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Appendix G
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Report

Western Sydney Airport EIS - Health Risk Assessment

GHD Pty Ltd

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EXECUTIVE SUMMARY

A health risk assessment (HRA) has been undertaken to assess the potential risks associated with air and noise emissions and potential surface and groundwater contamination that may arise from the construction and operation of the proposed Western Sydney Airport at Badgerys Creek (the airport).

While the EIS Guidelines (EPBC 2014/7391) do not specifically require the conduct of a health study, they require that the EIS include assessment in relation to the principles of ecologically sustainable development, which include the principle of inter-generational equity - "*that the present generation should ensure that the health, diversity and productivity of the environment is maintained or enhanced for the benefit of future generations*". The guidelines also require the EIS to consider impacts on the environment, which includes, amongst other things, impacts on people and communities.

Health is defined by the World Health Organization (WHO) as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO, 1948). Well-being is broadly described as an individual's self-assessment of their state of happiness, healthiness and prosperity. It relates to the quality of life and one's ability to enjoy it. There are many social and economic factors that influence well-being: including (enHealth 2012, NHC 2004):

- Social and cultural factors (e.g. social support, participation, access to cultural resources).
- Economic factors (e.g. income levels, access to employment).
- Environmental factors (e.g. land use, air quality).
- Population-based services (e.g. health and disability services, leisure services).
- Individual/behavioural factors (e.g. physical activity, smoking).
- Biological factors (e.g. biological age).

While the focus of this study is on the environmental factors and changes that might occur as a result of airport development, and the effects this may have on well-being, the social, cultural, economic and other environmental factors have also been analysed and documented in other parts of this EIS.

A HRA is an analysis that uses information about pollutants to estimate a theoretical level of risk for people who might be exposed to defined levels of these pollutants. The information on the pollutants comes from other technical papers that have been prepared for the Western Sydney Airport EIS, scientific studies and results if ambient monitoring and modelling. Where relevant, the results of international studies have been used to supplement the local evidence base of potential harmful effect on humans. The HRA is a document that assembles and synthesizes scientific information to determine whether a potential hazard exists and/or the extent of possible risk to human health.

The risk assessment process detailed in the enHealth HRA Guidelines, comprises five components which have been followed in the HRA:

1. **Issue Identification** – Identifies issues that can be assessed through a risk assessment and assists in establishing a context for the risk assessment.
2. **Hazard Assessment** – Identifies hazards and health endpoints associated with exposure to hazardous agents and provides a review of the current understanding of the toxicity and risk relationship of the exposure of humans to the hazards.
3. **Exposure Assessment** – This task identifies the groups of people who may be exposed to hazardous agents and quantifies the exposure concentrations.
4. **Risk Characterisation** – This task provides the qualitative evaluation of potential risks to human health. The characterisation of risk is based on the review of concentration response relationship and the assessment of the magnitude of exposure.

5. **Uncertainty Assessment** – identifies potential sources of uncertainty and qualitative discussion of the magnitude of uncertainty and expected effects on risk estimates.

Consultation was also conducted with NSW Health and local area health services and additional advice and requirements have also been incorporated into this report.

Three scenarios have been considered for the air quality and noise HRA:

1. Construction
2. Airport operations in 2030
3. Longer term airport operations in 2063.

Where data was available the airport operations for 2050 were also assessed for noise.

Air Quality

The HRA has examined the increase in risk resulting from air pollution generated by the construction and operation of the proposed airport. The health effects that have been considered are increases in both long-term and short-term mortality, increases in hospital admissions and increases in emergency department attendances for asthma. Emissions from construction activities, aircraft operations as well as on site and local road traffic have been included in the modelling. The pollutants considered were particulate matter, NO_2, SO_2, CO, benzene and diesel. Predictions of changes in local air quality were derived from the local air quality assessment (Pacific Environment 2015) which is included elsewhere in the EIS. For regional air quality, the health risk arising from changes to ozone levels was assessed. Changes in ozone levels were taken from the regional air quality assessment (Ramboll Environ 2015).

Mitigation measures to reduce NO_2 and PM are included in the local air quality technical report and when implemented, would have an effect of reducing PM and NO_2 emissions generated. It is also worth noting that particularly for the 2063 scenario assessment, the modelling assumed no future reductions in emissions technology either for aircraft or for vehicular traffic more generally. On the basis that emissions reduction has occurred over the past several decades and is expected to continue to occur in the future, the results of the HRA are likely to overestimate the actual level of risk that would be realised.

The predicted concentrations of all pollutants considered in this HRA are below the relevant NEPM standards. However, epidemiological studies into the health effects of air pollution have shown that there is no threshold for health effects associated with exposure to these pollutants. which means that there is a risk of health effects occurring below current air quality standards.

The highest risk during construction is predicted to be associated with PM_{10} during construction of aviation infrastructure. The highest predicted risk could result in an additional one additional death per hundred years from all causes (non-accidental). The most affected areas would be Luddenham, Bringelly, Kemps Creek and Badgerys Creek. As construction will only occur for a period of less than 10 years, the actual impact on the local community will be lower as the period of exposure is less.

Risks associated with air quality impacts from airport operation have been assessed for two scenarios:

- 2030 – the proposed Stage 1 airport development; and
- 2063 – a potential longer-term airport development scenario.

2030 operations

The results of the HRA show that the highest risk associated with airport operations relates to NO_2 concentrations. The highest risk would arise from a predicted increase in long-term mortality of an additional 5 deaths in 10 years. Increases in short-term mortality and hospital admissions are lower than

that for long-term mortality. The most affected areas are likely to be Luddenham, Bringelly, Kemps Creek, Mulgoa, Wallacia and Rossmore. It should be noted that these health predictions have been made based on the absence of mitigation measures, which will reduce the concentration of emissions generated and therefore also, the predicted health risks.

International agencies usually consider increases in risk of between 1 in one million and in in 100,000 as being acceptable. The predicted increase in mortality from NO_2 emissions exceeds this range. The predicted risks from PM_{10} and $PM_{2.5}$ are at the high end of the acceptable risk range established by international agencies but lower than that predicted for NO_2. The risks from all other pollutants, SO_2, CO, benzene and diesel articles are low and within the acceptable risk range.

2063 operations

For 2063, the levels of emissions are generally higher than in 2030 and the number of health outcomes attributable to the pollutants arising from airport operations is also higher. The highest predicted risk is associated with NO_2. The most affected areas are Luddenham, Bringelly, Kemps Creek, Mulgoa, Wallacia and Rossmore. The health risks for PM_{10} and $PM_{2.5}$ are at the higher end of the acceptable risk range established by international agencies. For all other pollutants the risk levels are all within the range established by international agencies.

The modelling used in the HRA has not considered the reductions that may occur due to the implementation of mitigation measures. The implementation of such measures will lead to reduction in ambient levels of the pollutants considered in this HRA and the associated health risk. In particular, it is assumed no future reductions in emissions technology either for aircraft or for vehicular traffic more generally. On the basis that emissions reduction has occurred over the past several decades and is expected to continue to occur in the future, the results of the HRA are likely to overestimate the actual level of risk that would be realised. Mitigation measures to reduce NO_2 and PM are included in the local air quality technical reports.

Aircraft and Ground Operations Noise

The HRA for noise has assessed three health outcomes – sleep disturbance (as awakenings), increases in ischaemic heart disease and impacts on cognitive development and learning in children and has been conducted for noise impacts from aircraft and ground operations noise sources. The results for the HRA show that ground-based operations may lead to a small increase in sleep disturbance (assessed as awakenings), increases in risk of cardiovascular disease and potential delays in childhood learning and cognitive development. Implementation of mitigation measures outlined in the noise technical reports will lead to reductions in these risks. These effects are predicted for suburbs close to the airport site, in particular Luddenham. The predicted impact of aircraft noise is less than for ground operations.

Based on the results of the noise assessment, the risk posed to the health of the exposed communities is generally low. No increase in cardiovascular outcomes is likely from aircraft noise as the predicted night noise levels are below the threshold for adverse effects. However, ground operations noise is predicted to be above the threshold value of 55 dB Lnight and may lead to a 10% increase in myocardial infarctions in Luddenham in 2063 if not mitigated.

A significant increase in EEG awakenings is predicted especially in Luddenham. In 2030, it is predicted that there would be 190 additional EEG awakenings per person per year and an additional 400 EEG awakenings per person per year with ground-based noise in 2063. For full awakenings, the number is lower but still highest in Luddenham. There is a greater predicted impact from ground- operations noise than aircraft noise. Mitigation measures in the noise assessment reports should be implemented and would reduce this impact.

Impacts on children's learning and cognitive development are predicted to be within acceptable risk levels for most locations for aircraft noise. The impact of ground operations noise is more substantial. In Luddenham, increases of up to 3 dB above the WHO guideline are predicted for outdoor noise levels in 2063. Mitigation measures should be implemented at these locations to reduce this risk to within acceptable levels. For the indoor assessment, assuming a 10 dB attenuation from predicted outdoor levels, the hazard quotients above 1 correspond to an increase in noise levels above the guideline value of between 19 and 23 dB in 2030 and 2063 respectively at Luddenham indicating a significant increase in noise levels. The risk of sleep disturbance and impacts on cognitive development are predicted to be higher for 2063 than 2030. Mitigation measures should be implemented to reduce this risk to within acceptable levels.

It should be noted that the noise HRA includes comparison of the runway operating mode options outlined in the EIS. As with the noise assessment results, the HRA will be considered in the process to finalise the flight paths and preferred operating mode.

The EIS noise assessment reports include mitigation and management measures which will reduce the potential impacts which have been assessed by the HRA. In particular, a noise amelioration strategy should be developed that considers both aircraft and ground operations in accordance with the requirements of AS202.

Surface and groundwater

There are 42 registered groundwater bores within a five kilometre radius of the centre of the site. Twelve of these bores are registered as being used for domestic, stock, industrial, farming and irrigation purposes. The depth of registered extraction bores indicates that the majority of groundwater users extract water from the Bringelly Shale and Hawkesbury Sandstone aquifers. The salinity in the Alluvial and Bringelly Shale aquifers is reported to be >1,000 mg/l (based on 1995-1998 data) and thus is considered unsuitable for potable uses.

The site has historically been used for a wide range of agricultural, industrial, commercial and rural-residential activities which have the potential to generate a range of contaminants in soil and groundwater. There is likely to be considerable overlap of those contaminants associated with historical site activities and the potential contaminants which may be associated with future construction and operation of the proposed airport. It is therefore important that baseline groundwater data are collected including all potential contaminants that may be already present, to enable identification of the current baseline conditions and from which to monitor future performance of the airport.

The contaminating activities which may occur during construction and operation of the airport may also have the potential to result in surface water contamination. Aircraft movements at the airport site during operation may result in increased deposition of particulates in surrounding waterways. Based on the results of the operational air quality modelling, the potential for deposition of particulates particularly at locations associated with potable water supply is very low with the maximum annual concentration of PM_{10} at Warragamba predicted to be 0.02 $\mu g/m^3$.

The commencement of aircraft operations at the proposed Western Sydney Airport also increases the potential for fuel jettisoning to occur on rare occasions. It is understood that the majority of fuel jettisoning instances for commercial aircraft occur in emergency conditions where an unscheduled landing is required. In 2014, there were only 10 reported instances nationwide of civilian aircraft fuel jettisoning out of 698,856 domestic and 31,345 international flights. Where fuel jettisoning is considered necessary, the pilot is required to take any reasonable precautions to ensure the safety of people and property on the ground and in the air, and where possible, undertake fuel jettisoning at a minimum altitude of 6,000 ft. Most fuel is considered to evaporate within 100 metres with only a small amount of fuel, if any, likely to reach ground level. There are no recorded instances in Australia of fuel reaching the ground after a fuel jettisoning incident.

Construction of the proposed airport also presents the potential for risks for contamination of groundwater and surface water. However these risks are common for other major infrastructure projects where standard construction measures are typically effective. Implementation of mitigation in the construction environmental management plans will minimise these risks.

The outcomes of the risk assessment suggest that it would be beneficial to collect additional data to inform the future management of potential risks to groundwater and surface water receptors. Consideration should be given to a pre-construction and post construction/operation monitoring program to test the quality of local tank water and monitor any changes to the quality of the water over time. This would enable informed decisions to be made about implementation of any mitigation measures in a timely manner.

GLOSSARY

µg/m3	Microgram per Cubic Metre
ACHAPS	Australian Child Health and Air Pollution Study
ADWG	Australian Drinking Water Guideline
AFFF	Aqueous Film Forming Foam
ANZECC	Australia New Zealand Environment Conservation Council
Airport	The proposed Western Sydney Airport
APHEA2	Air Pollution and Health a European Approach
APU	Auxiliary Power Unit
ASC NEPM	National Environment Protection (Assessment of Site Contamination) Measure
AWQG	Australian Water Quality Guideline
CAA	Civil Aviation Authority UK
CHETRE	Centre for Health Equity Training Research and Evaluation
CO	Carbon Monoxide
CoPC	Chemical of Potential Concern
COPD	Chronic Obstructive Pulmonary Disease
dB	Decibel
DEC	NSW Department of Environmental Conservation
DLWC	NSW Department of Land and Water Conservation
EC	European Commission
EC	Electrical Conductivity
ECG	Electrocardiography
EEG	Electroencephalography
EIS	Environmental Impact Statement
EMG	Electromyography
EOG	Electroculography
EPA	Environment Protection Authority

EPHC	Environment Protection and Heritage Council
EV	Environmental Value
FCV	Forced Vital Capacity
FEV1	Forced Expiratory Volume (in one second)
ft	Feet
GIL	Groundwater Investigation Level
GMRRW	Guideline for Managing Risks in Recreational Water
GSE	Ground Support Equipment
H2H	Head to Head
HIA	Health Impact Assessment
HQ	Hazard Quotient
HRA	Health Risk Assessment
HYENA	Hypertension and Exposure to Noise near Airports
IQ	Interquartile Range
km	Kilometre
L	Litres
L/day	Litres per Day
LAeq	Day-Time Aircraft Noise
Lday	Day-Time Noise Level Averaged between 9am-3pm
Lden	Day-Evening-Night Noise Level
Lnight	Aircraft noise at night averaged between 11pm and 7am
LOAEL	Lowest Observed Adverse Effects Level
m bgl	Metres Below Ground Level
MBA	Methylene Blue Activated Substances
min	Minutes
ml	Millilitres
NEPC	National Environment Protection Council
NEPM	National Environmental Protection Measure

NHMRC	National Health and Medical Research Council
NO	Nitric Oxide
NO2	Nitrogen Dioxide
NOEL	No Observed Effects Level
NREM	Non-Rapid Eye Movement
NSW	New South Wales
NSW GQPP	New South Wales Groundwater Quality Protection Policy
NWQMS	National Water Quality Management System
OEHHA	Office of Environmental Health Hazard Assessment
OR	Odds Ratio
PAH	Polycyclic Aromatic Hydrocarbons
PFC	Perfluorinated Compounds
PM	Particulate Matter
PM10	Particulate Matter (10 micrometre diameter or less)
PM2.5	Particulate Matter (2.5 micrometre diameter or less)
ppb	Parts per Billion
ppm	Parts per Million
RANCH	Road Traffic and Aircraft Noise and Children's Cognition and Health
REM	Rapid Eye Movement
REVIHAAP	Review of Evidence of Health Aspects of Air Pollution
RIVM	National Institute for Public Health and the Environment
SCEW	Standing Council on Environment and Water
SEIFA	Socio-Economic Indexes for Areas
SEL	Sound Exposure Level
SES	Socioeconomic Status
SO2	Sulphur Dioxide
TPH	Total Petroleum Hydrocarbon
UK	United Kingdom

URF	Unit Risk Factor
USEPA	United States Environment Protection Agency
WHO	World Health Organisation
WQO	Water Quality Objective

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1 INTRODUCTION

Pacific Environment Limited (PEL) was engaged by GHD Pty Ltd (GHD) to undertake a health risk assessment (HRA) to assess risks associated with noise, air emissions, and risks to surface water and groundwater resulting from the proposed Western Sydney Airport (the airport). The risk assessment will be incorporated into the environmental impact statement, for the proposed Western Sydney Airport at Badgerys Creek (the airport site).

1.1 Background

Planning investigations to identify a site for a second Sydney airport first commenced in 1946 with a number of comprehensive studies—including two previous environmental impact statements for a site at Badgerys Creek—having been completed over the last 30 years.

More recently, the *Joint Study on Aviation Capacity in the Sydney Region* (Department of Infrastructure and Transport, 2012) and *A Study of Wilton and RAAF Base Richmond for civil aviation operations* (Department of Infrastructure and Transport, 2013) led to the Australian Government announcement on 15 April 2014 that Badgerys Creek will be the site of a new airport for Western Sydney. The airport is proposed to be developed on approximately 1,700 hectares of land acquired by the Commonwealth in the 1980s and 1990s. Construction could commence as early as 2016, with airport operations commencing in the mid-2020s.

The proposed airport would provide both domestic and international services, with development staged in response to demand. The initial development of the proposed airport would include a single, 3,700 metre runway coupled with landside and airside facilities such as passenger terminals, cargo and maintenance areas, car parks and navigational instrumentation capable of facilitating the safe and efficient movement of up to 10 million passengers per year. While the proposed Stage 1 development does not currently include a rail service, planning for the proposed airport preserves flexibility for several possible rail alignments including a potential express service. A final alignment will be determined in consultation with the New South Wales Government, with any enabling work required during Stage 1 subject to a separate approval and environmental assessment process.

In the longer term, approximately 40 years after operations commence and in accordance with relevant planning processes, the airport development could include parallel runways and additional passenger and transport facilities for around 82 million passenger movements per year. To maximise the potential of the site, the airport is proposed to operate on a 24 hour basis. Consistent with the practice at all federally leased airports, non-aeronautical commercial uses could be permitted on the airport site.

On 23 December 2014, a delegate of the Australian Government Minister for the Environment determined that the construction and operation of the airport would require assessment in accordance with the *Environment Protection and Biodiversity Conservation Act 1999* (Cth) (EPBC Act). Guidelines for the content of an environmental impact statement (EIS) were issued in January 2015. Approval for the construction and operation of the proposed airport will be controlled by the *Airports Act 1996* (Cth) (Airports Act). The Airports Act provides for the preparation of an Airport Plan which will serve as the authorisation for the development of the proposed airport.

The Australian Government Department of Infrastructure and Regional Development is undertaking detailed planning and investigations for the proposed airport, including the development of an Airport Plan. The draft Airport Plan is the primary source of reference for, and companion document to, the EIS. The draft Airport Plan identifies a staged development of the proposed airport. It provides details of the initial development being authorised, referred to as Stage 1, as well as a long-term vision of the airport's development. This enables preliminary consideration of the implications of longer term airport

operations. Any stages of airport development beyond Stage 1 would be managed in accordance with the existing process in the Airports Act. This includes a requirement that for major developments (as defined in the Airports Act), a major development plan be approved by the Australian Government Minister for Infrastructure and Regional Development following a referral under the EPBC Act.

The Airport Plan will be required to include any conditions notified by the Environment Minister following this EIS. Any subsequent approvals for future stages of the development will form part of the airport lessee company's responsibilities in accordance with the relevant legislation.

1.2 Objectives

The EIS Guidelines for the Western Sydney Airport (EPBC 2014/7391) specify the information that must be included in the EIS. In particular, Section 5g lists a range of potential impacts on the environment and specific issues which must be analysed.

Attachment 1 of the EIS Guidelines outlines the objects and principles of the EPBC Act which include "*to promote ecologically sustainable development*". One of the key elements of ecologically sustainable development includes the principle of inter-generational equity which states "*that the present generation should ensure that the health, diversity and productivity of the environment is maintained or enhanced for the benefit of future generations*".

While not specifically included in the EIS Guidelines, a Health Risk Assessment (HRA) has been undertaken to inform decision makers of the potential for effects on human health. While the other EIS technical studies report impacts in relation to relevant Commonwealth and State criteria, which are often set in order to protect the environment and human health, it is considered that a specific health study is required to further analyse the potential impacts, particularly in relation to a number of recent literature studies linking health effects to aviation activity. Potential health effects may also be linked to different criteria and measures than examined elsewhere in the EIS.

This report provides the results of the HRA for air quality and noise as well as the potential impacts on surface and groundwater. The HRA has been conducted in accordance with the EIS Guidelines and the Australian Government *Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazard* 2012" (enHealth, 2012) as well as publications from other agencies such as the World Health Organisation (WHO).

2 OVERVIEW OF THE HEALTH RISK ASSESSMENT PROCESS

A Health Risk Assessment (HRA) aims to quantify the potential health effects arising from exposure to, in this case, environmental pollution. The information in this report on the pollutants comes from other studies undertaken for the Western Sydney Airport EIS, peer reviewed scientific studies, ambient monitoring and modelling. The HRA is a document that assembles and synthesizes scientific information to determine whether a potential hazard exists, the exposure to the hazard and the resultant risk to human health.

Risk assessments are often conducted by considering possible or theoretical community exposures predicted from air dispersion modelling or using environmental concentrations that have been measured in the potentially affected population. Conservative safety margins are built into a risk assessment analysis to ensure protection of public health. During the risk assessment analysis, the most vulnerable people (e.g. children, the sick and elderly) are carefully considered to make sure that all the risk to these more vulnerable groups is considered.

For air quality risk assessments the key health effects that are considered include increases in mortality and morbidity (eg. Hospital admissions for respiratory disease) which have been associated with exposure to air pollution in population based epidemiological studies. For noise the main health effects that are considered are sleep disturbance, increases in ischaemic heart disease and impacts on children's learning and cognitive development. These outcomes have been considered in this HRA.

The Australian guidance for conducting HRAs is set out in the enHealth Guidelines (2012). For the assessment of health risks from air pollution, the National Health and Medical Research Council (NHMRC) *Approach to Hazard Assessment for Air Quality*, 2006 and the National Environment Protection Council (NEPC) *Methodology for Setting Air Quality Standards in Australia*, 2011 provide detailed frameworks to assess health risks associated with air pollution.

2.1 Approach to Health Risk Assessment

Health is defined by the World Health Organization (WHO) as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO, 1948). Well-being is broadly described as an individual's self-assessment of their state of happiness, healthiness and prosperity. It relates to the quality of life and one's ability to enjoy it. There are many social and economic factors that influence well-being:, including (enHealth 2012, NHC 2004):

- Social and cultural factors (e.g. social support, participation, access to cultural resources).
- Economic factors (e.g. income levels, access to employment).
- Environmental factors (e.g. land use, air quality).
- Population-based services (e.g. health and disability services, leisure services).
- Individual/behavioural factors (e.g. physical activity, smoking).
- Biological factors (e.g. biological age).

While the focus of this study is on the environmental factors and changes that might occur as a result of the proposed airport development, the social, cultural, economic and other environmental factors have also been analysed and documented in other parts of this EIS.

The risk assessment process detailed in the enHealth HRA Guidelines comprises five components as outlined below:

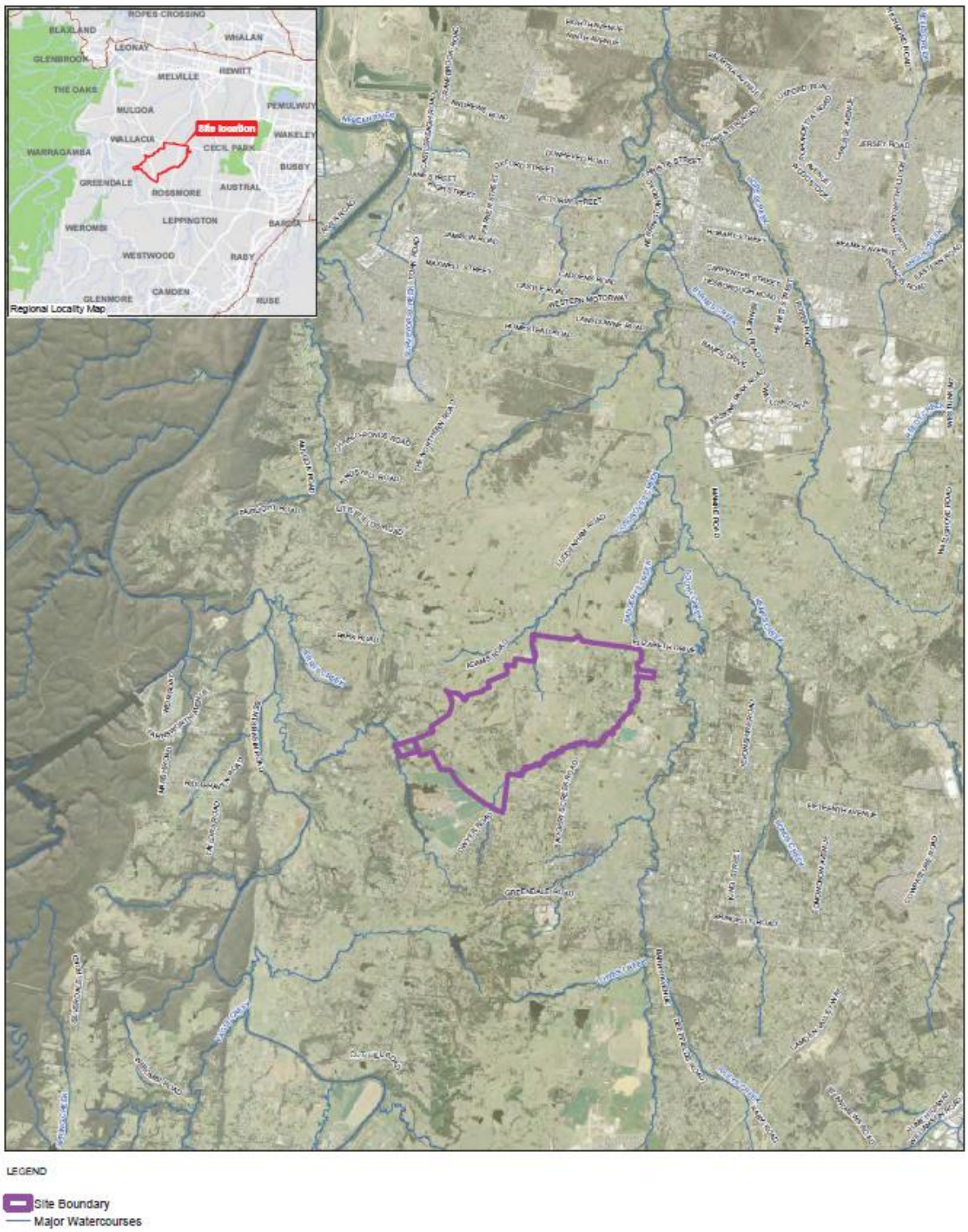
1. **Issue Identification** – Identifies issues that can be assessed through a risk assessment and assists in establishing a context for the risk assessment.
2. **Hazard Assessment** – Identifies hazards and health endpoints associated with exposure to hazardous agents and provides a review of the current understanding of the toxicity and risk relationship of the exposure of humans to the hazards.
3. **Exposure Assessment** – This task identifies the groups of people who may be exposed to hazardous agents and quantifies the exposure concentrations.
4. **Risk Characterisation** – This task provides the qualitative evaluation of potential risks to human health. The characterisation of risk is based on the review of concentration response relationship and the assessment of the magnitude of exposure.
5. **Uncertainty Assessment** – identifies potential sources of uncertainty and qualitative discussion of the magnitude of uncertainty and expected effects on risk estimates.

3 WESTERN SYDNEY AIRPORT SITE

The site for the proposed Western Sydney Airport covers an area of approximately 1,700 hectares located at Badgerys Creek in Western Sydney as shown in **Figure 1.** The site is located around 50 kilometres west of Sydney's Central Business District and 15 to 20 kilometres from major population centres such as Liverpool, Fairfield, Campbelltown and Penrith. The nearest suburbs include Luddenham, Badgerys Creek, Kemps Creek and Bringelly.

The Northern Road transects the western end of the airport site and Elizabeth Drive borders the site to the north. Badgerys Creek flows in a north-easterly direction and forms the south eastern boundary of the airport site. The airport site is located on undulating topography that has been extensively cleared with the exception of stands of remnant vegetation located predominantly along Badgerys Creek and the south western portion of the site.

Figure 1: Site Location



4 POPULATION AND HEALTH PROFILE

The airport site is located within the Liverpool LGA in the south west of Sydney. The Liverpool LGA encompasses a total land area of 305 square kilometres and it borders Fairfield and Penrith cities in the north, Camden Council and Campbelltown City areas in the south, the Wollondilly Shire in the west and Bankstown City in the east.

According to the Liverpool Community Health Profile (SWSLHD, 2014), the population in the Liverpool LGA is predicted to increase significantly from 188,088 people in 2011 to 288,959 in 2031. The predicted population growth in various age groups is shown in Figure 2 (taken from Liverpool Community Health Profile, SWSLHD, 2014).

Figure 2: Predicted Population Growth Liverpool LGA

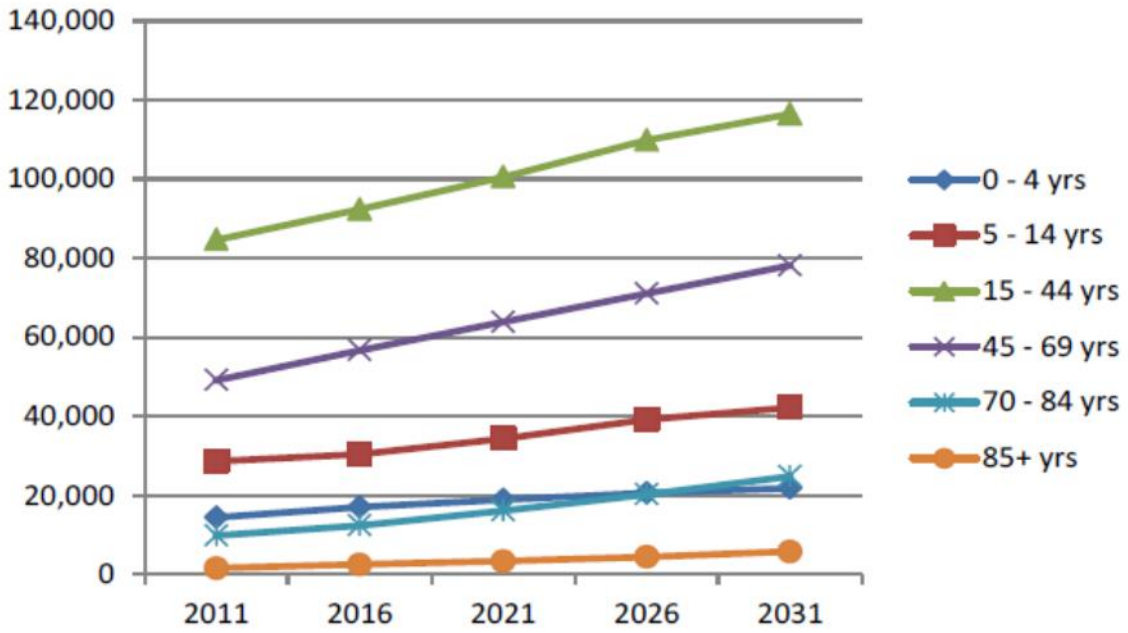


Figure 2 shows that the most significant population growth is predicted for people 15-44 and 45-69 years of age.

Population statistics for the 2011 Census have been obtained from ABS for the each of the suburbs which have been considered in the health risk assessment. These statistics are shown in Table 1 in order of increasing population size. It should be noted that the airport site will occupy significant parts of Badgerys Creek and Luddenham and a number of current residents will be relocated. Therefore the future populations in these areas is likely to be much lower than that recorded in 2011.

Table 1: Demographic Profile for Suburbs included in HRA

Suburb	Approx. distance to airport site (km)[1]	Total Population	% > 65 years of age	%< 15 years of age	SEIFA Index
Australia - avg	-	-	14	19	1000
Sydney – avg	-	-	13	19	1025
Greendale	8	352	11	22	986
Badgerys Creek	3	455	12	20	913
Mt Vernon	8	1036	11	20	1102
Warragamba	11	1236	12	22	914
Luddenham	3	1496	12	22	1034
Wallacia	8	1700	10	21	1032
Mulgoa	8.5	1792	12	20	1065
Horsley Park	13	1936	16	18	1007
Kemps Creek	6	2309	15	19	993
Bringelly	6	2387	10	21	1036
Rossmore	8	2412	13	22	997
Silverdale	11	3439	7	24	1077
Prospect	21	4621	9	21	1031
Erskine Park	11.5	6668	4	23	1041
Colyton	13	7993	11	22	930
Plumpton	18.5	8244	6	25	999
St Mary's	14	10961	14	21	881
Mt Druitt	16	15794	8	26	895
Rooty Hill	17	13377	12	22	970
St Clair	12	19837	6	21	1013

1. Distances estimated from approximate centre of the airport site to each town.
2. Demographic information based on ABS figures for 2011

People who are of low socioeconomic status (SES) have been identified as a vulnerable group for the effects of air and noise pollution. This is largely due to the fact that people within these groups usually have poorer health status than people within higher SES groups. They may also have poorer access to medical care. In addition, they usually live in areas that are more polluted (e.g., near major roads or near industry) as property is generally cheaper in these areas.

There are several indices of social deprivation used to assess SES status in Australia. One commonly used is the Socio-Economic Indexes for Areas (SEIFA) index. The SEIFA index is a measure of relative social disadvantage and takes into account 20 variables to assess relative social disadvantage. The lower the SEIFA index the greater the level of disadvantage. The index is relative to a score of 1000 which is considered as the Australian average.

The SEIFA Index of Relative Socio-Economic Advantage/Disadvantage is derived from attributes such as low income, low educational attainment, high unemployment, jobs in relatively unskilled occupations and variables that broadly reflect disadvantage rather than measuring specific aspects of disadvantage (e.g. Indigenous and Separated/Divorced). At the advantage end of the scale, households with high incomes, high education levels, large dwellings, high numbers of motor vehicles, spare bedrooms and professional occupations contribute to a higher score.

The SEIFA scores shown in Table 1 indicate that there are areas considered in the HRA that have a lower SES than the Australian average (1000) or Sydney as a whole (1025). In particular, Badgerys Creek, St Marys, Mount Druitt, Rooty Hill, Colyton and Warragamba all have low SEIFA scores indicating that the populations in these suburbs may form a vulnerable group to the effects of air pollution and noise from the proposed airport.

The Liverpool Community Health Profile (SWSLHD, 2014) presents the baseline health statistics for the local population. These are shown in Table 2:

Table 2: Baseline Health Status Liverpool LGA

Indicator	Liverpool	Proportion of NSW average (%)
Hospitalisations		
Hospitalisations (2009/10 to 2010/11) per year	58,010	99.9
Potentially preventable hospitalisations per year (2010/11 to 2011/12)	3,850	95.4
Alcohol attributable hospitalisations per year (2010/11 to 2011/12)	934	81.8
Smoking attributable hospitalisations per year (2010/11 to 2011/12)	905	100.5
High body mass index attributable hospitalisations per year (2010/11 to 2011/12)	719	101
Coronary heart disease hospitalisations per year (2009/10 to 2010/11)	821	91.2
Chronic obstructive pulmonary disease hospitalisations (persons aged over 65) per year (2009/10 to 2010/11)	262	112.9
Diabetes hospitalisations per year (2009/10 to 2010/11)	515	132.1
Fall-related injury overnight hospitalisations (persons aged 65 years and over) per year (2010/2011 to 2011/12)	572	116.9
Stroke hospitalisations per year (2010/11 to 2011/12)	196	97.6
Deaths		
Potentially avoidable deaths (persons aged under 75 years) per year (2006 to 2007)	211	99.5
Potentially avoidable deaths from preventable causes (persons aged under 75 years) (2006 to 2007)	122	96.6
Potentially avoidable deaths from causes amenable to health care (persons aged under 75 years) per year (2006 to 2007)	84	97.8
High body mass index attributable deaths (2006 to 2007)	46	91.1
Alcohol attributable deaths per year (2006 to 2007)	23	94.6
Smoking attributable deaths per year (2006 to 2007)	79	99.2

The data shown in Table 2 indicate that the baseline health status of the Liverpool LGA does not differ significantly from the data for NSW as a whole. Although the data for the whole LGA does not differ from NSW as a whole there may be parts of the LGA that have a lower health status due in part to having a lower SES.

According to the Liverpool Community Health Profile (SWSLHD), the asthma prevalence rate in people over 16 years of age in the area is 6.3%. This is lower than the NSW average for the same age group.

5 HEALTH RISK ASSESSMENT – AIR QUALITY

5.1 Introduction

The health effects attributable to PM_{10}, $PM_{2.5}$, NO_2, SO_2 and CO have been assessed in this HRA for increases in mortality, hospital admissions for respiratory and cardiovascular disease, and emergency department visits for asthma in children that may be attributable to emissions from the construction and operation of the proposed airport. Baseline health statistics for Sydney have been used in the assessment and the risk has been assessed for suburbs within 5 km from the airport site boundary (Pacific Environment, 2015). These include Bringelly, Luddenham, Greendale, Kemps Creek, Mulgoa, Wallacia, Badgerys Creek, Rossmore and Mount Vernon. The cancer risk due to diesel emissions and benzene has also been calculated for these areas. The focus of this HRA is on the potential health effects of local air quality which includes emissions from aircraft overflights, ground based airport sources and traffic to and from the airport site. The potential health risk from construction activities has also been assessed.

The predicted future air quality data used in the HRA have been generated by the EIS local air quality study (Pacific Environment, 2015) conducted for the initial and longer term airport developments which are expected to occur in or about 2030 and 2063 respectively. The modelling results that have been used in the HRA represent the worst case emission scenarios. The 2030 scenario involves a single runway with approximately 10 million passenger movements per year. The 2063 scenario includes two runways with approximately 82 million passenger movements per year. Given the uncertainties associated with predicting baseline emissions in 2063, as well as emissions from a future aircraft fleet, only PM_{10}, $PM_{2.5}$, and NO_2 have been assessed. Further details of these limitations are provided in the local air quality report and relevant EIS chapters.

The risk assessment for each of the pollutants is presented in the following sections in accordance with the enHealth HRA Guidelines (enHealth, 2012).

5.2 PM_{10} and $PM_{2.5}$

According to the local air quality study, the main source of PM_{10} and $PM_{2.5}$, would be airport traffic on roadways external to the airport site. Aside from road traffic, aircraft movements would be the next largest source of PM_{10}, $PM_{2.5}$, NO_X and SO_2. The operation of Auxiliary Power Units (APUs) and ground support equipment (GSE) would also have an influence on the predicted pollutant concentrations. The largest contributor of on-site emissions is anticipated to be associated with aircraft taking off and landing, for both Stage 1 and longer term development scenarios. The external road infrastructure was shown to be a significant contributor to predicted off-site ground level concentrations, particularly for those receptors located in close proximity to existing or proposed new roadways.

A key assumption integral to the assessment of the longer term development is that no improvement in aircraft emissions, either due to improvements in fuel or engine emissions was able to be incorporated into the modelling. This is based on the inability to predict the effect of future policies or technological developments which are expected to occur and which are likely to result in improvements in levels of combustion emissions and pollutants. This assumption embeds a high degree of conservatism into the longer term development assessment (Pacific Environment, 2015).

5.2.1 Literature Review of the Health Effects of PM_{10} and $PM_{2.5}$

The health effects of particles linked to ambient exposures have been well studied and reviewed by international agencies (NEPC, 2010; USEPA, 2004, 2009, 2012; WHO, 2013, 2006; OEHHA, 2000). Most information comes from population-based epidemiological studies that find increases in daily mortality, as well as morbidity outcomes such as increases in hospital admissions and emergency room attendances, and exacerbation of asthma associated with daily changes in ambient particle levels. In

recent years, there has been an increasing focus on the association between exposure to particles and cardiovascular outcomes. In addition to studies on the various size metrics for particles, recent research has also investigated the role of particle composition in the observed health effects.

Several studies conducted in Australia also show adverse effects of both PM_{10} and $PM_{2.5}$ on mortality and morbidity outcomes (Simpson et al., 2005a, b; Barnett et al., 2005; 2006) similar to those observed in overseas studies. The effects observed in the Australian studies appear to be greater per 1 µg/m increase in PM than those observed in the US and Europe but comparable to the results of Canadian studies.

A recent review conducted by the World Health Organization (WHO, 2013) concluded that both PM_{10} and $PM_{2.5}$ are related to increases in mortality from respiratory and cardiovascular causes, hospital admissions and emergency department attendances for respiratory and cardiovascular causes including asthma, exacerbation of asthma and increases in respiratory symptoms. In recent years, studies have provided much stronger evidence for the cardiovascular effects of particles, in particular $PM_{2.5}$. There has also been an increase in evidence to support a biological mechanism, the cardiovascular effects of which include interference with electrical process within the heart, systemic inflammation and oxidative stress. The WHO concluded that long-term exposure to $PM_{2.5}$ is the cause of both cardiovascular mortality and morbidity. The USEPA (2012) concluded that there was new evidence regarding cardiovascular mortality showing strong effects between $PM_{2.5}$ exposure and cardiovascular mortality, especially in women. In addition, there is evidence for long-term exposure to $PM_{2.5}$ and respiratory effects, including the incidence of lung cancer. Studies of cardiovascular effects provided evidence of an association between long-term exposure to $PM_{2.5}$ and myocardial infarction (heart attack), hypertension, diabetes and stroke especially among women.

A number of new studies as reported by WHO (2013) and USEPA (2012) linking long-term exposures have examined additional health outcomes apart from the previoulsy identified respiratory and cardiovascular outcomes. These outcomes include atherosclerosis, adverse birth outcomes and childhood respiratory disease. Studies have also shown possible links between long-term exposure to $PM_{2.5}$ and neurodevelopment and cognitive function as well as other chronic conditions such as diabetes. In recent years, the evidence for a link between exposure to particles and diabetes has been strengthened.

Birth cohort studies from Europe and elsewhere have found associations between $PM_{2.5}$ and respiratory infections and asthma in young children. Reduced lung function is also linked to $PM_{2.5}$ exposure. Findings of a cohort study conducted in the Netherlands supports the findings of previous studies conducted in the US and Europe linking exposure to particles and these health outcomes. Associations with birth outcomes such as low-birth-weight, preterm birth and small gestation age at birth have also been found with long-term exposure to $PM_{2.5}$. These outcomes may affect a child's development later in life. The USEPA (2012) also identified several recent studies that showed associations between long-term exposure to $PM_{2.5}$ and respiratory morbidity including hospital admissions and respiratory symptoms as well is the incidence of asthma. Studies of reproductive and developmental effects also provided evidence for long-term exposure to $PM_{2.5}$ and reduced birth weight.

With respect to short-term effects, the USEPA (2012) found that there were important new studies that increase the evidence for an association between $PM_{2.5}$ and mortality and morbidity outcomes and strengthen the previous US EPA conclusion that there is a causal association between short-term exposure to $PM_{2.5}$ and these outcomes. Associations were found for hospital admissions and emergency department attendances for all cardiovascular and respiratory causes as well as cause specific outcomes, in particular asthma.

The Australian Child Health and Air Pollution Study (ACHAPS), which used a similar study design as that used in the Southern Californian Children's Health Study, was conducted to inform the review of the

particle standards in the Ambient Air Quality NEPM (Standing Council on Environment and Water (SCEW, 2011). The results of a cross-sectional study of approximately 4,000 Australian school children aged 7-11 years showed varied results for the particulate matter exposures used in ACHAPS. PM_{10} was associated with a decline in lung function (FEV_1) post-bronchodilator use and increase in exhaled NO (an indicator of airway inflammation), but no overall increase in current respiratory symptoms. $PM_{2.5}$ was associated with an adverse effect on lung function (measured as Forced Vital Capacity (FVC)) post-bronchodilator use and on exhaled NO, with no overall effects on current symptoms, but showed increased risk of lifetime wheezing, asthma, and asthma medication use, and current asthma, use of beta-agonists and itchy rash in non-atopic children. Females had an increase in FEV_1/FVC ratio pre-bronchodilator for recent $PM_{2.5}$, and recent PM_{10} exposures, with non-significant effects in males. Despite the absence of effect on current symptoms, a reduction in lung volume at this age may have longer-term adverse consequences if it persists into later life (SCEW, 2011).

No studies investigating the long term effects of exposure to PM_{10} on health have been conducted in Australia, however there have been several international studies that have shown strong associations between long-term exposure to PM_{10} and increases in mortality (WHO, 2013; USEPA, 2012, 2009).

5.2.2 Exposure Assessment

The residential locations that have been used for the air quality HRA are Bringelly, Luddenham, Badgerys Creek, Greendale, Rossmore, Mount Vernon, Wallacia, Mulgoa and Kemps Creek. The data in the following sections have been predicted for these locations. The total population covered by these areas is 13,939 people.

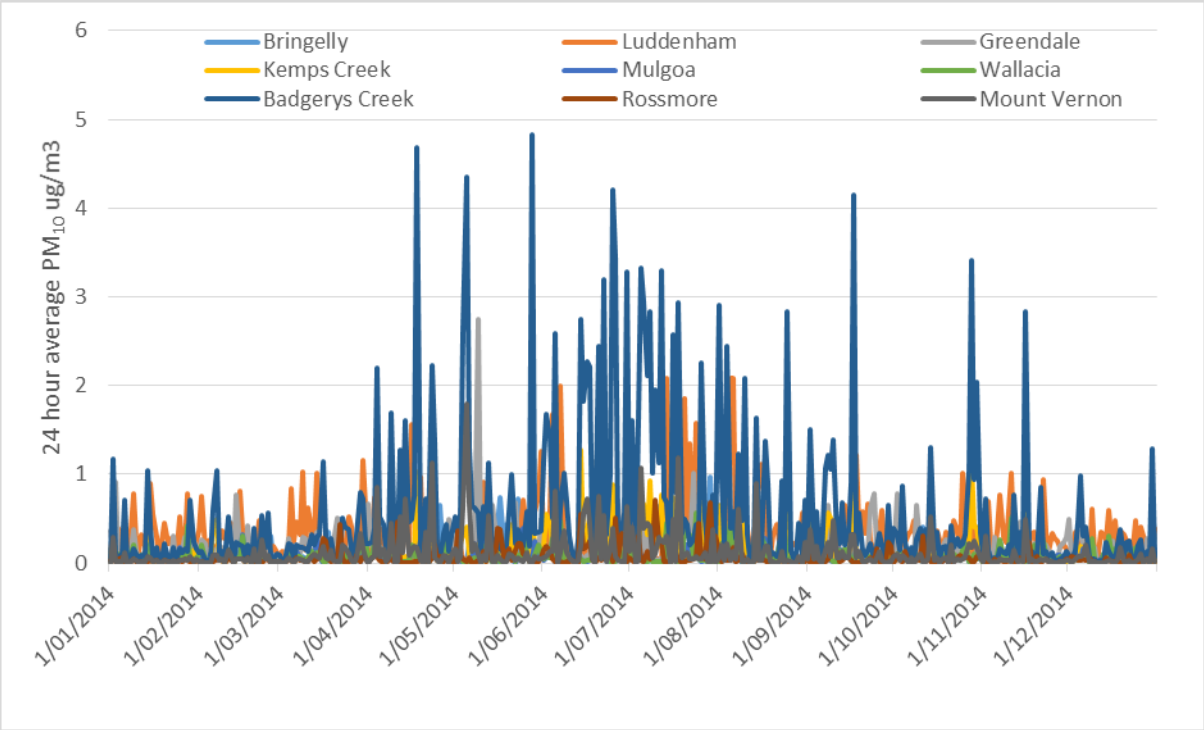
5.2.2.1 Construction phase

Air quality modelling conducted for the construction phase has provided annual average and 24-hour average PM_{10} and $PM_{2.5}$ for the following:

- bulk earthworks
- aviation infrastructure works
    - machinery, trucks, graders etc.
    - concrete batching plant

Details of the modelling and sources considered are provided in the Local Air Quality Assessment (Pacific Environment (2015). The 24 hour average PM_{10} and $PM_{2.5}$ concentrations predicted for each of the residential locations listed above are shown in Figures 3-6.

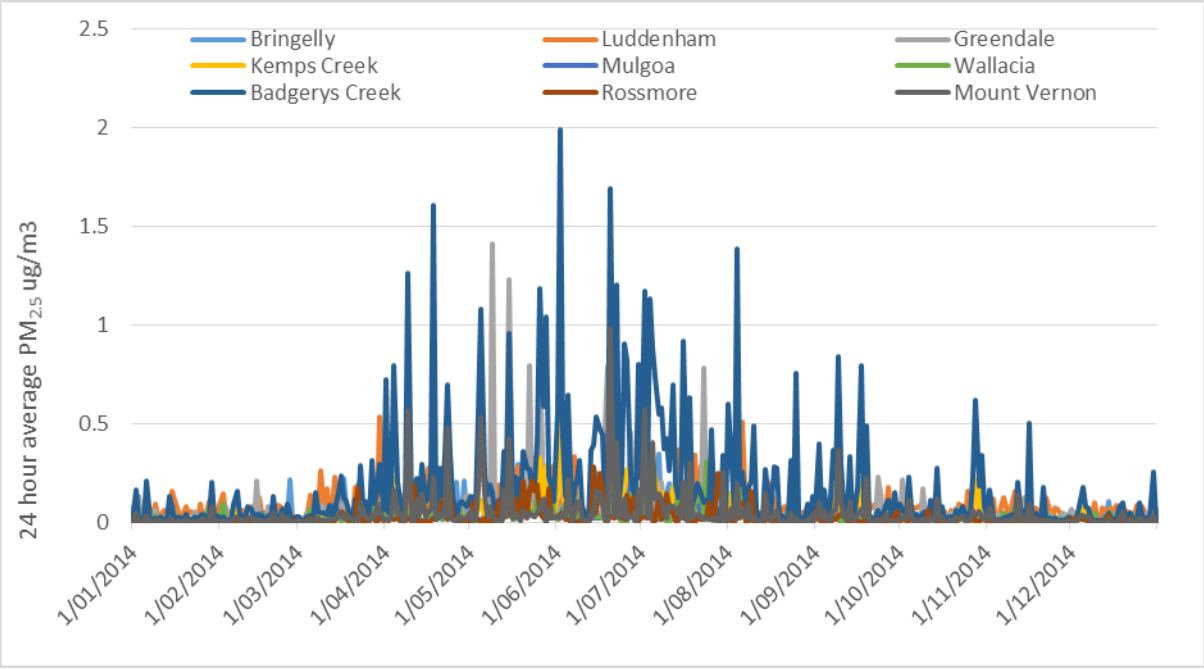
Figure 3: Predicted 24-hour Average PM_{10} Bulk Earthworks



The data shown in Figure 3 indicate that the PM_{10} levels from earthworks during construction are predicted to be well below the current NEPM standard of 50 µg/m^3 at all residential locations assessed. The highest impact is predicted for Badgerys Creek.

Figure 4 shows the predicted $PM_{2.5}$ concentrations from the construction earthworks.

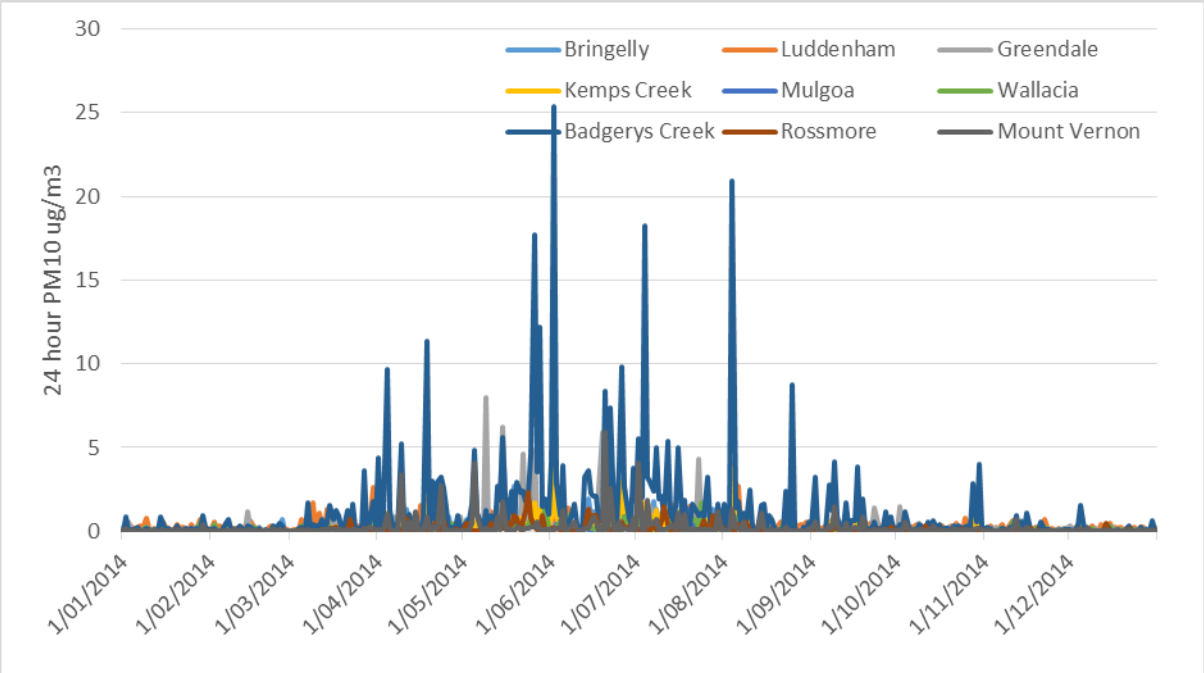
Figure 4: Predicted 24-hour average $PM_{2.5}$ Bulk Earthworks



As with the PM_{10}, the predicted $PM_{2.5}$ concentrations are low and below the advisory reporting standard of 25 µg/m^3 in the NEPM. The highest impacts are predicted for Greendale and Badgerys Creek.

Figure 5 shows the predicted PM_{10} concentrations from the construction of the aviation infrastructure.

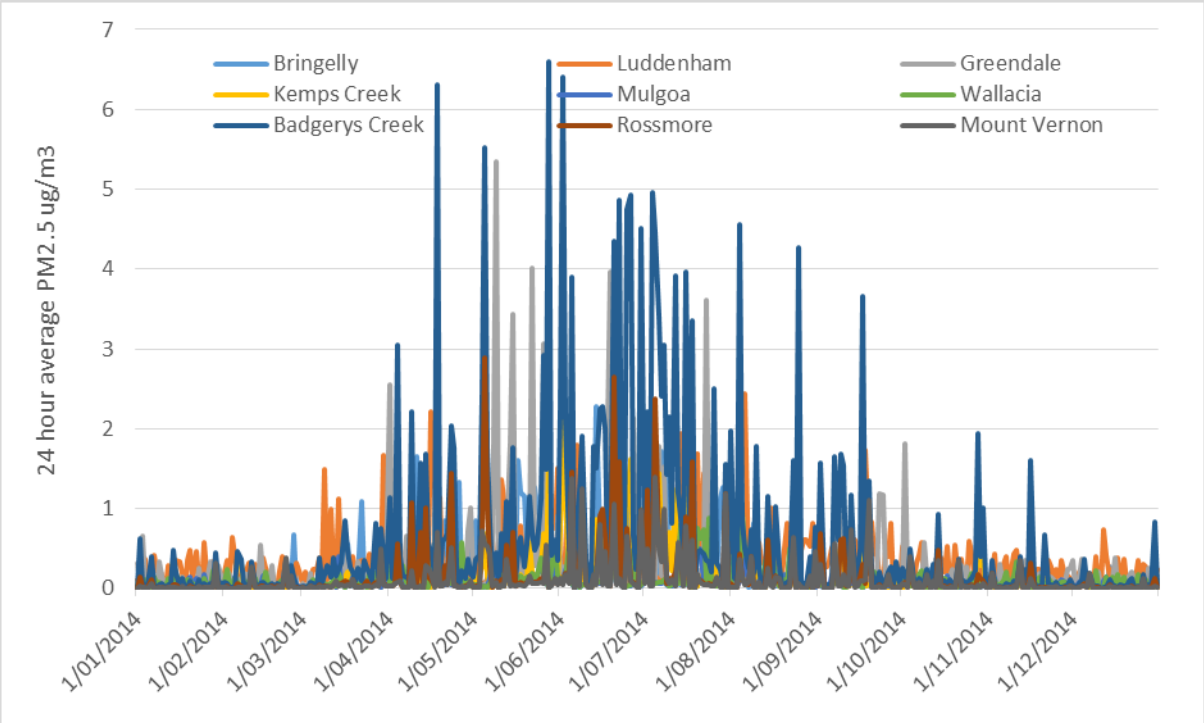
Figure 5: 24 Hour Average PM_{10} Aviation Infrastructure



The predicted PM_{10} concentrations are higher than those predicted during the bulk earthworks but are still below the NEPM standard of 50 µg/m^3. The highest impact is again at Badgerys Creek.

The data for $PM_{2.5}$ are shown in Figure 6.

Figure 6: 24 hour Average $PM_{2.5}$ Aviation Infrastructure



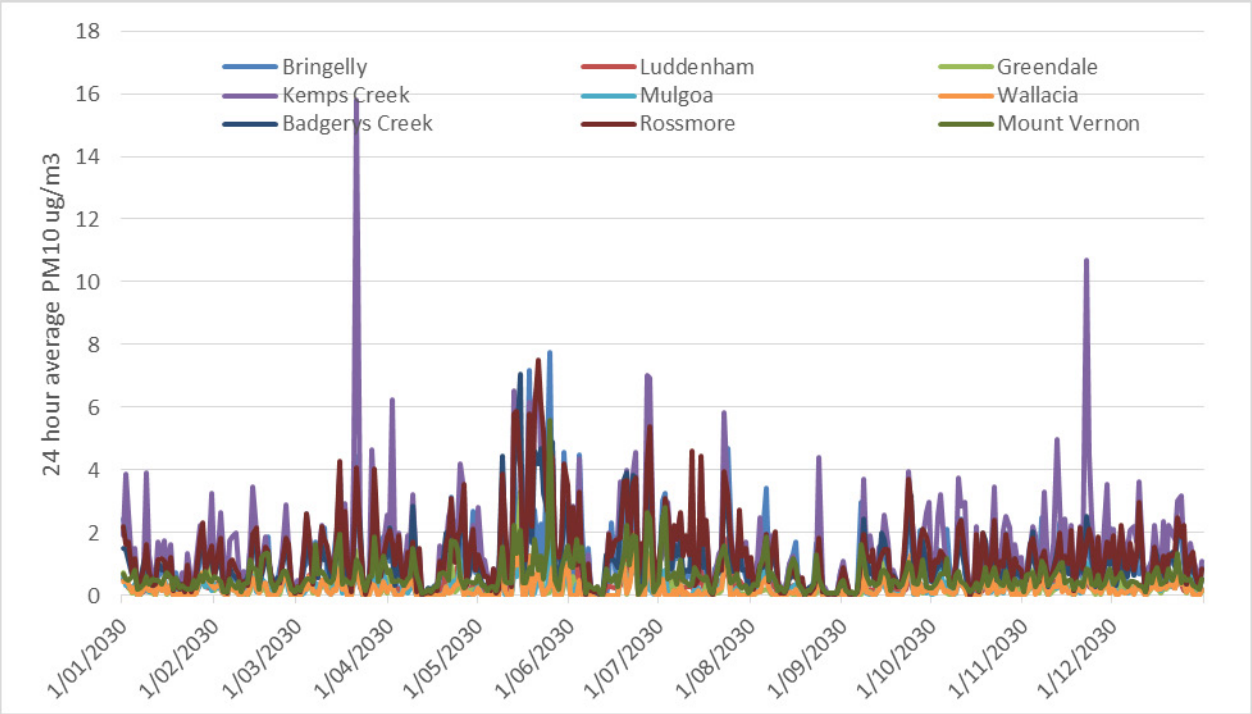
As with PM_{10}, the predicted concentrations are higher than those predicted for the bulk earthworks but still below the NEPM Advisory Reporting Standard of 25 µg/m^3. The highest concentrations are predicted for Badgerys Creek, Greendale and Rossmore.

5.2.2.2 Operation

Annual average and 24-hour PM_{10} and $PM_{2.5}$ have been modelled as part of the local air quality assessment for both 2030 and 2063. Figures 7 to 10 show the predicted 24-hour averages for the residential receptors in the surrounding area. The air quality standards for PM_{10} and $PM_{2.5}$ contained in the Ambient Air Quality NEPM are 50 µg/m^3 and 25 µg/m^3 respectively. The data shown in Figures 7 to 10 are below these standards.

The PM_{10} and $PM_{2.5}$ data generated by the local air quality assessment has been used to calculate the risk of adverse health outcomes associated with exposure to PM_{10} and $PM_{2.5}$ from the operation of the airport in 2030 and 2063 as well as the construction activities.

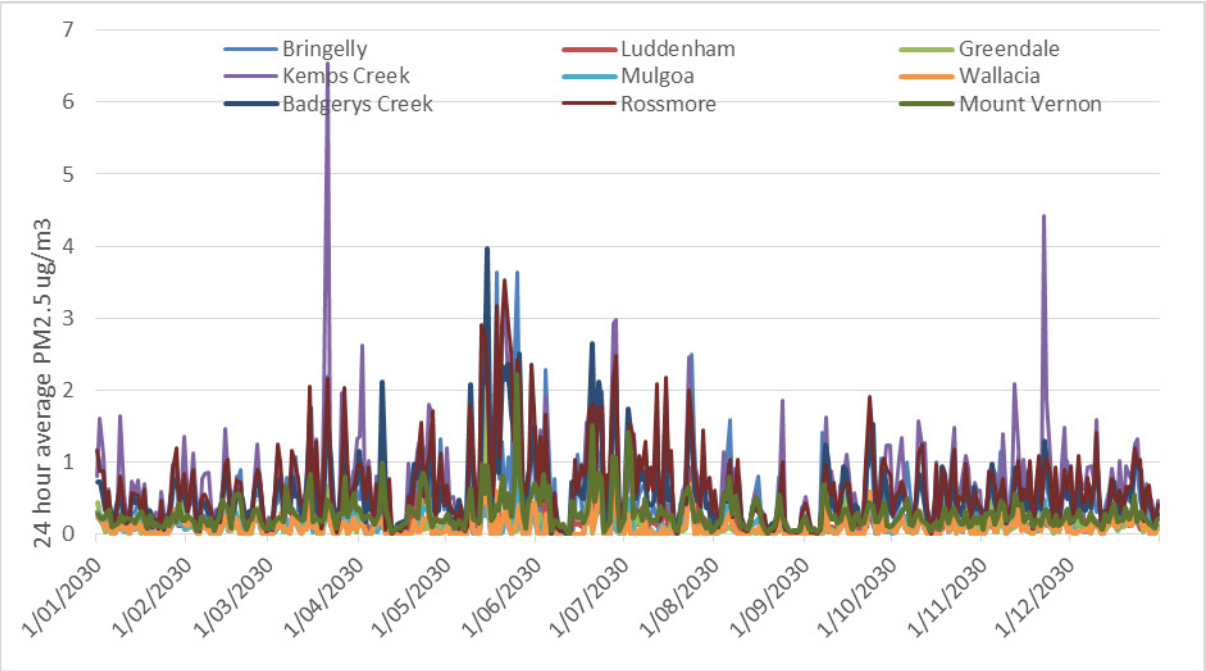
Figure 7: Daily average PM_{10} from operations 2030



The data shown in Figure 7 indicate that the highest impact of airport operations in 2030 is predicted for Kemps Creek and Rossmore.

Figure 8 shows the predicted concentrations for $PM_{2.5}$ for operations in 2030.

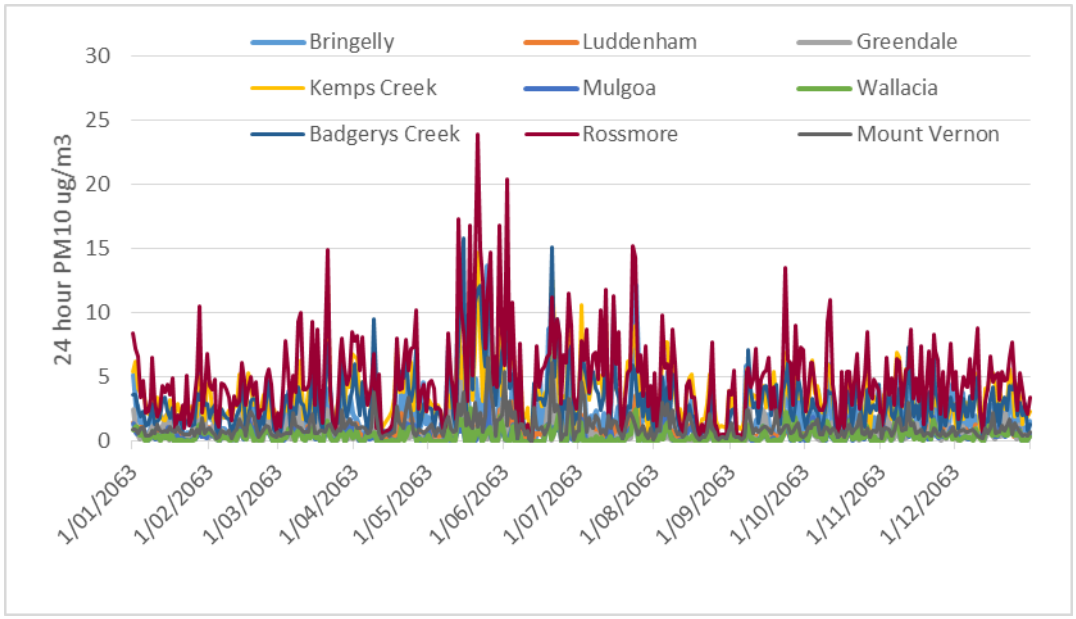
Figure 8: Daily average $PM_{2.5}$ from operations 2030



The data in Figure 8 show that the highest predicted impact is for Kemps Creek, Badgerys Creek, Rossmore and Mulgoa. All predicted concentrations are below the NEPM Advisory Reporting Standard of 25 µg/m^3.

The predicted PM_{10} concentrations for operations in 2063 are shown in Figure 9.

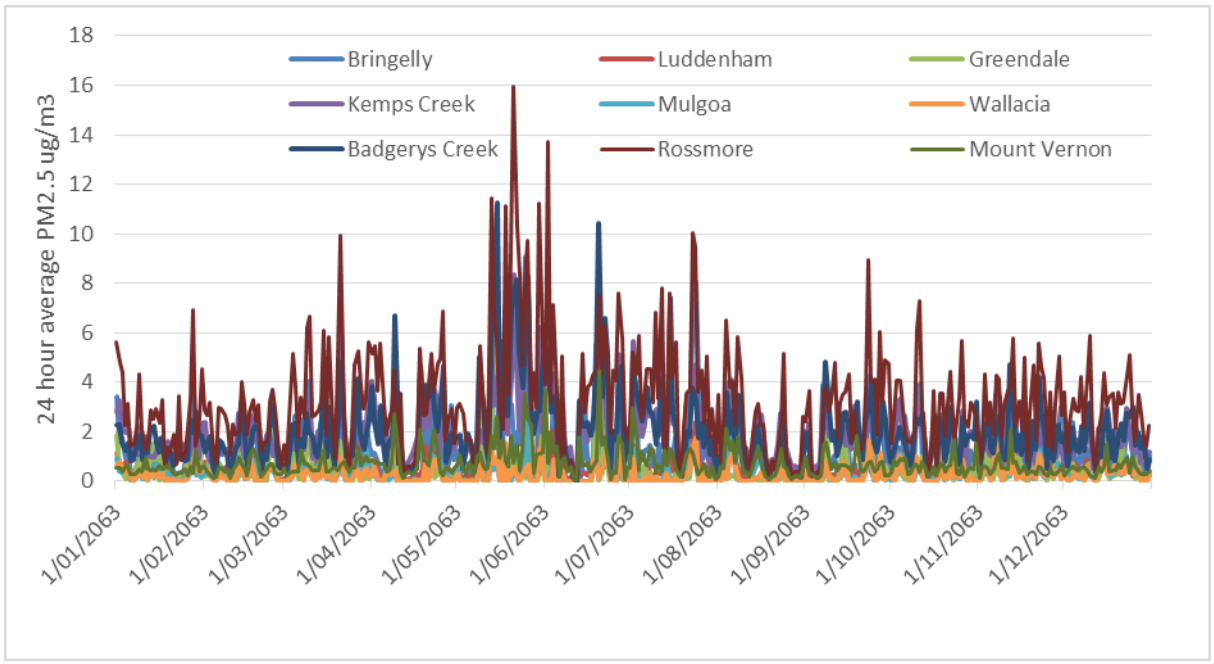
Figure 9: 24 Hour Average PM_{10} Operations 2063



As shown in Figure 9, the predicted PM_{10} concentrations are higher than predicted in 2030. The highest impact is at Rossmore and Mulgoa. The predicted concentrations are below the NEPM standard of 50 µg/m^3.

The $PM_{2.5}$ data for 2063 are shown in Figure 10.

Figure 10: Daily average $PM_{2.5}$ from operations 2063



As with PM_{10}, the predicted $PM_{2.5}$ concentrations in 2063 are higher than those predicted in 2030 but still below the NEPM standard. Again, the highest impact is predicted for Rossmore and Badgerys Creek.

5.2.3 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to PM_{10}. Australian studies (NEPC, 2012; EPHC 2006) have found associations between PM_{10} levels currently experienced in Australian cities and the following health outcomes:

- increases in daily mortality
- increases in hospital admissions
    - respiratory disease
    - cardiovascular disease
    - cardiac disease
    - pneumonia and bronchitis
- increases in emergency room attendances for asthma

These health outcomes have been assessed in this health risk assessment for the relevant age groups.

Although no studies investigating the long term effects of exposure to PM_{10} on health have been conducted in Australia, there have been several international studies that have shown strong associations between long-term exposure to PM_{10} and increases in mortality. On the basis of the findings of these studies, long-term mortality has also been assessed.

There are several groups within the general population that have been identified as being more vulnerable to the effects of air pollution. These include:

- the elderly
- people with existing cardiovascular and respiratory disease
- people with asthma
- low socio-economic groups
- children

Compared to healthy adults, children are generally more sensitive to air pollutants as their exposure is generally higher. The reasons for this are that children inhale more air per minute and have a larger contact lung surface area relative to their size compared to adults. Other factors that increase the potential for exposure in children are that children generally spend more time outdoors and more time exercising.

Recent studies have shown that people who have a low socioeconomic status (SES) also form a group within the population that is particularly vulnerable to the effects of air pollution. This is largely due to the fact that people within these groups usually have poorer health status than people within higher SES groups. They may also have poorer access to medical care. In addition, they usually live in areas that are more polluted (e.g., near major roads or near industry) as property is generally cheaper in these areas.

To calculate the number of people that might be affected by air pollution, exposure-response functions for each outcome being assessed are required. These functions are a measure of the change in the health outcome within the population for a given change in PM_{10} or $PM_{2.5}$ concentration.

The exposure-response functions in Table 3 and 4 have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005; EPHC, 2011). The use of Australian meta-analyses is consistent with the NHMRC (2006) and NEPC (2011) recommendations for selecting exposure-response functions.

The exposure-response functions for long-term exposure to PM_{10} and $PM_{2.5}$ have been taken from the American Cancer Society study (HEI, 2009). This study is considered by the WHO as the most reliable

study to assess long-term effects of air pollution. The use of these values is also consistent with the recommendations made by NHMRC (2006) and NEPC (2011).

Table 3 Exposure Response Functions for PM_{10} Selected Health Outcomes (Taken from EPHC, 2011; HEI, 2009)

Outcome	**Averaging Period**	**Exposure Response Function per 1 µg/m³ increase in PM_{10}**
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.004
Daily all-cause mortality (non-accidental) all ages	24 hours	0.002
Daily mortality cardiovascular disease - all ages	24 hours	0.002
Hospital Admissions respiratory disease 65+ years	24 hours	0.003
Hospital Admissions cardiac disease 65+ years	24 hours	0.002
Hospital Admissions pneumonia and bronchitis 65+ years	24 hours	0.0013
Hospital Admissions respiratory disease 15-64 years	24 hours	0.003
ED Visits Asthma 1-14 years	24 hours	0.015

Table 4 shows the exposure response functions used for $PM_{2.5}$.

Table 4: Exposure Response Functions for $PM_{2.5}$ Selected Health Outcomes Taken from EPHC, 2011; HEI, 2009)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in $PM_{2.5}$
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.006
Annual cardiopulmonary mortality 30+	Annual average	0.014
Annual mortality ischemic heart disease 30+ years	Annual average	0.024
Annual mortality lung cancer 30+ years	Annual average	0.014
Daily all-cause mortality (non-accidental) all ages	24 hours	0.0023
Daily mortality cardiovascular disease - all ages	24 hours	0.0013
Hospital Admissions respiratory disease 65+ years	24 hours	0.004
Hospital Admissions cardiac disease 65+ years	24 hours	0.005
Hospital Admissions cardiovascular disease 65+ years	24 hours	0.003
Hospital Admissions ischemic heart disease 65+ years	24 hours	0.004
Hospital Admissions COPD 65+ years	24 hours	0.004
Hospital Admissions pneumonia and bronchitis 65+ years	24 hours	0.005
Hospital Admissions respiratory disease 15-64 years	24 hours	0.003
ED Visits Asthma 1-14 years	24 hours	0.0015

Using the predicted annual average and 24 hour average PM_{10} and $PM_{2.5}$ concentrations for the residential receptors previously identified, the population in each of these locations (see Table 1) and the exposure response function in Tables 3 and 4, the health effects attributable to PM_{10} and $PM_{2.5}$ have been calculated using the following equation:

Number of attributable cases = exposure response function (Change in health outcome) per 1 µg/m³ increase in PM x PM concentration x baseline health incidence rate/ 100,000 population x actual population

In this assessment it, is assumed that the data for each receptor point, e.g., Bringelly, are representative of the whole population of that suburb.

The baseline health statistics for Sydney were used in this assessment. The number of attributable cases is shown in Table 5 (PM_{10}) and Table 6 ($PM_{2.5}$) for operations in 2030. The number of attributable cases is the increase in the number, for example hospital admissions for respiratory disease, that may arise from exposure to PM from Stage 1 airport operations. If the same example is used, the results in Table 5 show that for Rossmore, based on the predicted increase in daily PM_{10} concentrations, there would be an additional 0.05 hospital admissions per year which is equivalent to 5 additional hospital admissions per 100 years which may be attributed to emissions of PM_{10} from the Stage 1 operations of the airport. This

increase is the increase in the admissions relative to the existing situation for a given area, eg., Rossmore using the example above.

Table 5: Predicted attributable health outcomes due to PM_{10} from Stage 1 operation (2030)

Health Outcome	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
Annual Mortality 30+ years	0.06	0.01	0.002	0.1	0.001	0.03	0.013	0.08	0.02
Daily Mortality all causes all ages	0.03	0.005	0.001	0.05	0.005	0.01	0.006	0.04	0.007
Daily Mortality Cardiovascular Disease all ages	0.01	0.002	0.0003	0.02	0.002	0.004	0.002	0.01	0.003
Hospital Admissions Respiratory Disease 65+	0.03	0.007	0.001	0.07	0.006	0.01	0.007	0.05	0.009
Hospital Admissions Cardiac Disease 65+	0.03	0.007	0.001	0.07	0.006	0.01	0.007	0.05	0.009
Hospital Admissions Pneumonia and Bronchitis 65+	0.003	0.0008	0.0001	0.008	0.0007	0.002	0.0008	0.006	0.001
Hospital Admissions Respiratory Disease 15-64 years	0.04	0.007	0.001	0.06	0.007	0.02	0.008	0.05	0.01
Emergency Department Visits 0-14 years	0.009	0.002	0.0003	0.01	0.001	0.004	0.002	0.01	0.002

As can be seen from Table 5, the predicted number of attributable cases due to PM_{10} from proposed Stage 1 operation is low. The highest risk would be for all-cause mortality from long-term exposures with between 1 additional death per 1000 years and 1 additional death per 10 years attributable to PM_{10} from the airport operation in 2030. The highest risk is predicted for Kemps Creek with an additional 1 death per 10 years predicted in a population of 2,309 (Table 1). All other risks are lower than that predicted for long-term mortality. According to Health Statistics NSW in 2012-13 there were 10,127 deaths in the Western Sydney Local Health District due to all causes. This is in a population of 904,886 people.

The results for $PM_{2.5}$ for 2030 are shown in Table 6.

Table 6: Predicted attributable health outcomes due to $PM_{2.5}$ from Stage 1 operation (2030)

HEALTH OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.05	0.009	0.002	0.06	0.008	0.006	0.01	0.06	0.01
Cardiopulmonary mortality 30+years (long-term)	0.05	0.009	0.002	0.06	0.008	0.006	0.01	0.06	0.01
Ischemic Heart Disease 30+ years (long-term)	0.03	0.006	0.001	0.04	0.005	0.004	0.006	0.04	0.007
Lung cancer mortality 30+ years (long-term)	0.007	0.001	0.0002	0.009	0.001	0.0009	0.001	0.009	0.002
Daily mortality all causes all ages	0.02	0.003	0.0006	0.02	0.003	0.003	0.004	0.02	0.004
Daily mortality cardiovascular disease all ages	0.003	0.006	0.0001	0.004	0.0005	0.0005	0.0006	0.004	0.0007
Hospital admissions respiratory disease 65+ years	0.02	0.005	0.008	0.04	0.004	0.003	0.005	0.03	0.005
Hospital admissions cardiac disease 65+ years	0.04	0.01	0.002	0.08	0.008	0.006	0.01	0.06	0.01
Hospital admissions pneumonia and bronchitis 65+ years	0.006	0.002	0.0002	0.01	0.001	0.001	0.002	0.01	0.002
Hospital admissions cardiovascular disease 65+ years	0.03	0.008	0.001	0.07	0.007	0.005	0.009	0.06	0.009
Hospital admissions respiratory disease 15-64 years	0.02	0.004	0.0007	0.03	0.003	0.003	0.004	0.03	0.005
Emergency Department visits asthma 1-14 years	0.004	0.001	0.0002	0.006	0.0007	0.0007	0.0009	0.006	0.001

As with PM_{10}, the predicted number of attributable cases from $PM_{2.5}$ from proposed Stage 1 operation of the airport is low. The highest predicted risk is for all-cause mortality and cardiopulmonary mortality from long-term exposures with between 1 additional death per 1000 years and 6 additional deaths per 100 years that are attributable to $PM_{2.5}$. The highest risks are predicted for Kemps Creek and Rossmore which is a total population of 4,721. This increase in deaths is relative to the current situation in these areas. As previously noted, according to Health Statistics NSW in 2012-13 there were 10,127 deaths in the Western Sydney Local Health District due to all causes. This is in a population of 904,886 people. All other risks are lower than that predicted for these outcomes.

Tables 7 and 8 show the predicted health effects attributable to PM_{10} and $PM_{2.5}$ for the airport operation in 2063.

Table 7: Predicted attributable health outcomes due to PM_{10} from longer term airport operation (2063)

Health Outcome	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
Annual Mortality 30+ years	0.2	0.03	0.006	0.3	0.03	0.07	0.05	0.4	0.08
Daily Mortality all causes all ages	0.08	0.02	0.003	0.2	0.01	0.03	0.02	0.2	0.04
Daily Mortality Cardiovascular Disease all ages	0.03	0.006	0.001	0.05	0.004	0.01	0.007	0.06	0.01
Hospital Admissions Respiratory Disease 65+	0.08	0.02	0.004	0.2	0.02	0.03	0.03	0.2	0.04
Hospital Admissions Cardiac Disease 65+	0.08	0.02	0.004	0.2	0.02	0.04	0.03	0.3	0.05
Hospital Admissions Pneumonia and Bronchitis 65+	0.009	0.003	0.0004	0.03	0.002	0.004	0.003	0.03	0.005
Hospital Admissions Respiratory Disease 15-64 years	0.1	0.02	0.004	0.2	0.02	0.04	0.03	0.3	0.05
Emergency Department Visits 0-14 years	0.02	0.006	0.001	0.04	0.004	0.01	0.007	0.06	0.01

Table 8: Predicted attributable health outcomes due to $PM_{2.5}$ from longer term airport operation in (2063)

HEALTH OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.1	0.05	0.005	0.2	0.02	0.02	0.03	0.3	0.03
Cardiopulmonary mortality 30+years (long-term)	0.1	0.05	0.005	0.2	0.02	0.02	0.03	0.3	0.03
Ischemic Heart Disease 30+ years (long-term)	0.07	0.03	0.003	0.1	0.01	0.01	0.02	0.2	0.02
Lung cancer mortality 30+ years (long-term)	0.02	0.007	0.0007	0.02	0.003	0.002	0.005	0.04	0.004
Daily mortality all causes all ages	0.04	0.02	0.002	0.06	0.007	0.006	0.01	0.1	0.01
Daily mortality cardiovascular disease all ages	0.007	0.004	0.004	0.01	0.001	0.001	0.002	0.02	0.002
Hospital admissions respiratory disease 65+ years	0.04	0.03	0.003	0.1	0.01	0.008	0.02	0.2	0.01
Hospital admissions cardiac disease 65+ years	0.09	0.06	0.005	0.2	0.02	0.01	0.03	0.3	0.03
Hospital admissions pneumonia and bronchitis 65+ years	0.02	0.01	0.0009	0.04	0.003	0.003	0.008	0.05	0.004
Hospital admissions cardiovascular disease 65+ years	0.08	0.05	0.005	0.2	0.02	0.01	0.03	0.3	0.02
Hospital admissions respiratory disease 15-64 years	0.3	0.02	0.002	0.07	0.008	0.007	0.01	0.1	0.01
Emergency Department visits asthma 1-14 years	0.01	0.01	0.0006	0.02	0.002	0.002	0.003	0.03	0.002

The risks predicted for 2063 for both PM_{10} and $PM_{2.5}$ are higher than those predicted for 2030 operations. As can be seen from Table 7, the highest risk is for all-cause mortality from long-term exposures with between 5 additional deaths per 1000 years and 3 additional deaths per 10 years that are attributable to PM_{10}. This increase in deaths is relative to the current situation (10,127 deaths from all causes in 2012-13) in these areas. All other risks are lower than that predicted for long-term mortality.

As with PM_{10}, the number of attributable cases from $PM_{2.5}$ from the operations in 2063 at the airport are higher than those predicted for 2030. The highest predicted risk is for all-cause mortality and cardiopulmonary mortality from long-term exposures with between 5 additional deaths per 1000 years and 3 additional deaths per 10 years attributable to $PM_{2.5}$. All other risks are lower than that predicted for these outcomes. The highest predicted impacts are at Rossmore and Kemps Creek.

The potential health risks associated with proposed construction works have been calculated for the same receptors as used for the operational stages. The predicted results are shown in Tables 9 and 10 for earthworks and aviation infrastructure respectively for PM_{10} and Tables 11 and 12 for $PM_{2.5}$.

Table 9: Predicted attributable health outcomes due to PM_{10} from construction bulk earthworks

Health Outcome	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
Annual Mortality 30+ years	0.006	0.01	0.001	0.007	0.003	0.003	0.006	0.004	0.003
Daily Mortality all causes all ages	0.003	0.006	0.0006	0.003	0.001	0.002	0.003	0.002	0.001
Daily Mortality Cardiovascular Disease all ages	0.001	0.002	0.0002	0.001	0.0004	0.0005	0.001	0.0005	0.0005
Hospital Admissions Respiratory Disease 65+	0.003	0.007	0.0007	0.005	0.001	0.002	0.004	0.002	0.002
Hospital Admissions Cardiac Disease 65+	0.003	0.007	0.0007	0.005	0.001	0.002	0.004	0.002	0.002
Hospital Admissions Pneumonia and Bronchitis 65+	0.0003	0.0008	0.00007	0.0006	0.0002	0.0002	0.0004	0.0002	0.0002
Hospital Admissions Respiratory Disease 15-64 years	0.004	0.007	0.008	0.004	0.002	0.002	0.004	0.002	0.002
Emergency Department Visits 0-14 years	0.0009	0.002	0.0002	0.0009	0.0003	0.0003	0.0009	0.0005	0.0004

Table 10: Predicted attributable health outcomes due to PM_{10} from construction of aviation infrastructure

Health Outcome	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
Annual Mortality 30+ years	0.01	0.01	0.002	0.01	0.004	0.006	0.01	0.006	0.005
Daily Mortality all causes all ages	0.006	0.006	0.001	0.007	0.002	0.003	0.006	0.003	0.002
Daily Mortality Cardiovascular Disease all ages	0.002	0.002	0.0004	0.002	0.0007	0.0009	0.002	0.001	0.0003
Hospital Admissions Respiratory Disease 65+	0.006	0.007	0.001	0.009	0.003	0.003	0.008	0.004	0.003
Hospital Admissions Cardiac Disease 65+	0.006	0.008	0.002	0.01	0.003	0.003	0.008	0.004	0.003
Hospital Admissions Pneumonia and Bronchitis 65+	0.0007	0.0008	0.0002	0.001	0.0003	0.0003	0.0009	0.0005	0.0003
Hospital Admissions Respiratory Disease 15-64 years	0.008	0.008	0.002	0.009	0.003	0.003	0.009	0.004	0.003
Emergency Department Visits 0-14 years	0.002	0.002	0.0004	0.002	0.0006	0.0008	0.002	0.001	0.0007

Table 11: Predicted attributable health outcomes due to $PM_{2.5}$ from construction bulk earthworks

HEALTH OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.004	0.004	0.0007	0.003	0.001	0.002	0.003	0.002	0.002
Cardiopulmonary mortality 30+years (long-term)	0.004	0.004	0.0007	0.003	0.001	0.002	0.003	0.002	0.002
Ischemic Heart Disease 30+ years (long-term)	0.002	0.002	0.0005	0.002	0.0008	0.001	0.002	0.001	0.0009
Lung cancer mortality 30+ years (long-term)	0.0005	0.0005	0.0001	0.005	0.0002	0.0003	0.0005	0.0003	0.0002
Daily mortality all causes all ages	0.0009	0.001	0.0003	0.001	0.0005	0.0006	0.001	0.0007	0.0006
Daily mortality cardiovascular disease all ages	0.0002	0.0002	0.00005	0.0002	0.00008	0.0001	0.0002	0.0001	0.0001
Hospital admissions respiratory disease 65+ years	0.001	0.002	0.0004	0.002	0.0007	0.0007	0.002	0.001	0.0008
Hospital admissions cardiac disease 65+ years	0.002	0.004	0.0007	0.004	0.001	0.001	0.003	0.002	0.001
Hospital admissions pneumonia and bronchitis 65+ years	0.0003	0.0007	0.0001	0.0007	0.0002	0.0002	0.0005	0.0004	0.0003
Hospital admissions cardiovascular disease 65+ years	0.002	0.003	0.0003	0.004	0.001	0.001	0.003	0.003	0.001
Hospital admissions respiratory disease 15-64 years	0.007	0.002	0.00008	0.001	0.0006	0.0007	0.001	0.001	0.0007
Emergency Department visits asthma 1-14 years	0.0002	0.0004	0.0006	0.003	0.0001	0.0002	0.0003	0.0003	0.0002

Table 12: Predicted attributable health outcomes due to $PM_{2.5}$ from construction of aviation infrastructure

HEALTH OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.02	0.02	0.003	0.01	0.005	0.006	0.01	0.008	0.006
Cardiopulmonary mortality 30+years (long-term)	0.02	0.02	0.003	0.01	0.005	0.006	0.01	0.008	0.006
Ischemic Heart Disease 30+ years (long-term)	0.009	0.01	0.002	0.009	0.003	0.004	0.006	0.005	0.004
Lung cancer mortality 30+ years (long-term)	0.002	0.002	0.007	0.002	0.0007	0.0009	0.001	0.001	0.0008
Daily mortality all causes all ages	0.005	0.007	0.001	0.005	0.002	0.003	0.004	0.003	0.002
Daily mortality cardiovascular disease all ages	0.001	0.001	0.002	0.0009	0.0004	0.0005	0.007	0.0005	0.0004
Hospital admissions respiratory disease 65+ years	0.006	0.009	0.002	0.009	0.003	0.003	0.005	0.004	0.003
Hospital admissions cardiac disease 65+ years	0.01	0.02	0.003	0.02	0.005	0.006	0.01	0.008	0.005
Hospital admissions pneumonia and bronchitis 65+ years	0.002	0.003	0.0005	0.003	0.0009	0.001	0.002	0.001	0.0009
Hospital admissions cardiovascular disease 65+ years	0.01	0.02	0.003	0.006	0.005	0.005	0.007	0.007	0.005
Hospital admissions respiratory disease 15-64 years	0.007	0.008	0.001	0.001	0.002	0.003	0.005	0.003	0.003
Emergency Department visits asthma 1-14 years	0.001	0.002	0.0003	0.02	0.0005	0.0007	0.001	0.0008	0.0005

The risks predicted for the construction scenarios – bulk earthworks and aviation infrastructure - for both PM_{10} and $PM_{2.5}$ are lower than those predicted for both the 2030 and 2063 operational scenarios. As can be seen from Table 9, the highest predicted risk is for all-cause mortality from long-term exposures with between 1 additional death per 1000 years and 1 additional death per 100 years that are attributable to PM_{10}. All other risks are lower than that predicted for long-term mortality. For construction of aviation infrastructure, risks are similar to bulk earthworks with the highest risks for all-cause mortality from long-term exposures with between 2 additional death per 1000 years and 1 additional death per 100 years that are attributable to PM_{10}. The highest impacts are predicted at Luddenham, Bringelly, Kemps Creek and Badgerys Creek.

As with PM_{10}, the number of attributable cases from $PM_{2.5}$ from the construction scenarios are lower than those predicted for the operational scenarios in both 2030 and 2063. The highest predicted risk is for all-cause mortality and cardiopulmonary mortality from long-term exposures with between 7 additional deaths per 10,000 years and 4 additional deaths per 1000 years that are attributable to $PM_{2.5}$ from construction of bulk earthworks and between 3 additional deaths per 1000 years and 2 additional deaths per 100 years that are attributable to $PM_{2.5}$ from aviation infrastructure construction. All other risks are lower than that predicted for these outcomes. The highest predicted impacts are at Bringelly and Luddenham.

It should be noted that the construction of the airport will occur for a period of less than 10 years. Therefore the predicted risk levels associated with the construction phase discussed above are unlikely to be realised as they are predicted to occur over much longer timeframes – 100 to 10,000 years.

5.3 Nitrogen Dioxide (NO_2)

As with PM_{10} and $PM_{2.5}$, the main sources of NO_2 are roadways external to the airport site. The next main contributor would be aircraft movements. The operation of Auxiliary Power Units (APUs) and ground support equipment (GSE) would also influence the predicted pollutant concentrations. The largest contributor of on-site emissions is anticipated to be associated with aircraft taking off and landing, for both Stage 1 and longer term development scenarios. The external road infrastructure was shown to be a significant contributor of predicted off-site ground level concentrations, particularly for those receptors located in close proximity to existing or proposed new roadways.

5.3.1 Literature Review of the Health Effects of NO_2

In recent years, there has been an increased interest in the health effects of nitrogen dioxide. The REVIHHAP study (WHO, 2013) investigated the new studies of both long-term and short-term exposure to NO_2 and associations with mortality, hospital admissions and respiratory symptoms and concluded that these new studies show that short-term exposure to NO_2 is associated with increases in these outcomes. Studies of the long-term effects of exposure to NO_2 have shown associations with both mortality and morbidity outcomes. The effects that have been observed for both long-term and short-term exposure are occurring below current WHO air quality guidelines for NO_2 which are lower than the current NEPM standards. Controlled human exposure and toxicological studies provide support for biological mechanisms for the effects that are observed in epidemiological studies and provide evidence for a causal relationship between exposure to NO_2 and these outcomes. The most recent studies have provided evidence that NO_2 has an independent effect from other pollutants. Epidemiological studies of long-term effects of NO_2 exposure on mortality (both respiratory and cardiovascular causes) and with children's respiratory symptoms and lung function also support the conclusion that NO_2 has an independent effect on health.

Controlled human exposure studies show increased inflammation of the airways and airway hyper responsiveness at nitrogen dioxide levels down to 0.2 ppm in healthy individuals. As previously mentioned, the general population includes sensitive populations and effects are likely to occur at lower levels of NO_2 than those where adverse effects have been observed in controlled human exposure studies.

Short-term exposure to NO_2 has been linked to increases in 'all-cause', cardiovascular and respiratory mortality. The effects were greater in people 65 years of age and older and for respiratory mortality (WHO, 2013). Epidemiological studies provide no evidence of a threshold for the effect. Recent studies have provided evidence that has strengthened the association with hospital admissions and emergency department visits for respiratory disease including all respiratory causes, asthma and chronic obstructive pulmonary disease (COPD) (WHO, 2013). Strong associations have been observed for all respiratory causes in people 65 years and older and for children with asthma. The effects are not as strong for cardiovascular causes and in some cases, there is no consistent effect observed with cardiovascular effects. There is some evidence for an association with cardiac hospital admissions but these findings are not consistent across studies.

Panel studies of children with asthma show associations between nitrogen dioxide and reductions in lung function, increases in cough, night-time asthma and school absenteeism. There is also an increase in symptoms in asthmatic children and changes in lung function observed, as well as increases in airway inflammation and hyper responsiveness. Controlled human exposure and animal toxicological studies support the findings of the epidemiological and panel studies.

Long-term exposure to NO_2 has been linked to deficits in lung function growth. These findings have been found in studies in California, Mexico and Sweden. In these studies, the effects on nitrogen dioxide were greater than those observed for other pollutants including $PM_{2.5}$. There is also strong evidence of an association between long-term exposure to NO_2 and the incidence of asthma and

wheezing. This new evidence suggests that NO_2 exposure may actually cause asthma rather than just exacerbate existing asthma (WHO, 2013; USEPA, 2014). There have also been studies that have shown increases in mortality with long-term exposure NO_2 including all-cause, cardiovascular (especially ischaemic heart disease) lung cancer and respiratory mortality. These effects are similar to those observed for $PM_{2.5}$ if not larger and are independent of $PM_{2.5}$.

Australian studies have reported similar associations as overseas studies between hospitalisation for respiratory effects, including asthma, and daily NO_2 (Morgan et al. 1998a; Barnett et al. 2005; Erbas et al., 2005; Jalaludin et al. 2004; Rodriguez et al., 2007). In a meta-analysis of results from five Australian and two New Zealand cities, Barnett et al. (2005) analysed hospital admissions for three age groups of children. Significant increases in hospital admissions for respiratory disease (1–4, 5–14 years) and asthma (5–14 years) were associated with interquartile range increases in either 1-hr or 24-hr NO_2. The largest association reported was a 6.0% increase in asthma admissions with a 5.1 part per billion (ppb) increase in 24- 30 hr NO_2 and the effect was not reduced by inclusion of PM_{10} in the analysis. A meta-analysis of the associations between pollutants and cardiovascular hospital admissions in the elderly in Brisbane, Canberra, Melbourne, Perth, Sydney, Auckland and Christchurch found significant associations between CO, NO_2, and particles and five categories of cardiovascular disease admissions. The two largest statistically significant increases were for cardiac failure, with a 6.9% increase for a 5.1-ppb unit increase in NO_2 and a 6.0% increase for a 0.9-ppm increase in CO (Barnett et al, 2006).

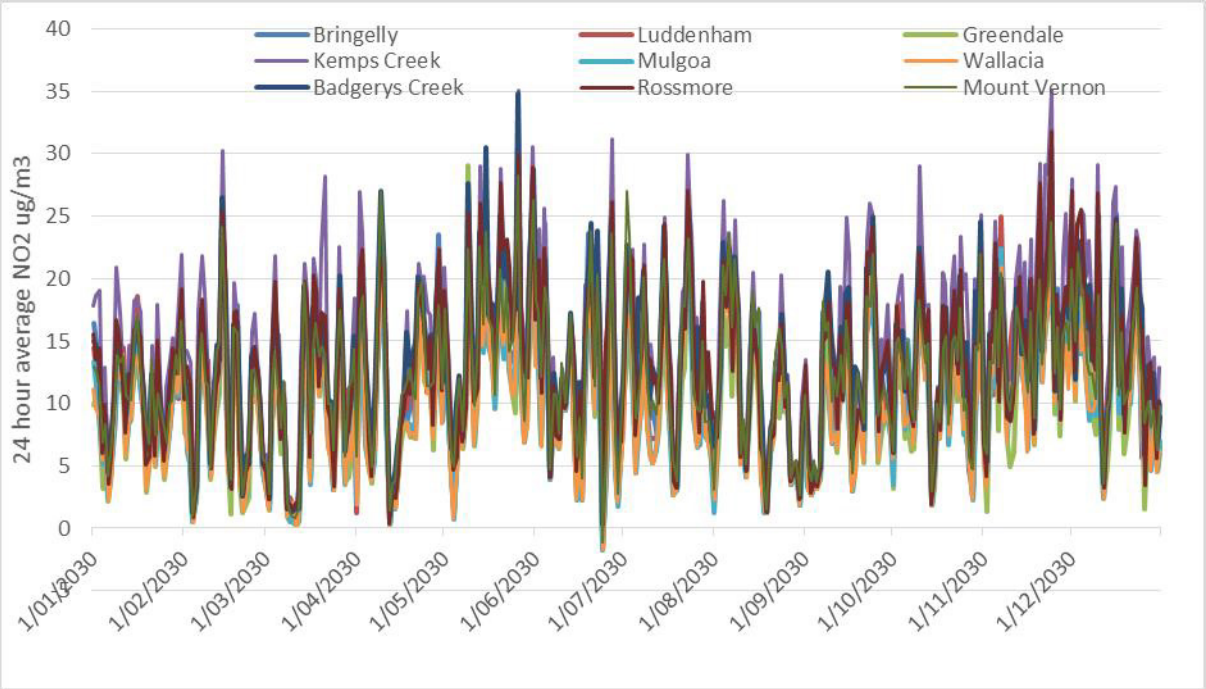
In the ACHAPS panel study (SCEW, 2012), the most consistent adverse effect was that increased NO_2 exposure was associated with an increased risk of cough and wheezing during the day and night, and increased use of bronchodilators for symptom relief. Relationships between NO_2 and night symptoms and effects were greater for NO_2 24-hr than for NO_2 1-hr and were more consistent.

The ACHAPS cross-sectional study shows consistent evidence of respiratory adverse effects of NO_2 for both recent and life-time exposure (SCEW, 2012). These adverse effects are manifested as increased risk of asthma-like symptoms (in particular, wheeze), increased airway inflammation and reduced lung volumes. For current asthma and per ppb recent exposure NO_2, the odds ratio (OR) was 1.06 (1.02, 1.10), with OR per interquartile range (IQR) NO_2 1.26 (1.08, 1.48). For recent wheeze after exercise, the OR was 1.07 (1.03, 1.120) per ppb and 1.32 (1.12, 1.57) per IQR. Airways inflammation as measured by exhaled nitric oxide (NO) increased by 3% (1%-5%) and lung volume as measured by pre-bronchodilator forced expiratory volume (FEV1) and forced vital capacity (FVC) decreased by 7.1 ml (2.8-11.4) and 6.8 ml (2.7-10.9) per ppb respectively. Effect estimates were slightly smaller for lifetime exposure. Per IQR decreases in lung function measured by FEV1 and FVC, pre- and post-bronchodilator ranged from 27.5 to 29 ml.

5.3.1.1 Exposure Assessment

Air dispersion modelling conducted as part of the local air quality assessment has predicted maximum 1-hour, 24-hour average and annual average NO_x concentrations for a range of residential receptors within a 5km radius from the airport site. The daily 24-hour NO_2 concentrations at the residential receptors used in the HRA are shown in Figures 11 and 12 for 2030 and 2063 respectively.

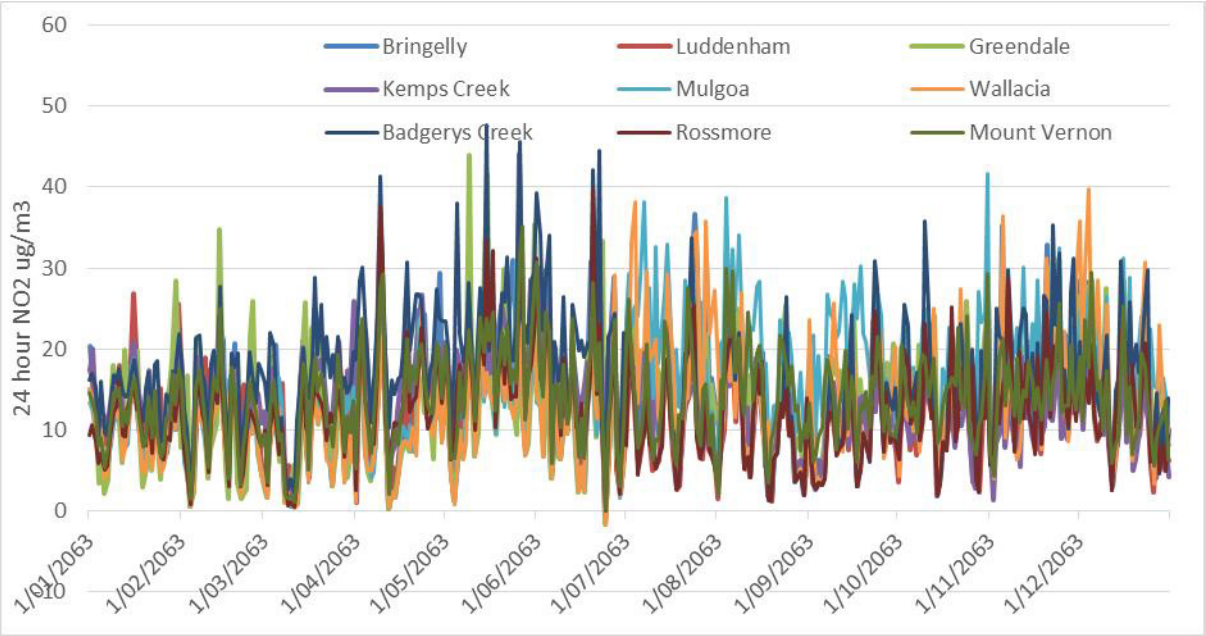
Figure 11: Predicted daily average NO_2 concentrations for 2030 operations



The data shown in Figure 11 show that all predicted levels are low. The local air quality assessment shows that for all relevant averaging periods, the predicted NO_2 levels due to the airport operations in 2030 are well below the current NEPM air quality standards. Note that the NEPM standards apply to 1-hour max and annual average concentrations. For health risk assessment the exposure response relationships are most reliable for 24-hour averages. The levels predicted at all residential locations are similar with slightly higher levels at Kemps Creek.

The data for 2063 is shown in Figure 12.

Figure 12: Predicted daily average NO_2 concentrations for 2063 operations



5.3.2 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to NO_2. Australian studies (NEPC, 2012; EPHC 2006) have found associations between NO_2 levels currently experienced in Australian cities and the following health outcomes:

- increases in daily mortality
- increases in hospital admissions
    - respiratory disease
    - cardiovascular disease
- increases in emergency room attendances for asthma

These health outcomes have been assessed in this health risk assessment for the relevant age groups.

Although no studies investigating the long term effects of exposure to NO_2 on health have been conducted in Australia, there have been several international studies that have shown strong associations between long-term exposure to NO_2 and increases in mortality. On the basis of the findings of these studies, long-term mortality has also been assessed.

The groups that were identified as being susceptible to the effects of NO_2 are:

- Elderly
- People with existing cardiovascular and respiratory disease
- People with asthma
- Low socioeconomic groups
- Children

The exposure-response functions in Table 13 have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005; EPHC, 2006). Exposure response functions are the increase in a health outcome observed per 1 $\mu g/m^3$ increase in pollutant concentration. The use of Australian meta-analyses is consistent with the NHMRC (2006) and NEPC (2011) recommendations for selecting exposure response functions.

The exposure-response functions for long-term exposure to NO_2 have been taken from the results of a cohort of more than a million adults in Rome (Cesaroni et al., 2013). This study was reviewed by the WHO as part of the REVIHHAP review (WHO, 2013). The use of this value is also consistent with the recommendations made by NHMRC (2006) and NEPC (2011).

Table 13: Exposure Response Functions for NO_2 Selected Health Outcomes (EPHC, 2005; Cesaroni et al. 2013)

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in NO_2
Annual all-cause mortality (non-accidental) 30+ years	Annual Average	0.0028
Annual cardiovascular mortality 30+ years	Annual Average	0.0028
Annual respiratory mortality 30+ years	Annual Average	0.0028
Daily all-cause mortality (non-accidental) all ages	24 –hour average	0.001
Daily mortality respiratory disease - all ages	24 –hour average	0.0023
Daily mortality cardiovascular disease - all ages	24 –hour average	0.001
Hospital Admissions respiratory disease 65+ years	24 –hour average	0.003
Hospital Admissions cardiovascular disease 65+ years	24 –hour average	0.0014
Hospital Admissions respiratory disease 15-64 years	24 –hour average	0.001
ED Visits Asthma 1-14 years	24 –hour average	0.0006

The data shown in Figures 11 and 12 have been combined with the exposure response functions in Table 13 and baseline health statistics for Sydney to calculate the number of cases attributable to NO_2 from Stage 1 operations, including traffic from roads outside of the airport site. The local population data have been used to estimate the number of people impacted in each suburb (Table 1). The number of attributable cases has been calculated using the following equation:

Number of attributable cases = exposure response function (increase in health outcome) per 1µg/m³ increase in NO_2 x NO_2 concentration x baseline health incidence rate/ 100,000 population x actual population

The number of attributable cases are summarised in Table 14 for 2030 and Table 15 for 2063.

Table 14: Predicted attributable health outcomes due to NO_2 from 2030 operations, including traffic

OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.5	0.3	0.06	0.6	0.3	0.3	0.1	0.5	0.3
Cardiovascular mortality 30+years (long-term)	0.2	0.09	0.02	0.2	0.1	0.1	0.03	0.2	0.09
Respiratory Mortality 30+ years (long-term)	0.05	0.03	0.005	0.06	0.03	0.03	0.009	0.05	0.03
Lung cancer mortality 30+ years (long-term)	0.04	0.02	0.005	0.05	0.03	0.03	0.008	0.04	0.02
Daily mortality all causes all ages	0.2	0.1	0.02	0.2	0.1	0.1	0.03	0.2	0.09
Daily mortality cardiovascular disease all ages	0.05	0.03	0.007	0.06	0.03	0.03	0.01	0.06	0.03
Daily mortality respiratory disease all ages	0.03	0.02	0.004	0.04	0.02	0.02	0.007	0.04	0.02
Hospital admissions respiratory disease 65+ years	0.4	0.2	0.05	0.6	0.3	0.2	0.09	0.5	0.2
Hospital admissions cardiovascular disease 65+ years	0.4	0.3	0.05	0.6	0.3	0.2	0.09	0.5	0.2
Hospital admissions respiratory disease 15-64 years	0.2	0.09	0.02	0.2	0.1	0.09	0.03	0.2	0.09
Emergency Department visits asthma 1-14 years	0.04	0.02	0.005	0.05	0.02	0.02	0.008	0.05	0.02

Table 15: Predicted attributable health outcomes due to NO_2 from 2063 operations, including traffic

OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.6	0.3	0.08	0.6	0.5	0.4	0.1	0.6	0.2
Cardiovascular mortality 30+years (long-term)	0.2	0.1	0.03	0.2	0.2	0.1	0.04	0.2	0.07
Respiratory Mortality 30+ years (long-term)	0.05	0.03	0.007	0.05	0.04	0.03	0.01	0.06	0.02
Lung cancer mortality 30+ years (long-term)	0.05	0.02	0.006	0.05	0.04	0.03	0.01	0.05	0.02
Daily mortality all causes all ages	0.2	0.1	0.03	0.2	0.2	0.1	0.04	0.2	0.07
Daily mortality cardiovascular disease all ages	0.06	0.03	0.009	0.06	0.05	0.04	0.01	0.07	0.02
Daily mortality respiratory disease all ages	0.04	0.02	0.006	0.04	0.03	0.03	0.009	0.04	0.02
Hospital admissions respiratory disease 65+ years	0.4	0.2	0.07	0.5	0.4	0.3	0.1	0.6	0.2
Hospital admissions cardiovascular disease 65+ years	0.09	0.07	0.02	0.1	0.09	0.07	0.02	0.1	0.05
Hospital admissions respiratory disease 15-64 years	0.2	0.09	0.03	0.2	0.2	0.1	0.04	0.2	0.07
Emergency Department visits asthma 1-14 years	0.04	0.03	0.007	0.04	0.04	0.03	0.01	0.06	0.02

The results shown in Tables 14 and 15 show that the risk from exposure to NO_2 from the operations in 2030 and 2063 are predicted to be higher than that predicted for PM_{10} and $PM_{2.5}$. Although the predicted NO_2 levels meet the NEPM standards, it is accepted that there is no threshold for exposure to NO_2 below which adverse health effects are not observed. This means that even meeting the air quality standards means that there is a level of risk associated with exposure to the relevant pollutant including NO_2.

Based on the modelling data provided for the HRA, the highest predicted risk is for long-term mortality in people over 30 years of age with between 6 additional deaths every 100 years and 6 additional deaths every 10 years predicted for 2030 and similar risks predicted for 2063. Evidence provided by NSW Health to a Parliamentary Inquiry into health effects of pollution showed that in 2006 it was estimated that between 600 and 1400 deaths per year were attributed to air pollution in the Sydney basin (NSW Parliament 2006). Based on these figures, the health impact associated with NO_2 emissions from the airport is expected to represent a small increase in current rates of long-term mortality associated with air pollution.

The results shown in Tables 14 and 15 are for the combined NO_2 emissions from traffic as well as emissions from the airport operations. To enable an assessment of the risk posed by NO_2 emissions from the airport operations in isolation, additional air dispersion modelling was conducted in the absence of traffic. This enabled an assessment of the contribution of the airport operations to the overall risk from exposure to NO_2 and helped to identify what mitigation could be implemented to reduce the overall risk. The results for 2030 and 2063 for airport operations alone are shown in Tables 16 and 17.

Table 16: Predicted attributable health outcomes due to NO_2 from 2030 operations excluding traffic

OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.4	0.2	0.05	0.4	0.3	0.3	0.07	0.3	0.2
Cardiovascular mortality 30+years (long-term)	0.1	0.08	0.02	0.1	0.09	0.08	0.02	0.1	0.05
Respiratory Mortality 30+ years (long-term)	0.03	0.02	0.005	0.03	0.03	0.02	0.006	0.03	0.01
Lung cancer mortality 30+ years (long-term)	0.03	0.02	0.004	0.03	0.02	0.02	0.006	0.03	0.01
Daily mortality all causes all ages	0.1	0.08	0.02	0.1	0.09	0.09	0.03	0.1	0.05
Daily mortality cardiovascular disease all ages	0.04	0.03	0.006	0.04	0.03	0.03	0.008	0.04	0.02
Daily mortality respiratory disease all ages	0.02	0.02	0.04	0.02	0.02	0.02	0.005	0.02	0.01
Hospital admissions respiratory disease 65+ years	0.2	0.2	0.01	0.3	0.2	0.2	0.06	0.3	0.1

Hospital admissions cardiovascular disease 65+ years	0.3	0.2	0.05	0.4	0.2	0.2	0.06	0.3	0.1
Hospital admissions respiratory disease 15-64 years	0.1	0.08	0.02	0.1	0.09	0.09	0.02	0.1	0.05
Emergency Department visits asthma 1-14 years	0.03	0.02	0.005	0.03	0.02	0.02	0.006	0.03	0.01

Table 17: Predicted attributable health outcomes due to NO_2 from 2063 operations excluding traffic

OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
All-cause mortality 30+years (long-term)	0.4	0.3	0.06	0.4	0.4	0.3	0.1	0.4	0.2
Cardiovascular mortality 30+years (long-term)	0.1	0.09	0.02	0.1	0.1	0.1	0.04	0.1	0.07
Respiratory Mortality 30+ years (long-term)	0.04	0.02	0.005	0.04	0.03	0.03	0.01	0.04	0.02
Lung cancer mortality 30+ years (long-term)	0.03	0.02	0.005	0.03	0.03	0.03	0.008	0.03	0.02
Daily mortality all causes all ages	0.1	0.09	0.02	0.1	0.1	0.1	0.04	0.1	0.07
Daily mortality cardiovascular disease all ages	0.05	0.03	0.007	0.04	0.04	0.04	0.01	0.05	0.02
Daily mortality respiratory disease all ages	0.03	0.02	0.004	0.03	0.03	0.02	0.007	0.03	0.01
Hospital admissions respiratory disease 65+ years	0.3	0.2	0.05	0.4	0.3	0.2	0.09	0.4	0.2

Hospital admissions cardiovascular disease 65+ years	0.09	0.2	0.05	0.4	0.4	0.3	0.1	0.4	0.2
Hospital admissions respiratory disease 15-64 years	0.1	0.08	0.02	0.2	0.1	0.1	0.03	0.1	0.06
Emergency Department visits asthma 1-14 years	0.03	0.02	0.006	0.03	0.03	0.03	0.008	0.04	0.02

As can be seen from Tables 16 and 17, the predicted impact of the airport operations in isolation on surrounding suburbs is variable. By removing traffic emissions, there was a reduction in risk in both 2030 and 2063 in Bringelly, Kemps Creek and Rossmore but there was no change in Luddenham in 2030 and a small reduction in 2063. At Rossmore, the modelling indicates that traffic emissions may contribute to the predicted risk in that area. However, even without traffic emissions, the airport operations alone may lead to 4 additional deaths every 10 years due to NO_2. As outlined earlier, evidence provided by NSW Health to a Parliamentary Inquiry into health effects of pollution showed that in 2006 it was estimated that between 600 and 1400 deaths per year were attributed to air pollution in the Sydney basin (NSW Parliament 2006). Based on these figures, the health impact associated with the NO_2 emissions from the airport are expected to represent a small increase in current rates of long-term mortality associated with air pollution.

These results suggest that mitigation measures outlined in the local air quality report should be implemented to reduce community exposure to NO_2. It should be noted that the air quality predictions used in the HRA do not include the implementation of any mitigation measures to reduce NO_x emissions.

The dispersion modelling conducted for the local air quality assessment (Pacific Environment, 2015) identified that NO_x emissions contributed 91% of the emissions from the airport and therefore mitigation and management measures focus on reducing NO_x emissions. It is noted that NO_x emissions from GSE and APUs also play an important role, so the mitigation measure such as the installation of fixed system APU and GSE should reduce NO_x emissions for the longer term development as well.

A literature review was conducted of NO_x mitigation measures that have been used at other airports to mitigate NO_x emissions. These are discussed in the Local Air Quality Report Pacific Environment, 2015 , Section 5.4.2) and include a range of measures that would lead to reduction in NO_2 concentrations and associated health risks.

5.4 Sulfur Dioxide (SO_2)

The main sources of SO_2 with the exception of roadways external to the airport site were aircraft movements. The operation of Auxiliary Power Units (APUs) and ground support equipment (GSE) also had an influence on the predicted pollutant concentrations. The largest contributor of on-site emissions is anticipated to be associated with aircraft taking off and landing, for both the proposed Stage 1 and longer term development. The external road infrastructure was shown to be a significant contributor to predicted off-site ground level concentrations, particularly for those receptors located in close proximity to existing or proposed new roadways.

5.4.1 Literature Review of the Health Effects of SO_2

The health effects of sulfur dioxide (SO_2) linked to ambient air exposures have been well studied and reviewed by international agencies such as NEPC (2010), USEPA (2008), WHO (2005) and California EPA (OEHHA, 2011, 2000).

A large number of population-based epidemiological studies have reported a link between short term SO_2 exposure and daily mortality and respiratory and cardiovascular effects. The associations persist when other pollutants, such as particles, are controlled for. The epidemiological evidence is supported by controlled human exposure studies and animal toxicology studies. The strongest evidence comes from controlled human exposure studies examining short term exposure to SO_2 and respiratory effects. These studies have exposed volunteers to SO_2 for periods ranging from 5–10 min up to one hour. Adverse effects, such as sneezing or shortness of breath, occur within the first few minutes after inhalation and are not changed by further exposure. The effects are greater when the person is exercising, and are most pronounced in people with asthma and other respiratory conditions such as COPD, and particularly in exercising asthmatics.

A large number of epidemiological studies in cities in various parts of the world, including the United States, Canada and Europe, have reported associations between exposure to ambient levels of sulfur dioxide and increases in all-cause (non-accidental) and respiratory and cardiovascular mortality, often at mean 24-h average levels of <10ppb (Biggeri et al. 2005; Samet et al., 2000a; Dominici et al., 2003; Burnett et al., 1998a, 2000, 2004; Katsouyanni et al. 1997, 2006; Samoli et al., 2001, 2003; US EPA, 2008; Stieb et al. 2002, 2003). The mortality effect estimates for cardiovascular and respiratory causes are generally larger than for all-cause mortality (Zmirou et al., 1998), and the effect estimates for respiratory mortality are larger than the cardiovascular mortality, suggesting a stronger association of SO_2 with respiratory mortality compared to cardiovascular mortality. The mortality effect estimates from the multipollutant models in the multicity studies suggest some extent of confounding between SO_2 and particles and/or NO_2 (USEPA, 2008).

The epidemiological evidence, supported by controlled human exposure studies and a limited number of animal toxicological studies conducted at near ambient concentrations, indicate an association between short-term exposure to SO_2 and several measures of respiratory health, including respiratory symptoms, inflammation, and airway hyperresponsiveness.

The epidemiological evidence further indicates that the SO_2-related respiratory effects (≥ 1-hour to 24-h average) are more pronounced in asthmatic children and older adults (65+ years). In the limited number of studies that examined potential confounding by co-pollutants through multipollutant models, the SO_2 effect was generally found to be robust after adjusting for particles and other co-pollutants (USEPA, 2008).

The strongest evidence for a causal relationship between respiratory morbidity and short term exposure to SO_2 comes from human clinical studies reporting respiratory symptoms and decreased lung function following peak exposures of 5–10min duration to SO_2. The exact duration is not critical, however, because responses occur very rapidly, within the first few minutes from commencement of inhalation; continuing the exposure further does not increase the effects. These effects have been observed consistently across studies involving mild to moderate asthmatics during exercise. Statistically significant decrements in lung function accompanied by respiratory symptoms including wheeze, chest tightness and shortness of breath have been clearly demonstrated following exposure to 0.4–0.6ppm SO_2.

Several studies have observed positive associations between ambient SO_2 concentrations and emergency department visits or hospital admissions for cardiovascular diseases (e.g., all cardiovascular diseases, cardiac diseases, cerebrovascular diseases) particularly among individuals 65+ years of age, but results are not consistent across studies. The strongest evidence comes from a large multicity study conducted in Spain (Ballester et al. 2006) that observed statistically significant positive associations between ambient SO_2 and cardiovascular disease admissions.

A large body of epidemiological studies generally report consistent and robust associations between ambient SO_2 concentrations and emergency department visits and hospitalizations for all respiratory causes, particularly among children and older adults (65+ years), and for asthma and chronic obstructive pulmonary disease (COPD) (USEPA, 2008).

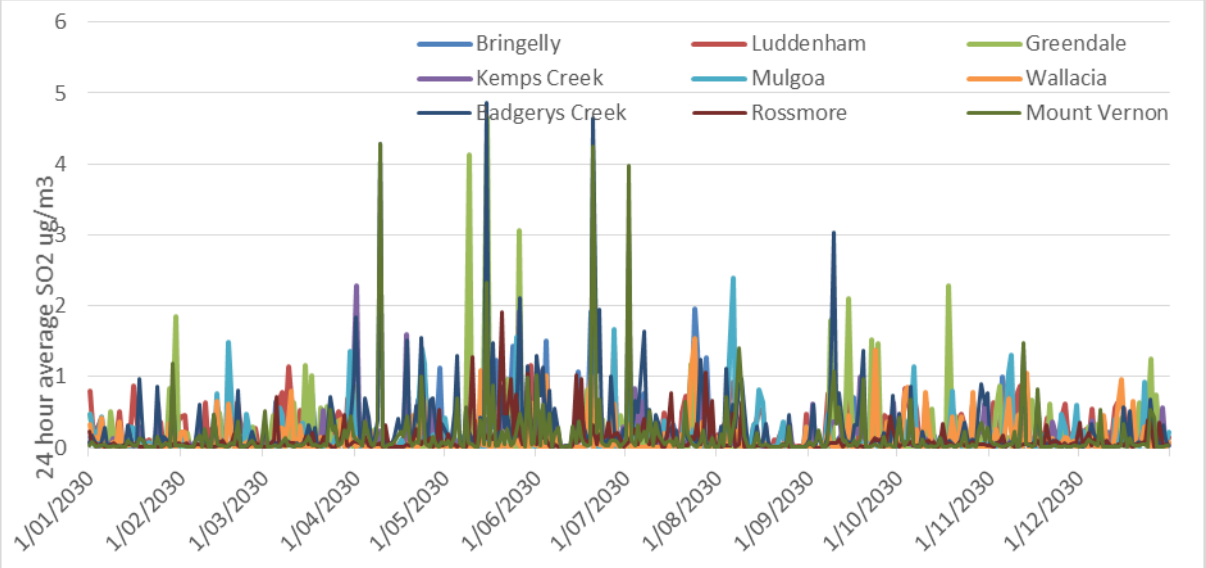
In a case-crossover study of air pollution and child respiratory health undertaken in five Australian and two New Zealand cities, Barnett et al. (2005) found a statistically significant increase in hospital admissions and SO_2 with an interquartile range of 5.4ppb for 1-hour SO_2. The ambient levels recorded during the study included: SO_2 1-hour mean (3 cities) 7.1ppb, range of means 3.7 to 10.1ppb; 24-hour mean (4 cities) 4.5ppb, range of means 0.9 to 4.3ppb. In the 1–4 year age group there was evidence of seasonal impacts on pneumonia and acute bronchitis admissions for SO_2 (May to October 4.9% increase 95% CI, 0.6–10.8%, November to April 10.4% increase 95% CI, 2.1–19.4%) (Barnett et al. 2005).

A study of 123,840 singleton births of over 20 weeks gestation in Sydney, between 1998 and 2000, found that 4.9% of babies were born at less than 37 weeks gestation. The mean of the one-hour maximum SO_2 levels was 3.6ppb. SO_2 level in early pregnancy had a large adverse impact on gestational age in those infants conceived in autumn and winter for a 1ppb increase in SO_2. The authors noted that SO_2 appears to be an important pollutant, despite SO_2 levels in Sydney being well below the national standard, with vehicular traffic being the primary source and it is conceivable that SO_2 is a marker for traffic related air pollutants in the study (Jalaludin et al 2007).

5.4.2 Exposure Assessment

Air dispersion modelling conducted as part of the local air quality assessment has predicted maximum 1-hour, 24-hour average and annual average SO_2 concentrations for a range of receptors within the surrounding suburbs. The daily 24-hour SO_2 concentrations at the most affected receptors used in the HRA are shown in Figure 13 for 2030. Air dispersion modelling for SO_2 for 2063 was not conducted.

Figure 13: Predicted daily 24-hour Average SO_2 for operations in 2030



The data in Figure 13 show that all levels are well below the current NEPM air quality standards of 228 µg/m^3. The levels are similar across most locations but higher at the Badgerys Creek, Greendale and Mount Vernon locations. The SO_2 data generated in the local air quality assessment has been used to calculate the risk of adverse health outcomes associated with exposure to SO_2 from the airport operations in 2030.

5.4.2.1 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to SO_2. Australian studies (NEPC, 2012; EPHC 2006) have found associations between SO_2 levels currently experienced in Australian cities and the following health outcomes:

- increases in daily mortality
- hospital admissions
    - respiratory disease
    - cardiovascular disease
- emergency room attendances for asthma

These health outcomes have been assessed in this health risk assessment for the relevant age groups. The groups that were identified as being susceptible to the effects of SO_2 are:

- the elderly
- people with existing cardiovascular and respiratory disease
- people with asthma
- children

The exposure-response functions in Table 18 for mortality outcomes, have taken from the results of the APHEA2 study in Europe (Katsouyanni et al., 2006). This study was a large meta-analysis across a number of cities and provides a robust exposure-response function in the absence of Australian data. The exposure-response function for hospital admission for respiratory disease has been taken from an Australian multicity meta-analysis (Simpson et al., 2005). A study by Jalaudin et al., (2008) examining the association between emergency department attendances for asthma in children conducted in Sydney has been used as the basis for the exposure-response function for this outcome. No studies investigating the long term effects of exposure to SO_2 on health were identified.

Table 18: Exposure Response Functions for SO_2

Outcome	Averaging Period	Exposure Response Function per 1 µg/m³ increase in SO_2
Daily all-cause mortality (non-accidental) all ages	24 –hour average	0.0006
Daily mortality respiratory disease - all ages	24 –hour average	0.0013
Daily mortality cardiovascular disease - all ages	24 –hour average	0.0008
Hospital Admissions respiratory disease 65+ years	1 –hour maximum	0.002
ED Visits Asthma 1-14 years	24 –hour average	0.008

The data shown in Figure 14 have been combined with the exposure response functions in Table 18 and baseline health statistics for Sydney to calculate the number of cases attributable to SO_2 from the airport operations. The number of attributable cases has been calculated using the following equation:

Number of attributable cases = exposure response function (increase in health outcome) per 1 µg/m³ increase in SO_2 x SO_2 concentration x baseline health incidence rate/ 100,000 population x actual population

The number of predicted attributable cases are summarised in Table 19.

Table 19: Predicted attributable health outcomes due to SO_2 from Stage 1 operation (2030)

OUTCOME	Bringelly	Luddenham	Greendale	Kemps Creek	Mulgoa	Wallacia	Badgerys Creek	Rossmore	Mount Vernon
Daily mortality all causes all ages	0.001	0.001	0.0002	0.001	0.0008	0.0007	0.0005	0.0009	0.0006
Daily mortality cardiovascular disease all ages	0.0005	0.0005	0.00009	0.0005	0.0004	0.0003	0.0002	0.0004	0.0003
Daily mortality respiratory disease all ages	0.0002	0.0002	0.00004	0.0002	0.0002	0.0001	0.00009	0.0002	0.0001
Hospital admissions respiratory disease 65+ years	0.003	0.003	0.0005	0.004	0.002	0.002	0.001	0.003	0.002
Emergency Department visits asthma 1-14 years	0.006	0.007	0.001	0.006	0.004	0.004	0.002	0.005	0.003

The results in Table 19 show that the predicted risk from exposure to SO_2 from the airport operations in 2030 is very low. Based on the modelling data provided for the HRA, the highest risk is for hospital admissions from respiratory causes with approximately 3 additional admissions per thousand years. All other predicted risks are lower than this.

5.5 Health Effects of Carbon Monoxide (CO)

5.5.1 Literature Review of the Health Effects of CO

The health effects of carbon monoxide are based on the ability of carbon monoxide to remove haemoglobin from blood forming carboxyhaemoglobin (COHb). The clearest evidence of the health effects associated with short-term exposure to CO is provided by studies of cardiovascular morbidity. The combined health effects evidence supports a likely causal relationship for this outcome. Controlled human exposure studies provide strong evidence of independent effects of CO on cardiac function with effects being observed in patients with chronic airways disease following short-term CO exposures resulting in 2 to 2.4% of COHb. Epidemiological studies of emergency department visits and hospital admissions for ischaemic heart disease report consistent positive associations with additional preliminary evidence for an increase in cardiovascular-related mortality provided by multi-city studies. This epidemiological evidence is coherent with ischemia related effects observed in control human exposure studies. New toxicological evidence suggests that other mechanisms involving altered cellular signalling may play a role in cardiovascular disease outcomes following carbon monoxide exposure.

Consistent decreases in time to onset of exercise induced angina along with changes in the heart that are indicative of myocardial ischaemia were observed in individuals with coronary artery disease (CAD) following controlled CO exposures resulting in COHb concentrations of 2 to 6% with no evidence of a threshold at the lowest levels tested. Volunteers who participated in controlled exposure studies were diagnosed with moderate to severe CAD may not be representative of the most sensitive individuals in the population. Variability in activity pattern and severity of disease combined with daily fluctuations in baseline COHb levels may influence a critical level of increased COHb which can lead to adverse cardiovascular effects in a particular individual.

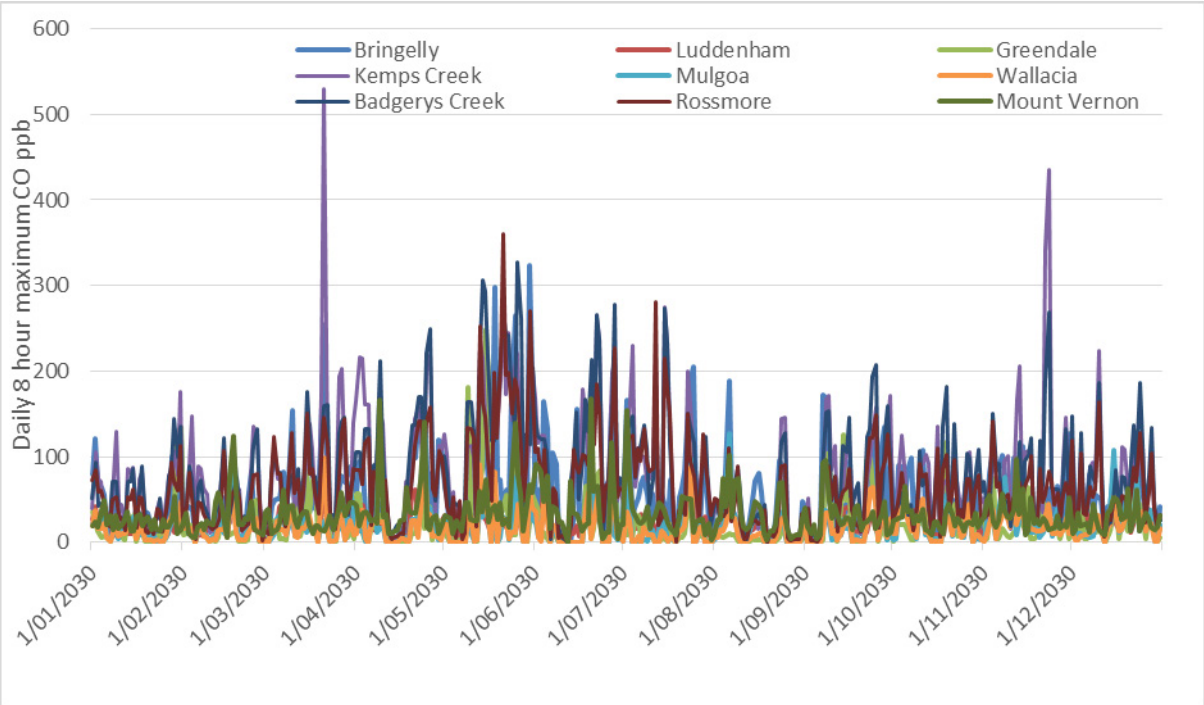
Controlled human exposure studies reviewed by the USEPA showed definitive evidence of cardiovascular effects among individuals with CAD following short-term CO exposure resulting in COHb concentrations as low as 2 to 2.4%. Evidence from control human exposure studies provide evidence of a causal relationship and reduce the uncertainties of previous assessments on the health effects of carbon monoxide. It is the consistent and coherent evidence from epidemiologic and human clinical studies along with biological plausibility provided by the role of CO in limiting oxygen availability that is sufficient for the US EPA to conclude that a causal relationship is likely to exist between relevant short-term carbon monoxide exposures and cardiovascular morbidity.

Recent studies observed associations between ambient CO concentration and emergency department visits and hospital admissions for ischaemic heart disease, congestive heart failure and cardiovascular diseases as a whole in locations with mean 24-hour average CO concentrations ranging from 0.5 ppm to 9.4 ppm. All but one of these studies that evaluated these outcomes reported positive associations. Although CO is often considered a marker of the effects of other traffic related pollutants or mix of pollutants, evidence indicates that CO associations generally remain robust in two pollutant models and supports a direct effect of short-term ambient CO exposure on cardiovascular morbidity. The known role of CO in limiting oxygen availability lends biological plausibility to ischaemia-related health outcomes following CO exposure, however it is not clear whether small changes in COHb associated with ambient CO exposures result in substantially reduced oxygen delivery to the tissues.

5.5.2 Exposure Assessment

The air dispersion modelling conducted as part of the local air quality assessment has provided daily 8-hour maximum CO levels for worst affected locations in the surrounding suburbs. These data are shown in Figure 14.

Figure 14: Predicted daily maximum 8-hour CO concentrations from Airport Operations in 2030



The data shown in Figure 14 indicates that the predicted CO levels are higher at Kemps Creek, Bringelly, Rossmore and Badgerys Creek. All predicted CO concentrations are well below the NEPM standard of 10 mg/m^3. The data from all locations have been used in the calculation of risk from CO for the proposed Stage 1 airport operations.

5.6 Risk Characterisation

The results of epidemiological studies have shown that a wide range of health effects are associated with exposure to CO. Recent Australian studies (NEPC, 2012; EPHC 2006) have found associations between CO levels currently experienced in Australian cities and the following health outcomes:

- increases in daily mortality
- increases in hospital admissions
    - cardiac disease
    - cardiovascular disease

These health outcomes have been assessed in this health risk assessment for the relevant age groups.

No studies investigating the long term effects of exposure to CO on health have been identified.

The groups that have been identified as being susceptible to the effects of CO are:

- the elderly
- people with existing cardiovascular disease

- low socioeconomic groups
- foetus

The exposure-response functions in Table 20 have been taken from Australian studies and in particular two multicity meta-analyses (Simpson et al., 2005; EPHC, 2006).

No exposure-response functions for long-term exposure to CO have been identified.

Table 20: Exposure response functions for CO selected health outcomes

Outcome	Averaging Period	Exposure Response Function per 1 mg/m³ increase in CO
Daily all-cause mortality (non-accidental) all ages	8 hours	0.001
Hospital admissions cardiac disease 65+ years	8 hours	0.003
Hospital admissions cardiovascular disease 65+ years	8 hours	0.0014

The number of attributable cases has been calculated using the following equation:

Number of attributable cases = exposure response function(increase in health outcome) per 1mg/m^3 increase in CO x CO concentration x baseline health incidence rate/ 100,000 population x actual population

The number of cases for each outcome was calculated for the population in each of the potentially affected suburbs using the data from the worst impacted residential receptor in each area. The number of cases for each day of the year were calculated and then summed to give the annual total. Table 21 shows the results for each study population for CO.

Table 21: Predicted Number of Attributable Cases due to Airport Operations in 2030

	Daily Mortality all causes all ages	Hospital Admissions Cardiac Disease 65+ years	Hospital Admissions Cardiovascular Disease 65+ years
Badgerys Creek	0.0002	0.0006	0.0008
Bringelly	0.0008	0.002	0.002
Greendale	0.00004	0.0001	0.0002
Luddenham	0.0002	0.0005	0.0007
Kemps Creek	0.001	0.003	0.005
Mulgoa	0.0002	0.0005	0.0008
Wallacia	0.0002	0.0004	0.0005
Rossmore	0.0009	0.003	0.004
Mount Vernon	0.0002	0.0005	0.0007

The results shown in Table 21 indicate that the predicted health effects attributable to CO arising from the Stage 1 airport operation are very low. The highest risk is for hospital admissions for cardiovascular disease in people 65 years of age and older with a maximum of an additional 5 hospital admissions in one thousand years due to the emissions from the operations in 2030. This risk is negligible.

5.7 Health Effects of Air Toxics

A number of air toxics are emitted from airport operations. As part of the local air quality assessment, air dispersion modelling has been conducted for benzene, toluene, xylenes and formaldehyde. The most significant potential health risk is cancer from exposure to benzene. The predicted data for benzene have been used in the risk assessment for the airport operations in 2030.

5.7.1 Literature Review of the Health Effects of Benzene

Acute (short-term) inhalation exposure of humans to benzene may cause drowsiness, dizziness, headaches, as well as eye, skin, and respiratory tract irritation, and, at high levels, unconsciousness. Chronic (long-term) inhalation exposure has caused various disorders in the blood, including reduced numbers of red blood cells and aplastic anaemia, in occupational settings. Reproductive effects have been reported for women exposed by inhalation to high levels, and adverse effects on the developing foetus have been observed in animal tests. Increased incidence of leukaemia (cancer of the tissues that form white blood cells) have been observed in humans occupationally exposed to benzene.

Benzene is a well-established cause of cancer in humans. The International Agency for Research on Cancer has classified benzene as carcinogenic to humans (Group 1). Benzene causes acute myeloid leukaemia (acute non-lymphocytic leukaemia), and there is limited evidence that benzene may also cause acute and chronic lymphocytic leukaemia, non-Hodgkin's lymphoma and multiple myeloma.

Individuals who have experienced benzene poisoning requiring treatment show a substantially increased risk of mortality from leukaemia. Benzene is a genotoxic carcinogen and does not have a threshold for effect.

Chronic inhalation of certain levels of benzene causes disorders in the blood in humans. Benzene specifically affects bone marrow (the tissues that produce blood cells). Aplastic anaemia (a risk factor for acute non-lymphocytic leukaemia), excessive bleeding, and damage to the immune system (by changes in blood levels of antibodies and loss of white blood cells) may develop. In animals, chronic inhalation and oral exposure to benzene produces the same effects as seen in humans.

5.7.2 Exposure Assessment

From the review of the health effects of the benzene presented above, the critical health endpoint for evaluation is cancer. To enable the potential increased risk of cancer arising from the airport operations to be evaluated, annual average concentrations of benzene have been modelled as part of the Air Quality Assessment (Pacific Environment, 2015). The maximum concentration predicted at any location was 0.1 µg/m^3. This value has been used to calculate the maximum cancer risk from benzene in the surrounding area.

5.7.3 Risk Characterisation Benzene

The lifetime (assumed to be 70 years) increase in cancer risk has been calculated for the inhalation pathway. International agencies have published unit risk factors (URF) for benzene. The URF is an estimate of the increase in risk with exposure to 1 µg/m^3 of the pollutant over a lifetime. To calculate the lifetime cancer risk associated with the concentrations of benzene predicted to arise from emissions from the airport operations the following equation has been used:

Increase in lifetime cancer risk = annual average concentration x unit risk factor (URF)

A review of the available unit risk factors has been undertaken. The derivation of these factors, like any standard or guideline, is based on a range of assumptions and key information on the concentrations at which these effects can be observed. To enable the risk from each of these substances to be compared, it is important that as far as practicable, all URFs are obtained from the same source and that the derivation of these values is well documented. Based on the review undertaken, the URF from the Californian EPA Office of Environmental Health Hazard Assessment (OEHHA) has been used in this study. The URFs used to calculate the increased risk of cancer in the HRA is 2.9 x 10^{-5}.

Combining the maximum annual average concentrations of 0.1 µg/m^3 and the unit risk factor using the equation shown above the increase in cancer risk has been calculated. The maximum predicted cancer risk is 2.9 x 10^{-6}.

It is generally accepted by regulatory agencies that an increase in risk between 1 x 10^{-6} (1 in a million) and 1x10^{-5} (1 in 100,000) is considered to be a low risk and within acceptable criteria. The maximum predicted cancer risk from exposure to benzene from the airport operation in 2030 is within this range. Therefore the emissions from the proposed Stage 1 development of the airport would pose a very small increase in cancer risk which is within the acceptable levels established by national and international agencies.

5.8 Diesel

Diesel emissions associated with the proposed Western Sydney airport would arise from machinery used during construction activities as well as truck movements and diesel powered equipment used on site during operation. In recent years, there has been increased community concern about the health effects of diesel emissions. The local air quality assessment has modelled diesel emissions from the construction activities for the airport development.

5.8.1 Literature Review of the Health Effects of Diesel Emissions

Exposure to diesel exhaust can have immediate health effects. Diesel exhaust can irritate the eyes, nose, throat and lungs, and it can cause coughs, headaches, light headedness and nausea. In studies with human volunteers, diesel exhaust particles made people with allergies more susceptible to the materials to which they are allergic, such as dust and pollen. Exposure to diesel exhaust also causes inflammation in the lungs, which may aggravate chronic respiratory symptoms and increase the frequency or intensity of asthma attacks.

In experimental studies, healthy subjects have shown increased symptoms of irritation and compromised pulmonary function after short-term exposure to diesel exhaust. Additional studies have shown that diesel exhaust particles influence localised immunological components involved with allergic reactions. There have also been cases of newly developed asthma reported in workers exposed to diesel exhaust.

The inhalation or direct application of diesel particles into the respiratory tract of animals in acute and sub chronic studies induced inflammatory airway changes, lung function changes, and increased susceptibility of exposed animals to lung infection. The morphological effects observed in the lungs of animals in chronic inhalation exposures are mainly related to chronic inflammatory responses. Animal data indicate that chronic respiratory disease can result from long-term exposure to diesel exhaust. In rats, laboratory studies have shown that exposure to diesel exhaust can decrease resistance to infection and increase chronic inflammation. Rats, mice, rabbits, guinea pigs, and other primates all exhibit significant adverse pulmonary non-carcinogenic effects from long-term exposures to diesel exhaust.

Diesel exhaust and many individual substances contained in it (including arsenic, benzene, formaldehyde and nickel) have the potential to contribute to mutations in cells that can lead to cancer. A meta-analysis of 30 studies showed that occupational exposure to diesel exhaust is associated with an increased risk of lung cancer. Pooled relative risk estimates from 30 studies clearly reflect the existence of a positive relationship between diesel exhaust and lung cancer in a variety of diesel-exposed occupations. Based upon a review of these epidemiological studies conducted by OEHHA and the meta-analysis, it was concluded that these epidemiological studies provide evidence consistent with a causal relationship between occupational diesel exhaust exposure and lung cancer. The majority of these studies have reported elevated estimates of relative risk for lung cancer, many of which are statistically significant. The WHO and OEHHA have classified diesel particles and diesel exhaust as a known human carcinogen.

5.8.2 Exposure Assessment

The carcinogenic effects of diesel are associated primarily with particle fraction of the diesel. The unit risk factors for diesel are for diesel particles. Air dispersion modelling conducted by Pacific Environment (2015) has included modelling of diesel emissions from the proposed airport. The annual average diesel PM concentrations are shown in Table 22.

Table 22: Annual Average Diesel PM Concentrations

Location	Annual Average Diesel PM (µg/m³)
Bringelly	0.000064
Luddenham	0.0004
Greendale	0.0004
Kemps Creek	0.0002
Mulgoa	0.0001
Wallacia	0.0002
Badgerys Creek	0.0008
Rossmore	0.0001
Mount Vernon	0.0002

These concentrations represent the highest predicted concentrations within the surrounding suburbs. These values have been used in the calculation of the cancer risk associated with diesel emissions from the operation of the airport in 2030.

5.8.3 Risk Characterisation

As with the risk characterisation for the air toxics, the URF from OEHHA has been used in the assessment of the increase in cancer risk associated with diesel particles from the operations of the airport at 2030. The URF from OEHHA for diesel particles is $3x10^{-4}$ per 1 µg/m³ increase in diesel particles. The resultant cancer risk is shown in Table 23:

Table 23: Predicted Increase in Cancer Risk attributable to diesel particles

Location	Lifetime Excess Cancer Risk
Bringelly	1.3×10^{-6}
Luddenham	8.4×10^{-6}
Greendale	8.4×10^{-6}
Kemps Creek	4.4×10^{-6}
Mulgoa	2.3×10^{-6}
Wallacia	3.2×10^{-6}
Badgerys Creek	1.6×10^{-5}
Rossmore	2.7×10^{-6}
Mount Vernon	4.2×10^{-6}

The risk estimates shown in Table 22 fall within the acceptable level for risk generally adopted by international agencies. International agencies have generally adopted risk levels of being between 1 in a million and 1 in 100,000 as being within an acceptable risk range.

5.9 Regional Air Quality – Ozone

A regional air quality assessment has been conducted to assess the impact of the airport operations on ozone levels in Sydney (Ramboll – Environ, 2015). Ozone is one of the key pollutants of concern in the Sydney region with exceedances of the NEPM standards observed each year.

The approach to the regional air quality assessment differs from that adopted for the local air quality assessment. The NSW EPA has established guidelines for modelling of ozone which focuses on identifying peak ozone periods with potential exceedances of the air quality standards. The regional modelling has been conducted in accordance with these guidelines. The results of the regional air quality assessment are based on a small selection of days when exceedances of the standards are predicted and when there is good correlation between the model outputs and existing monitoring data obtained from NSW EPA monitoring stations.

Given that there is only a limited ozone prediction dataset available to assess the potential risk associated with exposure to changes in ozone levels across Sydney, a full risk characterisation was not possible. This would require predictions of ozone concentrations for every day of the year as has been done with the other pollutants. A different approach has therefore been necessary which focusses on the increase in risk due to changes in ozone levels only on the peak ozone days that have been assessed and where exceedances are predicted. This necessarily constrained the assessment of the potential risk posed by changes in ozone concentrations and could under estimate or overestimate the results.

5.9.1 Literature Review of the Health Effects of Ozone

Ozone is a secondary pollutant and is formed from precursors such as oxides of nitrogen and VOCs. Ambient monitoring shows that there is significant year-to-year variability in the ozone levels observed in

the Sydney region. Ozone levels are influenced by meteorology and seasonality (i.e. warmer seasons, cloudless skies, stable atmosphere) and bushfires.

The main health effects associated with exposure to ozone are associated with effects on the respiratory tract. The mechanism by which ozone affects the respiratory tract includes the formation of secondary oxidation products in the lung, activation of neural reflexes, initiation of inflammation, alterations of epithelial barrier function, sensitization of bronchial smooth muscle, changes in immunity and airway remodelling. Systemic inflammation and oxidative stress, may be critical to the effects of ozone on the cardiovascular system that have been observed in some studies.

The REVIHAAP study (WHO, 2013) found that there are both long-term and short-term effects of ozone on health. Studies reviewed by WHO have shown that long-term exposure to ozone has an impact on people with existing disease, in particular people with chronic obstructive pulmonary disease (COPD), diabetes, congestive heart failure and myocardial infarction. Long term exposure to ozone has also been associated with an increase in asthma incidence, asthma severity, hospital care for asthma and lung function growth. In regards to long-term exposures, there was new epidemiological evidence and experimental animal studies showing inflammatory responses, lung damage and persistent structural airway muscle tissue changes early in life indicating that there were long-term effects of exposure to ozone. The findings were supported by the USEPA (2013). No dose-response relationships are available to quantify the long-term effects of ozone.

Short-term effects associated with daily maximum one hour and eight-hour ozone concentrations include all cause, cardiovascular and respiratory mortality as well as cardiovascular and respiratory hospital admissions (WHO, 2013). In the 2006 review of the ozone standards, the USEPA concluded that there was clear, consistent evidence of a causal relationship between short-term exposure to ozone and respiratory health effects (USEPA, 2006). This finding was supported by the coherence of effects across a range of epidemiological, controlled human exposure and toxicological studies. These findings indicated that the effects of short-term exposure to ozone can impact a range of respiratory health endpoints ranging from respiratory tract inflammation to respiratory-related emergency department visits and hospital admissions.

There is strong evidence that short-term ozone exposures induced or were associated with statistically significant declines in lung function (USEPA (2006). An equally strong body of evidence from controlled human exposure and toxicological studies demonstrated that ozone induced inflammatory responses, increased epithelial permeability and airway hyperresponsiveness. These findings supported the findings of epidemiological studies which showed that short-term increases in ozone concentrations were consistently associated with increases in respiratory symptoms and asthma medication use in children with asthma, respiratory-related hospital admissions and asthma-related emergency department visits.

In the 2013 review, the USEPA concluded that more recent studies built on the findings of the previous review and strengthened the evidence that short-term exposure to ozone is causally associated with respiratory health effects. Recent controlled human exposure studies have shown that ozone levels as low as 60-70ppb are associated with statistically significant group mean decreases in pulmonary function in young healthy adults. These results are supported by the findings of epidemiological studies that provide strong evidence of associations between ozone exposure and respiratory hospital admissions and emergency department visits across the US, Canada and Europe. Several multicity studies and multi-continent studies reported associations between short-term increases in ambient ozone concentrations and increases in respiratory mortality. A large body of individual level epidemiological panel studies have demonstrated associations between exposure to ozone and respiratory symptoms in children with asthma. The findings of these studies are supported by recent studies that found ozone-associated increases in indicators of airway inflammation and oxidative stress in children with asthma (USEPA, 2013).

The toxicological data supports the findings of epidemiological studies and has strengthened in recent years. The toxicological studies show evidence of chronic injury and long-term structural change to the airways of animals exposed to prolonged periods of ozone as well as ozone and allergens combined. The strongest evidence for a relationship between long-term exposure to ozone and respiratory morbidity comes from studies that demonstrate long-term exposures to ozone are associated with new onset asthma in children as well as increases in respiratory symptom effects in children with asthma.

Studies that have investigated the presence of a threshold for the effects of ozone have found no evidence of a threshold for long-term effects, in particular mortality. Controlled human exposure studies show that ozone at 60ppb causes impaired lung function and inflammation of the airways in healthy adults. It is expected that effects would occur at lower levels in susceptible groups, however controlled exposure studies cannot include these groups as only healthy volunteers or people with mild disease can be included. Studies conducted at children's summer camps have seen adverse effects at lower concentrations of ozone than those observed in controlled human exposure studies. This is thought to reflect the response of susceptible groups in the general population. The multi-city studies on mortality and hospital admission outcomes have shown no evidence for a threshold for adverse effects. The WHO concluded that if a threshold does occur, that it is below 45ppb one hour maximum ozone (WHO, 2013).

In 2008, the WHO published a report on the long-range transport of air pollution. The study focused on the health effects associated with ozone exposures related to long-term transport of ozone across Europe (WHO, 2008). The WHO identified that in terms of short-term exposures to ozone, that the recent epidemiological studies had strengthened the evidence that daily exposures to ozone increased mortality and respiratory morbidity rates. Studies on pulmonary function, lung inflammation, lung permeability, respiratory symptoms, increased medication usage, morbidity and mortality, indicated that ozone appears to have an effect independent of other pollutants including particulate matter. The WHO concluded that evidence that ozone may act independently of other pollutants is supported by the results of controlled human exposure studies and toxicological studies showing the potential of ozone to cause adverse health effects especially vulnerable people.

5.9.2 Exposure Assessment

Peak daily ozone concentrations have been predicted for a number of days for airport operations in both 2030 and 2063, as well as the largest change in ozone concentration. The data reported in the regional air quality assessment is shown in Table 24. Peak daily ozone levels for the Liverpool air monitoring station is also shown for the 2008/2009 summer period. The choice of 2008/2009 as the base year was made in the Regional Air Quality Assessment (Ramboll – Environ, 2015) as this is the ozone season with the highest number of exceedances in the previous 10 years.

Table 24: Maximum 1-hour ozone concentrations for Sydney region

Date	2009 (Liverpool data)	2030	2063
6/1/2009	86	149.1	149.3
7/1/2009	106	129.8	130.3
14/1/2009	95	106.6	106.7
29/1/2009	-	124.1	124.1
30/1/2009	-	107.4	107.4
31/1/2009	75	109.4	109.4
4/2/2009	-	103.8	103.8
5/2/2009	-	119.6	119.6
6/2/2009	-	112.5	112.5
7/2/2009	92	133.7	133.7
8/2/2009	91	148.6	148.8
20/2/2009	51	98.3	98.5

The data shown in Table 24 is the peak daily 1-hour ozone concentrations that were predicted across the modelling grid. The location at which peak concentrations were predicted are identified in contour plots presented in the regional air quality assessment, however results are presented on a regional scale. Decreases in daily maximum ozone occur only in the vicinity of the airport and are attributable to ozone suppression by fresh NOx emissions. Increases in ozone occur downwind of the airport which, on most days, is to the south and southwest.

For the purposes of the risk assessment, the ozone data from the Liverpool EPA monitoring station has been used as the background dataset as this was the most complete dataset for 2009 and that the maximum concentrations did not occur adjacent to the airport site.

5.9.3 Risk Characterisation

The review of the health effects of ozone has identified that the health endpoints that have reliable exposure-response functions are short-term increases in daily mortality from all causes (non-accidental), respiratory and cardiovascular disease as well as emergency department visits for asthma in children aged 1-14 years. The exposure response functions used in the risk characterisation are shown in Table 25 and have been taken from EPHC, 2005 and Jalaludin et al., 2008.

Table 25: Exposure Response Functions

Health Outcome	Exposure Response Function (% increase per 1ppb increase in daily 1-hour max ozone)
Daily all-cause mortality (all ages)	0.0014
Daily cardiovascular mortality (all ages)	0.0021
Daily respiratory mortality (all ages)	0.0023
Emergency Department Attendances Asthma 1-14 years	0.001

The increase in risk per 100,000 population has been calculated using the following equation:

Increase in Risk (per 100,000) = Daily 1-hour max ozone concentration (ppb) x exposure response function (% increase in health outcome per 1ppb increase in ozone concentration) x baseline incidence rate/100,000 population.

The resulting risk per 100,000 population for all days assessed in the Regional Air Quality Assessment is shown in Table 26 for the Base Year 2009, Airport Operations in 2030 and 2063.

Table 26: Attributable Risk from Ozone on Peak Ozone Days

Outcome	Base Year 2009	2030 Airport Operations	2063 Airport Operations
Daily All-Cause Mortality (all ages)	1.3×10^{-5}	2×10^{-5}	2×10^{-5}
Daily Cardiovascular Mortality (all ages)	7×10^{-6}	1×10^{-5}	1×10^{-5}
Daily Respiratory Mortality (all ages)	2×10^{-6}	3×10^{-6}	3×10^{-6}
Emergency Department Attendances 1-14 years	1.8×10^{-5}	2.7×10^{-5}	2.7×10^{-5}

For the base year, the resulting risk for the outcomes assessed is between 2 in a million (respiratory mortality) and 1.8 in 100,000. For the 2030 operations, the increase in risk ranges from 1 in 1 million for respiratory mortality to 9 in 1 million for emergency department attendances for asthma in children. There is no difference between the maximum risk between 2030 and 2063. This is because the peak predicted daily ozone is largely unchanged between base case (all other emissions source) and the airport case and for both 2030 and 2063 the base case is the same. The largest predicted ozone concentration changes from the airport occur in a different location to the predicted daily peak ozone concentrations.

There is general agreement by international agencies including the WHO and USEPA that acceptable risk levels fall between 1 in a million and 1 in 100,000. The increases in risk for the days assessed in the regional air quality assessment fall within these limits.

As indicated in the introduction, there are limitations to this assessment. The regional air quality assessment has been undertaken in accordance with NSW EPA guidelines and is limited to a small number of days when exceedances of ozone standards are likely. This approach does not provide a full dataset that would typically enable a full quantitative risk assessment to be conducted using a full year of data. Therefore the risk characterisation is limited to the number of exceedance days.

The location of the maximum ozone concentrations is not identified in the Regional Air Quality Assessment, therefore the risk analysis has been conducted using the Liverpool air quality data as the baseline dataset against which the increase in risk due to predicted ozone levels has been assessed. This may lead to an overestimate or underestimate of the risk. As no spatial data was available, the difference in risk at different locations was not possible to infer. There may be variability in the risk estimates based on variability in predicted ozone levels across the Sydney airshed.

5.10 Summary

The health risk assessment for the emissions to air from the operation of the airport in 2030 and 2063 as well as the construction activities has shown that the predicted highest risk arises from exposure to PM_{10}, $PM_{2.5}$ and NO_2. The predicted levels of these pollutants in 2030 did not exceed the NEPM standards. IN 2063 some exceedances of the NEPM standards were predicted (Pacific-Environment, 2015).

The results of the HRA show that the predicted risk from exposure to NO_2 from the airport operations in both 2030 and 2063 is higher than that predicted for PM_{10} and $PM_{2.5}$. Based on the modelling data provided for the HRA, the highest risk is for long-term mortality in people over 30 years of age with 6 additional deaths per 10 years predicted for 2063. The highest risk predicted for 2030 is 5 deaths in 10 years. As outlined earlier, evidence provided by NSW Health to a Parliamentary Inquiry into health effects of pollution showed that in 2006 it was estimated that between 600 and 1400 deaths per year were attributed to air pollution in the Sydney basin (NSW Parliament 2006). Based on these figures, the health impact associated with the emissions from the airport are expected to represent a small increase in current rates of long-term mortality associated with air pollution.

Nevertheless, these results suggest that mitigation strategies should be implemented to reduce community exposure to these pollutants. A range of mitigation measures are discussed in the local air quality report (Pacific-Environment, 2015.)The greatest impact is for Bringelly, Rossmore and Kemps Creek.

The increased cancer risk from benzene is low. It is generally accepted by regulatory agencies that an increase in risk between 1×10^{-6} (1 in a million) and 1×10^{-5} (1 in 100,000) is considered to be a low risk and within acceptable criteria. The maximum predicted cancer risk is well below these criteria. Therefore the predicted emissions of benzene from the Stage 1 operation of the airport pose a very small increase in cancer risk which is well within the acceptable levels established by national and international agencies.

For diesel emissions, the predicted cancer risk is also low and within acceptable risk criteria established by international agencies.

A risk assessment based on a small number of days with predicted ozone exceedance has been conducted to provide an indication of the potential risk associated with increases in ozone concentrations due to the airport operations in 2030 and 2063.

The risk assessment has shown that for these days, there are small increases in risk of daily mortality from all causes, cardiovascular and respiratory causes as well as emergency department visits for asthma in children. The risk estimates are within acceptable risk levels generally accepted by international agencies. There is no difference in risk associated with the maximum predicted levels between 2030 and 2063 airport operations. This is because the peak predicted daily ozone is largely unchanged between base case (all other emissions source) and the airport case and for both 2030 and 2063 the base case is the same. The largest predicted ozone concentration changes from the airport occur in a different location to the predicted daily peak ozone concentrations.

6 HEALTH RISK ASSESSMENT – NOISE

The health risk assessment for noise has been undertaken following the enHealth Guidance *Health Effects of Environmental Noise other than Hearing Loss* (enHealth, 2004) and WHO guidelines (2009; 1999). EnHealth are currently updating their environmental noise guidelines. Discussions with NSW Health and the Local Health Districts affected by the airport identified sleep disturbance – in particular awakenings, cardiovascular disease and cognitive impairment as the issues of main concern with respect to the development of Western Sydney Airport. The HRA has focussed on these issues.

6.1 Literature Review on Health Effects related to Noise

In recent years, evidence has accumulated regarding the health effects of environmental noise. Epidemiological studies have found that cardiovascular diseases are consistently associated with exposure to environmental noise. The WHO has released three reports on the health effects of environmental noise: *Guidelines for Community Noise* (1999), Night Noise Guidelines (2009) and the *Burden of Disease from Environmental Noise* (2011). In these documents, the main health effects associated with environmental noise are:

- Annoyance
- Sleep disturbance
- Cardiovascular disease
- Cognitive impairment

An increasing body of literature has shown traffic noise to have adverse short- and long-term health effects (Babisch 2006; Berglund et al. 1999; Bluhm et al. 2007; Stansfeld et al. 2000, 2005). One of the suggested mechanisms by which noise affects nonauditory health is through indirect or direct activation of the sympathetic nervous system and endocrine systems (Ising and Kruppa 2004; Stansfeld and Matheson 2003), resulting in autonomic reactions, including increased blood pressure, heart rate, and arrhythmia (Berglund et al. 1999). Therefore, research has focused on the impact of transportation noise on cardiovascular health. There is suggestive evidence that transportation noise, including aircraft noise, exposure is associated with an increase in ischemic heart disease (Babisch 2006). Associations between transportation noise and hypertension have been inconsistent (Babisch et al. 2006; Chang et al. 2009; Jarup et al. 2008; van Kempen et al. 2006).

6.1.1 Annoyance

Annoyance is the most prevalent community response in a population exposed to environmental noise. It is not in itself considered to be a health effect (WHO, 2009; enHealth, 2004). The term annoyance is used to describe negative reactions to noise such as disturbance, irritation, dissatisfaction and nuisance (Guski, 1999). Annoyance can also be accompanied by stress-related symptoms, leading to changes in heart rate and blood pressure. Acoustic factors, such as the noise source and sound level, account for only a small to moderate amount of annoyance responses: other factors such as the fear associated with the noise source, interference with activities, ability to cope, noise sensitivity, expectations, anger, attitudes to the source – both positive or negative, and beliefs about whether noise could be reduced by those responsible influence annoyance responses (WHO, 2000).

Exposure to aircraft noise at 60 dB Lden is estimated to be associated with 38% of the population reporting being "annoyed" and 17% being "highly annoyed" (EC, 2002). Exposure to aircraft noise at 65 dB Lden is estimated to be associated with 48% of the population reporting being "annoyed" and 26% being "highly annoyed" (EC, 2002). However, in recent years, several studies have suggested that aircraft noise annoyance around major airports in Europe has increased (Babisch et al., 2009; Janssen et al., 2011; Schreckenberg et al., 2010) indicating that the percentage of the population reporting

being "annoyed" or "highly annoyed" at each noise exposure level may have increased since these figures were put forward by the European Commission in 2002 (EC, 2002).

Annoyance responses can also increase in relation to a change in airport operations. A study around Zurich airport found that residents who experienced a significant increase in aircraft noise exposure due to an increase in early morning and late evening flight operations had a pronounced over-reaction of annoyance i.e. the annoyance reaction was greater than that which would be predicted by the level of noise exposure (Brink et al., 2008).

Children also report annoyance responses, although it is not known at what age children begin to exhibit annoyance responses. The RANCH study found that children aged 9-11 years of age living near London Heathrow, Amsterdam Schiphol, and Madrid Barajas airports, reported annoyance for aircraft noise exposure at school and at home (van Kempen et al., 2009). For school exposure the percentage of "highly annoyed" children increased from about 5.1% at 50 dB LAeq 16 hour, to 12.1% at 60 dB LAeq 16 hour.

The results for daytime noise impacts on learning and cognitive development in children are shown in Table 34. The results show that for both the 2030 and 2063 assessment scenarios, all predicted noise levels are below the WHO guideline criteria of 55 dB and that, with the exception of one scenario for the longer term operation of the airport in 2063, all predicted daytime levels at sensitive receivers are below 50 dB. No quantitative assessment of annoyance has been conducted in this HRA.

6.1.2 Sleep Disturbance

Possible effects of noise on sleep are generally grouped into three categories:

1. The immediate effects of noise on sleep (sleep disturbance and physiological effects)
2. The secondary effects of sleep disturbances (morning after effects)
3. Long term health effects.

Sleep disturbance is defined as any deviation, measurable or subjectively perceived, from an individual's habitual or desired sleep behaviour. This may include awakenings, sleep quality, medication use to control sleep, total sleep time, time spent in slow wave sleep (see Table 1), arousals and time spent in rapid eye movement sleep (WHO, 2009).

The WHO estimated sleep disturbance to be the most adverse non-auditory effect of environmental noise exposure (Basner et al., 2014; WHO, 2011). Undisturbed sleep of a sufficient number of hours is needed for alertness and performance during the day, for quality of life, and for health (Basner et al., 2014). Humans exposed to sound whilst asleep still have physiological reactions to the noise which do not adapt over time including changes in breathing, body movements, heart rate, as well as awakenings (Basner et al., 2014). The elderly, shift-workers, children and those with poor health are thought to be at risk for sleep disturbance by noise (Muzet, 2007).

The effect of night-time aircraft noise exposure has been explored for a range of sleep outcomes ranging from subjective self-reported sleep disturbance and perceived sleep quality, to more objective measures of interference with ability to fall asleep, shortened sleep duration, awakenings, and increased bodily movements as assessed by polysomnography (Michaud et al., 2007). Most evidence comes from studies of self-reported sleep disturbance. However, self-reported sleep disturbance outcomes are vulnerable to bias, as such measures are likely to be influenced by noise annoyance and other demographic factors (Clark and Stansfeld, 2011).

Reviews have concluded that there is evidence for an effect of night-time aircraft noise exposure on sleep disturbance from community based studies (Hume et al., 2012; Miedema & Vos, 2007). However, some reviews have concluded that the evidence is contradictory and inconclusive (Jones, 2009; Michaud et al., 2007), which might be explained by methodological differences between studies of noise effects on sleep disturbance. A meta-analysis of 24 studies, including nearly 23,000 individuals

exposed to night-time noise levels ranging from 45-65 dBA, found that aircraft noise was associated with greater self-reported sleep disturbance than road traffic noise (Miedema and Vos, 2007). However, another study, whilst confirming that aircraft noise was associated with greater self-reported sleep disturbance than road traffic noise, found that when [1]polysomnography measures of sleep disturbance were analysed, that road traffic noise was associated with greater disturbance than aircraft noise (Basner et al., 2011).

Polysomnography enables the assessment of noise effects on different stages of the sleep cycle. The average sleep cycle lasts between 90 to 110 minutes, and an individual experiences between four to six sleep cycles per night (Michaud et al., 2007). Table 27 summarises the duration and characteristics of each stage of the sleep cycle (Clark and Stansfeld, 2011) from wake, through non-rapid eye movement (NREM) stages 1 to 4, and rapid eye movement (REM) sleep. It is usual for people to move between NREM sleep stages several times before undergoing REM sleep. Slow-wave sleep (NREM stages 3 and 4) occurs more frequently in the first half of the night, and REM sleep propensity is greater in the second half of the night. Sleep disturbance is indicated by less stage 3, stage 4 and REM sleep, and by more wake and stage 1 sleep, as well as more frequent changes in sleep stage (Basner and Siebert, 2010).

Table 27: Stages of Sleep (taken from Clark and Stansfeld 2012)

Wake	
Non-rapid Eye Movement (NREM)	
Stage 1	Light stage of sleep Lasts 5-10 minutes Bridge between wakefulness and sleep
Stage 2	Light stage of sleep Lasts around 20 minutes Brain waves of increased frequency Increased heart rate variability
Stage 3	Transition to deeper stages of sleep Increased amount of delta waves of lower frequency
Stage 4	Deepest stage of sleep Characterised by a greater number of delta waves
Rapid Eye Movement (REM) sleep	Typically starts 70-90 minutes after falling asleep Characterised by rapid eye movements Increases in brain activity Greater variability in respiration rate, blood pressure

[1] Polysomnography records biophysiological changes that occur during sleep, including brain waves using electroencephalography (EEG), eye movements using electroculography (EOG), muscle activity using electromyography (EMG), and heart rhythm using electrocardiography (ECG).

	and heart rate

There is evidence that aircraft noise influences the time spent in different sleep stages, with aircraft noise reducing slow-wave sleep (NREM Stage 4) and REM sleep and increasing NREM Stages 1, 2 & 3 (Basner et al., 2008; Swift, 2010). This evidence, taken with the increase in REM sleep in the later stages of the night might have implications for early morning (04.00-06.30 hours) flight operations at airports.

A laboratory study compared the potential effects of changes in the night-time curfew at Frankfurt airport on sleep disruption (Basner and Siebert, 2010), using polysomnography on 128 subjects over 13 nights. Three different operational scenarios were compared: scenario 1 was based on 2005 air traffic at Frankfurt airport which included night flights; scenario 2 was as scenario 1 but cancelled flights between 23.00-05.00 hours; scenario 3 was as scenario 1 but with flights between 23.00-05.00 hours rescheduled to the day-time and evening periods. The study found that compared to the night without a curfew on night flights (scenario 1), small improvements were observed in sleep structure for the nights with curfew, even when the flights were rescheduled to periods before and after the curfew period. However, the change in the amount of time spent in the different sleep stages for the different scenarios was small, which might be explained by the small number of night-flights (on average 4 take-offs per hour) in the Frankfurt airport scenarios examined: larger effects may be observed for airports with a greater number of night-flights. The authors concluded that the benefits for sleep seen in the scenario involving rescheduling of flights rather than cancellation may be offset by the expected increase in air traffic during the late evening and early morning hours for those who go to bed before 22.30 or after 01.00 hours.

The WHO Europe Night Noise Guidelines (WHO, 2009) were based on expert-consensus that there was sufficient evidence that nocturnal environmental noise exposure was related to self-reported sleep disturbance and medication use, and that there was some evidence for effects of nocturnal noise exposure on high blood pressure (hypertension) and heart attacks. The WHO Europe Night Noise Guidelines state that the target for nocturnal noise exposure should be 40 dB Lnight, outside, which should protect the public as well as vulnerable groups such as the elderly, children, and the chronically ill from the effects of nocturnal noise exposure on health. The Night Noise Guidelines also recommend the level of 55 dB Lnight, outside, as an interim target for countries wishing to adopt a step-wise approach to the guidelines. It is worth noting that the 40 dB Lnight, outside guideline represents a very low level of noise exposure (equivalent to the noise level expected in a library).

There have been fewer studies on aircraft noise exposure and sleep in children (Stansfeld and Clark, 2015), even though children are a group thought to be vulnerable to the effects of sleep disturbance (Pirrera et al., 2010). Children sleep outside the typical hours used to denote night-time noise exposure around airports (e.g. Lnight is typically 23.00 hours to 07.00 hours), so exposures during the hours of the evening and morning, which would fall within day-time exposure metrics may also be relevant when considering sleep disturbance effects for children.

6.1.3 Cardiovascular Disease

In recent years, evidence that aircraft noise exposure leads to increased risk for poorer cardiovascular health has increased considerably. A recent review, suggested that risk for cardiovascular outcomes such as high blood pressure (hypertension), heart attack, and stroke, increases by 7 to 17% for a 10 dB increase in aircraft or road traffic noise exposure (Basner et al., 2014). A review of the evidence for children concluded that there were associations between aircraft noise and high blood pressure (Paunović et al., 2011), which may have implications for adult health (Stansfeld and Clark, 2015).

The HYENA study (HYpertension and Exposure to Noise near Airports) examined noise effects on the blood pressure (hypertension) of 4,861 people, aged 45-70 years, who had lived for over five years near seven major European airports including London Heathrow; Amsterdam Schiphol; Stockholm Arlanda

and Bromma; Berlin Tegel, Milan Malpensa; and Athens Eleftherios Venizelos (Jarup et al., 2008). High blood pressure was assessed via measurements and medication use. The HYENA study found that a 10 dB increase in aircraft noise at night (Lnight) was associated with a 14% increase in risk for high blood pressure but day-time aircraft noise (LAeq 16 hour) did not increase the risk for high blood pressure (Jarup et al., 2008). The HYENA study did not find an association between day-time aircraft noise and high blood pressure which might be because many residents work away from home during the day-time, leading to potential mis-classification of their day-time aircraft noise exposure. The HYENA study also found that a 10 dB increase in night-time aircraft noise was associated with a 34% increase in the use of medication for high blood pressure in the UK (Floud et al., 2011). The HYENA study is a high quality large-scale study of aircraft noise exposure effects on blood pressure, which includes a population sample around London Heathrow airport.

A further study conducted as part of the HYENA project demonstrated an association between noise and cardiovascular disease risk factors (Floud et al., 2013). The results are consistent with the hypothesis that noise exposure provokes a stress response causing a release of stress hormones, which in turn affect factors such as blood pressure and heart rate and thus cardiovascular disease risk. Night-time aircraft noise was statistically significantly associated with self-reported heart disease and stroke but was reduced and became non-significant after adjustment for confounders. However, there was a significant association for those who had lived for 20 years or more at their current address and aircraft noise. A statistically significant association (25 % increase in risk) was found between exposure to night-time aircraft noise and heart disease and stroke in people who had lived in the same home for 20 years or more, and this association was robust to adjustment for exposure to NO_2 air pollution.

A recent study around London Heathrow airport examined risks for hospital admission and mortality for stroke, coronary heart disease and cardiovascular disease for around 3.6 million people living near the airport (Hansell et al., 2013). Both day-time (LAeq 16 hour) and night-time (Lnight) aircraft noise exposure were related to increased risk for a cardiovascular hospital admission. Compared to those exposed to aircraft noise levels below 51 dB in the day-time LAeq, 16 hour, those exposed to aircraft noise levels over 63 dB LAeq, 16hour in the day-time had a 24% higher chance of a hospital admission for stroke; a 21% higher chance of a hospital admission for coronary heart disease; and a 14% higher chance of a hospital admission for cardiovascular disease. These estimates took into account age, sex, ethnicity, deprivation and lung cancer mortality as a proxy for smoking. These results were also not accounted for by air pollution, which was adjusted for in the analyses. Similar effects were also found between aircraft noise exposure and mortality for stroke, coronary heart disease, and cardiovascular disease. The study concluded that high levels of aircraft noise were associated with increased risks of stroke, coronary heart disease, and cardiovascular disease for both hospital admissions and mortality in areas near Heathrow airport.

Further longitudinal evidence for an association between aircraft noise exposure and mortality from heart attacks comes from a large-scale Swiss study of 4.6 million residents over 30 years of age (Huss et al., 2010). This study found that mortality from heart attacks increased with increasing level and duration of aircraft noise exposure (over 15 years), but there were no associations between aircraft noise exposure and other cardiovascular outcomes including stroke or circulatory disease. The lack of association between aircraft noise and stroke differs from the findings of the similar study conducted by Hansell et al., (2013) around Heathrow airport, which did find an association of aircraft noise on stroke mortality.

A multi-airport retrospective study of approximately 6 million older people residing near airports in the United States (Correia et al., 2013) found that averaged across all airports and using the 90th centile noise exposure metric, a zip code with 10 dB higher noise exposure had a 3.5% higher (95% confidence interval 0.2% to 7.0%) cardiovascular hospital admission rate, after controlling for covariates. Despite limitations related to potential misclassification of exposure, a statistically significant association between exposure to aircraft noise and risk of hospitalization for cardiovascular diseases among older

people living near airports was observed. The most recent meta-analysis of the field (Babisch, 2014) concluded that aircraft noise exposure was associated with increased risk for cardiovascular outcomes such as high blood pressure, heart attack and stroke.

It is biologically plausible that long-term exposure to environmental noise might influence cardiovascular health (Babisch, 2014). The proposed pathways between environmental noise exposure and cardiovascular diseases (Babisch, 2014) include increased stress associated with noise exposure that might cause physiological stress reactions in an individual, which in turn can lead to increases in established cardiovascular disease risk factors such as blood pressure, blood glucose concentrations, and blood lipids (blood fats). These risk factors lead to increased risk of high blood pressure (hypertension) and arteriosclerosis (e.g. narrowing of arteries due to fat deposits) and are related to serious events such as heart attacks and strokes (Babisch, 2014; Basner et al., 2014). The stress that triggers this pathway can operate directly via sleep disturbance or indirectly via interference with activities and annoyance.

To date, few studies have examined whether aircraft noise exposure influences metabolic risk factors for cardiovascular health, such as Type II diabetes, body mass index, and waist circumference. Such factors would lie on the proposed pathway between aircraft noise exposure and cardiovascular diseases. A recent study of long-term exposure to aircraft noise in Sweden found that exposure was associated with a larger waist circumference but less clearly with Type II diabetes and body mass index (Eriksson et al., 2014). Further studies are required to investigate these associations.

6.1.4 Children's Learning and Cognitive Development

Children may be particularly vulnerable to the effects of noise because they may have less cognitive capacity to understand environmental issues and anticipate stressors and they may lack appropriate coping strategies to deal with noise. Additionally, noise may interfere with learning at a critical developmental stage.

The impact of environmental noise on children's learning and memory has been known for many years. Epidemiological studies show effects of chronic noise exposure on tasks involving central processing and language, such as reading, comprehension, memory and attention. Experimental studies investigating acute (short-term) exposures have found similar effects. Exposure during critical periods of learning at school could potentially impair development and have a lifelong effect on educational attainment.

There are several ways in which aircraft noise could influence children's cognition: lost teaching time - as a teacher may have to stop teaching whilst noise events occur; teacher and pupil frustration; annoyance and stress responses; reduced morale; impaired attention; children might tune out the aircraft noise and over-generalise this response to other sounds in their environment missing out on information; and sleep disturbance from home exposure which might cause performance effects the next day (Stansfeld and Clark, 2015).

Many studies have found effects of aircraft noise exposure at school or at home on children's reading comprehension or memory skills (Evans and Hygge, 2007). The RANCH study (Road traffic and Aircraft Noise and children's Cognition and Health) of 2844 9-10 year old children from 89 schools around London Heathrow, Amsterdam Schiphol, and Madrid Barajas airports found that aircraft noise was associated with poorer reading comprehension and poorer recognition memory, after taking socioeconomic factors and road traffic noise into account (Stansfeld et al., 2005).

The exposure-response relationship between aircraft noise at school and reading comprehension from the RANCH study (Clark et al., 2006), showed that as aircraft noise exposure increased, performance on the reading test decreased. Reading began to fall below average at around 55 dB LAeq 16 hour at school. The development of cognitive skills such as reading and memory is important not only in terms

of educational achievement but also for subsequent life chances and adult health (Kuh and Ben-Shlomo, 2004). In the UK, reading age was delayed by up to 2 months for a 5 dB increase in aircraft noise exposure (Clark et al., 2006). The UK primary schools in the RANCH study ranged in aircraft noise exposure from 34 dB LAeq 16 hour to 68 dB LAeq 16 hour. The study found that a 20 dB difference in aircraft noise exposure between schools would result in an 8-month difference in reading age.

In the RANCH study, for primary school children, aircraft noise exposure at school and at home were very highly correlated: in the RANCH UK sample, this correlation was r=0.91 (Clark et al., 2006). Such a high correlation makes estimating the impact of aircraft noise exposure in both environments difficult. The RANCH study found that night-time aircraft noise at the child's home was also associated with impaired reading comprehension and recognition memory, but night-noise was not having an additional effect to that of day-time noise exposure on reading comprehension or recognition memory (Clark et al., 2006; Stansfeld et al., 2010). These findings suggest that indices of aircraft noise exposure in the day-time in the school environment should be sufficient to capture effects. Further analyses of the UK RANCH sample found that these associations for aircraft noise exposure remained after controlling for air pollution effects (Clark et al., 2012).

A further study investigating the effects of aircraft noise around Heathrow Airport in the home environments on children's cognition found a significant dose-response relationship between aircraft noise at home and performance on memory tests of immediate and/or delayed recall (Matsui et al., 2004). The study found no associations with other cognitive outcomes.

Two studies of interventions to reduce or remove aircraft noise exposure at school have been conducted. The longitudinal Munich Airport study (Hygge et al., 2002) found that prior to the relocation of the airport in Munich, high noise exposure was associated with poorer long-term memory and reading comprehension in children aged 10 years. Two years after the airport closed these cognitive impairments were no longer present, suggesting that the effects of aircraft noise on cognitive performance may be reversible if the noise stops. In the cohort of children living near the newly opened Munich airport impairments in memory and reading developed over the following two years.

A recent study of 6,000 schools exposed between the years 2000-2009 at the top 46 United States airports, (exposed to Day-Night-Average Sound Level of 55 dB or higher) found significant associations between aircraft noise and standardised tests of mathematics and reading, after taking demographic and school factors into account (Sharp et al., 2014). In a sub-sample of 119 schools, they found that the effect of aircraft noise on children's learning disappeared once the school had sound insulation installed.

Schools located near airports often also experience high levels of road traffic noise but it is important to note that aircraft noise exposure still influences children's learning, even if road traffic noise exposure is high. The results presented for the RANCH study are the association for aircraft noise exposure, after taking road traffic noise into account (Clark et al., 2006).

A study conducted by Haines et al.. (2002) studied a sample of approximately 11,000 11 year old children from 123 schools surrounding Heathrow Airport. The results of the study showed that chronic exposure to aircraft noise was significantly related to poorer reading and mathematics performance. However, after control for socioeconomic factors these associations were no longer statistically significant.

Children spend a considerable amount of time at school in the playground. Play is thought to be important for children's social, cognitive, emotional and physical development, as well as enabling relaxation between more formal teaching activities. The WHO (1999) established a community noise guideline of 55 dB for school playgrounds, during play, to protect against these effects. The WHO community noise guidelines that apply to schools and preschools are summarised in Table 28.

Table 28: WHO Community Noise Guidelines School Environments (WHO, 1999)

School Environment	Critical Health Effects	L_{Aeq} (dB(A))	Time base (hours)	L_{Amax} fast (dB)
School class rooms and preschools indoors	Speech intelligibility, disturbance of information extraction, message communication	35	During class	-
Preschool bedrooms indoors	Sleep disturbance	30	Sleeping time	45
School playground, outdoors	Annoyance (external source)	55	During play	-

6.1.5 Psychological health

It has been suggested that long-term noise exposure might influence psychological health. However, overall the evidence for aircraft noise exposure being linked to poorer well-being, lower quality of life, and psychological ill-health is not as strong or consistent as for other health outcomes, such as cardiovascular disease. A recent study of 2300 residents near Frankfurt airport found that annoyance but not aircraft noise levels per se (LAeq16 hour, Lnight, Lden) was associated with self-reported lower quality of life (Schreckenberg et al., 2010).

Several studies of children around London Heathrow airport have shown no effect of aircraft noise at school on children's psychological health or cortisol levels (Haines et al., 2001a; Haines et al., 2001b; Stansfeld et al., 2009): cortisol levels are known to be raised in children with depression. However, there may be a small effect of aircraft noise on hyperactivity symptoms. The West London Schools Study of 451 children around Heathrow airport, aged 8-11 years found higher rates of hyperactivity symptoms for children attending schools exposed to aircraft noise levels >63 dB LAeq 16 hour compared with <57 dB LAeq 16 hour (Haines et al., 2001a). A similar effect was observed in the RANCH study where a 10 dB LAeq 16 hour increase in aircraft noise exposure at school was associated with 0.13% increase in hyperactivity symptoms (Stansfeld et al., 2009). However, these increases in hyperactivity symptoms, whilst statistically significant, are extremely small and most likely not of clinical relevance. Aircraft noise exposure does not appear to be causing children to develop hyperactivity problems.

There have been fewer studies of aircraft noise effects on adult psychological health. The HYENA study, found that a 10 dB increase in day-time (LAeq 16 hour) noise exposure was associated with a 28% increase in anxiety medication use: similarly, a 10 dB increase in night-time (Lnight) aircraft noise was associated with a 27% increase in anxiety medication use. However, day-time and night-time aircraft noise exposure were not associated with sleep medication or anti-depressant medication use (Floud et al., 2011). Anxiety medication is prescribed for individuals experiencing levels of anxiety and worry that interfere with their ability to function effectively: they can also be prescribed for sleeping problems. A sub-study of the HYENA study found that salivary cortisol (a stress hormone which is higher in people with depression) was 34% higher for women exposed to aircraft noise > 60 dB LAeq 24 hour, compared to women exposed to less than 50 dB LAeq 24 hour (Selander et al., 2009). However, no association between aircraft noise and salivary cortisol was found for men.

6.2 Exposure Assessment

As part of the technical studies being completed for the EIS, an assessment of potential noise and vibration effects has been undertaken (Wilkinson-Murray, 2015 a and b). The exposure data for the noise HRA have been taken from these reports which include aircraft as well as ground operations noise sources.

A number of sensitive receptors were identified for use in the HRA. These are summarised in Table 29.

Table 29: Representative Sensitive Receptors

RECEPTOR	Receptor Number
RESIDENTIAL LOCATIONS	
Bringelly	1
Kemps Creek	4
Erskine Park	9
Kemps Creek 2	22
St Marys	26
Greendale	27
Silverdale	29
Rossmore	30
Horsley Park	32
Rooty Hill	150
Prospect	152
EDUCATIONAL	
Warragamba Preschool	43
Emmaus Catholic College, Kemps Creek	65
Horsley Park Public School	70
Luddenham Public School	73
Bringelly Public School	76
Mount Druitt Public School	142
St Marys South Public School	144
Bennett Road Public School, Colyton	145
Colyton High School	146
St Clair High School	147
Banks Public School, St Clair	148
Blackwell Public School, St Clair	149
Plumpton High School	151

The locations of these receptors are shown in Figure 15. It should be noted that although the schools have been identified primarily for assessment of the impacts of noise on child learning and cognitive development, they are also located in residential areas. Therefore the noise levels predicted at these locations may also be representative of the exposure to noise for the local community. As such, these locations have also been used in the assessment of sleep disturbance. Background noise levels that have been measured as part of the Noise and Vibration Assessment (Wilkinson-Murray, 2015 a and b) are shown in Table 30:

Table 30: Measured Background Noise Levels around Western Sydney Airport (source Wilkinson-Murray, 2015b)

Location	Measurement Duration	Rating Background Level (dBA)		
		Day (7am-6pm)	Evening (6pm-10pm)	Night (10pm-7am)
9 Harold Bentley Way, Glenmore Park	Monday 23/3/15 – Thursday 2/4/15	39	42	38
16 Park Avenue, Springwood	Wednesday 25/3/15 – Thursday 2/4/15	29	32	24
17 Blue Ridge Place, Orchard Hills	Monday 23/3/15 – Tuesday 31/3/15	34	38	36
25 Peter Pan Avenue, Wallacia	Monday 23/3/15 – Thursday 2/4/15	37	34	28
27 Dwyer Road, Bringelly	Monday 23/3/15 – Thursday 2/4/15	33	38	35
35 Ramsay Road, Rossmore	Friday 27/3/15 – Thursday 2/4/15	35	37	35
54 Ridgehaven Road, Silverdale	Thursday 26/3/15 – Thursday 2/4/15	36	36	31
114 Mount Vernon Road, Mount Vernon	Monday 23/3/15 – Thursday 2/4/15	34	35	33
120 Vincent Avenue, Mulgoa	Monday 23/3/15 – Tuesday 31/3/15	38	42	35
Twin Creeks Golf & Country Club (2 Twin Creeks Drive, Luddenham)	Thursday 26/3/15 – Thursday 2/4/15	34	38	33

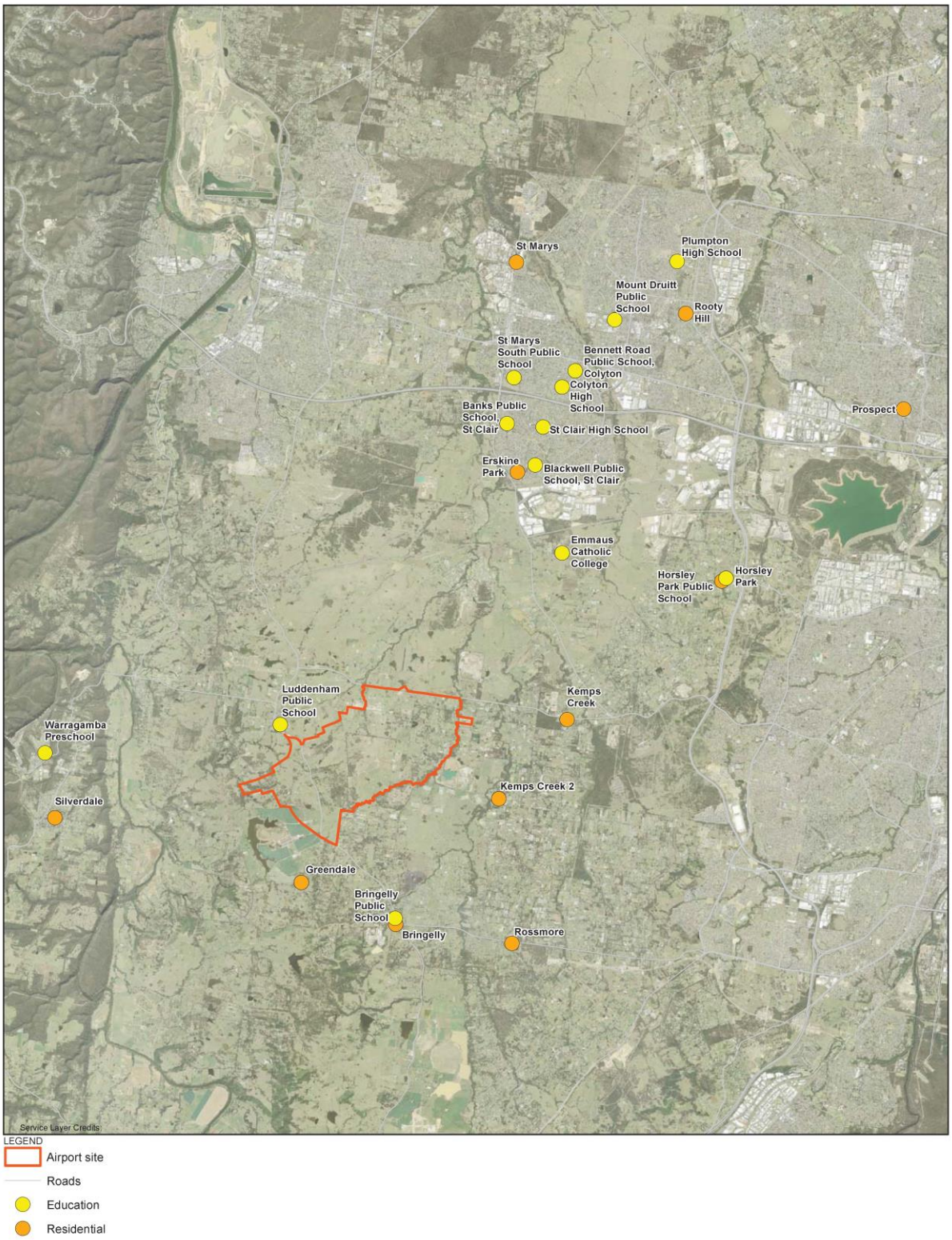
To enable assessment of the potential impacts of aircraft noise, Wilkinson Murray has provided measures of predicted noise exposure in terms of descriptors that are consistent with those used in health studies. The relevant units are:

- $L_{night,outside}$: This unit is used to describe night-time aircraft noise in documents produced by the WHO, and to estimate the risk of chronic health impacts associated with the noise. It represents the equivalent-continuous noise level due to aircraft noise over the period 11pm-7am, calculated on an annual basis as recommended by WHO.
- $L_{Aeq,9am-3pm}$: This unit is used to describe the impact of noise on school students and teachers. It represents the equivalent-continuous noise level due to aircraft noise over the period 9am-3pm, and is once again calculated on an annual basis.

Note that in all cases, these noise levels are calculated at an external point – the noise level within a building will be significantly lower, depending on the building fabric and whether windows and doors are open.

These units were calculated at specific nominated locations, using assumptions and procedures that are described in detail in the noise assessment reports (Wilkinson Murray, 2015 a and b).

Figure 15: Locations of Sensitive Receptors for Noise HRA



Three runway operating modes have been considered for the initial airport development, being "05", "23" and "Head to Head" modes as follows:

- Mode 05: Aircraft arrive from the South West and depart to the North East.
- Mode 23: Aircraft arrive from the North East and depart to the South West.
- Head to Head Mode: Aircraft arrive from the South West and depart to the South West.

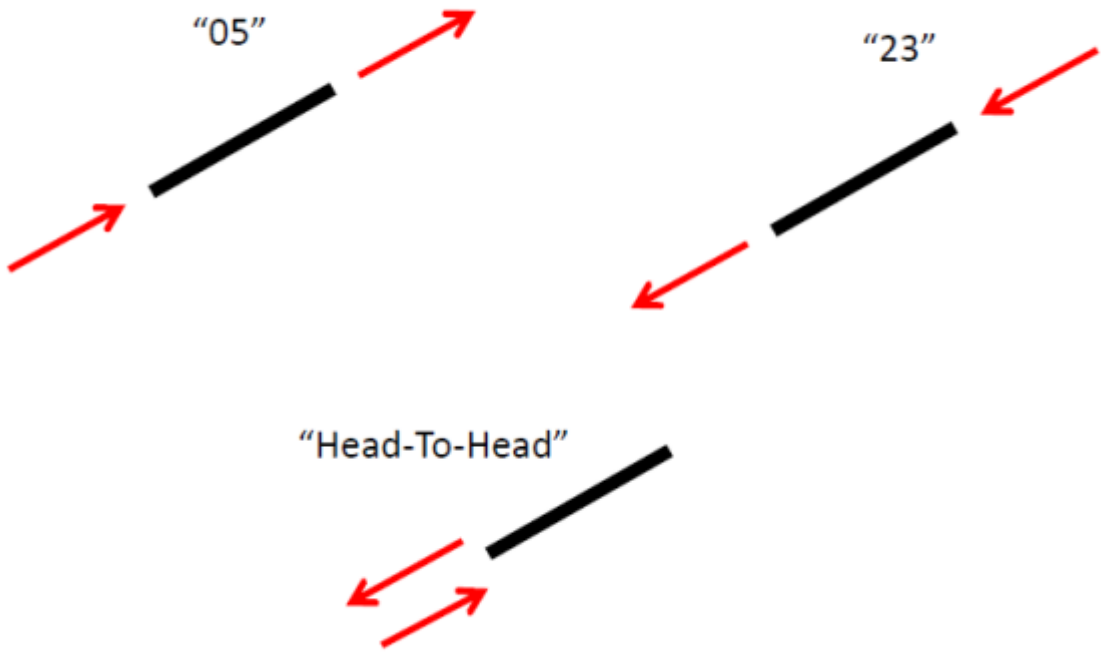
Figure 16 illustrates these modes. Each of these modes may or may not be available at a given time and more than one mode will be used each day, depending on meteorological conditions, particularly wind, number of presenting aircraft, and time of day. However, the assumed order for selection of the modes has an effect on the overall noise impact from the airport.

For daytime operations, two scenarios were considered, in which either the "Prefer 05" or the "Prefer 23" direction is adopted. For night-time operations, the possibility of "head-to-head" operations was also considered, in conjunction with either of the other two modes.

As outlined in Wilkinson Murray's noise assessment report, impacts of this development are considered for two nominal years, namely 2030 and 2050.

Potential impacts are also considered for the long-term case, with dual runways, in nominal year 2063. In this case, only the "Prefer 05" and "Prefer 23" modes are considered.

Figure 16: Potential Airport Operating Mode Options for WSA (Taken from Wilkinson Murray, 2015)



The predicted noise data used in the HRA are summarised in Tables 31 – 32 for aircraft noise and in Table 33 for ground operations noise sources.

Table 31: Daytime L_{Aeq} values 9am-3pm (dB) – aircraft noise (WHO Guideline 55 dB)

Receptor Number	Receptor Name	2030		2050		2063 (two-runway)	
		Prefer 05	Prefer 23	Prefer 05	Prefer 23	Prefer 05	Prefer 23
RESIDENTIAL LOCATIONS							
1	Bringelly	38	35	40	36	37	36
4	Kemps Creek	28	26	32	31	46	43
9	Erskine Park	39	35	45	41	38	35
22	Kemps Creek 2	34	32	37	34	44	41
26	St Marys	37	36	43	41	36	33
27	Greendale	34	36	38	39	48	52
29	Silverdale	32	35	37	40	43	46
30	Rossmore	26	24	28	26	39	35
32	Horsley Park	23	26	28	31	46	48
150	Rooty Hill	30	33	35	38	40	37
152	Prospect	19	23	24	28	42	44
EDUCATIONAL							
43	Warragamba Preschool	25	27	30	32	35	36
65	Emmaus Catholic College Kemps Creek	37	38	42	43	43	43
70	Horsley Park Public School	23	26	28	31	46	48
73	Luddenham Public School	44	46	48	49	49	51
76	Bringelly Public School	39	35	40	36	37	37
142	Mount Druitt Public School	32	34	37	39	43	39
144	St Marys South Public School	38	34	44	40	35	32

145	**Bennett Road Public School**	34	33	39	38	38	35
146	**Colyton High School**	33	34	38	39	40	36
147	**St Clair High School**	36	32	41	37	36	33
148	**Banks Public School**	39	35	45	41	36	32
149	**Blackwell Public School**	37	33	43	38	37	34
151	**Plumpton High School**	30	33	35	38	40	36

Bold indicates exceedance of the adopted criteria

Table 32: L_{Night} outside (equivalent to Leq 11pm-7am)– aircraft noise (WHO Guidelines – night 40 dB, interim target of 55 dB)

Receptor Number	Receptor Name	2030				2050				2063 (two-runway)	
		Prefer 05	Prefer 23	Prefer 05 + H2H	Prefer 23 + H2H	Prefer 05	Prefer 23	Prefer 05 + H2H	Prefer 23 + H2H	Prefer 05	Prefer 23
RESIDENTIAL											
1	**Bringelly**	34	24	24	24	34	27	33	27	33	31
4	**Kemps Creek**	26	24	22	22	30	28	28	27	**42**	37
9	**Erskine Park**	39	26	25	24	**44**	31	39	30	35	28
22	**Kemps Creek 2**	31	26	26	26	33	29	32	29	**40**	34
26	**St Marys**	37	35	30	29	**42**	**40**	38	37	34	26
27	**Greendale**	31	33	33	33	34	36	35	36	**43**	**47**
29	**Silverdale**	30	35	35	35	35	39	39	**40**	39	**44**
30	**Rossmore**	22	17	17	17	24	20	23	20	33	24
32	**Horsley Park**	22	27	22	22	26	32	26	29	**42**	**45**
150	**Rooty Hill**	29	34	28	28	34	39	33	36	38	27
152	**Prospect**	18	24	18	18	23	29	23	26	37	**43**
EDUCATIONAL											

43	**Warragamba Preschool**	24	28	28	28	28	32	32	32	32	34
65	**Emmaus Catholic College, Kemps Creek**	36	39	33	33	**41**	**42**	39	**41**	**41**	**41**
70	**Horsley Park Public School**	22	27	21	22	26	32	26	29	**42**	**46**
73	**Luddenham Public School**	**42**	**44**	**44**	**44**	**46**	**48**	**48**	**48**	**45**	**49**
76	**Bringelly Public School**	34	24	24	24	35	27	34	27	33	32
142	**Mount Druitt Public School**	31	35	29	29	35	39	34	37	**41**	28
144	**St Marys Sth Public School**	38	29	25	25	**43**	34	38	32	32	27
145	**Bennett Road Public School**	34	32	27	27	38	37	34	34	36	27
146	**Colyton High School**	33	34	28	28	37	38	34	35	38	28
147	**St Clair High School**	35	27	23	23	**40**	32	35	29	34	27
148	**Banks Public School**	39	26	25	24	**44**	31	39	30	33	27

149	**Blackwell Public School**	37	26	23	23	**42**	30	36	29	34	28
151	**Plumpton High School**	29	34	28	28	33	39	33	36	38	26

Bold indicates exceedance of the adopted criteria
(Note H2H refers to Head to Head mode)

Table 33: Predicted noise levels for day and night from ground operations noise (WHO Guidelines – night 40 dB, interim target 55 dB; daytime 55 dB)

Receptor	L_{night} dB			L_{Aeq} day (9am-3pm) dB		
	2030	2050	2063	2030	2050	2063
RESIDENTIAL						
Bringelly	36	38	**43**	39	40	45
Kemps Creek	32	35	39	35	37	41
Erskine Park	26	28	32	28	30	34
Kemps Creek 2	36	39	**45**	39	41	47
St Marys	17	20	22	20	21	24
Greendale	**42**	**43**	**47**	45	46	48
Silverdale	29	30	34	32	33	35
Rossmore	30	32	37	32	34	39
Horsley Park	21	24	27	23	25	29
Rooty Hill	15	18	21	18	19	23
Prospect	11	14	18	14	16	20
EDUCATIONAL						
Luddenham Public School	**51**	**53**	**55**	54	**55**	**58**
Warragamba Preschool	28	30	33	31	32	34
Bringelly Public School	37	38	**44**	39	41	46
Emmaus Catholic College, Kemps Creek	27	28	32	30	32	36
Blackwell Public School	25	27	31	27	29	33
Banks Public School	24	26	29	26	28	31
St Clair High School	23	25	29	25	27	31
St Marys South Public School	23	25	29	24	25	28
Horsley Park Public School	21	23	27	23	25	29
Bennett Road Public School	21	23	27	23	25	29

Colyton High School	20	22	26	22	24	28
Mount Druitt Public School	17	20	23	20	21	25
Plumpton High School	14	17	20	17	18	22

Bold indicates exceedance of the adopted criteria

The data shown in Tables 31 and 32 have been used to characterise the health risk associated with exposure to noise from aircraft. The data in Table 33 has been used to calculate the potential health risks associated with ground based operations for the proposed airport. The details of the noise modelling are contained within the EIS noise assessment reports (Wilkinson Murray, 2015 a and b).

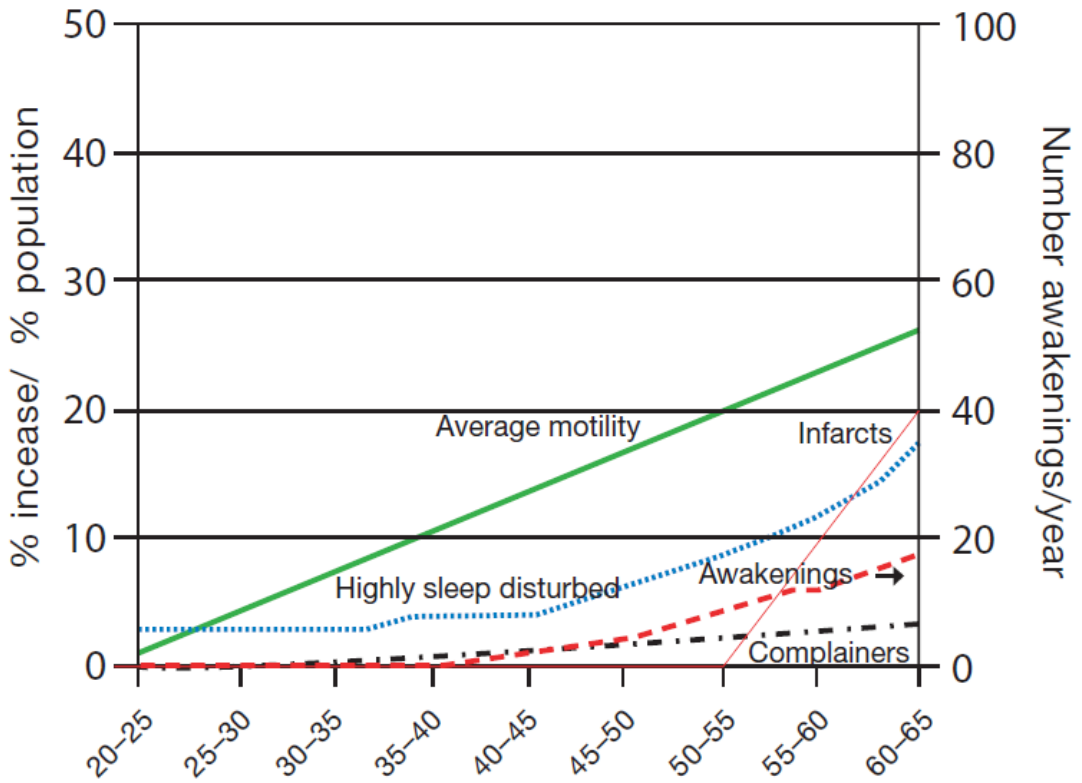
6.3 Risk Characterisation

To characterise the risk to aircraft noise from the proposed airport, three health outcomes have been assessed:

1. Sleep Disturbance assessed as awakenings
2. Increase in Myocardial Infarction
3. Impacts on learning and cognitive development in children

The dose-response relationships from WHO (2009) have been used in the risk calculations for myocardial infarctions and induced awakenings. The WHO dose-response relationships are shown in Figure 17:

Figure 17: Dose Response Relationships for Aircraft Noise Related Health Effects (Reproduced from WHO, 2009; original source European Commission, 2002). The noise metric used is $L_{night, outside}$.



In Figure 17, average motility and infarcts are expressed in percent increase (compared to the baseline number); the number of highly sleep disturbed people is expressed as percent of the population: complainers are expressed as a percent of the neighbourhood population; awakenings are expressed in number of additional awakenings per year.

WHO (2009) identified the health effects that are associated with noise (including aircraft noise) and these are summarised in Table 34:

Table 34: Effects of Different Levels of Night Noise on Population Health (WHO, 2009).

Average night noise level over a year $L_{night\ outside}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, up to this level no substantial biological effects are observed. $L_{night\ outside}$ of 30 dB is equivalent to the no observed effects level (NOEL) for night noise
30 to 40 dB	A number of effects on sleep are observed in this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effects depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst case the effects are modest. $L_{night\ outside}$ of 40 dB is equivalent to the lowest observed adverse effects level (LOAEL) for night noise.
40 to 55 dB	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with noise at night. Vulnerable groups are more severely affected.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable portion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

Below the level of 30 dB $L_{night,\ outside}$, no effects on sleep are observed except for a slight increase in the frequency of body movements during sleep due to night noise. There is insufficient evidence that the biological effects observed below 40 dB $L_{night,\ outside}$ are harmful to health (WHO, 2009). The WHO (2009) however concluded that adverse health effects are observed above 40 dB $L_{night,\ outside}$, such as self-reported sleep disturbance, environmental insomnia and increased use of sleeping pills and sedatives. Above 55 dB the cardiovascular effects become a major public health concern (WHO, 2009). These effects are likely to be less dependent on the nature of the noise. The WHO recommended that for the prevention of subclinical adverse health effects associated with night noise in the population, that the population should not be exposed to night noise levels greater than 40 dB $L_{night,\ outside}$. The WHO (2009) adopted 40 dB as the night noise guideline necessary to protect public health including the most vulnerable groups – children, the chronically ill and the elderly. An interim target of 55 dB was recommended in situations where the night noise guideline was not feasible in the short term but WHO emphasized that this value is not a health-based limit. Vulnerable groups cannot be protected at this level (WHO, 2009).

The purpose of the risk characterization is to estimate potential risks associated with exposure to noise from the proposed airport operations. For the assessment of health effects where there is a known threshold for effect, the predicted noise level for each averaging period is compared to the health

based guideline values as set by WHO (1999: 2009). The ratio of the predicted level to the guideline is termed the hazard quotient (HQ):

HQ = predicted noise level / health based guideline

The hazard quotients are estimated for each of the averaging periods relevant to the guidelines for a given health outcome. The hazard quotient approach has been used to assess the potential impact on children's learning and cognitive development. This applies to daytime noise only.

Where dose-response data are available, quantification of the increase in adverse health effects has been undertaken. This approach has been used for the assessment of awakenings and increases in myocardial infarction. This has been done fro night-time noise.

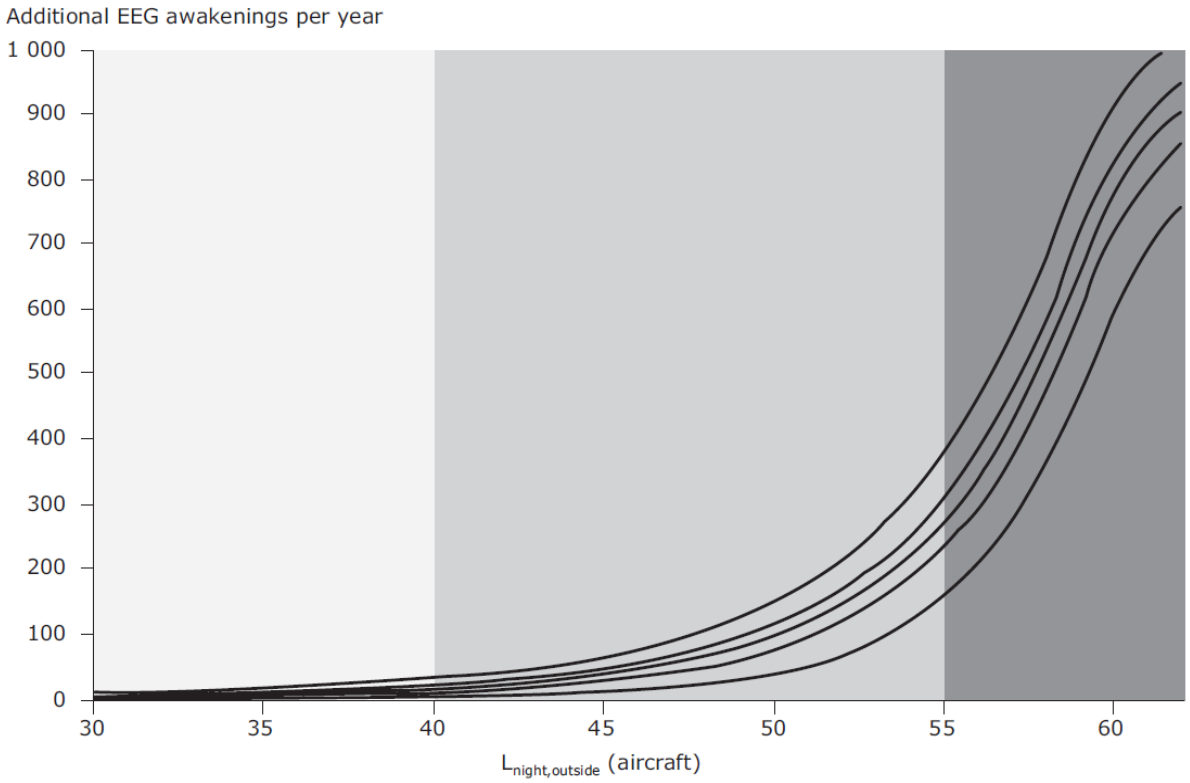
6.3.1 Awakenings

For night-time noise, the Wilkinson Murray (2015) report prepared as part of the EIS found that the extent of night time noise impact depends on the airport operating strategy, and in particular, the adoption of a "Head-to-Head" operating mode where practicable.

In terms of total population affected, the mode option with least impact is "Prefer 23 with Head-to-Head". In assessment year 2030, a "Prefer 05" operating mode option at night would result in an estimated 48,000 people experiencing more than 5 events above 60 dBA at night. This is reduced to approximately 6,000 with a "Prefer 23" operating mode option, or 4,000 if a "Head to Head" mode is included. Under a Prefer 23 with Head-to-Head mode option, an estimated 41,000 residents would experience an average of more than 5 aircraft noise events per night above 60 dBA in 2050. An external noise level of 60 dBA corresponds to an internal level of approximately 50 dBA if windows are open to a normal extent, which is the design criterion for aircraft noise in sleeping areas under Australian Standard 2021. Under the above scenario, the affected residents would be largely in areas to the north-east of the airport, including Horsley Park and parts of Blacktown. However some residents to the south-west of the airport would be more severely affected. Alternative airport operating modes are predicted to result in substantially greater numbers of residents impacted by night time noise, and in particular, a "Prefer 05" mode would result in large parts of St Marys experiencing more than 20 aircraft noise events per night above 60 dBA in 2050.

Two approaches have been taken to assessing awakenings. The first is to estimate the number of EEG awakenings that may be associated with aircraft noise and the second is to assess full awakenings. An EEG awakening is not a fully awakened state but is a measure of disturbed sleep. The dose response curves shown in the EEA *Good Practice Guide on Noise Exposure and Potential Health Effects* (EEA, 2010) have been used to estimate the number of EEG awakenings due to both aircraft and ground operational sources. The EEA identify that 33 dB $L_{night,outside}$ appears to be a threshold value for awakenings related to aircraft noise and below this, sleep disturbance is unlikely to occur. The dose response curves are shown in Figure 18:

Figure 18: Dose Response Curves for EEG Awakenings due to Aircraft Noise (reproduced from EEA, 2010)



The vertical axis in Figure 18 indicates the average number of additional EEG awakenings across a population per year. The shaded areas show the WHO guideline values for night-time noise L_{night} ranges 30-40dB, 40-55dB, >55dB

The data shown in Tables 31 and 32 have been used together with the dose-response data in Figure 18 to estimate the additional number of EEG awakenings per year an individual might experience due to the noise from airport operations. To put these results into context, the EEA (2010) noted that per person, there are usually 24 EEG awakenings per night even during undisturbed 8-hour night sleep. The results for aircraft noise are shown in Table 35 and for ground operations noise in Table 36.

Table 35: Predicted Number of Additional EEG Awakenings Per Person Per Year due to aircraft noise

RECEPTOR	Receptor Number	Lnight									
		2030				2050				2063	
		Prefer 05	Prefer 23	Prefer 05 + H2H	Prefer 23 + H2H	Prefer 05	Prefer 23	Prefer 05 + H2H	Prefer 23 + H2H	Prefer 05	Prefer 23
Bringelly I	1	5	0	0	0	5	0	0	0	0	0
Kemps Creek	4	0	0	0	0	0	0	0	0	40	25
Erskine Park	9	40	0	0	0	40	0	40	0	10	0
Kemps Creek 2	22	0	0	0	0	0	0	0	0	30	5
St Marys	26	25	10	0	0	40	30	40	0	5	0
Greendale	27	0	0	0	0	0	25	10	1	40	100
Silverdale	29	0	10	10	10	10	40	40	30	40	40
Rossmore	30	0	0	0	0	0	0	0	0	0	0
Horsley Park	32	0	0	0	0	0	0	0	0	40	50
Rooty Hill	150	0	5	0	0	5	40	0	10	40	0
Prospect	152	0	0	0	0	0	0	0	0	25	40
SCHOOL LOCATIONS REPRESENTATIVE OF LOCAL COMMUNITY EXPOSURES											

Warragamba Preschool	**43**	0	0	0	0	0	0	0	0	0	5
Emmaus Catholic College, Kemps Creek	**65**	25	40	0	0	30	40	40	30	30	30
Horsley Park Public School	**70**	0	0	0	0	0	0	0	0	40	50
Luddenham Public School	**73**	40	40	40	40	50	100	100	100	50	110
Bringelly Public School	**76**	5	0	0	0	10	0	5	0	0	0
Mount Druitt Public School	**142**	0	10	0	0	10	40	5	25	40	0
St Marys South Public School	**144**	40	0	0	0	40	5	40	0	0	0
Bennett Road Public School	**145**	5	0	0	0	40	25	5	5	25	0
Colyton High School	**146**	0	5	0	0	25	40	5	10	40	0
St Clair High School	**147**	10	0	0	0	30	0	10	0	5	0
Banks Public School	**148**	40	0	0	0	40	0	40	0	0	0

Blackwell Public School	**149**	25	0	0	0	40	0	25	0	5	0
Plumpton High School	**151**	0	5	0	0	0	40	0	10	40	0

The results for EEG awakenings for ground operations noise are shown in Table 36:

Table 36: Predicted Number of additional EEG Awakenings Per Person Per Year due to Ground Operations Noise

Name	Lnight			Awakenings		
	2030	2050	2063	2030	2050	2063
Residential locations						
Bringelly	36	38	43	30	40	60
Kemps Creek	32	35	39	15	25	42
Erskine Park	26	28	32	0	0	15
Kemps Creek 2	36	39	45	30	45	75
St Marys	17	20	22	0	0	0
Greendale	42	43	47	50	60	100
Silverdale	29	30	34	0	0	25
Rossmore	30	32	37	0	15	35
Horsley Park	21	24	27	0	0	0
Rooty Hill	15	18	21	0	0	0
Prospect	11	14	18	0	0	0
Educational locations representative of community exposures						
Luddenham Public School	51	53	55	190	240	400
Warragamba Preschool	28	30	33	0	00	20
Bringelly Public School	37	38	44	35	40	70
Emmaus Catholic College, Kemps Creek	27	28	32	0	0	15
Blackwell Public School, St Clair	25	27	31	0	0	10
Banks Public School, St Clair	24	26	29	0	0	0
St Clair High School	23	25	29	0	0	0
St Marys Sth Public School	23	25	29	0	0	0
Horsley Park Public School	21	23	27	0	0	0
Bennett Road Public School, Colyton	21	23	27	0	0	0
Colyton High School	20	22	26	0	0	0
Mount Druitt Public School	17	20	23	0	0	0
Plumpton High School	14	17	20	0	0	0

The results shown in Tables 35 and 36 indicate that there are several areas where there would be a significant increase in the number of awakenings from both aircraft and ground operations noise. The most affected area is Luddenham where in 2030, there are predicted to be up to 40 additional EEG awakenings per year due to aircraft noise and up to 190 additional EEG awakenings per year due to ground operations noise. In 2063, an additional 110 and 400 EEG awakenings are predicted for Luddenham from aircraft and ground operations noise respectively, The 2011 population of Luddenham was 1496 people. It should be noted that a significant proportion of the Luddenham and Badgerys Creek populations will be relocated as a consequence of the airport construction. This means that the number of people living in the affected areas will be substantially lower than the 2011

population figures. Other affected areas would include Bringelly, Greendale, Warragamba and Rossmore.

As discussed previously, EEG awakenings are not considered a health effect in itself, however they are considered to be early warning signals for other health effects when the values rise above the background levels of around 24 EEG awakenings per night (EEA, 2010).

It should also be noted that the dose-response curves shown in Figure 18 are derived from European studies where outdoor noise levels have been associated with sleep disturbance. In general, European buildings are better insulated than Australian buildings due to climatic conditions. Therefore, the attenuation achieved from outdoors to indoors is likely to be greater than in Australia, meaning that the indoor noise levels associated with EEG awakenings in Australia may be associated with lower outdoor noise levels than in Europe. This would mean that the number of predicted additional awakenings may be underestimated in the areas surrounding the Western Sydney airport site.

For full awakenings, the WHO dose-response curves shown in Figure 17 have been used to estimate the potential increases in awakenings for each of the runway operating modes assessed. The results shown in Table 37 are presented for aircraft noise and Table 38 for ground operations noise and show the number of additional awakenings per persone per year:

Table 37: Predicted **Number of Additional Awakenings per person per Year due to Aircraft Noise under each Operating Scenario**

RECEPTOR	Receptor Number	Lnight									
		2030				2050				2063	
		Prefer 05	Prefer 23	Prefer 05 + H2H	Prefer 23 + H2H	Prefer 05	Prefer 23	Prefer 05 + H2H	Prefer 23 + H2H	Prefer 05	Prefer 23
Bringelly	1	0	0	0	0	1	0	0	0	0	0
Kemps Creek	4	0	0	0	0	0	0	0	0	3	1
Erskine Park	9	1	0	0	0	5	0	1	0	0	0
Kemps Creek 2	22	0	0	0	0	0	0	0	0	3	1
St Marys	26	1	1	0	0	3	1	1	1	0	0
Greendale	27	0	0	0	0	0	1	1	1	3	10
Silverdale	29	0	1	1	1	1	1	1	1	3	5
Rossmore	30	0	0	0	0	0	0	0	0	0	0
Horsley Park	32	0	0	0	0	0	0	0	0	3	10
Rooty Hill	150	0	1	0	0	0	1	0	1	1	0
Prospect	152	0	0	0	0	0	0	0	0	1	3

SCHOOL LOCATIONS REPRESENTATIVE OF LOCAL COMMUNITY EXPOSURES											
Warragamba Preschool	**43**	0	0	0	0	0	0	0	0	0	0
Emmaus Catholic College, Kemps Creek	**65**	1	1	0	0	0	3	1	1	3	5
Horsley Park Public School	**70**	0	0	0	0	0	0	0	0	3	10
Luddenham Public School	**73**	3	3	5	5	10	10	10	10	5	10
Bringelly Public School	**76**	0	0	0	0	1	0	0	0	0	0
Mount Druitt Public School	**142**	0	1	0	0	1	1	0	1	3	0
St Marys Sth Public School	**144**	1	0	0	0	3	0	1	1	0	0
Bennett Road Public School	**145**	0	0	0	0	1	1	1	1	1	0
Colyton High School	**146**	0	0	0	0	1	1	1	1	1	0
St Clair High	**147**	1	0	0	0	3	0	1	0	0	0

School											
Banks Public School	**148**	1	0	0	0	3	0	1	0	0	0
Blackwell Public School	**149**	1	0	0	0	3	0	1	0	1	0
Plumpton High School	**151**	0	1	0	0	0	1	0	1	1	0

The results shown in Table 37 indicate that based on the $L_{night\ outside}$ noise levels, the predicted number of additional awakenings per person per year due to aircraft noise alone for all three scenarios is between 0-5 in 2030 and between 0-10 per person in 2063. The highest predicted number of awakenings is for 2063 with the most affected areas including Luddenham, Greendale and Horsley Park. For most areas assessed, aircraft noise would be unlikely to lead to any additional full awakenings. As with the EEG awakenings, the dose-response curves have been derived from European studies and may underestimate the impact in the area surrounding the Western Sydney airport site.

The results for ground operations are shown in Table 38.

Table 38: Predicted Number of Full Awakenings Per Person Per Year from Ground Operations

Name	Lnight			Awakenings		
	2030	2050	2063	2030	2050	2063
Residential locations						
Bringelly	36	38	43	1	2	2
Kemps Creek	32	35	39	0	0	2
Erskine Park	26	28	32	0	0	0
Kemps Creek 2	36	39	45	1	2	5
St Marys	17	20	22	0	0	0
Greendale	42	43	47	1	2	8
Silverdale	29	30	34	0	0	0
Rossmore	30	32	37	0	0	1
Horsley Park	21	24	27	0	0	0
Rooty Hill	15	18	21	0	0	0
Prospect	11	14	18	0	0	0
Educational locations representative of community exposures						
Warragamba Preschool	28	30	33	0	0	0
Emmaus Catholic College, Kemps Creek	27	28	32	0	0	0
Horsley Park Public School	21	23	27	0	0	0
Luddenham Public School	51	53	55	10	12	15
Bringelly Public School	37	38	44	1	2	3
Mount Druitt Public School	17	20	23	0	0	0
St Marys Sth Public School	23	25	29	0	0	0
Bennett Road Public School, Colyton	21	23	27	0	0	0
Colyton High School	20	22	26	0	0	0
Banks Public School, St Clair	24	26	29	0	0	0
St Clair High School	23	25	29	0	0	0
Blackwell Public School, St Clair	25	27	31	0	0	0
Plumpton High School	14	17	20	0	0	0

As can be seen from Table 38, the predicted noise from ground operations has a greater impact in the suburbs closest to the proposed airport, in particular Luddenham. In 2030, the number of additional awakenings is calculated to be between 0-10 and in 2063 between 0-15. The combined effects of aircraft and ground operations noise is predicted to lead to an additional 25 full awakenings per person per year in Luddenham in 2063. Other affected areas include Greendale, Kemps Creek, Rossmore and Bringelly.

6.3.2 Cardiovascular Effects

The WHO has identified that the NOAEL for increases for myocardial infarction (heart attacks) is 55 dB $L_{night, outside}$. For all receptors assessed for overflight noise, the $L_{night, outside}$ predicted levels were below 55 dB (see Table 28). This was observed for all years assessed and all operating modes. On the basis of these results, it can be concluded that the aircraft noise would not lead to any increased risk in myocardial infarction in nearby communities.

Based on the WHO exposure response curve presented in Figure 17, the ground operations noise levels predicted for Luddenham may result in an increase in myocardial infarction (an indicator of ischaemic heart disease) of approximately 2% in 2030. In 2063, the increase is predicted to be about 10%. Mitigation measures should be considered to reduce this impact. This assessment has assumed that the total population of Luddenham is exposed to the noise levels predicted by Wilkinson-Murray. The 2011 total population of Luddenham is 1496 people but would be substantially lower now following the relocation of the airport residents in 2015.

6.3.3 Learning and Cognitive Development in Children

The aircraft noise report found that the noise impact around the airport would depend primarily on the airport operating mode that was adopted by Air Traffic Control. The two basic operating scenarios that have been assessed are – "Prefer 05" in which aircraft approach and depart the airport in a south-west to north-east direction (unless this is not possible due to wind or other conditions), and "Prefer 23" in which the opposite direction is preferred. A "Head-to-Head" mode of operation, in which aircraft both approach and depart to the south-west, is also considered for night time operations.

In assessment year 2030, a "Prefer 05" operating scenario at night would result in an estimated 48,000 people experiencing more than 5 events above 60 dBA at night. This is reduced to approximately 6,000 with a "Prefer 23" operating scenario, or 4,000 if a "Head to Head" mode is included. However, as for daytime noise, "Prefer 23" or "Head to Head" result in slightly more people experiencing higher noise impacts. These will be residents in rural residential areas to the south and west of the airport (Wilkinson-Murray, 2015a).

The potential risk arising from aircraft noise on children's learning and cognitive development is related to daytime noise and has been undertaken using the hazard quotient approach. For this assessment, the WHO Community Noise Guidelines (1999) have been used.

Two approaches have been taken:

1. Assessment based on outside predicted levels
2. Assessment using the indoor guideline assuming a 10 dB reduction in noise levels due to noise attenuation by buildings.

The relevant WHO guidelines are shown in Table 27. The predicted daytime noise levels for each of the school locations identified as sensitive receivers are shown in Table 28. The resulting hazard quotients (HQ) are shown in Table 39 for outdoors and Table 40 for indoors for aircraft noise.

Table 39: Hazard Quotients for Learning and Cognitive Development in Children – outside for aircraft noise (WHO Guideline 55dB)

	2030						2050						2063 (two-runway)					
Receptor	**Prefer 05**			**Prefer 23**			**Prefer 05**			**Prefer 23**			**Prefer 05**			**Prefer 23**		
	L_{Aeq} 9am – 3pm (dB)	dB above or below guideline	HQ	L_{Aeq} 9am – 3pm (dB)	dB above /below guideline	HQ	L_{Aeq} 9am – 3pm (dB)	dB above /below guideline	HQ	L_{Aeq} 9am – 3pm (dB)	dB above /below guideline	HQ	L_{Aeq} 9am – 3pm (dB)	dB above /below guideline	HQ	L_{Aeq} 9am – 3pm (dB)	dB above /below guideline	HQ
Warragamba Preschool	25	-30	0.5	27	-28	0.5	30	-25	0.5	32	-23	0.6	35	-20	0.6	36	-19	0.7
Emmaus Catholic College, Kemps Creek	37	-18	0.7	38	-17	0.7	42	-13	0.8	43	-12	0.8	43	-12	0.8	43	-12	0.8
Horsley Park Public School	23	-32	0.4	26	-29	0.5	28	-27	0.5	31	-24	0.6	46	-9	0.8	48	-7	0.9
Luddenham Public School	44	-11	0.8	46	-9	0.8	48	-7	0.9	49	-6	0.9	49	-6	0.9	51	-4	0.9
Bringelly Public School	39	-16	0.7	35	-20	0.6	40	-15	0.7	36	-19	0.7	37	-18	0.7	37	-18	0.7
Mount Druitt Public	32	-23	0.6	34	-21	0.6	37	-18	0.7	39	-16	0.7	43	-12	0.8	39	-16	0.7

School																	
St Marys Sth Public School	38	-17	0.7	34	-21	0.6	44	-11	0.8	40	-15	0.7	35	-20	0.6	32	-23	0.6
Bennett Road Public School	34	-21	0.6	33	-22	0.6	39	-16	0.7	38	-17	0.7	38	-18	0.7	35	-20	0.6
Colyton High School	33	-22	0.6	34	-21	0.6	38	-17	0.7	39	-16	0.7	40	-15	0.7	36	-19	0.6
St Clair High School	36	-19	0.7	32	-23	0.6	41	-14	0.7	37	-18	0.7	36	-19	0.6	33	-22	0.6
Banks Public School	39	-16	0.7	35	-20	0.6	45	-10	0.8	41	-14	0.8	36	-19	0.6	32	-23	0.6
Blackwell Primary School	37	-18	0.7	33	-22	0.6	43	-12	0.8	38	-17	0.7	37	-18	0.7	34	-21	0.6
Plumpton High School	30	-25	0.6	33	-22	0.6	35	-20	0.6	38	-17	0.7	40	-15	0.7	36	-19	0.6

Table 40: Hazard Quotients for Learning and Cognitive Development in Children – inside assuming 10 dB attenuation (WHO Guideline 35dB)

	2030				2050				2063 (two-runway)			
Receptor	**Prefer 05**	**HQ**	**Prefer 23**	**HQ**	**Prefer 05**	**HQ**	**Prefer 23**	**HQ**	**Prefer 05**	**HQ**	**Prefer 23**	**HQ**
Warragamba Preschool	15	0.4	17	0.5	20	0.6	22	0.6	25	0.7	27	0.8
Emmaus Catholic College, Kemps Creek	27	0.8	28	0.8	32	0.9	33	**1**	33	**1**	33	**1**
Horsley Park Public School	13	0.4	16	0.5	18	0.5	21	0.6	36	**1**	38	**1.1**
Luddenham Public School	34	**1**	36	**1.0**	38	**1.1**	39	**1.1**	39	**1.1**	41	**1.2**
Bringelly Public School	29	0.8	25	0.7	30	0.9	26	0.8	27	0.8	27	0.8
Mount Druitt Public School	22	0.6	24	0.7	27	0.8	29	0.8	33	0.9	29	0.8
St Marys Sth Public School	28	0.8	24	0.7	34	**1**	30	0.9	25	0.7	22	0.6
Bennett Road Public School	24	0.7	23	0.7	29	0.8	28	0.8	28	0.8	25	0.7
Colyton High School	23	0.7	24	0.7	28	0.8	29	0.8	30	0.9	26	0.7

St Clair High School	26	0.7	22	0.6	31	0.9	27	0.8	26	0.8	23	0.7
Banks Public School	29	0.8	25	0.7	35	**1**	31	0.9	26	0.7	22	0.6
Blackwell Primary School	27	0.8	23	0.7	33	0.9	28	0.8	27	0.8	24	0.7
Plumpton High School	20	0.6	23	0.7	23	0.7	28	0.8	30	0.88	26	0.7

Note: 10 dB has been subtracted from outdoor noise levels in Table 40 to provide an estimate of indoor noise levels.
Number in bold indicate hazard quotient graeter than or equal to 1

As can be seen from Tables 39 and 40, most hazard quotients are less than 1 indicating that the risk from the aircraft noise from each of the proposed modes of operation generally does not pose an unacceptable risk with respect to children's learning and cognitive development. For outdoor noise, the actual predicted L_{day} values are between 6 dB and 25 dB below the relevant WHO guideline. Hazard quotients less than 1 are considered to be an acceptable level of risk (enHealth, 2012).

In some cases, there are marginal exceedances of 1. This does not mean that there will be an impact on children's learning and cognitive development but that there is an increased risk albeit very low. The main impact is at Luddenham where the predicted noise levels are between 4 and 6 dB below the indoor guidelines in 2063. According to enHealth (2004) and WHO (1999), a 10 dB increase in noise is associated with a doubling of the loudness and is noticeable. Noise mitigation measures recommended as part of the noise technical report will lead to a reduction in this potential risk.

For ground operations noise, the HQ results are shown in Tables 41 and 42:

Table 41: Hazard Quotients for Learning and Cognitive Development in Children – inside for ground-based noise assuming 10 dB attenuation (WHO guideline 35 dB)

Receptor	Lday 2030 dB	Hazard Quotient – indoors	Lday 2050 dB	Hazard Quotient – indoors	Lday 2063 dB	Hazard Quotient – indoors
Warragamba Preschool	31	0.6	32	0.6	34	0.7
Emmaus Catholic College, Kemps Creek	30	0.6	32	0.6	36	0.7
Horsley Park Public School	23	0.4	25	0.4	29	0.5
Luddenham Public School	54	**1.3**	55	**1.3**	58	**1.4**
Bringelly Public School	39	0.8	41	0.9	46	**1.0**
Mount Druitt Public School	20	0.3	21	0.3	25	0.4
St Marys Sth Public School	24	0.4	25	0.4	28	0.5
Bennett Road Public School	23	0.4	25	0.4	29	0.5
Colyton High School	22	0.3	24	0.4	28	0.5
St Clair High School	25	0.4	27	0.5	31	0.6
Banks Public School	26	0.5	28	0.5	31	0.6
Blackwell Public School	27	0.5	29	0.5	33	0.7
Plumpton High School	17	0.2	18	0.2	22	0.3

As can be seen from Table 41, the greatest impact from ground operations noise indoors is at Luddenham. The hazard quotients experienced at Luddenham exceed 1 suggesting that noise mitigation measures should be implemented. The hazard quotients above 1 correspond to an increase in noise levels above the guideline value of between 19 and 23 dB indicating a significant increase in

noise levels. The data shown in Table 42 show a similar pattern for outside noise with the hazard quotients exceeding 1 at Luddenham in 2063.

Table 42: Hazard Quotients for Learning and Cognitive Development in Children – outside (WHO Guideline 55 dB)

Receptor	2030	Hazard Quotient outdoors	2050	Hazard Quotient outdoors	2063	Hazard Quotient outdoors
Warragamba Preschool	31	0.6	32	0.6	34	0.6
Emmaus Catholic College	30	0.5	32	0.6	36	0.7
Horsley Park Public School	23	0.4	25	0.5	29	0.5
Luddenham Public School	54	1.0	55	**1.0**	58	**1.1**
Bringelly Public School	39	0.7	41	0.7	46	0.8
Mount Druitt Public School	20	0.4	21	0.4	25	0.5
St Marys Sth Public School	24	0.4	25	0.5	28	0.5
Bennett Road Public School	23	0.4	25	0.5	29	0.5
Colyton High School	22	0.4	24	0.4	28	0.5
St Clair High School	25	0.5	27	0.5	31	0.6
Banks Public School	26	0.5	28	0.5	31	0.6
Blackwell Public School	27	0.5	29	0.5	33	0.6
Plumpton High School	17	0.3	18	0.3	22	0.4

Bold numbers indicate hazard quotient greater than or equal to 1 indicating exceedance of guideline value..

6.4 Summary

The HRA has been conducted for noise impacts from aircraft and ground operations noise sources. Based on the results of the noise assessment, the risk posed to the health of the exposed communities is generally low. No increase in cardiovascular outcomes is likely from aircraft noise as the predicted night noise levels are below the threshold for adverse effects. However, ground operations noise is predicted to be above the threshold value of 55 dB Lnight and may lead to a 10% increase in myocardial infarctions in Luddenham in 2063 if not mitigated.

A significant increase in EEG awakenings is predicted especially in Luddenham. In 2030, it is predicted that there would be 190 additional EEG awakenings per person per year and an additional 400 EEG awakenings per person per year with ground-based noise in 2063. For full awakenings, the number is lower but still highest in Luddenham. There is a greater predicted impact from ground- operations noise than aircraft noise. Mitigation measures in the noise assessment report should be implemented and would reduce this impact.

Impacts on children's learning and cognitive development are predicted to be within acceptable risk levels for most locations for aircraft noise. The impact of ground operations noise is more substantial. In Luddenham, increases of up to 3 dB above the WHO guideline are predicted for outdoor noise levels in 2063. Mitigation measures should be implemented at these locations to reduce this risk to within acceptable levels. For the indoor assessment, assuming a 10 dB attenuation from predicted outdoor

levels, the hazard quotients above 1 correspond to an increase in noise levels above the guideline value of between 19 and 23 dB in 2030 and 2063 respectively at Luddenham indicating a significant increase in noise levels. Mitigation measures should be implemented to reduce this risk to within acceptable levels.

According to the Wilkinson-Murray reports there are practical limits on the mitigation measures that can be applied to reduce the impact of ground operations noise sources. Although in theory it is possible to locate buildings and construct earth mounds in positions that may shield affected receivers from noise sources, in practice this is difficult to achieve. Space is required around the runway for safety and other reasons and access to the runway is required. Also, to facilitate mitigation of aircraft noise would essentially comprise a wall almost the full length of the runway, which is unlikely to be feasible. Even if feasible, only small reductions of 5-8dBA may be achievable using 10m mounds, and significant residual impacts would still result.

Engine run-up on high power would normally be carried out during daytime and night time. High power run-up should be restricted to special circumstances where high power testing is required after maintenance activity prior to an aircraft taking off. Restricting the amount of high power run-up at night time will substantially reduce the impact of run-up noise. It may also be practical to construct buildings or mounds or barriers near the run-up area to provide greater noise shielding. It is possible that reductions of around 10dBA may be able to be achieved with mounds or buildings at least 10m high, but moderate residual impacts would still occur.

In relation to the high levels of exceedance predicted and on the assumption that the noise mound along the edge of the runway or airport boundary is not feasible, there would seem to be few practical options to avoid the need for acquisition in the most affected areas.

In any event, many of the localities affected by ground noise will also be affected by aircraft noise, but to a greater degree. Luddenham is one such locality. This means that, even if ground noise sources is reduced substantially, this locality will still be significantly affected by airport noise. This raises the possibility of at-receiver treatment to reduce internal noise levels, because this type of treatment will reduce both ground and aircraft noise.

7 HEALTH RISK ASSESSMENT – GROUNDWATER AND SURFACE WATER

7.1 Existing Groundwater Conditions

The following information provides an overview of the groundwater conditions beneath the proposed airport site. Information has been sourced primarily from the Groundwater Assessment prepared by GHD (2015) which provides a review of previous investigations conducted to inform the environmental impact statement (EIS) prepared in 1997 as well as a reanalysis of additional, more recent information. However, it is noted that groundwater has not been sampled since completion of the 1997 EIS, thus the following information may not be representative of current groundwater conditions in the area.

7.1.1 Local Hydrogeology

According to GHD, there are two main aquifers present beneath the airport site:

- A shallow alluvial aquifer which is considered to be localised around the main creeks which drain the site, and is generally encountered at depths of approximately 0.7 – 4.7 m below ground level (bgl).
- A confined regional aquifer within the Bringelly Shale which is present at approximately 20 m bgl.

The 1997 EIS notes that perched groundwater was intermittently encountered within the weathered shale profile, however it is not considered to be a continuous aquifer. GHD also report the presence of a deep regional aquifer interpreted to be present at depths of greater than 100 m bgl within the Hawkesbury Sandstone.

GHD indicate that it is unlikely the aquifers are interconnected based on geological information, water strike observations and groundwater elevation data. Details of the aquifer parameters are presented in GHD and have not been re-iterated herein, the following summary of information from GHD is provided for the purpose of the current assessment:

- Both aquifers present beneath the site are reported to have low hydraulic conductivity (0.0027 – 0.14 m/day).
- Storage parameters of the aquifers present beneath the site have not been assessed.
- Standing water level elevations (in surrounding registered bores) suggest that there is a strong downward head gradient between the Bringelly Shale aquifer and the underlying Hawkesbury Sandstone aquifer, which suggests that there is limited hydraulic connectivity between the aquifers.
- The Luddenham Dyke (which runs south-east to north-west in the south west area of the site) is observed to create a divide in the Bringelly Shale aquifer with flow on the eastern side towards Badgerys Creek and on the western side toward Duncan Creek.

7.1.2 Use of Groundwater in Study Area

According to GHD, there are 42 registered groundwater bores within a five (5) km radius of the centre of the site. Twelve (12) of these bores are registered as being used for domestic, stock, industrial, farming and irrigation purposes, these wells range in depth from 61 - 337 m bgl (GHD, 2015). The depth of registered extraction bores indicates that the majority of groundwater users extract water from the Bringelly Shale and Hawkesbury Sandstone aquifers.

Water quality within the aquifers beneath the site and potential environmental values considered relevant are discussed further below in **Section 7.1.3**.

7.1.3 Groundwater Environmental Values

The National Water Quality Management Strategy (NWQMS; Australian Government, 2013) defines 'Environmental Value' as:

'*the term applied to a particular category of value or use of groundwater that is important for a healthy ecosystems or for public benefit, welfare, safety or health*.'

The Environmental Values (EV) of an aquifer require protection from the effects of contamination, waste discharge and deposits. The NWQMS defines the following environmental values of groundwater aquifers which have been adopted in the NSW Groundwater Quality Protection Policy (NSW GQPP; Department of Land & Water Conservation [DLWC], 1998):

- Ecosystem protection;
- Recreation and aesthetics;
- Raw water for drinking water supply;
- Agricultural water; and
- Industrial water.

The NSW GQPP (DLWC, 1998) states that:

'*All groundwater systems should be managed such that their most sensitive identified beneficial use (or environmental value) is maintained*.'

'*Groundwater pollution should be prevented so that future remediation is not required.*'

'*For new developments, the scale and scope of work required to demonstrate adequate groundwater protection shall be commensurate with the risk the development poses to a groundwater system and the value of the groundwater resource.*'

The NSW GQPP (DLWC, 1998) outlines various levels of groundwater quality based on availability of groundwater and salinity as follows:

- 0-1,000 mg/l total salts (low yield): Good quality – usually potable and suitable for most uses.
- 0-1,000 mg/l total salts (high yield): Good quality – usually potable and yields may be sufficient for irrigation.
- 1,000-3,000 mg/l total salts: Suitable for all livestock, some domestic and limited industrial uses
- 3,000-14,000 mg/l total salts: Fair to poor stock quality

Based on the above groundwater classifications and the reported conductivity of groundwater sampled in 1995 and 1998 the following EV are considered to apply to the aquifers beneath the site:

- Ecosystem protection;
- Recreation and aesthetics;
- Agricultural water

Raw water for drinking water supplies is not considered to be an EV for the Alluvial aquifer or the Bringelly Shale aquifer. This is based on the salinity in the Alluvial and Bringelly Shale aquifers which is reported to be >1,000 mg/l (based on 1995-1998 data), and thus is unsuitable for potable water uses.

Industrial water use has not been considered further because the level of protection and chemicals of concern are considered likely to vary depending upon the type of industrial water use.

Consideration of potential risks to the relevant ecological values, as listed above, will be included in the assessment of risks to groundwater.

7.1.4 Source of Potential Groundwater Contamination

CURRENT

Table 43 summarises the findings of a review of current land use conditions at and in the vicinity of the site including potentially contaminating activities.

Table 43: Potentially Contaminating Activities - Current

Potentially Contaminating Activity	Associated Potential Contaminants (c)
Farming activities (use and storage of associated chemical products; and historical land filling practice on rural properties)	- Pesticide and herbicides - Fertilisers - Petroleum hydrocarbons - Polycyclic aromatic hydrocarbons (PAH) - Heavy metals - Chlorinated hydrocarbons (from de-greasing activities) - Asbestos - Paint and paint thinner associated compounds
Landfill operation (a)	- Petroleum Hydrocarbons - Heavy metals - PAH - Perfluorinated compounds (PFC) - Asbestos - Chlorinated hydrocarbons - Other household and industrial chemicals
Brickworks	- Asbestos - Fluoride - Petroleum hydrocarbons - PAH - Heavy metals
Landscape suppliers	- Fertilisers - Herbicides and pesticides - PAH - Heavy metals
Defence and Air Force base operations (b)	- Petroleum hydrocarbons - PAH - Heavy metals - Asbestos - PFC
Petrol Station operation (former and current)	- Petroleum hydrocarbons - Chlorinated hydrocarbons (from de-greasing activities) - Heavy metals - Asbestos

(a) The Elizabeth Drive Landfill Facility is noted to be present to the north of the site. There is potential for other historical land filling activities to have been conducted in the area.

(b) Defence Establishment Orchard Hills is located to the north-west of the site, and an Air Force Telecommunications facility (decommissioned) is located to the south-east of the site.

(c) This is not an exhaustive list of potential contaminants, but is intended to provide an indication of the types of contaminants which may be present at the site and in surrounding areas (thus posing a potential groundwater contamination issue).

AIRPORT CONSTRUCTION and OPERATION

During the construction and operation of the proposed airport, there would be potential for a number of activities which may result in environmental contamination if not properly managed. **Table 44** provides a list of potentially contaminating activities which may occur during airport construction and operation.

Table 44: Potentially Contaminating Activities – Airport Construction and Operation

Potentially Contaminating Activity	Associated Potential Contaminants (a)
Chemical and fuel storage	- Petroleum Hydrocarbons - Heavy metals - PAH - PFC (b)
Equipment Operation	- Petroleum Hydrocarbons - Heavy metals - PAH - PFC (b)
Equipment maintenance	- Petroleum Hydrocarbons - Heavy metals - PAH - Chlorinated Hydrocarbons - PFC (b)
Fire Fighting	- PFC (c) - Petroleum Hydrocarbons - Heavy metals - PAH

(a) This is not an exhaustive list of potential contaminants, but is intended to provide an indication of the types of contaminants which may be present at the site and in surrounding areas (thus posing a potential groundwater contamination issue).

(b) PFCs have been identified to be present in some hydraulic fluids used in the aviation industry.

(c) It is understood that Airservices Australia transitioned to 'fluorine-free' aqueous film forming foams (AFFF) for fire-fighting at airports across Australia from 2010. However there are some compounds in the fluorine-free foams which may break down to perfluorooctanoic acid (PFOA; one of the main PFCs in previously used AFFF). There is also potential for small amounts of PFCs to be present in these foams as a result of contamination which may occur during the production of the products.

Based on the activities and identified potential contaminants which may be associated with construction and operation of the proposed airport, there is likely to be considerable overlap of contaminants potentially present as a result of current activities at the site and in surrounding areas. It is therefore considered important that baseline groundwater data for these identified potential contaminants be collected to enable appropriate monitoring to be conducted during construction and operation phases of the proposed airport. This would enable any impacts from the airport construction and operation to be detected early and mitigation measures put in place to minimise any risk.

7.1.5 Hazard Identification

Contaminants of potential concern (CoPC) are considered to be those contaminants which are known or suspected to be present at concentrations which may warrant inclusion in the risk assessment. A CoPC is selected based on its reported presence in environmental media at concentrations above

adopted relevant screening criteria which have been derived to be protective of identified environmental values for the groundwater aquifer.

The ASC NEPM (NEPC, 2013) states that '*site assessment should consider the risks from contaminated groundwater to all potential receptors on and off the site of origin and potential effects on groundwater resources*.' Groundwater investigation levels (GIL) are adopted from the following sources (which have been individually referenced in **Table 45**:

- Australian Water Quality Guidelines 2000 (AWQG) (ANZECC, 2000)
- Australian Drinking Water Guidelines 2011 (ADWG) (NHMRC, 2011) and
- Guidelines for Managing Risk in Recreational Waters 2008 (GMRRW) (NHMRC, 2008)

Where guideline values could not be obtained from the above sources, additional sources were referenced as per the hierarchy listed in enHealth (2012).

Table 45 outlines the guidelines adopted for groundwater for each identified EV as per the guidance set out in the ASC NEPM (NEPC, 2013) and enHealth (2012).

In the absence of groundwater specific guidelines, surface water guidelines have been adopted for screening of groundwater. This approach is considered conservative as surface water guidelines only apply to groundwater at the point of discharge, however all available groundwater data have been collected from within the aquifer and not at the point of discharge to surface water.

Table 45: Adopted Groundwater Guidelines

Environmental Value	Guideline Hierarchy
Ecosystem protection	- ANZECC (2000) Freshwater Quality Criteria (95% Protection) (e) - ANZECC (2000) Freshwater Quality Criteria (Medium to Low Reliability) - USEPA (2013) Ambient Water Quality Criteria (Chronic – Freshwater) - USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria - USEPA (2003) Region 5 RCRA Ecological Screening Levels (water) - RIVM (2001) Dutch Groundwater Intervention Value
Recreation (a) and aesthetics	- NHMRC (2011) Drinking Water Guidelines (as amended 2013) - WHO (2011) Drinking Water Guidelines - WHO (2008) Petroleum Products in Drinking Water - USEPA (2015) Regional Screening Level – Tap Water
Agricultural water (b) (d)	- ANZECC (2000) Stock Water Screening Criteria - ANZECC (2000) Irrigation – Long Term Value (LTV) (c) - NHMRC (2011) Drinking Water Guidelines (as amended 2013) - WHO (2011) Drinking Water Guidelines - WHO (2008) Petroleum Products in Drinking Water - USEPA (2015) Regional Screening Level – Tap Water

(a) As per NHMRC (2008) Guidelines for managing risk in recreational waters, all drinking water guidelines have been multiplied by a factor of 10x based on the assumption that recreational exposure would result in a lower incidental intake (0.2 L) of water than direct ingestion of drinking water (assumed to be 2 L/day).

(b) Agricultural water is considered to consist of stock water and irrigation uses.

(c) The long term value for irrigation has been selected as a conservative approach as it is unclear how long irrigation activities using groundwater have been conducted for.

(d) Drinking water guidelines have been selected where no ANZECC Stock Watering guideline is available, as per ANZECC (2000).

(e) A 95% species protection value has been selected as the area is considered to be moderately disturbed, and thus 95% is likely to provide a suitable level of protection for current and proposed future land uses.

ANZECC – Australia New Zealand Environment Conservation Council

NHMRC – National Health and Medical Research Council

RIVM – Dutch National Institute for Public Health and the Environment

USEPA – United States Environment Protection Agency

WHO – World Health Organisation

7.1.6 Identification of Contaminants of Potential Concern

Groundwater data from samples collected in 1995 and 1998 were compared to GIL (as per the ASC NEPM [NEPC, 2013]) from guideline sources listed in **Table 45**. The analytical data screening is presented in **Table A1** in **Appendix A**. **Table 46** provides a summary of CoPC which were reported at concentrations which exceeded the adopted GILs.

Table 46: Groundwater Chemicals of Potential Concern

Environmental Value	Chemicals of Potential Concern
Ecosystem protection	- Aluminium - Copper - Lead - Zinc - Calcium - Chloride - Magnesium - Potassium - Sodium - Nitrogen - Phosphorus Reported electrical conductivity in groundwater (in both the Alluvial aquifer and the Bringelly Shale aquifer) was generally above the range identified for lowland river systems (ANZECC,2000). Therefore where groundwater has the potential to discharge to surface water it may result in increased salinity loading to the surface water environment.
Recreation and aesthetics (a)	- Phosphorus
Agricultural water (irrigation and stock watering)	- Iron (irrigation criteria only) - Nitrogen - Phosphorus
(a) Analytical data have been compared to health based screening values for primary contact recreation exposures via incidental ingestion. Further consideration of potential aesthetic impacts (as per NHMRC, 2008) should be made where groundwater is discharged to surface water bodies.	

The CoPCs listed in **Table 45** provide an indication of chemicals which may pose a risk to surface water at the point of discharge. The reported exceedances of screening criteria do not indicate that there is an actual risk to surface water environments from groundwater contaminants. It should be noted that there was only a limited number of contaminants analysed in the groundwater samples. Not all potential chemical contaminants which may be associated with current land uses at the site were assessed (**Section 7.1.4**). Given that there are sources of these pollutants identified, further investigation and baseline monitoring is required to identify conditions such that:

- risks to construction workers can be assessed;
- if required, remediation can be conducted prior to construction of the proposed airport; and
- monitoring can be conducted during airport construction and operation to assess the effectiveness of environmental management and mitigation measures.

7.1.7 Potential Exposure Pathways

A fundamental concept of risk assessment is the identification of an exposure pathway between the source of contamination and the identified receptors. An exposure pathway linkage is generally considered to include the following elements:

- **Contaminant Source**: A source of contamination and/or a mechanism for release of the chemical or physical agent;
- **Retention or Transport Medium**: Where the exposure point differs from the source (e.g. where soil contamination at the source has resulted in groundwater contamination at the point of

exposure) a transport/exposure medium or media (where chemicals are transferred between media) via which the chemical or physical agent is transported.

- **Exposure Point**: A location of potential contact between the organism and the chemical or physical agent;
- **Exposure Route**: A mechanism via which the chemical or physical agent comes in contact with the exposed receptors (e.g. inhalation, ingestion, dermal contact).

Where one or more of the above linkage elements is missing, the exposure pathway is considered to be incomplete and there is therefore no risk to the receptor. Where the exposure pathway linkage is identified to be complete, further assessment should be undertaken of the potential risks arising from exposure.

For the purpose of the current assessment, it is assumed that there is potential for contaminants to be present in groundwater, and thus the potential exposure points and exposure routes for groundwater have been considered further and are listed in **Table 47** below. If exposure occurs via the pathways identified in Table 46, there is a potential for health impacts in the exposed community.

Table 47: Groundwater Exposure Pathway Linkages

Receptor	Exposure Point	Exposure Route
Human users of extracted groundwater (e.g. farmers, residents etc.) (a)	Groundwater extraction point	- Incidental ingestion of groundwater during irrigation and other domestic activities - Dermal contact with groundwater during irrigation and other domestic activities - Inhalation of groundwater derived vapours at the point of extraction (c)
Human users of surface water bodies where groundwater discharges to surface water (e.g. recreational receptors)	Surface water body	- Incidental ingestion of groundwater derived contaminants in surface water - Dermal contact with groundwater derived contaminants in surface water - Inhalation of groundwater derived vapours at the point of discharge to surface water
Groundwater dependent ecosystems	Within the groundwater aquifer	- Uptake and accumulation of groundwater derived contaminants via plant root systems - Direct exposure to contaminants in groundwater
Aquatic organisms that inhabit surface water bodies where groundwater discharges	Surface water body where groundwater is discharging	- Ingestion of groundwater derived contaminants in surface water - Direct contact with groundwater derived contaminants in surface water - Ingestion of accumulated groundwater derived contaminants in the food chain (b)

(a) The drinking water exposure pathway has not been considered for these receptors as reported electrical conductivity in the Alluvial and Bringelly Shale aquifers is considered to exceed concentrations which would enable use of extracted groundwater for drinking water purposes.

(b) This exposure route may have wider implications where groundwater derived contaminants have the potential to bioaccumulate within the food chain. Bioaccumulation of contaminants in aquatic organisms may also result in biomagnification in higher order organisms (e.g. birds which consume aquatic organisms).

(c) Groundwater has not been analysed for the presence of volatile contaminants to date, however there is potential that they may be present as a result of current and proposed future activities at the site.

7.1.8 Risk Characterisation

The final step in the risk assessment process is to characterise the risks to identified receptors as a result of potential exposures to CoPC. This step involves integration of the information collected during the previous steps and qualitative (or where possible quantitative) assessment of the potential for unacceptable risks to occur.

For this assessment, only a qualitative evaluation of the potential for risks to identified receptors has been conducted. This is due to the limited data available which can be used to assess existing risks and risks under proposed future (construction and operation of the airport) conditions at the site. The following sections provide an assessment of the additional information required to further evaluate potential risks to environmental values of groundwater under proposed future conditions, and management and mitigation measures which can be implemented to reduce the potential for risks.

CURRENT POTENTIAL RISKS

Screening of available groundwater data against adopted GIL (as per the ASC NEPM [NEPC, 2013]) indicates that there are potential risks to the following EVs where groundwater is extracted or discharging to the surface:

- Ecosystem protection
- Recreation
- Agricultural water (irrigation and stock watering)

The following limitations and data gaps have been identified during the current assessment, and thus no further assessment or refinement of the risk assessment process could be completed:

- Groundwater hasn't been sampled since 1998 and thus current groundwater conditions at the site are uncertain.
- The potential for groundwater contamination to be present as a result of current land uses at and surrounding the site has not been assessed. Therefore it is unable to be determined what additional risks may be posed to groundwater from construction and operation of the airport(outside of identified CoPC).
- Groundwater samples have not been analysed for all contaminants which may be present as a result of proposed airport construction or operation. The absence of these data will make it difficult to analyse monitoring data to assess whether contamination is occurring and the effectiveness of any management and mitigation measures being implemented.
- No consideration of the potential for current PFC impacts has been included in the assessments conducted to date. It is understood that fire-fighting foams containing PFCs are unlikely to be employed during construction and operation of the airport. However there are a number of other products (e.g. hydraulic fluids) which may be used during airport operation which may contain PFCs and thus a robust understanding of baseline conditions will assist in future monitoring of these compounds.

POTENTIAL RISKS ASSOCIATED WITH CONSTRUCTION AND OPERATION

As outlined in **Section 7.1.3**, there is potential for groundwater contamination to occur as a result of a range of activities which would be undertaken during construction and operation of the proposed airport. Appropriate management and mitigation measures should be implemented to reduce the potential for these risks to be realised. It is therefore important to obtain a robust baseline groundwater dataset for the following reasons:

- to inform management and mitigation measures, to prevent exposure of construction and operation workers to existing groundwater contaminants, if present;

- to enable appropriate monitoring of groundwater quality during construction and operation to determine whether management and mitigation measures are appropriate; and
- to inform future decisions with regard to airport operations.

7.2 Existing Surface Water Conditions

The following information provides an overview of the surface water conditions in waterways within and surrounding the proposed airport site. Information has been sourced primarily from the Surface Water Quality Report prepared by GHD (2015) which provides a review of previous groundwater and surface water investigations conducted to inform the environmental impact statement (EIS) prepared in 1997. Additional surface water sampling has been conducted in 2014 and 2015, however this information was not available for this risk assessment.

7.2.1 Identification of Existing Water Bodies

Two main catchments have been identified by GHD to drain the site: South Creek Catchment and Nepean River Catchment. The following is a summary of the waterways which intersect the proposed airport site:

- Duncans Creek (located to the south-west of the site) drains to the Nepean River (west of the site);
- Oaky Creek (drains the central and northern area of the site) drains to Cosgroves Creek;
- Cosgroves creek (flows along to the north-west/north of the site) drains to South Creek to north-east of the site;
- Badgerys Creek (flows along the southern and south-eastern boundary of the site) drains to South Creek to the north-east of the site; and
- Thompsons Creek (located to the south-east of the site) drains to South Creek to the south-east of the site.

Each of the above listed creeks and rivers have a number of small tributaries which drain the site and areas immediately surrounding the site. GHD indicate that previous investigations have determined that the creeks which drain the site and surrounding area may not flow continuously, and that during dry periods only intermittent pools of water may remain along the creek beds.

There are a number of major water supply catchments located in the area surrounding the site. Warragamba Dam is located approximately 14 km west of the site and is one of Sydney's major drinking water supply dams. Prospect Reservoir is located approximately 8 km north-east of the site. Prospect Reservoir is a potable water supply dam which is used during periods of high demand (NSW SCA, 2015).

The rural setting of the site is currently such that GHD have noted that there are numerous farm storage dams present at and surrounding the site. GHD also noted the presence of a very large storage dam, located to the south-west of the site at the head waters of Duncans Creek. It is assumed this dam is used for agricultural purposes including irrigation as it is located adjacent a large agricultural facility.

7.2.2 Use of Surface Water in the Study Area

Review of the land uses in the area at and surrounding the site has identified the following uses of surface water (note these are the main identified surface water uses, however there may be other uses of surface water in the area):

- Agriculture (stock water and irrigation) – mostly farm dams
- Ecological habitat
- Potable domestic supply (Warragamba Dam and Prospect Reservoir)
- Non-potable domestic uses
- Collection of rain water for drinking and other purposes in surrounding rural residential areas
- Industrial use (a few large scale industrial operations in the area are observed to have large dams adjacent their facilities which indicates that this water may be used for industrial purposes)

Surface water quality must be protected during construction and operation of the airport to ensure that the above listed beneficial uses of surface water are not impacted.

7.2.3 Surface Water Environmental Values

According to the NSW DEC (2006) '*Environmental values are those values or uses of water that the community believes are important for a healthy ecosystem – for public benefit, welfare, safety or health.*'

The NSW DEC (2006) (now NSW EPA) define the following 'water quality objectives' (WQOs) which should be considered when assessing the potential impacts of activities on waterways:

- Aquatic ecosystems
- Aquatic foods (cook before eating)
- Drinking water at the point of supply
- Homestead water supply
- Irrigation water supply
- Livestock water supply
- Primary contact recreation
- Secondary contact recreation
- Visual amenity.

The above WQOs are intended to provide goals which will assist in selecting the most appropriate management options. The guiding principles of the WQOs are (NSW DEC, 2006):

- Where the environmental values are being achieved in a waterway, they should be protected, and
- Where the environmental values are not being achieved in a waterway, all activities should work towards their achievement over time.

Based on available information, the following WQOs (herein referred to as EVs) are considered to be relevant to waterways at the site:

- Aquatic ecosystems
- Aquatic foods (cook before eating)
- Irrigation water supply
- Livestock water supply
- Primary contact recreation
- Secondary contact recreation
- Visual amenity.

Drinking water at the point of supply is not considered a relevant environmental value of most waterways at and surrounding the site, however as noted in **Section 7.2.3** there are two waterways in the area surrounding the site which are currently used for drinking water supply purposes (e.g. Warragamba Dam and Prospect Reservoir). The potential for the proposed airport activities to impact on nearby municipal water supplies sourced from these storages has been assessed as there is potential for air emissions to impact these waterways.

7.2.4 Sources of Potential Surface Water Contamination

The potentially contaminating activities identified to be currently occurring at the site and which may occur during construction and operation of the site are considered to also have the potential to result in surface water contamination (**Section 7.1.4**).

In addition there is the potential for increased loading of suspended particles to surface water during construction activities. Air modelling of dust as PM_{10}, $PM_{2.5}$ and TSP conducted for construction and operation of the airport has shown that the impact on both Warragamba Dam and Prospect Reservoir is very low with an annual average increase at Warragamba for PM_{10} of 0.02 µg/m^3. This would not have any impact on surface water quality.

On rare occasions, aircraft may be required to jettison fuel to enable a safe landing in an emergency situation. Such incidents are managed under the direction of air traffic control and would be expected to occur at a considerable distance from the airport site and at high altitude to avoid the risk of fuel reaching the ground.

Aircraft movements at the site and during operation would also result in increased potential for deposition of particulates and aircraft emissions in surrounding waterways. Further qualitative discussion is provided in **Section 7.2.8**.

Through consultation on the EIS there have been concerns raised by the community about the potential for aircraft emissions to impact on the quality of tank water in the area close to the airport. Air dispersion modelling has been conducted and predicted ground level concentrations of VOCs and PM_{10} in areas close to the airport site. This is discussed further in the Local Air Quality Report (Pacific Environment, 2015). The predicted ground level concentrations of benzene and other VOC are very low. The maximum predicted concentration of benzene within 5 km of the airport site is 0.1 µg/m^3 and diesel particles 0.8 ng/m^3. These concentrations are very low and would not impact of the quality of tank water.

7.2.5 Hazard Identification

The ANZECC (2000) guidelines set out key 'indicators' which can be used to measure whether there is a potential risk to each environmental value. Indicators (or guidelines) have been selected based on the appropriate level of protection for the waterways at and surrounding the site, and provide a risk-based approach to assessing the potential for risks to environmental values. **Table 48** outlines the hierarchy of guideline sources from which indicator values have been selected as per ANZECC (2000), NEPC (2013) and enHealth (2012).

Surface water samples collected to date have not been compared to drinking water screening criteria as the waterways which have been sampled are not currently used for drinking water purposes. Qualitative consideration of the potential for airport construction and operation to result in impacts to potable water sources has been undertaken in **Section 7.2.8**.

Table 48: Adopted Surface Water Guidelines

Environmental Value	Guideline Hierarchy
Aquatic ecosystems	- ANZECC (2000) Freshwater Quality Criteria (95% Protection) (e) - ANZECC (2000) Freshwater Quality Criteria (Medium to Low Reliability) - USEPA (2013) Ambient Water Quality Criteria (Chronic – Freshwater) - USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria - USEPA (2003) Region 5 RCRA Ecological Screening Levels (water) - RIVM (2001) Dutch Groundwater Intervention Value
Aquatic foods	As per the ANZECC (2000) guidance, where surface waters are used for aquaculture purposes, contaminant concentrations in the tissue of the organisms must meet Australia New Zealand Food Standards. As such no surface water screening criteria have been adopted as part of the current assessment.
Primary and secondary contact recreation (a)	- NHMRC (2011) Drinking Water Guidelines (as amended 2013) - WHO (2011) Drinking Water Guidelines - WHO (2008) Petroleum Products in Drinking Water - USEPA (2015) Regional Screening Level – Tap Water
Agricultural water (b) (d)	- ANZECC (2000) Stock Water Screening Criteria - ANZECC (2000) Irrigation – Long Term Value (LTV) (c) - NHMRC (2011) Drinking Water Guidelines (as amended 2013) - WHO (2011) Drinking Water Guidelines - WHO (2008) Petroleum Products in Drinking Water - USEPA (2015) Regional Screening Level – Tap Water

(a) As per NHMRC (2008) Guidelines for managing risk in recreational waters, all drinking water guidelines have been multiplied by a factor of 10x based on the assumption that recreational exposure would result in a lower incidental intake (0.2 L) of water than direct ingestion of drinking water (assumed to be 2 L/day).

(b) Agricultural water is considered to consist of livestock water and irrigation uses.

(c) The long term value for irrigation has been selected as a conservative approach as it is unclear how long irrigation activities using groundwater have been conducted for.

(d) Drinking water guidelines have been selected where no ANZECC Stock Watering guideline is available, as per ANZECC (2000).

(e) A 95% species protection value has been selected as the area is considered to be moderately disturbed, and thus 95% is likely to provide a suitable level of protection for current and proposed future land uses.

7.2.6 Identification of Contaminants of Potential Concern

Surface water data from samples collected in 1996, 1998, 2014 and 2015 were compared to selected screening criteria (as per NSW DEC (2006) and ANZECC (2000)) from guideline sources as listed in **Table 45**. The analytical data screening is presented in **Table A2 and Table A3** in **Appendix A**. **Table 49** below provides a summary of CoPC (from all sampled waterways) which were reported at concentrations which exceeded the adopted screening criteria.

Table 49: Surface Water Chemicals of Potential Concern

Environmental Value	Chemicals of Potential Concern
Aquatic ecosystems	- Cadmium - Copper - Iron - Mercury (one sample only) - Zinc - Phosphorus - Anionic surfactants as MBAs (see discussion below) Reported electrical conductivity (EC) in samples from Badgerys Creek, Oaky Creek (one sample) and Cosgroves Creek were generally above the range identified for lowland river systems (ANZECC, 2000). According to GHD (2015) the elevated EC is expected to be due to the influence of the surrounding shale geology.
Primary and secondary contact recreation	- Phosphorus
Agricultural water	- Iron (irrigation only) - Mercury (stock watering only) - Phosphorus - TPH C15-C28/C16-C34 - TPH C29-C36
MBA = Methylene blue activated substances (considered, by ANZECC (2000), to include linear alkylbenzene sulfonates [LAS]) TPH = Total Petroleum Hydrocarbons	

Anionic surfactants as methylene blue activated substances were analysed in surface water samples collected in 1996, 1998, 2014 and 2015. In 1996, concentrations ranged between 12 mg/L (Duncans Creek) and 16 mg/L (Cosgroves Creek and Thompsons Creek), in 1998, concentrations ranged between <0.05 – 0.21 mg/L. At locations which were sampled during both 1996 and 1998 sampling events, reported concentrations of anionic surfactants as MBAs were between one and three orders of magnitude lower in samples collected in 1998 compared to results from 1996. Samples collected in 2015 correlated more closely with results obtained in 1998. The EIS conducted in 1997 states that

> 'No substantial rain had fallen for four months and western Sydney was officially declared "in drought". In April 1998, the drought ended with substantial rainfall over the following winter.'

The drought conditions may have caused the marked difference between anionic surfactants (as MBAs) concentrations in 1996 compared to other years. However, no other analytes were reported to have been affected to such an extent.

7.2.7 Potential Exposure Pathways

For the purpose of the current assessment, it is assumed that there is potential for contaminants to be present in surface water, and thus the potential exposure points and exposure routes for surface water have been considered further and are listed in **Table 50** below.

Table 50: Surface Water Exposure Pathway Linkages

Receptor	Exposure Point	Exposure Route
Human users of extracted surface water (e.g. farmers, residents etc.)	Tap or hose	- Ingestion of surface water as a potable water supply (c) - Incidental ingestion of contaminants in surface water during irrigation and other domestic activities - Dermal contact with contaminants in surface water during irrigation and other domestic activities - Inhalation of surface water derived vapours at the point of extraction (b) - Ingestion of contaminants accumulated in food crops irrigated with impacted surface water
Human users of surface water bodies (e.g. recreational receptors)	Surface water body	- Incidental ingestion of contaminants in surface water - Dermal contact with contaminants in surface water - Inhalation of surface water derived vapours
Aquatic and terrestrial (e.g. birds) organisms	Surface water body	- Ingestion of contaminants in surface water - Direct contact with contaminants in surface water - Ingestion of accumulated contaminants in the food chain (a)

(a) This exposure route may have wider implications where contaminants in surface water have the potential to bioaccumulate within the food chain. Bioaccumulation of contaminants in aquatic organisms may also result in biomagnification in higher order organisms (e.g. birds which consume aquatic organisms).

(b) Volatile contaminants have not been detected in surface water during sampling undertaken to date, however this exposure pathway should be considered where there is potential for surface water impacts from volatile contaminants during construction and operation of the airport.

(c) This exposure pathway is considered to be less common than the use of private tank water storages for potable water. However, consideration of the potential for current residents to be extracting water from farm dams for potable uses should be made.

7.2.8 Risk Characterisation

For the purposes of this assessment, a qualitative evaluation of the potential for risks to identified receptors under current conditions at the site and in surrounding areas has been conducted. There are limited surface water data available which can be used to assess risks under proposed future (construction and operation of the airport) conditions at the site. However, based on available information there is potential for the construction and operation of the airport to impact the environmental values of surface water. These potential risks are discussed further below.

CURRENT POTENTIAL RISKS

The currently available information indicates that there is potential risk to the following environmental values of surface water at the site and in the surrounding area:

- Aquatic ecosystems
- Primary and secondary contact recreation
- Agricultural water

There is no information available which can be used to determine whether the identified CoPC are a result of naturally elevated background concentrations in the area or as a result of a current contamination source. Further investigation is required to assess the potential risk to surface water bodies at and surrounding the site under current conditions. This will provide a baseline to assess any impacts from the airport operation and construction and identify key mitigation measures to minimise these risks.

POTENTIAL RISKS ASSOCIATED WITH CONSTRUCTION AND OPERATION

The proposed activities during construction and operation of the proposed airport that have the potential to impact surface water are as follows:

- increased loading of suspended particulates in surface run-off as a result of earthmoving activities and/or increased vegetation clearing
- increased potential for accidental spill of stored chemicals or fuels from construction or operation vehicles which may be released to nearby surface water environments;
- potential for release of stored groundwater which has not been adequately characterised (with regard to contamination concentrations) to surface water bodies;
- potential for fuel jettisoning to occur in emergency situations; and
- potential for vehicle and aircraft emissions to deposit in nearby surface water bodies which may result in increased contaminant loading to waterways.

Modelling of air toxics and particles from vehicles, ground based operations and aircraft overflights indicates that the levels of these pollutants near surface waters is low and therefore the resultant risk will also be very low.

Based on currently available information with regard to construction and operation of the proposed airport, there is a potential risk to surface waters from the other sources outlined above. However the implementation of management and mitigation measures through the construction environmental management and the airport environmental management plan once the airport is in operation will minimise these risks.

POTENTIAL FOR IMPACTS TO POTABLE WATER SUPPLIES

Although the proposed airport is not located within the catchment area for Warragamba Dam and Prospect Reservoir, there is the potential for aircraft flights over these areas. These waterways are major potable water storages which supply water to Sydney's municipal reticulated water system. The following activities could potentially result in impacts to these water storages:

- **Increased dust emissions** during construction activities which have the potential to result in increased deposition of particulates to the surrounding area. Dust management strategies implemented as part of the environmental management plan for construction will minimise this risk. Results for air dispersion modelling for PM_{10} conducted as part of this EIS show that the annual average concentrations at Warragamba are predicted to be 0.02 µg/m^3 which would have no impact on water quality at the Warragamba Dam. The Prospect Reservoir is approximately 8 km from the site. PM_{10} modelling for receptors close to the reservoir show that there is predicted to be a maximum annual average increase of 0.09 µg/m^3 during construction activities which would not affect water quality.
- **Surface water discharges**: The distance from the site, the topography of the area, and the number of waterways present between the site and the water supply storages would reduce the potential for overland flow of impacted surface run-off from the site. These factors are also considered likely to limit the potential for discharge of site-derived groundwater impacts to these water bodies. However, the management and mitigation measures contained in the construction environmental management plan will ensure that site-derived contaminants are not able to discharge to these waterways. Implementation of these measures will minimise any risk to surface waters and subsequently to people who may come in contact with these waters.
- **Fuel Jettisoning**: Instances of fuel jettisoning for commercial aircraft are very rare and only occur in emergency situations where an unscheduled landing is required. Where fuel jettisoning is considered necessary, the pilot is required to contact air traffic control to alert them of the need to undertake fuel jettisoning and is required to take any reasonable precautions to

ensure the safety of people and property on the ground and in the air, and where possible, jettison fuel at a minimum altitude of 6,000 ft. Most fuel is considered to evaporate within 100 metres with only a small amount of fuel likely to reach ground level (if at all). There are no recorded instances in Australia of fuel reaching the ground after a fuel jettison event.

- Most aircraft which fly shorter haul domestic routes do not have the capacity to jettison fuel as they can land fully fuelled, however larger aircraft used on longer domestic routes and international long haul flights do have the capacity to jettison fuel.
- Based on existing protocols and the available information, it is considered unlikely that fuel jettisoning would result in impacts to surface water bodies surrounding the proposed airport site including Warragamba Dam, Prospect Reservoir and private water storages (e.g. tanks).
- **Aircraft emissions**: There is potential that emissions from increased aircraft traffic during operation of the proposed airport may impact on water quality in Warragamba Dam and Prospect Reservoir (as well as other waterways in the area) as well as private tank water supplies. Consideration should be given to a pre-construction and post construction/operation monitoring program to test the quality of local tank water and monitor any changes to the quality of the water over time. This would enable informed decisions to be made about implementation of any mitigation measures in a timely manner.

7.3 Conclusions and Recommended Mitigation Measures

The groundwater and surface water risk assessment has been undertaken in accordance with the ASC NEPM (NEPC, 2013) and provides a preliminary assessment of the potential risks posed by the construction and operation of the proposed airport to groundwater and surface water receptors.

The 1997 EIS identified the following potential risks to groundwater and surface water as a result of the construction and operation of the proposed airport:

- 'Airport development would result in a lowering of groundwater levels due to reduced infiltration in paved areas. This effect may be locally counteracted by rising levels beneath proposed detention dams.'
- 'There is a possibility of groundwater contamination from a number of sources; however properly engineered surface water drainage facilities and fuel storage and delivery systems would protect it from obvious sources of pollution.'
- A range of potential impacts to local surface water bodies as a result of construction and operation of the proposed airport were also identified.

These risks are still valid and are consistent with the outcomes of the current risk assessment. Based on available data, with regard to existing groundwater and surface water conditions at the site, there is potential for risks to current users of groundwater without mitigation measures in place. The following management and mitigations measures are proposed in the surface and groundwater studies of this EIS:

- Any storage of material such as chemicals, fuel or concrete components should be bunded to contain any spills. Procedures should also be established to clean up spills as quickly as possible to reduce the potential for groundwater or surface water impacts.
- Dust control should be managed through the use of water sprays and stabilising or covering of stockpiles.
- Implementation of sediment and erosion control measures to reduce the potential for surface run-off to reach surface water bodies.
- Construction of suitable drainage and detention ponds (with gravel filter beds at discharge points) to prevent direct surface water run-off to waterways and thus prevent deposition of silt and increased turbidity.
- Monitoring of discharge from retention ponds to mitigate the potential for impacts to surface water quality in the receiving environment.

- Dosing of detention ponds with flocculants when monitoring detects excessive nutrients being discharged from the system, this will increase the sediment and nutrient removal from stormwater prior to release.
- Installation of gross pollutant traps to intercept and retain coarse sediment, rubbish and debris in storm water.
- Installation of flame traps to limit the release of oils and fuels in stormwater.
- Installation and maintenance of a waste water treatment system which incorporates: tertiary processes, includes disinfection and a high level of nutrient removal. It is noted that where reverse osmosis is proposed as a filtration system in the waste water treatment, monitoring for perfluorinated compounds should be conducted as these compounds are of a size which can reduce the efficiency of reverse osmosis filters.
- Ongoing monitoring of waste water discharges and the adequacy of chemical storage bunding as well as other management measures should be undertaken throughout construction and operation phases at the proposed airport.

With the implementation of mitigation measures described in the related technical reports (surface water, water quality and groundwater), the potential risks would be minimised.

7.4 Limitations of the Groundwater and Surface Water Assessment

The current risk assessment is based on limited data and provides a high level assessment of the potential for risks to groundwater and surface water receptors at and surrounding the site. The outcomes of the risk assessment indicate the need for further investigation into the current status of groundwater and surface water, especially with regard to spatial and temporal trends to ensure that a well-defined baseline is established to enable any changes in environmental quality due to airport construction and operation to be detected and mitigated.

8 CONCLUSIONS

A health risk assessment (HRA) has been undertaken to assess the potential risks associated with air and noise emissions and potential surface and groundwater contamination that may arise from the construction and operation of the proposed Western Sydney Airport at Badgerys Creek. Overall, the analysis has found that the health impacts of the proposed airport would be in line with national and international standards of acceptability.

Air Quality

The HRA has examined the increase in risk resulting from air pollution generated by the construction and operation of the proposed airport. The pollutants considered were particulate matter, NO_2, SO_2, CO, benzene and diesel. The prediction of changes in local air quality were derived from the local air quality assessment (Pacific Environment 2015) which is included elsewhere in the EIS. A limited assessment was also conducted for ozone as a regional air pollutant. Based on existing evidence, the health impacts associated with the emissions from the airport are expected to represent a small increase in current rates of health impacts associated with existing air pollution in the Sydney basin.The highest risk during construction is associated with PM_{10} during terminal construction. The highest risk could result in an additional 2 deaths per hundred years from all causes (non-accidental). The most affected areas would be Luddenham, Bringelly, Kemps Creek and Badgerys Creek.

For the 2030 and 2063 scenarios the results of the HRA show that the highest risk is associated with NO_2 with increases in mortality and hospital admissions predicted. The most affected areas are likely to be Luddenham, Bringelly, Kemps Creek, Mulgoa, Wallacia and Rossmore. The risks from PM_{10} and $PM_{2.5}$ are also at the high end of acceptable risk criteria established by international agencies. The risks from all other pollutants assessed is low and within acceptable risk criteria.

For regional air quality, the increase in risk of mortality and hospital admissions related to changes in ozone are small and within acceptable risk criteria. There are limitations to this assessment as the analysis, due to availability of air quality data, is limited to days when exceedances of the ozone standards are predicted. The assessment does not evaluate the full risk that may be experienced by changes in ozone levels due to airport operations.

Mitigation measures to reduce NO_2 and PM are included in the local air quality technical report and when implemented, would have an effect of reducing NO_2 emissions generated. It is also worth noting that particularly for the 2063 scenario assessment, the modelling assumed no future reductions in emissions technology either for aircraft or for vehicular traffic more generally. On the basis that emissions reduction has occurred over the past several decades and is expected to occur again in the future, the results of the HRA are likely to overestimate the actual level of risk that would be realised.

Noise

The noise HRA was conducted for the two categories of noise generated by: aircraft overflights and ground-based noise sources. The prediction of changes in the noise environment were derived from the noise assessments by Wilkinson Murray included in the EIS and additional data provided specifically for the HRA.

Three health outcomes were assessed – sleep disturbance (as awakenings), increases in ischaemic heart disease and impacts on cognitive development and learning in children. The results for the HRA for noise shows that ground based operations may lead to an increase in sleep disturbance (assessed as awakenings), increases in risk of cardiovascular disease and delays in childhood learning and cognitive development. These effects are predicted for suburbs close to the airport site, in particular Luddenham. The impact of aircraft overflights is lower than that for ground based operations. The risk of sleep disturbance and impacts on cognitive development are predicted to be higher for 2063 than 2030.

Based on the results of the noise assessment, the risk posed to the health of the exposed communities is generally low. No increase in cardiovascular outcomes is likely from aircraft noise as the predicted night noise levels are below the threshold for adverse effects. However, ground operations noise is predicted to be above the threshold value of 55 dB Lnight and may lead to a 10% increase in myocardial infarctions in Luddenham in 2063 if not mitigated.

A significant increase in EEG awakenings is predicted especially in Luddenham. In 2030, it is predicted that there would be 190 additional EEG awakenings per person per year and an additional 400 EEG awakenings per person per year with ground-based noise in 2063. For full awakenings, the number is lower but still highest in Luddenham. There is a greater predicted impact from ground- operations noise than aircraft noise. Mitigation measures in the noise assessment report should be implemented and would reduce this impact.

Impacts on children's learning and cognitive development are predicted to be within acceptable risk levels for most locations for aircraft noise. The impact of ground operations noise is more substantial. Mitigation measures should be implemented at these locations to reduce this risk to within acceptable levels.

The EIS noise assessments include mitigation and management measures which will reduce the potential impacts which have been assessed by the HRA. Particularly important, the Department will in future stages of assessment, consider the results of the EIS – both the noise technical study as well as the health risks identified in this report and incorporate the findings into the further analysis of airport operating mode options before determining the preferred flight tracks. Additionally, the Department would develop a noise amelioration strategy considering both overflight and ground-based noise sources with a range of mitigation options included for the most affected areas in accordance with AS2021.

Surface and groundwater

There are 42 registered groundwater bores within a five kilometre radius of the centre of the site. Twelve of these bores are registered as being used for domestic, stock, industrial, farming and irrigation purposes. The depth of registered extraction bores indicates that the majority of groundwater users extract water from the Bringelly Shale and Hawkesbury Sandstone aquifers. The salinity in the Alluvial and Bringelly Shale aquifers is reported to be >1,000 mg/l (based on 1995-1998 data) and thus is considered unsuitable for potable uses.

The site has historically been used for a wide range of agricultural, industrial, commercial and rural-residential activities from generate a range of potential contaminants in soil and groundwater. Based on these historical site activities and the potential contaminants which may be associated in future with construction and operation of the airport, there is likely to be considerable overlap of contaminants potentially present. It is therefore important that baseline groundwater data is collected including all potential contaminants that may be already present to enable identification of the current baseline conditions and from which to monitor future performance of the airport.

The contaminating activities which may occur during construction and operation of the airport may also have the potential to result in surface water contamination. Aircraft movements at the site during operation may result in increased deposition of particulates in surrounding waterways. Results of air dispersion modelling has shown that these impacts are predicted to be low. In addition, it is unlikely that fuel jettisoning would have an impact on surface water bodies around the airport site given the rarerity of fuel jettisoning, existing protocols in place, and the evaporative nature of aviation fuel.

Based on currently available information with regard to construction of the proposed airport, there is considered to be risks to environmental values of surface water. However these risks are not unique or unusual to the proposed airport and are common for other major infrastructure projects where standard construction measures are typically effective in reducing risks. Implementation of mitigation

measures through the construction environmental management plan and the site EMP during operation will minimise these risks.

The outcomes of the risk assessment suggest that there is a need to collect additional data to fill identified data gaps and enable a more robust assessment of current and potential future risks to groundwater and surface water receptors. Consideration should be given to a pre-construction and post construction/operation monitoring program to test the quality of local tank water and monitor any changes to the quality of the water over time. This would enable informed decisions to be made about implementation of any mitigation measures in a timely manner.

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Appendix A DATA TABLES

Table A1 - Selected Groundwater Screening Criteria

Chemical Name	Units	Primary Contact Recreation	Reference	Stock Watering	Reference	Irrigation	Reference	Freshwater Ecosystem	Reference
Alkalinity (Carbonate as CaCO3)	mg/L	-	-	-	-	-	-	-	-
Bicarbonate as CaCO3	mg/L	-	-	-	-	-	-	-	-
Electrical Conductivity (lab)	uS/cm	-	-	-	-	-	-	125-2200	ANZECC (2000) Lowland River Trigger Values
pH (Lab)	pH Units	-	-	-	-	-	-	6.0-8.0	ANZECC (2000) Lowland River Trigger Values
Aluminium	mg/L	200	USEPA (2015) Tap Water x 10	5	ANZECC (2000) Stock Watering	5	ANZECC (2000) Irrigation LTV	0.055	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Cadmium	mg/L	0.02	NHMRC (2011) Drinking Water Guidelines x 10	0.01	ANZECC (2000) Stock Watering	0.01	ANZECC (2000) Irrigation LTV	0.0002	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Copper	mg/L	20	NHMRC (2011) Drinking Water Guidelines x 10	0.4	ANZECC (2000) Stock Watering	0.2	ANZECC (2000) Irrigation LTV	0.0014	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Iron	mg/L	140	USEPA (2015) Tap Water x 10	14	USEPA (2015) Tap Water Guidelines	0.2	ANZECC (2000) Irrigation LTV	0.3	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Lead	mg/L	0.1	NHMRC (2011) Drinking Water Guidelines x 10	0.1	ANZECC (2000) Stock Watering	2	ANZECC (2000) Irrigation LTV	0.0034	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Zinc	mg/L	60	USEPA (2015) Tap Water x 10	20	ANZECC (2000) Stock Watering	2	ANZECC (2000) Irrigation LTV	0.008	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Calcium	mg/L	-	-	1000	ANZECC (2000) Stock Watering	-	-	116	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Chloride	mg/L	-	-	-	-	-	-	230	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Magnesium	mg/L	-	-	-	-	-	-	82	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Potassium	mg/L	-	-	-	-	-	-	53	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Sodium	mg/L	-	-	-	-	-	-	680	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Sulphate	mg/L	5000	NHMRC (2011) Drinking Water Guidelines x 10	2000	ANZECC (2000) Stock Watering	-	-	-	-
Total Kjeldahl Nitrogen	mg/L	-	-	-	-	-	-	-	-
Nitrate (as N)	mg/L	-	-	-	-	-	-	-	-
Nitrogen (Total)	mg/L	-	-	-	-	5	ANZECC (2000) Irrigation LTV	0.2-0.3	ANZECC (2000) Lowland River Trigger Values
Nitrate + Nitrate as N	mg/L	-	-	-	-	-	-	-	-
Phosphorus	µg/L	0.004	USEPA (2015) Tap Water x 10	0.0004	USEPA (2015) Tap Water Guidelines	0.05	ANZECC (2000) Irrigation LTV	0.01	ANZECC (2000) Lowland River Trigger Values

Table A2 - Historical Groundwater Analytical Data

Adopted Screening Criteria	Compounds / Units	Alkalinity (Carbonate as CaCO3) mg/L	Bicarbonate as CaCO3 mg/L	Electrical Conductivity (lab) µS/cm	pH (Lab) pH Units	Aluminium mg/L	Cadmium mg/L	Copper mg/L	Iron mg/L	Lead mg/L	Zinc mg/L	Calcium mg/L	Chloride mg/L	Magnesium mg/L	Potassium mg/L	Sodium mg/L	Sulphate mg/L
Primary Contact Recreation		-	-	-	-	200	0.02	20	140	0.1	60	-	-	-	-	-	5000
Stock Watering		-	-	-	-	5	0.01	0.4	14	0.1	20	1000	-	-	-	-	2000
Irrigation		-	-	-	-	5	0.01	0.2	0.2	2	2	-	-	-	-	-	-
Freshwater Ecosystem Protection		**-**	**-**	***125-2200***	***6.0-8.0***	***0.055***	***0.0002***	***0.0014***	***0.3***	***0.0034***	***0.008***	***116***	***230***	***82***	***53***	***680***	**-**
Location Code	**Well Depth (m)**	**Date Sampled**															
GW A	27.3	29/09/1998	<1	188	***29,700***	7.43	<0.01	<0.001	***0.013***	<1	***0.004***	***0.039***	***518***	***9660***	***186***	56	***5440***	4
GW B	38.5	29/09/1998	<1	921	***28,500***	6.72	<0.01	<0.001	***0.02***	<1	***0.005***	***0.042***	***650***	***9450***	***573***	***96***	***4600***	4
GW C	26	29/09/1998	<1	866	***5,520***	7.65	***0.06***	<0.001	***0.002***	<0.1	***0.022***	***0.014***	42	***1120***	51	15	***935***	9
GW E	Deep (11.3)	29/09/1998	<1	1140	***7,900***	7.42	<0.01	<0.001	***0.002***	<0.1	***0.006***	***0.012***	62	***1670***	***163***	11	***1260***	152
GW E	Shallow (5.0)	29/09/1998	<1	777	***3,990***	7.21	0.04	<0.001	***0.002***	<0.1	0.001	0.008	106	***648***	***113***	7	488	93
GW F	Deep (30.3)	29/09/1998	<1	621	***20,970***	7.08	<0.001	<0.001	***0.003***	<0.1	0.003	***0.009***	***291***	***6560***	***516***	36	***3260***	85
GW F	Shallow (6.0)	29/09/1998	<1	1060	***40,800***	7.02	0.01	<0.001	***0.004***	<0.1	0.003	***0.017***	***181***	***13,800***	***1500***	13	***6560***	390
GW G	Deep (24.3)	29/09/1998	<1	687	***23,900***	6.94	<0.01	<0.001	***0.008***	<1	***0.004***	***0.018***	***446***	***7740***	***464***	53	***3970***	1
GW G	Shallow (5.0)	29/09/1998	<1	630	***14,390***	7.5	0.02	<0.001	***0.003***	<0.1	0.001	0.006	***145***	***4070***	***364***	6	***2160***	249
GW Well G1a		15/06/1995	-	-	***17,700***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW Well G3a		15/06/1995	-	-	***22,190***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW G4a	-	15/06/1995	-	-	***36,600***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW G5	-	15/06/1995	-	-	***31,200***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW G6	-	15/06/1995	-	-	***28,000***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW G7	-	15/06/1995	-	-	***28,800***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW H	Deep (12.3)	29/09/1998	<1	862	***33,600***	6.98	<0.01	<0.001	***0.006***	<1	***0.007***	***0.014***	***237***	***10,600***	***1080***	31	***5370***	1080
GW H	Duplicate (4.5)	29/09/1998	<1	157	-	-	0.04	<0.001	***0.004***	<1	<0.001	***0.025***	23	***8410***	***799***	12	***4280***	677
GW H	Shallow (4.5)	29/09/1998	<1	155	***26,450***	7.07	0.04	<0.001	***0.004***	<1	<0.001	***0.024***	25	***8380***	***812***	12	***4270***	657
GW J	Deep (42.3)	29/09/1998	<1	549	***26,300***	6.72	<0.01	<0.001	***0.014***	***1.8***	0.002	***0.037***	***605***	***8300***	***217***	***62***	***4560***	1
GW J	Shallow (4.5)	29/09/1998	<1	255	***28,000***	7.51	0.03	<0.001	***0.004***	<1	<0.001	***0.009***	54	***8810***	***744***	13	***4580***	494
GW K	32.3	29/09/1998	<1	939	***27,400***	7.03	<0.01	<0.001	***0.005***	<1	***0.015***	***0.018***	***282***	***8160***	***825***	***59***	***4180***	977
GW Well D11	10.05	13/06/1995	-	-	***14,810***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW Well D13	10.35	13/06/1995	-	-	1350	-	-	-	-	-	-	-	-	-	-	-	-	-
GW Well D8	10.05	13/06/1995	-	-	***3010***	-	-	-	-	-	-	-	-	-	-	-	-	-
GW Well D9	10.25	13/06/1995	-	-	***14,290***	-	-	-	-	-	-	-	-	-	-	-	-	-
MW2D	-	29/09/1998	<1	907	-	-	<0.01	<0.001	***0.019***	<1	***0.005***	***0.038***	***633***	***9290***	***574***	***91***	***4530***	3
MW2D Duplicate	-	29/09/1998	<1	893	-	-	<0.01	<0.001	***0.019***	<1	***0.005***	***0.041***	***675***	***9290***	***611***	***92***	***4400***	3

Table A2 - Historical Groundwater Analytical Data

Adopted Screening Criteria	Compounds	Total Kjeldahl Nitrogen	Nitrate (as N)	Nitrogen (Total)	Nitrate + Nitrate as N	Phosphorus
	Units	mg/L	mg/L	mg/L	mg/L	mg/L
Primary Contact Recreation		-	-	-	-	0.004
Stock Watering		-	-	-	-	0.0004
Irrigation		-	-	5	-	0.05
Freshwater Ecosystem Protection		**-**	**-**	***0.2-0.3***	**-**	***0.01***

Location Code	Well Depth (m)	Date Sampled					
GW A	27.3	29/09/1998	11.9	0.06	***12***	0.06	***0.06***
GW B	38.5	29/09/1998	12.4	0.03	***12.4***	0.03	***0.02***
GW C	26	29/09/1998	2.5	0.03	***2.5***	0.03	***0.42***
GW E	Deep (11.3)	29/09/1998	0.5	0.06	***0.6***	0.06	***0.08***
GW E	Shallow (5.0)	29/09/1998	3.9	9.75	***13.7***	9.78	***1.2***
GW F	Deep (30.3)	29/09/1998	5	0.03	***5***	0.03	***0.08***
GW F	Shallow (6.0)	29/09/1998	0.8	0.03	***0.8***	0.03	***0.53***
GW G	Deep (24.3)	29/09/1998	6.6	0.02	***6.6***	0.02	***0.04***
GW G	Shallow (5.0)	29/09/1998	1	0.02	***1***	0.02	***0.11***
GW Well G1a		15/06/1995	-	-	-	-	-
GW Well G3a		15/06/1995	-	-	-	-	-
GW G4a	-	15/06/1995	-	-	-	-	-
GW G5	-	15/06/1995	-	-	-	-	-
GW G6	-	15/06/1995	-	-	-	-	-
GW G7	-	15/06/1995	-	-	-	-	-
GW H	Deep (12.3)	29/09/1998	1.8	0.02	***1.8***	0.02	***0.09***
GW H	Duplicate (4.5)	29/09/1998	1	0.06	***1.1***	0.06	***0.46***
GW H	Shallow (4.5)	29/09/1998	1	0.06	***1.1***	0.06	***0.47***
GW J	Deep (42.3)	29/09/1998	11.7	0.05	***11.8***	0.05	***0.07***
GW J	Shallow (4.5)	29/09/1998	1.3	0.4	***1.7***	0.4	***0.54***
GW K	32.3	29/09/1998	4.5	0.03	***4.5***	0.03	***0.06***
GW Well D11	10.05	13/06/1995	-	-	-	-	-
GW Well D13	10.35	13/06/1995	-	-	-	-	-
GW Well D8	10.05	13/06/1995	-	-	-	-	-
GW Well D9	10.25	13/06/1995	-	-	-	-	-
MW2D	-	29/09/1998	13.7	0.04	***13.7***	0.04	***0.11***
MW2D Duplicate	-	29/09/1998	13.3	0.03	***13.3***	0.03	***0.06***

Table A3 - Selected Surface Water Screening Criteria

Chemical Name	Units	Primary Contact Recreation	Reference	Stock Watering	Reference	Irrigation	Reference	Freshwater Ecosystem	Reference
Anionic Surfactants as MBAs	mg/L	-	-	-	-	-	-	0.28	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Electrical Conductivity (lab)	uS/cm	-	-	-	-	-	-	125-2200	ANZECC (2000) Lowland River Trigger Values
pH (Lab)	pH Units	-	-	-	-	-	-	6.0-8.0	ANZECC (2000) Lowland River Trigger Values
Cadmium	mg/L	0.02	NHMRC (2011) Drinking Water Guidelines x 10	0.01	ANZECC (2000) Stock Watering	0.01	ANZECC (2000) Irrigation LTV	0.0002	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Chromium (III+VI)	mg/L	0.5	WHO (2011) Drinking Water Guidelines x 10	1	ANZECC (2000) Stock Watering	0.1	ANZECC (2000) Irrigation LTV	0.085	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Copper	mg/L	20	NHMRC (2011) Drinking Water Guidelines x 10	0.4	ANZECC (2000) Stock Watering	0.2	ANZECC (2000) Irrigation LTV	0.0014	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Iron	mg/L	140	USEPA (2015) Tap Water x 10	14	USEPA (2015) Tap Water Guidelines	0.2	ANZECC (2000) Irrigation LTV	0.3	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Lead	mg/L	0.1	NHMRC (2011) Drinking Water Guidelines x 10	0.1	ANZECC (2000) Stock Watering	2	ANZECC (2000) Irrigation LTV	0.0034	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Mercury	mg/L	0.01	NHMRC (2011) Drinking Water Guidelines x 10	0.002	ANZECC (2000) Stock Watering	0.002	ANZECC (2000) Irrigation LTV	0.0006	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Nickel	mg/L	0.2	NHMRC (2011) Drinking Water Guidelines x 10	1	ANZECC (2000) Stock Watering	0.2	ANZECC (2000) Irrigation LTV	0.011	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Zinc	mg/L	60	USEPA (2015) Tap Water x 10	20	ANZECC (2000) Stock Watering	2	ANZECC (2000) Irrigation LTV	0.008	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
C6 - C 9 Fraction	mg/L	150	WHO (2008) Petroleum Products in Drinking Water x 10	15	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C10 - C14 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C15 - C28 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C29 - C36 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
Benzene	mg/L	0.01	NHMRC (2011) Drinking Water Guidelines x 10	0.001	NHMRC (2011) Drinking Water Guidelines	-	-	0.95	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Toluene	mg/L	8	NHMRC (2011) Drinking Water Guidelines x 10	0.8	NHMRC (2011) Drinking Water Guidelines	-	-	0.18	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Ethylbenzene	mg/L	3	NHMRC (2011) Drinking Water Guidelines x 10	0.3	NHMRC (2011) Drinking Water Guidelines	-	-	0.08	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Xylene Total	mg/L	6	NHMRC (2011) Drinking Water Guidelines x 10	0.6	NHMRC (2011) Drinking Water Guidelines	-	-	0.013	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
PAHs (Sum of total) - Lab calc	mg/L	-	-	-	-	-	-	-	-
Phenols (a)	mg/L	58	USEPA (2015) Tap Water x 10	5.8	USEPA (2015) Tap Water Guidelines	-	-	0.32	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
1,2-dichlorobenzene	mg/L	15	NHMRC (2011) Drinking Water Guidelines x 10	1.5	NHMRC (2011) Drinking Water Guidelines	-	-	0.16	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Phosphorus	mg/L	0.004	USEPA (2015) Tap Water x 10	0.0004	USEPA (2015) Tap Water Guidelines	0.05	ANZECC (2000) Irrigation LTV	0.01	ANZECC (2000) Lowland River Trigger Values

(a) The value for phenol has been adopted as a conservative approach

Table A4 - Historical Surface Water Analytical Data

Adopted Screening Criteria	Compounds	Fraction Organic Carbon	Soxhlet Grease	MBAS	Dissolved Oxygen (% saturated) (Field) (Filtered)	Dissolved Oxygen (Lab) (Filtered)	Electrical conductivity (lab)	pH (Lab)	Total Suspended Solids	Turbidity	Cadmium	Chromium (III+VI)	Copper	Iron	Lead	Mercury	Nickel	Zinc	C6 - C 9 Fraction	C10 - C14 Fraction	C15 - C28 Fraction
	Units	%	mg/L	mg/L	%S	mg/L	µS/cm	pH Units	mg/L	NTU	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	µg/L	µg/L	µg/L
Primary Contact Recreation		-	-	-	-	-	-	-	-	-	0.02	0.5	20	140	0.1	0.01	0.2	60	15,000	900	900
Stock Watering		-	-	-	-	-	-	-	-	-	0.01	1	0.4	14	0.1	0.002	1	20	1500	90	90
Irrigation		-	-	-	-	-	-	-	-	-	0.01	0.1	0.2	0.2	2	0.002	0.2	2	-	-	-
Freshwater Ecosystem Protection		**-**	**-**	***0.28***	**-**	**-**	***125-2200***	***6.0-8.0***	**-**	**-**	***0.0002***	***0.085***	***0.0014***	***0.3***	***0.0034***	***0.0006***	***0.011***	***0.008***	**-**	**-**	**-**
Location ID	**Date Sampled**																				
Badgerys Creek B1 (BCUS)	04-12-96	10	<5	<0.02	63	4.5	1170	6.9	2	1.1	<0.005	<0.005	***0.005***	0.22	<0.005	<0.001	<0.005	***0.013***	<20	<40	<200
Badgerys Creek B2	04-12-96	25	<5	0.22	150	12.2	2900	7.3	33	7	<0.005	0.01	<0.005	***1.8***	<0.005	<0.001	<0.005	<0.01	<20	<40	<200
Badgerys Creek B3 (BCDS)	04-12-96	<0.01	<5	**14**	24	2.2	1080	6.7	14	5.1	<0.001	<0.01	***0.005***	<0.005	<0.005	<0.02	<0.005	<0.005	<40	<200	<200
Badgerys Creek B3 (BCDS)	17-09-98	9	<5	0.13	107	11.3	1530	6.9	9	46	***0.001***	<0.005	<0.005	***1.93***	<0.005	<0.001	<0.005	<0.01	<20	<20	<100
Badgerys Creek B3 (BCDS)	08-10-98	11	<5	0.16	13	1.4	1895	7	15	19	<0.001	<0.005	<0.005	***1.51***	<0.005	<0.001	<0.005	***0.02***	<20	<20	<100
Badgerys Creek B3 (BCDS)	15-12-98	17	8	<0.05	35	2.9	2250	7.2	24	19	<0.0005	<0.005	<0.005	***3.02***	<0.002	<0.001	<0.005	<0.01	<20	<20	<100
Cosgroves Creek C1 (CCUS)	10-12-96	<0.01	<5	**16**	25	2.2	1500	6.7	5	2.9	<0.001	0.018	<0.005	<0.005	<0.005	<0.02	<0.005	<0.005	<40	<200	<200
Cosgroves Creek C3	10-12-96	<0.01	<5	**16**	25	2.2	1500	6.7	5	2.9	<0.001	0.018	<0.005	<0.005	<0.005	<0.02	<0.005	<0.005	<40	<200	<200
Cosgroves Creek C3	16-09-98	12	<5	0.19	65	6.6	1220	6.9	12	16	***0.001***	<0.005	<0.005	***1.76***	<0.005	<0.001	<0.005	<0.01	<20	<20	<100
Cosgroves Creek C3	07-10-98	15	<5	0.12	53	4.9	1820	7	8	10	<0.001	<0.005	<0.005	***2.28***	<0.005	<0.001	<0.005	<0.01	<20	<20	<100
Cosgroves Creek C3	14-12-98	18	9	0.1	2	0.2	2150	7.4	6	16	<0.0005	<0.005	<0.005	***3.17***	<0.002	<0.001	<0.005	<0.01	<20	<20	<100
Duncans Creek D1	10-12-96	<0.01	<5	**12**	15	1.3	1750	6.7	13	5.2	<0.001	0.01	<0.005	<0.005	<0.005	<0.02	<0.005	<0.005	<40	<200	<200
Duncans Creek D1	17-09-98	10	<5	0.17	28	2.7	1330	7	10	12	***0.001***	<0.005	<0.005	***1.26***	<0.005	<0.001	<0.005	<0.01	<20	<20	<100
Duncans Creek D1	08-10-98	8	<5	0.06	24	2.2	1950	7	8	10	***0.001***	<0.005	<0.005	***1.29***	0.003	<0.001	<0.005	***0.04***	<20	<20	<100
Duncans Creek D1	15-12-98	10	<5	0.09	50	4.1	2590	7.1	9	6.2	<0.0005	<0.005	<0.005	***0.58***	<0.002	<0.001	<0.005	<0.01	<20	<20	<100
South Creek S1	16-09-98	12	<5	0.09	105	10.1	673	7.2	12	65	0.001	<0.005	***0.006***	***1.5***	<0.005	<0.001	<0.005	***0.01***	<20	<20	<100
South Creek S1	07-10-98	10	<5	0.2	83	7.4	1000	7.1	8	15	<0.001	<0.005	<0.005	***1.41***	0.002	<0.001	<0.005	***0.03***	<20	<20	<100
South Creek S1	14-12-98	5	<5	0.05	100	7.7	1600	7	56	38	<0.0005	<0.005	<0.005	***0.81***	<0.002	<0.001	<0.005	<0.01	<20	<20	<100
South Creek S2	16-09-98	11	<5	0.12	66	6.6	727	6.9	11	82	***0.001***	<0.005	***0.006***	***1.81***	<0.005	<0.001	***0.012***	***0.01***	<20	<20	<100
South Creek S2	07-10-98	9	<5	0.16	60	5.5	989	7	19	32	<0.001	<0.005	<0.005	***1.5***	<0.005	<0.001	<0.005	***0.01***	<20	<20	<100
South Creek S2	14-12-98	7	<5	<0.05	87	6.8	1420	6.8	5	7	<0.0005	<0.005	<0.005	***0.35***	<0.002	<0.001	<0.005	***0.16***	<20	<20	<100
South Creek S3	16-09-98	12	<5	0.13	79	7.5	690	6.9	12	40	***0.001***	<0.005	<0.005	***2.03***	<0.005	<0.001	<0.005	<0.01	<20	<20	<100
South Creek S3	07-10-98	11	<5	0.21	44	4.1	645	6.9	14	23	***0.001***	<0.005	<0.005	***3.06***	0.003	<0.001	<0.005	***0.02***	<20	<20	<100
South Creek S3	14-12-98	12	<5	<0.05	39	3.1	850	7.4	4	12	<0.0005	<0.005	<0.005	***0.66***	<0.002	<0.001	<0.005	***0.03***	<20	<20	<100
Thompson Creek T1	10-12-96	<0.01	<5	**16**	23	2.2	4960	6.4	5	4.9	***0.01***	0.018	***0.005***	<0.005	<0.005	<0.02	<0.005	<0.005	<40	<200	<200
Thompson Creek T1	17-09-98	11	<5	0.14	50	5.5	2400	7	11	10	***0.001***	<0.005	<0.005	***1.21***	<0.005	<0.001	<0.005	<0.01	<20	<20	<100
Thompson Creek T1	09-10-98	13	<5	0.21	15	1.5	4240	7	8	11	<0.001	<0.005	<0.005	***1.39***	<0.005	***0.01***	<0.005	***0.03***	<20	<20	<100
Thompson Creek T1	15-12-98	18	<5	0.17	46	4.2	9550	7.3	11	14	<0.0005	<0.005	<0.005	***1.16***	<0.002	<0.001	0.006	<0.01	<20	<20	<100

Table A4 - Historical Surface Water Analytical Data

Adopted Screening Criteria	Compounds	C29 - C36 Fraction	Benzene	Toluene	Ethylbenzene	Xylene Total	PAHs (Sum of total) - Lab calc	Phenols	1,2-dichlorobenzene	Ammonia as N	Total Kjeldahl Nitrogen	Nitrate (as N)	Nitrite (as N)	Nitrogen (Total)	Phosphorus
	Units	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
Primary Contact Recreation		900	10	8,000	3,000	6,000	-	58,000	15,000	-	-	-	-	-	0.004
Stock Watering		90	1	800	300	600	-	5,800	1,500	-	-	-	-	-	0.0004
Irrigation		-	-	-	-	-	-		-	-	-	-	-	-	0.05
Freshwater Ecosystem Protection		**-**	***950***	***180***	***80***	***13***	**-**	**-**	***160***	**-**	**-**	**-**	**-**	**-**	***0.01***
Location ID	**Date Sampled**														
Badgerys Creek B1 (BCUS)	04-12-96	<200	<1	<1	<1	<1	<1	<10	<1	0.054	5	<0.02	<0.02	-	<0.02
Badgerys Creek B2	04-12-96	<200	<1	<1	<1	<1	<1	<10	<1	0.38	2.9	<0.02	<0.02	-	***1.2***
Badgerys Creek B3 (BCDS)	04-12-96	<1	<1	<1	<1	<1	<1	1100	<1	0.21	0.7	<0.02	<0.02	0.12	***0.26***
Badgerys Creek B3 (BCDS)	17-09-98	<100	<1	<1	<1	<1	<1	<10	<1	0.14	1.1	1.16	0.03	2.3	***0.35***
Badgerys Creek B3 (BCDS)	08-10-98	<100	<1	<1	<1	<1	<1	<10	<1	0.16	1.3	0.01	<0.01	1.31	***0.47***
Badgerys Creek B3 (BCDS)	15-12-98	<100	<1	<1	<1	<1	<1	<10	<1	0.13	1.4	0.02	<0.01	1.42	***0.32***
Cosgroves Creek C1 (CCUS)	10-12-96	<1	<1	<1	<1	<1	<1	<50	<1	0.12	0.9	<0.02	<0.02	-	<0.02
Cosgroves Creek C3	10-12-96	<1	<1	<1	<1	<1	<1	<50	<1	0.12	0.9	<0.02	<0.02	<0.02	<0.02
Cosgroves Creek C3	16-09-98	<100	<1	<1	<1	<1	<1	30	<1	0.11	1.2	0.13	0.01	1.3	***0.07***
Cosgroves Creek C3	07-10-98	<100	<1	<1	<1	<1	<1	<10	<1	0.06	1.2	0.03	<0.01	1.23	***0.07***
Cosgroves Creek C3	14-12-98	<100	<1	<1	<1	<1	<1	<10	<1	0.7	1.7	<0.01	<0.01	1.7	***0.05***
Duncans Creek D1	10-12-96	<1	<1	<1	<1	<1	<1	530	<1	0.025	0.2	<0.02	<0.02	<0.02	<0.02
Duncans Creek D1	17-09-98	<100	<1	<1	<1	<1	<1	<10	<1	0.02	1.1	0.2	<0.01	1.3	***0.03***
Duncans Creek D1	08-10-98	<100	<1	<1	<1	<1	<1	<10	<1	<0.01	0.8	<0.01	<0.01	0.8	***0.04***
Duncans Creek D1	15-12-98	<100	<1	<1	<1	<1	<1	<10	<1	<0.01	0.5	<0.01	<0.01	0.5	***0.02***
South Creek S1	16-09-98	<100	<1	<1	<1	<1	<1	<10	<1	0.04	0.9	0.67	0.02	1.6	***0.14***
South Creek S1	07-10-98	<100	<1	<1	<1	<1	<1	<10	<1	0.08	1.2	0.14	<0.01	1.34	***0.12***
South Creek S1	14-12-98	<100	<1	<1	<1	<1	<1	<10	<1	0.24	0.3	0.19	<0.01	0.49	0.01
South Creek S2	16-09-98	<100	<1	<1	<1	<1	<1	<10	<1	0.04	1	0.5	0.02	1.5	***0.11***
South Creek S2	07-10-98	<100	<1	<1	<1	<1	<1	<10	<1	0.03	0.7	<0.01	<0.01	0.7	***0.07***
South Creek S2	14-12-98	<100	<1	<1	<1	<1	<1	<10	<1	<0.01	0.4	0.04	<0.01	0.44	0.01
South Creek S3	16-09-98	<100	<1	<1	<1	<1	<1	120	<1	0.09	1.1	0.24	0.02	1.4	***0.47***
South Creek S3	07-10-98	<100	<1	<1	<1	<1	<1	<10	<1	0.21	1.3	0.18	0.04	1.52	***0.5***
South Creek S3	14-12-98	<100	<1	<1	<1	<1	<1	<10	<1	0.03	0.8	<0.01	<0.01	0.8	***0.05***
Thompson Creek T1	10-12-96	<1	<1	<1	<1	<1	<1	4900	<1	0.13	0.5	<0.02	0.026	<0.02	<0.02
Thompson Creek T1	17-09-98	<100	<1	<1	<1	<1	<1	<10	<1	0.18	1	0.1	0.01	1.1	0.01
Thompson Creek T1	09-10-98	<100	<1	<1	<1	<1	<1	<10	<1	0.29	1.1	0.02	<0.01	1.12	***0.04***
Thompson Creek T1	15-12-98	<100	<1	<1	<1	<1	<1	<10	<1	0.04	1.1	0.04	<0.01	1.14	***0.04***

Table A5 - Selected Surface Water Screening Criteria

Chemical Name	Units	Primary Contact Recreation	Reference	Stock Watering	Reference	Irrigation	Reference	Freshwater Ecosystem	Reference
Anionic Surfactants as MBAs	mg/L	-	-	-	-	-	-	0.28	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Electrical Conductivity (lab)	uS/cm	-	-	-	-	-	-	125-2200	ANZECC (2000) Lowland River Trigger Values
pH (Lab)	pH Units	-	-	-	-	-	-	6.0-8.0	ANZECC (2000) Lowland River Trigger Values
Total Phosphorus as P	mg/L	0.004	USEPA (2015) Tap Water x 10	0.0004	USEPA (2015) Tap Water Guidelines	0.05	ANZECC (2000) Irrigation LTV	0.01	ANZECC (2000) Lowland River Trigger Values
Calcium	mg/L	-	-	1000	ANZECC (2000) Stock Watering	-	-	116	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Magnesium	mg/L	-	-	-	-	-	-	82	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Potassium	mg/L	-	-	-	-	-	-	53	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Arsenic	mg/L	0.1	NHMRC (2011) Drinking Water Guidelines x 10	0.5	ANZECC (2000) Stock Watering	0.1	ANZECC (2000) Irrigation LTV	0.15	USEPA (2013) Ambient Water Quality Criteria (Chronic - Freshwater)
Cadmium	mg/L	0.02	NHMRC (2011) Drinking Water Guidelines x 10	0.01	ANZECC (2000) Stock Watering	0.01	ANZECC (2000) Irrigation LTV	0.0002	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Chromium	mg/L	0.5	WHO (2011) Drinking Water Guidelines x 10	1	ANZECC (2000) Stock Watering	0.1	ANZECC (2000) Irrigation LTV	0.085	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Copper	mg/L	20	NHMRC (2011) Drinking Water Guidelines x 10	0.4	ANZECC (2000) Stock Watering	0.2	ANZECC (2000) Irrigation LTV	0.0014	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Iron	mg/L	140	USEPA (2015) Tap Water x 10	14	USEPA (2015) Tap Water Guidelines	0.2	ANZECC (2000) Irrigation LTV	0.3	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Lead	mg/L	0.1	NHMRC (2011) Drinking Water Guidelines x 10	0.1	ANZECC (2000) Stock Watering	2	ANZECC (2000) Irrigation LTV	0.0034	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Mercury	mg/L	0.01	NHMRC (2011) Drinking Water Guidelines x 10	0.002	ANZECC (2000) Stock Watering	0.002	ANZECC (2000) Irrigation LTV	0.0006	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Nickel	mg/L	0.2	NHMRC (2011) Drinking Water Guidelines x 10	1	ANZECC (2000) Stock Watering	0.2	ANZECC (2000) Irrigation LTV	0.011	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Zinc	mg/L	60	USEPA (2015) Tap Water x 10	20	ANZECC (2000) Stock Watering	2	ANZECC (2000) Irrigation LTV	0.008	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Benzene	mg/L	0.01	NHMRC (2011) Drinking Water Guidelines x 10	0.001	NHMRC (2011) Drinking Water Guidelines	-	-	0.95	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Ethylbenzene	mg/L	3	NHMRC (2011) Drinking Water Guidelines x 10	0.3	NHMRC (2011) Drinking Water Guidelines	-	-	0.08	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Toluene	mg/L	8	NHMRC (2011) Drinking Water Guidelines x 10	0.8	NHMRC (2011) Drinking Water Guidelines	-	-	0.18	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Total Xylenes	mg/L	6	NHMRC (2011) Drinking Water Guidelines x 10	0.6	NHMRC (2011) Drinking Water Guidelines	-	-	0.013	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
C6 - C9 Fraction	mg/L	150	WHO (2008) Petroleum Products in Drinking Water x 10	15	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C6 - C10 Fraction	mg/L	150	WHO (2008) Petroleum Products in Drinking Water x 10	15	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C6 - C10 Fraction minus BTEX (F1)	mg/L	150	WHO (2008) Petroleum Products in Drinking Water x 10	15	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C10 - C14 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C15 - C28 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C29 - C36 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
>C10 - C16 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
>C10 - C16 Fraction minus Naphthalene (F2)	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
>C10 - C40 Fraction (sum)	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
>C16 - C34 Fraction	mg/L	0.9	WHO (2008) Petroleum Products in Drinking Water x 10	0.09	WHO (2008) Petroleum Products in Drinking Water	-	-	-	-
C10-C36 Fraction (sum)	mg/L	-	-	-		-	-	0.6	RIVM (2001) Dutch Groundwater Intervention Value
1,1-Dichloroethane	mg/L	0.027	USEPA (2015) Tap Water x 10	0.0027	USEPA (2015) Tap Water Guidelines	-	-	0.09	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
1,2-Dichloroethane	mg/L	0.03	NHMRC (2011) Drinking Water Guidelines x 10	0.003	NHMRC (2011) Drinking Water Guidelines	-	-	1.9	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
1,1-Dichloroethene	mg/L	0.3	NHMRC (2011) Drinking Water Guidelines x 10	0.03	NHMRC (2011) Drinking Water Guidelines	-	-	0.7	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
1,1,1-Trichloroethane	mg/L	80	USEPA (2015) Tap Water x 10	8	USEPA (2015) Tap Water Guidelines	-	-	0.27	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
1,1,2-Trichloroethane	mg/L	0.0028	USEPA (2015) Tap Water x 10	0.00028	USEPA (2015) Tap Water Guidelines	-	-	6.5	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
1,1,1,2-Tetrachloroethane	mg/L	0.0057	USEPA (2015) Tap Water x 10	0.00057	USEPA (2015) Tap Water Guidelines	-	-	-	-
1,1,2,2-Tetrachloroethane	mg/L	0.00076	USEPA (2015) Tap Water x 10	0.000076	USEPA (2015) Tap Water Guidelines	-	-	0.4	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
1,2,3-Trichlorobenzene	mg/L	0.07	USEPA (2015) Tap Water x 10	0.007	USEPA (2015) Tap Water Guidelines	-	-	0.01	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
1,2,4-Trichlorobenzene	mg/L	0.011	USEPA (2015) Tap Water x 10	0.0011	USEPA (2015) Tap Water Guidelines	-	-	0.17	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
1,2,3-Trichloropropane	mg/L	0.0000075	USEPA (2015) Tap Water x 10	0.00000075	USEPA (2015) Tap Water Guidelines	-	-	-	-
1,2-Dibromo-3-chloropropane	mg/L	0.01	WHO (2011) Drinking Water Guidelines x 10	0.001	WHO (2011) Drinking Water Guidelines	-	-	-	-
1,2-Dibromoethane	mg/L	0.01	NHMRC (2011) Drinking Water Guidelines x 10	0.001	NHMRC (2011) Drinking Water Guidelines	-	-	-	-
1,2-Dichlorobenzene	mg/L	15	NHMRC (2011) Drinking Water Guidelines x 10	1.5	NHMRC (2011) Drinking Water Guidelines	-	-	0.16	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
1,3-Dichlorobenzene	mg/L	-	-	-	-	-	-	0.26	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
1,4-Dichlorobenzene	mg/L	0.4	NHMRC (2011) Drinking Water Guidelines x 10	0.04	NHMRC (2011) Drinking Water Guidelines	-	-	0.06	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
1,2-Dichloropropane	mg/L	0.4	WHO (2011) Drinking Water Guidelines x 10	0.04	WHO (2011) Drinking Water Guidelines	-	-	0.9	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
1,3-Dichloropropane	mg/L	3.7	USEPA (2015) Tap Water x 10	0.37	USEPA (2015) Tap Water Guidelines	-	-	1.1	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
2-Chlorotoluene	mg/L	2.4	USEPA (2015) Tap Water x 10	0.24	USEPA (2015) Tap Water Guidelines	-	-	-	-
4-Chlorotoluene	mg/L	2.5	USEPA (2015) Tap Water x 10	0.25	USEPA (2015) Tap Water Guidelines	-	-	-	-
Bromobenzene	mg/L	0.62	USEPA (2015) Tap Water x 10	0.062	USEPA (2015) Tap Water Guidelines	-	-	-	-
Bromodichloromethane	mg/L	0.6	WHO (2011) Drinking Water Guidelines x 10	0.06	WHO (2011) Drinking Water Guidelines	-	-	-	-
Bromoform	mg/L	1	WHO (2011) Drinking Water Guidelines x 10	0.1	WHO (2011) Drinking Water Guidelines	-	-	0.32	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Bromomethane	mg/L	0.01	NHMRC (2011) Drinking Water Guidelines x 10	0.001	NHMRC (2011) Drinking Water Guidelines	-	-	0.016	USEPA (2003) Region 5 RCRA Ecological Screening Levels (Water)
Chloroethane	mg/L	210	USEPA (2015) Tap Water x 10	21	USEPA (2015) Tap Water Guidelines	-	-	-	-
Chloromethane	mg/L	1.9	USEPA (2015) Tap Water x 10	0.19	USEPA (2015) Tap Water Guidelines	-	-	-	-
cis-1,2-Dichloroethene	mg/L	0.36	USEPA (2015) Tap Water x 10	0.036	USEPA (2015) Tap Water Guidelines	-	-	-	-
cis-1,4-Dichloro-2-butene	mg/L	0.000013	USEPA (2015) Tap Water x 10	0.0000013	USEPA (2015) Tap Water Guidelines	-	-	-	-
Dibromochloromethane	mg/L	1	WHO (2011) Drinking Water Guidelines x 10	0.1	WHO (2011) Drinking Water Guidelines	-	-	-	-
Dibromomethane	mg/L	0.08	USEPA (2015) Tap Water x 10	0.008	USEPA (2015) Tap Water Guidelines	-	-	-	-
Dichlorodifluoromethane	mg/L	2	USEPA (2015) Tap Water x 10	0.2	USEPA (2015) Tap Water Guidelines	-	-	-	-
Hexachlorobutadiene	mg/L	0.007	NHMRC (2011) Drinking Water Guidelines x 10	0.0007	NHMRC (2011) Drinking Water Guidelines	-	-	0.00004	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Pentachloroethane	mg/L	0.0064	USEPA (2015) Tap Water x 10	0.00064	USEPA (2015) Tap Water Guidelines	-	-	0.08	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
trans-1,2-Dichloroethene	mg/L	3.6	USEPA (2015) Tap Water x 10	0.36	USEPA (2015) Tap Water Guidelines	-	-	0.97	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
trans-1,4-Dichloro-2-butene	mg/L	0.000013	USEPA (2015) Tap Water x 10	0.0000013	USEPA (2015) Tap Water Guidelines	-	-	-	-
Trichlorofluoromethane	mg/L	11	USEPA (2015) Tap Water x 10	1.1	USEPA (2015) Tap Water Guidelines	-	-	-	-
Acenaphthene	mg/L	5.3	USEPA (2015) Tap Water x 10	0.53	USEPA (2015) Tap Water Guidelines	-	-	0.0058	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Acenaphthylene	mg/L	-	-	-	-	-	-	4.84	USEPA (2003) Region 5 RCRA Ecological Screening Levels (Water)
Anthracene	mg/L	18	USEPA (2015) Tap Water x 10	1.8	USEPA (2015) Tap Water Guidelines	-	-	0.00001	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Benz(a)anthracene	mg/L	0.00034	USEPA (2015) Tap Water x 10	0.000034	USEPA (2015) Tap Water Guidelines	-	-	0.000018	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Benzo(a)pyrene	mg/L	0.0001	NHMRC (2011) Drinking Water Guidelines x 10	0.00001	NHMRC (2011) Drinking Water Guidelines	-	-	0.0001	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Benzo(g,h,i)perylene	mg/L	-	-	-	-	-	-	0.00764	USEPA (2003) Region 5 RCRA Ecological Screening Levels (Water)

Table A5 - Selected Surface Water Screening Criteria

Chemical Name	Units	Primary Contact Recreation	Reference	Stock Watering	Reference	Irrigation	Reference	Freshwater Ecosystem	Reference
Benzo(k)fluoranthene	mg/L	0.0034	USEPA (2015) Tap Water x 10	0.00034	USEPA (2015) Tap Water Guidelines	-	-	-	-
Chrysene	mg/L	0.034	USEPA (2015) Tap Water x 10	0.0034	USEPA (2015) Tap Water Guidelines	-	-	-	-
Dibenz(a,h)anthracene	mg/L	0.000034	USEPA (2015) Tap Water x 10	0.0000034	USEPA (2015) Tap Water Guidelines	-	-	-	-
Fluoranthene	mg/L	8	USEPA (2015) Tap Water x 10	0.8	USEPA (2015) Tap Water Guidelines	-	-	0.001	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Fluorene	mg/L	2.9	USEPA (2015) Tap Water x 10	0.29	USEPA (2015) Tap Water Guidelines	-	-	0.003	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Indeno(1,2,3-c,d)pyrene	mg/L	0.00034	USEPA (2015) Tap Water x 10	0.000034	USEPA (2015) Tap Water Guidelines	-	-	0.00431	USEPA (2003) Region 5 RCRA Ecological Screening Levels (Water)
Naphthalene	mg/L	0.0017	USEPA (2015) Tap Water x 10	0.00017	USEPA (2015) Tap Water Guidelines	-	-	0.016	ANZECC (2000) Fresh Water Quality Criteria (95% protection)
Phenanthrene	mg/L	-	-	-	-	-	-	0.0006	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Pyrene	mg/L	1.2	USEPA (2015) Tap Water x 10	0.12	USEPA (2015) Tap Water Guidelines	-	-	0.000025	USEPA (2006) Region 3 BTAG Fresh Water Quality Criteria
Carbon Tetrachloride	mg/L	0.03	NHMRC (2011) Drinking Water Guidelines x 10	0.003	NHMRC (2011) Drinking Water Guidelines	-	-	0.24	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Chlorobenzene	mg/L	3	NHMRC (2011) Drinking Water Guidelines x 10	0.3	NHMRC (2011) Drinking Water Guidelines	-	-	0.055	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Chloroform	mg/L	3	WHO (2011) Drinking Water Guidelines x 10	0.3	WHO (2011) Drinking Water Guidelines	-	-	0.37	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Tetrachloroethene	mg/L	0.5	NHMRC (2011) Drinking Water Guidelines x 10	0.05	NHMRC (2011) Drinking Water Guidelines	-	-	0.07	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Trichloroethene	mg/L	0.2	WHO (2011) Drinking Water Guidelines x 10	0.02	WHO (2011) Drinking Water Guidelines	-	-	0.33	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)
Vinyl chloride	mg/L	0.003	NHMRC (2011) Drinking Water Guidelines x 10	0.0003	NHMRC (2011) Drinking Water Guidelines	-	-	0.1	ANZECC (2000) Medium to Low Reliability Criteria (Freshwater)

Table A6 - Surface Water Analytical Data

							Location	Badgery's Creek - upstream	Badgery's Creek - upstream	Badgery's Creek - mid-catchment (a)	Badgery's Creek - mid-catchment (b)	Badgery's Creek - downstream	Badgery's Creek - downstream	Oaky Creek - downstream	Cosgrove Creek - upstream	Cosgrove Creek - upstream	Cosgrove Creek - downstream	Duncan's Creek - downstream
							Location ID	B1	BCUS (B1)	B2	BCMC	B3	BCDS (B3)	OCDS	C1	CCUS (C1)	CCDS	DN1
							Sample date:	22/09/2014	16/03/2015	22/09/2014	16/03/2015	22/09/2014	16/03/2015	16/03/2015	22/09/2014	16/03/2015	16/03/2015	22/09/2014
Analyte grouping/Analyte	**Units**	**LOR**	Primary Contact Recreation	Stock Watering	Irrigation	***Freshwater Ecosystem Protection***												
Electrical Conductivity @ 25°C	µS/cm	1	-	-	-	***125-2200***	-	**2710**	-	**3100**	-	**3050**	**4320**	-	**5020**	1050	-
Total Hardness as CaCO3	mg/L	1	-	-	-	-	-	550	-	521	-	535	604	-	806	160	-
Total Kjeldahl Nitrogen as N	mg/L	0.1	-	-	-	-	2.8	1.6	2.3	2.9	2.5	2.2	1.1	0.8	0.6	1.3	1.5
Total Nitrogen as N	mg/L	0.1	-	-	-	-	2.8	6.2	2.5	18.5	2.6	2.3	1.2	0.8	0.8	1.3	1.5
Ammonia as N	mg/L	0.01	-	-	-	-	0.01	0.17	0.013	0.28	0.006	0.22	0.22	<0.005	0.14	0.04	<0.005
Total Organic Carbon	mg/L	0.2	-	-	-	-	25	10.2	24	31.7	18	12.7	19.3	10	8.9	10.5	16
Anionic Surfactants as MBAS	mg/L	0.1	-	-	-	***0.28***	<0.1	0.1	<0.1	<0.1	<0.1	0.1	0.1	<0.1	0.1	0.1	<0.1
Nitrate as N	mg/L	0.01	-	-	-	-	<0.005	4.56	0.13	15.5	0.078	0.1	0.06	<0.005	0.15	0.03	<0.005
Nitrite + Nitrate as N	mg/L	0.01	-	-	-	-	-	4.64	-	15.6	-	0.1	0.06	-	0.15	0.03	-
Nitrite as N	mg/L	0.01	-	-	-	-	<0.005	0.08	0.011	0.08	0.01	<0.01	<0.01	<0.005	<0.01	<0.01	<0.005
Oil & Grease	mg/L	5	-	-	-	-	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5	<5
Suspended Solids (SS)	mg/L	5	-	-	-	-	10	23	17	<5	16	<5	19	32	<5	44	30
Total Phosphorus as P	mg/L	0.01	0.004	0.0004	0.05	***0.01***	***1.6***	***0.42***	***0.5***	***0.31***	***0.5***	***1***	***0.05***	***0.09***	***0.03***	***0.3***	***0.1***
Calcium	mg/L	1	-	1000	-	***116***	-	67	-	52	-	61	49	-	74	23	-
Magnesium	mg/L	1	-	-	-	***82***	-	***93***	-	***95***	-	***93***	***117***	-	***151***	25	-
Potassium	mg/L	1	-	-	-	***53***	-	13	-	***80***	-	***71***	19	-	8	9	-
Sodium	mg/L	1	-	-	-	***680***	-	430	-	497	-	449	***741***	-	***846***	162	-
Arsenic	mg/L		0.1	0.5	0.1	***0.15***	0.003	-	0.002	-	0.001	-	-	<0.001	-	-	0.001
Cadmium	mg/L	0.0001	0.02	0.01	0.01	***0.0002***	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Chromium	mg/L	0.001	0.5	1	0.1	***0.085***	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Copper	mg/L	0.001	20	0.4	0.2	***0.0014***	***0.007***	***0.007***	***0.01***	***0.009***	***0.008***	0.003	***0.002***	***0.003***	0.001	<0.001	***0.004***
Iron	mg/L	0.05	140	14	0.2	***0.3***	-	***0.38***	-	***1.35***	-	***0.72***	***2.35***	-	0.18	***3.82***	-
Lead	mg/L	0.001	0.1	0.1	2	***0.0034***	<0.001	<0.001	<0.001	0.002	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mercury	mg/L	0.0001	0.01	0.002	0.002	***0.0006***	<0.00005	<0.0001	<0.00005	<0.0001	<0.00005	<0.0001	<0.0001	<0.00005	<0.0001	<0.0001	<0.00005
Nickel	mg/L	0.001	0.2	1	0.2	***0.011***	0.004	0.001	0.005	0.01	0.003	0.005	0.003	0.001	0.002	0.001	0.003
Zinc	mg/L	0.005	60	20	2	***0.008***	***0.01***	0.008	***0.01***	***0.059***	0.007	0.008	0.008	0.003	<0.005	0.006	0.006
Benzene	µg/L	1	10	1	-	***950***	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Ethylbenzene	µg/L	2	3,000	300	-	***80***	<1	<2	<1	<2	<1	<2	<2	<1	<2	<2	<1
Toluene	µg/L	2	8,000	800	-	***180***	<1	<2	<1	<2	<1	<2	<2	<1	<2	<2	<1
meta- & para-Xylene	µg/L	2	-	-	-	-	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2	<2
ortho-Xylene	µg/L	2	-	-	-	-	<1	<2	<1	<2	<1	<2	<2	<1	<2	<2	<1
Total Xylenes	µg/L	2	6,000	600	-	***13***	-	<2	-	<2	-	<2	<2	-	<2	<2	-
Sum of BTEX	µg/L	1	-	-	-	-	-	<1	-	<1	-	<1	<1	-	<1	<1	-
C6 - C9 Fraction	µg/L	20	150,000	15,000	-	-	<10	<20	<10	<20	<10	<20	<20	<10	<20	<20	<10
C6 - C10 Fraction	µg/L	20	150,000	15,000	-	-	<10	<20	<10	<20	<10	<20	<20	<10	<20	<20	<10
C6 - C10 Fraction minus BTEX (F1)	µg/L	20	150,000	15,000	-	-	<10	<20	<10	<20	<10	<20	<20	<10	<20	<20	<10
C10 - C14 Fraction	µg/L	50	900	90	-	-	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50
C15 - C28 Fraction	µg/L	100	900	90	-	-	<100	<100	150	<100	230	<100	<100	<100	<100	<100	120
C29 - C36 Fraction	µg/L	50	900	90	-	-	190	<50	500	<50	340	<50	<50	220	<50	<50	320
>C10 - C16 Fraction	µg/L	100	900	90	-	-	<50	<100	<50	<100	<50	<100	<100	<50	<100	<100	<50
>C10 - C16 Fraction minus Naphthalene (F2)	µg/L	100	900	90	-	-	<50	<100	<50	<100	<50	<100	<100	<50	<100	<100	<50
>C10 - C40 Fraction (sum)	µg/L	100	900	90	-	-	-	<100	-	<100	-	<100	<100	-	<100	<100	-
>C16 - C34 Fraction	µg/L	100	900	90	-	-	240	<100	540	<100	480	<100	<100	260	<100	<100	380
>C34 - C40 Fraction	µg/L	100	-	-	-	-	<100	<100	280	<100	180	<100	<100	<100	<100	<100	150
C10 - C36 Fraction (sum)	µg/L	50	-	-	-	***600***	-	<50	-	<50	-	<50	<50	-	<50	<50	-

Table A6 - Surface Water Analytical Data

						Location	Badgery's Creek - upstream	Badgery's Creek - upstream	Badgery's Creek - mid-catchment (a)	Badgery's Creek - mid-catchment (b)	Badgery's Creek - downstream	Badgery's Creek - downstream	Oaky Creek - downstream	Cosgrove Creek - upstream	Cosgrove Creek - upstream	Cosgrove Creek - downstream	Duncan's Creek - downstream
						Location ID	B1	BCUS (B1)	B2	BCMC	B3	BCDS (B3)	OCDS	C1	CCUS (C1)	CCDS	DN1
						Sample date:	22/09/2014	16/03/2015	22/09/2014	16/03/2015	22/09/2014	16/03/2015	16/03/2015	22/09/2014	16/03/2015	16/03/2015	22/09/2014
Analyte grouping/Analyte	**Units**	**LOR**	Primary Contact Recreation	Stock Watering	Irrigation	***Freshwater Ecosystem Protection***											
1.1-Dichloroethane	µg/L	5	27	2.7	-	***90***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2-Dichloroethane	µg/L	5	30	3	-	***1900***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.1-Dichloroethene	µg/L	5	300	30	-	***700***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.1-Dichloropropylene	µg/L	5	-	-	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.1.1-Trichloroethane	µg/L	5	80,000	8,000	-	***270***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.1.2-Trichloroethane	µg/L	5	2.8	0.28	-	***6500***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.1.1.2-Tetrachloroethane	µg/L	5	5.7	0.57	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.1.2.2-Tetrachloroethane	µg/L	5	0.76	0.076	-	***400***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2.3-Trichlorobenzene	µg/L	5	70	7	-	***10***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2.4-Trichlorobenzene	µg/L	5	11	1.1	-	***170***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2.3-Trichloropropane	µg/L	5	0.0075	0.00075	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2-Dibromo-3-chloropropane	µg/L	5	10	1	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2-Dibromoethane (EDB)	µg/L	5	10	1	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2-Dichlorobenzene	µg/L	5	15,000	1,500	-	***160***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.3-Dichlorobenzene	µg/L	5	-	-	-	***260***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.4-Dichlorobenzene	µg/L	5	400	40	-	***60***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.2-Dichloropropane	µg/L	5	400	40	-	***900***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
1.3-Dichloropropane	µg/L	5	3700	370	-	***1100***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
2.2-Dichloropropane	µg/L	5	-	-	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
2-Chlorotoluene	µg/L	5	2400	240	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
4-Chlorotoluene	µg/L	5	2500	250	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Bromobenzene	µg/L	5	620	62	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Bromodichloromethane	µg/L	5	600	60	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Bromoform	µg/L	5	1000	100	-	***320***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Bromomethane	µg/L	50	10	1	-	***16***	<10	<50	<10	<50	<10	<50	<50	<10	<50	<50	<10
Chloroethane	µg/L	50	210,000	21,000	-	-	<10	<50	<10	<50	<10	<50	<50	<10	<50	<50	<10
Chloromethane	µg/L	50	1900	190	-	-	<10	<50	<10	<50	<10	<50	<50	<10	<50	<50	<10
cis-1.2-Dichloroethene	µg/L	5	360	36	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
cis-1.3-Dichloropropylene	µg/L	5	-	-	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
cis-1.4-Dichloro-2-butene	µg/L	5	0.013	0.0013	-	-	-	<5	-	<5	-	<5	<5	-	<5	<5	-
Dibromochloromethane	µg/L	5	1000	100	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Dibromomethane	µg/L	5	80	8	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Dichlorodifluoromethane	µg/L	50	2000	200	-	-	<10	<50	<10	<50	<10	<50	<50	<10	<50	<50	<10
Hexachlorobutadiene	µg/L	5	7	0.7	-	***0.04***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Iodomethane	µg/L	5	-	-	-	-	-	<5	-	<5	-	<5	<5	-	<5	<5	-
Pentachloroethane	µg/L	5	6.4	0.64	-	***80***	-	<5	-	<5	-	<5	<5	-	<5	<5	-
trans-1.2-Dichloroethene	µg/L	5	3600	360	-	***970***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
trans-1.3-Dichloropropylene	µg/L	5	-	-	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
trans-1.4-Dichloro-2-butene	µg/L	5	0.013	0.0013	-	-	-	<5	-	<5	-	<5	<5	-	<5	<5	-
Trichlorofluoromethane	µg/L	50	11,000	1,100	-	-	<10	<50	<10	<50	<10	<50	<50	<10	<50	<50	<10
Acenaphthene	µg/L	1	5300	530	-	***5.8***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Acenaphthylene	µg/L	1	-	-	-	***4840***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Anthracene	µg/L	1	18,000	1,800	-	***0.01***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Benz(a)anthracene	µg/L	1	0.34	0.034	-	***0.018***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Benzo(a)pyrene	µg/L	0.5	0.1	0.01	-	***0.1***	<0.1	<0.5	<0.1	<0.5	<0.1	<0.5	<0.5	<0.1	<0.5	<0.5	<0.1
Benzo(b+j)fluoranthene	µg/L	1	-	-	-	-	-	<1.0	-	<1.0	-	<1.0	<1.0	-	<1.0	<1.0	-
Benzo(g.h.i)perylene	µg/L	1	-	-	-	***7.64***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Benzo(k)fluoranthene	µg/L	1	3.4	0.34	-	-	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1

Table A6 - Surface Water Analytical Data

						Location	Badgery's Creek - upstream	Badgery's Creek - upstream	Badgery's Creek - mid-catchment (a)	Badgery's Creek - mid-catchment (b)	Badgery's Creek - downstream	Badgery's Creek - downstream	Oaky Creek - downstream	Cosgrove Creek - upstream	Cosgrove Creek - upstream	Cosgrove Creek - downstream	Duncan's Creek - downstream
						Location ID	B1	BCUS (B1)	B2	BCMC	B3	BCDS (B3)	OCDS	C1	CCUS (C1)	CCDS	DN1
						Sample date:	22/09/2014	16/03/2015	22/09/2014	16/03/2015	22/09/2014	16/03/2015	16/03/2015	22/09/2014	16/03/2015	16/03/2015	22/09/2014
Analyte grouping/Analyte	**Units**	**LOR**	Primary Contact Recreation	Stock Watering	Irrigation	***Freshwater Ecosystem Protection***											
Chrysene	µg/L	1	34	3.4	-	-	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Dibenz(a.h)anthracene	µg/L	1	0.034	0.0034	-	-	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Fluoranthene	µg/L	1	8,000	800	-	***1***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Fluorene	µg/L	1	2,900	290	-	***3***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Indeno(1.2.3.cd)pyrene	µg/L	1	0.34	0.034	-	***4.31***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Naphthalene	µg/L	1	1.7	0.17	-	***16***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Phenanthrene	µg/L	1	-	-	-	***0.6***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Pyrene	µg/L	1	1,200	120	-	***0.025***	<0.1	<1.0	<0.1	<1.0	<0.1	<1.0	<1.0	<0.1	<1.0	<1.0	<0.1
Sum of polycyclic aromatic hydrocarbons	µg/L	0.5	-	-	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Benzo(a)pyrene TEQ (zero)	µg/L	0.5	-	-	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Carbon Tetrachloride	µg/L	5	30	3	-	***240***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Chlorobenzene	µg/L	5	3,000	300	-	***55***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Chloroform	µg/L	5	3,000	300	-	***370***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Tetrachloroethene	µg/L	5	500	50	-	***70***	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1
Trichloroethene	µg/L	5	-	-	-	-	<1	<5	<1	<5	<1	<5	<5	<1	<5	<5	<1

Table A6 - Surface Water Analytical

						Location	Duncan's Creek - downstream	South Creek - Upstream	South Creek - recovery	South Creek - downstream	Thompson Creek - upstream	Thomsons Creek - mid-catchment
						Location ID	DCDS (DN1)	SCUS	SCREC	SCDS	TCUS	T1
						Sample date:	16/03/2015	16/03/2015	16/03/2015	16/03/2015	16/03/2015	22/09/2014
Analyte grouping/Analyte	**Units**	**LOR**	Primary Contact Recreation	Stock Watering	Irrigation	***Freshwater Ecosystem Protection***						
Electrical Conductivity @ 25°C	µS/cm	1	-	-	-	***125-2200***	847	1680	1540	1900	1640	-
Total Hardness as CaCO3	mg/L	1	-	-	-	-	162	281	253	292	179	-
Total Kjeldahl Nitrogen as N	mg/L	0.1	-	-	-	-	0.7	1.1	0.9	1	1	0.7
Total Nitrogen as N	mg/L	0.1	-	-	-	-	0.9	1.2	1	1.1	1	0.7
Ammonia as N	mg/L	0.01	-	-	-	-	0.23	0.3	0.14	0.06	0.04	<0.005
Total Organic Carbon	mg/L	0.2	-	-	-	-	6.3	10.1	14.4	16.6	20.3	11
Anionic Surfactants as MBAS	mg/L	0.1	-	-	-	***0.28***	0.1	<0.1	0.1	0.1	0.1	<0.1
Nitrate as N	mg/L	0.01	-	-	-	-	0.19	0.08	0.12	0.1	0.01	<0.005
Nitrite + Nitrate as N	mg/L	0.01	-	-	-	-	0.2	0.1	0.12	0.1	0.01	-
Nitrite as N	mg/L	0.01	-	-	-	-	0.01	0.02	<0.01	<0.01	<0.01	<0.005
Oil & Grease	mg/L	5	-	-	-	-	<5	<5	<5	<5	<5	<5
Suspended Solids (SS)	mg/L	5	-	-	-	-	14	<5	10	19	<5	31
Total Phosphorus as P	mg/L	0.01	0.004	0.0004	0.05	***0.01***	***0.06***	***0.27***	***0.05***	***0.08***	***0.09***	***0.07***
Calcium	mg/L	1	-	1000	-	***116***	32	40	32	38	24	-
Magnesium	mg/L	1	-	-	-	***82***	20	44	42	48	29	-
Potassium	mg/L	1	-	-	-	***53***	4	44	22	33	6	-
Sodium	mg/L	1	-	-	-	***680***	111	235	228	279	272	-
Arsenic	mg/L		0.1	0.5	0.1	***0.15***	-	-	-	-	-	<0.001
Cadmium	mg/L	0.0001	0.02	0.01	0.01	***0.0002***	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Chromium	mg/L	0.001	0.5	1	0.1	***0.085***	0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Copper	mg/L	0.001	20	0.4	0.2	***0.0014***	***0.002***	***0.004***	***0.01***	***0.002***	***0.002***	***0.003***
Iron	mg/L	0.05	140	14	0.2	***0.3***	***1.31***	***0.36***	***1.01***	***1.11***	***0.85***	-
Lead	mg/L	0.001	0.1	0.1	2	***0.0034***	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mercury	mg/L	0.0001	0.01	0.002	0.002	***0.0006***	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.00005
Nickel	mg/L	0.001	0.2	1	0.2	***0.011***	0.001	0.003	0.002	0.004	0.003	0.002
Zinc	mg/L	0.005	60	20	2	***0.008***	0.008	<0.005	***0.013***	0.005	0.007	0.007
Benzene	µg/L	1	10	1	-	***950***	<1	<1	<1	<1	<1	<1
Ethylbenzene	µg/L	2	3,000	300	-	***80***	<2	<2	<2	<2	<2	<1
Toluene	µg/L	2	8,000	800	-	***180***	<2	<2	<2	<2	<2	<1
meta- & para-Xylene	µg/L	2	-	-	-	-	<2	<2	<2	<2	<2	<2
ortho-Xylene	µg/L	2	-	-	-	-	<2	<2	<2	<2	<2	<1
Total Xylenes	µg/L	2	6,000	600	-	***13***	<2	<2	<2	<2	<2	-
Sum of BTEX	µg/L	1	-	-	-	-	<1	<1	<1	<1	<1	-
C6 - C9 Fraction	µg/L	20	150,000	15,000	-	-	<20	<20	<20	<20	<20	<10
C6 - C10 Fraction	µg/L	20	150,000	15,000	-	-	<20	<20	<20	<20	<20	<10
C6 - C10 Fraction minus BTEX (F1)	µg/L	20	150,000	15,000	-	-	<20	<20	<20	<20	<20	<10
C10 - C14 Fraction	µg/L	50	900	90	-	-	<50	<50	<50	<50	<50	<50
C15 - C28 Fraction	µg/L	100	900	90	-	-	<100	<100	<100	<100	<100	<100
C29 - C36 Fraction	µg/L	50	900	90	-	-	<50	<50	<50	<50	<50	<100
>C10 - C16 Fraction	µg/L	100	900	90	-	-	<100	<100	<100	<100	<100	<50
>C10 - C16 Fraction minus Naphthalene (F2)	µg/L	100	900	90	-	-	<100	<100	<100	<100	<100	<50
>C10 - C40 Fraction (sum)	µg/L	100	900	90	-	-	<100	<100	<100	<100	<100	-
>C16 - C34 Fraction	µg/L	100	900	90	-	-	<100	<100	<100	<100	<100	<100
>C34 - C40 Fraction	µg/L	100	-	-	-	-	<100	<100	<100	<100	<100	<100
C10 - C36 Fraction (sum)	µg/L	50	-	-	-	***600***	<50	<50	<50	<50	<50	-

Table A6 - Surface Water Analytical

			Location				Duncan's Creek - downstream	South Creek - Upstream	South Creek - recovery	South Creek - downstream	Thompson Creek - upstream	Thomsons Creek - mid-catchment
			Location ID				DCDS (DN1)	SCUS	SCREC	SCDS	TCUS	T1
			Sample date:				16/03/2015	16/03/2015	16/03/2015	16/03/2015	16/03/2015	22/09/2014
Analyte grouping/Analyte	**Units**	**LOR**	Primary Contact Recreation	Stock Watering	Irrigation	***Freshwater Ecosystem Protection***						
1.1-Dichloroethane	µg/L	5	27	2.7	-	***90***	<5	<5	<5	<5	<5	<1
1.2-Dichloroethane	µg/L	5	30	3	-	***1900***	<5	<5	<5	<5	<5	<1
1.1-Dichloroethene	µg/L	5	300	30	-	***700***	<5	<5	<5	<5	<5	<1
1.1-Dichloropropylene	µg/L	5	-	-	-	-	<5	<5	<5	<5	<5	<1
1.1.1-Trichloroethane	µg/L	5	80,000	8,000	-	***270***	<5	<5	<5	<5	<5	<1
1.1.2-Trichloroethane	µg/L	5	2.8	0.28	-	***6500***	<5	<5	<5	<5	<5	<1
1.1.1.2-Tetrachloroethane	µg/L	5	5.7	0.57	-	-	<5	<5	<5	<5	<5	<1
1.1.2.2-Tetrachloroethane	µg/L	5	0.76	0.076	-	***400***	<5	<5	<5	<5	<5	<1
1.2.3-Trichlorobenzene	µg/L	5	70	7	-	***10***	<5	<5	<5	<5	<5	<1
1.2.4-Trichlorobenzene	µg/L	5	11	1.1	-	***170***	<5	<5	<5	<5	<5	<1
1.2.3-Trichloropropane	µg/L	5	0.0075	0.00075	-	-	<5	<5	<5	<5	<5	<1
1.2-Dibromo-3-chloropropane	µg/L	5	10	1	-	-	<5	<5	<5	<5	<5	<1
1.2-Dibromoethane (EDB)	µg/L	5	10	1	-	-	<5	<5	<5	<5	<5	<1
1.2-Dichlorobenzene	µg/L	5	15,000	1,500	-	***160***	<5	<5	<5	<5	<5	<1
1.3-Dichlorobenzene	µg/L	5	-	-	-	***260***	<5	<5	<5	<5	<5	<1
1.4-Dichlorobenzene	µg/L	5	400	40	-	***60***	<5	<5	<5	<5	<5	<1
1.2-Dichloropropane	µg/L	5	400	40	-	***900***	<5	<5	<5	<5	<5	<1
1.3-Dichloropropane	µg/L	5	3700	370	-	***1100***	<5	<5	<5	<5	<5	<1
2.2-Dichloropropane	µg/L	5	-	-	-	-	<5	<5	<5	<5	<5	<1
2-Chlorotoluene	µg/L	5	2400	240	-	-	<5	<5	<5	<5	<5	<1
4-Chlorotoluene	µg/L	5	2500	250	-	-	<5	<5	<5	<5	<5	<1
Bromobenzene	µg/L	5	620	62	-	-	<5	<5	<5	<5	<5	<1
Bromodichloromethane	µg/L	5	600	60	-	-	<5	<5	<5	<5	<5	<1
Bromoform	µg/L	5	1000	100	-	***320***	<5	<5	<5	<5	<5	<1
Bromomethane	µg/L	50	10	1	-	***16***	<50	<50	<50	<50	<50	<10
Chloroethane	µg/L	50	210,000	21,000	-	-	<50	<50	<50	<50	<50	<10
Chloromethane	µg/L	50	1900	190	-	-	<50	<50	<50	<50	<50	<10
cis-1.2-Dichloroethene	µg/L	5	360	36	-	-	<5	<5	<5	<5	<5	<1
cis-1.3-Dichloropropylene	µg/L	5	-	-	-	-	<5	<5	<5	<5	<5	<1
cis-1.4-Dichloro-2-butene	µg/L	5	0.013	0.0013	-	-	<5	<5	<5	<5	<5	-
Dibromochloromethane	µg/L	5	1000	100	-	-	<5	<5	<5	<5	<5	<1
Dibromomethane	µg/L	5	80	8	-	-	<5	<5	<5	<5	<5	<1
Dichlorodifluoromethane	µg/L	50	2000	200	-	-	<50	<50	<50	<50	<50	<10
Hexachlorobutadiene	µg/L	5	7	0.7	-	***0.04***	<5	<5	<5	<5	<5	<1
Iodomethane	µg/L	5	-	-	-	-	<5	<5	<5	<5	<5	-
Pentachloroethane	µg/L	5	6.4	0.64	-	***80***	<5	<5	<5	<5	<5	-
trans-1.2-Dichloroethene	µg/L	5	3600	360	-	***970***	<5	<5	<5	<5	<5	<1
trans-1.3-Dichloropropylene	µg/L	5	-	-	-	-	<5	<5	<5	<5	<5	<1
trans-1.4-Dichloro-2-butene	µg/L	5	0.013	0.0013	-	-	<5	<5	<5	<5	<5	-
Trichlorofluoromethane	µg/L	50	11,000	1,100	-	-	<50	<50	<50	<50	<50	<10
Acenaphthene	µg/L	1	5300	530	-	***5.8***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Acenaphthylene	µg/L	1	-	-	-	***4840***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Anthracene	µg/L	1	18,000	1,800	-	***0.01***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Benz(a)anthracene	µg/L	1	0.34	0.034	-	***0.018***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Benzo(a)pyrene	µg/L	0.5	0.1	0.01	-	***0.1***	<0.5	<0.5	<0.5	<0.5	<0.5	<0.1
Benzo(b+j)fluoranthene	µg/L	1	-	-	-	-	<1.0	<1.0	<1.0	<1.0	<1.0	-
Benzo(g.h.i)perylene	µg/L	1	-	-	-	***7.64***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Benzo(k)fluoranthene	µg/L	1	3.4	0.34	-	-	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1

Table A6 - Surface Water Analytical

						Location	Duncan's Creek - downstream	South Creek - Upstream	South Creek - recovery	South Creek - downstream	Thompson Creek - upstream	Thomsons Creek - mid-catchment
						Location ID	DCDS (DN1)	SCUS	SCREC	SCDS	TCUS	T1
						Sample date:	16/03/2015	16/03/2015	16/03/2015	16/03/2015	16/03/2015	22/09/2014
Analyte grouping/Analyte	**Units**	**LOR**	Primary Contact Recreation	Stock Watering	Irrigation	***Freshwater Ecosystem Protection***						
Chrysene	µg/L	1	34	3.4	-	-	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Dibenz(a.h)anthracene	µg/L	1	0.034	0.0034	-	-	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Fluoranthene	µg/L	1	8,000	800	-	***1***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Fluorene	µg/L	1	2,900	290	-	***3***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Indeno(1.2.3.cd)pyrene	µg/L	1	0.34	0.034	-	***4.31***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Naphthalene	µg/L	1	1.7	0.17	-	***16***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Phenanthrene	µg/L	1	-	-	-	***0.6***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Pyrene	µg/L	1	1,200	120	-	***0.025***	<1.0	<1.0	<1.0	<1.0	<1.0	<0.1
Sum of polycyclic aromatic hydrocarbons	µg/L	0.5	-	-	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Benzo(a)pyrene TEQ (zero)	µg/L	0.5	-	-	-	-	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Carbon Tetrachloride	µg/L	5	30	3	-	***240***	<5	<5	<5	<5	<5	<1
Chlorobenzene	µg/L	5	3,000	300	-	***55***	<5	<5	<5	<5	<5	<1
Chloroform	µg/L	5	3,000	300	-	***370***	<5	<5	<5	<5	<5	<1
Tetrachloroethene	µg/L	5	500	50	-	***70***	<5	<5	<5	<5	<5	<1
Trichloroethene	µg/L	5	-	-	-	-	<5	<5	<5	<5	<5	<1