health and wellbeing from the temporary use of some green space areas for construction and permanent loss of some green space and recreation facilities (specifically the Bulleen Swim Centre) during operations. Management of changes in green space and access to recreational facilities during construction would be in accordance with EPRs SC1 to SC4 and AR1 to AR3. Changes to the visual landscape may affect community use of some areas, but health impacts associated with these changes are considered low, noting that many will adapt to visual changes associated with the project.	None identified
Community	A range of impacts related to the project on community cohesion, perceptions, amenity and wellbeing and visual amenity were identified. All impacts could be effectively managed with implementation of EPRs SC1 to SC4 so the impact of the project on the community is considered to be low.	Upgrades to pedestrian and cyclist access, reduced travel times and the reduction in some local traffic movements have the potential to improve community cohesion, safety and reduce stress and anxiety.
Equity	No impacts have been identified with potential to be unfairly or unequally distributed within the community.	None identified

13. Conclusions

An assessment of health impacts associated with the project has been undertaken. The health impact assessment considered changes in emissions to air and noise and vibration resulting from the construction and operation of the project. In addition, the assessment considered impacts (negative and positive) on health associated with social changes associated with the project.

Based on the assessment undertaken and presented in this report, the following is concluded:

Construction

Where appropriate management mitigation measures as set out in the EPRs are implemented to manage dust emissions, noise and vibration during construction and property acquisition, residual risks to human health are considered low. It is expected there may be some disruptions to local traffic, pedestrian and cyclist access during construction. In addition, there would be some temporary loss of green space during construction, but these losses are not considered to significantly affect community access to existing sporting fixtures or alternative facilities.

Operation

Changes in air quality (impacts) due to emissions from the tunnel ventilation structures as well as the redistribution of traffic on surface roads within the broader study area (community) are not considered to be associated with significant or measurable impacts on community health.

There are some localised changes associated with the redistribution of traffic on surface roads that would improve air quality, and the potential for some health benefits. Some other localised areas would experience impacts (or decreased air quality) from the redistribution of surface road traffic. These impacts are not considered to be associated with significant or measurable impacts on community health.

Changes in noise due to the project are expected to reduce overall noise impacts from road traffic, potentially resulting in some health benefits. Where localised changes in noise are considered (including localised areas of increased noise), and where proposed noise mitigation measures are considered (including at-property treatments) there would be no significant health impacts.

A range of other changes are associated with the project, including faster travel times, greater connectivity, more employment opportunities and jobs growth, and improvements in public transport and active transport networks. These all have potential to generate health benefits within the community. However, some changes may increase stress and anxiety levels. Where these impacts are managed appropriately with implementation of the project EPRs, there would be no significant impacts to community health.

14. Report limitations

This publication is prepared to inform the public about the North East Link.

This publication may be of assistance to you but the North East Link Project (a division of the Major Transport Infrastructure Authority (MTIA)) and its employees, contractors or consultants (including the issuer of this report) do not guarantee the report is without any defect, error or omission of any kind or is appropriate for your particular purposes, and therefore disclaims all liability for any error, loss or other consequence which may arise from relying on any information in this publication.

Environmental Risk Sciences has prepared this report 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 report.

This report was prepared in accordance with the scope of work and for the purpose outlined in **Sections 1 and 3**.

The methodology adopted and sources of information used are outlined in this report. 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.

This report was prepared between September 2018 and February 2019 and is based on the information provided and reviewed at that time. Environmental Risk Sciences disclaims responsibility for any changes that may have occurred after this time.

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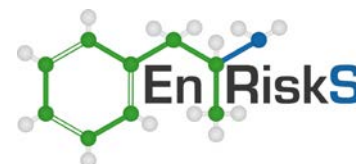
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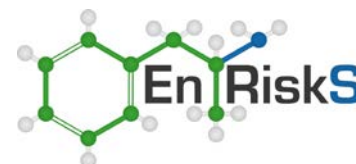
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Appendix A Definition of Terms used in health impact assessment



Health

The World Health Organisation defines health as '*a (dynamic) state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity*'.

The assessment of health should thus include the traditional and medical definition that focuses on illness and disease as well as the more broad social definition that includes the general health and wellbeing of a population.

Health hazard

These are aspects of the project, or specific activities that present a hazard or source of negative risk to the health or wellbeing.

In relation to the health impact assessment, these hazards may be associated with specific aspects of the proposed development/construction or operational activities, incidents or circumstances that have the potential to directly affect health. In addition, some activities may have a flow-on effect that results in some effect on health. Health hazards may thus be identified on the basis of the potential for direct and indirect effects on health.

Health outcomes

These are the effects of the activity on health. These outcomes can be negative (such as injury, disease or disadvantage), or positive (such as good quality of life, physical and mental wellbeing, reduction in injury, diseases or disadvantage).

It is noted that where health effects are considered these are also associated with a time or duration with some effects being experienced for a short period of time (acute) and other for a long period of time (chronic). The terminology relevant to acute and chronic effects is most often applied to the assessment of negative/adverse effects as these are typically the focus of technical evaluations of various aspects of the project.

Likelihood

This refers to how likely it is that an effect or health outcome would be experienced. It is often referred to as the probability of an impact occurring.

Risk

This is the chance of something happening that would have an impact on objectives. In relation to the conduct of the health impact assessment, the concept of risk more specifically relates to the chance that some aspect of the project would result in a reduction or improvement in the health and or wellbeing of the local and regional community.

Equity

Equity relates to the potential for the project to lead to impacts that are differentially distributed in the surrounding population. Population groups may be advantaged or disadvantaged based on age, gender, socioeconomic status, geographic location, cultural background, aboriginality, and current health status and existing disability.



Appendix B Approach to risk assessment using exposure-response relationships

B1 Mortality and morbidity health endpoints

A quantitative assessment of risk for these endpoints uses a mathematical relationship between an exposure concentration (concentration in air) and a response (namely a health effect). This relationship is termed an exposure-response relationship and is relevant to the range of health effects (or endpoints) identified as relevant (to the nature of the emissions assessed) and robust (as identified in the main document). An exposure-response relationship can have a threshold, where there is a safe level of exposure, below which there are no adverse effects; or the relationship can have no threshold (and is regarded as linear) where there is some potential for adverse effects at any level of exposure.

In relation to the health effects associated with exposure to nitrogen dioxide and particulate matter, no threshold has been identified. For the assessment of noise, exposures above a threshold have been defined on the basis of an exposure-response relationship. Non-threshold exposure-response relationships have been identified for the health endpoints considered in this assessment.

B2 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)¹¹ where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

The calculation of changes in health endpoints associated with exposure to nitrogen dioxide, particulate matter or noise as outlined by the WHO (Ostro, 2004) has considered the following four elements:

- Estimates of the changes in particulate matter exposure levels or noise levels (incremental impacts) due to the project for the relevant modelled scenarios
- Estimates of the number of people exposed to particulate matter or noise at a given location
- Baseline incidence of the key health endpoints that are relevant to the population exposed
- Exposure-response relationships expressed as a percentage change in health endpoint per $\mu\text{g}/\text{m}^3$ change in NO_2 or particulate matter exposure or per dB(A) for noise, where a relative risk (RR) is determined.

¹¹ For regional guidance, such as that provided for Europe by the WHO (WHO 2006b) regional background incidence data for relevant health endpoints are combined with exposure-response functions to present an impact function, which is expressed as the number/change in incidence/new cases per 100,000 population exposed per microgram per cubic metre change in particulate matter exposure. These impact functions are simpler to use than the approach adopted in this assessment, however in utilising this approach it is assumed 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 the population is similar to that in Europe (where these relationships are derived).

By applying these elements, the increased incidence of a health endpoint corresponding to a particular change in exposure can be calculated using the following approach:

Noise

Noise impacts have been calculated on the basis of:

$$\text{Equation 1} \quad \text{AF}_{\text{Noise}} = \frac{RR_{dB} - 1}{RR_{dB}} \times P \times B$$

Where:

B = baseline incidence of a given health effect (such as mortality rate per person per year)

P = relevant exposed population

RR_{dB} = relative risk, which is given per 10 dB increase, which is then scaled to be a change per dB as outlined in Equation 2

$$\text{Equation 2} \quad RR_{dB} = 1 + \left((RR_{10} - 1) \times \frac{dB}{10} \right)$$

Where:

dB = is the noise exposure, or change in noise exposure

P = relevant exposed population

RR₁₀ = relative risk per 10 dB increase from publications

Air quality

For the assessment of changes in air pollution, the attributable fraction/portion (AF) of health effects from air pollution, or impact factor, can be calculated from the relative risk as:

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

The assessment of potential risks associated with these exposures 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 linear¹². The calculation of a relative risk based on the change in relative risk exposure concentration from baseline/existing (that is, based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro, 2004):

¹² 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 micrograms per cubic metre, (relevant for evaluating potential impacts associated with air quality goals or guidelines) but lower relative risks below and above this range. For this assessment (where impacts from a particular project are being evaluated) the impacts assessed relate to concentrations of PM_{2.5} that are well below 10 micrograms per cubic metre and so use of the linear relationship is expected to provide a more conservative estimate of relative risk.

Equation 4 $RR = \exp[\beta(X-X_0)]$

Where:

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

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 $\mu\text{g}/\text{m}^3$ increase in particulate matter exposure

Based on this equation, where the published studies have derived relative risk values that are associated with a 10 $\mu\text{g}/\text{m}^3$ increase in exposure, the β coefficient can be calculated using the following equation:

Equation 5 $\beta = \frac{\ln(RR)}{10}$

Where:

RR = relative risk for the relevant health endpoint as published ($\mu\text{g}/\text{m}^3$)

10 = increase in particulate matter concentration or noise level associated with the RR (where the RR is associated with a 10 $\mu\text{g}/\text{m}^3$ increase in concentration)

The total number of cases attributable to exposure to the change in exposure (where a linear dose-response is assumed) can be calculated as:

Equation 6 $E = AF \times B \times P$

Where:

B = baseline incidence of a given health effect (such as mortality rate per person per year)

P = relevant exposed population

The above approach (while presented slightly differently) is consistent with that presented in Australia (Burgers & Walsh, 2002), US (OEHHA, 2002; USEPA, 2005a, 2010) and Europe (Martuzzi et al., 2002; Sjoberg et al., 2009).

The calculation of an increased incidence (that is, number of cases) of a particular health endpoint is not relevant to a specific individual, rather this is relevant to a statistically relevant population. This calculation has been undertaken for populations within the suburbs surrounding the proposed project.

When considering the potential impact of the project on the population for changes in air quality, the calculation has been undertaken using the following:

- The relative risk has been calculated for a population weighted annual average incremental increase in concentrations. The population weighted average has been calculated on the basis of the smallest statistical division provided by the Australian Bureau of Statistics within a suburb (that is, mesh blocks – which are small blocks that cover an area of approximately 30 to 60 urban residences). For each mesh block in a suburb the average incremental increase in concentration has been calculated and multiplied by the population living in the mesh block (data available from the ABS for the 2016 census year). The weighted average has been calculated by

summing these calculations for each mesh block in a suburb and dividing by the total population in the suburb (in all the mesh block)

- The attributable fraction has then been calculated
- Equation 6 has been used to calculate the increased number of cases associated with the incremental impact evaluated. The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered and the population (for the relevant age groups) present the area evaluated.

The above approach can be simplified (mathematically, where the incremental change in particulate concentration is low, in the order of one microgram per cubic metre or less):

Equation 7 $E = \beta \times B \times \sum_{mesh} (\Delta X_{mesh} \times P_{mesh})$

Where:

β = slope coefficient relevant to the per cent change in response to a 1 $\mu\text{g}/\text{m}^3$ change in exposure concentration

B = baseline incidence of a given health effect per person (such as annual mortality rate)

ΔX_{mesh} = change (increment) in exposure concentration in $\mu\text{g}/\text{m}^3$ as an average within a small area defined as a mesh block (from the ABS – where many mesh blocks make up a suburb)

P_{mesh} = population (residential – based on data from the ABS) within each small mesh block

An additional risk is calculated as:

Equation 8 $\text{Risk} = \beta \times \Delta X \times B$

Where:

β = slope coefficient relevant to the per cent change in response to a 1 $\mu\text{g}/\text{m}^3$ change in exposure

ΔX = change (increment) in exposure concentration in $\mu\text{g}/\text{m}^3$ relevant to the project at the point of exposure

B = baseline incidence of a given health effect per person (such as annual mortality rate)

This calculation provides an annual risk for individuals exposed to changes in air quality from the project at specific locations (such as the maximum, or at specific sensitive receiver locations). The calculated risk does not take into account the duration of exposure at any one location and so is considered to be representative of a population risk.

The above calculation of additional risk can also be undertaken for changes in noise levels in the community.

B3 Quantification of short-and long-term effects

The concentration-response functions adopted for the assessment of exposure are derived from long and short-term studies and relate to short or long-term effects endpoints (such as a change in incidence from daily changes in nitrogen dioxide or particulate matter, or chronic incidence from long-term exposures to particulate matter).

Long-term or chronic effects are assessed on the basis of the identified exposure-response function and annual average concentrations. These then allow the calculation of a chronic incidence of the assessed health endpoint.

Short-term effects are also assessed on the basis of an exposure-response function that is expressed as a percentage change in endpoint per microgram per cubic metre change in concentration. For short-term effects, the calculations relate to daily changes in nitrogen dioxide and particulate matter exposures to calculate changes in daily effects endpoints. While it may be possible to measure daily incidence of the evaluated health endpoints in a large population study specifically designed to include such data, it is not common to collect such data in hospitals nor are effects measurable in smaller communities. Instead these calculations relate to a parameter that is measurable, such as annual incidence of hospitalisations, mortality or lung cancer risks. The calculation of an annual incidence or additional risk can be undertaken using two approaches (Ostro, 2004; USEPA, 2010):

- Calculate the daily incidence or risk at each receiver location over every 24-hour period of the year (based on the modelled incremental 24-hour average concentration for each day of the year and daily baseline incidence data) and then sum the daily incidence/risk to get the annual risk
- Calculate the annual incidence/risk based on the incremental annual average concentration at each receiver (and using annual baseline incidence data).

In the absence of a threshold, and assuming a linear concentration-response function (as is the case in this assessment), these two approaches result in the same outcome mathematically (calculated incidence or risk). Given that it is much simpler computationally to calculate the incidence (for each receiver) based on the incremental annual average, compared with calculating effects on each day of the year and then summing, this is the preferred calculation method. It is the recommended method outlined by the WHO (Ostro, 2004).

The use of the simpler approach, based on annual average concentrations should not be taken as implying or suggesting the calculation is quantifying the effects of long-term exposure.

For the calculations presented in this technical working paper that relate to the expected use of the project tunnel—for long-term and short-term effects—annual average concentrations of nitrogen dioxide and particulate matter have thus been utilised.

Where short-term worst-case exposures are assessed (such as those related to a breakdown in the tunnel) short-term, daily, calculations have been undertaken to assessed short-term health endpoints. This has been undertaken as the exposure being assessed relates to an infrequent short-duration event. It would not occur each day of the year and so it is not appropriate to assess on the basis of an annual average.



Appendix C Approach to the assessment of carcinogenic risks

C1 Overall approach

For the assessment of potential risks associated with exposure to diesel particulate matter, carcinogenic PAHs, 1,3-dioxane and benzene, a non-threshold cancer risk is calculated. Non-threshold carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime due to exposure to a potential non-threshold carcinogen. The numerical estimate of excess lifetime cancer risk is calculated as follows for inhalation exposures (enHealth, 2012a; USEPA, 2009b):

$$\text{Carcinogenic Risk (inhalation)} = \text{Concentration in Air} \times \text{Inhalation Unit Risk} \times \text{AF}$$

Where the adjustment factor (AF) is equal to 1, the above calculation assumes the receptor is exposed at the same location for 24 hours of the day, every day, for a lifetime (which is assumed to be 70 years). This assumption is overly conservative for residents and workers in the community surrounding the project. Residents do not live in the one home for a lifetime. Guidance from enHealth indicates that an appropriate assumption for the time living in the one home is 35 years (enHealth, 2012b). For the calculation of carcinogenic risks for residents at this site, an AF of 0.5 has been adopted. This reflects exposure over 35 years at the one home, as a factor of the 70 years assumed as the lifetime of concern for the assessment of carcinogenic risk (enHealth, 2012a).

Assuming that a resident is at home 24 hours per day, every day for a lifetime is considered to be conservative.

C2 Diesel particulate matter

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 (2002a) indicates that diesel exhaust as measured as diesel particulate matter made up about six per cent of the total ambient/urban air PM_{2.5}. In this project, emissions to air from the operation of the tunnel include a significant proportion of diesel powered vehicles. Available evidence indicates 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 (such as episodic) exposure to diesel particulate matter can cause acute irritation (such as eye, throat, bronchial), neurophysiological symptoms (such as 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, 2002a) 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 the diesel particulate matter chronic guideline would also be met if the PM_{2.5} guideline was met.

Review of exposures to diesel particulate matter (USEPA, 2002a) 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 so 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 (2002a) 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 $\mu\text{g}/\text{m}^3$ (mean value of 3.4×10^{-5} per $\mu\text{g}/\text{m}^3$). This would suggest that an increase in lifetime exposure to diesel particulate matter between 0.14 and 0.625 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$. This would suggest that an increase in lifetime exposure to diesel particulate matter of 0.033 $\mu\text{g}/\text{m}^3$ could result in a 1 in 100,000 excess risk of cancer. This estimate has been widely criticised as overestimating the risk and so 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 $\mu\text{g}/\text{m}^3$) 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 AQAR; 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).

The assessment of exposure to diesel particulate matter has utilised an assumption that 80 per cent of PM_{2.5} comprises diesel particulate matter. This assumption is based on a review by Golder of PM_{2.5} sources, as assumed in the emissions inventory for the project. The relevant advice is attached to this Appendix.

Golder memo

TECHNICAL MEMORANDUM

DATE 31 October 2018

Reference No. 1897019-014-M-Rev0

TO David Hyett, AECOM

CC Frank Fleer

FROM Bruce Dawson

EMAIL bdawson@golder.com.au

HEALTH IMPACT ASSESSMENT INPUT: DIESEL PARTICULATE FRACTION OF PM_{2.5}

Golder Associates Pty Ltd (Golder) understands that as part of the health impact assessment (HIA), diesel particulate matter risks are conservatively calculated assuming that 100% of the PM_{2.5} impacts associated with the ventilation structure and surface road emissions are due to diesel particulate.

The ventilation structure and surface road impact assessments detailed in Technical Appendix B Air Quality (Golder document 1897019-007-R-RevE) include traffic emissions calculated using COPERT Australia traffic emission factors in conjunction with predicted project traffic volumes. The PM_{2.5} emission factors combine exhaust and non-exhaust (tyre and brake wear) emissions, therefore the PM_{2.5} impact assessment considers both types of emissions.

COPERT Australia also permits the calculation of exhaust particulate emissions (PM_{ex}), though these have not been included specifically in the Technical Appendix B Air Quality assessments. PM_{ex} refers to PM_{2.5}, as the coarser fraction PM_{2.5-10} is negligible in vehicle exhausts.

Table 1 presents and compares the calculated daily PM_{ex} emissions and PM_{2.5} emissions for the northbound and southbound tunnel ventilation structures, for the 2026 and 2036 scenarios. These values are dependent on the tunnel traffic fleet mix and road gradient, however a similar ratio of PM_{ex} to PM_{2.5} is expected for most surface road emissions.

Table 1: Assessment PM_{2.5} (exhaust and non-exhaust) and corresponding PM (exhaust only) emissions

Scenario	Ventilation structure	PM _{2.5} (kg/day)	PM _{ex} (kg/day)	PM _{ex} / PM _{2.5} (%)
A (2026)	Northern	7.2	5.7	79%
	Southern	4.7	3.7	79%
B (2036)	Northern	8.4	6.6	79%
	Southern	5.4	4.3	79%

The majority of PM_{ex} is attributable to diesel vehicles, e.g. the PM_{ex} emission factor for a diesel passenger car at 80 kph calculated using COPERT Australia is 18 times greater than that of a petrol passenger car; at 20 kph it is 40 times greater. Diesel particulate matter can therefore be conservatively considered to be represented by PM_{ex}.

It follows that approximately 80% of the predicted PM_{2.5} concentrations presented in Technical Appendix B Air Quality and provided for the HIA can be considered diesel particulate matter, the remaining 20% attributed to tyre and brake wear.

If you have any queries, please contact either of the undersigned directly.



Jason Shepherd
Senior Air Quality Consultant



Bruce Dawson
Principal

JAS/BED/jas

<https://golderassociates.sharepoint.com/sites/1897019/deliverables/1897019-014-m-diesel-particulate-fraction-of-pm2.5/1897019-014-m-rev0.docx>



Appendix D Acceptable risk levels

D1 General

The acceptability of an additional population risk is the subject of some discussion as there are currently no guidelines available in Australia, or internationally, in relation to an acceptable level of population risk associated with exposure to particulate matter (and other road-related matters that may impact human health). More specifically, there are no guidelines available that relate to an acceptable level of risk for a small population (associated with impacts from a specific activity or project) compared with risks that are relevant to whole urban populations (that are considered when deriving guidelines). The following provides additional discussion in relation to evaluating calculated risk levels.

'The solution to developing better criteria for environmental contaminants is not to adopt arbitrary thresholds of 'acceptable risk' in an attempt to manage the public's perception of risk, or develop oversimplified tools for enforcement or risk assessment. Rather, the solution is to standardize the process by which risks are assessed, and to undertake efforts to narrow the gap between the public's understanding of actual vs. perceived risk. A more educated public with regard to the actual sources of known risks to health, environmental or otherwise, will greatly facilitate the regulatory agencies' ability to prioritize their efforts and standards to reduce overall risks to public health'. (Kelly, 1991).

Most human activities that have contributed to economic progress present also some disadvantages, including risks of different kinds that adversely affect human health. These risks include air or water pollution due to industrial activities (coal power generation, chemical plants, and transportation), food contaminants (pesticide residues, additives), and soil contamination (hazardous waste). Despite all possible efforts to reduce these threats, it is clear the zero-risk objective is unobtainable or simply not necessary for human and environmental protection and that a certain level of risk in a given situation is deemed 'acceptable' as the effects are so small as to be negligible or undetectable. Risk managers need to cope with some residual risks and thus must adopt some measure of an acceptable risk.

Much has been written about how to determine the acceptability of risk. The general consensus in the literature is that 'acceptability' of a risk is a judgment decision properly made by those exposed to the hazard or their designated health officials. It is not a scientifically derived value or a decision made by outsiders to the process. Acceptability is based on many factors, such as the number of people exposed, the consequences of the risk, the degree of control over exposure, and many other factors.

The USEPA (Hoffman, 1988) 'surveyed a range of health risks that our society faces' and reviewed acceptable-risk standards of government and independent institutions. The survey found that 'No fixed level of risk could be identified as acceptable in all cases and under all regulatory programs...', and that: '...the acceptability of risk is a relative concept and involves consideration of different factors'. Considerations may include:

- The certainty and severity of the risk
- The reversibility of the health effect

- The knowledge or familiarity of the risk
- Whether the risk is voluntarily accepted or involuntarily imposed
- Whether individuals are compensated for their exposure to the risk
- The advantages of the activity
- The risks and advantages for any alternatives.

To regulate a technology in a logically defensible way, one must consider all its consequences (that is, risks and benefits).

D2 10⁻⁶ as an 'acceptable' risk level?

The concept of 1×10^{-6} (10^{-6}) was originally an arbitrary number, finalised by the US Food and Drug Administration (FDA) in 1977 as a screening level of 'essentially zero' or de minimis risk. The term de minimis is an abbreviation of the legal concept, 'de minimis non curat lex: the law does not concern itself with trifles'. In other words, 10^{-6} was developed as a level of risk below which risk was considered a 'trifle' and not of concern in a legal case.

This concept was traced back to a 1961 proposal by two scientists from the National Cancer Institute regarding methods to determine 'safety' levels in carcinogenicity testing. The FDA applied the concept in risk assessment in its efforts to deal with diethylstilboestrol as a growth promoter in cattle. The threshold of one-in-a-million risk of developing cancer was established as a screening level to determine what carcinogenic animal drug residues merited further regulatory consideration. In the FDA legislation, the regulators specifically stated that this level of 'essentially zero' was not to be interpreted as equal to an acceptable level of residues in meat products. Since then, the use of risk assessment and 10^{-6} (or variations thereof) have been greatly expanded to almost all areas of chemical regulation, to the point where today one-in-a-million (10^{-6}) risk means different things to different regulatory agencies in different countries. What the FDA intended to be a lower regulatory level of 'zero risk' below which no consideration would be given as to risk to human health, for many regulators it somehow came to be considered a maximum or target level of 'acceptable' risk (Kelly, 1991).

When evaluating human health risks, the quantification of risk can involve the calculation of an increased lifetime chance of cancer (as is calculated for diesel particulate matter in this assessment) or an increased probability of some adverse health effect (or disease) occurring, over and above the baseline incidence of that health effect/disease in the community (as is calculated for exposure to particulate matter).

In the context of human health risks, 10^{-6} is a shorthand description for an increased chance of 0.000001 in 1 (one chance in a million) of developing a specific adverse health effect due to exposure (over a lifetime or a shorter duration as relevant for particulate matter) to a substance. The number 10^{-5} represents 1 chance in 100,000, and so on.

Where cancer may be considered, lifetime exposure to a substance associated with a cancer risk of 1×10^{-6} would increase an individual's current chances of developing cancer from all causes (which is 40 per cent, or 0.4 – the background incidence of cancer in a lifetime) from 0.4 to 0.400001, an increase of 0.00025 per cent.

For other health indicators considered in this assessment, such as cardiovascular hospitalisations for people aged 65 years and older (for example), an increased risk of 10^{-6} (one chance in a million) would increase an individual's (aged 65 years and older) chance of hospitalisation for cardiovascular disease (above the baseline incidence of 23 per cent, or 0.23) from 0.23 to 0.230001, an increase of 0.00043 per cent.

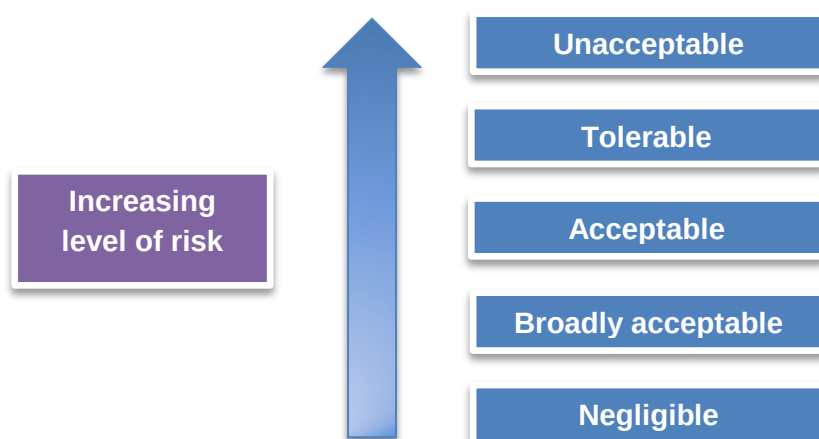
To provide more context in relation to the concept of a one in a million risk, the following presents a range of everyday life occurrences. The activity and the time spent undertaking the activity that is associated with reaching a risk of one in a million for mortality are listed below (Higson, 1989; NSW Planning, 2011):

- Motor vehicle accident – 2.5 days spent driving a motor vehicle to reach one in a million chance of having an accident that causes mortality (death)
- Home accidents – 3.3 days spent within a residence to reach a one in a million chance of having an accident at home that causes mortality
- Pedestrian accident (being struck by vehicles) – 10 days spent walking along roads to reach a one in a million chance of being struck by a vehicle that causes mortality
- Train accident – 12 days spent travelling on a train to reach a one in a million chance of being involved in an accident that causes mortality
- Falling down stairs [1] – 66 days spent requiring the use of stairs in day-to-day activities to reach a one in a million chance of being involved in a fall that causes mortality
- Falling objects – 121 days spent in day-to-day activities to reach a one in a million chance of being hit by a falling object that causes mortality.

This risk level should also be considered in the context that everyone has a cumulative risk of death that ultimately must equal one and the annual risk of death for most of one's life is about one in 1000.

While various terms have been applied, it is clear the two ends of what is a spectrum of risk are the 'negligible' level and the 'unacceptable' level. Risk levels intermediate between these are frequently adopted by regulators with varying terms often used to describe the levels. When considering a risk derived for an environmental impact it is important to consider that the level of risk that may be considered acceptable would lie somewhere between what is negligible and unacceptable, as illustrated below.

[1] Mortality risks as presented by: <http://www.riskcomm.com/visualaids/riskscale/datasources.php>



The calculated individual lifetime risk of death or illness due to an exposure to a range of different environmental hazards covers many orders of magnitude, ranging from well less than 10^{-6} to levels of 10^{-3} and higher (in some situations). However, most figures for an acceptable or a tolerable risk range between 10^{-6} to 10^{-4} , used for either one year of exposure or a whole life exposure. It is noteworthy that 10^{-6} as a criterion for 'acceptable risk' has not been applied to all sources of exposure or all agents that pose risk to public health.

A review of the evolution of 10^{-6} reveals that perception of risk is a major determinant of the circumstances under which this criterion is used. The risk level 10^{-6} is not consistently applied to all environmental legislation. Rather, it seems to be applied according to the general perception of the risk associated with the source being regulated and where the risk is being regulated (with different levels selected in different countries for the same sources).

A review of acceptable risk levels at the USEPA (Schoeny, 2008) points out that risk assessors can identify risks and possibly calculate their value but cannot determine what is acceptable. Acceptability is a value judgment that varies with type of risk, culture, voluntariness and many other factors. Acceptability may be set by convention or law. The review also states the USEPA aims for risk levels between 10^{-6} and 10^{-4} for risks calculated to be linear at low dose, while for other endpoints, not thought to be linear at low dose, the risk is compared to Reference Dose/Concentrations or guideline levels. The USEPA typically uses a target reference risk range of 10^{-4} to 10^{-6} for carcinogens in drinking water, which is in line with World Health Organization (WHO) guidelines for drinking water quality which, where practical, base guideline values for genotoxic carcinogens on the upper bound estimate of an excess lifetime cancer risk of 10^{-5} .

There are many different ways to define acceptable risk and each way gives different weight to the views of different stakeholders in the debate. No definition of 'acceptable' would be acceptable to all stakeholders. Resolving such issues, therefore, becomes a political (in the widest sense) rather than a strictly health process.

The following is a list of standpoints that could be used as a basis for determining when a risk is acceptable or, perhaps, tolerable. The WHO (Fewtrell & Bartram, 2001) address standards related to water quality. They offer the following guidelines for determining acceptable risk. A risk is acceptable when:

- It falls below an arbitrary defined probability
- It falls below some level that is already tolerated
- It falls below an arbitrary defined attributable fraction of total disease burden in the community
- The cost of reducing the risk would exceed the costs saved
- The cost of reducing the risk would exceed the costs saved when the 'costs of suffering' are also factored in
- The opportunity costs would be better spent on other, more pressing, public health problems
- Public health professionals say it is acceptable
- The general public say it is acceptable (or more likely, do not say it is not)
- Politicians say it is acceptable.

In everyday life, individual risks are rarely considered in isolation. It could be argued that a sensible approach would be to consider health risks in terms of the total disease burden of a community and to define acceptability in terms of it falling below an arbitrary defined level. A problem with this approach is that the current burden of disease attributable to a single factor, such as air pollution, may not be a good indicator of the potential reductions available from improving other environmental health factors. For diseases, such as cardiovascular disease, where causes are multifactorial, reducing the disease burden by one route may have little impact on the overall burden of disease.

Further discussion (McClure, 2014) on the level of acceptable risk indicates that the actual size of the exposed population needs to be taken into account. Where the exposed population is, say, 100 people exposed over a lifetime, then if each person is subject to a 1 in 10,000 individual risk from the exposure, then the risk to the 100 person population is 0.0001 times 100, which equals 0.01; (that is, there would not be one person affected by the health outcome evaluated). And this is still conservative because it is unlikely that all 100 persons would be in the one place for their lifetime and the exposure would stay the same for that time. In such a case, it is suggested that using the 1 in 10,000 individual risk threshold (which EPA considers to be at the upper end of the acceptable range) because that is not expected to result in even one person impacted by the health effect evaluated.

There is no rational basis for using 1 in 100,000 criteria, much less 1 in million or 1 in 1 million as an acceptable non-threshold risk criteria for all situations.

This is why it is important to also evaluate the population health incidence for the health endpoints evaluated in the health impact assessment as this part of the assessment does not provide an individual risk, but considers the risk over a larger population to determine the incidence. The health impact assessment has evaluated incidence associated with changes in particulate and nitrogen dioxide exposures and there are no population impacts where there is more than one person that may be affected.

D3 Additional context on defining acceptable risks for nitrogen dioxide, particulates and noise

The population is always be exposed to particulates and noise at some level. In addition, it is noted the calculation of incremental risks associated with changes in particulates and noise have not been commonly undertaken for specific projects in Australia or Internationally. Typically, the exposure-response relationships adopted are used to determine population-wide guidelines based on health benefits/costs. No acceptable risk level is defined by the NEPC or WHO in establishing any of the current air quality or noise guidelines.

When considering risks posed by stressors/pollutants to which a population is constantly exposed, some analogy can be made to radon. Acceptable cancer risk levels adopted in the US for inside homes (where natural radon levels affect populations) range from 3 in 100 for residents who are smokers to 2-7 in 1000.

For particulate exposures, the change in particulate levels evaluated for the project should also be considered in the context of the variability of particulate exposures that occur throughout any one day. Particulates are generated from a wide range of activities, with many of these occurring indoors. This can result in higher levels of particulate exposures occurring indoors from a wide range of indoor sources, well in excess of outdoor air or any change in outdoor air levels. **Figure D1** presents a comparison of the maximum change in $PM_{2.5}$ concentrations predicted for the project with comparison against other changes in $PM_{2.5}$ associated with daily activities. This figure shows that over a day (24 hours) the maximum change in $PM_{2.5}$ from the project is very small when compared with changes from other sources people are exposed to over the course of the day which would result in changes in risk levels (for the health endpoints evaluated in the health impact assessment) in excess of 10^{-4} . Similarly, when considering short duration events the maximum change on $PM_{2.5}$ from the project is similar to the changes that occur during vacuuming and cooking.

To provide further context, particulate risks calculated for other major projects in Australia (completed, approved or being completed) are summarised in **Table D1**. This table shows the levels of risk associated with changes in $PM_{2.5}$ from a range of projects. These risks generally lie in the range 10^{-4} to 10^{-6} with some resulting in risks in excess of 10^{-4} . It could be inferred that these risks have been accepted by the community/regulators, in most cases without having had the actual level of risk calculated (as is presented in this report).

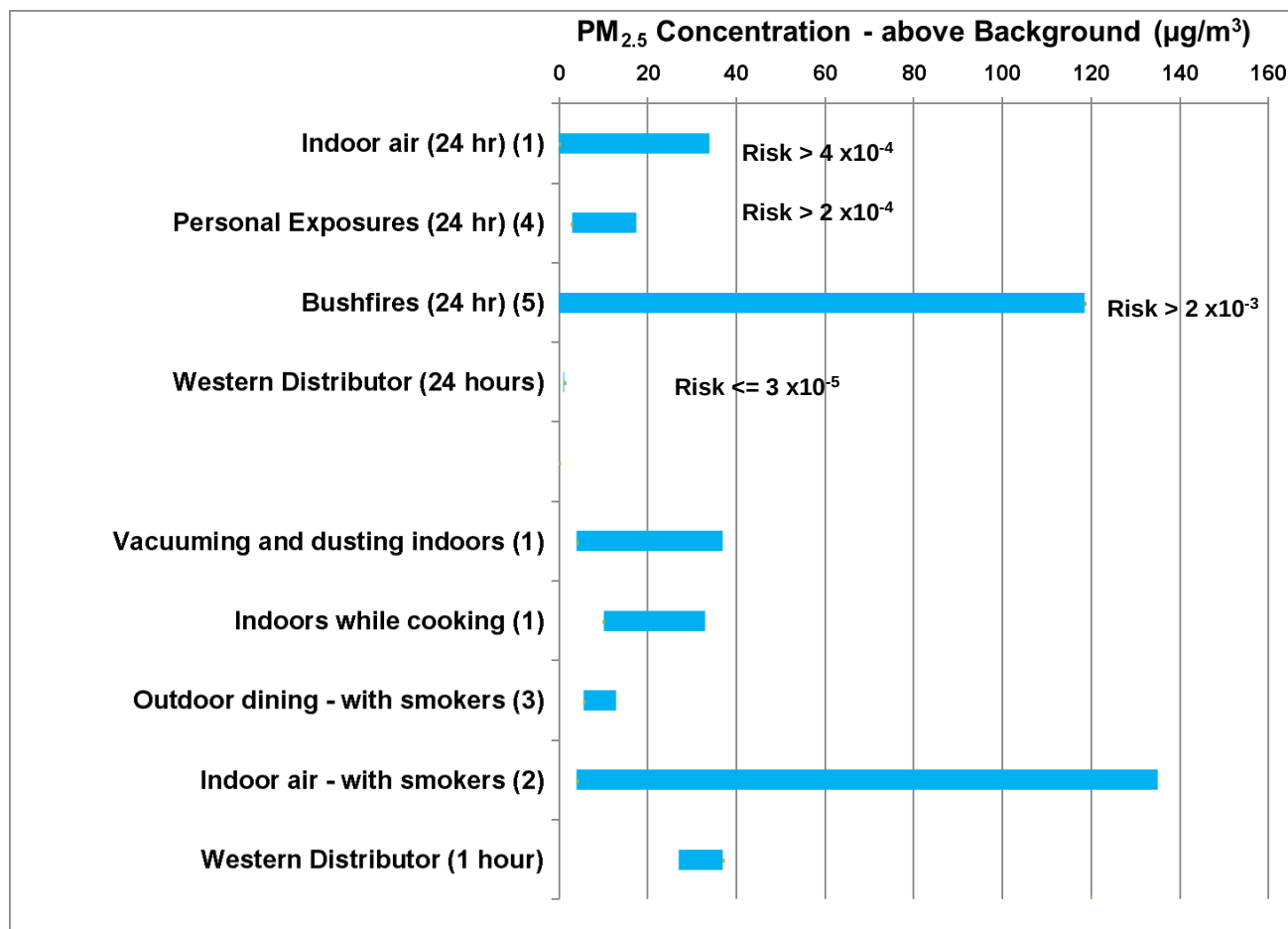


Figure D1 Comparison of incremental (above background) PM_{2.5} concentrations for a range of events and activities

1 – Data for range of indoor activities for homes in Brisbane (Morawska et al. 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/m³ (He et al. 2004). The range reported for cooking activities in Australia are similar to the range reported in other countries (Abdullahi et al. 2013).

2 – Data for PM_{2.5} levels in indoor venues in Western Australia (Stafford et al. 2010).

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

4 – 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).

5 – 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/m³) are often reported when bushfires are present (CSIRO 2008)

Table D1: Summary and comparison of calculated PM_{2.5} risks in off-site community areas for projects completed, approved or under construction in Australia

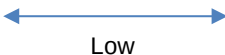
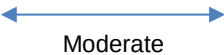
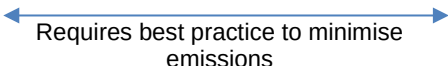
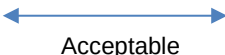
Max Incremental PM increase from project (µg/m ³ , annual average)	Max Incremental Individual Risk
0.1 (NorthConnex and WestConnex)	2x10 ⁻⁵ , 2 in 100,000
0.07 (M5 stack and Brisbane Northern Link Project)	1 in 100,000
0.1 to 1.3 (major roadway widening/upgrade)	2 in 100,000 to 2 in 10,000
0.2 to 1.4 (thermal desorption remediation projects – Homebush Bay and Villawood)	4 in 100,000 to 3 in 10,000
0.6 to 1.5 (long-term development/construction)	1 to 3 in 10,000
Up to 4 (quarry)	7 in 10,000

D4 Determining project-specific risk criteria

Determining an acceptable risk level for the assessment of incremental risks from exposure to air pollutants and noise is challenging as there is currently no specific guidance available. When determining what may be an acceptable risk level, approaches that are available from other regulatory guidance has been considered. These guidance relate to an incremental lifetime or annual risk level that may be applied at either a community exposure level or an individual level. The calculation of risks associated with nitrogen dioxide, particulate and noise exposures relates to an annual risk and so reference to other guidance to determine an acceptable risk relates to chronic risks.

Table D2 summarises the available guidance on chronic risk levels available in other guidance, particularly related to the assessment of air pollution.

Table D2: Risk levels in other Australian regulatory guidance

Source	Incremental risk		
	1 in 1,000,000	1 in 100,000	1 in 10,000
Air pollution based guidance			
NEPC (NEPC, 2011) (community/population mortality risks)	As low as possible but this is upper limit		
NSW Health 2017 (community/population mortality from smoke events up to 3 months)	< Negligible	 Low	 ≥High
NSW EPA (NSW, EPA 2016) for the assessment of localised impacts from specific projects	<Negligible	 >Unacceptable	
Other guidance (not specifically air pollution based)			
NSW Planning (NSW, Planning 2011) for annual fatality risks from hazardous industry	 Acceptable		
NEPC (contaminated land) (NEPC, 1999 amended 2013b) for the assessment of lifetime exposures to genotoxic carcinogens – localised impacts	<Negligible	≤Acceptable >Unacceptable	
NHMRC (drinking water) (NHMRC, 2011 updated 2018) for the assessment of lifetime exposures to genotoxic carcinogens – population impacts	≤Acceptable (basis for drinking water guidelines)		

In addition to the above, a number of recent road tunnel projects (NorthConnex and WestConnex in New South Wales and the West Gate Tunnel Project in Victoria) have adopted project-specific criteria of <1 in 1,000,000 (10^{-6}) as a negligible risk, > 1 in 10,000 (10^{-4}) as an unacceptable risk and between 1 in 1,000,000 (10^{-6}) and 1 in 10,000 (10^{-4}) as an acceptable or tolerable risk. These projects have been approved.

Based on the discussion presented above it is apparent that providing a clear definition of an acceptable risk is challenging. However, for this project, the following has been determined and adopted.

Negligible risk

There is a general consensus that risks below 10^{-6} are considered to be negligible.

Acceptable risk

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

If the level of 10^{-6} (one chance in a million) were retained as a level of increased risk that would be considered as a negligible risk in the community, then the level of risk that could be considered to be acceptable would lie between this level and a higher level that may be considered to be unacceptable.

The acceptability of risk also depends on the population exposed to the pollutant or stressor, and the type of risk calculation undertaken. As discussed in **Sections 8.1 and 9.1** of this report, the assessment of health impacts has considered community (or population) risks as well as localised impacts.

For the assessment of community, or population, exposures the following has been adopted as indicative of acceptable risks:

- Incremental carcinogenic risk, acceptable risk = $\leq 1 \times 10^{-6}$ (that is, negligible risk and the risk level adopted in population wide guidance for the assessment of genotoxic carcinogens such as drinking water guidelines (NHMRC, 2011 updated 2018))
- Incremental population risk from nitrogen dioxide, particulate matter and noise, acceptable risk = $\leq 1 \times 10^{-5}$ (NEPC, 2011). It is noted these criteria have not been applied to the calculation of localised risks.

For the assessment of localised impacts relevant to the assessment of nitrogen dioxide, particulate matter and noise, there is limited guidance available. The assessment of community/population risk provides an evaluation of potential health impacts within a larger population based on the average (or population weighted average) change in exposure that occurs within that population or region. For the assessment of nitrogen dioxide, particulate matter and noise such calculations are appropriate as they draw on exposure-response relationships that are derived from population-wide epidemiological studies (where regional or average air quality is evaluated against changes in population health).

Within any such region or larger population there will be areas where exposures and risks will be higher, as some individuals are located closer to localised sources, and some areas where exposures and risks will be lower, as some individuals will be well away from localised sources. This will also be the case, but not evaluated, with the populations considered in the underlying epidemiological studies from which the exposure-response relationships are derived. For the assessment of a local source, it is important to provide an upper limit for the localised exposures and risks to minimise health impacts associated with these sources. Such a limit will be higher than that adopted for the assessment of community/population risks as noted above. However, it should not be so large that risks are in the range that is considered to be unacceptable.



A level of 10^{-4} for increased risk (one chance in 10,000) has been generally adopted by health authorities as a point where risk is considered to be unacceptable in the development of drinking water guidelines (that impact on whole populations) (for exposure to carcinogens as well as for annual risks of disease (Fewtrell & Bartram, 2001)), from the USEPA and in the evaluation of exposures from air pollutants from specific sources (NSW DEC, 2005). It is therefore relevant to consider an upper limit for a localised risk that is no greater than 10^{-4} (above which risk would be considered unacceptable).

This upper level of risk for the assessment of localised impacts, 10^{-4} , is 10 times higher than the level adopted for the assessment of community or population risks from changes in nitrogen dioxide, particulate matter and noise. Adopting an upper limit for the assessment of localised impacts that is 10 times higher than that adopted for population exposures is consistent with the difference in acceptable risks adopted for population exposures to carcinogens (10^{-6} as outlined by the NHMRC (NHMRC, 2011 updated 2018)) and the assessment of localised carcinogenic risks from contaminated land (10^{-5} as outlined by NEPC (NEPC, 1999 amended 2013b)).

On the basis of the above an upper limit or management risk level of 10^{-4} has been determined to assist in the interpretation of localised impacts.

Health impacts from exposure to changes in nitrogen dioxide, particulate matter and noise for the community are only considered to be acceptable where the community/population risks are below 10^{-5} , AND localised impacts are less than the upper limit or management risk limit of 10^{-4} .

The above criteria are specific to this project and relate to the assessment of localised risks and implementation of risk management measures and should not be considered to be policy for the assessment of all projects in Victoria.

D5 Determination of significance of population impacts

The assessment of potential health impacts associated with emissions to air from the project has not only calculated an increased annual risk, relevant to the health endpoints considered, but also a change in the incidence (that is, the additional (or saving of) number of cases), of the adverse effects occurring within the population potentially exposed. The calculated change in incidence need to be considered in terms of what may be significant.

In relation to the calculated change in incidence of an adverse health effect occurring in a population this has relied on statistics on mortality and hospitalisations. This data reflects records held in the health system and the variability in this data is typically in the order of approximately 2 to 10 per cent. For the health endpoints considered in this assessment this variability translates to a variability in the order of ± 10 to 100 cases per year for mortality and hospitalisations for cardiovascular and respiratory disease. In relation to the incidence of hypertension this translates to approximately ± 1000 to 9000.

The change in the population relevant to the assessment of mortality, hospitalisations and incidence has been reviewed in the context of the above. A change that is considered to be significant would be one which is outside of the normal range of variability expected within the data.

D6 Evaluating the significance of noise annoyance and sleep disturbance

The assessment of changes in annoyance and sleep disturbance requires some consideration of what level of change may be of concern in terms of health, and complaints. Most health-based noise guidelines are set at a level that corresponds with 10 per cent of the residents highly annoyed (NSW DECCW, 2011; WHO, 2018). In relation to sleep-disturbance the WHO (2018) also recommends the use of 3 per cent highly sleep disturbed for the development of guidelines.

Where noise levels change, community reaction to these changes can vary. There is evidence to suggest that reaction to a newly introduced noise source (including expanded rail lines and roads) is considerably higher than a source that has been present for a long time (NSW DECCW, 2011).

There are no specific guidelines available for determining what would be an acceptable, or unacceptable increase in annoyance and sleep disturbance from a project. The New South Wales Road Noise Policy (NSW DECCW, 2011) indicates that a relative increase of 12 dB represents slightly more than a doubling of perceived loudness and is likely to trigger community reaction (complaints). A change of 12 dB corresponds to an increase of 9 per cent population highly annoyed and 8 per cent highly sleep disturbed. This is similar to the 10 per cent population highly annoyed which is the basis for many health-based guidelines, including the WHO (WHO, 1999, 2011, 2018). However, this is a total annoyance level, not a change in annoyance. When evaluating noise annoyance in an urban environment, where there are a number of existing sources, the application of a total 10 per cent highly annoyed criteria is not helpful.

Further review of criteria (Schomer, 2005) that may be considered for evaluating noise annoyance identified a change of 5 dB as a level that would be considered an acceptable change in noise levels in a residential home. A change in noise level of 5 dB results in 5 per cent of the population being highly annoyed – approximately half of the percentage that would be highly annoyed used to determine health-based guidelines and on which the New South Wales guidelines have determined to result in noise complaints. On this basis, a change that exceeds 5 per cent population highly annoyed/disturbed was adopted for this assessment.

In relation to changes in sleep disturbance, the percentage of the population that is highly sleep disturbed would be lower than the percentage that would highly annoyed at the same change in noise level. No guidelines are available for the assessment of changes in sleep disturbance and the health-based reviews relate to determining noise thresholds. The calculation of the change in percentage of population highly sleep disturbed is linked to the change in the percentage of the population highly annoyed (as the noise measures adopted are linked, refer to discussion above). Where a 5 per cent change in the population highly annoyed is adopted, this corresponds to a change of approximately 3 to 4 per cent population highly sleep disturbed. The lower end of this range, 3 per cent is similar to that adopted for the development of guidelines by the WHO (2018). On this basis, a change that exceeds 3 per cent of the population highly sleep disturbed was adopted for this assessment.



Appendix E Characterising health effects of particulate exposure

E1 Introduction

The characterisation of health impacts from changes in particulate matter derived from road traffic has been undertaken on the basis of concentration-response functions. The concentration-response functions have been selected following a decision-making process (as detailed in **Section E3**) that has been developed with consideration of key and relevant health effects associated with exposure to particulate matter (summarised in **Section E2**).

E2 Health effects

E2.1 General

There are numerous publications available that provide statistical associations between exposure to particulate matter (specifically PM_{2.5}) and a variety of health outcomes. These evaluations come from epidemiological studies. However, not all epidemiological studies are of the same quality, or show a consistency of health outcome from the pollutant exposure. Quality and consistency affect the ability of a particular epidemiological study to be used, and for an assessor to be confident about the true nature of the health outcome assessed. For those studies where there is strong evidence that PM_{2.5} exposure is related to the health outcome, that health outcome is considered a core health outcome.

A core health outcome is defined as one where a strength or weight of evidence approach has been undertaken on the epidemiological evidence defining the health outcome, and this strength or weight has been assessed as strong. In other words, we are sure that what has been reported is true and not just due to random chance, bias or confounding.

In the conduct of a health impact assessment it is important the health effects considered and characterised relate to core health outcomes. These core health outcomes are most clearly determined by detailed reviews of the available literature, typically following a documented robust approach to appraising the published epidemiological studies and supporting mechanistic studies to determine core health outcomes that may be considered causal or an association.

The available detailed reviews where core health outcomes are identified include the recent review undertaken for the NEPM review (Jalaludin & Cowie, 2012), WHO and USEPA (including the most current draft science update published in October 2018), summarised below. In addition to these reviews, EPA Victoria has provided an overview of the current understanding of the health effects of PM_{2.5} and a more recent literature review is available that has focused on health effects relate to specific sources of air pollution, including traffic air pollution. The following presents a summary of these reviews.

E2.2 NEPM review

The scope of the NEPM review undertaken by Jalaludin et al (Jalaludin & Cowie, 2012) specifically excluded undertaking a weight of evidence approach, as this involves *evaluating the quality of measurement methods, size and power of study design, consistency of results across studies, and biological plausibility of CRFs* (Concentration Response Functions) *and statistical associations*. This was beyond what could be done for this project. Instead the NEPM review undertaken (Jalaludin & Cowie, 2012) pointed to the weight of evidence reviews undertaken by organisations such as the World Health Organization (WHO) and the US EPA, while documenting CRFs found in the literature that may be considered relevant to Australia. The report does provide recommended CRFs, but this needs to be considered in the context that a weight of evidence approach was not applied when recommending these CRFs.

The selection of recommended CRFs within the NEPM process are identified in the 2013 NEPM Report Exposure Assessment and Risk Characterisation to Inform Recommendations for Updating Ambient Air Quality Standards for PM_{2.5}, PM₁₀, O₃, NO₂, SO₂ (Golder 2013). For particulates, these are summarised below:

■ Short-term (24 hour) PM₁₀:

□ Mortality

- Cardiovascular (all ages)

□ Morbidity

- Asthma (Emergency Department)
- Cardiovascular (65+ years)
- Cardiac including Cardiac Failure (ICD 10:I50)
- Respiratory (<= 14 years)
- Pneumonia & Acute bronchitis (65+ years)

■ Short-term (24 hour) PM_{2.5}:

□ Mortality

- Cardiovascular (all ages)

□ Morbidity

- Asthma (Emergency Department)
- Cardiovascular (65+ years)
- Cardiac including Cardiac Failure (ICD 10:I50)

- Long-term (Annual) PM_{2.5}

- Mortality

- Cardiopulmonary (30+ years)
 - Ischaemic Heart Disease (30+ years)
 - Lung Cancer (30+ years)

As this report was designed to look at regional effects of air pollution, some of the above health endpoints may not have the localised data necessary to calculate out the impact at a local scale.

Referring to the NEPM review undertaken by Jalaludin et al (Jalaludin & Cowie, 2012) and its reference to the weight of evidence approach undertaken by the WHO and US EPA, these organisations recommend the following health endpoints.

E2.3 World Health Organization

In 2013 the WHO released its *Review of evidence on health aspects of air pollution – REVIHAAP Project* (WHO, 2013b). While the report made suggestions for core health outcome related to NO₂ exposure, for particulate matter it summarised the health endpoints identified in published Health Impact Assessments. The following health outcomes are noted:

- Short-term (24 hour) PM_{2.5}:

- Mortality

- All Cause (all ages)

- Long-term (Annual) PM_{2.5}

- Mortality

- All Cause (30+ years)
 - Cardiovascular (30+ years)
 - Cardiopulmonary and Lung Cancer (30+ years)

- Other effects associated with PM exposure (not critically reviewed by WHO):

- Mortality

- Infant (0-1 years)

- Morbidity (noting that many of these outcomes will be difficult to estimate given the lack of baseline incidence rates)
 - Bronchitis symptoms (<18 years)
 - Chronic bronchitis (30+ years)
 - Asthma attacks (all ages)
 - Cardiovascular, cerebrovascular (possibly) and respiratory hospital admissions (all ages)
 - Urgent care visits due to asthma (and possible other respiratory outcomes) and cardiovascular disease (all ages)
 - Restricted activity days (adults)

E2.4 United States Environmental Protection Agency (US EPA)

The US EPA undertook a 'strength of evidence' approach to assess the quality and consistency of epidemiological evidence regarding health effects from exposure to PM_{2.5} in 2009 (USEPA, 2009a) with an update and further review of the science based on published studies to January 2018 (USEPA, 2018). A strength of evidence approach will generally follow key parameters outlined by Bradford Hill (Hill, 1965). In the case of the US EPA, they assessed 'strength of evidence' based on consistency of finding the health outcome across multiple epidemiological studies, coherence of the evidence across disciplines and across related health endpoints, and biological plausibility of the health outcome (USEPA, 2016a, 2018). In doing so, they classify the relationship between the pollutant and health outcome as either;

1. Causal
2. Likely to be causal
3. Suggestive
4. Not sufficient to infer a causal relationship
5. Not likely to be causal.

In estimating the health impacts from PM_{2.5} the US EPA only used health outcomes that were classified as either causal or likely to be causal (USEPA, 2009a, 2018), those being;

For short-term PM_{2.5} exposure

- Mortality (causal relationship)
 - non-accidental
 - cardiovascular-related
 - respiratory-related
- Cardiovascular effects (causal relationship)
 - cardiovascular-related hospital admissions

- Respiratory effects (likely causal relationship)
 - respiratory-related hospital admissions
 - asthma-related emergency department visits

For long-term PM_{2.5} exposure:

- Mortality (causal relationship)
 - all-cause
 - ischemic heart disease (IHD)-related
 - cardiopulmonary-related
 - lung cancer
- Cardiovascular effects (causal relationship)
 - cardiovascular-related hospital admissions
- Respiratory effects (likely causal relationship)
 - respiratory-related hospital admissions
 - asthma-related emergency department visits
- Nervous system effects (likely causal relationship, noting evidence for these effects most strongly observed in animal studies)
 - Cognitive deficits
 - all cause dementia.

Only suggestive relationships were identified for effects associated with short-term PM_{10-2.5} exposure, and inadequate evidence is available for the assessment of long-term exposure to PM_{10-2.5}.

Note it's important to understand the use of multiple endpoints may involve double counting of the health impacts.

E2.5 Summary of health effects of particulate exposure from EPA Victoria

EPA Victoria has recently released an overview of air pollution issues relevant to Victoria (EPA Victoria, 2018). This included a summary of the health effects of exposure to air pollution, including particulates (principally reviews from open access publications, noting the review does not provided critical appraisal of the studies). The following are relevant excerpts from the 2018 review:

General

There is a large body of evidence that demonstrates that air pollution, even at concentrations below the current air quality standards, is associated with adverse health effects (Brook et al., 2010; Burnett et al., 2014; Lim et al., 2012; USEPA, 2009a; WHO, 2006b). The strongest evidence relates to premature mortality and effects on the respiratory and cardiovascular system. In 2013, the International Agency for Research on Cancer classified outdoor air pollution and particulate matter as carcinogenic to humans (IARC, 2016).

Particulate matter is estimated to be the individual pollutant responsible for the largest burden of disease from outdoor pollution (GBD Risk Factors Collaborators, 2017). This is mainly due to its effects on the cardiovascular and respiratory system as the small particles can penetrate deep in to the lung (GBD Risk Factors Collaborators, 2017). In fact, on a global scale, ambient particulate matter is estimated to be responsible for approximately 4.1 million premature deaths (7.5 per cent of global deaths). These deaths are largely caused by chronic lung diseases and lung cancer, heart disease and stroke, and respiratory infections.

Outdoor air pollution is a complex mixture of pollutants that often have similar sources which generally result in a high correlation between pollutants. This can make it difficult to determine the health effects attributable to individual air pollutants. However, PM₁₀, PM_{2.5} and ozone are of most concern in Victoria, as these pollutants present in the highest concentrations with relation to the air quality standards, and they have well-documented adverse health effects such as premature mortality, and acute and chronic respiratory morbidity (Jerrett et al., 2009; Peng et al., 2013; WHO, 2006a).

Population level impacts and susceptibility

Most epidemiological studies that investigate the association between air pollution and health have used existing health registries, such as mortality registries, and registries of hospital admissions and emergency presentations to establish an association. These are all high-level health outcomes in the pyramid of health effects (see below **Figure 23**), meaning they are the least common but have the most severe health effects. There are a whole range of effects that are less severe and are generally not captured by existing health registries, such as symptoms, and sub-clinical health effects. Generally, the only way to measure these less severe health effects are by collecting information on individuals, such as blood markers of inflammation or coagulation which can indicate an effect on the heart, or exhaled nitric oxide which can indicate lung inflammation. Therefore, the studies of the higher-level health outcomes are very informative, but it is important to consider that these only include a small part (the tip of the pyramid) of the wide range of health effects of air pollution.

The impacts on individuals exposed to similar levels of air pollutants can vary considerably, depending on their susceptibility to the effects of air pollutants. Individuals that are generally considered to be more susceptible are those who have existing lung or heart disease, the young and the elderly (Pope, CA & Dockery, 2006).



Figure 23: The air pollution health effects pyramid (Dennekamp & Abramson 2011; adapted from ATS 2000)
(from EPA Victoria, 2018)

Ambient particulate matter

PM_{2.5} is a component of PM₁₀ and both size fractions show clear evidence of being associated with health effects, with PM_{2.5} generally showing stronger associations. Ultrafine particles (UFP), which are a subset of PM_{2.5}, are much smaller and can therefore penetrate further into the lungs. They can only be measured by number due to their small size and are therefore more difficult to measure. Unlike PM_{2.5} and PM₁₀, UFPs are not regulated. A smaller number of studies have investigated the health effects of UFPs. There is some evidence of an association of UFPs with cardiovascular health effects (HEI, 2013; Pieters et al., 2015; Stone et al., 2017). In epidemiological studies, it is difficult to determine whether UFPs have independent effects, with more research needed. This section (as provided by EPA Victoria) will therefore focus on the health effects of PM_{2.5}.

Systematic reviews and meta-analyses investigating the association between PM_{2.5} and effects on health have mainly focused on effects on the respiratory and cardiovascular system. There is clear evidence there is an association between increases in daily average PM_{2.5}, and emergency presentations and hospital admissions for respiratory and cardiovascular conditions and mortality (Atkinson et al., 2014; Katsouyanni et al., 2009; Simpson et al., 2005; WHO, 2006a, 2013b). There is now also evidence from a meta-analysis of an association between increased PM_{2.5} with increased out-of-hospital cardiac arrest using data from Europe, North America, Asia and Australia (Zhao et al., 2017). This included a study conducted in Melbourne (Dennekamp & Carey, 2010). In addition, many studies have also shown an association between exposure to PM₁₀ and PM_{2.5}, and reduced lung function, respiratory symptoms, and physiological and sub-clinical changes, such as heart rate variability, blood markers of inflammation and coagulation (Gold et al., 2000; Gotschi et al., 2008; WHO, 2013b; Brook et al. 2010). It is generally accepted there is a linear relationship

between exposure to particulate matter PM₁₀ and PM_{2.5}, and health outcomes, and there is no safe PM₁₀ and PM_{2.5} level below which no effects are expected (Pope, CA & Dockery, 2006).

Studies investigating long-term exposure to PM_{2.5} have also shown associations with effects on the respiratory and cardiovascular system, in particular cardiopulmonary mortality (WHO, 2013b). A comprehensive review by the American Heart Association concluded that long-term exposures increased the risk of cardiovascular mortality to an even greater extent than exposures over a few days (Brook et al., 2010). They also concluded that reductions in PM_{2.5} levels were associated with decreases in cardiovascular mortality. These conclusions have been confirmed by more reviews and meta-analyses published subsequently (Cesaroni et al., 2014; Hoek et al., 2013). A critical review by the Health Effects Institute (HEI, 2010) concluded that exposure to traffic air pollution adversely affected lung development in childhood. A recent meta-analysis supported the hypothesis that childhood exposure to traffic air pollution contributes to the development of childhood asthma (Khreis et al., 2017).

Using the results from a large meta-analysis done for the WHO (WHO, 2013b, 2013a), a study by Hoek et al. (Hoek et al., 2013) concluded that for every 10 µg/m³ increase in PM_{2.5}, the risk of all-cause mortality increased by 6.2 per cent (95 per cent confidence interval 4.1–8.4 per cent). This is similar to results from the earlier American Cancer Society Study (Pope, 2002). This estimate is most commonly used in health impact assessments (as stated by EPA Victoria). However, a more recent analysis using data from European Cohorts found an even greater increase in all-cause mortality of 13 per cent per 10 µg/m³ increase (95 per cent confidence interval 1–25 per cent) (Beelen et al., 2014). However, the confidence interval around this estimate was much larger, reducing the confidence in the estimate.

E2.6 Summary of health effects from vehicle emissions

A recent review conducted by Hime et al. (2018) involved a review of evidence of health effects associated with exposure to particulate matter from five common outdoor emission sources, that include traffic emissions and diesel emissions.

The review was not a comprehensive assessment of all evidence of health effects associated with particulate matter, but presents a review of the available studies and findings of comparative studies. The paper did not include a systematic review of the quality of the available studies.

The review is noted to be limited, as the different methods that have been used in epidemiological studies, along with the differences in populations, emission sources, and ambient air pollution mixtures between studies, make the comparison of results between studies problematic.

In relation to traffic emissions, the review identified that (Hime et al., 2018):

- Traffic generates airborne particles via exhaust emissions from fuel combustion, as well as the resuspension of non-exhaust PM from road, tyre, and brake wear. Non-exhaust PM is predominantly in the coarse fraction between 2.5 and 10 µm in diameter and is an important source of trace metals in PM in urban environments. Particles from vehicle exhaust constitute the major source of ultrafine particles, <0.1 µm in diameter (PM_{0.1}), in urban environments.

Traffic-generated PM includes secondary PM formed from hot exhaust gases (carbon dioxide, carbon monoxide, hydrocarbons, and nitrogen oxides) expelled from vehicle tailpipes.

- Traffic is a significant contributor to urban air pollution and the health effects of exposure to traffic-related air pollution (TRAP) have been extensively reviewed. An expert panel for the US Health Effects Institute concluded that while many health effects have been associated with exposure to TRAP, only the evidence related to the exacerbation of childhood asthma was sufficient to assign causality. The panel categorized the evidence linking the onset of childhood asthma, respiratory symptoms, impaired lung function, all-cause mortality, and cardiovascular morbidity with exposure to TRAP, as 'suggestive but not sufficient' to infer causation.
- The US Environmental Protection Agency (EPA) has implicated TRAP as a risk factor for myocardial infarction and also concluded that associations between ambient PM_{2.5} and cardiovascular disease hospitalisations may be primarily due to particles from traffic.
- Various toxicological and epidemiological studies implicate traffic-related PM as likely to be causal in the associations between TRAP and cardiovascular health effects. PM_{2.5} apportioned to traffic has been associated with all-cause, respiratory, and cardiovascular mortality, and daily hospital admissions for cardiovascular disease, stroke, and heart failure. In the Harvard Six Cities cohort the magnitude of the effect of exposure to traffic-related PM_{2.5} on daily mortality was greater than that for PM_{2.5} from coal combustion, crustal dust, or total PM_{2.5} mass. In the multi-country study, Air Pollution on Health: A European Approach Study (APHEA2), PM from areas with higher ambient nitrogen dioxide (a marker of traffic emissions) was associated with greater acute health effects, suggesting that PM emitted by traffic is more toxic than PM from other sources.
- Findings from toxicological studies conducted within the NPACT project suggest that PM_{2.5} from vehicle exhaust emissions has greater cardiovascular toxicity than non-exhaust PM_{2.5}, however epidemiological investigations within the project were inconclusive in their support. Particles from traffic have high oxidative potential, possibly due to metals arising from engine and brake abrasion. Some studies, but not all, have demonstrated that as traffic density increases, the capacity of roadside PM to generate tissue-damaging reactive oxygen species increases.

In relation to diesel emissions the review identified that (Hime et al., 2018):

- Diesel exhaust particles from modern, optimal combustion engines are primarily PM_{2.5}, a considerable component of which are PM_{0.1}. They are highly complex particles with a core of elemental carbon and adsorbed organic compounds, as well as small amounts of sulphate, nitrate, metals, and many trace elements.
- The study of the health effects of diesel exhaust PM is complicated by the fact that diesel exhaust PM varies in chemical composition and size according to engine type (heavy-duty, light-duty, method of fuel injection), engine operating conditions (idle, accelerating, decelerating), and fuel formulations (high/low sulphur fuel, petroleum-based diesel, biodiesel). It is unclear how these differences change toxicity.

- A more recent analysis of pooled data from 11 case-control occupational epidemiology studies conducted in Europe and Canada found that cumulative diesel exhaust exposure was associated with an increased lung cancer risk, and in 2012 the International Agency for Research on Cancer (IARC) classified diesel engine exhaust as 'carcinogenic to humans' based on findings from occupational epidemiological studies and toxicological investigations conducted in research animals. The occupational epidemiological studies on which the IARC conclusions were based were limited by a general lack of objective measure of diesel exposure. Recent cohort and nested case-control studies in 12,000 US mine workers, which included PM measurements in their exposure assessment, observed that exposure to diesel exhaust PM was associated with lung and oesophageal cancer mortality. However, not all reports have found such links between diesel exposure and cancer. A systematic review published in 2014 of 42 cohort and 32 case-control studies did not find a clear relationship between diesel exposure and lung cancer. A literature review published in 2012 concluded the occupational epidemiological evidence was inadequate to confirm a link between diesel exhaust exposure and lung cancer, and suggested that weak exposure–response associations could be explained by bias, confounding, chance, or exposure misclassification.
- Due to the difficulty in distinguishing PM derived from diesel exhaust from PM arising from other emission sources, most epidemiological studies have not assessed the effects of exposure to ambient diesel exhaust PM. It is noteworthy the IARC deliberately excluded evidence from non-occupational exposure of diesel exhaust in their assessment of the carcinogenicity of diesel exhaust emissions because of the difficulty in assessing the contribution to cancer risk of diesel exhaust in ambient air.
- The majority of evidence indicating the potential for diesel exhaust PM to cause health effects has come from human chamber studies and studies in research animals. Controlled exposures of humans to diesel exhaust have resulted in various cardiovascular changes indicative of increased acute coronary event risk, mild constriction and inflammation of lung airways, nose and throat irritation, and changes in lung function.
- Many research animal studies support the biological plausibility of the health effects observed in humans exposed to diesel exhaust. As with PM from other sources, it is thought that oxidative stress underpins the mechanism by which diesel exhaust causes health effects, and the effects of diesel exhaust PM may be accentuated in individuals with conditions associated with oxidative stress, such as diabetes and obesity. Diesel exhaust PM has also been shown to enhance susceptibility to infection and increase the atopic response to allergens. Exposure of pregnant mice to diesel exhaust PM has been found to affect the central nervous and immune systems of offspring, as well as their susceptibility to asthma and heart failure. However, there is no evidence of inherited health effects from exposure to diesel exhaust at levels that are typical of ambient environments.

E2.7 Assessment of asthma

In relation to respiratory effects associated with exposure to $PM_{2.5}$, the quantitative assessment has focused on children aged 1-14 years where emergency department admissions has been used as the health endpoint. This health endpoint is considered to provide an indication of the impact of changes in $PM_{2.5}$ (and NO_2) on the exacerbation of asthma, utilising the key public health measure (namely emergency department admissions) where exposure-response relationships can be measured and risks calculated. Children with asthma as a pre-existing health condition are considered to be a sensitive sub-group in relation to respiratory effects. Individuals, particularly children, with asthma tend to have a higher degree of oronasal breathing that can result in a greater penetration of PM into the lower respiratory tract (USEPA, 2018). There is also evidence those with asthma may have altered particulate clearance mechanisms (USEPA, 2018). Review of the epidemiological evidence by the USEPA determined a causal relationship between short-term exposure to $PM_{2.5}$ and exacerbation of asthma. The available studies show the strongest relationships with exacerbation of asthma in children, with some long-term studies providing suggestive evidence of impaired lung function growth in children (USEPA, 2018).

Review of exposures to traffic related air pollution (TRAP), in particular particulate matter, by Hime et al. (2018) indicated that detailed reviews by an expert panel for the US Health Effects Institute concluded that while many health effects have been associated with exposure to TRAP, only the evidence related to the exacerbation of childhood asthma was sufficient to assign causality.

A current review of exposures to $PM_{2.5}$, ozone and NO_2 on asthma (Anenberg et al., 2018) provides a review of the available exposure-response relationships derived from a range of epidemiological studies of varying quality. For the assessment of the exacerbation of asthma and exposure to $PM_{2.5}$ there are no long-term studies (that develop exposure-response relationships) for adults aged 18 to 64 or older adults aged 65 years and over. The only studies relate to children aged <18 years. In relation to short-term exposures to $PM_{2.5}$ most studies identify relationships for children (<18 years) with only a few identifying relationships for adults. Review of these relationships, and relationships relevant to emergency department visits for asthma by the USEPA (2018) indicates the relationships are more significant for children than for adults.

Where NO_2 is considered the most significant exposure-response relationships relate to exposures to children, again with few relationships identified for adults and, where they are identified they are of less significance.

Within the project area the rate of asthma hospitalisations for children is significantly higher than for adults. This same pattern is also expected for emergency department admissions. The calculation of incremental risks associated with exposure to $PM_{2.5}$ (and NO_2) and exacerbation of asthma (where emergency department admissions are used as the public health measure) for children will therefore provide a conservative (they overestimate) estimates of potential risks relevant to asthmatic adults. No separate assessment of asthma health effects on adults has been presented in the assessment.

E3 Approach to identifying concentration response functions

This section has been developed to provide transparency to the decision-making process regarding the concentration response functions used by enRiskS in this assessment to estimate health impacts from changes in PM_{2.5} and PM₁₀. The intention of this appendix is to provide a framework on which to assess papers presented to enRiskS outlining a relationship between PM_{2.5} & PM₁₀ concentration and health outcomes.

Currently used concentration response functions

Table E1 provides the concentration response functions used by enRiskS in a number of large scale development applications, including the WestConnex and NorthConnex road tunnel developments in New South Wales and the West Gate Tunnel Project in Victoria. These values are drawn from recognised and authoritative Australian sources, such as those presented in the National Environment Protection Measure (NEPM) Ambient Air Quality review and are reviewed and agree to by the relevant government authorities in New South Wales prior to use.

Table E1: Frequently used health endpoints and their values by enRiskS – those bolded are considered primary outcomes

Pollutant	Health Endpoint	Health Endpoint	Age group	Relative risk per 10 µg/m ³ pollutant	Adopted β coefficient (also as per cent) for 1 µg/m ³ increase in pollutant	Reference
Short-term (24 hour) PM _{2.5}	Mortality	All causes	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)
		Cardiovascular	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)
		Respiratory (including lung cancer)	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)
	Morbidity	Cardiovascular hospital admissions	≥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)

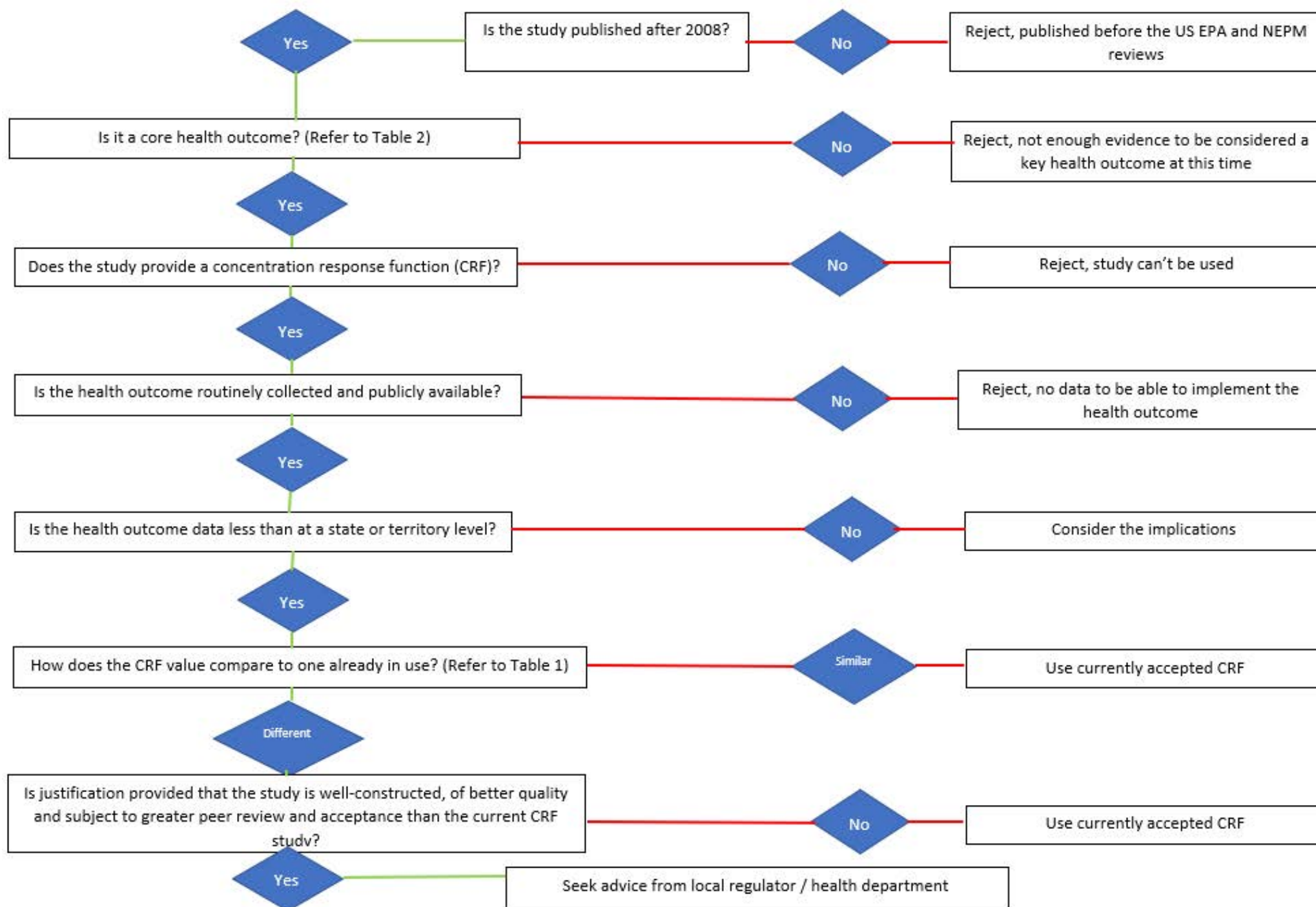
Pollutant	Health Endpoint	Health Endpoint	Age group	Relative risk per 10 $\mu\text{g}/\text{m}^3$ pollutant	Adopted β coefficient (also as per cent) for 1 $\mu\text{g}/\text{m}^3$ increase in pollutant	Reference
		Respiratory hospital admissions	≥ 65 yrs	1.0041 [1.0009–1.0074]	0.00041 (0.041)	Relationship 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)
		Asthma (emergency department admissions)	1–14 years	–	0.00148 (0.148)	Relationship established from review conducted on Australian children (Sydney) for the period 1997 to 2001 (Jalaludin et al. 2008)
Long-term (annual) PM 2.5	Mortality	All causes	≥ 30 yrs	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).
		Cardio pulmonary	≥ 30 yrs	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).

Pollutant	Health Endpoint	Health Endpoint	Age group	Relative risk per 10 $\mu\text{g}/\text{m}^3$ pollutant	Adopted β coefficient (also as per cent) for 1 $\mu\text{g}/\text{m}^3$ increase in pollutant	Reference
Short-term (24 hour) PM 10		All causes	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)

Decision flow chart

The concentration response functions used by enRiskS in development applications to are drawn from recognised Australian and international sources. These sources have undertaken significant review of the health evidence in the area (including its quality) when recommending a concentration response function. Further, prior to use in an assessment the relevant concentration response functions are reviewed for appropriateness by the local regulator/health department. Under this pretext a decision flow chart has been developed that can efficiently consider papers presented in situations such as a Planning Panels, which is common in the State of Victoria (**Figure E1**).

Table E3 provides an example of how this has been applied.



Is the study published after 2008?

There are three main authoritative sources that have extensively reviewed the literature related to $PM_{2.5}$, PM_{10} and their health outcome concentration response functions. The oldest was published in 2009 by the United States Environmental Protection Agency (USEPA)—*USEPA Integrated Science Assessment for Particulate Matter*—meaning that all papers before 2009 were available for review and critique by the USEPA (2009a). This review has been recently updated with a draft available in 2018 (USEPA, 2018). In 2012 an Australian review of the NEPM specifically examining concentration response functions was published—*Health Risk Assessment – Preliminary Work to Identify Concentration-Response Functions for Selected Ambient Air Pollutants* (Jalaludin & Cowie, 2012). Finally, in 2013 the World Health Organization published its *Review of evidence on health aspects of air pollution – REVIHAAP Project* (WHO, 2013b). Given these key reviews had access to papers published before 2009 (at the very least), a cut-off date criterion of ‘after 2008’ has been applied.

Is it a core health outcome?

Core health outcomes have been determined from the USEPA Integrated Science Assessment for Particulate Matter which undertook a weight of evidence approach to determine the strength of associations between particulate matter exposure and health outcomes, as well as those used in the Australian NEPM process. It should be noted the NEPM work examining concentration response functions (*Health Risk Assessment – Preliminary Work to Identify Concentration-Response Functions for Selected Ambient Air Pollutants*) specifically excluded undertaking a weight of evidence approach, as this involves *evaluating the quality of measurement methods, size and power of study design, consistency of results across studies, and biological plausibility of CRFs* (Concentration Response Functions) *and statistical associations*. This was beyond what could be done for this project. Instead the NEPM review pointed to the weight of evidence reviews undertaken by the USEPA, while documenting concentration response functions found in the literature that may be considered relevant to Australia. **Table E2** lists the core health outcomes determined.

Table E2: Core health outcomes

Pollutant	Timeframe	Mortality	Morbidity
$PM_{2.5}$	Short term	All cause Cardiovascular Respiratory	Cardiovascular Respiratory (including asthma)
$PM_{2.5}$	Long term	All cause Cardiovascular Respiratory	Cardiovascular Respiratory (including asthma)
PM_{10}	Short term	Cardiovascular	Cardiovascular Respiratory (including asthma)

Does the study provide a concentration response function?

Does the study provide a relationship between increase in pollutant and health outcome, and define the shape of that relationship such that it can be used to estimate impacts? If it does not, it cannot be used for the purpose of estimating health impacts.

Is the health outcome routinely collected and publicly available?

If the health outcome is not routinely collected or publicly available in the population of interest, then there is no ability to be able to estimate the impact.

Is the health outcome data less than at a state or territory level?

Health outcome data at a state or territory level may not provide the impact at a localised level, however small unit (localised) health outcome data is subject to instability which may provide an unrealistic estimate of impact.

How does the value found in the paper relate to what is currently being used?

Is the value the same or close to the same?

Has justification been provided that the study is well-constructed, of better quality and subject to greater peer review and acceptance than the current concentration response function study?

Has a case been provided regarding the quality of the advocated study and how it is more applicable than the current study used to define the concentration response function? Age of the study is not an appropriate justification and the study quality along with comprehensive review and acceptance by other government authorities is an important consideration. Undertaking a literature of studies involving particulate matter exposure and summarising them within providing context for their use or justification for their use is not appropriate.

Table E3: Example from papers presented at a Victorian planning panel

Paper	Published after 2008	Core health outcome	Provide a CRF	Routinely collected and publicly available health data	Health data at smaller area than state level	CRF value. Similar CRF to currently used?	Justified, well-constructed and highly peer reviewed study accepted by well-known government agencies
Pope (1989) Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley	No						
Heinrich (2003) Nonallergic Respiratory morbidity improved along with a decline of traditional air pollution levels: a review	No						
Raaschou Nielsen (2013) Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE)	Yes	Yes, Long-term PM _{2.5} and lung cancer	Yes	Yes	?	Lung cancer 1.18 (0.96–1.46) per 5 µg/m ³ ?	No
Pedersen (2013) Ambient air pollution and low birthweight: a European cohort study (ESCAPE)	Yes	No					
Oudin (2016) Traffic-Related Air Pollution and Dementia Incidence in Northern Sweden: A Longitudinal Study	Yes	No					
MacIntyre (2014) Air Pollution and Respiratory Infections during Early Childhood: An Analysis of 10 European Birth Cohorts within the ESCAPE Project	Yes	No					
Li (2016) Short-Term Exposure to Air Pollution and Biomarkers of Oxidative Stress: The Framingham Heart Study	Yes	No					

Paper	Published after 2008	Core health outcome	Provide a CRF	Routinely collected and publicly available health data	Health data at smaller area than state level	CRF value. Similar CRF to currently used?	Justified, well-constructed and highly peer reviewed study accepted by well-known government agencies
Lepeule (2012) Chronic Exposure to Fine Particles and Mortality: An Extended Follow-up of the Harvard Six Cities Study from 1974 to 2009	Yes	Yes, Long-term PM _{2.5} and mortality (all cause, cardiovascular, lung cancer, COPD)	Yes	Yes	?	All cause 1.14 per 10ug/m³ increase Cardiovascular 1.26 per 10ug/m³ increase Lung cancer 1.37 per 10ug/m³ increase COPD 1.17 per 10ug/m³ increase	No
Kioumourtzoglou (2016) Long-term PM _{2.5} Exposure and Neurological Hospital Admissions in the Northeastern United States	Yes	No					
Hamra (2014) Outdoor Particulate Matter Exposure and Lung Cancer: A Systematic Review and Meta-Analysis	Yes	Yes, Long-term PM _{2.5} and mortality (lung cancer)	Yes	Yes	?	Lung cancer 1.09 per 10ug/m ³ increase	No (Provides an estimate that includes the studies by Lepeule (2012) and Raaschou Nielsen (2013))

Paper	Published after 2008	Core health outcome	Provide a CRF	Routinely collected and publicly available health data	Health data at smaller area than state level	CRF value. Similar CRF to currently used?	Justified, well-constructed and highly peer reviewed study accepted by well-known government agencies
Gehring (2013) Air Pollution Exposure and Lung Function in Children: The ESCAPE Project	Yes	No, but long-term PM _{2.5} and lung function is mentioned in the US ISA but not taken up in the US EPA Risk Assessment					
Gauderman (2015) Association of Improved Air Quality with Lung Development in Children	Yes	No, but long-term PM _{2.5} and lung function is mentioned in the US ISA but not taken up in the US EPA Risk Assessment					
Gasana (2012) Motor vehicle air pollution and asthma in children: A meta-analysis	Yes						No (Did not find a significant association between PM _{2.5} and asthma or PM ₁₀ and asthma. Difficult paper as exposure periods are all over the place)

Paper	Published after 2008	Core health outcome	Provide a CRF	Routinely collected and publicly available health data	Health data at smaller area than state level	CRF value. Similar CRF to currently used?	Justified, well-constructed and highly peer reviewed study accepted by well-known government agencies
Feigin (2016) Global burden of stroke and risk factors in 188 countries, during 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013	Yes		No				
Di (2017) Air Pollution and Mortality in the Medicare Population	Yes	Yes, Long-term PM _{2.5} and mortality	Yes			1.073 (1.071 – 1.075)	No (Possible use of the Medicare cohort, so sensitive subpopulation?)
Dennekamp (2010) Outdoor Air Pollution as a Trigger for Out-of-hospital Cardiac Arrests	Yes	Yes, Short-term PM _{2.5} and out of hospital cardiac arrest	? Interquartile range				No Was it considered by Jal
Clifford (2016) Exposure to air pollution and cognitive functioning across the life course – A systematic literature review	Yes	No					
Cesaroni (2014) Long-term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE Project	Yes						Combined morbidity and mortality
Bowatte (2015) The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies	Yes	No					

Paper	Published after 2008	Core health outcome	Provide a CRF	Routinely collected and publicly available health data	Health data at smaller area than state level	CRF value. Similar CRF to currently used?	Justified, well-constructed and highly peer reviewed study accepted by well-known government agencies
Beelen (2014) Effects of long-term exposure to air pollution on natural-cause mortality: an analysis of 22 European cohorts within the multicentre ESCAPE project	Yes	? PM _{2.5} Long-term non accident mortality				1.07 (1.02 – 1.07) per 5ug/m ³	No
Balti (2014) Air pollution and risk of type 2 diabetes mellitus: A systematic review and meta-analysis	Yes	No					



Appendix F Health risk calculations: changes in nitrogen dioxide exposures

Health impacts associated with changes in nitrogen dioxide have been calculated on the basis of predicted changes in annual average nitrogen dioxide concentrations for the conservative emissions scenario for 2026 and 2036 and the realistic emissions scenario for 2036.

Risks and population incidence have been calculated for each individual receptor for the health endpoints and exposure-response functions listed in **Table F1** (which is a repeat of **Table 8.9**).

Table F1: Adopted exposure-response relationships for assessment of changes in nitrogen dioxide concentrations

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

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

The attached spreadsheets present the calculations undertaken for population/community risks and incidence as well as localised changes.

To assist in understanding the calculations presented an example calculation is presented below in relation to mortality (all cause), for the maximum localised change in nitrogen dioxide concentration from the tunnel ventilation structures – realistic scenario in 2036. The air quality modelling provided the change in nitrogen dioxide from all individual receptors (4288 receptors), with the maximum change in annual average for this scenario being 0.24 $\mu\text{g}/\text{m}^3$.

$$\text{Risk} = \beta \times \Delta X \times B$$

ΔX = change in annual average concentration of nitrogen dioxide with the project = 0.24 $\mu\text{g}/\text{m}^3$

B = baseline incidence relevant to all causes of mortality (all ages). Where the maximum risks are being calculated, the highest baseline incidence from all the LGAs has been adopted to ensure the risk calculations are protective of all LGAs = 653 per 100,000 population = 0.00653 per person (refer to **Table 6.4**)

$$\text{Risk} = 0.00188 \times 0.24 \times 0.00653 = 2.9 \times 10^{-6} = 3 \times 10^{-6} \text{ rounded to 1 significant figure}$$

For population risks for each LGA, the baseline incidence relevant to each LGA has been used (as indicated by the colour coding in the attached tables).

In general, Population incidence = Risk x population exposed



In practice, this is calculated based on the populated weighted change in nitrogen dioxide for each ABS mesh block within the modelled domain. This is the average change in annual average nitrogen dioxide concentration for each mesh block x population in each mesh block, with all mesh blocks added up.

$$\text{Population incidence } E = \beta \times B \times \sum_{\text{mesh}} (\Delta X_{\text{mesh}} \times P_{\text{mesh}})$$



Appendix G Health risk calculations: changes in particulate exposures

Health impacts associated with changes in particulate matter have been calculated on the basis of predicted changes in annual average PM_{2.5} (and PM₁₀) concentrations for the conservative emissions scenario for 2026 and 2036 and the realistic emissions scenario for 2036.

Risks and population incidence have been calculated for each individual receptor for the health endpoints and exposure-response functions listed in **Table G1** (which is a repeat of **Table 8.14**).

Table G1: 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					
PM _{2.5} : 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)
PM _{2.5} : 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)
PM _{2.5} : 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					
PM ₁₀ : Mortality, all causes	Short-term	All ages*	1.006 [1.004-1.008]	0.0006 (0.06)	Based on analysis of data from European studies from 33 cities and includes panel studies of symptomatic children (asthmatics, chronic respiratory conditions) (Anderson et al. 2004)
PM _{2.5} : 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)
PM _{2.5} : Cardio-pulmonary mortality	Long-term	≥30yrs	1.14 [1.11-1.17]	0.013 (1.3)	Relationship derived for all follow-up time periods to the year 2000 (for approx. 500,000 participants in the US) with adjustment for seven ecologic (neighbourhood level) covariates (Krewski et al. 2009)
PM _{2.5} : 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)
PM _{2.5} : Asthma (emergency department admissions)	Short-term	1-14 years	--	0.00148 (0.148)	Relationship established from review conducted on Australian children (Sydney) for the period 1997 to 2001 (Jalaludin et al. 2008)
PM _{2.5} : 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



The attached spreadsheets present the calculations undertaken for population/community risks and incidence as well as localised changes.

To assist in understanding the calculations presented an example calculation is presented below in relation to mortality (all cause for ages 30 years and older), for the maximum localised change in PM_{2.5} concentration from the tunnel ventilation structures – realistic scenario in 2036. The air quality modelling provided the change in PM_{2.5} from all individual receptors (4288 receptors), with the maximum change in annual average for this scenario being 0.044 µg/m³.

$$\text{Risk} = \beta \times \Delta X \times B$$

ΔX = change in annual average concentration of PM_{2.5} with the project = 0.044 µg/m³

B = baseline incidence relevant to all causes of mortality (ages 30 years and older). Where the maximum risks are being calculated, the highest baseline incidence from all the LGAs has been adopted to ensure the risk calculations are protective of all LGAs = 1061 per 100,000 population = 0.01061 per person (refer to **Table 6.4**)

$$\text{Risk} = 0.0058 \times 0.044 \times 0.01061 = 2.7 \times 10^{-6} = 3 \times 10^{-6} \text{ rounded to 1 significant figure}$$

For population risks for each LGA, the baseline incidence relevant to each LGA has been used (as indicated by the colour coding in the attached tables).

In general, Population incidence = Risk x population exposed

In practice, this is calculated based on the populated weighted change in PM_{2.5} for each ABS mesh block within the modelled domain. This is the average change in annual average nitrogen dioxide concentration for each mesh block x population in each mesh block, with all mesh blocks added up.

$$\text{Population incidence } E = \beta \times B \times \sum_{\text{mesh}} (\Delta X_{\text{mesh}} \times P_{\text{mesh}})$$



Appendix H Health risk calculations: changes in noise exposures

Health impacts associated with changes in noise have been calculated on the basis of noise level predictions, with and without the project, for the year 2036. The modelling of noise impacts from the operation of the project has provided noise levels and changes in noise associated with the project for all individual receptors evaluated, which is approximately 12,000 individual receptors.

Risks and population incidence has been calculated for each individual receptor for the health endpoints listed in **Table H1**, with consideration of the noise threshold, above which the exposure-response relationship listed in **Table H1** is utilised.

Table H1: Exposure-response relationships for assessment of noise impacts on health

Health effect	Noise measure*	Threshold or range (dB)	Exposure-response relationship (per 10 dB increase in noise [95% confidence interval], unless other relationship presented)	Reference
Ischaemic heart disease: hospitalisations	L _{den}	53	RR = 1.08 [1.01-1.15]	(WHO 2018)
Hypertension (incidence)	L _{den}	47	RR# = 1.07 [1.02-1.12]	(van Kempen & Babisch 2012)
Stroke: hospital admissions	L _{Aeq,16h}	55-60 >60	RR = 1.04 [1.02-1.07] RR = 1.05 [1.02-1.09]	(Halonen et al. 2015)
Mortality: all causes	L _{Aeq,16h}	55-60 >60	RR = 1.03 [1.01-1.05] RR = 1.04 [1.00-1.07]	
Annoyance	L _{den}	42	%HA = 116.4304 – 4.7342 x L _{den} + 0.0497 x L _{den} ²	(Guski et al. 2017)
Sleep disturbance	L _{night}	Threshold for health effects ranges from 32 to 60 WHO (2018) threshold for %HSD = 43	%HSD = 20.8 - 1.05L _{night} + 0.01486(L _{night}) ²	(EEA 2010; WHO 2009, 2011)

RR = relative risk

* Noise measure relates to noise levels measured over different time periods (as noted) outdoors (for all health effects evaluated)

It is not possible to include each individual calculation of risk and health incidence for each individual receptor, so this appendix presents example calculations relevant to one example receptor.

For this example, a residential receptor located within the Banyule local government area (LGA) (suburb of Macleod) has been selected. For this receptor the following data was provided from the noise modelling, for the noise measures relevant to the calculation of health impacts:

Noise level (without project) (L _{Aeq,16hour} dB)	Noise level (with project) (L _{Aeq,16hour} dB)	Change in L _{Aeq,16hour} (dB)	Noise level (without project) (L _{night} dB)	Noise level (with project) (L _{night} dB)	Change in L _{night} (dB)	Noise level (without project) (L _{den} dB)	Noise level (with project) (L _{den} dB)	Change in L _{den} (dB)
56.7	57.2	0.5	51.1	51.6	0.5	53.6	54.1	0.5

Mortality: all causes

The calculation of risk for mortality all causes depends on the noise level as L_{Aeq,16hr}. With the project, this is 57.2 dB, so the calculation utilises the RR = 1.03 as per **Table H1**.

Where RR = 1.03 for a 10 dB change in noise,

$\beta = \ln(RR)/10 = 0.003$ for a 1 dB change in noise

Risk = $\beta \times \Delta X \times B$

ΔX = change in noise with the project = 0.5 dB

B = baseline incidence relevant to all causes of mortality (all ages) for Banyule LGA = 634 per 100,000 population = 0.00634 per person (refer to **Table 6.4**)

Risk = $0.003 \times 0.5 \times 0.00634 = 9.4 \times 10^{-6}$

Population risks are calculated as the average change in risk over all receptors within each LGA.

Population incidence at this individual receptor = Risk x population exposed

Population exposed = 2.6 people (average population per household in Banyule LGA)

Population incidence = 2.4×10^{-5}

Note the population incidence per receptor is not presented, however for all receptors in Banyule the population incidence calculated is the total or sum for every individual receptor within the Banyule LGA.

Hospitalisations for stroke

The calculation of risk for hospitalisations for stroke depends on the noise level as L_{Aeq,16hr}. With the project, this is 57.2 dB, so the calculation utilises the RR = 1.04 as per **Table H1**.

Where RR = 1.04 for a 10 dB change in noise,

$\beta = \ln(RR)/10 = 0.0039$ for a 1 dB change in noise

Risk = $\beta \times \Delta X \times B$

ΔX = change in noise with the project = 0.5 dB



B = baseline incidence relevant to stroke hospitalisations for Banyule LGA = 132 per 100,000 population = 0.00132 per person (refer to **Table 6.4**)

$$\text{Risk} = 0.0039 \times 0.5 \times 0.00132 = 2.6 \times 10^{-6}$$

Population risks are calculated as the average change in risk over all receptors within each LGA.

Population incidence at this individual receptor = Risk x population exposed

Population exposed = 2.6 people (average population per household in Banyule LGA)

$$\text{Population incidence} = 6.7 \times 10^{-6}$$

Note the population incidence per receptor is not presented, however for all receptors in Banyule the population incidence calculated is the total or sum for every individual receptor within the Banyule LGA.

Hospitalisations for IHD

The calculation of risk for hospitalisations for IHD is only undertaken where LDEN > 53 dB. With the project, LDEN = 54.1 dB, so risk is calculated and the calculation utilises the RR = 1.08 as per **Table H1**.

Where RR = 1.08 for a 10 dB change in noise,

$$\beta = \ln(RR)/10 = 0.0077 \text{ for a 1 dB change in noise}$$

$$\text{Risk} = \beta \times \Delta X \times B$$

ΔX = change in noise with the project = 0.5 dB

B = baseline incidence relevant to IHD hospitalisations for Banyule LGA = 537 per 100,000 population = 0.00537 per person (refer to **Table 6.4**)

$$\text{Risk} = 0.0077 \times 0.5 \times 0.00537 = 2.1 \times 10^{-5}$$

Population risks are calculated as the average change in risk over all receptors within each LGA.

Population incidence at this individual receptor = Risk x population exposed

Population exposed = 2.6 people (average population per household in Banyule LGA)

$$\text{Population incidence} = 5.4 \times 10^{-5}$$

Note the population incidence per receptor is not presented, however for all receptors in Banyule the population incidence calculated is the total or sum for every individual receptor within the Banyule LGA.

Incidence of hypertension

The calculation of risk for hospitalisations for hypertension is only undertaken where LDEN > 47 dB and < 77 dB. With the project, LDEN = 54.1 dB, so risk is calculated and the calculation utilises the RR = 1.07 as per **Table H1**.

Where RR = 1.07 for a 10 dB change in noise,

$\beta = \ln(RR)/10 = 0.0068$ for a 1 dB change in noise

Risk = $\beta \times \Delta X \times B$

ΔX = change in noise with the project = 0.5 dB

B = baseline incidence relevant to hypertension for Banyule LGA = 21960 per 100,000 population = 0.2196 per person (refer to **Table 6.4**)

Risk = $0.0068 \times 0.5 \times 0.2196 = 7.5 \times 10^{-4}$

Population incidence at this individual receptor = Risk x population exposed

Population exposed = 2.6 people (average population per household in Banyule LGA)

Population incidence = 1.9×10^{-3}

Note the population incidence per receptor is not presented, however for all receptors in Banyule the population incidence calculated is the total or sum for every individual receptor within the Banyule LGA.

Change in per cent population highly annoyed

The calculation the change in per cent of population highly annoyed by noise is only undertaken where LDEN > 42 dB. With the project, LDEN = 54.1 dB, so calculations are undertaken as per **Table H1**.

Without the project LDEN = 53.6 dB

$$\begin{aligned} \%HA \text{ (no project)} &= 116.4304 - 4.7342 \times L_{den} + 0.0497 \times L_{den}^2 \\ &= 5.5\% \end{aligned}$$

With the project LDEN = 54.1 dB

$$\begin{aligned} \%HA \text{ (with project)} &= 116.4304 - 4.7342 \times L_{den} + 0.0497 \times L_{den}^2 \\ &= 5.8\% \end{aligned}$$

The change in %HA associated with the project = %HA (with project) - %HA (no project) = 0.3%

Population impacts relate to the average change from all receptors within each LGA or in the whole project area.

Change in per cent population highly sleep disturbed

The calculation the change in per cent of population highly sleep disturbed by noise is only undertaken where $L_{night} > 43$ dB. With the project, $L_{night} = 51.6$ dB, so calculations are undertaken as per **Table H1**.

Without the project $L_{night} = 51.1$ dB

$$\begin{aligned}\%HSD \text{ (no project)} &= 20.8 - 1.05L_{night} + 0.01486(L_{night})^2 \\ &= 6.0\%\end{aligned}$$

With the project $L_{night} = 51.6$ dB

$$\begin{aligned}\%HSD \text{ (with project)} &= 20.8 - 1.05L_{night} + 0.01486(L_{night})^2 \\ &= 6.2\%\end{aligned}$$

The change in %HSD associated with the project = %HSD (with project) - %HSD (no project) = 0.2%

Population impacts relate to the average change from all receptors within each LGA or in the whole project area.



Appendix I Risk pathways

Risk ID	Potential threat and effect on the environment	INITIAL RISK							RESIDUAL RISK							Reasoning	
		Initial EPR	Magnitude of consequence			Overall consequence	Likelihood	Risk level	Reasoning	Final EPR	Magnitude of consequence			Overall consequence	Likelihood		Risk level
			Extent	Severity	Duration						Extent	Severity	Duration				
CONSTRUCTION																	
HE01	Noise and vibration emissions from construction activities affecting the health of the local community	EPR NV3 - Minimise construction noise impacts to sensitive receptors EPR NV4 - Implement a Construction Noise and Vibration Management Plan (CNVMP) to manage noise and vibration impacts	Corridor	Medium	3 months to 2 years	Moderate	Possible	Medium		EPR NV3 - Minimise construction noise impacts to sensitive receptors EPR NV4 - Implement a Construction Noise and Vibration Management Plan (CNVMP) to manage noise and vibration impacts	Corridor	Low	3 months to 2 years	Minor	Possible	Low	
HE02	Dust generated and emissions to air from construction equipment and construction activities affecting the health of local community	EPR AQ1 - Implement a Dust and Air Quality Management and Monitoring Plan to minimise air quality impacts during construction EPR SC2 - Implement a Communications and Community Engagement Plan	Local	Medium	3 months to 2 years	Minor	Possible	Low		EPR AQ1 - Implement a Dust and Air Quality Management and Monitoring Plan to minimise air quality impacts during construction EPR SC2 - Implement a Communications and Community Engagement Plan	Local	Medium	3 months to 2 years	Minor	Unlikely	Low	
HE03	Inappropriate handling, stockpiling, transport and treatment/disposal of contaminated soil resulting in exposure and potential health impacts to the local community	EPR CL1 Implement a Spoil Management Plan EPR CL2 Minimise impacts from disturbance of acid sulfate soil	Local	Medium	3 months to 2 years	Minor	Unlikely	Low		EPR CL1 Implement a Spoil Management Plan EPR CL2 Minimise impacts from disturbance of acid sulfate soil	Local	Medium	3 months to 2 years	Minor	Unlikely	Low	
HE05	Changes within the community such as the altered access or connectivity to/between recreational areas, community facilities, commercial premises and active transport infrastructure resulting in potential implications to public safety and wellbeing of individuals during construction	EPR B4 Minimise access and amenity impacts on businesses EPR SC2 - Implement a Communications and Community Engagement Plan EPR T2 - Transport Management Plans	Local	Low	2-7 years	Minor	Likely	Medium		EPR B4 Minimise access and amenity impacts on businesses EPR SC2 - Implement a Communications and Community Engagement Plan	Local	Low	2-7 years	Minor	Possible	Low	
HE06	Changes within the community from the temporary use of some green space areas for construction resulting in impacts on the health and wellbeing of the community and permanent loss of some green space during operation.	EPR SC4 - Minimise impacts on sporting, recreation and other facilities EPR LV2 - Minimise landscape impacts during construction	Local	Low	2-7 years	Minor	Likely	Medium		EPR SC4 - Minimise impacts on sporting, recreation and other facilities EPR LV2 - Minimise landscape impacts during construction	Local	Low	2-7 years	Minor	Possible	Low	
OPERATION																	
HE07	Noise generated by the redistribution of traffic due to operation causes an increase in noise potentially affecting the health and amenity of the local community. In particular these impacts may increase annoyance affecting cognitive function in schools and workplaces, or increase sleep disturbance causing an increase in hypertension and increased risks of cardiovascular morbidity or premature mortality	EPR NV1 - Achieve traffic noise objectives EPR NV2 - Monitor traffic noise	Local	High	7+ years	Major	Possible	High		EPR NV1 - Achieve traffic noise objectives EPR NV2 - Monitor traffic noise	Local	High	7+ years	Major	Unlikely	Medium	
HE08	Tunnel operation leading to higher in car pollutant concentrations and exposures by occupants using the tunnels	EPR AQ2 - Design tunnel ventilation system to meet EPA requirements for air quality	Local	Medium	7+ years	Moderate	Unlikely	Low		EPR AQ2 - Design tunnel ventilation system to meet EPA requirements for air quality	Local	Low	7+ years	Minor	Unlikely	Low	

Risk ID	Potential threat and effect on the environment	Initial EPR	INITIAL RISK					Reasoning	Final EPR	RESIDUAL RISK					Reasoning		
			Magnitude of consequence			Overall consequence	Likelihood			Risk level	Magnitude of consequence			Overall consequence		Likelihood	Risk level
			Extent	Severity	Duration						Extent	Severity	Duration				
HE09	Redistribution of traffic on surface roads during operation changes ambient pollutant levels (particulate matter, nitrogen dioxide, carbon monoxide, volatile organic compounds and polycyclic aromatic hydrocarbons) and emissions from tunnel ventilation structures potentially impacting on the health of the surrounding the community. Health effects may include respiratory and cardiovascular effects morbidity or pre-mature mortality	EPR EMF2 - Deliver project in accordance with an Environmental Strategy and Management Plans EPR EMF3 - Audit and report on environmental compliance EPR AQ2 - Design tunnel ventilation system to meet EPA requirements for air quality EPR AQ4 - Monitor ambient air quality	Wlder region	Medium	7+ years	Major	Possible	High	EPR EMF2 - Deliver project in accordance with an Environmental Strategy and Management Plans EPR EMF3 - Audit and report on environmental compliance EPR AQ2 - Design tunnel ventilation system to meet EPA requirements for air quality EPR AQ4 - Monitor ambient air quality	Wider region	Medium	7+ years	Major	Unlikely	Medium		
HE10	Changes within the community, such as altered access or connectivity to/ between recreational areas and community facilities and active transport infrastructure may have implications to public safety and the wellbeing of individuals from the operation of the project	EPR SC2 - Implement a Communications and Community Engagement Plan EPR T2 - Transport Management Plans	Corridor	Low	7+ years	Moderate	Possible	Medium	EPR SC2 - Implement a Communications and Community Engagement Plan EPR T2 - Transport Management Plans	Corridor	Low	7+ years	Moderate	Unlikely	Low		
HE11	Changes within the community from the permanent loss of green space and tree canopy areas resulting in impacts on the health and wellbeing of the community.	EPR LV2 - Minimise landscape impacts during construction	Corridor	Low	7+ years	Moderate	Planned	Planned	EPR LV2 - Minimise landscape impacts during construction	Corridor	Low	7+ years	Moderate	Planned	Planned		



Appendix J Peer review report

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Peer Review EnRiskS Technical Report

Report reference: GNE/18/HIAR001

Health Impact Assessment of the North East Link project

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14 February 2019

¹ The relevant experience brought to this task by the author includes:

- Fifteen years of leadership of the Australian Centre for Human Health Risk Assessment at Monash University (part time from 2009 to 2018)
- Experience in regulatory toxicology in former leadership appointments to the Commonwealth Department of Health in areas of toxicological assessment of agricultural & veterinary chemicals, regulation of medicines, and assessment of chemicals for poisons scheduling
- More than 45 years experience with government expert committees and panels assessing chemical toxicity and chemicals risk management, including issues of air quality assessment
- Management of the re-writing and updating of seminal Australian guidance on health risk assessment of environmental hazards (enHealth 2012).
- Peer-reviewed recognition as a Fellow of the Australasian College of Toxicology & Risk Assessment (ACTRA), a professional organisation that I helped to found and for which I served as its inaugural President.

The opinions set out in this report are my own, and do not reflect views of any previous employers.

The purpose of this peer review report is primarily to consider the approach taken by Environmental Risk Sciences (EnRiskS) in its Health Impact Assessment (HIA) report relating to the proposed major road development called the North East Link. This proposed development comprises freeways connecting the M80 Ring Road to the Eastern Freeway and includes both above-ground multi-lane highway and tunnel constructions.

The EnRiskS HIA report has been prepared to inform an Environmental Effects Statement (EES) and Public Environment Report (PER) in accordance with the requirements of relevant legislation and Ministerial authority. It addresses potential health impacts in both the construction and operational phases of the project, and is focussed primarily on changes in air quality, noise and vibration during the construction phase, as well as potential impacts on amenity and social functions as a result of traffic diversions. The EnRisk Report is part of a larger suite of technical reports prepared by various consultants that address issues relating to traffic management, air quality, noise and vibration impacts, business and social impacts and other environmental impacts. My peer review report only addresses the HIA aspects of the EES, as contained in the EnRiskS report.

The EnRiskS report includes a summary of the relevant legislation that impacts on such major development projects and includes citations of the various ambient air and noise reduction policy and guidance documents against which the potential health impacts are measured.

General Comments and overall assessment

In summary, I find the EnRiskS report to be a thorough, professional and well-presented review of the potential health risks and benefits associated with the proposed development. It generally follows prescribed Australian and Victorian guidance on how a HIA is to be structured and it includes appropriate illustrations, tabulated data and discussion. Quite correctly, it focusses on potential health impacts on residents and the general community, and notes that issues of occupational health and safety for workers on the project are out of scope and addressed by other regulatory measures.

The layout of the report is quite standard and the Table of Contents allows for easy location of the key elements. I note that the Table of Contents has been modified based on comments made on earlier drafts, with the inclusion of lists of Figures and Tables.

It is noted that the report has been meticulous in identifying places where 'sensitive receptors' may be exposed to NO₂ and particulates from tunnel vents (Table 8.1 and Figure 8.1) and incorporating these sites into the risk assessments. For volatile organic compounds (VOCs) there is an assessment of both acute and chronic health effects.

The report is also meticulous in identifying 'receptors' likely to be impacted by noise and vibration from the construction phases and from altered traffic flows (Section 9 and Appendix H). Figures 9.1_{a-e}, and 9.2 provide a useful graphic illustration of these issues.

The amount of data presented in the report is very extensive, and I will not attempt to comment on all of it. Nor have I attempted to verify calculations based on the equations outlined in Appendix B, other than to check a few of the more critical ones. This disclaimer is consistent with those claimed by EnRiskS in Section 14, indicating

reliance on, but no responsibility for, the validity of the data used. Confidence in the risk calculations is enhanced by the detailed descriptions in Appendices F, G and H, on how these risks have been quantitatively assessed.

The overall outcomes of the risk assessments are summarised at the end of each section and in Table 12.1 in relation to air quality, vibration, noise and socio-economic impacts. There is also a useful summary (Appendix I) of the initial and residual risks associated with defined risk pathways for both the construction and operational phases of the project. The conclusions drawn are measured and reasonable and I can find no reason to challenge any of them.

It appears that, while health effects associated with changes in air quality are generally in the 'acceptable' range, impacts associated with noise, vibration and social dislocations may exceed these benchmarks at specific locations around the project if not specifically addressed (Sections 9.5.3 and 9.6). The EnRiskS report notes that risk management of the localised noise and vibration impacts has been covered in EES Technical Report C *Surface Works noise and vibration impact assessment*. I am satisfied that these remedial works should mitigate the identified localised risks and that application of relevant Environmental Performance Requirements (EPR NV1, 2, 7 and 13) will result in compliance with the risk management targets.

Comments on selected elements of the EnRiskS Report

1. Acceptable risk levels

In any health risk assessment, the benchmark against which the risk estimates are compared is a crucial element. In some cases, where a threshold is deemed to exist, the benchmark is generally conservatively set as a Tolerable Daily Intake (TDI), where exposures less than the TDI are assumed to have no appreciable health impacts over a lifetime of exposure. In other cases, where the exposure-response relationships do not exhibit a clear threshold (e.g. for carcinogens and some air pollutants), the exposure-response relationship is extrapolated down to a defined level of excess risk (e.g. 1 in 10,000 or 10^{-4} to 1 in 1,000,000 or 10^{-6}). This defined risk level is termed 'acceptable' in the EnRiskS HIA report, although it is sometimes referred to as a 'target risk level' in some cases to avoid argument over to whom the risk level meets 'acceptable' criteria (see also discussion in Section 5.10 enHealth 2012)

The approach taken for airborne pollutants in the EnRiskS HIA report is explained in some detail in Appendices B, C and D. This discussion clearly sets out the complex methodologies used to estimate additional or incremental risks and to calculate the likely number of additional cases. There is discussion of when an annual average exposure is used rather than summation of individual day exposure, as recommended by WHO guidance, but it is noted that daily exposure calculations have been used for short-term infrequent incidents, such as a breakdown in the tunnel. I am satisfied that these are the most appropriate approaches for this HIA.

There is a further detailed discussion in Appendix D of where to set the 'acceptable' level of risk, including some thoughtful probing of the origins of the 10^{-6} risk level as applied to carcinogens, and to comparisons of this risk level with some other everyday risks. Ultimately, the EnRiskS HIA report settles on a 10^{-4} to 10^{-6} range of risks for the prescription of various risk management actions. These levels are further justified in Section D4, with comparisons made with other projects comparable to the North East Link. The caveat is noted that the adopted 'acceptable' risk level for

carcinogens (10^{-6}) and NO_2 (10^{-5}) are “*specific to this project ...and should not be considered to be policy for the assessment of all projects in Victoria*”. I note that even using the more conservative risk level (10^{-6} , rather than the 10^{-5} endorsed in enHealth 2012 and justified in the discussion in Appendix D), the estimated risks for exposure to the four carcinogens assessed (benzene, 1,3-butadiene, PAHs and diesel particulates) is lower than this benchmark.

There is also discussion (in Sections 8.1 and 9.1 and Appendix D) of the need to differentiate population risk estimates from those of localised impacts, such as redistribution of traffic. While it is acknowledged that there is no specific guidance on how to manage these different risk scenarios in relation to air pollutants, and that maximum localised risk is ‘*only semi-quantitative*’, increasing the risk management level from 10^{-5} to 10^{-4} for localised risk has been mainly justified on the basis that fewer people are likely to be impacted, compared to a ‘population risk’. While this argument seems to be driven more by comparison with ‘tolerable risks’ adopted for other comparable construction projects, I find the approach adopted to be acceptable.

In the case of noise annoyance vibration and social dislocation, where the risk assessments are based on more nebulous endpoints, the risk estimates have been compared in some cases to some statutory guidance. In the case of health data for cardiovascular disease, morbidity/mortality and sleep deprivation, where the quantitative exposure-response relationships are more robust, the ‘acceptable’ risk levels (10^{-4} localised to 10^{-5} project-wide) are based on the change of relative risk (RR) per 10dB increase in noise. I also find these approaches to the risk assessments to be acceptable.

A further difficulty is the factoring in of background levels of noise and determining the different noise impacts likely during construction and operational phases of the project. The approach adopted in the EnRiskS HIA report is acceptable, with the calculations of noise impacts outlined in more detail in Appendix H.

2. Data inputs

Data on exposures to airborne particulates and vehicle exhaust fumes is largely drawn from reports and modelling prepared by others. However, the exposure assessments include various assumptions that are crucial to the EnRiskS HIA, such as the mix and magnitude of traffic volumes, tunnel emissions, locations of sensitive receptors, baseline health and amenity conditions.

I am satisfied that these inputs have been chosen using appropriately conservative assumptions.

3. Treatment of elements of uncertainty

The issue of how the HIA deals with uncertainties in the reviewed data and information is explained in some detail in Section 11. This section sets out clearly the ways in which these uncertainties have been managed and stresses the conservatism that is normally built into the models and paradigms that inform the health risk estimates. I found that the Forest plots in Figures 11.1 to 11.3 to be a particularly useful visual depiction of the extent to which variability between different studies affects the interpretation of exposure-response relationships for the three major health impacts associated with respirable dusts ($\text{PM}_{2.5}$). Likewise, the summarised data in Figures 6.6 to 6.9 and Tables 6.4 and 6.5 provides useful

baseline information on the variability in acute and chronic health statistics across different Local Government Areas (LGA) compared to a State-wide comparison.

There is a useful discussion (Sections 11.8 and 11.10) on how population size and demographics can impact on the use of broader baseline health-based statistics against which risks can be measured, as well a pragmatic approach to how a possibly higher incidence of asthma in localised suburbs around the proposed development could be factored into the risk assessment processes (section 11.9). Doubling of the LGA baseline for asthma incidence showed that the incremental level of risk associated with the project is still within the prescribed risk levels of 10^{-4} to 10^{-5} .

Table 8.11 is also an important data set. It demonstrates the range of individual risk estimates from maximum NO₂ emissions at various points along the proposed road and tunnel development. These data suggest that, at some points, air quality may actually be improved as a result of changed traffic flows. These calculations are also made more informative by presenting data derived from both conservative and more realistic emission estimates².

4. Selection of emission standards

Section 8, and in particular Table 8.12, sets out the 24h and annual average air quality goals (NEPM/EPA Victoria, WHO, EU and US EPA) against which the health risks of airborne particulates (PM₁₀ and PM_{2.5}) have been assessed. In the case of PM_{2.5} goals, the more stringent 2025 goals have been more generally applied. I believe this is an appropriate approach. Table 8.13 summarising the cumulative background and background + project incremental exposures from tunnel vents and surface traffic. It is notable that, while the project-attributable incremental risk is small, it is overlaid on more substantial background levels associated with existing traffic. In the case of PM_{2.5}, these background emissions meet neither the current NEPM guidelines nor the 2025 air quality goals.

² In order to understand how the different emission estimates are derived, it is necessary to refer back to Section 8.4.5, where the differences in fleet volumes and emission standards are discussed in more detail

CURRICULUM VITAE

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Current appointments: Adjunct Professor, Department of Epidemiology & Preventive Medicine, School of Public Health & Preventive Medicine, Monash University, Vic 3004

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Ph: (08) 8431 7042 (home landline)
Mobile: 0413 607 285
Email: brian.priestly@monash.edu or brianpriestly8@gmail.com

DEGREES AND DIPLOMAS:

1963 B. Pharm – Sydney University.

1965 M.Pharm – Sydney University

1968 PhD – Sydney University

PAST APPOINTMENTS:

1964-67 Teaching Fellow (Demonstrator) Pharmacy School, University of Sydney

1968 Research Associate, Department of Pharmacology, University of Iowa

1968-70 Canadian MRC Postdoctoral Fellow, Department of Pharmacology, University of Montreal

1970-80 Lecturer/Sen. Lecturer, Dept. of Human Physiology and Pharmacology, University of Adelaide

1975-76 Professeur invite, Department of Pharmacology, University of Montreal,

1980 -92 Senior Lecturer, Dept. of Clinical and Experimental Pharmacology, University of Adelaide

1992-95 Senior Principal Research Scientist, (SPRS), & Scientific Director, Chemicals Safety Unit, Commonwealth Dept of Community Services & Health

1995-96 Scientific Director, (SPRS) Environmental Health & Safety Unit, Commonwealth Dept. Human Services & Health

1996-2001 Scientific Director, (SPRS) Chemicals & Non-Prescription Medicines Branch, Therapeutic Goods Administration, Commonwealth Dept. Health & Aged Care

2001-03 Director (SPRS), Laboratories Branch, Therapeutic Goods Administration (TGA), Commonwealth Dept. of Health & Ageing

2011-16 SPRS, Office of Chemical Safety (OCS), Department of Health (DoH), limited term, casual.

2004-18. Director, Australian Centre for Human Health Risk Assessment (ACHHRA), School of Public Health & Preventive Medicine, Monash University

CAREER RESEARCH AND OTHER PROFESSIONAL INTERESTS:

- Pre-1992 Mechanisms of chemically-induced cytotoxicity, with particular interest in hepatotoxic chemicals and the use of isolated hepatocytes/cell cultures to determine drug metabolism and toxicological mechanisms. Metabolic activation/inactivation and genotoxicity of carcinogenic chemicals with applications to biological monitoring of occupational chemical exposure.
- 1992-2001: Risk assessment and regulatory control of hazardous chemicals, including: management of toxicological and public health assessments for agricultural, veterinary and industrial chemicals for the relevant Australian regulatory authorities; responsibility for policy development for national and international chemicals regulatory activities; advice on regulation of OTC and complementary medicines; community liaison on hazardous chemicals
- 2001- 03 As Director of the TGA Laboratories: management of the delivery of scientific and laboratory testing services in support of the regulation of therapeutic goods in Australia (medicines, medical devices); budget management responsibilities \$14.2m; policy formulation on therapeutic goods regulation; liaison with stakeholders (industry, governments and the community) on laboratory testing of therapeutic goods.
- 2004-18 As Director of the Australian Centre for Human Health Risk Assessment (ACHHRA), to establish ACHHRA as a leading Australian centre of excellence in health risk assessment; to co-ordinate workforce development and training programs in health risk assessment; to maintain appropriate linkages with government public & environmental health and regulatory programs; to promote Australian contributions to national and international chemicals risk assessment and risk management programs.
- 2011-16 Limited-term part-time appointment to act as the Delegate of the Secretary, Department of Health, to make decisions relating to the scheduling of chemicals in the *Standard for Uniform Scheduling of Medicines & Poisons*.

MEMBERSHIP OF EXTRAMURAL COMMITTEES:

1. **Editorial Boards:**
Toxicology (1977-78); Journal of Environmental Toxicology (guest editor; 2006 special issue on Australian environmental toxicology)
2. **National Health and Medical Research Council:**
Committee on Toxicity (1985-92); Drugs and Poisons Schedule Committee (1980-85)
Food Science & Technology Comm. (1982-91); Consumer Products Safety Committee (1979-82)
WP on Heavy Metal Contamination of seafoods (1973)
WP on the use and safety of 2,4,5-T (1978-80); WP on Fluoridation (1979 & 1989-90)
WP on urea-formaldehyde foam insulation (1982); WP on dichlorvos (1984-86)
WP on plasticisers (1987); WP on cancer risk assessment (1995-96)
Special Expert Committee on Transmissible Spongiform Encephalopathies (Observer; 2001-03)
Working Group on Toxicity & Risk Assessment (2003-04)
Expert panel; health-based methodology for setting ambient air quality standards (2006)
Round Table conference on nanotechnology (2006)
Advisory Committee on Health & Nanotechnology (Chair; 2007-08)
Disinfection By-Products Advisory Committee (2016-18)
Working Group on environmental health standard setting (2016)
PFAS Targeted Call for Research (TCR) Reference Group (2018-)
3. **Commonwealth Department of Health:**
Australian Drug Evaluation Committee (Assoc member; 1992; 2007-09)
Advisory Committee on Prescription Medicines (Assoc member; 2010-12)
Advisory Committee on Pesticides & Health (Chair; 1996-2001)
Chemicals Clearing House (Inter-Departmental Committee) (Chair 1996-2001)
IDC on Health Monitoring program (Chair; 2000-01);
National Drugs & Poisons Schedule Committee (1997-2003);
TGA-Industry Consultative Committee (2001-03); TGA Expert Advisory Group on Vit E (2006)
TGA/Medsafe Interim Joint Expert Advisory Committee on Complementary Medicines (2006)
FSANZ Panels: Dairy Scientific Advisory Panel (2005); Iodine Scientific Advisory Group (2006)
phytosterols SAG (2006); health claims SAG (2006); safety assessment GM foods (2007)
Scientific Nanotechnology Advisory Group (2016-); New Breeding techniques (2017-)
Appointed FSANZ Science Fellow (2007-)
Office of Chemical Safety, Advisory Group on Chemical Safety (2006-09)
Gene Technology Technical Advisory Committee (2009-13)
enHealth workshop to review HILs proposed for the Contaminated Sites NEPM (2010)
enHealth workshops on perfluoralkyl chemicals (2015-16)

4. **Other Government Committees & Working Parties:**
 Forest Herbicides Management Working Group; 1995-1999
 National Profile on Chemicals Management Infrastructure in Australia, team member 1997-98
 National Pollutants Inventory Technical Advisory Panel, 1997-98
 WP on hazardous chemicals, Trans-Tasman Mutual Recognition Arrangements, 1999-2002
 Workshop participant ; Draft framework for Environmental Health Risk Assessment, 1999-2000
 Working Party on the Health Impacts of Pesticide Odours 2000-01
 EPHC Working Party to develop a risk management framework for recycled water (2004-08)
 NSW Food Authority; Expert panel on dioxins in seafood (Chair; 2005-06)
 Vic EPA; Expert panel on fish contamination (Chair; 2006-07); Port Phillip Bay fish issues (2009)
 Standards Australia; Committee NT-001 nanotechnologies (2006-09)
 Queensland Water Commission; Expert Panel (to advise on water recycling) (2007-13)
 NSW Health Expert Panel on health risk assessment (2007-)
 APVMA Science Fellow (2007-); advice on nanotechnology regulation (2009-)
 WP to advise Vic DHS on management of algal blooms (2008-11)
 NICNAS Nanotechnology Advisory Group (2008-); NICNAS Human Health Expert WG (2009-10)
 AICIS Evaluations Roadmap WG (2018-) CASA Expert Panel on Aircraft Air Quality (2009-10)
 DCCEE Peer Review Panel for a project on introduction of Compact Fluorescent Lights (2010-11)
 Consultant to AG/AFP on chemicals risk assessment (2010-11)
 Panel advising Dept Industry, Innovation etc; community consultation on nanotechnology (2013)
 Victorian Dept. Health; Expert Advisory Group for Potential Cancer Cluster Investigations (2016-)
 NSW Chief Scientist Office; Lord Howe Island rodent eradication HHRA Expert Panel (2016-17)
 NT Government Expert Panel on Hydraulic Fracturing ('Fracking') (2016-18)
 NICNAS Evaluation Roadmap WG Facilitator (2018)
5. **SA Government (1979-1992; 2014-):**
 Food and Drug Advisory Committee 1979 - 1992.
 Controlled Substances Advisory Council 1985-92; Working Party on Termiticides 1988-89.
 Animal Welfare Advisory Committee 1988-1992; Scientific Data Review Working Party
 (Chairman) 1980-1985 ; Working Party to review the Level of Concern for Lead 1986
 Ministerial Working Party on Toxicity of Aldrin, 1987; Ministerial Scientific & Advisory Committee
 on Aldrin Contamination, Streaky Bay Area School 1987-1989
 Advisory Panel on TCE contamination at Clovelly/Mitchell Park (2014-15)
6. **University & Hospital Committees (1971-92) (2004-):**
 Adelaide University Animal Ethics Committee (1990-92; medical sub-group 1980-92;
 agricultural & natural sciences sub-group 1990-92)
 Adelaide University Biosafety Committee (1985-92)
 Faculty of Science (Dept representative) 1971-92)
 Anti-Cancer Foundation of the Universities of South Australia Executive Board (1980-91)
 Queen Elizabeth Hospital Ethics of Research Committee (1983-89)
 Aust Centre of Excellence for Risk Assessment (Uni Melb) Scientific Advisory Comm (2007-10)
 National Research Centre for Environmental Toxicology - Research Advisory Comm (1992-2006)
 National Research Centre for Environmental Toxicology (EnTox) – Management Board (2007-11)
 Scientific Reference Group, Monash University; Hazlewood Health Study (2015-17)
7. **International:**
 International Agency for Research on Cancer (IARC) (Member, Monograph Working Party on
 Pesticides, 1990)
 Joint FAO/WHO Expert Committee on Food Additives (JECFA) (Temporary Advisor 39th.
 JECFA Meeting, 1992. Member, Expert Panel on Food Safety 1993-12
 Joint FAO/WHO Meeting on Pesticide Residues (JMPR) (Member 1997-2001)
 International Programme on Chemical Safety (IPCS)
 INTOX-VI 1993; Harmonisation of Risk Assessment 1993; Workshop/Planning
 meeting on Biomarkers in Risk Assessment 1993; IPCS/OECD Consultation on
 Priority Chemicals & Related Issues 1995; Internationalization of National & Regional
 Criteria Documents (Chair) 1995; Consultation to Review the Terms of Reference of
 the Programme Advisory Committee, 1995; Member, Programme Advisory
 Committee (PAC) 1996-2001; PAC Standing Committee 1998-2001 ; Cancer Risk
 Assessment Workshop 1998; Meeting on Peer Review Processes for Risk
 Assessment 1998
 Codex Committee on Veterinary Drug Residues (Member, Australian delegation, 1992)
 Intergovernmental Forum on Chemical Safety (IFCS)
 2nd Interessional Meeting, Canberra 1996, member of Australian delegation
 2nd Session IFCS, Ottawa 1997, Deputy Head of Australian delegation
 ILSI/HESI Agricultural Chemical Safety Steering Group (2002)
 Official Medicines Control Laboratories network (2002)
 International Laboratory Forum on Counterfeit Medicines (2002-03)
 FAO core group planning a consultation on nanotechnology in food & agriculture (2008)
 WHO Expert Panel assisting Philippines Dept Health – aerial spraying of pesticides (2009)

WHO Consultation on DDT risk assessment (2010)
WHO 2nd Meeting on Global Collaboration in Risk Assessment (2012)

8. **Miscellaneous**

International Advisory Board, Xth International Congress of Pharmacology, Member 1986-1987.
International Union of Toxicology (IUTOX), Director 1992-1995
Australasian Society of Clinical and Experimental Pharmacologists & Toxicologists (ASCEPT).
Member 1971-2001; Served on Council, 1982-1985; 1992; 1999-2000;
Toxicology Committee, Convenor 1982-1983; Secretary 1990-94;
Member of the Executive Committee of the International Congress of Toxicology IX
Organising Committee, and Chair of the Programme Committee, 1995-2000.
Aust College Toxicology & Risk Assessment (ACTRA) Comm convenor 2006-7; President 2008-11
Program Advisory Committee; CRC-CARE project on HSLs for petroleum hydrocarbons (2007-11)
Program Advisory Committee; CRC-CARE project on emerging soil contaminants (2014-)
CRC-CARE Australian Remediation Industry Cluster (ARIC) Steering Committee (2008-12)
Independent Monitoring Committee; Botany industrial site remediation project (2006-14)
Independent Expert Panel: Botany industrial site carpark remediation project (2010-14)
CRC-CARE Technical Working Group on PFOS/PFOA (2015-)
Advisor to Airservices Australia & Dept of Defence on PFOS contamination issues (2010-11; 2015-)
NSW Health; invited speaker at training workshop on health risk assessment (2016-17)
Evaluation consultant; Adelaide University School of Public Health project to review SWA Workplace
Exposure Standards (2018)

HONOURS AND PROFESSIONAL RECOGNITION

Included as a Fellow on the peer-reviewed register of the Australasian College of Toxicology & Risk Assessment (FACTRA) 2014-; ACTRA Achievement Award 2016

MEMBERSHIP OF SOCIETIES:

1. Australasian Society of Clinical and Experimental Pharmacologists and Toxicologists (1971-2001)
2. Australasian College of Toxicology & Risk Assessment (ACTRA) 2006-
3. Society for Risk Analysis 2012-13
4. Australian Land & Groundwater Association (ALGA) 2016-

GRADUATE STUDENT SUPERVISION BY BRIAN PRIESTLY

1970-92 11 PhD; 2 M.Env.studies; 17 BSc (Hons)

2004- 1 PhD

PhD Theses examined: 8 (since 2012)

TEACHING ACTIVITIES

Monash University

- MPH2045 *Principles of health risk assessment* (course convenor and main lecturer); 2004-10
- MPH2075 *Health risk management and communication* (course convenor and main lecturer); 2005-07
- MPH2069 *Health systems policy*; invited lecture on risk perception 2006
- MPH1003/5203 *Environmental influences on health* (lecturer 2004-18; convenor 2007)
- MPH2041 *Introduction to occupational health & safety* (invited lecture on nanotechnology OHS aspects, 2008)
- HSC3102 *Environmental determinants of health and disease* (invited lecture 2012)
- MPH5243 *Practical toxicology* (invited lecture, 2014)
- AFOEM Short course on Environmental Medicine (2016-17)

Flinders University

- National short course in environmental health; invited lecturer 2003-04; 2010-16

Adelaide University

- Public Health Sciences module on environmental health sciences; invited lecture *Principles of toxicology*; 2012

Queensland University

- National Research Centre for Environmental Toxicology (EnTox); Short course on environmental health risk assessment; invited lecturer 2005, 2008

Sydney University

- Short course in public health toxicology, invited lecturer 2005

ALGA

- Online course in basic toxicology; invited lecture on advanced toxicology and mixtures assessment

Conferences and Symposia – invited speaker and topics (past 8 years)

2012

- Australian Faculty of Occupational & Environmental Medicine; Annual Scientific Conference, Brisbane 8 May; *Health impact assessment (HIA) and health risk assessment (HRA) – essential differences and processes*
- Flinders University School of the Environment seminar series; Adelaide, 6 June; *Water quality standards and guidance for the protection of human health and the environment: How they are established*
- APVMA training session; Canberra 26 June; *Dose response modelling (DRM) and benchmark dose (BMD) assessment – the way of the future?*
- Monash University Department of Chemical Engineers, Platinum seminar series, Melbourne 3 Sept; *Health & safety issues in the nanotechnologies – if we are at the crossroads, which direction to take?*
- 1st Malaysian Congress on Toxicology, Kuala Lumpur, 2-3 Oct; *Environmental Health risk assessment: The Australian experience*
- International Workshop on the risk assessment of manufactured nanomaterials; Adelaide 8-9 Oct; *Nanomaterials regulation in Australia*

2013

- ACCORD Cleaning & Hygiene sector conference, Melbourne, 7 Nov; *The Precautionary Principle: Use and misuse in chemicals risk assessment.*

2014

- Australian Pesticides & Veterinary Medicines Authority (APVMA); Canberra 28 October; Panellist in a symposium on nanotechnology regulation
- Malaysian Society of Toxicology Workshop on oil spills; 10-11 Nov; (2 talks); *Oil spill effects on human health; Is there a cancer risk associated with oil spills?*

2015

- Australian Water Recycling Centre of Excellence (AWRCE); Workshop on linking bio-analytical tools to human health issues related to water. Leura 9-11 February; *BAT comparisons with conventional HHRA tools & workshop panellist*
- Centre for Air quality & health Research (CAR); air pollution workshop; Melbourne 2 June; (2 talks); *Understanding health-based air quality guidelines; Clean-and Healthy-Air-for-Gladstone(CHAG) case study.*
- Society for Risk Analysis; World Congress on Risk, Singapore 20-22 July; *Australian guidance on EHRA; including guidance on community engagement.*
- Regulatory Sciences network; Canberra Nov 2015; Keynote address; *Regulating in the face of uncertainty; dealing with data gaps and chasms.*
- enHealth summit on PFCs; Sydney Dec 2015; *Potential health impacts from exposures to PFOS and PFOA.*

2016

- NSW Health Public Health training workshop; Sydney 16 Feb; *Explaining risks to the community; what is acceptable and what is not.*
- 2nd Shanghai Summit on cosmetics regulation in the Asia-Pacific; Shanghai 27-28 June; *Australian cosmetics and cosmetic ingredient management.*
- Ecoforum 2016; Fremantle 26-27 Oct; Keynote address: *Does a formal Human Health Risk Assessment (HHRA) really assist with the management of contaminated sites?*

2017

- NSW Health Public Health training workshop; Sydney 13 Feb; *Some issues in risk assessment, risk management & stakeholder engagement; Development of a checklist for EHOs for HHRAs.*
- Aust. Contaminated Land Consultants Assoc. Melbourne 30 May; *PFAS - emerging environmental contaminants;*
- ACTRA workshop on chemical sensitization; Melbourne 2 June; *Toxicology of skin & respiratory sensitization – what do the testing data tell us?*
- 5th Asia Safety Experts Workshop. Singapore 6 June; Test strategies for genotoxicity (workshop panellist).
- RACI Victoria. Melbourne 15 August *Nanotechnology: Risk assessment, risk perception and risk management of nanoscale materials.*
- ACTRA/EPA workshop on education & training opportunities; Melbourne 10 November; *Challenges and issues in the academic sector.*
- Sydney University; 12 December; *Safety assessment and regulation of nanoscale materials.*

2018

- Victoria EPA; Melbourne 15 June; *PFAS - emerging environmental contaminants*

2019

- ASEAN Product Safety Forum 2019; Singapore 27-28 Feb; *Labelling and post-market regulatory aspects of labelling in Australia.*

Conferences & Symposia – submitted papers and topics (past 8 years)

2011

- Society of Toxicology (SOT); 50th Annual Congress; Washington 6-10 March; *Registration of specialists in toxicology and risk assessment in Australasia* (poster)
- ACTRA workshop; The use of epidemiology studies in regulatory risk assessment; Canberra 10 Aug; *Integration of epidemiology and toxicology in risk assessment – case study with DDT*
- ACTRA & CRC-CARE CE session in conjunction with CleanUp11 conference; Adelaide 11 Sept; *Environmental health risk assessment – update to enHealth guidance*
- ACTRA Annual Scientific Meeting; Melbourne 27-28 Oct; ACTRA Debate – speaker FOR the motion *That regulatory toxicology in Australia has not engaged ‘drive’ in order to move forward, but it remains stuck in ‘neutral’*

2012

- ACTRA Annual Scientific Meeting; Adelaide, 24-25 Oct; *Exposure and the AEF – where next in relation to lifestage-specific risk assessment*

2013

- ACTRA workshop; Life stage-specific health risk assessment; Sydney, 21 March; *Australian and US EPA exposure factors handbooks: Similarities and key differences*
- ACTRA & CRC-CARE CE session in conjunction with CleanUp13 conference; Melbourne 15 Sept; *enHealth guidance on exposure assessment*
- ACTRA Annual Scientific Meeting; Canberra 17-18 Oct; *Precautionary Principle; use and misuse in environmental risk assessment.*

2015

- ACTRA & CRC-CARE CE session in conjunction with CleanUp2015 conference; Melbourne 13 Sept; *enHealth and NEPM guidance on mixtures toxicology assessment*
- ACTRA Annual Scientific Meeting; Brisbane 14-16 Oct; *HD_M^I – an alternative to the ADI/TDI encompassing variability?*

2016

- ACTRA Workshop on regulatory toxicology; Canberra 22 July; *Glyphosate carcinogenesis – how did IARC and EFSA reach different conclusions?*
- ACTRA CE session on HHRA; Adelaide 21 Sept; *Mechanistic data framework for evaluating carcinogens;*
- ACTRA Annual Scientific Meeting; Adelaide 22-23 Sept; *Systemic effects generally drive toxicity classifications - but what weight should be given to localised topical effects (skin/eye irritancy & sensitisation)?*

2017

- ACTRA Annual Scientific Meeting; Canberra 27-28 Sept; *Fit-for-purpose TDIs; Matching short-term exposures to TDIs based on lifetime exposure.*

PUBLICATIONS:

- 1 PRIESTLY BG & O'REILLY WJ (1966). Protein binding and the excretion of some azo dyes in rat bile. *J. Pharm. Pharmacol.* **18**, 41-45.
- 2 PRIESTLY BG & PLAA GL. (1969). Effects of benzydaron on the metabolism and biliary excretion of sulfobromophthalein and related dyes. *Proc. Soc. Exp. Biol. Med.*, **132**, 881-885.
- 3 PRIESTLY BG & PLAA GL. (1970). Sulfobromophthalein metabolism and excretion in rats with iodomethane-induced depletion of hepatic glutathione. *J. Pharmacol. Exp. Ther.*, **174**, 221-231.
- 4 PRIESTLY BG & PLAA GL. (1970). Reduced bile flow after sulfobromophthalein administration in the rat. *Proc. Soc. Exp. Biol. Med.*, **135**, 373-376.
- 5 PRIESTLY BG & PLAA GL. (1970). Temporal aspects of carbon tetrachloride-induced alteration of sulfobromophthalein excretion and metabolism. *Toxicol. Appl. Pharmacol.*, **17**, 786-794.
- 6 PRIESTLY BG, COTE MG & PLAA GL. (1971). Biochemical and morphological parameters of tauroolithocholate-induced cholestasis. *Canad. J. Physiol. Pharmacol.*, **49**, 1078-1091.
- 7 O'REILLY WJ, COATES PE, MCMAHON KA, TING E & PRIESTLY BG. (1972). Pharmacokinetic models for the biliary excretion of amaranth in the rat. *Life Sci.*, **11**, 197-208.
- 8 WILLIAMS DE & PRIESTLY BG (1973). Evaluation of prophylactic efficacy of silymarin in CCl₄-induced hepatotoxicity. *Res. Comm. Chem. Path. Pharmacol.*, **6**, 185-194.
- 9 DREW R & PRIESTLY BG. (1976). Microsomal drug metabolism during α -naphthylisothiocyanate-induced cholestasis. *Toxicol. Appl. Pharmacol.* **35**, 491-499.
- 10 DREW R & PRIESTLY BG. (1976). Hexobarbital sleeping time and drug metabolism in rats with ligated bile ducts - a lack of correlation. *Biochem. Pharmacol.*, **25**, 1659-1663.
- 11 HANASONO GK, DE REPENTIGNY L, PRIESTLY BG & PLAA GL. (1976). The effects of oral diazepam pretreatment on the biliary excretion of sulfobromophthalein in rats. *Canad. J. Physiol. Pharmacol.*, **54**, 603-612.
- 12 STACEY NH & PRIESTLY BG (1976). The bicarbonate ion concentration of Krebs-Henseleit solution. *Aust. J. Exp. Biol. Med. Sci.*, **54**, 103-105.
- 13 PRIESTLY BG & PLAA GL. (1976). Hepatic function after acute or subchronic nicotine administration in untreated mice and mice treated with hepatotoxic chemicals. *Arch. Int. Pharmacodyn. Ther.*, **223**, 132-141.
- 14 PLAA GL & PRIESTLY BG. (1977). Intrahepatic cholestasis induced by drugs and chemicals. *Pharmacol. Rev.*, **28**, 207-273.
- 15 DREW R, PRIESTLY BG & O'REILLY WJ. (1977). Hexobarbital pharmacokinetics in rats after ligation of the common bile duct. *J. Pharmacol. Exp. Ther.*, **201**, 534-540.
- 16 HARMAN AW, PENHALL RK, PRIESTLY BG, FREWIN DB, PHILLIPS PJ & CLARKSON A. (1977). Salivary antipyrine kinetics in hepatic and renal disease and in patients on anticonvulsant therapy. *Aust. N.Z. J. Med.*, **7**, 385-390.
- 17 HARMAN AW, PRIESTLY BG & FREWIN DB. (1977). A comparative study of antipyrine pharmacokinetics in saliva and plasma using a colourimetric method of antipyrine analysis. *Clin. Exp. Physiol. Pharmacol.*, **4**, 593-596.
- 18 STACEY NH, PRIESTLY BG, HALL RC. (1978). Toxicity of halogenated volatile anesthetics in isolated rat hepatocytes. *Anesthesiology*, **48**, 17-22.
- 19 STACEY NH, COOK R & PRIESTLY BG. (1978). Comparative stability of alanine aminotransferase in rat plasma and hepatocyte suspensions. *Aust. J. Exp. Biol. Med. Sci.*, **56**, 379-381.
- 20 HARMAN AW, RUSSELL WJ, FREWIN DB & PRIESTLY BG. (1978). Altered drug metabolism in anaesthetists exposed to volatile anaesthetic agents. *Anaesth. Inten. Care*, **6**, 210-214.
- 21 LAVIGNE J, PRIESTLY BG & PLAA GL. (1978). Effects of a cholestatic agent on isolated rat hepatocyte function. *Toxicol. Appl. Pharmacol.*, **45**, 263.
- 22 DREW R & PRIESTLY BG. (1978). Failure of hypoactive hypertrophic smooth endoplasmic reticulum to produce cholestasis in rats. *Toxicol. Appl. Pharmacol.*, **45**, 191-199.

- 23 STACEY NH & PRIESTLY BG. (1978). Lipid peroxidation in isolated rat hepatocytes: relationship to toxicity of CCl₄, ADP/Fe³⁺, and diethyl maleate. *Toxicol. Appl. Pharmacol.*, **45**, 41-48.
- 24 STACEY NH & PRIESTLY BG. (1978). Dose-dependent toxicity of CCl₄ in isolated rat hepatocytes and the effects of hepatoprotective treatments. *Toxicol. Appl. Pharmacol.*, **45**, 29-39.
- 25 HARMAN AW, FREWIN DB, PRIESTLY BG & ALEXANDER CBJ. (1979). Impairment of hepatic metabolism in alcoholics. *Brit. J. Clin. Pharmacol.*, **7**, 45-48.
- 26 DREW R & PRIESTLY BG. (1979). Choleretic and cholestatic effects of infused bile salts in the rat. *Experientia*, **35**, 809-810.
- 27 DREW R & PRIESTLY BG. (1979). Effect of chlorpromazine and erythromycin on bile salt-induced cholestasis in the rat. *Pharmacology*, **18**, 202-209.
- 28 PENHALL RK, HARMAN AW, FREWIN DB & PRIESTLY BG. (1979). A comparison of the efficacy of atenolol given once a day with either propranolol or pindolol in the management of hypertension. *Current Therapeutics*, **20** Suppl, 4-10.
- 29 HARMAN AW, FREWIN DB & PRIESTLY BG. (1980). Comparative enzyme-inducing effects of chlorpromazine and fluphenazine therapies in psychotic patients. *Psychopharmacol.*, **69**, 35-37.
- 30 HARMAN AW, FREWIN DB & PRIESTLY BG. (1981). Induction of microsomal drug metabolism in man and in the rat by exposure to petroleum. *Brit. J. Indust. Med.*, **38**, 91-97.
- 31 PRIESTLY BG. (1984). Interpretation of drug mutagenicity tests (commentary). *Med. J. Aust.*, **141**, 166-167.
- 32 PRIESTLY BG. (1984). Handling cytostatic drugs. *Q.E.H. Newsletter*, p.3, September.
- 33 STOCK JK & PRIESTLY BG. (1986). Urinary thioether output as an index of occupational chemical exposure in petroleum retailers. *Brit. J. Indust. Med.*, **43**, 718-720.
- 34 BRUSCHI S & PRIESTLY BG. (1989). Effects of β-naphthoflavone induction on microsomal oxygenase activity and acetaminophen-mediated cytotoxicity in differentiated human hepatoma cell line (HH₁). *In Vitro Toxicol.* **2**, 91-99.
- 35 BRUSCHI S & PRIESTLY BG. (1990). Implications of alteration in intracellular calcium ion homeostasis in the advent of paracetamol-induced cytotoxicity in primary mouse hepatocyte monolayer cultures. *Toxicol In Vitro*, **4**, 743-749, 1990.
- 36 SIMULA AP & PRIESTLY BG. (1991). The influence of vegetable oil vehicles on bone marrow proliferation in the mouse micronucleus test. *Mutation Res.* **261**, 83-84
- 37 SIMULA AP & PRIESTLY BG. (1992). Species differences in the genotoxicity of cyclophosphamide and styrene in three in vivo assays. *Mutation Res.* **271**, 49-58
- 38 EDWARDS JW & PRIESTLY BG. (1992). Biological and biological-effect monitoring of workers exposed to 4,4'-methylene bis (2-chloroaniline) (MOCA). *Human & Expt. Toxicol.* **11**, 229-236
- 39 NICHOLLS-GRZEMSKI FA, CALDER IC & PRIESTLY BG. (1992). Peroxisome proliferators protect against paracetamol hepatotoxicity in mice. *Biochem. Pharmacol.* **43**, 1395-1396
- 40 EDWARDS JW & PRIESTLY BG (1993) Sister chromatid exchange in lymphocytes of petroleum retailers. *Brit. J. Indust. Med.* **50**, 149-154.
- 41 EDWARDS JW & PRIESTLY BG (1994) Effect of occupational exposure to aldrin on urinary D-glucaric acid, plasma dieldrin and lymphocyte sister chromatid exchange. *Int. Arch. Occup. Environ. Health.* **66**, 229-234.
- 42 NICHOLLS-GRZEMKI FA, BURCHAM PC, CALDER IC & PRIESTLY BG (1996). Pretreatment with peroxisome-proliferators protects mice against some, but not all, hepatotoxins. *Ann. NY Acad. Sci.* **804**, 742-744
- 43 DI MARCO PN, DREW R, PRIESTLY BG & WRIGHT PFA (1996). Health based risk assessment of contaminated land: Focus on carcinogens. Report of the ASCEPT Workshop. In *The health risk assessment and management of contaminated sites*, Procs. Third National Workshop on the health risk assessment and management of contaminated sites. Edited by Langley A, Markey B & Hill H. South Australian Health Commission, Contaminated Sites Monograph series **5**: pp 275-278.

- 44 DI MARCO PD, PRIESTLY B.G. & BUCKETT KJ (1998). Carcinogen risk assessment. Can we harmonise? *Toxicology Letters* **102-103**; 241-246.
- 45 DI MARCO PD, ANDERSSON R, BADENOCH-JONES J, FITZGERALD J, LANGLEY J, MARKEY B, PRIESTLY B, PORTIER CJ, ROBERTSON D & CALLAN P (1999). Toxicity assessment for carcinogenic soil contaminants. National Health & Medical Research Council, Canberra <http://www.nhmrc.gov.au/publications/pdf/eh21.pdf>
- 46 NICHOLLS-GRZEMSKI FA, CALDER IC, PRIESTLY BG & BURCHAM PC (2000). Clofibrate-induced in vitro hepatoprotection against acetaminophen is not due to altered glutathione homeostasis. *Toxicological Sciences* **56**, 220-228
- 47 NICHOLLS-GRZEMSKI FA, BELLING GB, PRIESTLY BG, CALDER IC & BURCHAM PC (2000) Clofibrate treatment in mice confers resistance against lipid peroxidation. *J. Biochem. Mol. Toxicol.* **14**, 335-345
- 48 PRIESTLY BG, NOLLER B, MOORE M & NG J (2004) Human health risk assessment on the potable water contamination incident at Ranger Mine March 2004. Supervising Scientist Report 184, Supervising Scientist, Australian Government Department of the Environment & Heritage, pp 106-172. <http://www.deh.gov.au/ssd/publications/ssr/184.html>
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