Blackwattle Bay

State Significant Precinct

Attachment 19:

Human Health Risk Assessment

June 2021

NSW GOVERNMENT

BLACKWATTLE BAY HEALTH RISK ASSESSMENT

Particulate Matter and Nitrogen Dioxide
Volume I of II

Prepared for:

Infrastructure NSW
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SYDNEY NSW 2000

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BASIS OF REPORT

This report has been prepared by SLR Consulting Australia Pty Ltd (SLR) with all reasonable skill, care and diligence, and taking account of the timescale and resources allocated to it by agreement with Infrastructure NSW (the Client). Information reported herein is based on the interpretation of data collected, which has been accepted in good faith as being accurate and valid.

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DOCUMENT CONTROL

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EXECUTIVE SUMMARY

Background and Objective

This Human Health Risk Assessment (HHRA) has been prepared by SLR and on behalf of Infrastructure NSW, to form part of the Blackwattle Bay State Significant Precinct Study (SSP Study). This HHRA provides a comprehensive investigation of risk to human health from potential exposure to particulate matter (as $PM_{2.5}$ and PM_{10}) and nitrogen dioxide (NO_2) to address a part of the Study Requirements and support the development of a new planning framework for Blackwattle Bay.

The SSP Study seeks a rezoning for new planning controls for Blackwattle Bay, located on the south-western side of Pyrmont in Sydney, New South Wales. The outcome of the Blackwattle Bay SSP process will be a new planning framework that will enable further development applications for the renewal of the Precinct, connected to the harbour and centred around a rejuvenated Sydney Fish Market (SFM). The framework will also provide for new public open spaces including a continuous waterfront promenade, community facilities, and other compatible uses. Of particular interest and relevance to the HHRA are future plans to undertake residential development in the area, with current plans consisting of construction of a number of high-rise mixed commercial/residential towers. The objective of this HHRA was to assess the risk of harm for future residents, commercial/retail workers, and recreational users at the proposed development from exposures to PM and NO_2.

It is important to note the rezoning itself does not contribute to an increase in emissions of these air pollutants (existing emissions of PM and NO_2 originate predominantly from existing traffic on the surrounding road network as well as operations at a nearby concrete batch plant operated by Hymix); however, peoples' exposures to these air pollutants may theoretically change as a result of the rezoning.

The HHRA has utilised the results of air dispersion modelling undertaken by SLR as part of an Air Quality Impacts Assessment (AQIA). The assumptions used as inputs into the modelling err on the side of safety (i.e. they are conservative).

The AQIA and HHRA herein have been undertaken for the following two scenarios:

- Scenario 1: Redevelopment of the entire Study Area. This scenario assumes the Hymix concrete batch plant is no longer operational, and two buildings ('PLO 03 – Hymix 1' and 'PLO 03 – Hymix 2') are constructed where Hymix is currently located.
- Scenario 2: Partial redevelopment of the Study Area with the Hymix concrete batching facility remaining in place.

The AQIA predicted $PM_{2.5}$, PM_{10}, and NO_2 concentrations for a large number of equally spaced façade and roof locations on the proposed mixed-use towers, as well as a number of grid and boundary locations within and around the Study Area. This resulted in a total number of 2,354 modelled locations for Scenario 1 (Without Hymix) and 1,987 locations for Scenario 2 (With Hymix), the difference between the two scenarios being the absence of Buildings 'PLO 03 – Hymix 1' and 'PLO 03 – Hymix 2' in Scenario 2. While most of the grid based and boundary receptor locations were 1m above ground level (i.e. Ground Level Receptors), a number of elevated locations (i.e. Elevated Receptors) south, east, and north of the proposed development were also included both to enable the prediction from elevated roads bordering the Study Area but also to inform potential residential exposure to air emissions at existing residences at height (e.g. in apartment buildings) and residential/commercial/retail exposure at the proposed mixed-use towers forming part of the Precinct Plan.

EXECUTIVE SUMMARY

Methodology

The HHRA followed Australian guidance for completing such assessments (enHealth 2012a).

The relevant sources of emissions, exposure pathways and exposed populations were identified with the help of a Conceptual Site Model (CSM). Health effects data were gathered from publicly available scientific literature to establish a relationship between concentrations of $PM_{2.5}$, PM_{10} and NO_2 in air and relevant adverse health effects or health impacts. Effects were discussed in terms of health effects in individuals (e.g. observation of low effect levels in controlled human studies for NO_2) and/or population health endpoints (e.g. changes in mortality/morbidity incidence from baseline incidences with change in pollutant concentrations for all three pollutants). The latter relationships, termed concentration response functions or CRFs have been established/recommended by authoritative organisations based on studies examining associations of health endpoints with ambient exposures of large populations to these compounds in air.

Potential health risk to population groups exposed to $PM_{2.5}$, PM_{10} and NO_2 in air was determined by estimating a potential change in the annual incidence of specific health endpoints (as a rate per 100,000 people in the region) considered relevant to PM and NO_2 long-term (i.e. annual average) or short-term (i.e. 24-hour or in the case of NO_2, 1-hour) exposures. The potential change in population annual health outcomes was estimated using:

- CRFs gathered from the literature and authoritative reviews,
- the potential change in people's exposure calculated from the air dispersion modelling information, and
- baseline health incidence data for Sydney.

This type of health impact assessment assumes a non-threshold concentration response relationship for all three pollutants is appropriate. Whilst this assumption is commonly applied in health impact assessments on a population level, it may not be entirely appropriate for consideration of short-term health effects from exposure to NO_2 on an individual level. Therefore, for NO_2 the potential for short-term health effects to eventuate in individuals has also been assessed by comparing modelled 99.9^{th}, 99.7^{th} and 99^{th} percentile 1-hour NO_2 concentrations with the lowest available Low Observed Adverse Effect Concentration (LOAEC) from controlled human exposure studies in sensitive asthmatic adults (i.e. 188 $\mu g/m^3$). These percentile concentrations are estimated to occur for 9, 44, or 263 hours of the year, respectively.

Results and Conclusions

Population Health Endpoint Assessment

The population health endpoint assessment undertaken in this HHRA has shown that for both long-term and short-term exposure, the altered exposure circumstances of the proposed development are predicted to provide an average net health benefit. This means that on average lower population exposures to $PM_{2.5}$, PM_{10}, and NO_2 are anticipated to occur within the totality of the Study Area relative to other residences and commercial properties south, southeast, and north of the Study Area.

EXECUTIVE SUMMARY

When comparing Scenario 1 (without Hymix) and Scenario 2 (with Hymix), the estimated change in population health outcomes from long-term and short-term exposures to $PM_{2.5}$ within the Study Area are slightly better (i.e. provide higher benefit) for Scenario 1 (without Hymix) than Scenario 2 (with Hymix), albeit the proposed redevelopment in both Scenarios provides a net overall benefit relative to baseline conditions. The difference in population health outcomes for PM_{10} and NO_2 long-term exposures between Scenarios 1 and 2 are negligible.

There are a number of locations at which, when modelled individually, from a societal perspective, an unacceptable increase (i.e. >10 per 100,000 population) in a particular health outcome from short-term or long-term exposures to the pollutants evaluated may result. This should be interpreted as follows: if a large population of people were to live in that one receptor location, then there may be an unacceptable increase from baseline incidence in the health outcome. Clearly a large population could never live in the one location. This unacceptability at a few locations is more than balanced out by the numerous locations where there is an overall net health benefit (i.e. reduced exposure, and therefore reduced population incidence of an adverse health outcome). The majority of locations where this occurred were either retail or commercial locations on the façade of proposed buildings.

The analysis of short-term exposure health outcomes also revealed that, of the three pollutants investigated, the highest individual occurrences of increases in incidence of adverse population health outcomes occurred for exposures to NO_2.

Complementary NO_2 Assessment of Individual Exposures

The comparison of cumulative modelled 1-hour average NO_2 concentrations with the lowest LOAEC available from controlled exposure studies described in the literature for asthma exacerbation in individuals showed that where the same buildings are assumed to be present, the number and identity of locations where the LOAEC is predicted to be exceeded for 0.1-3% of the time are similar for Scenarios 1 and 2.

Scenario 1 (without Hymix) exhibits a larger total number of exceedances than Scenario 2 (with Hymix) since there are a large number of additional locations modelled due to the presence of the two additional buildings (PLO3 – Hymix 1 and PLO3 – Hymix 2), which are absent in Scenario 2.

Depending on the scenario evaluated, for the 99.9th percentile NO_2 concentrations (which may occur for 9 hours out of the year), a number of exceedances of the LOAEC were predicted to occur for different building locations. These were:

- BLD02: Three residential locations at 6 to 15m and one to two retail locations at ground level.
- BLD04: One to two retail locations at ground level.
- BLD05: Two retail locations at ground level.
- PLO1 – Poulos: Two commercial locations at 10-20m elevation and 3-5 retail locations at ground level.
- PLO2 – Celestino: 3-6 commercial locations at 6-10m elevation, 7 retail locations at ground level, and one podium roof location at 10-15m elevation.
- PLO3 – Hymix 1 (only applicable for Scenario 1): 7 commercial locations at 6-15m elevation, 9 retail locations at ground level, and 4 podium roof location at 10-15m elevation.
- PLO3 – Hymix 2 (only applicable for Scenario 1): 11 commercial locations at 6-20m elevation, 7 retail locations at ground level.

EXECUTIVE SUMMARY

Although the likelihood of sensitive asthmatics being present at these building locations at the exact time that concentrations of NO_2 greater than the LOAEC may be found is considered low (especially considering the inbuilt conservatism in the modelling), the possibility cannot be excluded. It is noted, however, that a sensitivity analysis was undertaken for Scenario 1 predictions in the AQIA to assess the impacts of using vehicular emission factors expected to be more representative of future emissions. This analysis showed that the incremental NO_2 concentrations are likely to be markedly overestimated (by approximately 56%) by the modelling predictions using the 2010 vehicle fleet emissions. Using the revised predictions for Scenario 1, the 99.9th percentile 1-hour NO_2 concentration is only predicted to be equal to or exceed the NO_2 LOAEC at three locations within the Study Area, all of these being retail locations (at 1m elevation) in PLO 03- Hymix 2 or BLD02.

It is therefore recommended (where practical) that management measures be considered to reduce potential NO_2 exposures at these retail locations.

Based on a comparison of modelled NO_2 concentrations at grid and boundary locations, it is considered unlikely that adverse effects resulting from exposure to NO_2 would be experienced by people using the proposed new public open spaces within the Study Area.

Uncertainties

Although uncertainties in the risk assessment may influence its accuracy, reliability and interpretation, the assumptions used to cope with the uncertainties in this HHRA tend to err on the side of safety and therefore bias the evaluation to over estimation rather than under estimation of health risk. This is appropriate for an assessment for possible impacts on public health. The principal uncertainties/assumptions in the HHRA are:

- *The air quality dispersion modelling provides a true reflection of Scenario 1 and 2 air quality.* A number of assumptions were made in undertaking the dispersion modelling which render the modelling results conservative. This conservatism is unlikely to markedly influence the results of the population health outcome assessment. However, potential overestimates of cumulative modelled air pollutant concentrations could also have resulted in an overestimation of the likelihood of adverse health effects in the complementary NO_2 assessment against a LOAEC, as demonstrated by the sensitivity analysis undertaken for Scenario 1 predictions (described above).
- *Concentration Response Functions (CRFs) are appropriate for the population under consideration.* The use of the 95% confidence interval CRFs would not alter the conclusions of this report.
- *Baseline health incidence data available at the time of writing this report was from 2010 and was only available for a wider area (i.e. Sydney) instead of the local population.* If the baseline incidence of a health outcome for the region which includes the Study Area is lower than what has been assumed in the HHRA, this would decrease the estimated change in population health outcomes and vice versa. It is therefore recommended that once the updated and more location-specific baseline incidence data become available, the potential impact of the updated information on the overall conclusions of the HHRA be considered.
- *No exposure adjustment was undertaken for commercial or retail locations in the population health impact assessment.* It was considered inappropriate to adjust the calculations to assume only 8 out of 24 hours exposure, since it is feasible that someone may both live and work in the proposed development. Exposure to air pollutants in general also does not cease when someone leaves work. The effect on the assessment is a potential overestimation for increased population incidence (from baseline) for the health endpoint assessment at commercial and retail locations.

EXECUTIVE SUMMARY

CONTENTS

CONTENTS

DOCUMENT REFERENCES

TABLES

CONTENTS

FIGURES

CONTENTS

APPENDICES

1 Introduction

This Human Health Risk Assessment (HHRA) has been prepared by SLR and on behalf of Infrastructure NSW, to form part of the Blackwattle Bay State Significant Precinct Study (SSP Study). The need for this HHRA was identified to address a part of the Study Requirements and support the development of a new planning framework for Blackwattle Bay.

The SSP Study seeks a rezoning for new planning controls for Blackwattle Bay, located in the south-western portion of Pyrmont in Sydney, New South Wales. The outcome of the Blackwattle Bay SSP process will be a new planning framework that will enable further development applications for the renewal of the Precinct, connected to the harbour and centred around a rejuvenated Sydney Fish Market (SFM). The framework will also provide for new public open spaces including a continuous waterfront promenade, community facilities, and other compatible uses. Of particular interest and relevance to the HHRA are future plans to undertake residential development in the area, with current plans consisting of construction of a number of high-rise mixed commercial/residential towers. Infrastructure NSW wish to understand the potential for health risks to people living in or using areas in the Study Area resulting from exposure to particulate matter (PM, i.e. $PM_{2.5}$, PM_{10}) and nitrogen dioxide (NO_2). It is important to note the rezoning itself does not contribute to an increase in emissions of these air pollutants[1]; however, peoples' exposures to these air pollutants may theoretically change as a result of the rezoning. The aim of this HHRA is to assess the risk of harm for future residents, commercial/retail workers, and recreational users who may be exposed to PM and NO_2.

This HHRA provides a comprehensive investigation of risk to human health from potential exposure to PM and NO_2 to address a part of the Study Requirements and support the development of a new planning framework for Blackwattle Bay. Relevant background information for the SSP Study is outlined in Section 2.1.

An overview of the methodology which was followed to undertake the HHRA is provided in Section 3. Section 4 presents a Conceptual Site Model (CSM) which helped identify the potential exposure pathways and relevant population groups for consideration in the HHRA. Hazard and exposure assessments are presented in Section 5 and Section 6, respectively. Human health risks have then been characterised in Section 7 of the report.

[1] Existing emissions of PM and NO_2 originate predominantly from existing traffic on the surrounding road network as well as operations at a nearby concrete batch plant operated by Hymix (SLR 2021).

2 Study Area information

2.1 Background Information

In 2015 the NSW Government recognised The Bays Precinct as one of the highest potential urban transformation sites in Australia with the release of The Bays Precinct, Sydney Transformation Plan (Urban Growth NSW 2015). Following this, the Minister for Planning identified the renewal of Blackwattle Bay and the broader Bays Precinct as a matter of State planning significance to be investigated for rezoning through the State Significant Precinct (SSP) process. Study Requirements for the Blackwattle Bay (formerly known as 'Bays Market District') investigation area were issued by the Minister on 28 April 2017.

The Blackwattle Bay SSP Investigation Area ('Study Area') encompasses the land and water area, known as Blackwattle Bay, between Bank Street (adjacent to the Western Distributor) and the Glebe foreshore shown in Figure 2-1. The land is located within the City of Sydney local government area (LGA).

SFM = Sydney Fish Market, UTS = University of Technology Sydney, SSP = State Significant Precinct

Figure 2-1 The Blackwattle Bay SSP Investigation Area ('Study Area')

The land within the Study Area is approximately 10.4 hectares (ha) in size. It is largely government owned land containing the Sydney Fish Market (SFM) (existing wholesale and retail)[2], recreation and boating operations and facilities. There are three privately owned sites including a concrete batching plant operated by Hymix, seafood wholesaler Poulos Brothers and private developer Celestino which owns further wholesaling facilities. The Blackwattle Bay land area wraps around the southern and eastern edges of Blackwattle Bay and is bounded by Bridge Road to the south and Bank Street to the east. The Western Distributor motorway / Anzac Bridge viaduct is located adjacent to the eastern boundary before traversing over the northern section of the site. The water area of Blackwattle Bay is approximately 21 hectares.

Key characteristics of the rezoned Blackwattle Bay Precinct Plan include (shown in Figure 2-2 below):

- New homes, jobs and services close to the CBD including:
    - 5,636 jobs / or approximately 5,600 jobs.
    - 1,546 dwellings for approximately 2,800 new residents.
- A continuous waterfront promenade providing a 15 km foreshore walk from Woolloomooloo to Rozelle (blue dotted line) and new and improved pedestrian and cycling links.
- Improved public transport options and minimised vehicle usage strategies including:
    - Minimising car parking spaces with limited on-street parking.
    - Ferry wharf.
    - Opportunity for buses to service through site link.
    - Connections to the existing light rail and access to a future Sydney Metro West Station in Pyrmont.
- New parks and green space with 30,000 m^2 of new open space.
- A new (larger) SFM at the heart of Blackwattle Bay.

[2] A critical part of Blackwattle Bay's revitalisation and vision has been the NSW Government's decision to relocate the Sydney Fish Market (SFM) from its existing location on Bank Street to the head of Blackwattle Bay. This approval was sought through a State Significant Development Application (SSDA) process and was approved in June 2020. The new SFM was designed alongside the baseline Blackwattle Bay studies to ensure that key aspects of the project are consistent with the vision and principles for Blackwattle Bay.

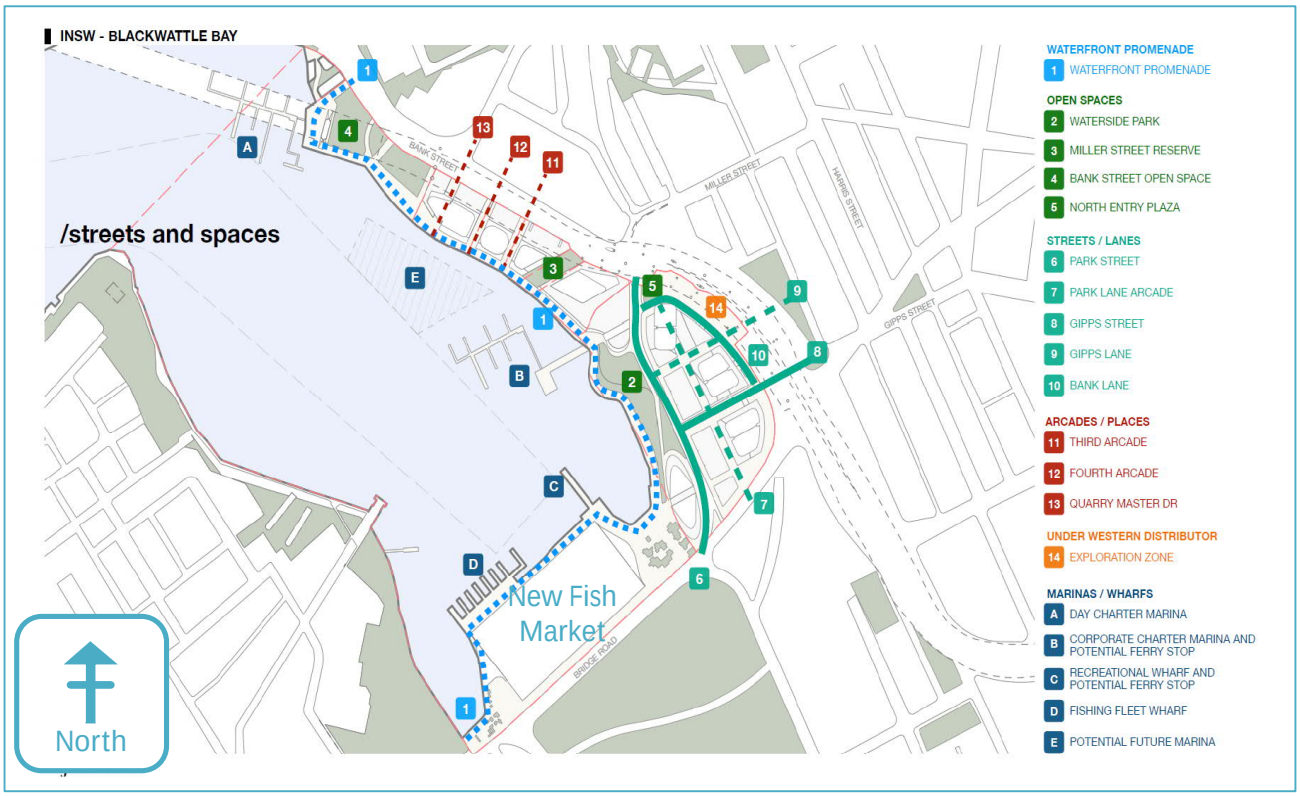


Figure 2-2 Proposed New Infrastructure in the Blackwattle Bay State Significant Precinct (SSP)

2.2 Study Area and Surrounds

A summary of the study area information is detailed below in Table 2-1. The study area location is shown above in Figure 2-1. The extent of land area and water area within Blackwattle Bay will change with the construction of the new SFM. Previously, the total land area was 10.4 hectares and water area was 21 hectares. However, part of the new SFM is being developed below the mean high-water mark, increasing the overall land area of the study area to 10.4 hectares and reducing the water area to 21 hectares. The site is currently zoned Commercial Core (B3) or Public Recreation (RE1).

Table 2-1 Summary of study area information

Site Information	Details
Local Government	City of Sydney Council
Study Area	Approximately 10.4 hectares of predominantly government owned land located approximately 1km from the Sydney Central Business District.
Area – Prior to development.	• Land - 10.4ha (land), 21ha (water). • Private landowners – 1.6ha (15,735sqm).
Area – Post development.	• Land - 10.4ha (land), 21ha (water). • Private landowners – 1.6ha (15,735sqm).
Current Zoning	B3 – Commercial Core, RE1 – Public Recreation
Zoning in Surrounding Areas	B2 – Local Centre, B3 – Commercial Core, B4 – Mixed Use, SP2 – Infrastructure, RE1 – Public Recreation and R1 – General residential

The SSP Study is proposing to rezone Blackwattle Bay with a new planning framework and planning controls to enable its future urban renewal.

The rezoning proposal is based on the Blackwattle Bay Precinct Plan ('Precinct Plan') which provides a conceptual layout to guide the development of planning controls for the precinct and has informed this report. The Precinct Plan provides overarching guidance about how the area should be developed based on community and stakeholder input, local character and place, current and future demographics, economic and social trends, cultural and environmental considerations, and urban renewal aspirations and needs regarding land use, community recreation, transportation, housing, and jobs. Key characteristics of the Precinct Plan are outlined in Section 2.1.

Once the Study Area is rezoned and the new planning controls are in place, future development will need to seek development approval through the relevant approval pathway. This will include detailed development proposals and further associated environmental, social and economic assessments.

The rezoning proposal responds to the Study Requirements issued for Blackwattle Bay (formerly Bay's Market District) by the Department of Planning and Environment in April 2017.

An aerial image of the Precinct Plan is shown in Figure 2-3, and a concept plan of the proposed new buildings is provided in Figure 2-4. Locations of proposed buildings 'PLO 01 - Poulos', 'PLO 02 - Celestino', 'PLO 03 – Hymix 1' and 'PLO 03 – Hymix 2' are currently on privately owned land, whereas the land for the remainder of the proposed buildings is owned by Infrastructure NSW.

The area surrounding the Study Area includes lands zoned as local centre, commercial core, mixed use, general residential, public recreation and infrastructure as seen in Figure 2-5. There are a number of existing residences located southeast, northeast, southwest and west of the Study Area.

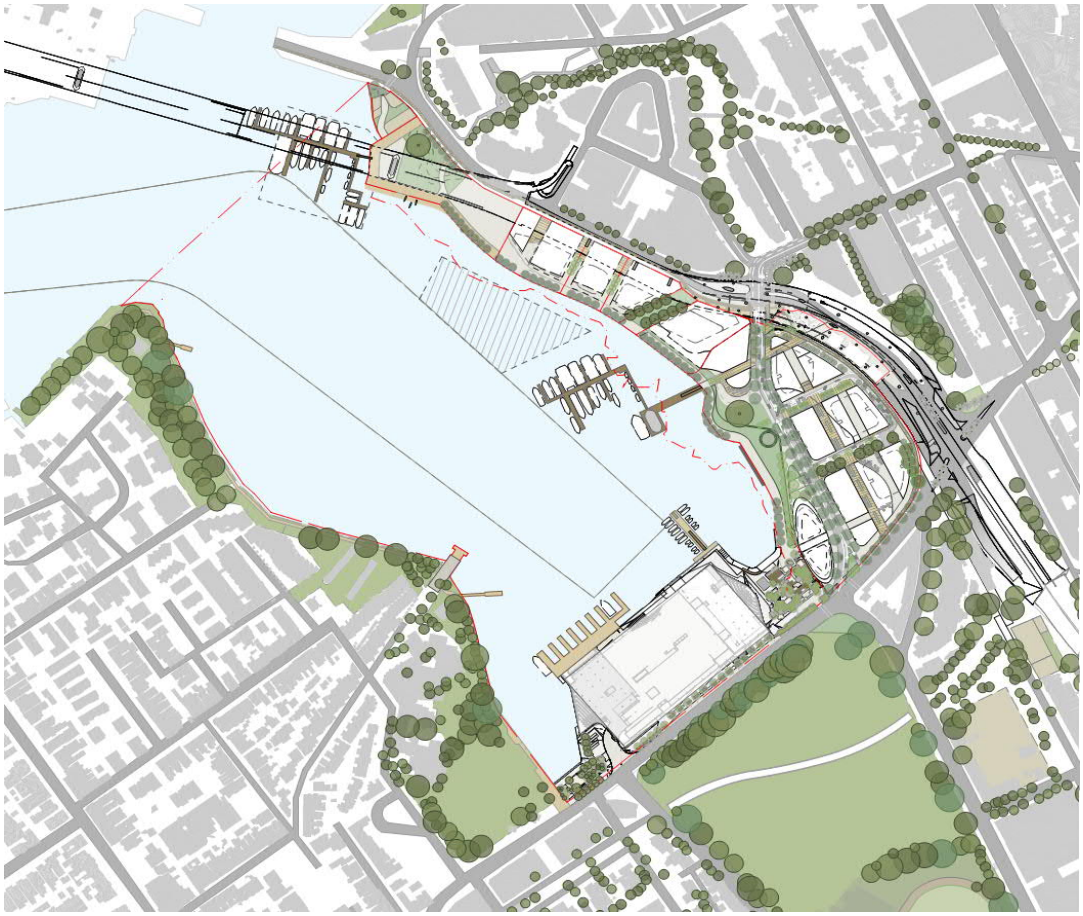


Figure 2-3 Blackwattle Bay Precinct Plan (supplied by Infrastructure NSW)

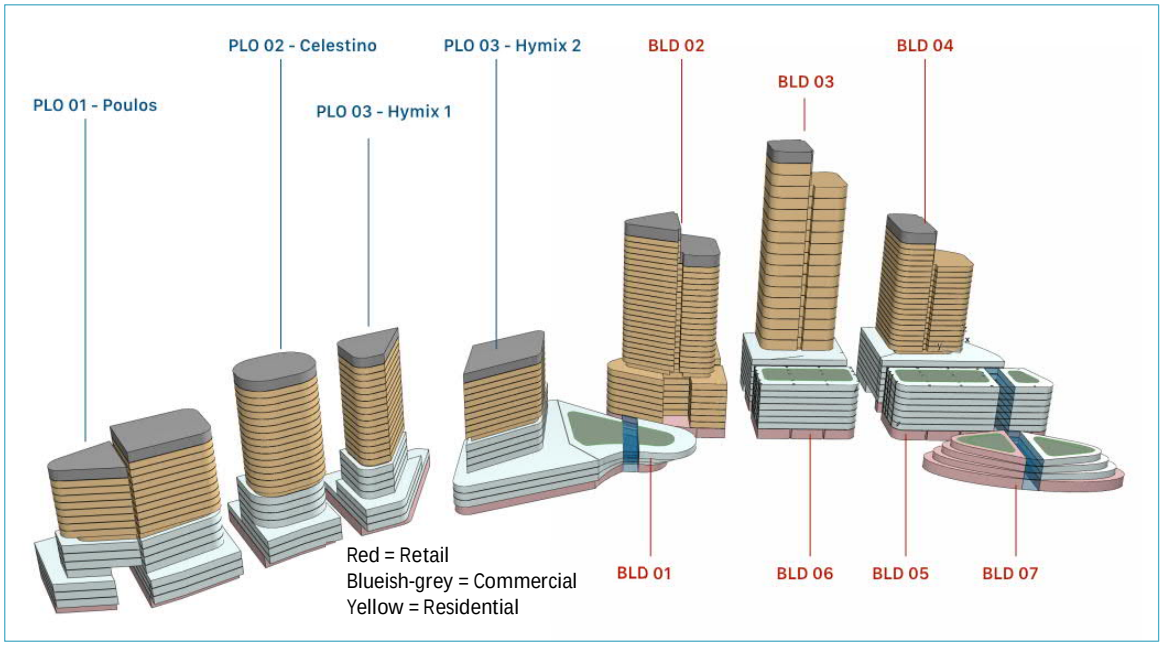


Figure 2-4 Proposed new buildings in Blackwattle Bay Precinct Plan (supplied by Infrastructure NSW)

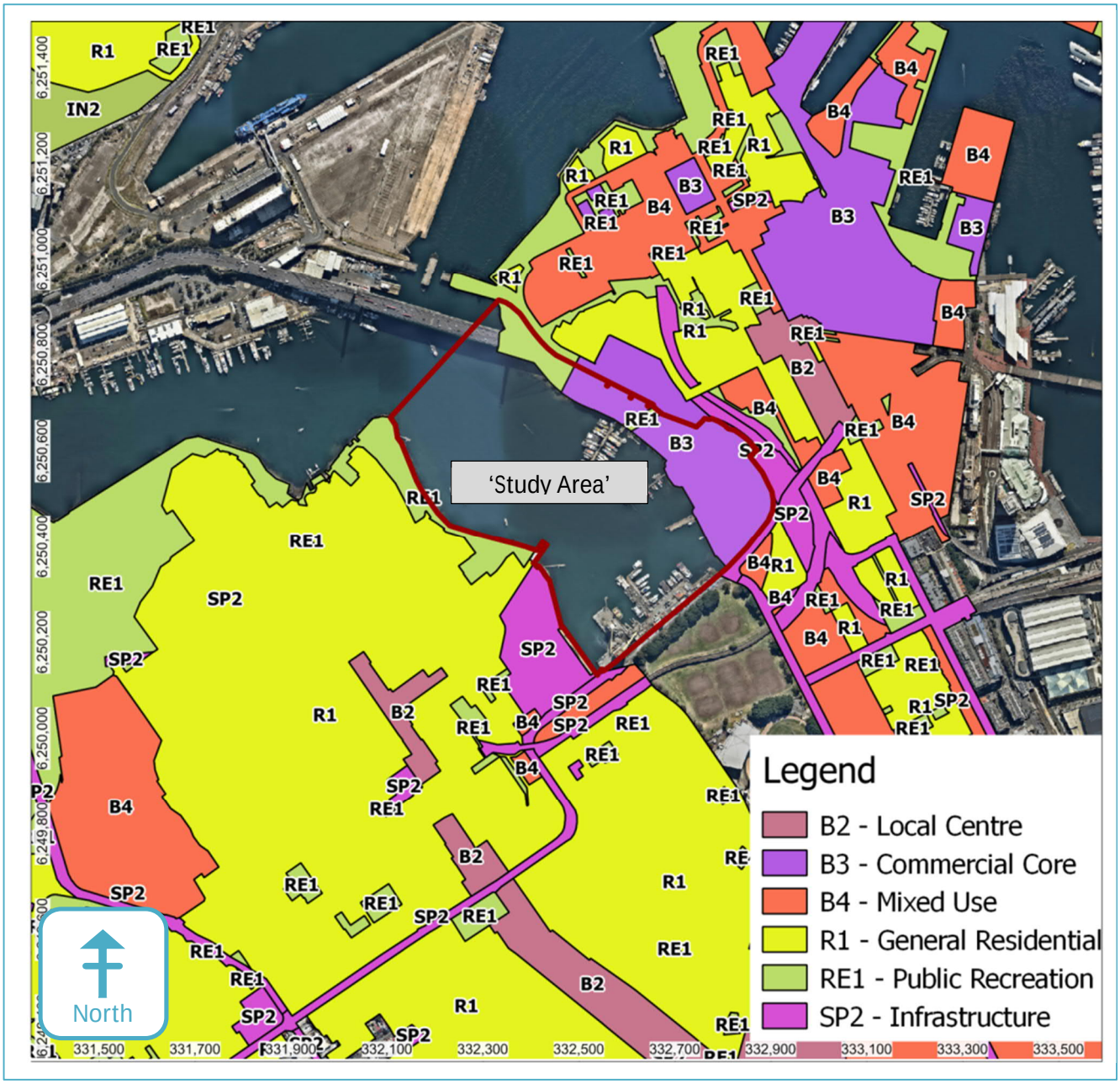


Figure 2-5 Existing Land Uses surrounding Study Area

2.3 Demographics

The Study Area is located in Central Sydney, NSW, < 1.5 kms to the west of the Central Business District (CBD), within the City of Sydney LGA. The Blackwattle Bay land area wraps around the northeast, south and south-west edges of Blackwattle Bay and the surrounding suburbs relevant to this HHRA are Glebe, Pyrmont and Ultimo. The Western Distributor Motorway/Anzac Bridge is located adjacent to the eastern and northern boundaries of the Study Area.

The nearest existing residential areas can be found within 50 metres to the south of the Study Area boundary on Wattle Crescent (Ultimo), north of the Study Area on Bank St (Pyrmont), southwest of the Study Area on Bridge Rd (Glebe), and along the majority of the south-western boundary of the Study Area, from Bridge Rd to Blackwattle Bay Park and Wharf (Glebe). The Blackwattle Bay campus of Sydney Secondary College occupies 250 m of the south-western boundary of the Study Area (Figure 2-6). The closest childcare centres are Thrive Early Learning approximately 150m north-east of the Study Area and Kindy Patch child care approximately 700m south-east of the Study Area.

Figure 2-6 Places of Interest Surrounding Study Area

Based on 2016 census data, the populations of the local suburbs were Glebe: 11,532, Pyrmont: 12,813, and Ultimo: 8,845 (ABS 2016). Table 2-2 summarises the age distribution for residents, which shows a lower proportion of children under 15 years in all three suburbs compared to the national average (7.6% compared to 18.7% for Australia) and a lower proportion of people over the age of 55 in Pyrmont and Ultimo (18.5% and 8%) compared to the national average (27.6%), but this was not the case in Glebe (26.1%) (ABS 2016). In general, the suburbs surrounding Blackwattle Bay have a higher proportion of young to middle aged adults, and fewer children and older people, compared to the national average.

Table 2-2 The age distribution for residents of Glebe, Pyrmont and Ultimo compared to the national average [1]

Age Group	Glebe	Pyrmont	Ultimo	Blackwattle Bay Average	National Average
0-4 years	3.4%	4.7%	2.3%	3.5%	6.3%
5-14 years	5.9%	4.5%	2.0%	4.1%	12.4%
15-24 years	15.4%	11.2%	38.9%	21.8%	12.8%
25-54 years	49.2%	61.0%	48.9%	53.0%	41.2%
55-64 years	12.0%	9.5%	3.8%	8.4%	11.8%
65 years +	14.1%	9.0%	4.2%	9.1%	15.8%

1. Data sourced from ABS (2016).

During specific periods of life (i.e. childhood and advanced age), individuals may be more susceptible to the health effects of particulate matter. An evaluation of age-related health effects suggests that older adults have heightened responses for cardiovascular health effects with particulate matter exposure (US EPA 2010). The toxicological profiles in Appendix A provide additional information.

2.4 Population Health Statistics

This section provides an overview of the health of the community currently living in the vicinity of the Study Area. The key focus of the assessment presented is the local community within the City of Sydney LGA, however some aspects of the assessment require consideration of health indicator statistics that are derived from larger populations, such as those within the South Eastern Sydney Local Health District (LHD, which includes the Botany Bay, Hurstville, Kogarah, Randwick, Rockdale, Sutherland Shire, Waverley and Woollahra LGAs), the Central and Eastern Sydney Primary Health Network, and the greater Sydney Area. Hence, where relevant, information related to both the local community and other areas within Sydney (and NSW) has been presented.

There are a large number of factors to consider when describing the health of a local community. The health of the community is influenced by a range of factors including age, socio-economic status, behaviours, lifestyle, country of origin, genetics and access to health care and social support. Population statistics for health endpoints of interest (see Section 2.4.2) are not readily available in the format which match concentration-response functions for the pollutants considered in this report. Therefore, the specific health statistics of interest were requested from the Australian Bureau of Statistics (ABS – for mortality data) or the Australian Institute of Health and Welfare (AIHW – for morbidity data). At the time of writing this report, the requested data had not yet been received by SLR. For this reason, baseline health statistics for the wider Sydney area from 2010 (which were publicly available in Frangos and Di Marco 2013) have been assumed to be representative of the smaller population located in the vicinity of the Study Area. It is anticipated that the influence on the HHRA conclusions of using the more up-to-date health statistics for the LHD will be considered when they become available.

2.4.1 Health-related behaviours

Information about health-related behaviours that are linked to poorer health status and chronic disease is available for large population areas in Sydney and NSW. This includes information on alcohol consumption, smoking, fruit and vegetable consumption, body weight and physical activity. Review of the general health for residents in South Eastern Sydney LHD indicates that high level health indicator measures such as life expectancy at birth and deaths from all causes for these residents are slightly better than the NSW average. Residents from South Eastern Sydney LHD, on average, reflected a lower proportion of obese adults (10% lower), and higher levels of physical activity compared to the NSW average (9% higher), with similar levels of smoking and fruit and vegetable consumption, but higher levels of risky alcohol consumption (4% higher) (NSW Gov 2019).

2.4.2 Health Statistics

In relation to some more specific health indicators[3] that are of particular relevance for the more detailed assessment of exposure to PM and NO_2, the subject of this HHRA, Table 2-3 presents the available data for population areas defined under the South Eastern Sydney LHD, Sydney, and/or NSW as a whole.

As discussed above, for the assessment of potential health impacts due to rezoning and potential future residential development in the Study Area, where specific health statistics for the smaller population adjacent to the Study Area were not available (and/or not reliable due to the small size of the population), adopting health statistics from the whole of Sydney or whole of NSW is considered to provide a reasonably representative summary of the existing health of the population of interest. In addition, it is feasible that people may move into the rezoned Study Area from elsewhere in NSW or indeed elsewhere in Australia.

Review of the data from the South Eastern Sydney LHD compared to the average of all NSW LHDs indicates the following:

- Deaths from all causes in the South Eastern Sydney LHD were lower than for NSW as a whole, and mortality rates for circulatory disease and respiratory disease (including asthma and Chronic Obstructive Pulmonary Disease, COPD) were lower in South Eastern Sydney LHD.
- Hospitalisation rates for circulatory disease and coronary heart disease in South Eastern Sydney LHD are higher than the NSW average, while hospitalisation rates for asthma and COPD are lower than the NSW average. Asthma prevalence is also lower in South Eastern Sydney LHD than the NSW average.

Table 2-3 Summary of key health indicators in South Eastern Sydney compared to NSW average

Health Indicator	Rate per 100,000 population		
	2010 (1)	2016-2019(2)	
	Sydney	South Eastern Sydney Local Health District	All NSW Local Health Districts
Mortality			
All causes (non-trauma) – all ages	569	-	-
All causes (non-trauma) – 30+	930	-	-
All causes – 30+	977	-	-
All causes – all ages	-	443.6 (3)	506.4 (3)

[3] The health indicators include those that are specifically relevant to the quantification of exposure to PM and NO_2 presented in Section 6.

Health Indicator	Rate per 100,000 population		
	2010 (1)	2016-2019(2)	
	Sydney	South Eastern Sydney Local Health District	All NSW Local Health Districts
Circulatory disease – all ages	-	118.9	136
Cardiopulmonary – 30+	392	-	-
Cardiovascular disease – all ages	192	-	-
Ischemic heart disease – 30+	147	-	-
Lung cancer – 30+	57	-	-
Respiratory disease – all ages	52	37.5	49.3
Asthma – all ages	-	1.3	1.6
COPD – all ages	-	16.6	24.5
COPD – 65+ years	-	120.9	175.4
Hospital admissions			
Asthma – all ages	-	120.6	144.2
Asthma – 5-34 years	-	147.4	154.7
Asthma – 1-14 years *	1221	-	-
Asthma – 65+	165	-	-
Circulatory disease – all ages	-	1728.6	1672.4
Coronary heart disease – All ages	-	570.8	493.8
Coronary heart disease – 75+ years	-	3481.3	2947.8
Cardiovascular disease – 1-14 yrs	55	-	-
Cardiovascular disease – 15-64 yrs	1170	-	-
Cardiovascular disease – 65+	9065	-	-
Cardiac disease – 1-14 yrs	29	-	-
Cardiac disease – 15-64 yrs	668	-	-
Cardiac disease – 65+	6208	-	-
Cardiac failure – 1-14 yrs	1.4	-	-
Cardiac failure – 65+	1217	-	-
Pneumonia + bronchitis – 1-14 yrs	330	-	-
Pneumonia + bronchitis – 65+	1072	-	-
Respiratory disease – 1-4 yrs	-	-	-
Respiratory disease – 5-14 yrs	-	-	-
Respiratory disease – 1-14 yrs	2431	-	-
Respiratory disease – 15-64 yrs	792	-	-
Respiratory disease – 65+	3905	-	-

Health Indicator	Rate per 100,000 population		
	2010 (1)	2016-2019(2)	
	Sydney	South Eastern Sydney Local Health District	All NSW Local Health Districts
COPD – all ages	-	131.3	224.8
COPD – 65+ years	-	855.6	1351.9
All respiratory disease – all ages	-	1359.9	1675.2
Asthma prevalence			
Asthma – 2-15 years	-	10.7	13.1

Bolded values indicate higher prevalence of health indicator in South Eastern Sydney LHD compared with all NSW LHDs. Grey shaded cells indicate health indicators with concentration-response functions recommended for use in Australia as per the work underpinning the update of the ambient air quality NEPM (i.e. Jalaludin and Cowie 2012, Frangos and Di Marco 2013).

* Emergency department visits.

1. Source = Frangos and Di Marco (2013), the data used for the HHRA underpinning the NEPM update. Population incidence (per 100,000) for the most recent year available (2010) were calculated using the incidence data for Sydney provided in Frangos and Di Marco (2013, Appendix D, Tables 1-11) combined with the population data in Tables 19-23 of the same publication in order to calculate the incidence rate per 100,000 people in a particular age group.
2. Source = HealthStats NSW (NSW Gov 2020). Data for different endpoints was variably available for different years between 2016 and 2019.
3. It is not clear from the HealthStats NSW website (NSW Gov 2020) whether this value includes or excludes deaths from trauma.

2.4.3 Uncertainties

There are limitations in the use of these data for the quantification of health impact and risk. The data are calculated from statistics recorded by hospitals and medical practices, reported by postcode of residence, and are dependent on the correct categorisation of health problems. There is expected to be some under-reporting of less serious conditions as individuals may not seek medical assistance. The baseline data considered is only a general indicator of the incidence of these health endpoints.

2.5 Available Air Quality Information

The primary sources of air emissions immediately surrounding the Study Area are vehicles travelling along the Western Distributer and other local roads as well as the existing Hymix concrete batching facility (in the event that this land is not rezoned as part of the proposed development). Engine exhaust emissions will also be generated by marine traffic within Blackwattle Bay and the wider Sydney Harbour, including ferries and water taxis, fishing trawlers, cruise ships visiting Darling Harbour and recreational boating (SLR 2021). Different emission sources have different effects on PM particle size. For example, exhaust particulate emissions from road vehicles are almost entirely in the $PM_{2.5}$ fraction, whereas non-exhaust particles arising from processes such as tyre wear and brake wear are more likely to be in the coarse fraction ($PM_{10-2.5}$). Dust storms tend to have high PM_{10} levels but only moderate effects on those of $PM_{2.5}$, whereas bush fires tend to have high levels of $PM_{2.5}$ but only moderate levels of PM_{10} (NEPC 2014). Vehicle exhaust is the principal source of NO_2 in the area.

Ambient air quality monitoring performed in the Sydney area over the last few decades has shown that the city's air quality has improved and is continuing to improve. A major driver of this improvement in urban air quality is the fact that newer vehicles produce significantly less emissions than older vehicles (SLR 2021).

A search of the EPA public register and NPI database within a 3 km radius of the Study Area identified several industries which could potentially impact local air quality (SLR 2021). Considering the separation distances, prevailing winds and activity types associated with the identified emission sources, significant air quality impacts at the Study Area from facilities other than the Hymix Australia operations are considered unlikely (SLR 2021). It is noted the Environmental Protection License for the Hymix facility is no longer in force. However, it is understood that Hymix may remain in its existing location and continue concrete batching and possibly bulk shipping activities in the medium and long term, either in its existing guise or as a redeveloped / upgraded facility (SLR 2021). Hymix is also anticipated to be a potential source of PM and NO_2. For this reason, the air quality impact investigation undertaken by SLR (2021) and this HHRA Report have considered two potential exposure scenarios: with and without the Hymix facility (see Section 6.3).

The NSW Office of Environment and Heritage (OEH) maintains a network of air quality monitoring stations (AQMS) across NSW. The nearest such OEH stations are located at Cook and Philip Park and Rozelle, approximately 1.9 km east and 2.6 km to the northwest of the Study Area respectively. The Cook and Philip Park AQMS does not include long term data as it was commissioned in September 2019. The AQIA used data from the Rozelle AQMS to inform background air quality for the Study Area (SLR 2021). Although a review of the 2015-2019 air quality data for this station revealed a number of exceedances of the NSW EPA (2017) 24-hour average PM_{10} and 24-hour average $PM_{2.5}$ criteria, this was mainly due to exceptional events including bushfires, dust storms and hazard reduction burning (SLR 2021). Ambient $PM_{2.5}$ concentrations exceeded the annual average NSW EPA (2017) criterion at Rozelle in 2019, and frequently exceed the annual criterion across the Sydney Greater Metropolitan Area (SLR 2021). The NO_2 criteria were not exceeded at this monitoring station in any of the years reviewed (SLR 2021).

2.5.1 Air quality dispersion modelling

Air dispersion modelling was undertaken by SLR (2021) to model the incremental levels of PM and NO_2 in the Study Area from the Hymix concrete batch plant and the surrounding road network. It is noted the proposal for rezoning the Study Area would itself make negligible contribution to altering existing air quality. The assumptions, inputs and methodology employed to undertake the air dispersion modelling are detailed in the AQIA Report (SLR 2021). The assumptions used as inputs into the modelling err on the side of safety (i.e. they are conservative) and include:

- Vehicular emissions were estimated based on a low vehicle speed of 10 km/hr (potential worst-case emission rate that would be representative of congested traffic conditions) from 6am to 10pm every day of the year.
- In addition, potential improvements in emissions performance of newer cars in the future were not accounted for. It was assumed the 2033 vehicle fleet emission rates are similar to the 2010 vehicle fleet.
- Particulate emissions from aggregate and sand transfer and weigh hopper loading activities at the concrete batch plant are uncontrolled.
- All available ambient ozone was assumed to react immediately with emitted NO to form NO_2.

PM and NO_2 concentrations were predicted for a large number of equally spaced façade and roof receptor locations on the proposed mixed-use towers, as well as a number of grid and boundary receptors within and around the Study Area. This resulted in a total number of 2,354 modelled receptor locations for Scenario 1 (Without Hymix) and 1,987 receptor locations for Scenario 2 (With Hymix), the difference between the two scenarios being the absence of Buildings 'PLO 03 – Hymix 1' and 'PLO 03 – Hymix 2'. While most of the grid based and boundary receptor locations were 1m above ground level (i.e. Ground Level Receptors), a number of elevated locations (i.e. Elevated Receptors) south, east, and north of the Study Area were also included both to enable the prediction from elevated roads bordering the Study Area but also to inform potential residential exposure to air emissions at existing residences at height (e.g. in apartment buildings) and residential/commercial/retail exposure at the proposed mixed-use towers forming part of the Precinct Plan (SLR 2021).

2.5.2 Modelled Air Concentrations for PM and NO_2

The health impact assessment undertaken within this HHRA report requires quantification of an incremental change in potential exposure to PM and NO_2 due to the proposed rezoning of the Study Area. The proposed rezoning itself does not contribute to a change in air quality *per se* except for a potential improvement in air quality for Scenario 1 (i.e. should the development proceed without Hymix). However, the location in which retail, commercial and residential activities take place (i.e. within the proposed mixed-use towers) may signify a change in the location where exposures to existing air emissions occur.

For example, a resident may live in an apartment on Wattle Crescent, just south of the Study Area. That resident will be exposed to existing air emissions from the surrounding roads and Hymix concrete batch plant. However, if they were to move into one of the proposed residential towers the resident's exposure may conceptually be somewhat different due to closer proximity to the concrete batch plant (for Scenario 2 only), height of the apartment (i.e. modelled PM and NO_2 concentrations generally decrease with height due to more effective dispersion) and/or proximity to the road (e.g. an apartment facing the bay may be subject to different emissions than an apartment facing the road). Therefore, in this HHRA, the incremental change in potential exposure to PM and NO_2 has been calculated by estimating the difference between modelled air emissions for a group of existing receptor locations (at particular heights) and the proposed new receptor locations (at comparable heights) forming part of the Precinct Plan.

The locations where people may be exposed (i.e. receptor locations), including existing receptor locations, are further shown and discussed in Section 6.2.

The AQIA modelling found that emissions from local traffic and activities at Hymix (for Scenario 2 only) have the potential to result in exceedances of the ambient air quality criteria for PM_{10}, $PM_{2.5}$ and NO_2 within the Study Area, particularly on lower floors of buildings at locations closest to the Western Distributor and Hymix (SLR 2021). In summary (SLR 2021):

- exceedances of the 24-hour average PM_{10} criterion (50 µg/m^3) are predicted to occur at 5% of residential receptor locations modelled for Scenario 1 and 11% of residential receptor locations modelled for Scenario 2.
- exceedances of the annual average PM_{10} criterion (25 µg/m^3) are predicted to occur at none of residential receptor locations modelled for Scenario 1 and 2% of residential receptor locations modelled for Scenario 2.
- exceedances of the 24-hour average $PM_{2.5}$ criterion (25 µg/m^3) are predicted to occur at 1% of residential receptor locations modelled for Scenario 1 and 3% of residential receptor locations modelled for Scenario 2.

- exceedances of the annual average $PM_{2.5}$ criterion (8 µg/m^3) are predicted to occur at 2% of residential receptor locations modelled for Scenario 1 and 7% of residential receptor locations modelled for Scenario 2.
- exceedances of the 1-hour average NO_2 criterion (246 µg/m^3) are predicted to occur at 4% of residential receptor locations modelled for Scenario 1 and 4% of residential receptor locations modelled for Scenario 2.
- exceedances of the annual average NO_2 criterion (62 µg/m^3) are predicted to occur at none of the residential receptor locations modelled for Scenario 1 and 0.5% of residential receptor locations modelled for Scenario 2.

This indicates there are a higher number of residential receptors where modelled PM or NO_2 concentrations exceeded relevant criteria for Scenario 2 which assumes Hymix continues to operate as currently permitted. It is noted the proposed Precinct Plan incorporates a number of mitigation measures including:

- Minimising the formation of urban canyons by having buildings of different heights interspersed.
- No residential units are proposed within a 20 m radius of the major roads.
- For all proposed buildings (with the exception of BLD 02) the lower eight floors are proposed to be used for commercial/retail purposes. For BLD 02, the ground floor is proposed to be used for retail purposes.

The AQIA concluded from an air quality perspective, the Study Area is suitable for the intended uses within the SSP proposal subject to the findings of the HHRA and future design development on final proposals of high-level mitigation measures summarised within this study.

The HHRA herein will help inform if any additional air quality mitigations are recommended for each building.

For each receptor location, the HHRA used the following data from the AQIA investigation:

- Annual average concentrations for PM_{10}, $PM_{2.5}$ and NO_2 (to evaluate the potential health effects/impacts from long-term exposures).
- Time series data for 24-hour average concentrations for PM_{10} and $PM_{2.5}$ (to evaluate the potential health effects/impacts from short-term exposures to PM).
- Time series 1-hour maximum concentrations for NO_2 (to evaluate the potential health effects/impacts from short-term exposures).

3 HHRA Methodological Considerations

The HHRA herein broadly follows four steps outlined in Australian guidance for completing such assessments, namely 'the enHealth Risk Assessment guidelines' (enHealth 2012a). These four steps are consistent with risk assessment guidelines for contaminated sites in Australia (Schedule 4 from NEPM 2013):

1. Issue Identification (Section 2 and Section 4): Background information (Precinct Plan details) is discussed along with details of the Study Area and its surroundings, local demographics and population health statistics. Information on available air quality data used in the HHRA is also summarised (Section 2.5). Identification of the relevant sources of emissions, exposure pathways and exposed populations is undertaken with the help of a Conceptual Site Model (CSM, Section 4).
2. Hazard Identification (Section 5 and Appendix A): Health effects data are discussed to establish a relationship between concentrations of $PM_{2.5}$, PM_{10} and NO_2 in air and relevant adverse health effects or health impacts. Effects from exposure to $PM_{2.5}$, PM_{10} and NO_2 are discussed in terms of health effects (e.g. observation of low effect levels in controlled human studies) or population health endpoints (e.g. changes in mortality/morbidity from baseline incidences with change in pollutant concentrations). The latter relationships, termed concentration response functions or CRFs have been established/recommended by authoritative organisations based on studies examining associations of health endpoints with ambient exposures of populations to these compounds in air.
3. Exposure Assessment (Section 6): In this step of the HHRA, the relevant exposure scenarios, parameter values and equations necessary for the quantification of health risk and health impact are provided. The exposure assessment also provides the methodology for quantifying the potential change in incidence of population health outcomes as a result of the proposed development.
4. Risk Characterisation (Section 7): Potential health risk to population groups exposed to $PM_{2.5}$, PM_{10} and NO_2 in air was determined by estimating a potential change in the annual incidence of specific health endpoints (as a rate per 100,000 people in the region) considered relevant to PM and NO_2 long-term (i.e. annual average) or short-term (i.e. 24-hour or in the case of NO_2, 1-hour) exposures. The potential change in population annual health outcome incidence is estimated using CRFs from the hazard identification section (Section 5.2) and baseline health incidence data for Sydney (Section 2.4.2). This type of health impact assessment assumes a non-threshold concentration response relationship for all three pollutants is appropriate. Whilst this assumption is commonly applied in health impact assessments on a population level, it may not be entirely appropriate for consideration of short-term health effects from exposure to NO_2 on an individual level. Therefore, for NO_2 the potential for short-term health effects to eventuate in individuals has also been assessed by comparing modelled 99.9^{th} percentile 1-hour NO_2 concentrations with a Low Observed Adverse Effect Concentration (LOAEC) from controlled human exposure studies in sensitive asthmatic adults.

The methodological process followed in this HHRA is illustrated Figure 3-1 below.

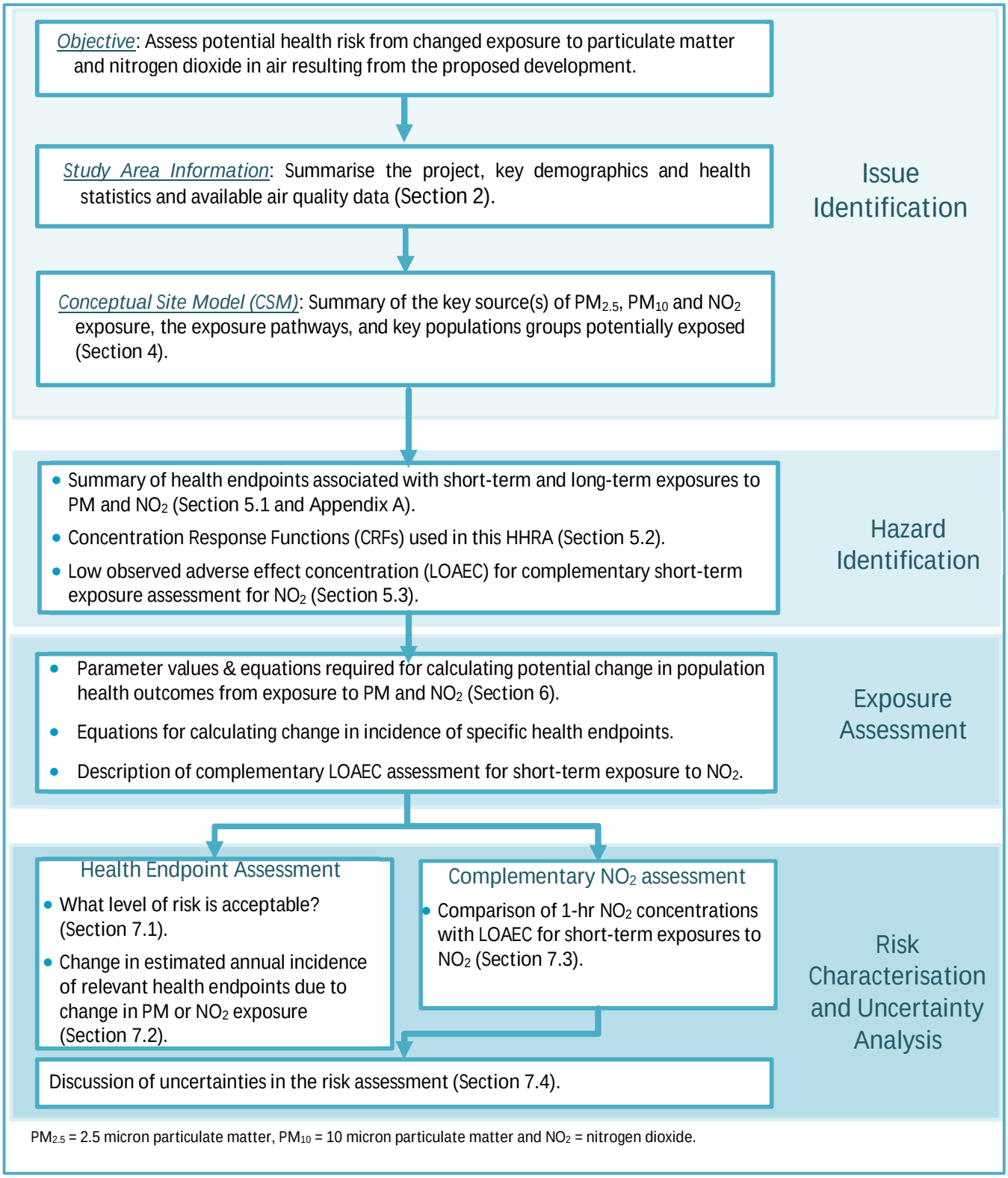


Figure 3-1 Summary of HHRA Methodological Process

4 Conceptual site model

In health risk assessments, a conceptual site model (CSM) is typically developed to assist in understanding the key source(s) of exposure to the chemicals being assessed, the pathways through which people may be exposed to these sources, and the key population groups (including the most sensitive people) that may be involved in the exposure. The subsequent sections outline these aspects for the Study Area.

4.1 What are the sources of exposure?

As discussed in Section 2.5, the primary sources of PM and NO_2 in air in the vicinity of the Study Area are vehicular emissions from vehicles travelling along the Western Distributer and other local roads as well as the existing Hymix concrete batching facility (in the event that this land is not rezoned as part of the proposed development, i.e. Scenario 2). It is reiterated the proposed rezoning and development does not itself contribute to an increase in emissions of these air pollutants; however, peoples' exposures to these air pollutants may theoretically change as a result of the rezoning.

The PM and NO_2 emissions disperse in air from where they are released; the degree of dispersion in the Study Area is related to meteorological conditions, height, and obstructions (e.g. buildings) (SLR 2021).

4.2 What are the relevant pathways of exposure?

An exposure pathway consists of two components, the migration pathway (the manner in which PM and NO_2 emissions move away from the sources) and the exposure route (the process by which the emissions come into contact with a person). The exposure pathway considered relevant in this HHRA for PM_{10}, $PM_{2.5}$, and NO_2 emissions from vehicles and the Hymix concrete batch plant is direct inhalation.

4.3 Who is potentially exposed?

The following potentially exposed population groups have been identified within the proposed development (i.e. the Study Area) and are considered in this HHRA:

- Residents living in apartment buildings to be constructed.
- Commercial and retail workers in buildings to be constructed.
- Visitors/patrons at retail locations to be constructed.
- People spending time (e.g. walking, jogging, playing sports, picnicking) in proposed new open space areas (i.e. Bank Street open space, Waterside Park, Miller Street Reserve) or along the Waterfront Promenade.

The people included in the HHRA are people of all ages (i.e. children, adults and the elderly). Note defensible concentration-response functions (CRFs) exist for a particular number of age groups depending on the health endpoint evaluated (Section 5.2). The age groups for which defensible CRFs were available and which have been assessed in this HHRA include all ages, ≥30 years, 1-4 years, 5-14 years, 1-14 years, 15-64 years, and ≥65 years (Section 5.2).

4.4 Summary CSM

For a complete exposure pathway to be present in the environment there must be a link between the source, a migration pathway / exposure route and an exposed population. Potentially complete exposure pathways are presented below in Table 4-1. Note that a potentially complete exposure pathway does not necessarily mean there is a risk of harm; rather it provides guidance on the data that may be required in order to confirm whether the pathway is complete and to identify people for which potential risk of adverse health effects may need to be further assessed.

Table 4-1 Conceptual Site Model Summary

Source	Pathway and Exposure Route	Exposed Populations (1)	Included in this HHRA
Vehicular emissions from vehicles travelling along the Western Distributer and other local roads	Inhalation of PM_{10}, $PM_{2.5}$, and NO_2	Residents	✓
		Commercial Workers	✓
		Retail Workers	✓
		Visitors/Patrons	✓ (2)
Emissions from existing Hymix concrete batching facility		People spending time in proposed new public open spaces (includes exercising individuals)	✓

✓ = This Source-Pathway-Receptor (SPR) linkage is considered in this HHRA, ✘ = This SPR linkage is NOT considered in this HHERA

1. Exposed populations include all age groups (see Section 4.3).
2. Not specifically assessed, but inherently any assessment for residents or workers will be conservative for visitors/patrons at the same locations.

5 Hazard Assessment

5.1 Health Effects and Health Endpoint Summary

Health effects information for PM and NO_2 is provided in Appendix A. The information has been gleaned primarily from toxicological profiles and reviews written by reputable national or international agencies and has been supplemented to some extent with recent information published since the date the reviews were written. It was beyond the scope of this assessment to undertake detailed updated literature searches and reviews. Nevertheless, the information provided and used in this HHRA is the information recommended for use in Australia during the National Environment Protection Council (NEPC) considerations to update the ambient air quality standards in Australia (e.g. Frangos and Di Marco 2013, Jalaludin and Cowie 2012, NEPC 2016, 2019a, b).

The vast majority of health effect information for exposures to PM and NO_2 in the literature stems from epidemiology studies. Epidemiology is the study of diseases in human populations. These studies examine associations or correlations between concentrations of a pollutant in ambient air and incidence of a particular health endpoint in the general population under consideration. Epidemiology is an observational science. The clear advantages of using such observational data over information generated in a laboratory are that they are based on health outcomes in real people, living in conventional environments exposed to typical concentrations of a mixture of pollutants in air. However, it must be appreciated that epidemiology is not an exact science. There can be many confounding factors in these types of studies which may affect the results, including different air pollutant measurement techniques and averaging times, different statistical treatment of data, varying individual exposures, different sources and contributions of individual pollutants within an air shed, lag times and latencies for health effects and differences in classification and recording of health status. The health outcomes are generally reported as number of deaths attributable to air pollution and/or hospitalisations / emergency department visits for particular ailments. It must be appreciated that these are very broad health outcomes and there may be a large number of societal, dietary or lifestyle factors that also affect these outcomes. Epidemiological studies attempt to account for many of the latter factors when assessing whether air pollution exposure was likely associated with a health outcome.

Epidemiological studies typically measure an association between at least one specific pollutant and health outcomes. Specific components in an ambient air mixture are usually highly correlated with one another; it is therefore often unclear what proportion of the associations are due to independent effects of a specific pollutant. This means that it is inappropriate to sum the pollutant-specific estimated health impacts in an HHRA (Frangos and Di Marco 2013).

There is a threshold concentration for PM and NO_2 below which an individual is unlikely to experience adverse health effects. However, there is a mounting amount of scientific information which indicates there is no threshold for PM (for both short- and long-term exposures) or NO_2 (for long-term exposures) at the population level. This is because a population consists of a large number of individuals with varying sensitivities, some of whom may be particularly vulnerable to the effects of PM and NO_2 exposures (e.g. the elderly, people with pre-existing medical conditions, asthmatics, etc). Assessment of population health outcomes undertaken in this HHRA has assumed no threshold for effects from either PM or NO_2 exposures. In addition, a threshold assessment has also been undertaken for short-term exposures to NO_2.

$PM_{2.5}$ and PM_{10} have been shown in numerous epidemiological studies to be associated with mortality and hospitalisation from cardiovascular and respiratory causes (NEPC 2014). The $PM_{2.5}$ fraction appears to be the most significant in relation to health outcomes. Similarly, short-term and long-term exposure to ambient NO_2 has been associated with increases in all-cause, respiratory and cardiovascular mortality, as well as increased hospital admissions and emergency department visits for respiratory disease (NEPC 2019a, WHO 2013b). An increase in symptoms in asthmatic children and increases in airway inflammation and hyper-responsiveness have also been observed (NEPC 2019a). Short-term exposure to NO_2 has also been investigated in controlled exposure studies with sensitive asthmatics; this information provides a good indication for clear LOAECs for NO_2 from short-term exposure and was used in the derivation of short-term ambient air quality standards for NO_2 (see also Section 5.3 and Appendix A).

A summary of the health outcomes considered in this HHRA for PM and NO_2 is provided below in Table 5-1. The list is based on the impact assessments conducted for PM (NEPC 2014) and NO_2 (NEPC 2019a) in Australia and the associated health risk assessment and concentration-response function (CRF) review (Frangos and Di Marco 2013, Jalaludin and Cowie 2012). CRFs are used to relate a change in an air pollutant concentration to a change in a particular health outcome. CRFs relevant for this HHRA are presented in Section 5.2.

Table 5-1 Summary of Health Outcomes Associated with Short-Term and Long-Term Exposures to $PM_{2.5}$, PM_{10} and NO_2

Health outcome	$PM_{2.5}$	PM_{10}	NO_2
Long-term (relevant averaging time)	Annual average	Annual average	Annual average
All-cause mortality 30+ years	✓	✓	✓
Cardiopulmonary mortality 30+	✓	✗	✗
Mortality ischemic heart disease 30+ years	✓	✗	✗
Mortality lung cancer 30+ years	✓	✗	✗
Life expectancy lost (YLL)	✓	✓	✗
Short-term (relevant averaging time)	24-Hour Average	24-Hour Average	1-hour
All-cause mortality (non-trauma) all ages	✓	✗	✓
Mortality cardiovascular disease- all ages	✓	✓	✓
Respiratory Mortality – all ages	✗	✗	✓
ED visits asthma 1-14 years	✓	✓	✓
Hospital admissions cardiovascular disease 15-64 years	✗	✗	✓
Hospital admissions cardiovascular disease 65+ years	✓	✗	✓
Hospital admissions cardiac disease 15-64 years	✗	✗	✓
Hospital admissions cardiac disease 65+ years	✓	✓	✓
Hospital admissions cardiac failure 65+ years	✓	✓	✓
Hospital admissions respiratory disease 1-4 years	✗	✓	✓
Hospital admissions respiratory disease 5-14 years	✗	✓	✓
Hospital admissions respiratory disease 15-64 years	✗	✗	✓
Hospital admissions respiratory disease 65+ years	✗	✗	✓
Hospital admissions pneumonia and bronchitis 65+ years	✗	✓	✗

Health outcome	$PM_{2.5}$	PM_{10}	NO_2
CRF = Concentration Response Function. ✓ = Health endpoint considered in this HHRA for which defensible CRFs are available (as per NEPC 2014, Frangos and Di Marco 2013, Jalaludin and Cowie 2012 – see Appendix A for basis), × = Health endpoint *NOT* considered in this HHRA as no defensible CRF is available (see Appendix A), Orange shading indicates health endpoints that are typically considered in economic analyses of air pollution impacts but not typically used in health risk assessments (as per NEPC 2014, Frangos and Di Marco 2013). However, in this HHRA all-cause mortality (non-trauma) for all ages has been considered.			

5.2 Summary of Concentration Response Functions (CRFs)

As part of the NEPC (2014, 2019a) review of the ambient air quality standards for PM_{10}, $PM_{2.5}$ and NO_2, Jalaludin and Cowie (2012) identified relevant defensible epidemiology studies in the general population and recommended CRFs within these studies for each pollutant for use in HHRAs (Frangos and Di Marco 2013).

The mathematical form of the CRF relationship between the change in pollutant concentration, x, and the change in population health response (e.g. incidence rate), y, depends on the functional form of the CRF from which it is derived; this depends on the underlying relationship assumed in the epidemiological study chosen to estimate a given effect (Frangos and Di Marco 2013). The recommendations made by Jalaludin and Cowie (2012) were reviewed at a Panel Meeting convened by EPA Victoria on 22 August 2012 in Melbourne. The panel reviewed and confirmed the health endpoints/outcomes and CRFs selected.

Jalaludin and Cowie (2012) and Frangos and Di Marco (2013) presented the functional forms of the CRFs as reported in the epidemiology studies (e.g. as relative risks, odds ratios, or percentage incidence/prevalence). The CRFs were converted in this HHRA into ß coefficients consistent with the approach taken by Frangos and Di Marco (2013) (see Table 5-2).

Table 5-2 Equation for conversion of Relative Risk to ß coefficient

Parameter	Symbol	Units	Summary Description
Equation 5-1 Equation for converting a RR to β			$\beta = \ln(RR) \div \Delta x$ [1]
Change in pollutant concentration	Δx	$\mu g/m^3$	Represents the incremental change in pollutant concentration as specified for CRFs in the relevant epidemiological study.
Relative Risk	RR	$(\mu g/m^3)^{-1}$	RR is a comparison of risks for two different groups that is typically expressed as a risk per unit change in pollutant concentration.
Exposure-response function	β	$(\mu g/m^3)^{-1}$	The relationship between a change in health outcome per 1 $\mu g/m^3$ change in exposure concentration for a given health outcome/endpoint.

1. Relative risk (RR) may be converted to an exposure response functions (as β) using this Equation which is reproduced from Equation 2 in Frangos and Di Marco (2013).

The ß coefficients for various health endpoints used in this HHRA are presented in Table 5-3. Refer to Appendix A for the CRFs as provided by the individual studies and study details.

Table 5-3 Concentration Response Functions (β) [1] for Various Health Outcomes

Health endpoint/outcome	<2.5 μm Particulate Matter ($PM_{2.5}$)	<10 μm Particulate Matter (PM_{10})	Nitrogen Dioxide (NO_2)
Long-term (Annual average)			
All-cause mortality 30+ years	0.006015 (0.003922 – 0.007973)*	0.003853 (0.002946 – 0.004759)	0.005354 (0.003053 – 0.007696)*
Cardiopulmonary mortality 30+	0.01310 (0.01044 – 0.0157)	-	-
Mortality ischemic heart disease 30+ years	0.02151 (0.01740 – 0.02469)	-	-
Mortality lung cancer 30+ years	0.01310 (0.005827 – 0.0116)	-	-
Short-term (24-Hour Average)			Short-term (1-hour Average)
All-cause mortality (non-trauma) – all ages	0.00237 (0.000529 – 0.004199)	-	
All-cause mortality (non-trauma) 30+ years	-	-	0.000997 (0.000177 – 0.001864)
Mortality cardiovascular disease- all ages	0.003939 (0.001845 – 0.006016)	0.002369 (0.000794 – 0.003925)	0.000939 (0.000236 – 0.001634)
Respiratory Mortality – all ages	-	-	0.002264 (0.000354 – 0.004224)
ED visits asthma 1-14 years	0.001479 (0.000953 – 0.001898)	0.001829 (0.001048 – 0.002606)	0.000611 (0.000334 – 0.000887)
Hospital admissions cardiovascular disease 15-64 years	-	-	0.000764 (0.000177 – 0.001346)
Hospital admissions cardiovascular disease 65+ years	0.003417 (0.001583 – 0.005239)	-	0.001519 (0.001056 – 0.001921)
Hospital admissions cardiovascular disease – all ages	-	0.001213 (0.000409 – 0.00201)	-
Hospital admissions cardiac disease 15-64 years	-	-	0.000706 (0 – 0.001403)
Hospital admissions cardiac disease 65+ years	0.004979 (0.002632 – 0.007048)	0.001846 (0.000662 – 0.00289)	0.001921 (0.001403 – 0.002491)
Hospital admissions cardiac failure 65+ years	0.009356 (0.00472 – 0.013913)	0.004697 (0.00263 – 0.006732)	0.004279 (0.003056 – 0.005478)
Hospital admissions respiratory disease 1-4 years	-	0.00302 (0.00119 – 0.004953)	0.002093 (0.000881 – 0.00328)
Hospital admissions respiratory disease 5-14 years	-	0.00302 (0.000265 – 0.005718)	0.002321 (0.000647 – 0.004059)
Hospital admissions respiratory disease 15-64 years	-	-	0.000939 (0.000295 – 0.001634)
Hospital admissions pneumonia and bronchitis 65+ years	-	0.001321 (0.000265 – 0.004953)	0.00085 (0.000319 – 0.001381)

(1) Average β function provided. The Lower and Upper Confidence Interval (CI) for β are provided in brackets. ED = Emergency Department; PM = Particulate Matter. * = Non-trauma.

5.3 Low Observed Adverse Effect Concentration for NO_2

As discussed in Section 3, the type of health impact assessment undertaken in this HHRA assumes a non-threshold concentration response relationship for all three pollutants is appropriate to describe effects at a population level. Whilst this assumption is commonly applied in health impact assessments, it may not be entirely appropriate for consideration of short-term health effects from exposure to NO_2 to individuals. Therefore, for NO_2 the potential for short-term health effects to eventuate in individuals has also been assessed by comparing modelled 1-hour average NO_2 concentrations with a Low Observed Adverse Effect Concentration (LOAEC) from controlled human exposure studies in sensitive asthmatic individuals. These exposure studies provide the key evidence that short-term NO_2 exposure independently can trigger an asthma attack (US EPA 2016). Asthmatic individuals are considered a sensitive sub-population since, in the general population, airway responsiveness is lognormally distributed with individuals having asthma generally being more responsive than healthy age-matched controls (Brown 2015). Allergic inflammation and airway responsiveness are hallmark measures of asthma attacks, which supports the epidemiological findings of an association between NO_2 exposure and increased emergency department visits for asthma.

Brown (2015) recently undertook a meta-analysis of data from controlled human exposures studies investigating the effect of NO_2 on airway responsiveness in individuals with asthma. Although not well understood, several mechanisms have been proposed by which NO_2 exposure could lead to increases in airway responsiveness; these may be neurally mediated or via histamine release from mast cells.

The following information summarises the principal findings from the meta-analysis by Brown (2015):

- Exposure concentrations tested in the included studies ranged from 100 ppb (i.e. 188 µg/m^3) for 60 minutes to 600 ppb (i.e. 1,128 µg/m^3) for 120 minutes.
- The general tendency of most studies was toward increased airway responsiveness following NO_2 exposure by individuals with asthma with some studies reaching statistical significance. Fewer studies showed no effect or a tendency for decreased airway responsiveness following NO_2 exposure.
- Statistically significant increases in non-specific airway responsiveness following resting NO_2 exposures were observed across all exposure concentrations tested.
- Increases in airway responsiveness were not observed following the exercising exposures to NO_2.
- No significant effects of NO_2 exposure were observed on airway responsiveness to allergen challenge, except at NO_2 concentrations over 300 ppb (i.e. 564 µg/m^3).
- About 70% of asthmatic individuals in the studies included in the meta-analysis had an increase in non-specific airway responsiveness following 30-minute exposures to NO_2 in the range of 200-300 ppb (i.e. 376-564 µg/m^3) and following 60-minute exposures to 100 ppb (i.e. 188 µg/m^3). About a quarter (25%) of the exposed individuals experienced a clinically relevant, doubling dose reduction in their provocative dose due to NO_2, i.e. commonly the provocative dose is the dose of a challenge agent required to produce a 20% reduction in forced expiratory volume in 1 second or a 100% increase in specific airway resistance.

These data indicate a LOAEC of approximately 100 ppb (i.e. 188 µg/m^3) for 1-hour exposures to NO_2 for a clinically significant increased airway responsiveness in asthmatic individuals is defensible. Increased airway responsiveness has been associated with increased severity and longer duration of asthma exacerbation (Weiss et al. 2000).

6 Exposure Assessment

6.1 People who may be exposed

As indicated in Section 4.3, population groups who may be exposed to PM and NO_2 within the proposed development (i.e. the Study Area) and are considered in this HHRA include:

- Residents living in apartment buildings to be constructed.
- Commercial and retail workers in buildings to be constructed.
- Visitors/patrons at retail locations to be constructed (these individuals are inherently included in the assessments undertaken for residents and workers).
- People spending time (e.g. walking, jogging, playing sports, picnicking) in proposed new open space areas (i.e. Bank Street open space, Waterside Park, Miller Street Reserve) or along the Waterfront Promenade.

6.2 Locations where exposure may occur

As indicated previously, the rezoning of the Study Area itself does not contribute to an increase in emissions of PM and NO_2 however peoples' exposures to these air pollutants may theoretically change as a result of people spending more time in the Study Area instead of elsewhere. As discussed in Section 2.5.1, a large number of locations were included in the air quality investigation (SLR 2021), i.e. a total number of 2,354 modelled receptor locations for Scenario 1 (Without Hymix) and 1,987 receptor locations for Scenario 2 (With Hymix) (Figure 6-1), including residential, commercial, retail, roof, boundary (i.e. of the Study Area) and grid-based receptors. Figure 6-1 shows the receptor locations, where the blue shading indicates buildings proposed to be erected as part of the development.

In order to evaluate any potential change in people's exposure circumstances if they were to spend time in the Study Area, a number of additional existing locations south, east and north of the Study Area were selected to represent existing exposures to PM and NO_2 by people already inhabiting the area. The underlying logic is that the existing air quality would remain relatively unchanged by the proposed development, and baseline incidence of health outcomes for the population living in this area is already linked to existing air quality (as well as a number of other factors, see Section 2.4.1). Modelling of PM and NO_2 exposure concentrations at these locations (denoted 'baseline receptors' in Figure 6-1) was undertaken to represent 'baseline' exposure at a number of elevations (i.e. 1m, 4m, 7m, and 10m) to inform potential exposure to air emissions at existing residences at height (e.g. in apartment buildings) for comparison with residential / commercial / retail / recreational exposure within the proposed development.

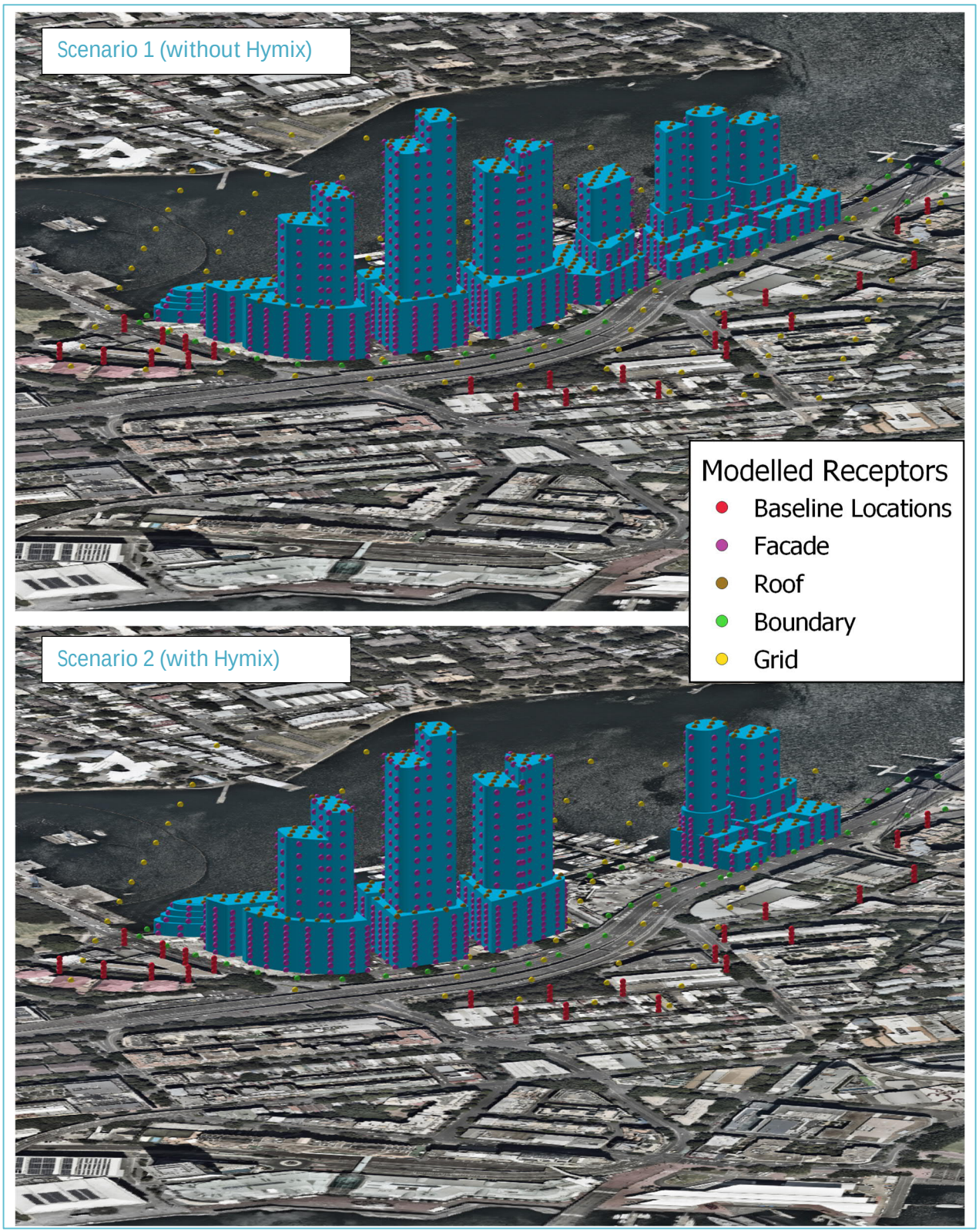


Figure 6-1 Modelled receptor locations

6.3 Scenarios evaluated

The AQIA and HHRA herein have been undertaken for the following two scenarios:

- Scenario 1: Redevelopment of the entire Study Area. This scenario assumes the Hymix concrete batch plant is no longer operational, and two buildings ('PLO 03 – Hymix 1' and 'PLO 03 – Hymix 2') are constructed where Hymix is currently located.
- Scenario 2: Partial redevelopment of the Study Area with the Hymix concrete batching facility remaining in place.

6.4 Exposure equations

6.4.1 Incidence Equation for the Population Health Endpoint/Outcome Assessment

The change in the annual incidence for a given health endpoint (Δy) can be expressed as a function of three factors; i) the change in pollutant concentration (Δx), ii) baseline incidence rate of a disease (y_o) and a concentration-response function (CRF), refer to Equation 6-1 (NEPC 2019b) shown in the table below. CRF is the estimated relationship between ambient air concentrations (x) and a health endpoint of interest (y) that is typically derived from epidemiological studies in people. There are a variety of CRF that are derived however the two most common types are the Relative Risk (RR) and the Odds Ratio (OR). The RR is the most common CRF available for $PM_{2.5}$, PM_{10} and NO_2. CRFs used in this HHRA are summarised in Appendix A.

The RR (or OR) may be converted to an exposure-response function (β) using the natural log as shown by Equation 6-2 (Frangos and Di Marco 2013, USEPA 2018, NEPC 2019b, NEPC 2002). The change in the incidence of a given health endpoint (Δy) may then be estimated based on CRF derived using a modified log-linear equation based on β (see Equation 6-3). This is the same equation used by Frangos and Di Marco (2013)[4]. These equations and others for estimating Δy are outlined in the environmental Benefits Mapping and Analysis Program (BenMap)[5] user manual (USEPA 2018).

[4] Health risk assessments by Frangos and Di Marco (2013) and NEPC (2019b) were prepared to support impact statements prepared by the National Environment Protection Council (NEPC) for particulate matter (NEPC 2014) and nitrogen dioxide (NEPC 2019a).

[5] BenMAP is a tool used to answer one of two types of questions i) What are the human health and economic benefits associated with a policy improving air quality? And ii) What is the human health burden attributable to total air pollution levels? (USEPA 2018)

Table 6-1 Equations Used for Estimating a Change in Health Outcome

Parameter	Symbol	Units	Summary Description
Equation 6-1 Generic equation expressed using CRF			$\Delta y = f(\Delta x, y_o, CRF)$ (1)
Equation 6-2 Equation for converting a RR to β function			$\beta = \ln(RR) \div \Delta x$ (2)
Equation 6-3 Modified log-linear equation expressed using β			$\Delta y = y_o \times \left(e^{\beta \times \Delta x} - 1\right)$ (3)
Change in health outcome	Δy	Incidence/yr	The change in annual incidence for a given health endpoint/outcome in a population
Change in pollutant concentration	Δx	µg/m^3	Difference in before and after modelled ground level (or at specific elevations) air concentrations (1-hour, 24-hour or annual values) at select locations. In this HHRA, this is the difference from modelled concentrations at 'baseline' locations. See Appendix B for individual values.
Baseline incidence rate	y_o	Incidence/yr	The baseline health incidence rate in a relevant population per year for a given health endpoint/outcome. Summarised in Section 2.4.2.
Concentration-response function	CRF	(µg/m^3)$^{-1}$	A relationship estimated between ambient concentrations and health endpoints of interest. CRFs used in this HHRA are summarised in Appendix A.
Risk Ratio	RR	(µg/m^3)$^{-1}$	RR is a comparison of risks for two different groups that is typically expressed as a risk per unit change in pollutant concentration.
Exposure-response function	β	(µg/m^3)$^{-1}$	The relationship between a change in health outcome per 1 µg/m^3 change in air concentration for a given health endpoint/outcome. ß functions used in this HHRA are summarised in Section 5.2.

1. Generic equation to estimate a change in health outcome (Δy) as quoted in the risk assessments used to support the impact statements for NO_2 (NEPC 2019a, 2019b).
2. Relative risk (RR) may be converted to an exposure response functions (as β) using Equation 6-2 (USEPA 2018, Frangos and Di Marco 2013, NEPC 2019b, NEPC 2002).
3. Generic equation to estimate a change in health outcome (Δy) using exposure response functions (β) (refer to the health risk assessment to support the impact statement for particulate matter, (Frangos and Di Marco 2013) used for log-linear models (USEPA 2018).

As discussed in Section 5.1, population health endpoints/outcomes for different pollutants (including $PM_{2.5}$, PM_{10} and NO_2) are often correlated therefore the estimates of Δy are not typically added together as this may result in double counting (WHO 2013b, EEA 2018). This means that single pollutant models (used to estimate CRFs) will invariably include effects from a pollutant that could be (in part) attributed to another pollutant.

6.4.2 Comparison with LOAEC for NO_2

In addition to the estimation of potential changes in population incidence of health outcomes, for NO_2 a complementary risk assessment approach has been undertaken to estimate the potential risk of asthma exacerbation in individuals, rather than the population as a whole. This was done by comparing cumulative modelled 1-hour average NO_2 concentrations (99.9th, 99.5th, and 97th percentiles) with the LOAEC at each modelled location within the Study Area. Thus, no exposure equations were required for this. The percentiles used in the HHRA can be put into context as follows:

- 99.9th: This concentration of NO_2 may occur for approximately 9 out of the 8,760 hours in a year (i.e. 0.1% of the time).
- 99.5th: This concentration of NO_2 may occur for approximately 44 out of the 8,760 hours in a year (i.e. 0.5% of the time).

- 97th: This concentration of NO_2 may occur for approximately 263 out of the 8,760 hours in a year (i.e. 3% of the time).

6.5 Exposure concentrations

Baseline health incidence data are reported as an annual incidence rate per population of 100,000 people. For PM, the averaging times used to evaluate long-term and short-term population health outcomes are annual averages and 24-hour averages, respectively. For NO_2, the averaging times used are annual averages and 1-hour averages, respectively.

While theoretically it may be possible to measure daily incidence of particular health outcomes related to short-term exposures in large populations, these effects are not generally measurable in smaller communities. Instead the calculations relate to a measurable parameter, i.e. annual incidence of mortality, hospital admissions or emergency department visits in a population. Thus, the change in modelled air concentration (from 'baseline') needs to be averaged over a year in order to relate this to a change in population annual incidence of a health endpoint/outcome. It is important to remember that these types of assessments do not inform on the health risk to a particular individual but rather a whole population living in the area.

For health outcomes related to short-term (i.e. 24-hour) PM_{10} and $PM_{2.5}$ exposure, this is achieved by estimating the average of all 24-hour averages over the course of a year. This is the same value as the 'annual average' concentration. This approach results in the same value mathematically as calculating daily change in health incidence at each modelled location over every 24-hour period of the year and then summing the daily incidences to get an annual change in incidence. The simplified calculation method is that recommended by the World Health Organization (Ostro 2004).

For assessment of population incidence changes in health outcomes related to short-term NO_2 exposures (i.e. 1-hour averages), the approach utilised by Frangos and Di Marco (2013) has been used in this HHRA. This involved taking the 1-hour maximum NO_2 concentration from every day of the year and averaging these values over the year. This effectively provides an average daily maximum 1-hour NO_2 concentration, which can then be related to annual baseline incidence data for a particular health endpoint. The relevant incremental modelled concentrations are provided for each receptor location and scenario in Appendix B.

Modelled annual average incremental[6] PM and NO_2 and average daily maximum 1-hour NO_2 concentrations for the 'baseline' receptors were grouped into those located south (n=8), southeast (n=6), east (n=5) and north (n=4) of the Study Area, and also grouped by elevation (1, 4, 7, or 10m). It is evident from the data there is a clear difference between average concentrations by elevation (i.e. concentrations decrease with increasing elevation) but no significant difference between areas was found (Kruskall-Wallis, $p>0.05$) (Appendix C). Therefore, the data were combined into an overall average concentration for all 'baseline' locations (n=23) at each elevation (see Table 6-2). For calculation of an incremental change in exposure concentration (i.e. Δx) relative to 'baseline' locations, modelled data within the Study Area were compared as follows:

- Predictions at ground level (i.e. 1m) were compared with 'baseline' concentrations at 1m.
- Predictions at 6m were compared with 'baseline' concentrations at 4m.
- Predictions at ≥ 10m were compared with 'baseline' concentrations at 10m.

[6] For the population health impact assessment, it is only the change in exposure that is important. As the background concentration of each pollutant was assumed to be the same at every location including at 'baseline' locations (SLR 2021), this does not influence the calculation of change in exposure. Therefore, incremental concentrations, rather than cumulative concentrations, were used.

It is noted there were no predictions generated within the Study Area for elevations between 1 and 6m or between 6 and 10m.

Table 6-2 Modelled incremental concentrations [(1)] of PM_{10}, $PM_{2.5}$ and NO_2 at 'baseline' locations

Elevation	Modelled incremental concentration (µg/m^3)					
	Scenario 1 (No Hymix)			Scenario 2 (with Hymix)		
	$PM_{2.5}$	PM_{10}	NO_2	$PM_{2.5}$	PM_{10}	NO_2
Annual average						
1m	3.9 ± 2.3	3.8 ± 2.5	25 ± 10	3.9 ± 2.3	4.1 ± 2.3	24 ± 10
4m	3.1 ± 1.6	3.2 ± 1.9	23 ± 9	3.1 ± 1.7	3.5 ± 1.7	22 ± 9
7m	2.7 ± 1.3	2.8 ± 1.5	22 ± 8	2.7 ± 1.3	3.0 ± 1.4	21 ± 8
10m	2.3 ± 1.0	2.4 ± 1.1	20 ± 7	2.3 ± 0.9	2.6 ± 1.0	20 ± 7
Average Daily 1-hour Maximum						
1m	-	-	61 ± 13	-	-	60 ± 12
4m	-	-	56 ± 10	-	-	55 ± 10
7m	-	-	53 ± 9	-	-	52 ± 9
10m	-	-	51 ± 8	-	-	50 ± 7.5

- This averaging time is not relevant to PM.

1. Concentrations are overall averages ± standard deviations for all 'baseline' locations (n=23).

For the complementary LOAEC assessment undertaken for NO_2, three percentiles (the 99.9th, 99.5th, and 97th) of 1-hour NO_2 concentrations were calculated for each location within the Study Area. These data are in Appendix B.

7 Risk characterisation

7.1 What Level of Risk is Acceptable?

All aspects of life involve a certain level of risk. What society considers to be an 'acceptable' level of risk varies due to numerous elements such as type of activity, familiarity/unfamiliarity, number of people who may be affected, severity of potential outcome, reversibility of outcome, voluntariness of risk, individual perceptions, social factors, advantages/benefits of the activity, ethics and equity (Hergon et al. 2004).

For societal risk, NSW Government (2011) suggested criteria for what should be considered an 'acceptable' risk incorporate the ALARP (As Low As Reasonably Possible) approach. The limit of risk to a member of the public was considered tolerable above 1 in 10,000 and acceptable above 1 in 1,000,000. In the tolerable region, the risk was accepted only if a benefit is desired (NSW Government 2011). In 2017, both the NSW EPA (2017) and NSW Government (2017) issued guidance on risk levels which roughly follows the societal risk levels suggested in 2011 (NSW Government 2011). A summary of these risk levels is provided below in Table 7-1.

Table 7-1 Risk Level for Pollutants

<table>
<tr><th>Risk Level</th><th>NSW Government 2011 (Societal Risk)</th><th>NEPC 2011 (AQS)</th><th>NSW EPA 2016</th><th>NSW Health 2017 (2)</th></tr>
<tr><td><1 in 1,000,000</td><td>Acceptable</td><td rowspan="2">Acceptable (1)</td><td>Acceptable</td><td>Negligible</td></tr>
<tr><td>> 1 in 1,000,000 – <1 in 100,000</td><td rowspan="2">Tolerable (based on ALARP approach) (1)</td><td rowspan="2">Requires best practice</td><td>Low</td></tr>
<tr><td>>1 in 100,000 – <1 in 10,000</td><td rowspan="2">Unacceptable</td><td>Moderate</td></tr>
<tr><td>>1 in 10,000</td><td>Unacceptable</td><td>Unacceptable</td><td>High</td></tr>
<tr><td colspan="5">ALARP = As low as reasonably practical, AQS = Level for setting air quality standards
1) Acceptable level for individual risk ranges from 0.5 -50 in 1,000,000 depending on land use.
2) For events no greater than 3 months.</td></tr>
</table>

Based on available guidance an unacceptable level of risk can be taken to mean a risk of 1 in 10,000 (or higher) and a tolerable risk lies somewhere between 1 in 10,000 and less than 1 in 1,000,000 assuming that best practice or the ALARP approach is demonstrated, particularly where a project provides benefits to the community.

Criteria for the assessment of incremental impacts of long-term $PM_{2.5}$ exposure on increased risk of mortality have been derived by Capon and Wright (2019), refer to Table 7-2. The acceptability of the population risk of this effect and suggested interpretation by Capon and Wright (2019) have also been used in this HHRA for all pollutants when interpreting Δy (or change in incidence of population health outcomes). It is noted the suggested interpretation of reducing pollutant load is not directly applicable to this HHRA, since the proposed development is not the source of pollutants *per se*. Instead, this can be interpreted as a level of estimated risk (of increased incidence of health outcomes) at which exposure reduction measures may need to be considered.

It should be reiterated that the level of risk discussed in this section relates to changes in population incidence of a particular health outcome, rather than an individual level of risk. Thus, the emphasis of the health impact assessment undertaken in this report is on the overall net effect of the proposed development as a whole, rather than individual locations. Nevertheless, information is provided for individual locations to gain an appreciation of locations where exposure management measures may be most effective at reducing overall net health risk.

Table 7-2 Suggested interpretation of risk acceptability for $PM_{2.5}$ exposure and risk of increased mortality (Capon and Wright 2019).

Risk Level - Increased risk of mortality	Risk acceptability and suggested interpretation
<1 in 1,000,000	Negligible.
1 in 1,000,000 to 1 in 100,000	Acceptable: Development needs to show use of best practice with consideration of reasonable and feasible measures to reduce pollutant load (or in this case, reduce exposure).
>1 in 100,000 to 1 in 10,000	Tolerable: Only if best practice is proven and reasonable, and feasible measures have been demonstrated. At this level, costly interventions are now considered reasonable and feasible, that would not have been in the acceptable range.
>1 in 10,000	Unacceptable.

7.2 Health Endpoint Assessment

7.2.1 Long-term Exposures

7.2.1.1 Health benefit vs. detriment

The overall estimated change from baseline incidence of mortality (for people >30 years from all-causes) as a result of the change in exposure which may be experienced by people living, working or spending time in the Study Area is shown in Table 7-3. The table shows the number of locations in each category (i.e. residential, commercial, retail, roof, grid, and boundary) and summary statistics for the change from baseline incidence for Scenario 1 (without Hymix) and Scenario 2 (with Hymix). A negative change signifies a net health benefit to the population, i.e. a reduction in the incidence of the particular health outcome for every 100,000 people living in the area. For example, for $PM_{2.5}$ the HHRA has estimated in Scenario 1 (without Hymix) for the population living in the area an overall average net reduction in risk of all-cause mortality for people >30 years of age ranging from 10 to 18 less deaths per 100,000 people. A positive change would signify a net health detriment for a particular endpoint.

As can be seen from Table 7-3, for all-cause mortality in people >30 years of age, the altered exposure circumstances of the proposed development are predicted to provide an average net health benefit at each category of locations. This means that on average lower population exposures to the pollutants considered in this HHRA are anticipated to occur within the totality of the Study Area relative to other residences and commercial properties south, southeast, and north of the Study Area. The exceptions to this are PM_{10} exposures at Study Area boundary locations for Scenario 2 (average increase of ~2 per population of 100,000 people) and NO_2 exposures at Study Area boundary locations for Scenario 1 (average increase of ~1.5 per population of 100,000 people). The same finding of an average net health benefit to the population living or working within the Study Area (relative to baseline conditions) was also found for the other health endpoints evaluated for long-term exposures to $PM_{2.5}$ (i.e. for people >30 years mortality due to cardiopulmonary causes, ischaemic heart disease, and lung cancer). The results of these calculations can be found in Appendix B.

Table 7-3 Summary statistics for estimated change in all-cause mortality (non-trauma) for people >30 years (rate per 100,000 people in local population)

Pollutant	Average (1st quartile, 3rd quartile)					
	Residential	Commercial	Retail	Roof	Grid	Boundary
# of locations (Scenario 1)	721	845	213	276	238	61
# of locations (Scenario 2)	629	706	149	204	238	61
$PM_{2.5}$						
Scenario 1	-12.7 (-13, -12.4)	-9.5 (-12.4, -8.42)	-12.2 (-17.6, -9.91)	-11.5 (-13, -11.9)	-17.9 (-19.8, -18.2)	-11.6 (-17.6, -9.35)
Scenario 2	-11.8 (-12.8, -12.2)	-5.8 (-8.38, -2.82)	-5.09 (-7.14, 1.79)	-10.5 (-12.2, -10.6)	-13.6 (-16, -12.1)	-3.63 (-5.47, 2.91)
PM_{10}						
Scenario 1	-8.2 (-8.72, -8.37)	-3.1 (-7.66, -1.59)	-1.8 (-8.66, 0.82)	-6.5 (-8.72, -6.94)	-8.8 (-11.9, -9.17)	-0.26 (-9.2, 2.6)
Scenario 2	-8.6 (-9.42, -9.07)	-4.1 (-8.36, -1.95)	-3.05 (-10, 1.73)	-7.7 (-9.42, -7.65)	-7.8 (-12.5, -6.85)	1.95 (-6.85, 4.97)
NO_2						
Scenario 1	-87.5 (-96.2, -87.2)	-35.6 (-73.6, -11.8)	-16 (-64.3, 11.7)	-72.1 (-96.2, -69)	-59.8 (-83.9, -51.6)	1.46 (-51.6, 37.3)
Scenario 2	-84.5 (-94.2, -85.2)	-38.6 (-76.2, -14.6)	-17.6 (-59.5, 13.5)	-76.2 (-94.2, -71.6)	-60.2 (-82.3, -49.9)	-13.7 (-45.2, 18.6)

Bolded values represent an estimated increase in all-cause population mortality incidence. Bolded values in red indicate instances where the 75th percentile (i.e. upper quartile) of data is an estimated increase (from baseline incidence) of more than 10 in 100,000 people (i.e. in accordance with the criteria outlined in Section 7.1, this would be considered to be an unacceptable increase). However, it is the average statistic in this table that best represents the net overall effect on population incidence. All average estimated changes are considered to be acceptable; indeed, most average changes are negative indicating an overall net health benefit of the proposed development on population health outcomes.

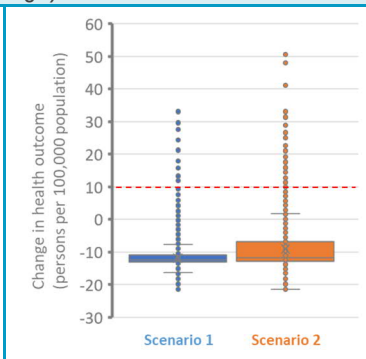
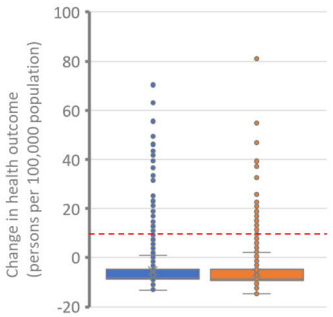
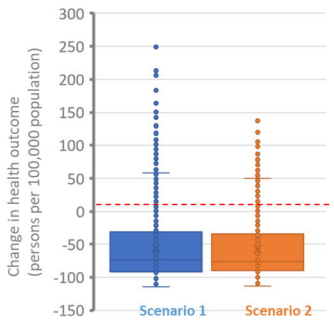
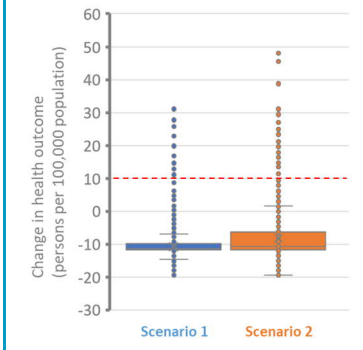
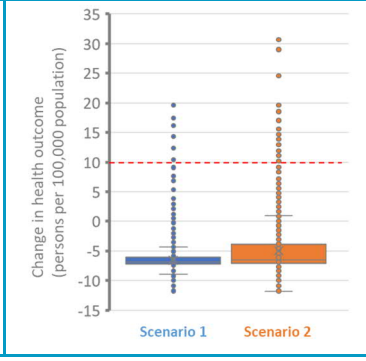
7.2.1.2 Comparison by scenario

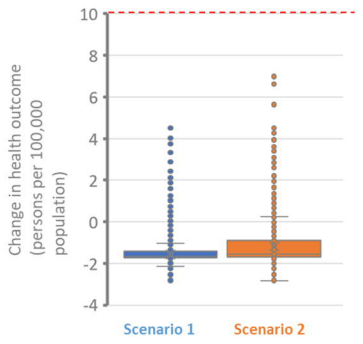
Table 7-4 compares estimated changes in population incidence for health outcomes related to long-term exposures to $PM_{2.5}$, PM_{10} and NO_2 for Scenario 1 (without Hymix) with Scenario 2 (with Hymix). Box and whisker plots show the following data:

- The ends of the box are the upper and lower quartiles (so the box spans the interquartile range).
- The median is marked by a horizontal line inside the box, the average by an 'x' in the box.
- The minimum and maximum are marked by small horizontal black lines above and below the box.
- Outliers are shown as dots above and below the minimum and maximum of the distribution.

The box and whisker plots in Table 7-4 show that for $PM_{2.5}$ exposures, the upper quartiles (Quartile 3) are higher for Scenario 2 than for Scenario 1, indicating the estimated change in population health outcomes from long-term exposures to $PM_{2.5}$ within the Study Area are slightly better (i.e. provide higher benefit) for Scenario 1 (without Hymix) than Scenario 2 (with Hymix), albeit the proposed redevelopment in both Scenarios provides a net overall benefit. The difference in population health outcomes for PM_{10} and NO_2 long-term exposures between Scenarios 1 and 2 are negligible (Table 7-4).

Table 7-4 Box and whisker plots showing estimated change in population incidence for long-term health outcomes for Scenario 1 vs. Scenario 2

Health endpoint/outcome	<2.5 µm Particulate Matter ($PM_{2.5}$)	<10 µm Particulate Matter (PM_{10})	Nitrogen Dioxide (NO_2)
Long-term (Annual average)			
All-cause mortality 30+ years			
Cardiopulmonary mortality 30+ years			
Mortality ischemic heart disease 30+ years			

Health endpoint/outcome	<2.5 µm Particulate Matter ($PM_{2.5}$)	<10 µm Particulate Matter (PM_{10})	Nitrogen Dioxide (NO_2)
Mortality lung cancer 30+ years			

The ends of the box are the upper and lower quartiles (so the box spans the interquartile range); the median is marked by a horizontal line inside the box; the average by an 'x' in the box; the minimum and maximum are marked by small horizontal black lines above and below the box; outliers are shown as dots above and below the minimum and maximum of the distribution.

The red dotted line indicates an estimated increase (from baseline incidence) of more than 10 in 100,000 people (i.e. in accordance with the criteria outlined in Section 7.1, this would be considered to be an unacceptable increase). However, it is the median or average statistic in this table that best represents the net overall effect on population incidence. All average estimated changes are considered to be acceptable; indeed, most average changes are negative and indicate an overall net health benefit of the proposed development on population health outcomes. See Appendix B for calculation spreadsheets.

7.2.1.3 Comparison by Building

Figure 7-1 shows the estimated change in all-cause mortality (non-trauma, >30 years) from baseline incidence for all modelled receptor locations and for each pollutant. The figures show colour-coded locations based on which proposed building they correspond to, or whether they are grid or boundary receptors. As has been discussed above, the figures clearly show for the vast number of modelled locations the proposed development results in a net health benefit with respect to population health outcomes. There are a number of locations at which, when modelled individually, from a societal perspective, an unacceptable increase in mortality from long-term exposures may result. This should be interpreted as follows: if a large population of people were to live in that one receptor location, then there may be an unacceptable increase from baseline incidence in mortality from all-causes. Clearly a large population could never live in the one location. This unacceptability at a few locations is more than balanced out by the numerous locations where there is an overall net health benefit (i.e. reduced exposure, and therefore reduced population mortality). For an individual living at a particular location, a >10 in 100,000 population increased risk in mortality from baseline would equate to a 0.01% increase in risk of mortality for an individual.

Nevertheless, examining the locations which are estimated to be associated with >10 in 100,000 population increase from baseline can help provide an indication where management measures in building design could be considered.

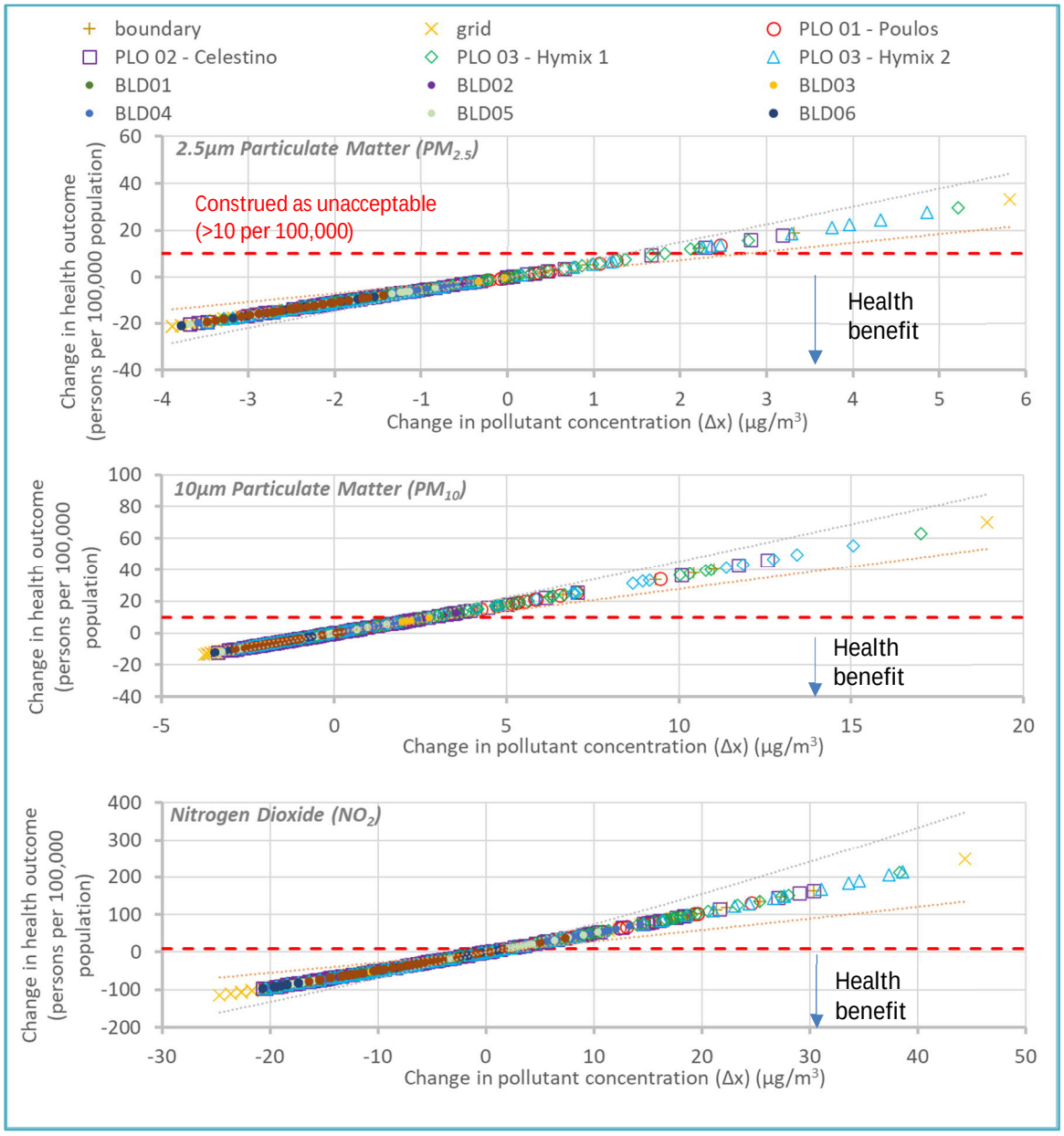


Figure 7-1 Estimated change in all-cause mortality (persons >30 years per 100,000 population) from baseline incidence from long-term exposure to $PM_{2.5}$, PM_{10} and NO_2 by building.
Fine dotted lines above and below individual location markers indicate the estimated change in health outcome plotted using the CRF upper and lower confidence intervals.

Instances where individual modelled locations were associated with >10 in 100,000 population increases (when using the mean CRF) in all-cause mortality from baseline are summarised in Table 7-5, Figure 7-2, and Figure 7-3.

Table 7-5 Instances where individual modelled locations were associated with >10 in 100,000 population increases in all-cause mortality from baseline

Building	Scenario 1 (without Hymix)				Scenario 2 (with Hymix)			
	Residential	Retail	Commercial	Podium Roof	Residential	Retail	Commercial	Podium Roof
PM_{10}								
BLD01	-	-	-	-	-	1/13 (7.7%), 1m	1/52 (1.9%), 6m	-
BLD02	-	1/16 (6.25%), 1m	-	-	-	-	-	-
BLD03	-	-	-	-	-	-	1/98 (1%), 10m	-
BLD04	-	-	-	-	-	-	-	-
BLD05	-	-	-	-	-	-	-	-
BLD06	-	-	-	-	-	-	-	-
BLD07	-	-	-	-	-	-	-	-
PLO 01 – Poulos	-	6/19 (31.6%), 1m	14/103 (13.6%), 6-20m	-	-	6/19 (31.6%), 1m	12/103 (11.7%), 6-20m	1/36 (2.8%), 20.2m
PLO 02 – Celestino	-	6/24 (25%), 1m	9/121 (7.4%), 6,10m	1/18 (5.6%), 12.6m	-	6/24 (25%), 1m	9/121 (7.4%), 6-10m	2/18 (11.1%), 12.6m
PLO 03 – Hymix 1	-	9/17 (53%), 1m	15/77 (19.5%), 6-15m	5/23 (21.7%), 12.6m	NA	NA	NA	NA
PLO 03 – Hymix 2	-	4/17 (23.5%), 1m	11/107 (10.3%), 6-20m	-	NA	NA	NA	NA
$PM_{2.5}$								
BLD01	-	-	-	-	-	-	-	-
BLD02	-	-	-	-	-	-	-	-
BLD03	-	-	-	-	-	-	4/98 (4.1%), 6-15m	-
BLD04	-	-	-	-	-	1/15 (6.7%), 1m	1/98 (1%), 10m	-
BLD05	-	-	-	-	-	1/14 (7.1%), 1m	-	-
BLD06	-	-	-	-	-	-	-	-
BLD07	-	-	-	-	-	-	-	-
PLO 01 – Poulos	-	-	1/103 (0.97%), 15-20m	-	-	6/19 (31.6%), 1m	16/103 (15.5%), 6-30m	1/36 (2.8%), 20.2m

Building	Scenario 1 (without Hymix)				Scenario 2 (with Hymix)			
	Residential	Retail	Commercial	Podium Roof	Residential	Retail	Commercial	Podium Roof
PLO 02 – Celestino	-	1/24 (4.2%), 1m	2/121 (1.7%), 6m	-	-	8/24 (33.3%), 1m	11/121 (9.1%) (6-20m)	2/18 (11.1%), 12.6m
PLO 03 – Hymix 1	-	4/17 (23.5%), 1m	1/77 (1.3%), 6m	-	NA	NA	NA	NA
PLO 03 – Hymix 2	-	1/17 (5.9%), 1m	7/107 (6.5%), 6-20m	-	NA	NA	NA	NA
NO_2								
BLD01	-	-	-	-	-	-	1/52 (1.9%), 10m	-
BLD02	2/254 (0.8%), 6,10m	3/16 (18.8%), 1m	-	-	7/247 (2.8%), 6-15m	3/16 (18.8%), 1m	-	-
BLD03	-	4/14 (28.6%), 1m	17/98 (17.3%), 6-30m	-	-	4/14 (28.6%), 1m	17/98 (17.3%), 6-30m	-
BLD04	-	6/15 (40%), 1m	19/105 (18.1%), 6-20m	-	-	6/15 (40%), 1m	20/105 (19%), 6-20m	-
BLD05	-	5/14 (35.7%), 1m	7/98 (7.1%), 6-15m	-	-	5/14 (35.7%), 1m	8/98 (8.2%), 6-15m	-
BLD06	-	-	-	-	-	1/11 (9.1%), 1m	-	-
BLD07	-	2/56 (3.6%), 1,6m	-	-	-	2/23 (8.7%), 1,6m	1/45 (2.2%), 10m	-
PLO 01 – Poulos	-	9/19 (47.4%), 1m	24/103 (23.3%), 6-30m	1/36 (2.8%), 20.2m	-	10/19 (52.6%), 1m	24/103 (23.3%), 6-30m	1/36 (2.8%), 20.2m
PLO 02 – Celestino	-	8/24 (33%), 1m	15/121 (12.4%), 6-20m	6/18 (33%), 12.6m	-	8/24 (33%), 1m	17/121 (14.1%), 6-20m	5/18 (27.8%), 12.6m
PLO 03 – Hymix 1	-	11/17 (64.7%), 1m	23/77 (29.9%), 6-30m	6/23 (26%), 12.6m	NA	NA	NA	NA
PLO 03 – Hymix 2	-	6/17 (35.3%), 1m	17/107 (15.9%), 6-20m	-	NA	NA	NA	NA

- = no instances with >10 in 100,000 population increase in all-cause mortality. NA = Not applicable (i.e. these buildings would not exist in Scenario 2).

Overall, when the comparison is made for the same buildings between Scenario 1 (without Hymix) and Scenario 2 (with Hymix), there are a similar or a larger number of locations with an estimated change in health outcome (if a population of people were to live in the one location) of >10 in 100,000 in Scenario 2 (with Hymix) than Scenario 1 (without Hymix).

It is noted:

- Most of these locations are designated to be used for commercial or retail purposes. It is recognised long-term exposures to air pollutants at these locations will not occur 24 hours per day and 7 days per week, as the assessment conservatively assumes. However, it was considered inappropriate to adjust the calculations to assume only 8 out of 24 hours exposure, since it is feasible that someone may both live and work in the proposed development. Exposure to air pollutants in general also does not cease when someone leaves work.
- There are two buildings which are only present in Scenario 1 (without Hymix). These are 'PLO 03 – Hymix 1' and 'PLO 03 – Hymix 2' which represent an additional 152 and 230 modelled locations, respectively. This includes an additional 85 modelled retail locations on the ground floor and 284 commercial locations on lower floors of both these buildings.
- The only residential locations where this applies are in BLD02 at 6 or 10 m (Scenario 1) or 6-15m (Scenario 2).

The other long-term health outcomes evaluated for $PM_{2.5}$ exposures showed similar patterns to those found for all-cause (non-trauma) mortality. These analyses can be found in Appendix B.

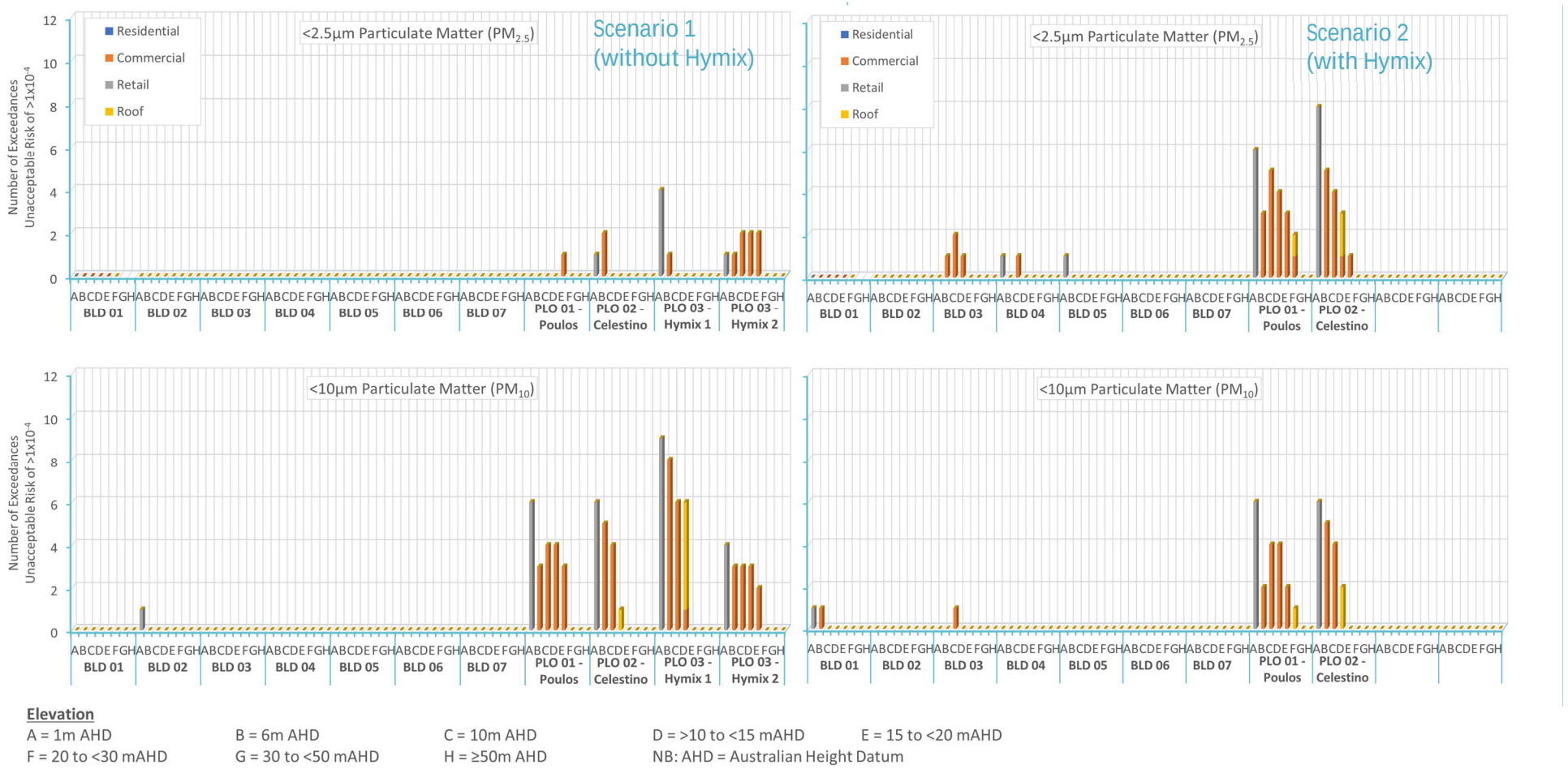


Figure 7-2 Number of modelled locations by building with an estimated change in all-cause (non-trauma) mortality (>30 years) incidence of >10 people per 100,000 population (i.e. a 0.01% increased risk) from long-term exposures to PM

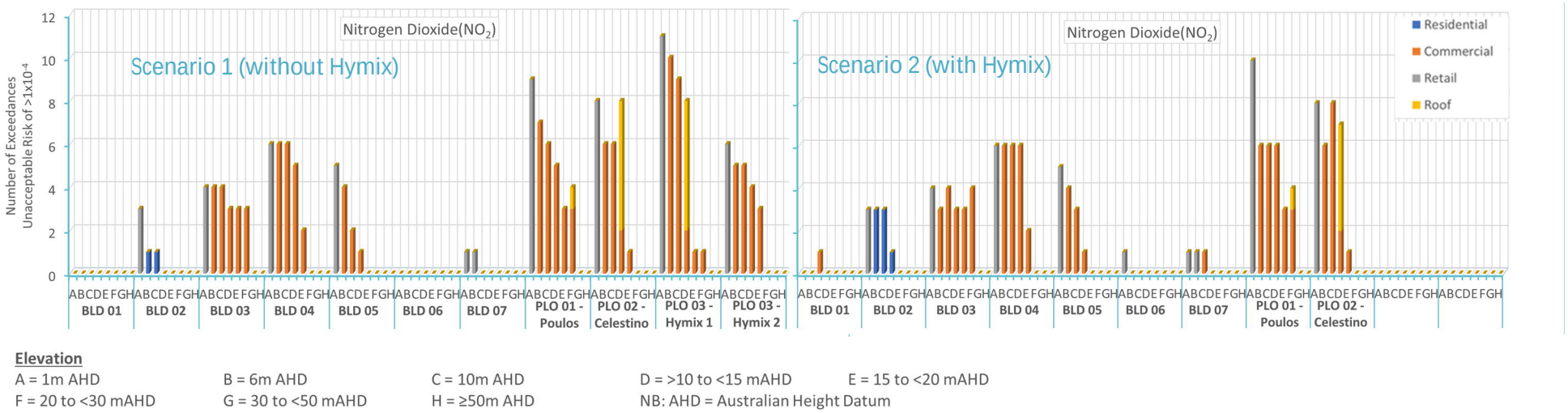


Figure 7-3 Number of modelled locations by building with an estimated change in all-cause (non-trauma) mortality (>30 years) incidence of >10 people per 100,000 population (i.e. a 0.01% increased risk) from long-term exposures to NO_2

7.2.2 Short-term Exposure

7.2.2.1 Health benefit vs. detriment

Similar to the health outcomes associated with long-term exposures to PM_{10}, $PM_{2.5}$ and NO_2, the overall estimated population change from baseline in incidence of health outcomes associated with short-term exposures to these pollutants within the Study Area is one of improvement, i.e. a net health benefit is predicted to be conferred by the proposed development. This is evident both from examining the summary statistics for a few select health outcomes in Table 7-6 as well as when comparing the box and whisker plots for each health outcome and scenario in Table 7-7. In total 13 different combinations of health outcomes and age groups were evaluated; data for all health outcomes and both scenarios are presented in Appendix B.

Table 7-6 Summary statistics for estimated change in selected health outcomes for short-term exposure (rate per 100,000 people in local population)

Pollutant	Average (1st quartile, 3rd quartile)					
	Residential	Commercial	Retail	Roof	Grid	Boundary
# of locations (Scenario 1)	721	845	213	276	238	61
# of locations (Scenario 2)	629	706	149	204	238	61
Emergency Department visits for asthma 1-14 years						
$PM_{2.5}$						
Scenario 1	-4.1 (-4.2, -4.0)	-3.1 (-4.0, -2.7)	-3.98 (-5.7, -3.2)	-3.7 (-4.2, -3.9)	-5.8 (-6.5, -5.9)	-3.8 (-5.7, -3.0)
Scenario 2	-3.83 (-4.2, -3.97)	-1.9 (-2.7, -0.9)	-1.7 (-2.3, 0.58)	-3.4 (-3.97, -3.4)	-4.4 (-5.2, -3.9)	-1.2 (-1.8, 0.9)
PM_{10}						
Scenario 1	-5.1 (-5.5, -5.2)	-1.9 (-4.8, -0.99)	-1.1 (-5.4, 0.5)	-4.1 (-5.5, -4.3)	-5.5 (-7.4, -5.7)	-0.19 (-5.7, 1.6)
Scenario 2	-5.4 (-5.9, -5.7)	-2.6 (-5.2, -1.2)	-1.9 (-6.3, 1.1)	-4.8 (-5.9, -4.8)	-4.9 (-7.8, -4.3)	1.2 (-4.3, 3.1)
NO_2						
Scenario 1	-30.4 (-36, -27.7)	-10.3 (-21.3, -2.4)	-5.8 (-17.4, 1.7)	-23 (-34.2, -18.1)	-18.2 (-24.8, -14.1)	-2.1 (-13.1, 4.5)
Scenario 2	-29.5 (-35.4, -27)	-11.1 (-22.3, -2.9)	-6.3 (-15.4, 2.8)	-24.6 (-34.4, -19.7)	-18 (-23.9, -13.5)	-5.53 (-12.6, 2.0)
Hospital Admissions cardiac disease 65+ years						
PM2.5						
Scenario 1	-70.1 (-71.9, -68.8)	-52.6 (-68.8, -46.6)	-67.8 (-97.5, -54.8)	-63.6 (-71.9, -65.8)	-99 (-110, -101)	-64.1 (-97.5, -51.7)
Scenario 2	-66.3 (-71.9, -68.8)	-32.9 (-46.6, -16.6)	-28.2 (-39.5, 9.9)	-59.4 (-68.8, -59.6)	-75.3 (-88.4, -67)	-20.1 (-30.2, 16.1)
PM_{10}						
Scenario 1	-26.1 (-28, -26.8)	-9.9 (-24.5, -5.1)	-5.88 (-27.8, 2.6)	-20.8 (-28, -22.3)	-28.3 (-38.2, -29.4)	-0.96 (-29.4, 8.4)

Pollutant	Average (1st quartile, 3rd quartile)					
	Residential	Commercial	Retail	Roof	Grid	Boundary
Scenario 2	-25.4 (-28, -26.8)	-10.8 (-24.5, -3.77)	-5.96 (-28.3, 9.5)	-22.4 (-28, -22.3)	-21.1 (-36.2, -18)	10 (-18, 19.8)
NO_2						
Scenario 1	-473 (-557, -433)	-161 (-334, -38.8)	-89.4 (-274, 27.8)	-357 (-530, -284)	-284 (-387, -223)	-30.4 (-207, 72.4)
Scenario 2	-468 (-558, -431)	-184 (-359, -55.6)	-108 (-253, 34.9)	-393 (-543, -319)	-291 (-383, -223)	-96.7 (-208, 22.5)

Bolded values represent an estimated increase in the population incidence of the health outcome cited. Bolded values in red indicate instances where the 75th percentile (i.e. upper quartile) of data is an estimated increase (from baseline incidence) of more than 10 in 100,000 people (i.e. in accordance with the criteria outlined in Section 7.1, this would be considered to be an unacceptable increase). However, it is the average statistic in this table that best represents the net overall effect on population incidence. All average estimated changes are considered to be acceptable; indeed, most average changes are negative indicating an overall net health benefit of the proposed development on population health outcomes.

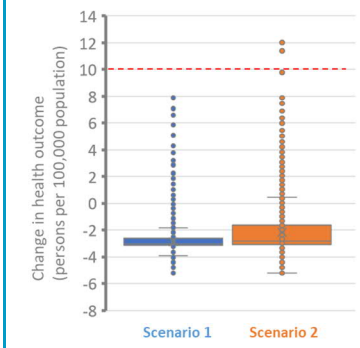
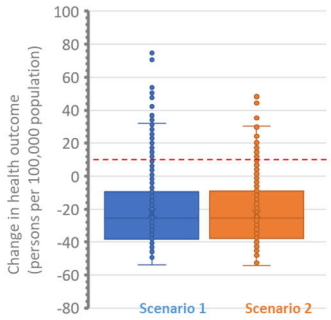
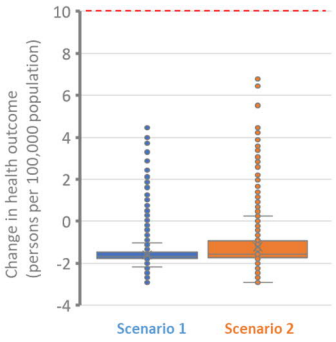
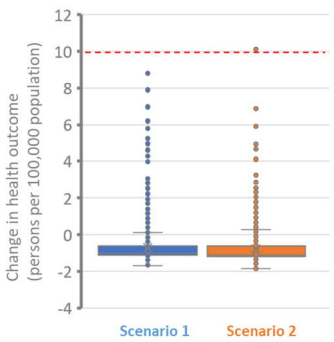
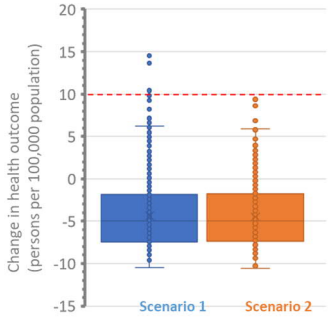
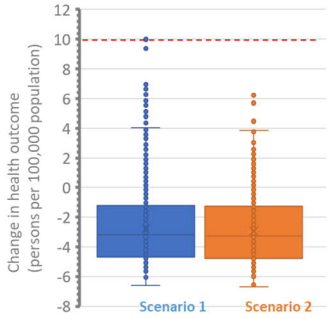
7.2.2.2 Comparison by Scenario

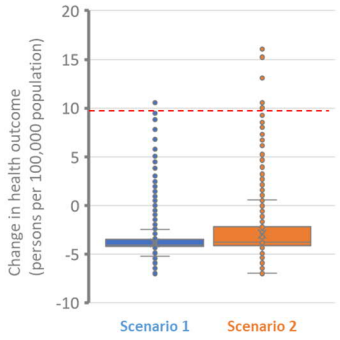
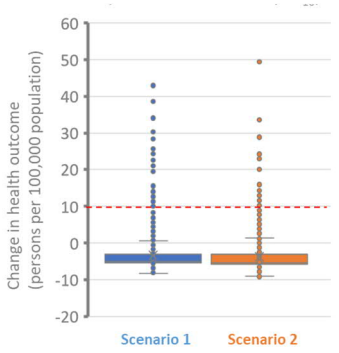
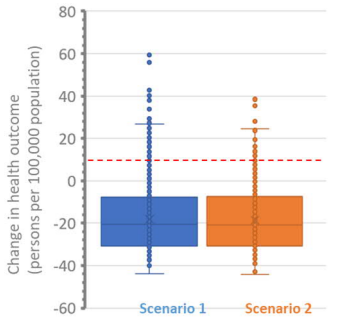
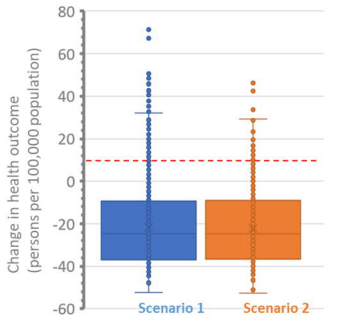
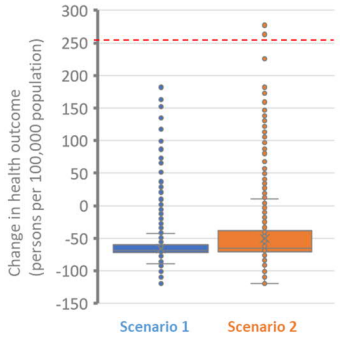
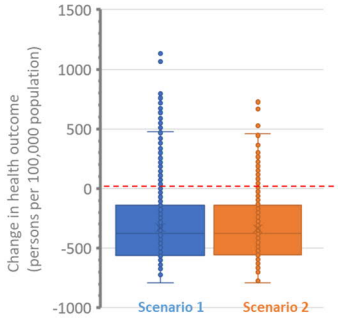
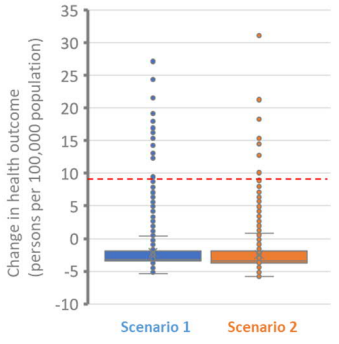
Similar to the population health outcomes from long-term exposure to the air pollutants evaluated, the box and whisker plots constructed for short-term exposure health outcomes in Table 7-7 show that for $PM_{2.5}$ exposures, the upper quartiles are higher for Scenario 2 than for Scenario 1, indicating the estimated change in population health outcomes from short-term exposures to $PM_{2.5}$ within the Study Area are slightly better (i.e. provide higher benefit) for Scenario 1 (without Hymix) than Scenario 2 (with Hymix), albeit the proposed redevelopment in both Scenarios provides a net overall benefit. The difference in population health outcomes for PM_{10} and NO_2 short-term exposures between Scenarios 1 and 2 are negligible (Table 7-7). The only pollutant for which some of the estimated maximums of the ranges (excluding outliers) displayed in Table 7-7 exceed a >10 in 100,000 population increase in the incidence of short-term exposure health outcomes is NO_2.[7] This occurs for a number of health outcomes (i.e. all-cause mortality non-trauma ≥30 years, ED visits for asthma 1-14 years, hospital admissions for cardiovascular disease 15-64 years and ≥65 years, hospital admissions for cardiac disease 15-64 years and ≥65 years, hospital admissions for cardiac failure ≥65 years, and hospital admissions for respiratory disease 1-4 years and 5-14 years and hospital admissions pneumonia and bronchitis 65+ years).

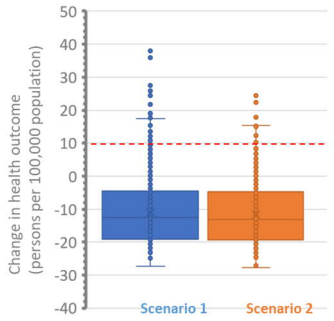
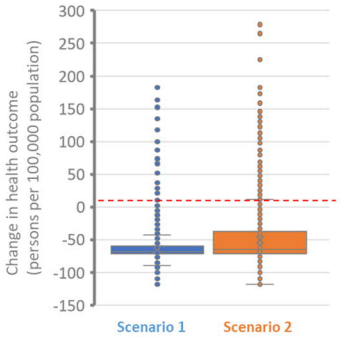
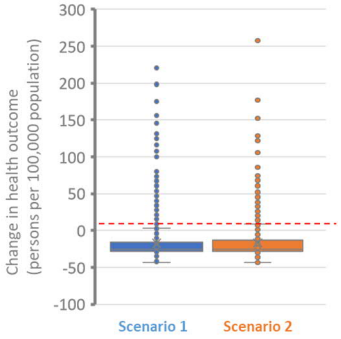
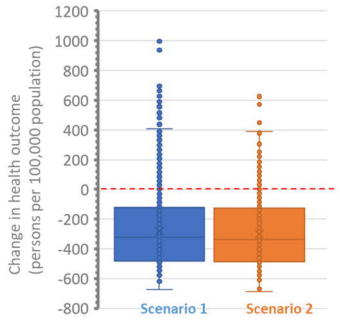
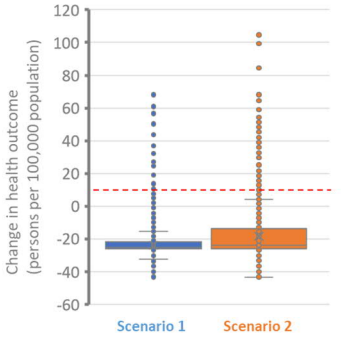
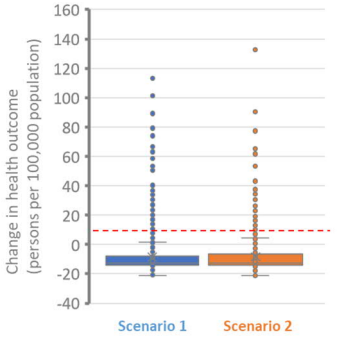
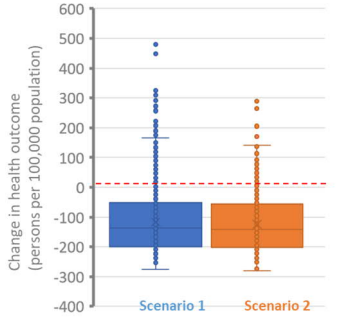
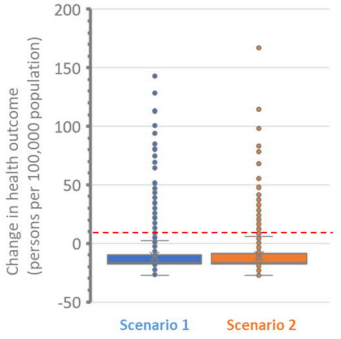
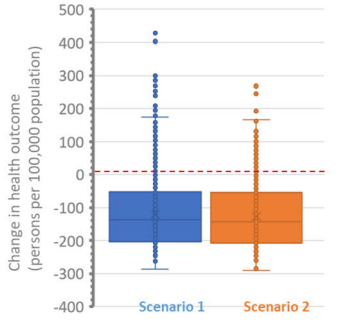
The individual locations and building localities at which >10 in 100,000 population increase in incidence of short-term exposure health outcomes have been predicted are examined more closely in Section 7.2.2.3.

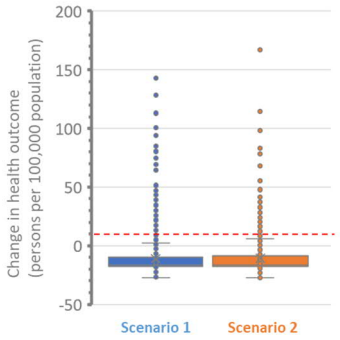
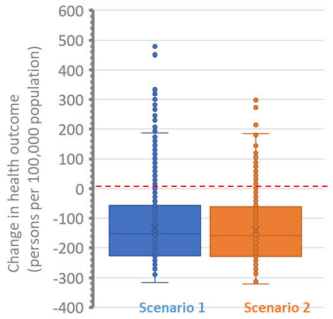
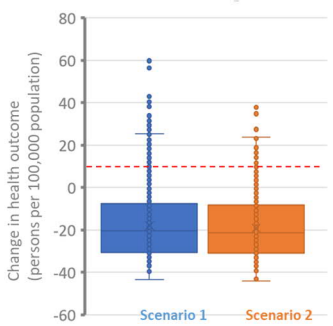
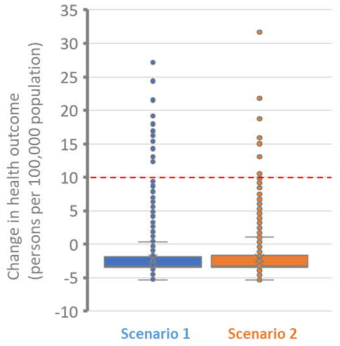
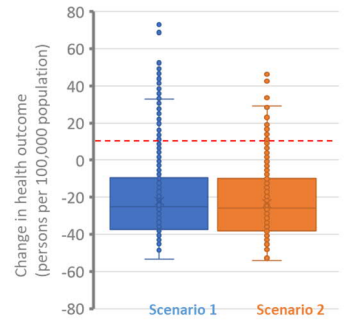
[7] It should be noted the interquartile ranges in Table 7-7 were calculated using all data combined. As the number of locations is predominated by residential and commercial locations (see Table 7-6), estimates at these locations heavily influence the overall distributions. Since the interquartile ranges presented in Table 7-6 were calculated separately for each location category, they clearly differ from those presented in Table 7-7 when using all data.

Table 7-7 Box and whisker plots showing estimated change in population incidence for short-term health outcomes for Scenario 1 vs. Scenario 2

Health endpoint/outcome	<2.5 µm Particulate Matter ($PM_{2.5}$)	<10 µm Particulate Matter (PM_{10})	Nitrogen Dioxide (NO_2)
Short-term (24-hour average or average 1-hour maximums)			
All-cause mortality (non-trauma) – all ages			
All-cause mortality (non-trauma) 30+ years			
Mortality cardiovascular disease- all ages			
Respiratory Mortality – all ages			

Health endpoint/outcome	<2.5 µm Particulate Matter ($PM_{2.5}$)	<10 µm Particulate Matter (PM_{10})	Nitrogen Dioxide (NO_2)
Emergency Department visits asthma 1-14 years			
Hospital admissions cardiovascular disease 15-64 years			
Hospital admissions cardiovascular disease 65+ years			
Hospital admissions cardiovascular disease – all ages			

Health endpoint/outcome	<2.5 µm Particulate Matter ($PM_{2.5}$)	<10 µm Particulate Matter (PM_{10})	Nitrogen Dioxide (NO_2)
Hospital admissions cardiac disease 15-64 years			
Hospital admissions cardiac disease 65+ years			
Hospital admissions cardiac failure 65+ years			
Hospital admissions respiratory disease 1-4 years			

Health endpoint/outcome	<2.5 µm Particulate Matter ($PM_{2.5}$)	<10 µm Particulate Matter (PM_{10})	Nitrogen Dioxide (NO_2)
Hospital admissions respiratory disease 5-14 years			
Hospital admissions respiratory disease 15-64 years			
Hospital admissions pneumonia and bronchitis 65+ years			

The ends of the box are the upper and lower quartiles (so the box spans the interquartile range); the median is marked by a horizontal line inside the box; the average is marked by an 'x' in the box; the minimum and maximum are marked by small horizontal black lines above and below the box; outliers are shown as dots above and below the minimum and maximum of the distribution.

The red dotted line indicates an estimated increase (from baseline incidence) of more than 10 in 100,000 people (i.e. in accordance with the criteria outlined in Section 7.1, this would be considered to be an unacceptable increase). However, it is the median or average statistic in this table that best represents the net overall effect on population incidence. All average estimated changes are considered to be acceptable; indeed, most average changes are negative and indicate an overall net health benefit of the proposed development on population health outcomes. See Appendix B for calculation spreadsheets.

7.2.2.3 Comparison by building/locality

Figure 7-4 summarises the estimated change in ED visits for asthma (in 1-14 year olds) and hospital admissions for cardiac disease (in ≥65 year olds) per population of 100,000 people from baseline as a result of short-term exposures to PM and NO_2 by building, location type and scenario.

Dots above the red dotted line indicate individual locations where an estimated change (from baseline incidence) of more than 10 in 100,000 people was calculated (i.e. in accordance with the criteria outlined in Section 7.1, this would be considered to be an unacceptable increase). However, as indicated previously the overall net change to population incidence of health outcomes is predicted to be negative, i.e. the proposed development is predicted to confer an overall benefit in terms of population health outcomes.

Figure 7-4 Estimated change in selected population health outcomes (persons per 100,000 population) from baseline incidence associated with short-term exposure to $PM_{2.5}$, PM_{10} and NO_2 by building and location type.
Dots above the red dotted line indicate individual locations where an estimated change (from baseline incidence) of more than 10 in 100,000 people was calculated (i.e. in accordance with the criteria outlined in Section 7.1, this would be considered to be an unacceptable increase). However, as indicated previously the overall net change to population incidence of health outcomes is predicted to be negative, i.e. the proposed development is predicted to confer an overall benefit in terms of population health outcomes.

Nevertheless, examining the locations which are estimated to be associated with >10 in 100,000 population increase from baseline can help provide an indication where management measures in building design could be considered to reduce exposure.

It is evident from Figure 7-4 that the vast majority of individual locations estimated to be associated with >10 in 100,000 increase of short-term health outcomes from baseline are retail and commercial locations.

The buildings and elevations at which this occurs are (Figure 7-5 and Figure 7-6):

- BLD01: Retail at ground level and commercial between 6 and 15m (Scenario 2 only).
- BLD02: Retail at ground level and residential between 6 and 15m (Scenario 1) or between 6 and 30m (Scenario 2).
- BLD03: Retail at ground level and commercial between 6 and 30m (Scenario 1 and 2).
- BLD04: Retail at ground level and commercial between 6 and 20m (Scenario 1 and 2).
- BLD05: Retail at ground level and commercial between 6 and 15m (Scenario 1) or between 6 and 10m (Scenario 2).
- BLD06: None.
- BLD07: Retail at 6 and 10m (Scenario 1 and 2) and commercial at 10m (Scenario 2).
- PLO 01 – Poulos: Retail at ground level, commercial between 6 and 30m and podium roof at 15-30m (Scenario 1 and 2).
- PLO 02 – Celestino: Retail at ground level, commercial between 6 and 20m, and podium roof at 10-15m (Scenario 1 and 2).
- PLO 03 – Hymix 1: Retail at ground level, commercial between 6 and 30m, and podium roof at 10-15m (Scenario 1 only).
- PLO 03 – Hymix 2: Retail at ground level and commercial between 6 and 20m (Scenario 1 only).

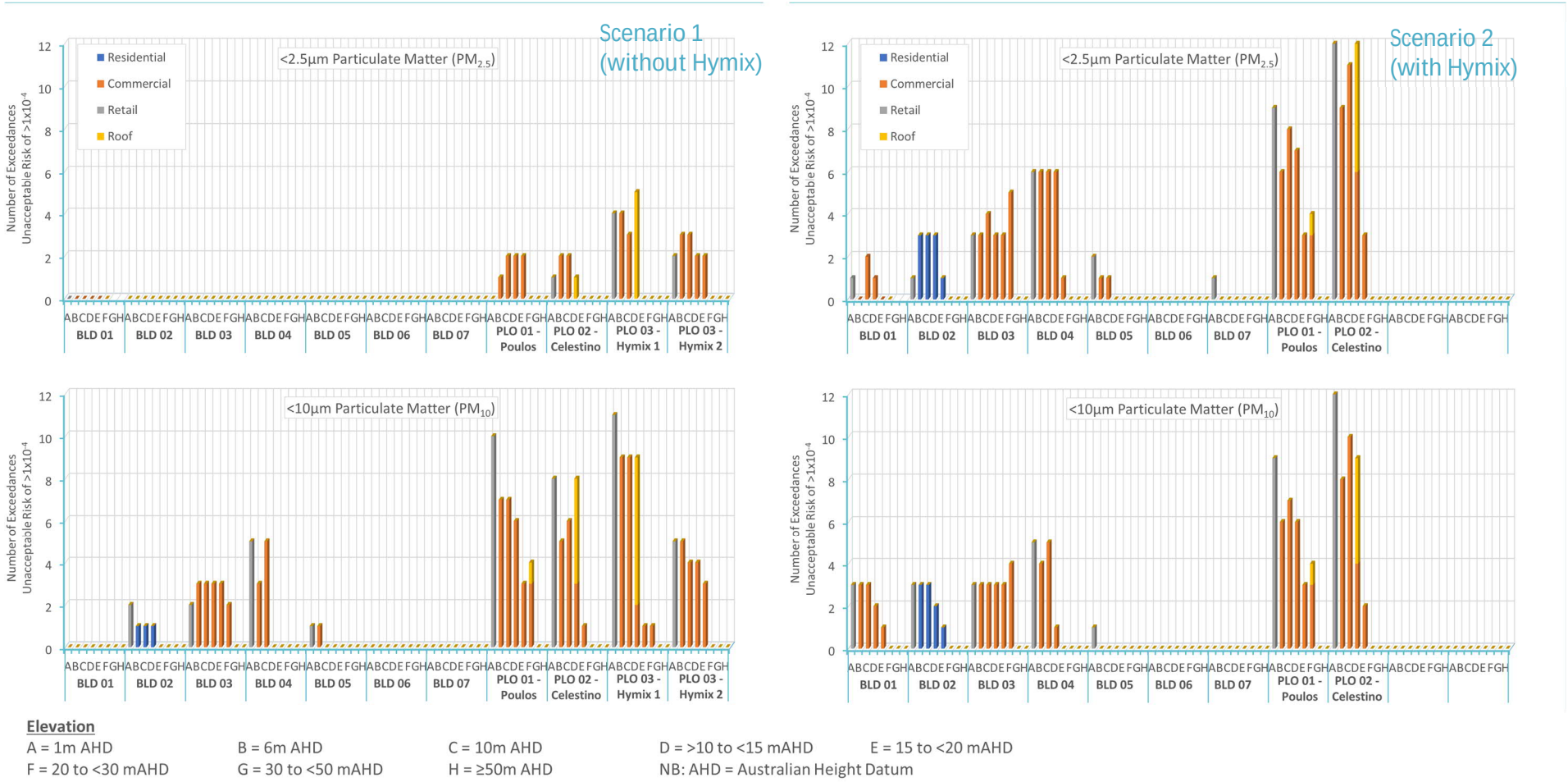
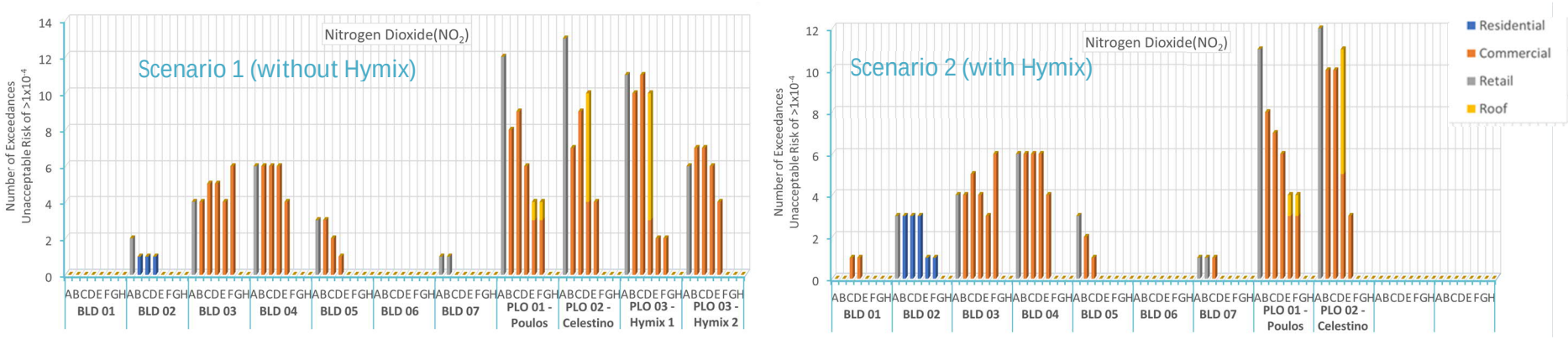


Figure 7-5 Number of modelled locations by building with an estimated change in hospital admissions for cardiac disease (≥65 years) of >10 people per 100,000 population (i.e. a 0.01% increased risk) from short-term exposures to PM



Elevation

A = 1m AHD B = 6m AHD C = 10m AHD D = >10 to <15 mAHD E = 15 to <20 mAHD
F = 20 to <30 mAHD G = 30 to <50 mAHD H = ≥50m AHD NB: AHD = Australian Height Datum

Figure 7-6 Number of modelled locations by building with an estimated change in hospital admissions for cardiac disease (≥65 years)of >10 people per 100,000 population (i.e. a 0.01% increased risk) from short-term exposures to NO_2

7.3 Complementary NO_2 Assessment

The assessment undertaken in Section 7.2 has shown that on a population level, the proposed development is predicted to result in a net benefit to population health outcomes related to both short- and long-term exposures to evaluated air pollutants from current baseline. The analysis of short-term exposure health outcomes also revealed that, of the three pollutants investigated, the highest individual occurrences of increases in incidence of population health outcomes occurred for exposures to NO_2. In order to provide an indication of potential likelihood of detectable adverse effects occurring to sensitive individuals at specific locations, a comparison was undertaken of cumulative modelled 1-hour average NO_2 concentrations with the lowest LOAEC available from controlled exposure studies described in the literature for asthma exacerbation (i.e. 188 µg/m^3, described in Section 5.3). This is the concentration (as a 1-hour average) at which 25% of exposed asthmatics experienced a clinically relevant airway response to NO_2. It is noteworthy that exercise does not exacerbate the response.

Figure 7-7 shows the 99.9th, 99.7th and 99th percentile 1-hour NO_2 concentrations at individual modelled locations. The concentrations above the red dotted line in the Figure are greater than the LOAEC. At these locations, there is a possibility that a sensitive asthmatic may experience airway responsiveness if they spend ~1 hour in these areas at the particular time that high NO_2 concentrations are present. An appreciation of the likelihood of this possibility can be gained by considering that the 99.9th percentile concentration may occur 0.1% of the time, the 99.5th 0.5% of the time, and the 97th 3% of the time. The number of locations where the various percentile modelled 1-hour NO_2 concentrations are predicted to exceed the LOAEC are shown in Figure 7-8 for Scenario 1 and Figure 7-9 for Scenario 2.

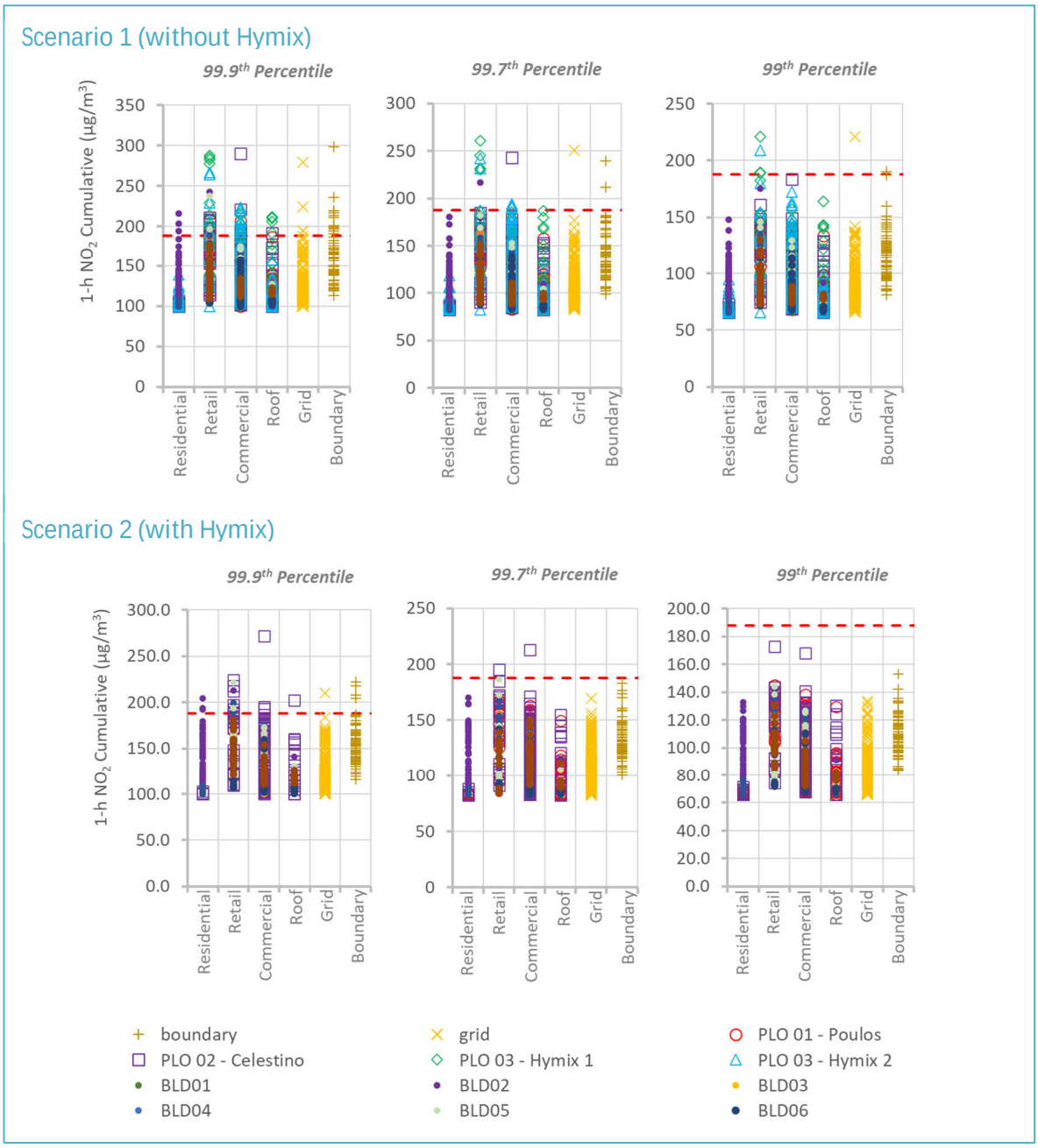


Figure 7-7 99.9th, 99.7th and 99th percentile 1-hour NO_2 concentrations at individual modelled locations by location type and building.
Dots above the red dotted line indicate individual locations where a sensitive asthmatic may experience airway responsiveness if they spend ~1 hour or more at the particular time that high NO_2 concentrations are present (i.e. concentrations >LOAEC). The concentration may occur for 9 hours (for the 99.9th percentile), 44 hours (for the 99.7th percentile) or 263 hours (for the 99th percentile) of the year.

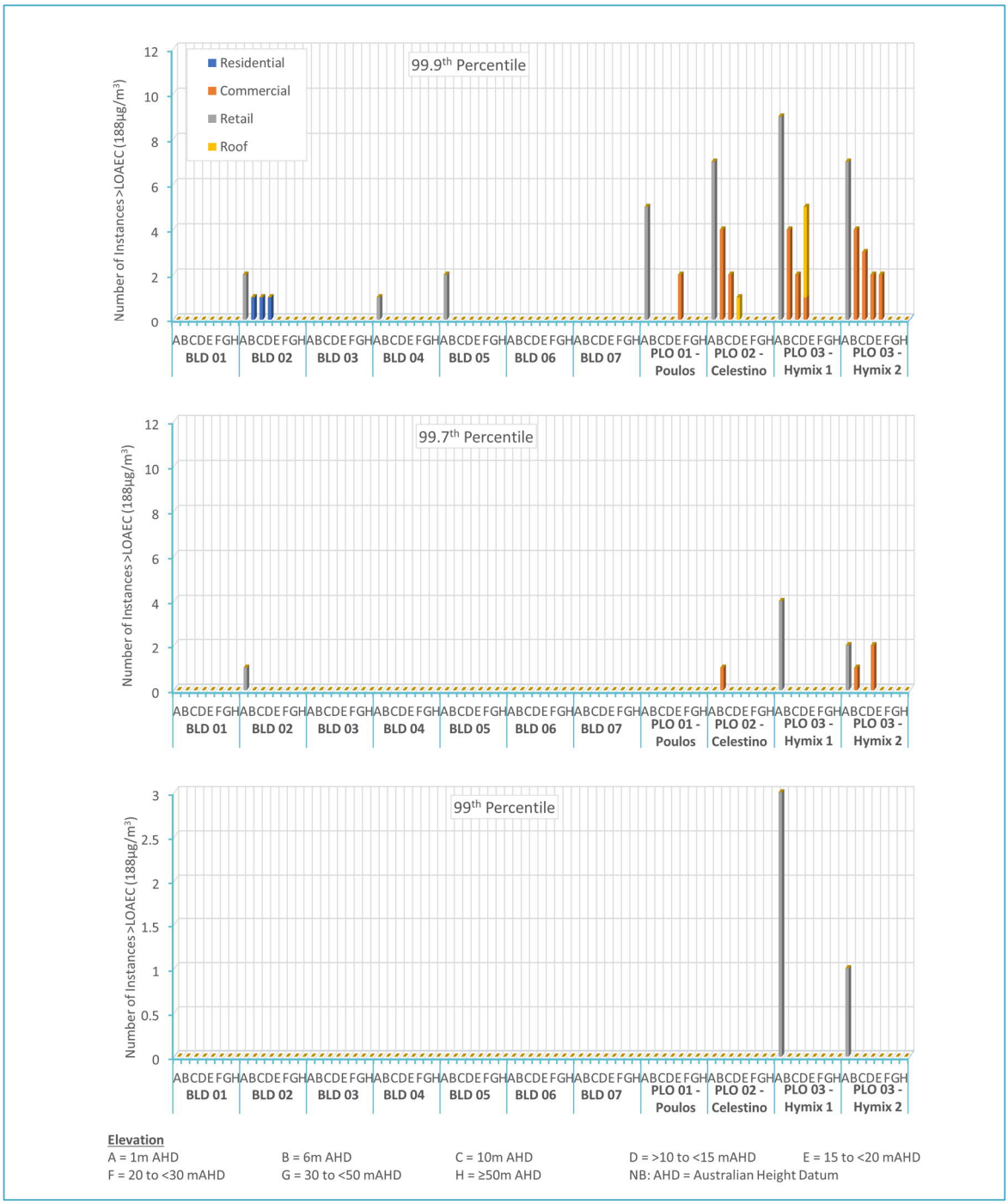


Figure 7-8 Number of building locations where modelled 99.9th, 99.7th and 99th percentile 1-hour NO_2 concentrations are predicted to exceed the LOAEC for sensitive asthmatics for Scenario 1 (without Hymix).
These data show individual locations where a sensitive asthmatic may experience airway responsiveness if they spend ~1 hour or more at the particular time that high NO_2 concentrations are present (i.e. concentrations >LOAEC). The relevant concentration may occur for 9 hours (for the 99.9th percentile), 44 hours (for the 99.7th percentile) or 263 hours (for the 99th percentile) of the year.

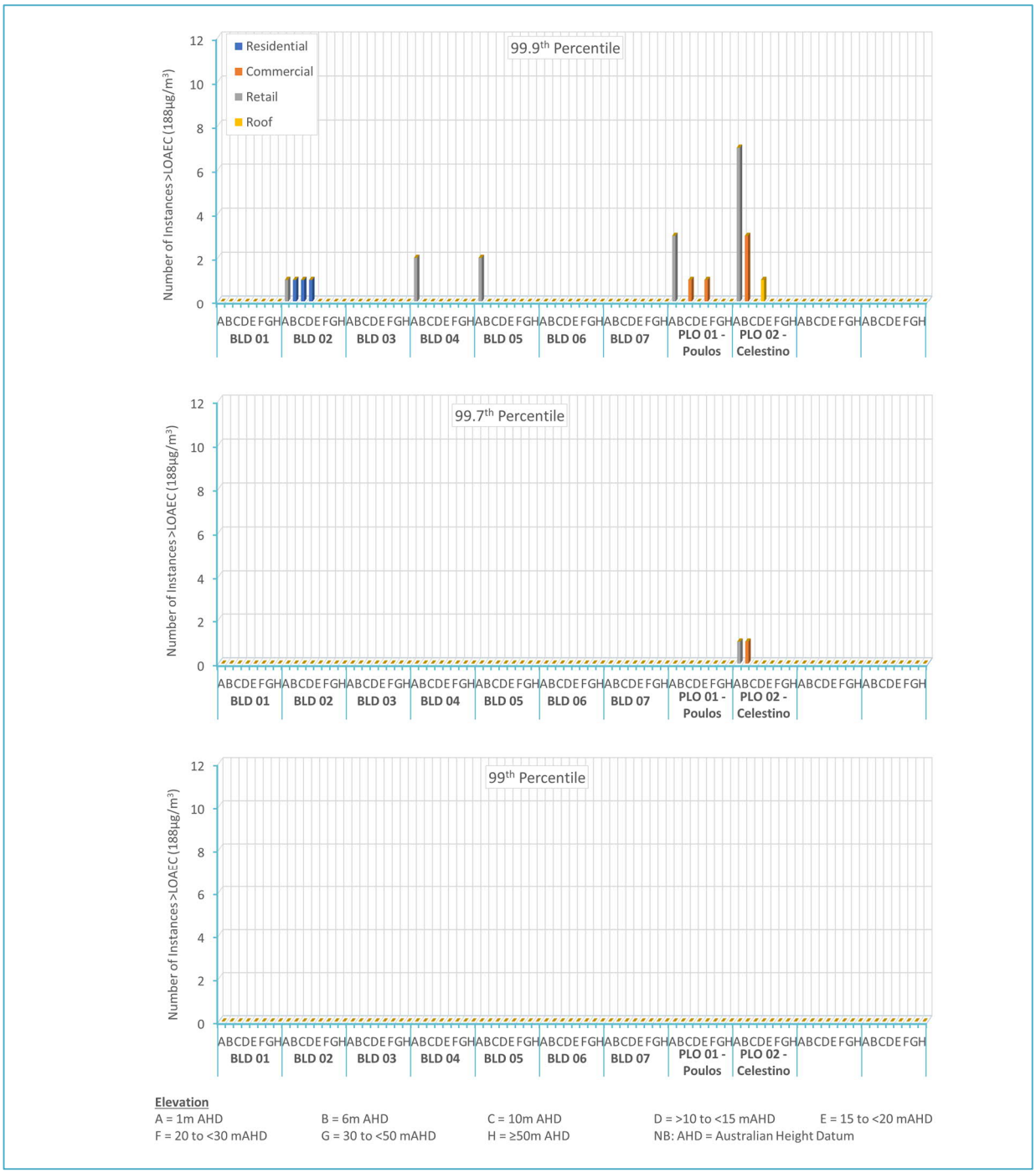


Figure 7-9 Number of building locations where modelled 99.9th, 99.7th and 99th percentile 1-hour NO_2 concentrations are predicted to exceed the LOAEC for sensitive asthmatics for Scenario 2 (with Hymix).
These data show individual locations where a sensitive asthmatic may experience airway responsiveness if they spend ~1 hour or more at the particular time that high NO_2 concentrations are present (i.e. concentrations >LOAEC). The relevant concentration may occur for 9 hours (for the 99.9th percentile), 44 hours (for the 99.7th percentile) or 263 hours (for the 99th percentile) of the year.

From the Figures it is evident:

- Comparison between scenarios shows that where the same buildings are present, the number and identity of locations where the LOAEC is predicted to be exceeded for 0.1-3% of the time are similar for Scenarios 1 and 2, indicating the roadways (rather than Hymix) are the primary contributor to NO_2 at these locations.
- Scenario 1 (without Hymix) exhibits a larger total number of exceedances than Scenario 2 (with Hymix) since there are a large number of additional locations modelled due to the presence of the two additional buildings (PLO 03 – Hymix 1 and PLO 03 – Hymix 2), which are absent in Scenario 2.
- Depending on the scenario evaluated, for the 99.9th percentile NO_2 concentrations (which may occur for 9 hours out of the year), exceedances of the LOAEC are predicted to occur for the following building locations:
    - BLD01: None.
    - BLD02: Three (out of 254 modelled, i.e. 1.2%) residential locations at 6 to 15m and one to two (out of 16 modelled, i.e. 6.3-12.5%) retail locations at ground level.
    - BLD03: None.
    - BLD04: One to two (out of 15 modelled, i.e. 6.7-13.3%) retail locations at ground level.
    - BLD05: Two (out of 14 modelled, i.e. 14.3%) retail locations at ground level.
    - PLO 01 – Poulos: Two (out of 103 modelled, i.e. 1.9%) commercial locations at 10-20m elevation and 3-5 (out of 19 modelled, i.e. 15.8-26.3%) retail locations at ground level.
    - PLO 02 – Celestino: 3-6 (out of 121 modelled, i.e. 2.5-5%) commercial locations at 6-10m elevation, 7 (out of 24 modelled, i.e. 29.2%) retail locations at ground level, and one (out of 18 modelled, i.e. 5.6%) podium roof location at 10-15m elevation.
    - PLO 03 – Hymix 1 (only applicable for Scenario 1): 7 (out of 77 modelled, i.e. 9.1%) commercial locations at 6-15m elevation, 9 (out of 17 modelled, i.e. 52.9%) retail locations at ground level, and 4 (out of 23 modelled, i.e. 17.4%) podium roof location at 10-15m elevation.
    - PLO 03 – Hymix 2 (only applicable for Scenario 1): 11 (out of 107 modelled, i.e. 10.3%) commercial locations at 6-20m elevation and 7 (out of 17 modelled, i.e. 41.2%) retail locations at ground level.

Although the likelihood of sensitive asthmatics being present at these locations at the exact time that concentrations of NO_2 greater than the LOAEC may be found is considered low (especially considering the inbuilt conservatism in the modelling, see Section 7.4), the possibility cannot be excluded.

It is noted that a sensitivity analysis was undertaken for Scenario 1 predictions in the AQIA to assess the impacts of using vehicular emission factors expected to be more representative of future emissions. This was done by scaling vehicle emission factors using ratios calculated from the 2016 and 2026 emission factors derived from the Roads and Maritime air quality screening model TRAQ (SLR 2021). This analysis showed that the incremental NO_2 concentrations are likely to be markedly overestimated (by approximately 56%) by the modelling predictions using the 2010 vehicle fleet emissions (see also Section 7.4). Using the revised predictions for Scenario 1, the 99.9th percentile 1-hour NO_2 concentration is only predicted to be equal to or exceed the NO_2 LOAEC at three locations within the Study Area, all of these being retail locations (at 1m elevation) in PLO 03-Hymix 2 or BLD02. It is therefore recommended that management measures be considered (where practical) to reduce potential NO_2 exposures at these locations.

In addition to the locations on the façade or roof of the proposed buildings, it is also important to consider whether any LOAEC exceedances are predicted to occur in proposed new public open spaces within the Study Area. Although depending on Scenario, the 99.9th percentile of all NO_2 concentrations was predicted to exceed the NO_2 LOAEC at a total of one to four (out of 238 modelled, i.e. 1.7%) grid locations and 4-13 (out of 61 modelled, i.e. 6.6%) boundary locations, these locations are all located directly adjacent to the busy Western Distributor and none of these locations are located in the proposed new parks where people might spend a significant amount of time outdoors (Figure 7-10, Figure 7-11).

The only exception is location No. 2312 in Scenario 1 which is along the proposed new Waterfront Promenade. At this location the 99.9th percentile modelled NO_2 concentration is 190 µg/m^3, just above the LOAEC of 188 µg/m^3. Considering the infrequency that this concentration might occur (both the 99.7th and 99th percentiles are markedly below the LOAEC), the overall conservatism in the modelling, and the unlikelihood that someone would spend 1 hour or more in one particular location along the promenade, the likelihood of adverse effects being experienced at this location is considered very low. Indeed, the sensitivity analysis undertaken in the AQIA for Scenario 1 incorporating more realistic vehicular emission factors revealed that none of the 99.9th percentile NO_2 concentrations in proposed new public open spaces within the Study Area are predicted to exceed the LOAEC.

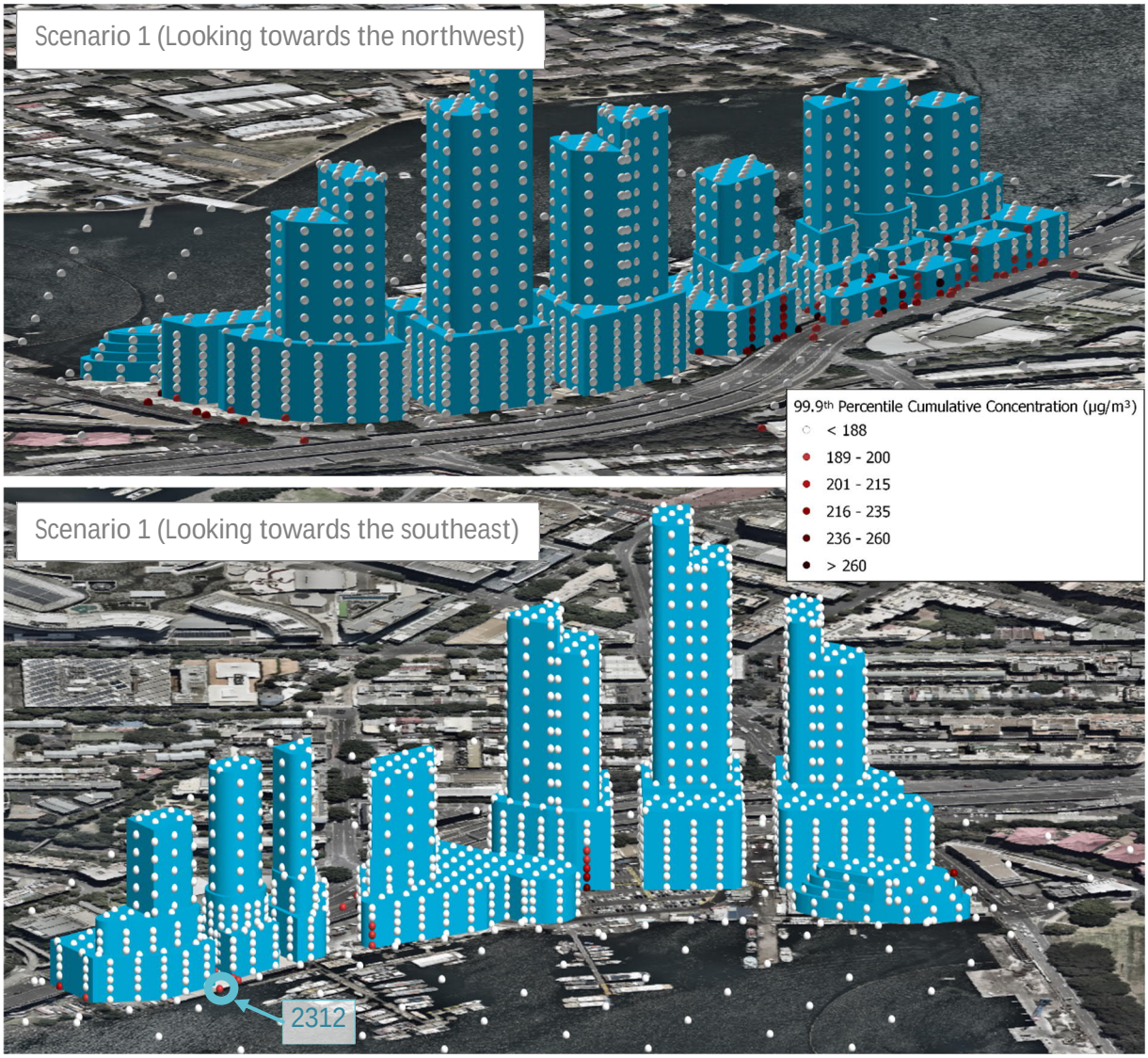


Figure 7-10 Locations in Scenario 1 where modelled 99.9th percentile NO_2 concentrations are predicted to be ≥ LOAEC in sensitive asthmatics (i.e. ≥188 µg/m^3).

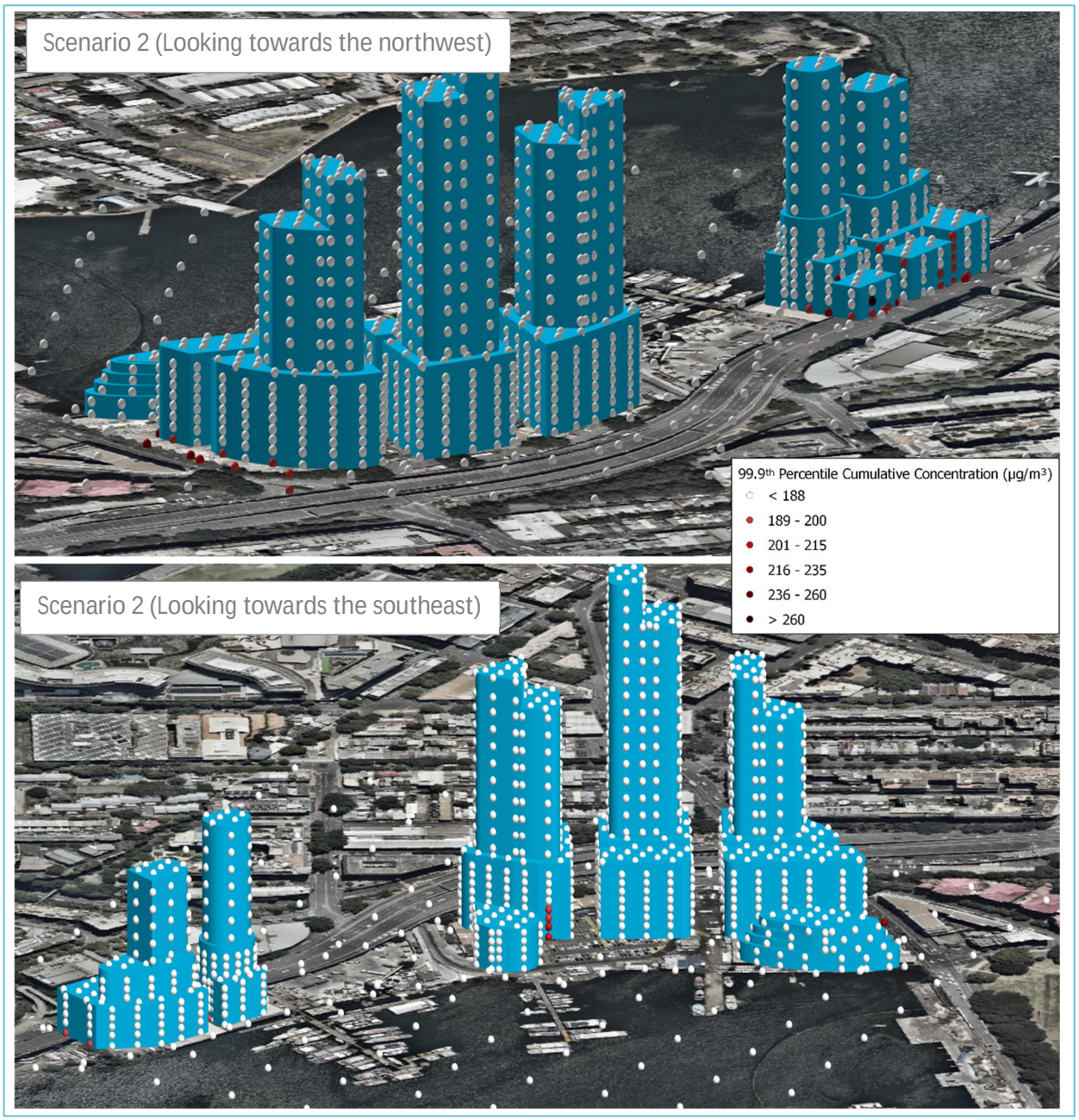


Figure 7-11 Locations in Scenario 2 where modelled 99.9th percentile NO_2 concentrations are predicted to be ≥ LOAEC in sensitive asthmatics (i.e. ≥188 µg/m^3).

7.4 Uncertainty analysis

Health risk assessments involve a number of steps (e.g. exposure assessment, toxicity assessment and risk characterisation), each of which may incorporate the use of assumptions and simplifications to manage uncertainty or lack of knowledge about the 'real' value. Without such assumptions and simplifications, it would not be possible to quantitatively evaluate the potential for health effects. Although uncertainties in the risk assessment may influence its accuracy, reliability and interpretation, the assumptions used to cope with the uncertainties tend to err on the side of safety and therefore bias the evaluation to over estimation of health risk. This is appropriate for an assessment for possible impacts on public health.

This section contains a general qualitative discussion of the principal uncertainties and their potential influence on the health risk assessment. The main uncertainties/assumptions and the effect they have on the risk assessment are summarised in Table 7-8.

Elsewhere in this report, information on the uncertainty of specific assumptions is provided to enable the reader to integrate the uncertainties with the assessment at that point in the report.

Table 7-8 Principal uncertainties in this HHRA

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
Air quality dispersion modelling provides a true reflection of Scenario 1 and 2 air quality.	The modelling undertaken as part of the AQIA has been taken on face value in this risk assessment. A number of assumptions were made in undertaking the dispersion modelling which render the modelling results conservative. These assumptions included: • Vehicular emissions were estimated based on a low vehicle speed of 10 km/hr (potential worst-case emission rate that would be representative of congested traffic conditions) from 6am to 10pm every day of the year. • In addition, potential improvements in emissions performance of newer cars in the future were not accounted for. It was assumed the 2033 vehicle fleet emission rates are similar to the 2010 vehicle fleet. • Particulate emissions from aggregate and sand transfer and weigh hopper loading activities at the concrete batch plant were assumed to be uncontrolled. • All available ambient ozone was assumed to react immediately with emitted NO to form NO_2.	The use of these assumptions ensure that the modelling is conservative and more likely to over-estimate than under-estimate modelled air pollutant concentrations. Since the same assumptions were made for modelling the 'baseline' locations, this is unlikely to markedly affect the conclusions in the population health impact assessment, since the metric of interest is the modelled change in air pollutant concentrations from baseline. Nevertheless, any overestimation of this incremental change would also overestimate the increased incidence of health outcomes in the HHRA. Potential overestimation of cumulative modelled air pollutant concentrations could also have resulted in an overestimation of the likelihood of adverse health effects in the complementary NO_2 assessment against a LOAEC. If the extent of model overestimation is greater than a factor of 1.6x, then the conclusions in relation to NO_2 would change and none of the modelled locations would be predicted to exceed the LOAEC. In fact, an additional sensitivity analysis was undertaken for Scenario 1 predictions in the AQIA to assess the impacts of using vehicular emission factors expected to be more representative of future emissions. This was done by scaling vehicle emission factors using ratios calculated from the 2016 and 2026 emission factors derived from the Roads and Maritime air quality screening model TRAQ (SLR 2021). This analysis showed that the incremental NO_2 concentrations are likely to be markedly overestimated (by 56%) by the modelling predictions using the 2010 vehicle fleet emissions. Using the revised predictions for Scenario 1, the 99.9th percentile 1-hour NO_2 concentration is only predicted to be equal to or exceed the NO_2 LOAEC at three locations within the Study Area (all of these being retail locations in PLO 03- Hymix 2 or BLD02).

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
Concentration Response Functions (CRFs) are appropriate for the population under consideration.	The HHRA used CRFs sourced and recommended for use in Australia by a review undertaken to update the NEPC (2016) air quality standards. The available literature was independently reviewed by expert epidemiologists (Jalaludin and Cowie 2012) and recommendations were agreed upon in a meeting of experts at the Victorian Environmental Protection Authority. The CRFs are expressed as means with upper and lower 95% confidence intervals.	As the CRFs were compiled by a number of expert epidemiologists from studies in the peer reviewed literature, they are considered appropriate for use in this HHRA. The majority of the information presented in Section 7.2 is based on use of the average CRF, however all calculations have also been undertaken using the upper and lower 95% confidence intervals around the mean CRF (see Appendix B). The use of the 95% confidence interval CRFs would not alter the conclusions of this report.
Baseline health incidence data available at the time of writing this report was from 2010 and was only available for a wider area (i.e. Sydney) instead of the local population.	At the time of writing this report, updated baseline health incidence data had been requested but not yet received from ABS and AIHW, both for the wider Sydney and NSW region, as well as for a more localised area which includes the Study Area. The data summarised in Section 2.4.2 suggests there may be a lower incidence of most health outcomes in the area in the LGA in which the Study Area is located relative to the rest of NSW. However, it is unknown to SLR at this time whether the incidence of these health endpoints is also lower than for greater Sydney (which are the data used in the HHRA).	If the baseline incidence of a health outcome for the region which includes the Study Area is lower than what has been assumed in the HHRA, this would decrease the estimated change in population health outcomes and vice versa. It is recommended that once the updated and more location-specific baseline incidence data become available, the potential impact of the updated information on the overall conclusions of the HHRA be considered.
Protectiveness of NO_2 LOAEC for sensitive asthmatics.	The LOAEC used in this HHRA is a 1-hour exposure concentration of NO_2 which, in controlled exposure studies, was the lowest concentration at which a clinically relevant airway response was observed in 25% of asthmatics exposed. Every asthmatic has their individual sensitivity to particular pollutants in air. It is possible the LOAEC may not be protective for all individuals. However, other studies reviewed by Brown (2015) determined higher LOAECs which does suggest the LOAEC used in the HHRA is likely a true LOAEC for the majority of individuals.	This is unlikely to markedly affect the conclusions of the HHRA, especially considering the conservativeness incorporated into the modelling of air pollutant concentrations.

Uncertainty/ Assumption	Comment	Effect on Risk Assessment
No exposure adjustment was undertaken for commercial or retail locations in the population health impact assessment.	Since people are likely present at commercial and retail locations for less than 24 hours per day, the assessment undertaken herein is conservative for commercial and retail locations. However, it was considered inappropriate to adjust the calculations to assume only 8 out of 24 hours exposure, since it is feasible that someone may both live and work in the proposed development. Exposure to air pollutants in general also does not cease when someone leaves work.	The effect on the assessment is a potential overestimation for increased population incidence (from baseline) for the health endpoint assessment at commercial and retail locations.
Uncertainties related to co-pollutant exposure and effects.	It is likely that some of the baseline health outcomes, as well as the outcomes described by CRF functions, relate to a number of related/co-related air pollutants.	This means that it is inappropriate to add together the health outcomes from different pollutants. As this has not been done in this HHRA, the effect of this uncertainty on the HHRA is considered negligible.

8 Overall conclusions

Population Health Endpoint Assessment

The population health endpoint assessment undertaken in this HHRA has shown that for both long-term and short-term exposure, the altered exposure circumstances of the proposed development are predicted to provide an average net health benefit. This means that on average lower population exposures to $PM_{2.5}$, PM_{10}, and NO_2 are anticipated to occur within the totality of the Study Area relative to other residences and commercial properties south, southeast, and east of the Study Area.

When comparing Scenario 1 (without Hymix) and Scenario 2 (with Hymix), the estimated change in population health outcomes from long-term and short-term exposures to $PM_{2.5}$ within the Study Area are slightly better (i.e. provide higher benefit) for Scenario 1 (without Hymix) than Scenario 2 (with Hymix), albeit the proposed redevelopment in both Scenarios provides a net overall benefit relative to baseline conditions. The difference in population health outcomes for PM_{10} and NO_2 long-term exposures between Scenarios 1 and 2 are negligible.

There are a number of locations at which, when modelled individually, from a societal perspective, an unacceptable increase in a particular health outcome from short-term or long-term exposures to the pollutants evaluated may result. This should be interpreted as follows: if a large population of people were to live in that one receptor location, then there may be an unacceptable increase from baseline incidence in the health outcome. Clearly a large population could never live in the one location. This unacceptability at a few locations is more than balanced out by the numerous locations where there is an overall net health benefit (i.e. reduced exposure, and therefore reduced population incidence of an adverse health outcome). The majority of locations where this occurred were either retail or commercial locations on the façade of buildings.

The analysis of short-term exposure health outcomes also revealed that, of the three pollutants investigated, the highest individual occurrences of increases in incidence of adverse population health outcomes occurred for exposures to NO_2.

Complementary NO_2 Assessment of Individual Exposures

The comparison of cumulative modelled 1-hour average NO_2 concentrations with the lowest LOAEC available from controlled exposure studies described in the literature for asthma exacerbation in individuals showed that where the same buildings are assumed to be present, the number and identity of locations where the LOAEC is predicted to be exceeded for 0.1-3% of the time are similar for Scenarios 1 and 2.

Scenario 1 (without Hymix) exhibits a larger total number of exceedances than Scenario 2 (with Hymix) since there are a large number of additional locations modelled due to the presence of the two additional buildings (PLO 03 – Hymix 1 and PLO 03 – Hymix 2), which are absent in Scenario 2.

Depending on the scenario evaluated, for the 99.9th percentile NO_2 concentrations (which may occur for 9 hours out of the year), a number of exceedances of the LOAEC were predicted to occur for different building locations. These were:

- BLD02: Three residential locations at 6 to 15m and one to two retail locations at ground level.
- BLD04: One to two retail locations at ground level.
- BLD05: Two retail locations at ground level.
- PLO 01 – Poulos: Two commercial locations at 10-20m elevation and 3-5 retail locations at ground level.

- PLO 02 – Celestino: 3-6 commercial locations at 6-10m elevation, 7 retail locations at ground level, and one podium roof location at 10-15m elevation.
- PLO 03 – Hymix 1 (only applicable for Scenario 1): 7 commercial locations at 6-15m elevation, 9 retail locations at ground level, and 4 podium roof location at 10-15m elevation.
- PLO 03 – Hymix 2 (only applicable for Scenario 1): 11 commercial locations at 6-20m elevation, 7 retail locations at ground level.

Although the likelihood of sensitive asthmatics being present at these building locations at the exact time that concentrations of NO_2 greater than the LOAEC may be found is considered low (especially considering the inbuilt conservatism in the modelling), the possibility cannot be excluded. It is noted, however, that a sensitivity analysis was undertaken for Scenario 1 predictions in the AQIA to assess the impacts of using vehicular emission factors expected to be more representative of future emissions. This analysis showed that the incremental NO_2 concentrations are likely to be markedly overestimated (by approximately 56%) by the modelling predictions using the 2010 vehicle fleet emissions. Using the revised predictions for Scenario 1, the 99.9th percentile 1-hour NO_2 concentration is only predicted to be equal to or exceed the NO_2 LOAEC at three locations within the Study Area, all of these being retail locations (at 1m elevation) in PLO 03- Hymix 2 or BLD02.

It is therefore recommended that management measures be considered (where practical) to reduce potential NO_2 exposures at these retail locations.

Based on a comparison of modelled NO_2 concentrations at grid and boundary locations, it is considered unlikely that adverse effects resulting from exposure to NO_2 would be experienced by people using the proposed new public open spaces within the Study Area.

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APPENDIX A

Toxicological Profiles

A1. Particulate Matter

Airborne particulate matter (PM) is a heterogenous mixture of solid and liquid particles with a variety of chemical components and physical features. Commonly used indicators describing PM that are relevant to health refer to the mass concentration of particles with a mass median aerodynamic diameter of less than 10 µm (PM_{10}) and of particles with a diameter less than 2.5 µm ($PM_{2.5}$). The latter is often called fine PM, which also comprises ultrafine particles having a diameter of less than 0.1 µm (WHO 2013b). By way of comparison, a human hair is about 100 µm, so roughly approximately 40 fine particles could be placed on its width (NPI 2019) (see Figure A.1). PM can originate from a variety of anthropogenic stationary or mobile sources (e.g. combustion engines, solid fuel combustion for energy production, mining, building, smelting, etc), as well as from natural sources (e.g. soil erosion and dust re-suspension, volcanic emissions, etc).

Inhalation of airborne particles dispersed to ambient air has been associated with various health effects. These include (NEPC 2014):

- premature mortality
- aggravation of cardiovascular disease (e.g. atherosclerosis)
- aggravation of respiratory disease (e.g. asthma) disease
- changes to lung tissue, structure, and function
- reproductive and developmental effects
- changes in the function of the nervous system
- cancer[8].

[8] The International Agency for Research on Cancer (IARC) has classified outdoor air pollution as carcinogenic to humans, with an emphasis on PM in general and especially PM in diesel engine exhaust (IARC 2012, 2013).

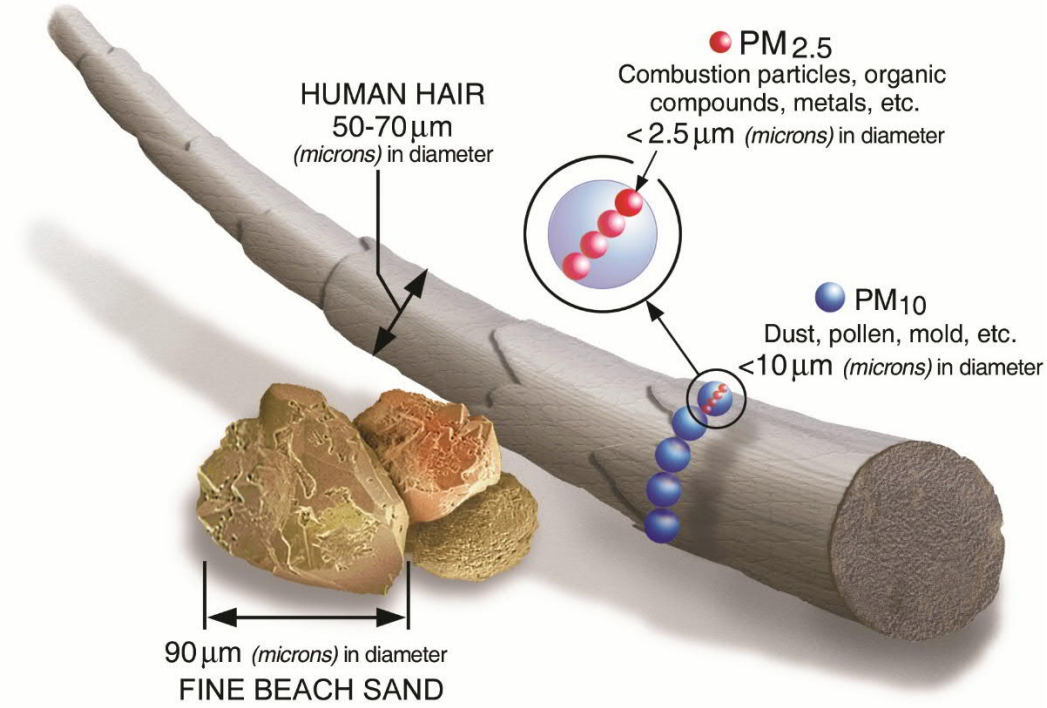

Figure A.1 Diagrammatic size comparison for PM particle sizes (US EPA 2020)

The health effects of inhaled particles are defined by their physical and chemical properties, by the sites of deposition, and by their mechanisms of action (NEPC 2014). There is the clear link between the particle size of PM and the potential for causing health effects (WHO 2013b). Small particles of concern include inhalable coarse particles with a diameter of 2.5 to 10 µm and fine particles smaller than 2.5 µm in diameter (Kim et al. 2015). During normal nasal breathing larger particles with an aerodynamic diameter between 10 µm and 100 µm are deposited in the extrathoracic region (nose, mouth and throat) of the respiratory tract, and are easily eliminated from the body through expiration or by ingestion (NEPC 2014). Particles in the 5–10 µm range are mostly deposited in the proximity of the larynx and enter the thoracic region (Brown et al. 2013). Particles with a diameter of less than 2.5 µm can penetrate deep into the human respiratory system (Xing et al. 2016). $PM_{2.5}$ and PM_{10} have been shown in numerous epidemiological studies to be associated with mortality and hospitalisation from cardiovascular and respiratory causes (NEPC 2014). A growing body of research has pointed towards the $PM_{2.5}$ fraction as being the most significant in relation to health outcomes (NEPC 2014). PM is considered a pollutant that exerts its effects in a non-threshold manner (NEPC 2014, WHO 2013b, US EPA 2019), i.e. in health risk assessment for PM, potential health effects are quantitated as potential increased incidence of morbidity or mortality due to a unit increase in PM concentration.

Cardiovascular effects

Exposure to ambient PM is a risk factor for the development of ischemic cardiovascular events via exacerbation of atherosclerosis, coronary artery disease, and triggering of myocardial infarction, sometimes occurring within hours following exposure (Sun et al. 2010; Peters et al. 2001). In their 2019 Integrated Science Assessment for PM, US EPA (2019) concluded that there is a causal relationship between short-term and long-term $PM_{2.5}$ exposure and cardiovascular effects (e.g. emergency department visits and hospital admissions for ischemic heart disease and heart failure, as well as cardiovascular mortality), whereas the relationship for $PM_{10-2.5}$ and ultrafine particles was either suggestive but insufficient to infer causality or inadequate. For example, one study has demonstrated a significant elevation in the incidence of life-threatening myocardial infarctions for an hourly increase (1-h) of 25 µg/m³ in $PM_{2.5}$ or a daily increase (24-h) of 20 µg/m³ $PM_{2.5}$ (Dockery et al. 2001). Amsalu et

al. (2019) reported an increase in cardiac arrhythmias in the hours to days following exposure to high levels of atmospheric $PM_{2.5}$. For every 10 µg/m^3 increase in the $PM_{2.5}$ concentration from the previous day, there was a significant increase in total cardiovascular disease admissions, with a strong association for older adults (aged ≥ 65 years) (Amsalu et al. 2019). Another study evaluating heart rate showed an association between previous day exposure to PM_{10} at 100 µg/m^3 and significantly increased heart rate, suggesting that changes in cardiac autonomic function may be part of the pathophysiological mechanism linking cardiovascular mortality and PM_{10} (Pope et al. 1999).

Respiratory effects

Elevated levels of PM in air are strongly correlated with the onset and development of various respiratory diseases with measures used in epidemiological studies including aggravation of asthma, respiratory symptoms, increase in respiratory-related hospital admissions and mortality. The most sensitive vulnerable populations suffering from respiratory effects associated with airborne particulate matter are primarily those with existing respiratory conditions such as asthma, bronchitis, and chronic obstructive pulmonary disease (COPD), pneumonia patients, and children and the elderly (> 65 years) (NEPC 2019).

US EPA (2019) concluded that the relationship between short-term and long-term $PM_{2.5}$ exposure and respiratory effects is likely to be causal, whereas the relationship for $PM_{10-2.5}$ and ultrafine particles was either suggestive but insufficient to infer causality or inadequate. The WHO (2013b) stated there is good evidence of the effects of short-term exposure to PM_{10} on respiratory health but for mortality, and especially as a consequence of long-term exposure, $PM_{2.5}$ is a stronger risk factor than the coarse part of PM_{10} (i.e. $PM_{10-2.5}$). All-cause daily mortality is estimated to increase by 0.2-0.6% per 10 µg/m^3 of PM_{10}; long-term exposure to $PM_{2.5}$ is associated with an increase in the long-term risk of cardiopulmonary mortality by 6-13% per 10 µg/m^3 of $PM_{2.5}$ (WHO 2013b).

Zanobetti et al. (2009) reported that when the daily $PM_{2.5}$ increased by 10 µg/m^3 the prevalence rate of respiratory diseases increased by 2.07%, while hospitalisation rate increased by 8%. Peacock et al. (2011) showed that there was an increase of COPD symptoms and exacerbations in patients living in London particularly for PM_{10} at daily average outdoor concentrations >37 µg/m^3.

Other effects

Epidemiological studies have shown that long-term exposure to $PM_{2.5}$ significantly increases mortality from lung cancers (Valavanidis et al. 2013, Ciabattini et al. 2020, Raaschou-Nielsen et al. 2013, Ni et al. 2015). Ciabattini et al. (2020), using the results from 15 cohort studies, recently found the relative risk (RR) of lung cancer mortality for an increase of 10 µg/m^3 in $PM_{2.5}$ exposure was 1.16 (95% confidence interval [CI] 1.09, 1.23). The corresponding RR for PM_{10} exposure was 1.23 (95 CI 1.05, 1.40; seven studies). This indicates magnitude of the risk might be higher than previously estimated.

Correia et al. (2013) conducted a 7-year study (from 2000 to 2007) in the United States which showed that the average life span was extended by 0.35 years for every 10 µg/m^3 decrease of $PM_{2.5}$. Nevertheless, US EPA (2019) concluded that the relationship between $PM_{2.5}$ and $PM_{10-2.5}$ exposures and cancer is only *likely to be causal* or *suggestive but insufficient to infer causality*, respectively. This may stem from the fact that chronic health effects of air pollution are more difficult to investigate than acute effects, because of the need for longitudinal studies with valid assessment of exposure, data on potential confounders, adequate latency between exposure and outcome (Ciabattini et al. 2020).

With respect to other health effects, US EPA (2019), in their detailed Integrated Science Assessment of PM, concluded:

- There is likely to be a causal relationship between long-term $PM_{2.5}$ exposures and certain neurological effects including changes in brain morphology, cognitive decrements and dementia.
- For other health effects (e.g. reproductive and fertility effects, metabolic changes etc), the evidence was considered to be suggestive but insufficient to infer causality for $PM_{2.5}$ or inadequate for $PM_{10-2.5}$ and ultrafine particles.

Concentration response

Reviews of the concentration-response relationships for PM exposures have indicated the most informative relationships exist for studies of short-term $PM_{2.5}$ exposure and respiratory-related emergency department visits, hospital admissions and mortality. For long-term $PM_{2.5}$ exposures, most of the concentration-response information comes from studies of mortality with some initial recent evidence from studies of respiratory and cardiovascular effects, as well as lung cancer mortality and incidence (US EPA 2019). Overall, the evidence continues to support a linear, no-threshold concentration-response relationship for $PM_{2.5}$ exposure but with less certainty in the shape of the curve at lower concentrations (i.e. ~8 $\mu g/m^3$).

Concentration-response functions for various health endpoints are summarised in Table A1.1.

Susceptible sub-populations

Of the various different populations and life-stages of people examined in epidemiological studies with PM exposures, US EPA (2019) concluded some populations may be at disproportionately increased risk from a $PM_{2.5}$-related health effect, including non-white populations, children, people with specific genetic variants in genes in the glutathione transferase pathway, people who are overweight or obese, people with pre-existing cardiovascular or respiratory diseases, people of low socioeconomic status, and people who smoke or were former smokers. This also agrees with other reviews (e.g. NEPC 2014, WHO 2013a).

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Table A1.1: Concentration Response Functions for $PM_{2.5}$

Health endpoint	Study	Model	Relative Risk per 10 µg/m^3 unless otherwise stated (95%CI)	Calculated ß value [1]	Source
Long-term (Annual average)					
All-cause mortality 30+ years (ICD-10, codes A-R)	Meta-analysis of 13 cohort studies; data compiled from European mortality database (MDB) rates for deaths from all-natural causes in each of the 53 countries of the WHO European Region Hoek et al. (2013)	Log-linear	1.062 (1.040–1.083)	0.006015 (0.003922 – 0.007973)	CRF derived by Hoek et al. (2013) and recommended by WHO (2013b) as part of the HRAPIE project.
Cardiopulmonary mortality 30+ (ICD10, I26-I28)	Meta-analysis of 13 cohort studies in United States. The extended 18-year follow up included vital status data for the CPS-II cohort multiple cause - of-death codes (through December 2000) and more recent exposure data from air pollution monitoring sites from the suburban areas (Krewski et al. 2009).	Log-linear	1.14 (1.11–1.17)	0.013103 (0.010436 – 0.0157)	CRF derived by US EPA (2010) recommended for use in Australia by Jalaludin and Cowie (2012).
Mortality ischemic heart disease 30+ years (ICD10, I20-I25)		Log-linear	1.24 (1.19–1.28)	0.021511 (0.017395 – 0.024686)	
Mortality lung cancer 30+ years (ICD10, C34)		Log-linear	1.14 (1.06–1.123)	0.013103 (0.005827 – 0.0116)	
Life expectancy lost (YLL)	A dynamic model was developed that accounted for the decrease of risk after the termination of an exposure to pollution to analyse the loss of life expectancy (LLE) due to air pollution and the associated social cost (Leksell and Rabl 2001).	Log-linear	6.02 x 10^{-4} YOLL/ (person/year/µg/m^3)	-	CRF derived by Leksell and Rabl (2001) and recommended by European Commission (2005) and recommended for use in Australia by Jalaludin and Cowie (2012).

Health endpoint	Study	Model	Relative Risk per 10 µg/m^3 unless otherwise stated (95%CI)	Calculated ß value [(1)]	Source
Short-term (24-Hour Average)					
All-cause mortality (non-trauma) – all ages (ICD10, A-R)	Meta-analysis of four cities – Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants (Simpson et al 2005; EPHC 2005).	Non-linear	0.9% (0.2-1.6%) per 3.78 µg/m^3	0.00237 (0.000529 – 0.004199)	CRF from Environment Protection and Heritage Council (EPHC 2005) and recommended for use in Australia by Jalaludin and Cowie (2012).
Mortality cardiovascular disease – all ages (ICD10, I00-I99, excluding I67.3, I68.0, I88, I97.8, I97.9, I98.0; G45 excluding G45.3; G46, M30, M31, R58)		Log-linear	1.5% (0.7-2.3%) per 3.78 µg/m^3	0.003939 (0.001845 – 0.006016)	
ED visits asthma 1-14 years (ICD10, J45)	The study was based on ED hospital visits for children aged 1–14 years (mean of 174 ED visits for asthma/day). Most asthma ED visits were for children aged 1–4 years (60.9%, mean visits/day = 109) (Jalaludin et al. 2008).	Time-stratified approach in case-crossover design.	1.4% (0.9-1.8%) per 9.4 µg/m^3	0.001479 (0.000953 – 0.001898)	CRF derived and recommended for use in Australia by Jalaludin and Cowie (2012).

Health endpoint	Study	Model	Relative Risk per 10 µg/m^3 unless otherwise stated (95%CI)	Calculated ß value [1]	Source
Hospital admissions cardiovascular disease 65+ years (ICD10, I00-I99 excluding I67.3, I68.0, I88, I97.8, I97.9, I98.0; G45 excluding G45.3; G46, M30, M31, R58)	Meta-analysis of four cities – Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants (Simpson et al 2005; EPHC 2005).	Linear regression model	1.3% (0.6-2.0%) per 3.78 µg/m^3	0.003417 (0.001583 – 0.005239)	CRF from Environment Protection and Heritage Council (EPHC 2005) and recommended for use in Australia by Jalaludin and Cowie (2012).
Hospital admissions cardiac disease 65+ years (ICD10, I00-I52, I97.0, I97.1, I98.1)	Meta-analysis of four cities – Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants (Simpson et al 2005; EPHC 2005).	Linear regression model	1.9% (1.0-2.7%) per 3.78 µg/m^3	0.004979 (0.002632 – 0.007048)	CRF derived and recommended for use in Australia by EPHC (2005) and Jalaludin and Cowie (2012).
Hospital admissions cardiac failure 65+ years (ICD10, I50)		Linear regression model	3.6% (1.8-5.4%) per 3.78 µg/m^3	0.009356 (0.00472 – 0.013913)	

[1] Calculated as per Equation 2 in Frangos and Di Marco (2013): $ß = \frac{\ln(RR)}{\Delta x}$

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Table A1.2: Concentration Response Functions for PM_{10}

Health endpoint	Study design	Model	Relative Risk per 10µg/m^3 unless otherwise stated (95%CI)	Calculated ß value (1)	Source
Long-term (Annual average)					
All-cause mortality 30+ years (ICD-10, all)	Cohort study (n=552,138 adults, >30 yrs), investigating the effects of air pollution on mortality from metropolitan areas of fifty states in US (Pope, Thun et al. 1995).	Cox proportional hazards regression modelling	0.386% (0.295-0.477%) per 1 µg/m^3	0.003853 (0.002946 – 0.004759)	CRF derived by Pope, Thun et al. (1995), recommended for use in Australia by Jalaludin and Cowie (2012)
Cardiopulmonary mortality 30+	-	-	-	-	No CRF recommended. Jalaludin and Cowie (2012)
Mortality ischemic heart disease 30+ years	-	-	-	-	No CRF recommended. Jalaludin and Cowie (2012)
Mortality lung cancer 30+ years	-	-	-	-	No CRF recommended. Jalaludin and Cowie (2012

Health endpoint	Study design	Model	Relative Risk per 10µg/m^3 unless otherwise stated (95%CI)	Calculated ß value [(1)]	Source
Life expectancy lost (YLL)	A cohort study (n = 552,138) drawn from the American Cancer Society (ACS) Cancer Prevention Study II (CPS-II) assessing relationship between mortality and particulate pollution adjusting for individual differences in smoking status, gender, age, education, and other risk factors (European Commission 2005).	Multiple regression	2.69 x10^{-4} YOLL / (person/yr/µg/m^3)	-	CRF recommended by European Commission (2005) applying CRF from Pope (1995) to whole population and recommended for use in Australia by Jalaludin and Cowie (2012)
Short-term (24-Hour Average)					
All-cause mortality (non-trauma) all ages	-	-	-	-	No CRF recommended. Jalaludin and Cowie (2012)
Mortality cardiovascular disease-all ages (ICD10, I00-I99, excluding I67.3, I68.0, I88, I97.8, I97.9, I98.0; G45 excluding G45.3; G46, M30, M31, R58)	Meta-analysis of four cities – Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants (Simpson et al 2005; EPHC 2005).	Linear regression analysis	1.8 (0.6-3.0%) per 7.53 µg/m^3	0.002369 (0.000794 – 0.003925)	CRFs derived by EPHC (2005) recommended for use in Australia by Jalaludin and Cowie (2012).

Health endpoint	Study design	Model	Relative Risk per 10µg/m^3 unless otherwise stated (95%CI)	Calculated ß value [1]	Source
Respiratory Mortality – all ages	-	-	–	-	No CRF recommended. Jalaludin and Cowie (2012)
ED visits asthma 1-14 years (ICD10, J45)	The cohort study based on emergency department (ED) visits data for asthma and air pollution from metropolitan Sydney, Australia for the period between 1997-2001 (Jalaludin et al 2008).	The case-crossover design	1.4% (0.8 – 2.0%) per 7.6 µg/m^3	0.001829 (0.001048 – 0.002606)	CRF derived by Jalaludin et al. (2008) andrecommended for use in Australia by Jalaludin and Cowie (2012).
Hospital admissions cardiovascular disease -all ages (ICD10, I00-I99, excluding I67.3, I68.0, I88, I97.8, I97.9, I98.0; G45 excluding G45.3; G46, M30, M31, R58)	Meta-analysis study based on daily time series of mortality, hospital admissions, meteorologic factors, and air pollutants from urban sources in Sydney, Australia from 1994 through 2002 (Morgan et al. 2010).	Not reported.	1.22% (0.41 - 2.03%)	0.001213 (0.000409 – 0.00201)	CRFs derived by Morgan et al. (2010) recommended for use in sensitivity analysis in Australia by Jalaludin and Cowie (2012).
Hospital admissions cardiac disease 65+ years (ICD10, I00-I52, I97.0, I97.1, I98.1)		Poisson regression modelling	1.4% (0.5-2.2%) per 7.53 µg/m^3	0.001846 (0.000662 – 0.00289)	CRF derived by Simpson et al. (2005), EPHC (2005) and recommended for use in Australia by EPHC (2005) and Jalaludin and Cowie (2012).
Hospital admissions cardiac failure 65+ years (ICD10, I50)			3.6% (2.0 – 5.2%) per 7.53 µg/m^3	0.004697 (0.00263 – 0.006732)	
1-4 yrs			2.3% (0.9% to 3.8%) per 7.53 µg/m^3	0.00302 (0.00119 – 0.004953)	

Health endpoint		Study design	Model	Relative Risk per 10µg/m^3 unless otherwise stated (95%CI)	Calculated ß value (1)	Source
Hospital admissions respiratory disease (ICD10, J00-J99 excluding J95.4-J95.9; R09.1, R09.8)	5-14 yrs	Meta-analysis of four cities - Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants.		2.3% (0.2% to 4.4%) per 7.53 µg/m^3	0.00302 (0.000265 – 0.005718)	CRF derived by EPHC (2005) and recommended for use in Australia by Jalaludin and Cowie (2012).
Hospital admissions pneumonia and bronchitis 65+ years (ICD10, J12-J17, J18.0, J18.1, J18.8, J18.9, J20, J21)				1% (0.2-3.8%) per 7.53 µg/m^3	0.001321 (0.000265 – 0.004953)	

[1] Calculated as per Equation 2 in Frangos and Di Marco (2013): $ß = \frac{\ln(RR)}{\Delta x}$

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Table A1.3: Summary of available Guideline Values for PM_{10}

Criterion Name	Averaging Time	Derivation	Guideline Value ($\mu g/m^3$)	Source
Australian Ambient Air Quality (AAQ) Standard	24 hour	The Australian National Environmental Protection Council ambient air quality standard for PM_{10} is based on the health-based evidence that was available in the mid-to-late 1990s to inform the making of the AAQ NEPM. The standard is based on Streeton (1997) who reported an increases incidence of respiratory or cardiac disease, of the order of 1% for every 10 $\mu g/m^3$ increase in PM_{10} levels. • increases in hospital admissions for respiratory, and (probably) cardiac conditions, • increases in hospital casualty and medical surgery visits for asthma and other respiratory conditions, • increases in functional limitation as indicated by restricted activity days or, in the case of children, by increased frequency of absence from school, • increases in the daily prevalence of respiratory symptoms, and • small decreases in the level of pulmonary function in healthy children, and in adults with obstructive airways disease.	50	NEPC (2014) NEPM (2016) Streeton (1997)
Australian Ambient Air Quality (AAQ) Standard	Annual mean	No background documentation was found for the basis of this value.	25	NEPC (2014) NEPM (2016)
WHO Ambient Air Quality Guidelines (AQG)	24 hour	The health risks associated with short-term exposures to PM_{10} are likely to be similar in cities in developed and developing countries, associated with an increase in mortality of around 0.5% for each 10 $\mu g/m^3$ increment in the daily average concentration. Thus, a PM_{10} concentration of 150 $\mu g/m^3$ would be expected to transform into nearly a 5% increase in daily mortality, an impact that would be of significant concern. For PM_{10}, the WHO AQG for the 24-hour average is 50 $\mu g/m^3$ and reflects the relationship between the distributions of 24-hour means (and its 99th percentile) and annual average concentrations.	50	WHO (2006) WHO (2012)
	Annual mean	The WHO AQG for PM is based on studies that use $PM_{2.5}$ as an indicator. The $PM_{2.5}$ guideline values were converted to the corresponding PM_{10} guideline values by application of a $PM_{2.5}/PM_{10}$ ratio of 0.5. A $PM_{2.5}/PM_{10}$ ratio of 0.5 is typical of developing country urban areas and is at the bottom of the range found in developed country urban areas (0.5–0.8).	20	WHO (2006) WHO (2013b)

Criterion Name	Averaging Time	Derivation	Guideline Value ($\mu g/m^3$)	Source
National Environmental Standards for Air Quality New Zealand	24- hour	Based on higher level of mortality, morbidity, hospitalisation, work-affected days, increased use of medication related to elevated concentrations of PM_{10}. According to the New Zealand Air Quality Guidelines, there is no evidence of a threshold below which adverse health effects will not be seen.	50	NZ MfE (2002)
	Annual mean	No background documentation was found for the basis of this value, but it is likely NZ MfE (2002) adopted the WHO (2006) limit values.	20	NZ MfE (2002)
US National Air Quality Standards	24 hour	The national air quality standard of 150 $\mu g/m^3$ with no more than one expected exceedance per year on average over three years was first set in 1979. Upon finalising an update to the National Ambient Air Quality Standards, the standards for PM_{10} were unchanged.	150	US EPA (2012)
European Union Air Guidelines	24 hour	The European limit value is based on the lowest reasonably practical value. No background documentation was found for the basis of this value. Likely adopted from EU Directive (EU 2008).	50	EU Directive (2008) EU Directive (2015)
	Annual mean	No background documentation was found for the basis of this value. Likely adopted from EU Directive (EU 2008).	40	EU Directive (2008) EU Directive (2015)
UK Air Quality Guideline	Annual mean	No background documentation was found for the derivation of this value. Likely adopted from EU Directive (EU 2008).	50	EU Directive (2008)

Table A1.4: Summary of available Guideline Values for $PM_{2.5}$

Criterion Name	Averaging Time	Derivation	Guideline Value ($\mu g/m^3$)	Source
Australian Ambient Air Quality (AAQ) Measure	24-hour	No background documentation was found for the basis of this value, but it is likely the NEPM adopted the WHO (2006) limit values.	25	NEPC 2014; NEPM 2016
Australian Ambient Air Quality (AAQ)	Annual mean	No background documentation was found for the basis of this value.	8	NEPC 2014; NEPM 2016
WHO ambient air quality guidelines	24-hour	No detailed background documentation was found for the basis of this short term (24-h) value. The WHO set the value based on the information on hospital admissions, incidence of asthma symptoms in asthmatic children and adults.	25	WHO (2006)
WHO ambient air quality guidelines	Annual mean	An annual average concentration of 10 $\mu g/m^3$ was chosen as the long-term guideline value for $PM_{2.5}$ as it places a significant weight on the long-term exposure epidemiological studies (Dockery et al. 1993, Pope et al. 1995). In these studies, robust associations were reported between long-term exposure to $PM_{2.5}$ and mortality. The mean $PM_{2.5}$ concentration was 18 $\mu g/m^3$ in the American Cancer Society's (ACS) study (Pope et al. 2002), but the risk estimates become apparent at concentrations of about 13 $\mu g/m^3$, below which the confidence bounds significantly widen since the concentrations are relatively far from the mean. In the Dockery et al. (1993) study, the increase in risk was apparent in the city with the next-lowest long-term $PM_{2.5}$ mean (e.g., 14.9 $\mu g/m^3$), indicating that health effects can be expected when annual mean concentrations are in the range of 11-15 $\mu g/m^3$. Thus, an annual mean concentration of 10 $\mu g/m^3$ was considered by WHO to be an appropriate guideline value.	10	WHO (2006)
US National Air Quality Standards	24-hour	In September 2006, EPA revised the previous PM standards by lowering the level of the 24-hour $PM_{2.5}$ standard from 65 $\mu g/m^3$ in 1997 to 35 $\mu g/m^3$, based on $PM_{2.5}$ exposure and increased risk of all-cause, cardiovascular and lung cancer mortality as per a study by Lepeule et al. (2012). These authors found a linear concentration-response relationship down to $PM_{2.5}$ concentrations of 8 $\mu g/m^3$, and that mortality rate ratios for $PM_{2.5}$ fluctuated over time, but without clear trends.	35	US EPA (2012)
European Union Air Guidelines	24-hour	No background documentation was found for the basis of this value. Likely adopted from EU Directive (EU 2008).	20	EU (2008) EU Directive (2015)
UK Air Quality Guideline	Annual average	No background documentation was found for the derivation of this value.	25	UK Air Quality Standard Regulations (2020)

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A2. Nitrogen Dioxide

The term 'oxides of nitrogen' refers to all forms of oxidised nitrogen (N) compounds, including nitric oxide (NO), nitrogen dioxide (NO_2) and all other oxidised N-containing compounds formed from NO and NO_2. NO and NO_2 are the main nitrogen oxides associated with combustion. The abbreviation NO_x refers specifically to the sum of NO and NO_2. Oxidised N compounds are emitted into the atmosphere mainly as NO, with only 10% or less emitted as NO_2 (US EPA 2016). Freshly emitted NO is primarily converted to NO_2 by reacting with ozone, and NO is recycled during the day by photolysis of NO_2 (US EPA 2016). NO_2 has a greater impact on health than NO (NEPC 2019).

Ambient concentrations of NO and NO_2 vary widely based on local environmental conditions, but in dense urban areas can exceed a total (for both gases) of 500 $\mu g/m^3$ (Jarvis et al. 2010). The main sources of NO_2 pollution in Australian cities originating from human activities are the combustion of fossil fuels (coal, gas and oil), with the largest contributor being road vehicle exhaust. Oxides of nitrogen are also produced from making nitric acid, welding, and using explosives, refining of petrol and metals, commercial manufacturing, and food manufacturing. Natural sources of NO_2 include volcanoes and bacterial respiration (NEPC 2019, NZ MfE 2002, US EPA 2016).

The general population is exposed to NO_2 by inhalation. Once inhaled, NO_2 reacts with the moisture in the respiratory tract, resulting in the formation of nitric acid (HNO_3), which dissociates into nitrates and nitrites (NRC 1998). At low concentrations, NO_2 reacts with moisture in the upper respiratory tract, but as the exposure concentration increases, that reaction diffuses into the lower respiratory tract (NRC 1998).

NO_2 or its chemical derivatives, particularly NO, can be transported to extrapulmonary sites via the bloodstream, where they can react with haemoglobin to form methaemoglobin (MetHb), which is an ineffective oxygen carrier (Weinberger et al. 2001). Transformation of haemoglobin to MetHb has important health implications and can increase health risks to vulnerable individuals who have hypoxia associated with pulmonary and cardiac disease (Frostell et al. 1991, Hunter et al. 2004, Amaducci and Downs 2020). Increased levels of nitrates have been reported in the blood and urine following exposure to NO_2, indicating that NO_2 reacts to produce nitrates (NRC 1998).

Health effects of ambient exposure to NO_2 have been studied and reviewed by Australian and international regulatory agencies (NEPC 2011b, WHO 2013b, OEHHA 2008, EEA 2019, US EPA 2016). Short-term exposure to ambient NO_2 has been associated with increases in all-cause, and respiratory and cardiovascular mortality (NEPC 2019, WHO 2013b). Recent studies have provided evidence which has strengthened the association with increased hospital admissions and emergency department visits for respiratory disease including all respiratory causes, asthma, and chronic obstructive pulmonary disease (COPD) (NEPC 2019, WHO 2013b). An increase in symptoms in asthmatic children and increases in airway inflammation and hyper-responsiveness have also been observed (NEPC 2019).

Epidemiological studies of long-term effects of NO_2 exposure on mortality (both respiratory and cardiovascular) and on children's respiratory symptoms and lung function also support the conclusion that NO_2 has an independent effect on health, with long-term NO_2 exposure linked to deficits in lung function growth, as well as increased incidence of asthma and wheeze (NEPC 2019). However, there is much greater uncertainty as to the quantitative health impact on large populations of long-term exposure to NO_2 compared with the evidence for PM (NEPC 2019).

Cardiovascular effects

Exposure to ambient NO_2 was found to be a risk factor for systolic and diastolic blood pressure (SBP and DBP), and inversely associated with the central retinal venular equivalent (CRVE) and mean baseline brachial artery diameter (Everson et al. 2019). Duan et al. (2019), using cause-specific death data, weather conditions, and air pollutant concentrations in Shenzhen, China from 2013 to 2017 derived linear models to analyse the effect of season on the NO_2-associated cardiovascular mortality. The results showed that in the cold season, the percentage increase in daily mortality for every 10 µg/m^3 increment in NO_2 concentration over lags of 0–2 days was 4.45% (95% CI: 2.71–6.21%). Nevertheless, US EPA (2016) in their Integrated Science Assessment for oxides of nitrogen, concluded that the available evidence is suggestive but not sufficient to infer a causal relationship between short-term or long-term NO_2 exposure and cardiovascular effects. This is similar to the conclusions made by NEPC (2011b) in their review.

Respiratory effects

Effects observed for exposure to NO_2 in epidemiological studies are greatest for respiratory outcomes (NEPC 2011b).

The current NEPM standards for NO_2 are 120 ppb (246 µg/m^3) for 1-hour acute exposure, and 30 ppb (62 µg/m^3) for an annual average chronic NO_2 exposure (NEPC 2019). The 1-hour exposure standard is based on a meta-analysis undertaken by Folinsbee (1992) and reviewed by Streeton (1997) which determined a short-term lowest observed adverse effect level (LOAEL) for NO_2, at between 0.2 and 0.3 ppm (i.e. 376 – 564 µg/m^3) over 1h exposure. This is similar to the 1-hour reference exposure level (REL) of 470 µg/m^3 derived by OEHHA (2008) for the general population[9]. Chronic exposure concentrations from 0.04-0.08 ppb (75 – 150 µg/m^3) have been associated with recurring respiratory infections, and onset of mild symptoms in asthmatics (Streeton 1997).

The current LOAEL for NO_2 suggested by Streeton (1997) is in the range of 0.2-0.3 ppm (1-hour), but an increased body of knowledge suggest that chronic exposure to concentrations from 0.04-0.08 ppm during early and middle childhood can initiate a recurrent upper and lower respiratory symptom. Therefore, Streeton (1997) suggested concentrations of 0.2-0.3 ppm (1-hour) represent an acute LOAEL, and 0.04-0.08 ppm represent a chronic exposure LOAEL, and an uncertainty factor of 2 should be applied to account for susceptible population categories.

[9] OEHHA (2008) considered a 1-hour NO_2 exposure of 0.25 ppm a NOAEL for increased airway reactivity in sensitive humans (asthmatics) and did not apply any uncertainty factors to this value. The resulting REL of 0.25 ppm (i.e. 470 µg/m^3) used a similar point of departure as the current NEPM standard except that the value used to derive the NEPM standard was considered a LOAEL instead of a NOAEL.

The 1998 NEPM standards were based on the understanding that concentrations of NO_2 below 1.0 ppm (i.e. 1,880 µg/m^3), for short-term exposures (2 hours or less) do not appear to cause adverse effects in healthy subjects, as indicated by conventional measurement of pulmonary function (Hesterberg et al. 2009, Streeton 1997). Healthy individuals exposed to NO_2 for 2 hours at a concentration of 5.0 ppm (i.e. 9,400 µg/m^3) exhibited increased airway resistance and impaired oxygen exchange in the lung (NRC 1998). The study also reported a decrease in CO_2 diffusion capacity following a 15-min exposure at 5.0 ppm by healthy individuals (NRC 1998). However, the latest NEPM review (NEPC 2011b) highlighted that infants, children and elderly people are more susceptible to the effects of NO_2 and people with asthma and other chronic respiratory and cardiovascular disease are particularly vulnerable. Observed effects are independent of other pollutants and do occur at ambient levels of NO_2 (NEPC 2011b). As the effects appeared to be greater in Australia than those observed in the US and Europe and effects were observed at concentrations between 0.03 and 0.04 ppm (i.e. 56 and 75 µg/m^3), NEPC (2011b) concluded that revision of the NEPM NO_2 standards should include consideration of the sensitive groups of the population.

In their 2016 Integrated Science Assessment for oxides of nitrogen, US EPA (2016) concluded that the evidence for NO_2 being able to independently trigger asthma attacks supports a causal relationship between short-term NO_2 exposure and respiratory effects, whereas they concluded evidence for development of asthma supports a likely to be causal relationship between long-term NO_2 exposure and respiratory effects.

Increases in airway responsiveness and doubling reduction in provocative dose have been demonstrated in adults with asthma following 0.2-0.3 ppm (i.e. 376 – 564 µg/m^3) NO_2 exposures at rest for 30 minutes and 0.1 ppm (i.e. 188 µg/m^3) for 1 hour (Folinsbee 1992, Brown 2015). Further linking short-term NO_2 exposure to asthma exacerbation is evidence for NO_2 exposures of 0.26-0.4 ppm (i.e. 489 – 752 µg/m^3) enhancing allergic inflammation in humans with allergic asthma and a rat model of allergic disease (US EPA 2016). In epidemiological studies, asthma hospital admissions and emergency department visits were associated with 24-hour average and 1-hour maximum NO_2, with risk estimates ranging from a 4.5-34% increase per 0.02 ppm (i.e. 38 µg/m^3) increase in 24-hour average NO_2 or 0.03 ppm (i.e. 56 µg/m^3) increase in 1-hour maximum NO_2 (US EPA 2016). There is likely a sensitive sub-group of asthmatics with increased airway reactivity following inhalation exposure to NO_2 present in the general population; this contributes to the wide range of responsiveness present among asthmatics to inhaled NO_2 (OEHHA 2008).

There have also been associations of ambient NO_2 concentrations with non-asthma respiratory effects and mortality. All-cause daily mortality has been estimated to increase by 0.27% (95% CI = 0.16–0.38%) per 10 µg/m^3 NO_2 (maximum 1 hour) adjusted for PM_{10}; long-term exposure to NO_2 has been associated with all cause (natural) mortality as well as cardiopulmonary mortality risk corresponding to relative risks (RR) of 1.055 (95% CI = 1.031, 1.08) per 10 µg/m^3 annual average NO_2 (WHO 2013b). According to WHO (2013b), this indicates that the mortality risk of NO_2 exposures is likely to be higher than previously estimated. WHO (2013b) also indicate that impacts should only be calculated for levels of NO_2 above 20 µg/m^3, implicating that there is a threshold for all-cause mortality below which this effect does not occur.

Other effects

Recent epidemiological studies have shown an association between exposure to traffic-related NO_2 and prevalence of lung cancer. Bai et al. (2020) showed a showed positive associations of lung cancer incidence with NO_2 (HR = 1.05 [95% CI: 1.03–1.07] per 26 µg/m^3 increase). Hamra (2015) found the meta-estimate for the change in lung cancer incidence associated with a 10-µg/m^3 increase in exposure to NO_2 was 4% (95% CI: 1%, 8%). One study has also found an association between NO_2 exposure and increased risk of postmenopausal incidence of breast cancer (Goldberg et al. 2017). However, with respect to health effects other than respiratory effects, US EPA (2016), in their detailed Integrated Science Assessment of NO_x concluded that:

- The available evidence is inadequate to conclude a causal relationship between long-term NO_2 exposures and certain reproductive and developmental effects including changes in fertility, reproduction and pregnancy. However, exposures are likely associated with some birth outcomes, but the effect is only suggestive and not sufficient to infer causality.
- For other health effects (e.g. reproductive and fertility effects, total mortality, cancer etc), the evidence was considered to be suggestive but insufficient to infer causality for NO_2.

Concentration response

Concentration-response relationships for short-term NO_2 exposure and asthma-related effects have not been well examined in controlled human exposure or animal toxicological studies (US EPA 2016). According to US EPA (2016), experimental studies do not provide insight on whether asthma responses increase with increasing NO_2 concentration because few studies examined multiple NO_2 exposure concentrations, and the range of these NO_2 concentrations (all greater than 0.1 ppm, i.e. 188 µg/m3) exceed those examined in epidemiological studies of concentration-response. In epidemiological studies, linear relationships were observed for all-cause mortality associated with short-term NO_2 averages in the U.S., Canada, and Asia, and non-linear relationships for health effects for which the concentration-response relationship has not been widely examined (cough in children or cardiovascular hospital admissions in adults). According to the US EPA (2016), analysis of the concentration-response of long-term average NO_2 concentrations does not provide a strong foundation for assessing whether there is a threshold for respiratory effects.

According to US EPA (2016), the shape of the concentration-response relationship for NO_2 is better described in epidemiological studies for short-term NO_2 exposures rather than long-term exposures. US EPA (2016) concluded the available evidence indicates a linear relationship between short-term NO_2 exposure and hospital admissions or emergency department (ED) visits for asthma and multiple combined respiratory conditions. For example, in Atlanta, GA, a linear relationship with asthma ED visits was shown for 1-h max NO_2 concentrations averaged over 3 days, with comparable confidence in the relationship across the range of 11 to 37 ppb (i.e. 21 – 70 µg/m^3). There is uncertainty in the relationship at concentrations less than 11 ppb (i.e. 21 µg/m^3). Another source of uncertainty is that 24-h avg or 1-h max NO_2 concentrations were averaged across multiple central site monitors within a city, which may not indicate changing distributions of concentrations within the city or population exposures (US EPA 2016).

In contrast, authorities in Australia (NEPC 2019) and California (OEHHA 2008) have so far evaluated short-term NO_2 exposure as having a threshold, even in sensitive asthmatics. WHO (2013b), with their recommendation that health effects of NO_2 should only be evaluated for concentrations above 20 µg/m^3, also seem to support the notion that NO_2 has an effect threshold.

Concentration-response functions for various health endpoints are summarised in Table A2.1.

Susceptible populations

As discussed above, the latest NEPM review (NEPC 2011b) highlighted that infants, children and elderly people are more susceptible to the effects of NO_2 and people with asthma and other chronic respiratory and cardiovascular disease are particularly vulnerable. US EPA (2016) made a similar conclusion identifying people with asthma, children (especially ages 0–14 years), and older adults (especially ages 65 years and older) as being at increased risk of NO_2-related health effects.

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Table A2.1: Concentration Response Functions for NO_2

Health endpoint	Study	Model	Relative Risk per 10 µg/m^3 unless otherwise stated (95%CI)	Calculated ß value (1)	Source
All-cause mortality 30+ years	Meta-analysis of 13 cohort studies; data compiled from European mortality database (MDB) rates for deaths from all-natural causes in each of the 53 countries of the WHO European Region Hoek et al. (2013)	RR based on single-pollutant models	1.055 (1.031–1.080)	0.005354 (0.003053 – 0.007696)	WHO (2013b). * Although Jalaludin and Cowie (2012) did not recommend a CRF for use in Australia, this CRF has been included here as it was recommended by WHO after the Jalaludin and Cowie (2012) review was published
Short-term (1-Hour Average)					

Health endpoint	Study	Model	Relative Risk per 10 µg/m^3 unless otherwise stated (95%CI)	Calculated ß value [1]	Source
All-cause mortality (non-trauma) all ages (ICD10, A-R)	Meta-analysis of four cities – Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants (Simpson et al 2005; EPHC 2005).	Linear regression model	1.7% (0.3-3.2%) per 8.98 ppb (i.e. 16.9 µg/m^3)	-	CRF as per EPHC (2005) recommended for use in Australia by Jalaludin and Cowie (2012)
Mortality cardiovascular disease- all ages (ICD10, I00-I99, excl. I67.3, I68.0, I88, I97.9, I98.0; G45 excl. G45.3; G46, M30, M31, R58			1.6% (0.4-2.8%) per 8.98 ppb (i.e. 16.9 µg/m^3)	0.000939 (0.000236 – 0.001634)	CRF as per EPHC (2005) recommended for use in Australia by Jalaludin and Cowie (2012)
Respiratory Mortality – all ages (ICD10, J00-J99, excl. J95.4 to J95.9; R09.1, R09.8)			3.9% (0.6-7.4%) per 8.98 ppb (i.e. 16.9 µg/m^3)	0.002264 (0.000354 – 0.004224)	
ED visits asthma 1-14 years (ICD10, J45)	The study was based on ED hospital visits for children aged 1–14 years (mean of 174 ED visits for asthma/day). Most asthma ED visits were for children aged 1–4 years (60.9%, mean visits/day = 109) (Jalaludin et al. 2008).	Two pollutant model with $PM_{2.5}$	1.1% (0.6-1.6%) per 9.5 ppb (i.e. 17.9 µg/m^3)	0.000611 (0.000334 – 0.000887)	CRF from Jalaludin et al. (2008) recommended for use in Australia by Jalaludin and Cowie (2012)

Health endpoint	Study	Model	Relative Risk per 10 µg/m^3 unless otherwise stated (95%CI)	Calculated ß value [1]	Source
Hospital admissions cardiovascular disease 15-64 years (ICD10, I00-I99, excl. I67.3, I68.0, I88, I97.9, I98.0; G45 excl. G45.3; G46, M30, M31, R58)	Meta-analysis of four cities – Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants (Simpson et al 2005; EPHC 2005).	Linear regression model	1.3% (0.3-2.3%) per 8.98 ppb (i.e. 16.9 µg/m^3)	0.000764 (0.000177 – 0.001346)	CRF as per EPHC (2005) recommended for use in Australia by Jalaludin and Cowie (2012)
Hospital admissions cardiovascular disease 65+ years (ICD10, I00-I99, excl. I67.3, I68.0, I88, I97.9, I98.0; G45 excl. G45.3; G46, M30, M31, R58)			2.6% (1.8-3.3%) per 8.98 ppb (i.e. 16.9 µg/m^3)	0.001519 (0.001056 – 0.001921)	
Hospital admissions cardiac disease 15-64 years (ICD10, I00-I52, I97.0, I97.1, I98.1)			1.2% (0.0-2.4%) per 8.98 ppb (i.e. 16.9 µg/m^3)	0.000706 (0 – 0.001403)	
Hospital admissions cardiac disease 65+ years (ICD10, I00-I52, I97.0, I97.1, I98.1)			3.3% (2.4-4.3%) per 8.98 ppb (i.e. 16.9 µg/m^3)	0.001921 (0.001403 – 0.002491)	
Hospital admissions cardiac failure 65+ years (ICD10, I50)			7.5% (5.3-9.7%) per 8.98 ppb (i.e. 16.9 µg/m^3)	0.004279 (0.003056 – 0.005478)	

Health endpoint	Study	Model	Relative Risk per 10 µg/m^3 unless otherwise stated (95%CI)	Calculated ß value (1)	Source
Hospital admissions respiratory disease 1-4 years (ICD10, J00-J99 excl. J95.4-J95.9; R09.1, R09.8)	Meta-analysis of four cities – Brisbane, Melbourne, Perth and Sydney conducted by the Environment Protection and Heritage Council (2005). The study used all available data sets from 1998 to 2001 for daily mortality, daily hospital admissions and air pollution data for all available pollutants (Simpson et al. 2005; EPHC 2005).	Linear regression model	3.6% (1.5-5.7%) per 9 ppb (i.e. 16.9 µg/m^3)	0.002093 (0.000881 – 0.00328)	
Hospital admissions respiratory disease 5-14 years (ICD10, J00-J99 excl. J95.4-J95.9; R09.1, R09.8)			4% (1.1-7.1%) per 9 ppb (i.e. 16.9 µg/m^3)	0.002321 (0.000647 – 0.004059)	CRF as per EPHC (2005) recommended for use in Australia by Jalaludin and Cowie (2012)
Hospital admissions respiratory disease 15-64 years (ICD10, J00-J99 excl. J95.4-J95.9; R09.1, R09.8)			1.6% (0.5-2.8%) per 9 ppb (i.e. 16.9 µg/m^3)	0.000939 (0.000295 – 0.001634)	
Hospital admissions respiratory disease 65+ years (ICD10, J00-J99 excl. J95.4-J95.9; R09.1, R09.8)			1.0016 (1.0006 – 1.0026) per 1 ppb (i.e. 1.88 µg/m^3)	0.00085 (0.000319 – 0.001381)	CRF from Simpson, et al. (2005), as recommended in Australia by Jalaludin and Cowie (2012)

[1] Calculated as per Equation 2 in Frangos and Di Marco (2013): $ß = \frac{\ln(RR)}{\Delta x}$

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Table A2.2: Summary of available Guideline Values for Nitrogen Dioxide

Criterion Name	Averaging Time	Derivation	Guideline Value ($\mu g/m^3$)	Guideline Value ppb	Source
Acute guidelines					
Australian Ambient Air Quality (AAQ) Standard	1 hr	Based on a lowest observed adverse effect level (LOAEL) of 0.2 to 0.3 ppm adopted from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in young children including asthma, and adults compromised by chronic cardiac and respiratory disorders (Streeton 1997). To protect susceptible people (i.e. asthmatic children) an uncertainty factor of 2 was added to the LOAEL (Streeton 1997).	246	120	NEPC (1998) NEPM(2016) NEPC (2019)
WHO Ambient Air Quality Guidelines (AQG)	1 hr	A number of short-term experimental human toxicology studies have reported acute health effects following exposure to 1-hour NO_2 concentrations of 500 $\mu g/m^3$. Although the NO_2 exposure show a direct effect on pulmonary function in asthmatics at concentrations around 560 $\mu g/m^3$, studies of bronchial responsiveness among asthmatics suggest an increase in responsiveness at levels higher than 200 $\mu g/m^3$. This latter concentration was considered a NOAEL and no uncertainty factor was applied.	200	97	WHO (2006) WHO (2013b)
The California Ambient Air Reference Exposure Level (REL)	1 hr	The California Ambient Reference Exposure Level (REL) was based on a 1-hour NOAEL of 0.25 ppm (i.e. 470 $\mu g/m^3$) from controlled acute exposure studies with NO_2 in asthmatics showing an increase in airway reactivity at exposure concentrations between 0.25 and 0.5ppm (OEHHA 2008). No uncertainty factor was applied to the NOAEL.	470	250	OEHHA (2008)

Criterion Name	Averaging Time	Derivation	Guideline Value ($\mu g/m^3$)	Guideline Value ppb	Source
US National Air Quality Standard	1 hr	Derived based on meta-analysis including epidemiological and rat studies (Brown 2015) where subjects (8-50 years old) were exposed to NO_2 for: • 30-min to 20-300 ppb (i.e. 37.6 – 564 $\mu g/m^3$) • 60-min to 100 ppb (188 $\mu g/m^3$) Short term exposure was causal for respiratory effects including asthma exacerbation. There was no indication of a concentration-response relationship for exposures between 100 and 500 ppb NO_2 and increased airway responsiveness (AR) in individuals with asthma. Thus, US EPA (2016) considered 97 ppb a NOAEL. US EPA (2016) notes lack of an apparent dose-response relationship adds uncertainty to interpretation of controlled human exposure studies of AR but does not necessarily indicate lack of an NO_2 effect.	188	100	US EPA (2016)
Chronic guidelines					
Australian Ambient Air Quality (AAQ) standard	Annual	A LOAEL for respiratory effect (asthma exacerbation) of the order of 40 - 80 ppb (approx. 75-150 $\mu g/m^3$) was used as a point of departure and was assumed to be protective of long-term adverse health effects. However, NEPC (1998, 2019) noted an increasing body of data suggest that longer term chronic indoor exposures to these concentrations of 0.04 - 0.08 ppm during early and middle childhood can lead to the development of recurrent upper and lower respiratory symptoms. An uncertainty factor of 2 was applied to the LOAEL range to account for susceptible people within the population resulting in a guideline range of 20-40 ppb (38-75 $\mu g/m^3$) (Streeton 1997). A guideline of 30 ppb (in the middle of this range) was adopted as the AAQ standard.	56	30	NEPC (1998) NEPM (2016) NEPC (2019)
WHO Ambient Air Quality Guidelines (AQG)	Annual	This value was set based on epidemiological studies that have shown that bronchitis symptoms of asthmatic children increase in association with annual NO_2 concentration, and that reduced lung function growth in children is linked to elevated NO_2 concentrations.	40	21	WHO (2006) WHO (2013b)

Criterion Name	Averaging Time	Derivation	Guideline Value ($\mu g/m^3$)	Guideline Value ppb	Source
US National Air Quality Standards	Annual	The value was based on NOAEL for asthma development and other respiratory effects associated with long term exposure to ambient NO_2. Brown (2015) identified a positive association between asthma incidence in children and long tern NO_2 exposure measured near children's homes and schools. No uncertainty factor was applied to the NOAEL.	100	53	US EPA (2016)

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