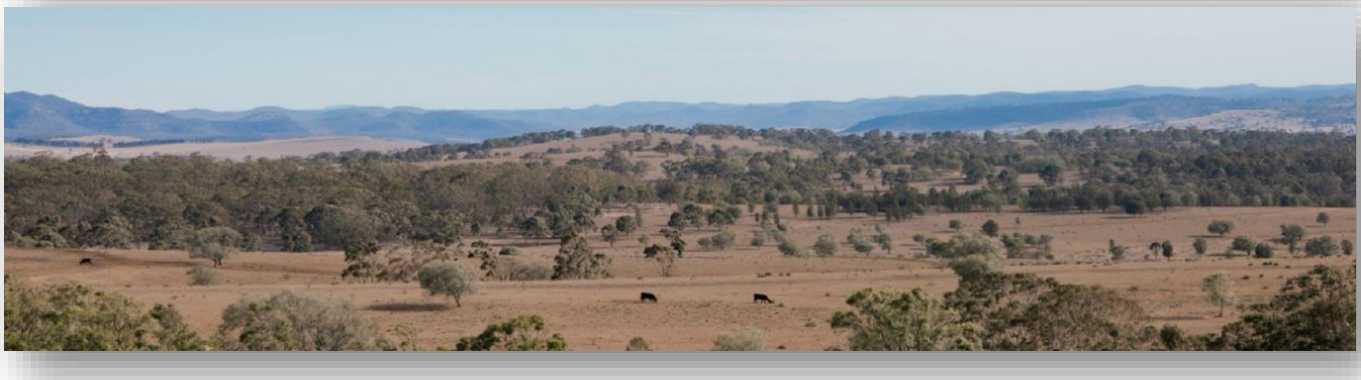
MALABAR COAL

MAXWELL PROJECT

APPENDIX R

Human Health Risk Assessment



Maxwell Project: Human Health Risk Assessment

Prepared for: Maxwell Ventures (Management) Pty Ltd

10 July 2019

Document History and Status

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Limitations

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Glossary of Terms and Abbreviations

Term	Definition
AAQ	Ambient air quality.
ABS	Australian Bureau of Statistics.
Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days).
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems.
Aerodynamic diameter	Airborne particles have irregular shapes, their aerodynamic behaviour is expressed in terms of the diameter of an idealised spherical particle.
AIHW	Australian Institute of Health and Welfare.
ANZECC	Australia and New Zealand Environment and Conservation Council.
AQGGA	Air Quality and Greenhouse Gas Assessment.
ATSDR	Agency for Toxic Substances and Disease Register.
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
Biodegradation	Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).
Body burden	The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.
Carcinogen	A substance that causes cancer.
CCC	Community Consultative Committee.
CCME	Canadian Council of Ministers of the Environment.
CHPP	Coal handling and preparation plant.
Chronic exposure	Contact with a substance or stressor that occurs over a long time (more than one year) [compare with acute exposure and intermediate duration exposure].
CL	Coal Lease.
COMEAP	Committee on the Medical Effects of Air Pollutants.
dB(A)	Decibels (A-weighted).
DEC	NSW Department of Environment and Conservation.
DECC	NSW Department of Environment and Climate Change.
DECCW	NSW Department of Environment, Climate Change and Water.
DEFRA	Department for Environment, Food & Rural Affairs.
DEH	Australian Department of Environment and Heritage.
Detection limit	The lowest concentration of a substance that can reliably be distinguished from a zero concentration.
Dose	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An 'exposure dose' is how much of a substance is encountered in the environment. An 'absorbed dose' is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
EIS	Environmental Impact Statement.
EL	Exploration Licence.
ENM	Environmental Noise Model.
EPHC	Environment Protection and Heritage Council.
EU	European Union.

Term	Definition
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Also includes contact with a stressor such as noise or vibration. Exposure may be short term [acute exposure], of intermediate duration, or long term [chronic exposure].
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.
Exposure pathway	The route a substance takes from its source (where it began) to its endpoint (where it ends), and how people can come into contact with (or get exposed) to it. An exposure pathway has five parts: a source of contamination (such as chemical substance leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.
Genotoxic carcinogen	These are carcinogens that have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement). Where this occurs, the damage may be sufficient to result in the initiation of cancer at some time during a lifetime.
Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the NSW Department of Environment and Conservation (DEC) or institutions such as the National Health and Medical Research Council (NHMRC), Australia and New Zealand Environment and Conservation Council (ANZECC) and World Health Organization (WHO)), that is used to identify conditions below which no adverse effects, nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines would have different names, such as investigation level, trigger value and ambient guideline.
HHRA	Human health risk assessment.
HI	Hazard Index.
IARC	International Agency for Research on Cancer.
ICNG	Interim Construction Noise Guideline.
I-INCE	International Institute of Noise Control Engineering.
Inhalation	The act of breathing.
Intermediate exposure	Contact with a substance that occurs for more than 14 days and less than a year [compared with acute exposure and chronic exposure].
LGA	Local Government Area.
LOAEL	Lowest-observed-adverse-effect level.
LOR	Limit of Reporting.
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
ML	Mining Lease.
Morbidity	This is the condition of being ill, diseased or unhealthy. This can include acute illness (which has a sudden onset and may improve or worsen over a short period of time) as well as chronic illness (which can present and progress slowly over a long period of time).
Mortality	This is the condition of being dead. It may be presented as the number of deaths in a population over time, either in general or due to a specific cause.
NCAs	Noise catchment areas.
NCG	Noise Criteria Guideline (various, as referenced in the report).

Term	Definition
NEPC	National Environment Protection Council.
NEPM	National Environment Protection Measure.
NHMRC	National Health and Medical Research Council.
NO_2	Nitrogen dioxide.
NOx	Nitrogen oxides.
NSW	New South Wales.
NSW EPA	NSW Environment Protection Authority.
OEH	NSW Office of Environment and Heritage.
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA).
PM	Particulate matter.
PM_1	Particulate matter of aerodynamic diameter 1 micrometre (µm) and less (termed ultrafine particles).
$PM_{2.5}$	Particulate matter of aerodynamic diameter 2.5 micrometres (µm) and less.
PM_{10}	Particulate matter of aerodynamic diameter 10 micrometres (µm) and less.
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].
Population	A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).
RBL	Rating Background Level.
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something would cause injury or harm.
ROM	Run-of-mine.
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
SEARs	Secretary's Environmental Assessment Requirements.
SEIFA	Socio-Economic Index for Areas.
SIA	Social Impact Assessment.
TCEQ	Texas Commission on Environmental Quality.
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
Toxicological profile	An assessment that examines, summarises, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.
Toxicology	The study of the harmful effects of substances on humans or animals.
TSP	Total suspended particulates.
UK	United Kingdom.
US	United States of America.
USEPA	United States Environmental Protection Agency.
WHO	World Health Organization.
$\mu g/m^3$	Micrograms per cubic metre.
µm	Micrometre.

Section 1. Introduction

1.1 Background

Maxwell Ventures (Management) Pty Ltd, a wholly owned subsidiary of Malabar Coal Limited (Malabar), is seeking consent to develop an underground coal mining operation, referred to as the Maxwell Project (the Project).

The Project is in the Upper Hunter Valley of New South Wales (NSW), east-southeast of Denman and south-southwest of Muswellbrook.

Underground mining is proposed within Exploration Licence (EL) 5460, which was acquired by Malabar in February 2018. Malabar also acquired existing infrastructure within Coal Lease (CL) 229, Mining Lease (ML) 1531 and CL 395, known as the "Maxwell Infrastructure". The Project would include the use of the substantial existing Maxwell Infrastructure, along with the development of some new infrastructure.

This assessment forms part of an Environmental Impact Statement (EIS) which has been prepared to accompany a Development Application for the Project in accordance with Part 4 of the NSW *Environmental Planning and Assessment Act, 1979* (EP&A Act).

More specifically this report presents a human health risk assessment (HHRA) relevant to the proposed Project.

1.2 SEARs

The Secretary's Environmental Assessment Requirements (SEARs) for this State Significant Development requires that a health risk assessment be prepared as part of the EIS:

> "a health risk assessment that considers the adverse effects from human exposure to acute and cumulative project related environmental hazards, in accordance with *Environmental Health Risk Assessment: Guidelines for assessing human health risk* [sic] *from environmental hazards*;"

On the basis of the above, the HHRA presented in this report has considered impacts to the surrounding community related to: air quality, noise, vibration and water.

1.3 Objectives

The overall objective of the HHRA is to provide an assessment of potential impacts to human health in relation to the Project, to specifically address the SEARs.

This report addresses impacts relevant to community health. No assessment of impacts to on-site workers is presented. Workplace health and safety is expected to be managed separately through application of the NSW *Work Health and Safety Act 2011* and NSW *Work Health and Safety (Mines and Petroleum Sites) Act 2013*, and associated regulations.

1.4 Approach and scope of works

The HHRA has been undertaken in accordance with the following guidance (and associated references as relevant):

- enHealth Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012) (as required in the SEARs).
- State Environmental Planning Policy No. 33 - Hazardous and Offensive Development (NSW Government 2014).
- National Environment Protection Council (NEPC) National Environment Protection (Ambient Air Quality) Measure (NEPM) (NEPC 2016).
- National Environmental Protection Measure – Assessment of Site Contamination including:
    - Schedule B1 Investigation Levels for Soil and Groundwater (NEPC 1999 amended 2013a).
    - Schedule B4 Guideline on Health Risk Assessment Methodology (NEPC 1999 amended 2013b).
    - Schedule B6 Guideline on Risk Based Assessment of Groundwater Contamination (NEPC 1999 amended 2013e).
    - Schedule B7 Guideline on Health-Based Investigation Levels (NEPC 1999 amended 2013d).
    - Schedule B8 Guideline on Community Consultation and Risk Communication (NEPC 1999 amended 2013c).
- Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales (NSW EPA 2016b)[1].
- NSW Noise Policy for Industry (NSW EPA 2017).
- National Health and Medical Research Council (NHMRC) Australian Drinking Water Guidelines (NHMRC 2011 updated 2018).
- Australian and New Zealand Guidelines for Fresh and Marine Water Quality (ANZG 2018).

Where relevant, additional guidance has been obtained from relevant Australian and International guidance, such as that available from the United States Environmental Protection Agency (USEPA) and the World Health Organisation (WHO), consistent with current industry best practice.

1.5 Available information

The HHRA has been prepared on the basis of information available for the Project, including information and data provided by other technical specialists, as detailed below:

- Todoroski Air Sciences Pty Ltd, 2019. Maxwell Project, Air Quality and Greenhouse Gas Assessment (AQGGA).
- Wilkinson Murray Pty Limited (Wilkinson Murray), 2019. Maxwell Project, Noise Impact Assessment.
- HydroSimulations, 2019. Maxwell Project, Groundwater Assessment.
- WRM Water & Environment Pty Ltd (WRM), 2019. Maxwell Project, Surface Water Assessment.

[1] NSW EPA – NSW Environment Protection Authority.

Section 2. Project description

2.1 Site description and location

The Project would involve an underground mining operation that would produce high quality coals over a period of approximately 26 years.

At least 75% of coal produced by the Project would be capable of being used in the making of steel (coking coals). The balance would be export thermal coals suitable for the new generation High Efficiency, Low Emissions power generators.

The Project would involve extraction of run-of-mine (ROM) coal from four seams within the Wittingham Coal Measures using the following underground mining methods:

- underground bord and pillar mining with partial pillar extraction in the Whynot Seam; and
- underground longwall extraction in the Woodlands Hill Seam, Arrowfield Seam and Bowfield Seam.

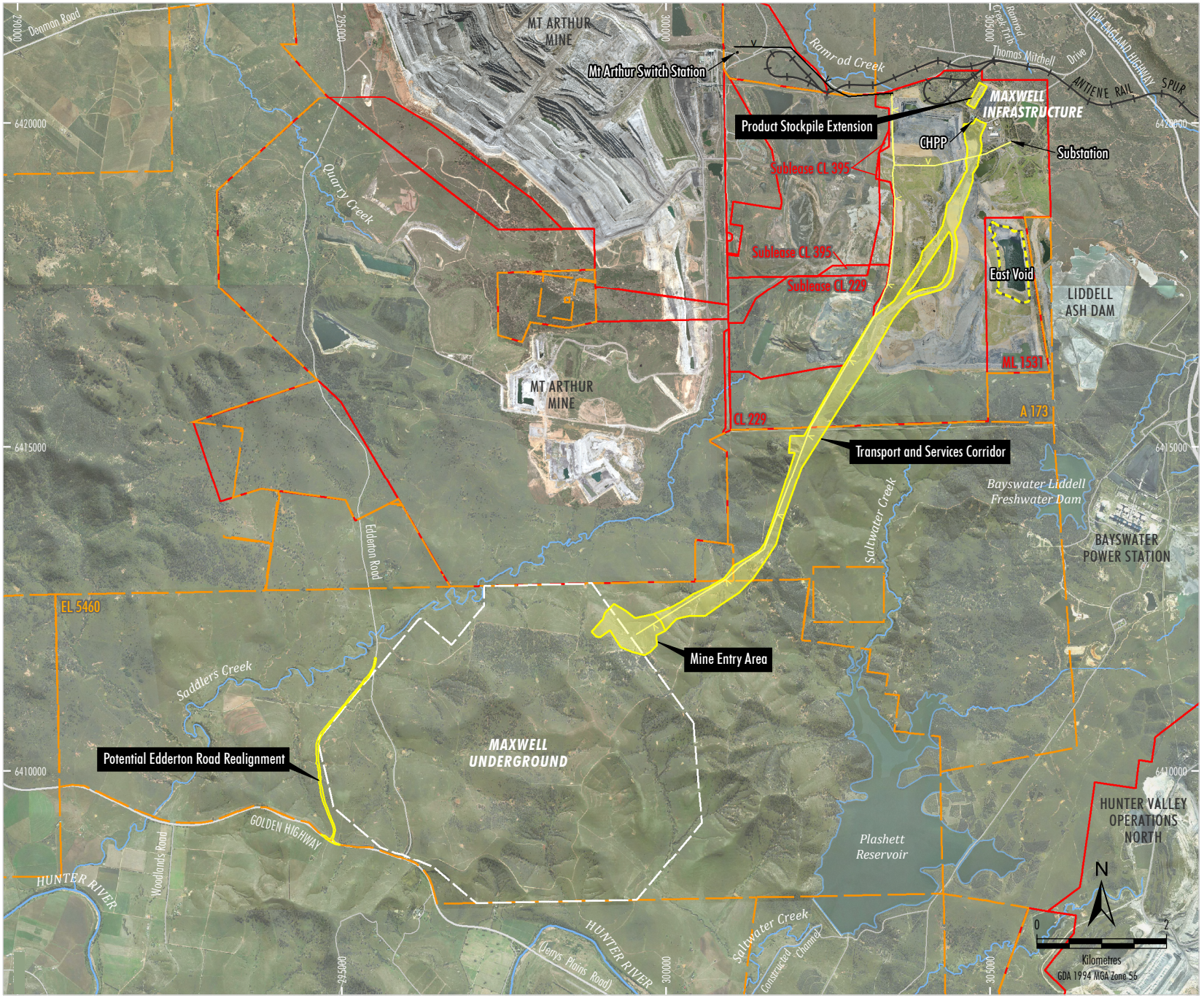
The substantial existing Maxwell Infrastructure would be used for handling, processing and transportation of coal for the life of the Project. The Maxwell Infrastructure includes an existing coal handling and preparation plant (CHPP), train load-out facilities and other infrastructure and services (including water management infrastructure, administration buildings, workshops and services).

A mine entry area would be developed for the Project in a natural valley in the north of EL 5460 to support underground mining and coal handling activities and provide for personnel and materials access.

ROM coal brought to the surface at the mine entry area would be transported to the Maxwell Infrastructure area. Early ROM coal would be transported via internal roads during the construction and commissioning of a covered overland conveyor system. Subsequently, ROM coal would be transported to the Maxwell Infrastructure area via the covered overland conveyor system.

The Project would support continued rehabilitation of previously mined areas and overburden emplacements areas within CL 229, ML 1531 and CL 395. The volume of the East Void would be reduced through the emplacement of reject material generated by Project coal processing activities and would be capped and rehabilitated at the completion of mining.

An indicative Project general arrangement showing the underground mining area and key infrastructure is provided on **Figure 2-1**. A detailed description of the Project is provided in the main document of the EIS.



LEGEND

- Railway
- Exploration Licence Boundary
- Mining and Coal Lease Boundary
- Indicative Extent of Underground Development
- Indicative Surface Development Area
- CHPP Reject Emplacement Area
- Proposed 66 kV Power Supply
- Proposed Ausgrid 66 kV Power Supply Extension#

Subject to separate assessment and approval.

Source: © NSW Department of Planning and Environment (2019); NSW Department of Finance, Services & Innovation (2019) Orthophoto Mosaic: 2018, 2016, 2011

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Project General Arrangement

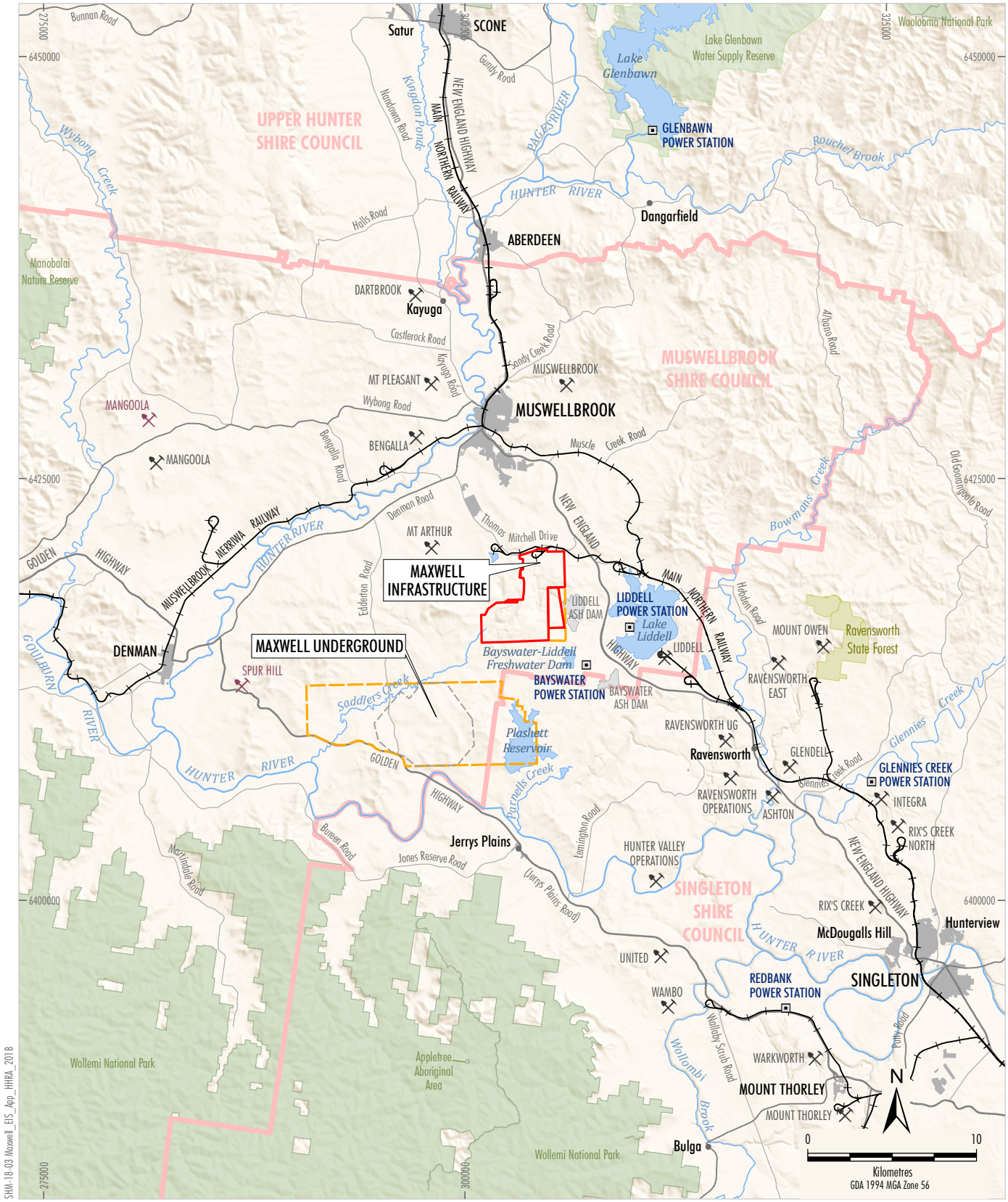
Figure 2-1

SHM-18-03 Maxwell_EIS_App_HHRA_202C

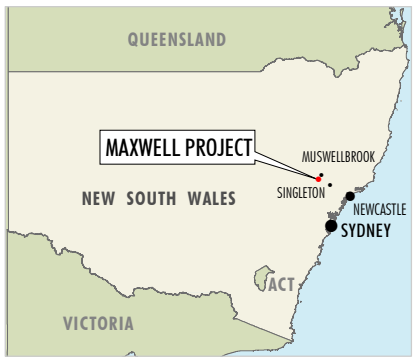
2.2 Local setting

The Project sits within the Upper Hunter Valley, in an area that includes Bayswater and Liddell Power Stations as well as a number of operating open cut coal mines. The local area also includes agricultural land, rural residences and the towns of Muswellbrook to the north, Jerrys Plains to the southeast and Denman to the west, refer to **Figure 2-2**.

The topography in the area of the Project comprises principally flat plains associated with the Hunter River, interspersed with low undulating to steeply sloped hills, ridges and crests over open farmland. Further away, the topographical features include the north to southwest aligned Hunter River floodplain to the west which then turns east and flows to the southeast down the valley and the foothills of the Barrington Tops mountain range to the northeast. The terrain features of the surrounding area, and the location of off-site properties are of importance in the assessment of various impacts relevant to the Project (including air quality and noise).



SHA-18-03 Maxwell_EIS_App_HHRA_201B



LEGEND

- Mining Operation
- Proposed Mining Operation
- Railway
- Local Government Boundary
- State Forest
- National Parks and Wildlife Service Estate
- Maxwell Project Exploration Licence Boundary
- Maxwell Project Mining and Coal Lease Boundary
- Indicative Extent of Underground Development

Source: © NSW Department of Planning and Environment (2019); NSW Department of Finance, Services and Innovation (20198); Office of Environment and Heritage NSW (2019)

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MAXWELL PROJECT

Regional Location

Figure 2-2

Section 3. Community profile

This section provides an overview of the community potentially impacted by the Project. It is noted that the key focus of this assessment is the local community surrounding the site.

The Project is situated in an area that includes existing agricultural and rural properties. Properties close to the Project include privately-owned and mine-owned properties.

Remote from the Project site are the larger population areas of Muswellbrook, Denman and Jerrys Plains.

The boundary of the community evaluated in this assessment has been determined based on modelling completed to evaluate key potential health impacts, specifically air quality and noise.

These assessments have focused on properties (individual receptors with receptor IDs) located within an area of 26 kilometres (km) x 26 km, which encompasses the Project along with rural residential properties to the north, south, west and southwest, as illustrated in **Figure 3-1** (as well as the figures in **Section 4**).

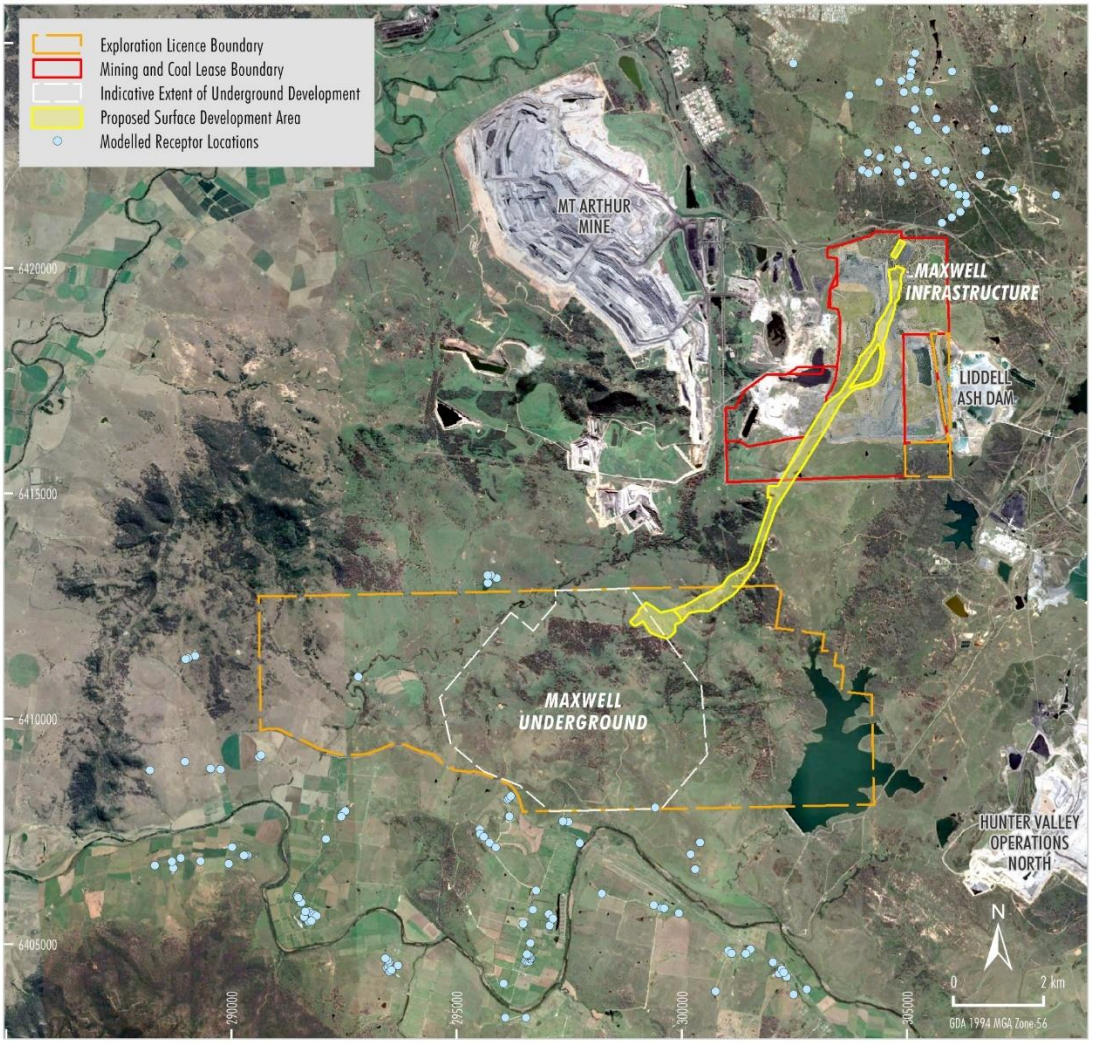


Figure 3-1: Study area (air quality and noise) (also refer to **Figure 5-2** for further detail on receptor IDs)

These receptors are located within the Muswellbrook and Singleton Local Government Areas (LGAs).

Table 3.1 presents a summary of the populations within the Muswellbrook and Singleton LGAs (based on 2016 Census and 2016 Socio-Economic data from the Australian Bureau of Statistics [ABS]) with comparison to NSW and Australia.

Table 3.1: Summary of populations surrounding the Project

Indicator	LGA		NSW	Australia
	Muswellbrook	Singleton		
Total population	16,086	22,987	7,480,231	23,401,892
Population 0 - 4 years	7.7% (1,242)	6.7% (1,537)	6.2% (465,135)	6.3% (1,464,779)
Population 5 - 19 years	20.9% (3,369)	21.8% (5,010)	18.3% (1,369,618)	18.5% (4,321,427)
Population 20 - 64 years	58.4% (9,397)	58.8% (13,521)	59.2% (4,427,843)	59.6% (13,938,918)
Population 65 years and over	12.9% (2,073)	12.7% (2,929)	16.3% (1,217,646)	15.7% (3,676,758)
Median age	35	36	38	38
Average household size	2.5	2.7	2.6	2.6
Unemployment (in 2016)	8.2%	6.1%	6.3%	6.9%
Unemployment (in December 2018)	7.7%	4.4%	4.3%	4.3%
Tertiary or technical institution	11.4%	13.5%	22.4%	22%
SEIFA IRSAD	917	974	--	--
SEIFA IRSAD rank	3	7	--	--
SEIFA IRSD	930	994	--	--
SEIFA IRSD rank	3	7	--	--
Indigenous	8.3%	5.7%	2.9%	2.8%
Born overseas	15.3%	16%	34.5%	26.3%

Most data presented in the table derived from the ABS 2016 Census (ABS 2016).
* Data presented for unemployment is based on available data (Australian Government 2018) to December 2018: https://docs.jobs.gov.au/documents/lga-data-tables-small-area-labour-markets-december-quarter-2018.
SEIFA IRSAD = index of socioeconomic advantage and disadvantage, rank relates to rank in Australia that ranges from 1 = most disadvantaged to 10 = least disadvantaged. Ranks lower than 5 are more disadvantaged than Australia on average.
SEIFA IRSD = index of socioeconomic disadvantage, rank relates to rank in Australia that ranges from 1 = most disadvantaged to 10 = least disadvantaged. Ranks lower than 5 are more disadvantaged than Australia on average.
Shading relates to comparison against NSW:

- statistic/data suggestive of a potential higher vulnerability within the population to health stressors.
- statistic/data suggestive of a potential lower vulnerability within the population to health stressors.
- statistics/data materially different to that of NSW and Australia, however this indicator is not a clear determinant of higher or lower vulnerability to health stressors.

Based on the population data available and presented in **Table 3.1**, the communities of Muswellbrook and Singleton have a similar age distribution as NSW and Australia, lower levels of tertiary and technical institution education and a lower percentage of people born overseas. Muswellbrook has a higher rate of unemployment, a higher proportion of indigenous population as well as lower rankings on the indices of socioeconomic disadvantage, (IRSAD and IRSD) (i.e. more disadvantaged), when compared with Singleton.

The indicators outlined in **Table 3.1** reflect the vulnerability of the population and its ability to adapt to environmental stresses. While it is not possible to provide more refined data for smaller pockets of these LGAs (in particular the properties evaluated in this assessment), in general the Muswellbrook population has a higher level of social disadvantage relative to the rest of NSW.

The health of the community is influenced by a complex range of interactive factors including age, socio-economic status, social capital, behaviours, beliefs and lifestyle, life experiences, country of origin, genetic predisposition and access to health and social care. The health indicators available and reviewed in this report (**Table 3.2**) generally reflect a wide range of these factors.

The population adjacent to the proposed site is relatively small and health data is not available that specifically relates to this population.

The Project is located within the Hunter New England Health District. This district covers a region of 131,785 square kilometres from Newcastle in the south to Tenterfield in the north, and past Narrabri in the west. There are approximately 920,000 people residing in the district, including residents of a major metropolitan centre (Newcastle) and regional communities. The populations of Muswellbrook and Singleton LGAs represent approximately 4% of the total population in the Hunter New England Health District.

Table 3.2 presents a summary of the general population health relevant to the area, based on currently available data. The table presents available information on health-related behaviours (i.e. key lifestyle and behaviours factors known to be important to health) and indicators for the burden of disease within the relevant LGAs (where available), the Hunter New England Health District and NSW. The values noted in bold are those utilised in this assessment.

Table 3.2: Summary of health indicators/data

Health indicator/data	Hunter New England	NSW
Health behaviours		
Adults - compliance with fruit consumption guidelines (2017)[1]	44.5%	46.4%
Adults - compliance with vegetable consumption guidelines (2017)[1]	8.7%	6.6%
Children - compliance with fruit consumption guidelines (2017) [1]	67.7%	66.8%
Children - compliance with vegetable consumption guidelines (2017) [1]	8.6%	7.4%
Adults - increased lifetime risk of alcohol related harm (2017) [1]	37.2%	32.4%
Adults - body weight (pre-obese) (2017) [1]	33.6%	32.5%
Adults - body weight (obese) (2017) [1]	25.1%	21%
Adults – sufficient physical activity (2017) [1]	51.8%	58.4%
Children – adequate physical activity (2017) [1]	29.3%	24.2%
Current smoker (2017) [1]	16%	15.2%
Burden of disease		
Morbidity - cardiovascular disease hospitalisations (2016/2017)[1]	1,713.6*	1,724.6*
Cardiovascular disease hospitalisations (ages 65 years and older)[2]	--	**Sydney = 9,235***
Morbidity – respiratory disease hospitalisations (2016/2017)[1]	1,707.5*	1750.0*
Respiratory disease hospitalisations (ages 65 years and older)[2]	--	**Sydney = 4,168***
Mortality – all causes, all ages (2016)[1]	606.4* **Muswellbrook LGA = 643.2*** Singleton LGA = 606.0*	534.6*
Mortality (all causes, ages 30 years and older)[2]	--	**Sydney = 1,026**
Mortality – respiratory (all ages)	**51.9***	49.1*
Adults - prevalence of high blood pressure (2013)[1]	32.9%	28.4%
Adult asthma – prevalence (2017)[1]	13.3%	10.9%

Health indicator/data	Hunter New England	NSW
Adolescent (2015 years) – prevalence of current asthma (2016/2017)[1]	15.8%	12.9%

* Rate per 100,000 population.

1 Data from NSW Health (2010) Statistics: http://www.healthstats.nsw.gov.au/.

2 Data for Sydney Metropolitan area for 2010 based on hospital statistics as reported for 2010 and population data from the ABS for 2011 (relevant to each age group considered) used in review of exposure and risks to inform recommendations for updating the NEPM (Golder 2013).

Shading relates to comparison against NSW:

- statistic/data suggestive of a potential higher vulnerability within the population to health stressors.
- statistic/data suggestive of a potential lower vulnerability within the population to health stressors.
- statistics/data materially different to that of NSW and Australia, however this indicator is not a clear determinant of higher or lower vulnerability to health stressors.

As described above, the Hunter New England Health District covers a large area. A detailed review of respiratory and cardiovascular disease relevant to the Upper Hunter[2] (and other) areas was completed in 2010 (Health 2010) to provide a more detailed review of health impacts that may be attributable to mining in the area. While the report identified that the data could not establish that differences observed in some health statistics could be attributable to air pollution or any other specific cause (including lifestyle factors), **Table 3.3** provides a summary of more localised data in relation to selected cardiovascular and respiratory health indicators relevant to the area. The values noted in bold are those utilised in this assessment.

Table 3.3: Summary of older health data for cardiovascular and respiratory health in more localised areas (Health 2010)

Health indicator	Singleton LGA	Muswellbrook LGA	Hunter New England	NSW
Cardiovascular disease (rate per 100,000)				
Hospitalisations (2004-2009)	2,347.7	2,869.6	2,096.3	2,102.5
Respiratory disease (rate per 100,000)				
Hospitalisations (2004-2009)	1,535.8	1,923.6	1,425.9	1,597.9
Asthma hospitalisations	153.3	237.8	161.2	190.4
Asthma hospitalisations (children aged under 15 years)	450.5	715.1	467.8	591.3
Asthma emergency department presentations (children aged under 15 years)	1,263*	**2,284***	--	--
Current asthma (children aged under 15 years)	18.2% - Upper Hunter[2]		17.5%	13.4%
Mortality all causes (all ages) 2004-2009	555.96 – Upper Hunter[2]		677.32	624.01

* Rate for Singleton and Muswellbrook postcode areas.

Shading relates to comparison against NSW:

- statistic/data suggestive of a potential higher vulnerability within the population to health stressors.
- statistic/data suggestive of a potential lower vulnerability within the population to health stressors.
- statistics/data materially different to that of NSW and Australia, however this indicator is not a clear determinant of higher or lower vulnerability to health stressors.

[2] The Upper Hunter cluster, as defined by NSW Health is the region defined by Merriwa, Murrurundi, Muswellbrook, Denman and Scone and includes the LGAs of Muswellbrook, Upper Hunter Shire and Liverpool Plains. It is noted that the region defined as Upper Hunter by the NSW EPA also includes Singleton.

In general, the 2010 NSW Health report identified the following, when compared with the rest of NSW:

- Muswellbrook LGA had higher levels of cardiovascular and respiratory hospitalisations, as well as asthma hospitalisations (adults and children).
- Singleton LGA had higher levels of cardiovascular hospitalisations, but the rates of hospitalisations for respiratory disease and asthma were not significantly different.

It is noted that the data considered in the NSW Health 2010 report is now dated. Not all the health statistics included in the report are publicly available as updated information, hence it is difficult to provide a more current data comparison for these LGAs.

Data presented in **Table 3.3**, along with data presented in **Table 3.1**, suggest some of the population in the areas surrounding the site may be more vulnerable to health-related impacts associated with the Project, than the general population of NSW. The underlying reasons for this increased vulnerability are expected to be complex, and may include a broad range of lifestyle, behaviour and environmental factors.

Section 4. Community engagement

Malabar has established ongoing community consultation programs in the region, which include Community Consultative Committees (CCCs) for the Maxwell Infrastructure and its adjoining Spur Hill Exploration Licence. Malabar has also sought specific input on the Project through consultation during the preparation of the EIS and Social Impact Assessment (SIA).

The Muswellbrook Shire Council, in its input to the SEARs, raised the following issue of relevance to the HHRA:

> *There are a number of operating Coal Mines in the Muswellbrook Shire Local Government Area and in close proximity to the Muswellbrook Township. The cumulative impact of dust and air pollution issues associated with mining operations is of increasing concern to the local community and Council...*

Other consultation conducted for the Project also identified that potential health impacts associated with cumulative dust levels is a concern amongst some local stakeholders. These concerns related to both airborne dust and accumulation of dust in rainwater tanks. These concerns are addressed in **Section 5** of this report.

The use of underground mining methods for the Project, as opposed to open cut mining methods, significantly mitigates concerns regarding potential dust impacts.

Other environmental issues of relevance to the HHRA raised in consultation included the following:

- The potential for noise impacts associated with recommencing activities at the Maxwell Infrastructure, including train noise (**Section 6**).
- The potential for impacts on private properties' access to water resources for irrigation, stock and domestic use (**Section 7**).

The potential for the Project to change demand for local health services as a result of population changes is addressed in the SIA.

Further information on the community engagement conducted in relation to the Project is outlined in the EIS and SIA.

Section 5. Health impacts: Air emissions

5.1 Approach

This section presents a review of impacts on health associated with predicted air emissions, relevant to the operation of the Project. The assessment presented has relied on the following:

- Todoroski Air Sciences, 2019. Maxwell Project, Air Quality and Greenhouse Gas Assessment. This report is referred to as the AQGGA.

The estimation of risk follows the general principles outlined in the enHealth document Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012).

5.2 Background on particulate matter

The focus of the AQGGA and this assessment of potential health impacts is the emissions to air of dust or particulate matter.

Dust or Particulate Matter (PM) is a widespread air pollutant (that has and will always be present in air) with a mixture of physical and chemical characteristics that vary by location (and source). Unlike many other pollutants, particulates comprise a broad class of diverse materials and substances, with varying morphological, chemical, physical and thermodynamic properties, with sizes that vary from <0.005 micrometres (μm) to >100 μm. Particulates can be derived from natural sources such as crustal dust (soil), pollen and moulds, and other sources that include combustion and industrial processes. Secondary particulate matter is formed via atmospheric reactions of primary gaseous emissions. The gases that are the most significant contributors to formation of secondary particulates include: nitrogen oxides, ammonia, sulfur oxides, and certain organic gases (derived from vehicle exhaust; combustion sources; and agricultural, industrial and biogenic emissions).

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

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

The common measures of particulate matter that are considered in the assessment of air quality and health risks are:

- **Total Suspended Particulates (TSP):** This refers to all particulates with an equivalent aerodynamic particle[3] size below 50 μm in diameter[4]. It is a gross indicator of the presence of dust with a wide range of sizes. The larger particles included in TSP (termed “inspirable”,

[3] The term equivalent aerodynamic particle is used to reference the particle to a particle of spherical shape and particle of density 1 gram per cubic centimetre (g/cm^3)

[4] The size, diameter, of dust particles is measured in micrometers.

comprise particles around 10 µm and larger) are more of a nuisance as they will deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory tract[5] and do not reach the lungs, hence, there is no potential for adverse health effects. Finer particles included in TSP (smaller than 10 µm, termed "respirable", as described below) tend to be transported further from the source and are of more concern with respect to human health as these particles can penetrate into the lungs. Not all of the dust characterised as TSP is relevant for the assessment of health impacts, and hence TSP as a measure of dust impact in the community, is difficult to directly include in this assessment. TSP can be used as a measure of dust that may give rise to nuisance impacts close to the source, where the heavier particles readily deposit out of the air causing dust to deposit onto surfaces (including vegetation and within homes). The deposition of dust is more often directly measured using dust deposition gauges, however, these data relate to an assessment of nuisance effects only. The assessment of potential health impacts relates to particles of a size where significant associations have been identified between exposure and adverse health effects.

- **PM_{10}, particulate matter below 10 µm in diameter, $PM_{2.5}$, particulate matter below 2.5 µm in diameter and PM_1, particulate matter below 0.1 µm in diameter (termed ultrafine particles):** These particles are small and have the potential to penetrate beyond the body's natural filter mechanisms of cilia and mucous in the nose and upper respiratory system, with the smaller particles able to further penetrate into the lower respiratory tract[6] and lungs. Once in the lungs, adverse health effects may occur that include mortality and morbidity, which may be associated with a range of adverse cardiovascular and respiratory effects (OEHHA 2002)[7].

It is well accepted nationally and internationally that monitoring for PM_{10} is a good method of determining the community's exposure to potentially harmful dust (regardless of the source) and is most commonly measured in local and regional air quality monitoring programs. Reliable methods for the monitoring of PM_{10} concentrations has been available for a long time and hence these data are most widely available in urban and rural areas.

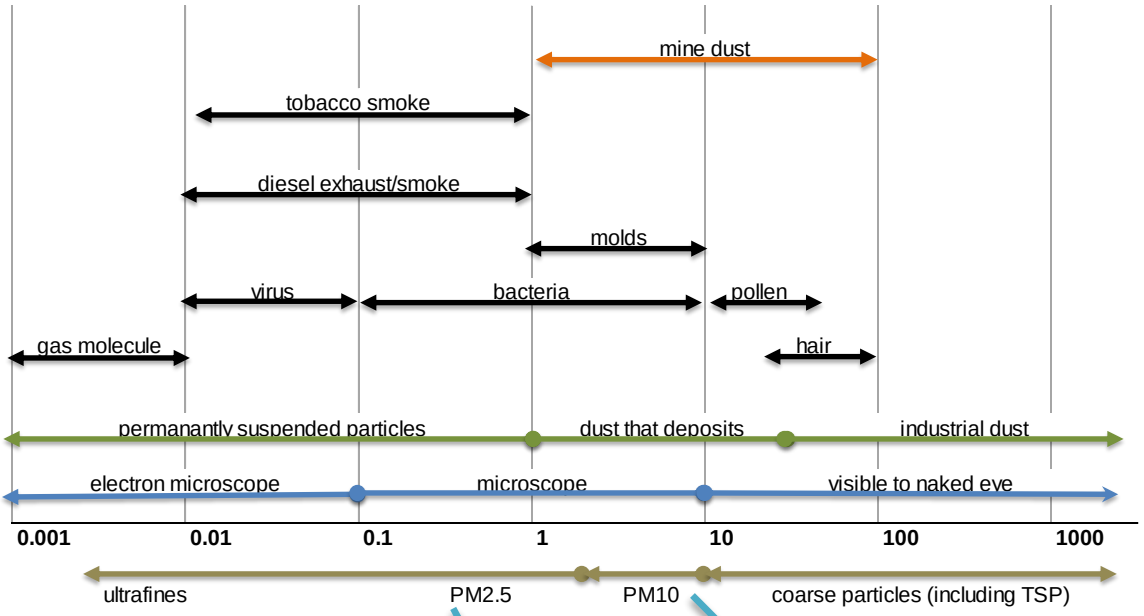
Smaller particles such as $PM_{2.5}$, however, are seen as more significant with respect to evaluating health effects, as a higher proportion of these particles penetrate deep into the lungs. Very fine particles, specifically ultrafine particles (PM_1 or $PM_{0.1}$), are also considered to be of importance for the assessment of health effects as these particles penetrate the deepest into the respiratory system.

Figure 5-1 provides a general illustration to provide some context in relation to the size of different particles (discussed above) and relevance/importance for the assessment of inhalation exposures.

[5] The upper respiratory tract comprises the mouth, nose, throat and trachea. Larger particles are mostly trapped by the cilia and mucosa and swept to the back of the throat and swallowed.

[6] The lower respiratory tract comprises the smaller bronchioles and alveoli, the area of the lungs where gaseous exchange takes place. The alveoli have a very large surface area and absorption of gases occurs rapidly with subsequent transport to the blood and the rest of the body. Small particles can reach these areas, be dissolved by fluids and absorbed.

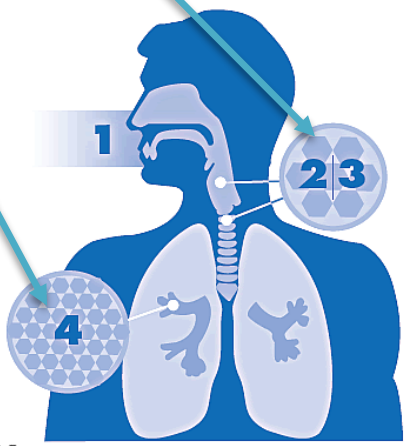
[7] OEHHA – Office of Environmental Health Hazard Assessment.



1 Particulate matter enters our respiratory (lung) system through the nose and throat.

2|3 The larger particulate matter (PM_{10}) is eliminated from the respiratory system through coughing, sneezing and swallowing.

4 $PM_{2.5}$ can penetrate deep into the lungs. It can travel all the way to the alveoli, causing lung and heart problems, and delivering harmful chemicals (where present) to the blood system.



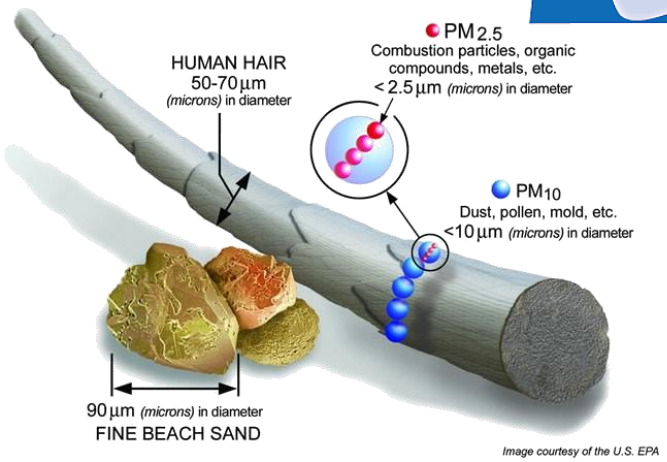


Figure 5-1: Illustrative Comparison of Relative Particle Sizes and Importance for Health

5.3 Summary of air modelling

5.3.1 Existing air quality

The main sources of particulate matter in the area surrounding the Project include mining, agriculture, cropping, commercial and industrial (including power generation) activities, urban activity and emissions from local anthropogenic activities such as motor vehicle exhaust and domestic wood heaters.

Data in relation to the existing air quality has been evaluated based on data from 16 stations that encompass the Maxwell Infrastructure and surrounding mining operations air quality monitoring networks as well as the Upper Hunter Air Quality Monitoring Network.

Data from these monitoring stations indicate the following:

- In relation to dust:
  - Reported dust deposition levels are well below the relevant guideline, suggesting dust deposition levels are generally good (or low) in the vicinity of the Project.
  - TSP monitoring, which includes all the large particulates which cannot be inhaled, reports levels below the relevant criteria, with higher results typically reported in the warmer months, likely as a result of drier conditions and higher levels of dust from agricultural and mining activities and more fire activity.
  - PM_{10} data shows similar variability and likely influences as per TSP. The PM_{10} data in the local area has some exceedances of the relevant guidelines. Data from monitors close to Muswellbrook and Jerrys Plains recorded the highest annual average levels, and the monitors further away from these urban centres reported lower annual average concentrations.
  - $PM_{2.5}$ data for monitoring stations near mining operations do not exceed the relevant guidelines, and do not show any seasonal variations. Monitoring data from Muswellbrook and Spur Hill have reported some exceedances of the relevant guidelines, suggesting influences from non-mining related sources such as wood fired heaters, vehicles and bushfires.
- The region reports low levels of nitrogen dioxide (NO_2), well below the current guideline, with the available data showing little seasonal variability.

5.3.2 Modelling impacts from the Project

Modelling of air quality impacts requires consideration of the local area, specifically the local terrain and meteorological conditions, as well as emissions to air from the various activities relevant to the Project.

The local meteorological conditions have been evaluated on the basis of data collected from the Maxwell Infrastructure Automatic Weather Station, along with data from 11 other local and regional meteorological stations. The influence of the local terrain of the Project areas and surrounding environments on meteorological conditions have also been taken into account.

Dust emissions from the Project have been estimated on the basis of emission factors for all the relevant activities, volumes to be handled and equipment proposed to be used. The emission factors have been locally developed and also derived from the USEPA. The assessment also considered emissions to air of oxides of nitrogen (NOx) from diesel powered equipment, with impacts of NO_2 evaluated in the assessment.

Modelling was undertaken using CALPUFF for three scenarios:

- Scenario 1, nominally Year 1, which mainly relates to construction activities as well as some operational activities.
- Scenario 2, nominally Year 3, which includes some construction activity as well as more operational activities.
- Scenario 3, nominally Year 4, which has the Project fully operational (and construction complete).

The modelling has also considered emissions to air from other nearby approved mining operations, and background (i.e. non-modelled) dust levels. Dust mitigation measures to be used within the Project have also been considered.

Impacts related to the Project have been evaluated at a number of receptors, representing privately-owned and mine-owned properties, as shown in **Figure 5-2**.

Figure 5-2: Location of sensitive receptors

5.4 Assessment of health impacts – particulates

5.4.1 Health effects

Evaluation of size alone as a single factor in determining the potential for particulate toxicity is difficult since the potential health effects are not independent of chemical composition. There are certain particle size fractions that tend to contain certain chemical components, such as metals or other organic compounds.

There is strong evidence to conclude (USEPA 2012; WHO 2003, 2013b) that fine particles (< 2.5 µm, $PM_{2.5}$) are more hazardous than larger ones (coarse particles), primarily on the basis of studies conducted in urban air environments where there is a higher proportion (as a percentage of all particulates) of fine particles and other gaseous pollutants present from fuel combustion sources, as compared to particles derived from crustal origins.

A significant amount of research, primarily from large epidemiology studies, has been conducted on the health effects of particulates with causal effects relationships identified for exposure to $PM_{2.5}$ (acting alone or in conjunction with other pollutants) (USEPA 2012). A more limited body of evidence suggests an association between exposure to larger particles, PM_{10} and adverse health effects (USEPA 2009; WHO 2003).

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

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

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

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

- Aggravation of existing respiratory and cardiovascular disease (as indicated by increased hospital admissions and emergency room visits).
- Changes in cardiovascular risk factors such as blood pressure.

- Changes in lung function and increased respiratory symptoms (including asthma).
- Changes to lung tissues and structure.
- Altered respiratory defence mechanisms.

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

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

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

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

5.4.2 Assessment of cumulative exposures to particulates

The assessment of cumulative exposures to $PM_{2.5}$ and PM_{10} is based on a comparison of the predicted cumulative concentrations to the current air quality standards and goals presented in the NEPM (NEPC 2016).

In relation to the current NEPM PM_{10} standard, the following is noted (NEPC 1998, 2010, 2014, 2016):

- The standard was derived through a review of appropriate health studies by a technical review panel of the NEPC where short term exposure-response relationships for PM_{10} and mortality and morbidity health endpoints were considered.
- Mortality health impacts were identified as the most significant and were the primary basis for the development of the standard.

- On the basis of the available data for key air sheds in Australia, the criterion of 50 micrograms per cubic metre (µg/m^3) was based on analysis of the number of premature deaths that would be avoided and associated cost savings to the health system (using data from the US). The development of the standard is not based on any acceptable level of risk and hence simply meeting the standard does not cover all aspects that need to be considered in terms of health impacts.
- The assessment undertaken considered exposures and issues relevant to urban air environments that are expected to also be managed through the PM_{10} standard. These issues included emissions from vehicles and wood heaters.

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

Table 5.1 presents a comparison of the current NEPC standards and goals with those established by the WHO (WHO 2005), the European Union (EU) and the USEPA (2012). The 2025 goals established by the NEPM for $PM_{2.5}$ (and adopted in this assessment) are similar to but slightly more conservative (health protective) than those provided by the WHO, EU and the USEPA. The NEPM PM_{10} guidelines are also similar to those established by the WHO and EU, however the guidelines are significantly lower than the 24-hour average guideline available from the USEPA.

Table 5.1: Comparison of particulate matter air quality goals

Pollutant	Averaging period	Criteria/guidelines/goals			
		NEPC	WHO (2005)	EU #	USEPA (2012)
PM_{10}	24-hour	50 µg/m^3	50 µg/m^3	50 µg/m^3 as limit value to be met, with 35 exceedances permitted each year	150 µg/m^3 (not to be exceeded more than once per year on average over 3 years)
	Annual	25 µg/m^3	20* µg/m^3	40 µg/m^3 as limit value to be met	NA
$PM_{2.5}$	24-hour	25 µg/m^3 20 µg/m^3 (goal for 2025)	25 µg/m^3	NA	35 µg/m^3 (98th percentile, averaged over 3 years)
	Annual	8 µg/m^3 7 µg/m^3 (goal for 2025)	10* µg/m^3	25 µg/m^3 as target value to be met from 2010 and limit value to be met from 2015. 20 µg/m^3 as a 3 year average (average exposure indicator) from 2015 with requirements for ongoing percentage reduction and target of 18 µg/m^3 as 3 year average to be attained by 2020	12 µg/m^3 (annual mean averaged over 3 years)

Current EU Air Quality Standards (EU 2015) available from **http://ec.europa.eu/environment/air/quality/standards.htm**

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

The air quality standards and goals for $PM_{2.5}$ and PM_{10} relate to total concentrations in the air (from all sources including the Project). This has been modelled as part of the AQGGA.

Table 5.2 summarises the maximum 24-hour average and annual average concentrations of $PM_{2.5}$ and PM_{10} estimated at any sensitive receptor relevant to Scenarios 1, 2 and 3, with comparison against the NEPC criteria.

Table 5.2: Review of cumulative PM concentrations

Location and scenario	Maximum 24-hour average concentration (µg/m^3)		Maximum annual average concentration (µg/m^3)	
	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}
Concentrations in the absence of the Project (all other cumulative sources) – maximum for all the receptors				
Scenario 1: Year 1	21.4	70	5.3	18.0
Scenario 2: Year 3	21.4	70	5.4	18.5
Scenario 3: Year 4	21.4	70	5.4	18.7
Cumulative concentrations with the Project – maximum from all the receptors				
Scenario 1: Year 1	21.5	70	5.4	18.4
Scenario 2: Year 3	21.4	70	5.5	18.8
Scenario 3: Year 4	21.4	70	5.5	19.5
Standards and goals	**25 (20 as goal for 2025)**	**50**	**8 (7 as goal by 2025)**	**25**

Review of **Table 5.2** indicates:

- The maximum 24-hour average concentrations of $PM_{2.5}$, for all scenarios considered, are below the relevant NEPC criteria. It is noted that the maximum concentrations predicted are dominated by existing background levels.
- The maximum 24-hour average concentrations of PM_{10}, for all scenarios considered, are above the relevant NEPC criteria at some locations for one or two days of the year. This is due to the elevated background level considered in the assessment on these days. It is noted that on these days the Project either does not contribute to the background, or only contributes up to approximately 6% of the total PM_{10} levels, which is a very minor contribution to the total. There were no additional days above the relevant NEPC criteria as a result of the Project.
- The total/cumulative annual average concentration of $PM_{2.5}$ and PM_{10}, for all scenarios considered, are below the relevant NEPC criteria.
- On this basis, there are no cumulative impacts of concern in relation to the Project.

5.4.3 Assessing incremental impacts associated with particulates

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

The available evidence does not suggest that there is a threshold below which health effects do not occur. Hence there are likely to be health effects associated with background levels of $PM_{2.5}$ and PM_{10}, even where the concentrations are below the current guidelines. Guidelines are currently available for the assessment of $PM_{2.5}$ and PM_{10} in Australia (NEPC 2002, 2003, 2016). These guidelines are not based on any acceptable level of risk, rather they are based on levels that are desirable in the community to balance background/urban sources with lowering impacts on health and cost savings in the health system.

A detailed assessment of potential health effects associated with exposure to a specific source, or a change in air quality as a result of a specific source has been undertaken. The assessment of impacts on health has utilised robust, published, quantitative relationships (exposure-response relationships) that correlate a change in $PM_{2.5}$ or PM_{10} concentration with a change in a health indicator. **Appendix A** presents an overview of the methodology adopted for using exposure-response relationships for the assessment of health impacts in a community.

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

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

- Long term exposure to $PM_{2.5}$ and PM_{10} and changes in all-cause mortality. This effect relates to exposures that may occur over all ages, however the most robust quantitative study used to calculate health risks and impacts relates to people aged 30 years and older.
- Short term exposure and changes to the rate of hospitalisations with cardiovascular and respiratory disease (equal or greater than 65 years of age). These effects have also been reported in other age groups, however the relationships between $PM_{2.5}$ and these effects are poor for younger age groups. The most robust relationships established are for people aged 65 years and older.

The above endpoints are robust and generally relate to $PM_{2.5}$. Exposure-response relationships are not as robust for PM_{10}, however, an assessment of PM_{10} has also been included for the key health endpoint (all-cause mortality), as particulate emissions derived from coal mining activities also include a significant proportion of particulates that are classified as PM_{10} but not $PM_{2.5}$.

The above endpoints are considered to be primary health indicators addressing the most significant health risks/impacts. Other effects and indicators reported in the literature are subsets of these and as a result have not been specifically presented. Notwithstanding, it is noted that in any community, asthma in children is typically of key concern and hence the following additional endpoint has also been considered:

- Short term exposure to $PM_{2.5}$ and changes in emergency department admissions for asthma in children aged 1–14 years. These effects have also been reported in other age groups, however it should be noted that the relationships between exposure to $PM_{2.5}$ and asthma effects are not as strong or robust for adults. The impact of air pollution on asthma has been the subject of a review by the Australian Institute of Health and Welfare (AIHW)

(AIHW 2010). This review makes it clear there are multiple contributors to the exacerbation of asthma in any individual (including respiratory infections, weather, seasonal allergens, indoor allergens, household chemicals, dietary factors and presence of smoking) so that isolating any one single factor is very difficult. Regardless of these many other factors, the presence of air pollution and its impacts on children with asthma are a common key concern in communities.

Table 5.3 presents a summary of the health endpoints considered in this assessment, the relevant health impact functions (from the referenced published studies) and the associated β coefficient relevant to the calculation of the relative risk (refer to **Appendix A** for details on the calculation of a β coefficient from published studies).

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

It should be noted that the approach adopted for assessing health impacts associated with $PM_{2.5}$ and PM_{10} relates to $PM_{2.5}$ and PM_{10} from any source. All sources of $PM_{2.5}$ and PM_{10} have the potential to impact on the health of individuals and the community. In rural and urban areas these sources include wood smoke, industrial emissions, vehicle emissions and sea salt. For example, Upper Hunter Valley Particle Characterisation Study (Hibberd et al. 2013) identified woodsmoke as the largest contributor to annual $PM_{2.5}$ at 30% in Muswellbrook, with the contribution of woodsmoke to $PM_{2.5}$ increasing to 62% in winter.

Table 5.3: Adopted health impact functions and exposure-responses relationships – $PM_{2.5}$ and PM_{10}

Health endpoint	Exposure period	Age group	Published relative risk [95 confidence interval] per 10 µg/m^3	Adopted β coefficient (as per cent) for 1 µg/m^3 increase in PM	Reference
$PM_{2.5}$: Mortality, all causes	Long term	≥30 years	1.06 [1.04-1.08]	0.0058 (0.58)	Relationship derived for all follow-up time periods to the year 2000 (for approx. 500,000 participants in the US) with adjustment for seven ecologic (neighbourhood level) covariates (Krewski et al. 2009). This study is an extension (additional follow-up and exposure data) of the work undertaken by Pope et al. (Pope et al. 2002), is consistent with the findings from California (1999–2002) (Ostro et al. 2006) and is more conservative than the relationships identified in a more recent Australian and New Zealand study (EPHC 2010)[8]

[8] EPHC – Environmental Protection and Heritage Council.

Health endpoint	Exposure period	Age group	Published relative risk [95 confidence interval] per 10 µg/m^3	Adopted β coefficient (as per cent) for 1 µg/m^3 increase in PM	Reference
PM_{10}: Mortality, all causes	Short term	All ages	1.006 [1.004-1.008]	0.0006 (0.06)	Based on analysis of data from European studies from 33 cities and includes panel studies of symptomatic children (asthmatics, chronic respiratory conditions) (Anderson et al. 2004)
$PM_{2.5}$: Cardiovascular hospital admissions	Short term	≥65 years	1.008 [1.0059–1.011]	0.0008 (0.08)	Relationship established for all data and all seasons from US data for 1999 to 2005 for lag 0 (exposure on same day) (strongest effect identified) (Bell 2012; Bell et al. 2008)
$PM_{2.5}$: Respiratory hospital admissions	Short term	≥65 years	1.0041 [1.0009–1.0074]	0.00041 (0.041)	Relationship established for all data and all seasons from US data for 1999 to 2005 for lag 2 (exposure 2 days previous) (strongest effect identified) (Bell 2012; Bell et al. 2008)
$PM_{2.5}$: Asthma (emergency department admissions)	Short term	1–14 years	–	0.00148 (0.148)	Relationship established from review conducted on Australian children (Sydney) for the period 1997 to 2001 (Jalaludin et al. 2008)

The assessment of health impacts for a population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004) (also outlined in **Appendix A**) where the exposure-response relationships (presented in **Table 5.3**) have been directly considered.

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

- Estimates of the changes in $PM_{2.5}$ and PM_{10} exposure levels or concentrations due to emissions from the Project.
- Baseline incidence of the key health endpoints that are relevant to the population exposed. This is specific to populations in the Upper Hunter Valley.
- Exposure-response relationships expressed as a percentage change in health endpoint per micrograms per cubic metre change in particulate matter exposure (see **Table 5.3**).

The change in incidence of each health endpoint relevant to changes in population exposure to $PM_{2.5}$ and PM_{10} has been calculated on the following basis:

- The average change in $PM_{2.5}$ and PM_{10} concentration over all receptors has been determined.
- A change in the number of cases associated with the change in $PM_{2.5}$ and PM_{10} impact evaluated in the population within the study area has been calculated (refer to **Appendix A** for details on the methodology). The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered and the population (for the relevant age groups) present in the area assuming each receptor has the average persons per household relevant to the LGAs evaluated.

Based on the above modelling and assumptions, health impacts associated with the Project for all three scenarios have been evaluated.

Table 5.4 presents a summary of the calculated impact of exposure to $PM_{2.5}$ and PM_{10} from the Project for the scenarios considered. The calculated incremental risks presented relate to the maximum risk for all receptors evaluated. Calculations of incremental risk for each individual receptor are included in **Appendix B**.

The incremental risk relates to the maximum individual risk within the community or area evaluated and does not consider the size of the population. The calculated population incidence reflects the increased risks for all members of the population in terms of the number of cases.

Assessment of what constitutes an acceptable risk level (as an individual risk for members of the community) for changes in exposure to $PM_{2.5}$ and PM_{10} within a community is a complex issue. For new and expanding developments in NSW, NSW EPA (NSW EPA 2016b) states that the following should be considered in relation to carcinogenic risks, which is inferred to also apply to other non-threshold risks:

- Unacceptable risks are ≥ 1 x 10^{-4}, or 1 in 10,000 and where risk management measures are required to be implemented.
- Acceptable risks are in the range <1x10^{-4} (1 in 10,000) and >1x10^{-6} (1 in 1,000,000) and where best practice is required.
- Negligible risks are ≤1x10^{-6} or 1 in 1,000,000.

Table 5.4: Population health impacts associated with exposure to $PM_{2.5}$ and PM_{10}

Location	Population incidence (increase in number of cases in population per year) and calculated incremental risk				
	$PM_{2.5}$				PM_{10}
	Mortality (all causes, ≥30 years)	Cardiovascular hospitalisations (≥65 years)	Respiratory hospitalisations (≥65 years)	Asthma ED admissions (1-14 years)	Mortality (all causes, all ages)
Population incidence – population in study area					
Scenario 1 – Year 1	0.00019	0.00005	0.00001	0.00004	0.000044
Scenario 2 – Year 3	0.00014	0.00004	0.000009	0.00003	0.000074
Scenario 3 – Year 4	0.00012	0.00003	0.000007	0.00003	0.00011
Incremental risk – maximum from all receptors					
Scenario 1 – Year 1	4x10^{-6}	5x10^{-6}	1x10^{-6}	2x10^{-6}	1x10^{-6}
Scenario 2 – Year 3	5x10^{-6}	6x10^{-6}	1x10^{-6}	3x10^{-6}	1x10^{-6}
Scenario 3 – Year 4	5x10^{-6}	7x10^{-6}	2x10^{-6}	3x10^{-6}	3x10^{-6}

Review of **Table 5.4** and **Appendix B** indicates the following:

- The calculated population health incidence values are very low and would never be measurable within the population surrounding the Project.
- There are no incremental risks that would be considered to be unacceptable. The maximum individual risk would not be considered to be unacceptable. Calculated risks for all individual receptors indicate population average risks in the order of 1x10^{-6} which are considered to be negligible.

- On the basis of the above, there are no health impacts of concern in relation to potential emissions of $PM_{2.5}$ and PM_{10} from the Project.

5.4.4 Assessment of dust deposition

Dust deposition is generally considered to pose an aesthetic impact, as it relates to the deposition of predominantly coarse particles (i.e. particles too large to be of concern in relation to inhalation) onto surfaces. Dust deposition is measured by Malabar in areas surrounding the site, with the existing levels (from 2013-2017) all below the relevant guideline of 4 g/m^2/month (as an annual average), which is an amenity-based guideline. This includes amenity issues related to rainwater tanks.

The monitoring of dust deposition reports the deposition of dust from all sources. While no specific study has been undertaken in the Upper Hunter region, the NSW EPA has conducted a dust deposition study in Newcastle (NSW EPA 2016a), to address concerns about the presence of black visible dust and to better understand the composition of dust deposited. Dust deposition levels in 2014-2015 were below the relevant guideline and principally comprised soil or rock (40% to 90%), with coal comprising an average of 10% (0% to 25%). The remainder comprised insects and plant debris, rubber dust, soot, salt, fly ash, alumina, paint and miscellaneous fibres. Although this study is not directly applicable to the Project, it indicates that the presence of black visible dust cannot be entirely attributed to the presence of coal dust.

The potential for any coal dust to contribute to, and impact on, the quality of water within rainwater tanks depends on the likelihood of coal dust depositing on the roof and being washed into the tank, the potential for leaching of trace elements into tank water and the quality of water at the point of use (i.e. as used form taps) (Lucas et al. 2009).

The AQGGA considered dust deposition that may occur as a result of the Project. The maximum predicted total annual average dust deposition (from all sources including the Project) at all receptors considered is 1.8 g/m^2/month, well below the guideline of 4 g/m^2/month. The maximum increase in dust deposition as a result of the Project, at any of the receptors surrounding the Project is less than 0.1 g/m^2/month, which is considered to be a negligible contribution to existing dust deposition levels in the area. This represents a negligible impact to dust deposition and accumulation of dust in rainwater tanks.

The study conducted by Lucas et al. (2009) evaluated the potential for trace elements in coal dust (from an Australian coal terminal) to leach into rainwater. This study concluded that negligible amounts of trace elements from coal dust leached into rainwater, and the presence of coal dust resulted in the removal of trace elements present in the initial rainwater. Any concentrations leached were below the Australian Drinking Water Guidelines and were of no concern to human health. Hence if there were some coal dust deposited onto a roof (which will be negligible as per the discussion above), there would be negligible impacts to health where tank water was used for drinking water.

It should be noted that NSW Health's information on Rainwater Tanks (https://www.health.nsw.gov.au/environment/water/Pages/rainwater.aspx) provides advice on how to maintain water tanks for safe drinking for those landholders concerned about drinking water quality.

5.5 Assessment of health impacts – nitrogen dioxide

5.5.1 General

Nitrogen oxides (NOx) refer to a collection of highly reactive gases containing nitrogen and oxygen, most of which are colourless and odourless. Nitrogen oxide gases form when fuel is burnt. Motor vehicles, along with industrial, commercial and residential (e.g. gas heating or cooking) combustion sources, are primary producers of nitrogen oxides.

In terms of health effects, nitrogen dioxide (NO_2) is the only oxide of nitrogen that may be of concern (WHO 2000). NO_2 is a colourless and tasteless gas with a sharp odour. NO_2 can cause inflammation of the respiratory system and increase susceptibility to respiratory infection. Exposure to elevated levels of NO_2 has also been associated with increased mortality, particularly related to respiratory disease, and with increased hospital admissions for asthma and heart disease patients (WHO 2013a). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of NO_2 (Morgan, Broom & Jalaludin 2013; NEPC 2010). The health effects associated with exposure to NO_2 depend on the duration of exposure as well as the concentration.

In relation to potential exposures to NO_2, the following should be considered:

- Whether the evidence suggests that associations between exposure to NO_2 concentrations and effects on health are causal: The most current review undertaken by the USEPA (USEPA 2015) specifically evaluated evidence of causation. The review identified that a causal relationship existed for respiratory effects (for short term exposure, with long term exposures also likely to be causal). All other associations related to exposure to NO_2 (specifically cardiovascular effects, mortality and cancer) were considered to be suggestive.
- Whether the reported associations are distinct from, and additional to, those reported and assessed for exposure to particulate matter: Co-exposures to NO_2 and particulate matter complicates review and assessment of many of the epidemiology studies as both these air pollutants occur together in urban areas. There is sufficient evidence (epidemiological and mechanistic) to suggest that some of the health effect associations identified relate to exposure to NO_2 after adjustment/correction for co-exposures with particulate matter (COMEAP 2015)[9].
- Whether the assessment of potential health effects associated with exposure to different levels of NO_2 can be undertaken on the basis of existing guidelines, or whether specific risk calculations are required to be undertaken: The current guidelines in Australia for the assessment of NO_2 in air relate to cumulative (total) exposures and adopt criteria that are considered to be protective of short and long-term exposures. However, for the assessment of impacts from a specific emission source, where background is not being considered, the exposure-response relationships relevant to NO_2 require consideration.

[9] COMEAP – Committee on the Medical Effects of Air Pollutants.

The evidence base supports quantification of effects of short-term exposure to NO_2, using the averaging time as in the relevant studies. The strongest evidence is for respiratory effects, in particular exacerbation of asthma, with some support also for all-cause mortality.

5.5.2 Assessment of cumulative exposures to nitrogen dioxide

The NEPC ambient air quality guideline for the assessment of acute (short-term) exposures to NO_2 relates to the maximum predicted total (cumulative) 1-hour average concentration in air. The guideline of 246 µg/m^3 (or 0.12 parts per million [ppm]) is based on a lowest-observed-adverse-effect level (LOAEL) of 409–613 µg/m^{3}derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of two to protect susceptible people (i.e. asthmatic children) was applied to the LOAEL (NEPC 1998). On this basis, the NEPC acute guideline is protective of adverse health effects in all individuals, including sensitive individuals.

The NEPC ambient air quality standard for the assessment of chronic (long-term) exposures to NO_2 relates to the maximum predicted total (cumulative) annual average concentration in air. The standard of 62 µg/m^3 (or 0.03 ppm) is based on a LOAEL of the order of 40–80 parts per billion by volume (around 75–150 µg/m^3). This relates to the early and middle childhood years when exposure can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent ‘colds’, a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school.

An uncertainty factor of two was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 parts per billion by volume (38–75 µg/m^3) (NEPC 1998). On this basis, the NEPC standard is protective of adverse health effects in all individuals, including sensitive individuals.

Table 5.5 summarises the maximum predicted cumulative 1-hour average and annual average concentrations of NO_2, using a conservative 25% rate of conversion of NOx to NO_2. Background NO_2 monitoring data is based on monitoring Muswellbrook township in 2015. It is noted that the background levels measured in Muswellbrook are likely to be higher than the levels for the majority of receptor locations, as there are many densely positioned sources of NO_X in Muswellbrook, such as motor vehicles. The measured levels are considered to be conservative and likely to overestimate actual levels.

Table 5.5: Review of potential acute and chronic health impacts – nitrogen dioxide

Location and scenario	Maximum 1-hour average concentration (µg/m^3)	Maximum annual average concentration (µg/m^3)
Cumulative concentrations with the Project – maximum at all the receptors		
Scenario 1: Year 1	99.6	40.1
Scenario 2: Year 3	94.3	39.8
Scenario 3: Year 4	93.1	39.7
Standards and goals	**246**	**62**

Based on **Table 5.5**, there are no cumulative concentrations of NO_2 that exceed the relevant guidelines and hence there are no cumulative exposure issues for the local community.

To further address potential risks to human health that may be associated with population exposures and localised changes in NO_2 that relate to the Project, incremental risk calculations have been undertaken and are presented in **Section 5.4.3**.

5.5.3 Assessment of incremental impacts

The approach adopted for the assessment of exposures and impacts is consistent with that adopted for particulates as outlined above (and **Appendix A**). This involves the calculation of a change in relative risk, and the change in incidence, or the number of cases, that occur in the community.

Table 5.6 presents a summary of the health endpoints considered in this assessment, the β coefficient relevant to the calculation of a relative risk. The coefficients adopted for the assessment of impacts on mortality and asthma emergency department admissions are derived from the detailed assessment undertaken for the current review of health impacts of air pollution undertaken by NEPC (Golder 2013) and are considered to be robust.

Table 5.6: Adopted exposure-responses relationships for assessment of changes in nitrogen dioxide concentrations

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

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

Table 5.7 presents a summary of the calculated impact of exposure to NO_2 from the Project for the scenarios considered. The calculated incremental risks presented relate to the maximum risk for all receptors evaluated. Calculations of incremental risk for each individual receptor are included in **Appendix C**. It is not possible to calculate a population incidence relevant to changes in NO_2 as only the maximum impacts predicted from the Project for each scenario is available.

Table 5.7: Population health impacts associated with exposure to nitrogen dioxide

Location	Calculated incremental risk		
	Mortality (all causes, all ages)	Respiratory mortality (all ages)	Asthma ED admissions (1-14 years)
Incremental risk – maximum from all receptors			
Scenario 1 – Year 1	$5x10^{-6}$	$9x10^{-7}$	$1x10^{-5}$
Scenario 2 – Year 3	$1x10^{-6}$	$2x10^{-7}$	$3x10^{-6}$
Scenario 3 – Year 4	$4x10^{-7}$	$7x10^{-8}$	$8x10^{-7}$

Review of **Table 5.7** and **Appendix C** indicates the following:

- There are no incremental risks relevant to NO_2 impacts from the Project that would be considered to be unacceptable. It is noted that NO_2 impacts are reduced over time where there are fewer diesel emissions from construction activities. The maximum individual risk for the operational phase of the Project (as represented by Scenario 3 – Year 4) are all considered to be negligible.
- On the basis of the above there are no health impacts of concern in relation to potential emissions of NO_2 from the Project.

5.6 Uncertainties

It is expected that the assessment of health impacts in relation to changes in air quality, associated with the Project, will be conservative. This is due to the incorporation of a number of conservative assumptions in the modelling of air quality impacts (specifically the continual operation of the proposed mine at maximum extraction rates, the use of conservative emission rates for the equipment proposed to be used, assuming locomotives are continually idling on the rail loop and the approach adopted for the estimation of nitrogen dioxide concentrations as a proportion of oxides of nitrogen).

In addition, the assessment of potential health impacts has assumed that the off-site community remains at home (or on their property) all day, every day for a lifetime. This will overestimate actual exposures where residents will spend time away from the home, and the changes in air quality evaluated remain the same for a lifetime.

As a result of the above, the risk calculations presented are considered to be conservative.

5.7 Outcomes of health risk assessment

Table 5.8 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in air quality, associated with the Project, on community health.

Table 5.8: Summary of health risks – air quality

Air emissions	
Impacts	Based on the available data and information in relation to emissions of dust and nitrogen dioxide from the Project, potential impacts on the health of the community have been assessed. The impact assessment has concluded there are no health risk issues of concern relevant to the Project (including construction and operational phases).
Mitigation	Development of an Air Quality Management Plan prior to commencement of operations at the Project that would outline the measures to manage air emissions (consistent with the AQGGA) and include aspects such as key performance indicators, monitoring methods, response mechanisms, compliance reporting and complaints management.

Section 6. Health impacts: Noise

6.1 Background

This section presents a review and further assessment of impacts on health associated with noise, relevant to the Project. The assessment presented has relied on the information provided in the following report:

- Wilkinson Murray 2019, Maxwell Project, Noise Impact Assessment.

The noise impact assessment has considered the same receptors (or receivers) that were evaluated within the air quality assessment (refer to **Figure 5-1**). These receptors (146 in total) include privately owned and mine-owned properties/premises surrounding the Project. These receptors are located in areas with different existing noise environments. Properties to the north experience noise from the Mt Arthur Mine and surrounding road network, and properties to the south experience a quieter noise environment with less road noise and minimal industrial noise.

Existing, or background, noise levels in the community, at the receptors evaluated, have been determined on the basis of available noise monitoring data. The background noise levels adopted in the assessment, termed a Rating Background Level (RBL, which relates to noise over a 15-minute period) are 35 decibels (A-weighted) (dBA) during the day, and range from 30 to 33 dBA during the evening and night.

6.2 Health impacts associated with noise

Environmental noise has been identified (I-INCE 2011; WHO 2011, 2018)[10] as a growing concern because it has negative effects on quality of life and wellbeing and has the potential for causing harmful physiological health effects. With increasingly urbanised or developed societies, impacts of noise on communities have the potential to increase over time.

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

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

[10] I-INCE – International Institute of Noise Control Engineering.

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

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

Within a community the severity of the health effects of exposure to noise and the number of people who may be affected are schematically illustrated in **Figure 6-1**.

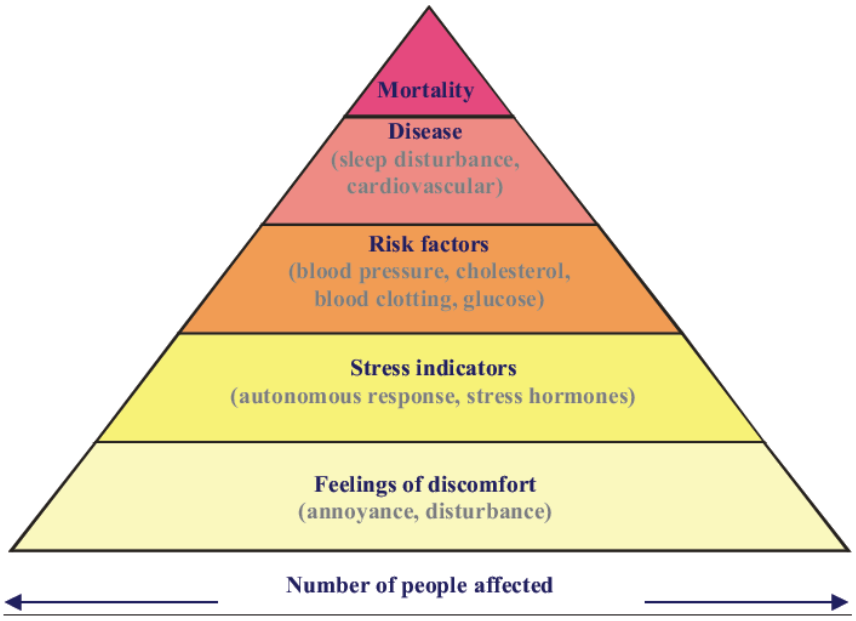


Figure 6-1: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011)

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

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

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

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

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

In relation to the available noise guidelines, the most recent review of noise by the WHO (WHO 2018) provided an update in relation to environmental noise guidelines (and targets) that more specifically relate to transportation (road, rail and air), wind turbines and leisure noise sources. The more comprehensive guideline levels for noise (related to all sources) remain the older WHO guidelines (WHO 1999) and night noise guidelines (WHO 2009).

6.3 Review of the noise guidelines adopted

Noise guidelines adopted in the Noise Impact Assessment are those outlined in the Noise Policy for Industry (NPfI) (NSW EPA 2017), which indicate that intrusive noise from a specific industrial source should not exceed the RBLs by more than 5 dBA. In addition, consideration has also been made to noise amenity, with the project noise trigger levels adopted based on the lower noise criteria relevant to intrusiveness and amenity. The noise trigger levels adopted were $LA_{eq,15\text{-}minute}$ of 40 dBA during the day and 35-38 dBA during the evening and night. These noise trigger levels are sufficiently low to be protective of health, based on available guidance from the WHO (WHO 1999, 2011). The NPfI provides guidance on the interpretation of noise impacts in relation to these trigger levels, particularly in relation to predicted/estimated changes in noise levels.

Noise amenity criteria (noted above) as $LA_{eq,15\text{-}minute}$ are 48 dBA during the day, 43 dBA during the evening and 38 dBA during the night-time period. The noise amenity criteria are more specifically used to evaluate cumulative noise from a number of industrial sources. These criteria will remain protective of health, including annoyance and sleep disturbance where they relate to outside noise levels (WHO 1999, 2009).

Maximum noise levels were also established based on the NPfI guidance (NSW EPA 2017). The maximum noise criteria are set to protect residence from sleep disturbance and for this Project, an LA_{Fmax} of 52 dBA is relevant to the night-time period. This maximum noise level is sufficiently low to be protective of health, based on available guidance from the WHO (WHO 1999).

Blasting impacts have been evaluated in accordance with criteria established to protect human annoyance and structural damage (NSW DEC 2006)[11]. Provided the human comfort criteria are met, there would be no concern in relation to health impacts.

Road traffic noise was assessed on the basis of the NSW Road Noise Policy (NSW DECCW 2011)[12], as it applies to existing residence affected by additional traffic. This provides a guideline of 60 dBA as $LA_{eq,15\ hour}$ (day and evening) and 55 dBA as $LA_{eq,9\ hour}$ (night). These guidelines are higher than the health based goals relevant to road noise traffic from the WHO (WHO 2018) but consistent with the upper end of noise criteria established in previous WHO guidelines for outdoor noise predictions (WHO 1999, 2009).

Rail noise was assessed on the basis of the Rail Infrastructure Noise Guideline (NSW EPA 2013) which provides acceptable noise levels in rural areas, as 50 dBA $LA_{eq,day}$ during the day, 45 dBA $LA_{eq,evening}$ during the evening and 40 dBA $LA_{eq,night}$ at night. These guidelines are lower than the health based goals relevant to rail noise from the WHO (WHO 2018).

Construction noise criteria have been adopted from the Interim Construction Noise Guideline (ICNG) (NSW DECC 2009)[13] which provide management levels relevant to the assessment of noise impacts above the RBL during standard hours (guideline is RBL + 10 dBA = 45 dBA for the Project) and outside standard hours (guideline is RBL + 5 dBA = 35-38 dBA for evening and night and 40 dBA for day), with noise levels (total noise from all sources) above 75 dBA during standard hours considered to be highly noise affected. While these criteria may result in some construction noise being noticeable, the noise criteria adopted for the Project will be protective of health, including annoyance and sleep disturbance, where they relate to outside noise levels (WHO 1999, 2009).

6.4 Review and assessment of health impacts from noise

6.4.1 Construction noise

Assessment of noise impacts during construction involved consideration of the relevant construction activities (equipment used, hours of use and location of use), including blasting (where required).

For potential blasting activities the distance required to achieve compliance with the guideline (with human comfort as the lowest guideline considered) is 1.5 km (Wilkinson Murray 2019). The closest private residence is 4.7 km. On this basis there are no heath related issues of concern in relation to potential blasting activities.

[11] DEC – NSW Department of Environment and Conservation.

[12] DECCW – NSW Department of Environment, Climate Change and Water.

[13] DECC – NSW Department of Environment and Climate Change.

In relation to the assessment of noise generated during a range of construction activities, these have been assessed separately as well as during key Project years (that include construction and varying levels of operations). The combined assessment is further discussed in **Section 6.4.2**. The assessment of construction noise was undertaken using the Environmental Noise Model (ENM), which provides noise predictions at each individual receptor – as an outdoor noise level.

Assessment of construction noise impacts only (i.e. not including operational noise) did not identify any receptors where noise impacts would result in exceedance of the relevant construction noise guidelines. Noise levels at one receptor were predicted to exceed the 'Noise Affected' management level by 1 dBA if works occurred outside of standard hours and during noise-enhancing meteorological conditions, which is unlikely to occur.

As the construction noise guidelines adopted for this assessment are protective of health, there are no health impacts of concern in relation to construction activities (where assessed separately). It is expected, however that some construction noise may be noticeable at some receptors.

6.4.2 Operational noise

Approach

The operational noise assessment has considered noise impacts from the Project operations as well as road and rail noise impacts. The noise assessment has utilised the ENM (Environmental Noise Model) that provides predictions of noise impacts at each modelled receptor as an outdoor noise level. Noise modelling considered operations during:

- Year 1, which includes construction works and some operations;
- Year 3, which also includes some construction works as well as more operational activities; and
- Year 4, which has the Project fully operational (and construction complete).

Assessments conducted for Year 1 and Year 3 include construction activities that would be indistinguishable from operational mining and coal processing activities, and would be representative of general noise emissions throughout the year.

Activities that are proposed to be undertaken during these Project years, including the time and location of operation, and sound power levels generated by these equipment/activities, have been considered in the noise model, along with terrain and meteorological conditions.

The noise modelling undertaken has been conducted in an iterative manner, incorporating and evaluating various combinations or noise management and mitigation measures. As a result, a range of specific mitigation measures have been identified in the noise impact assessment to reduce noise emissions from the Project. The assessment has also considered the use of a range of mitigation measures, with modelling being done with and without these measures.

Noise impacts during Years 1, 3 and 4

Assessment of noise impacts associated with the Project has not identified any impacts that exceed the adopted noise guidelines for receptors located to the south of the Project. In areas to the north of the Project, some noise impacts have been identified that exceed the adopted noise guidelines (trigger levels) during the day in Year 1, and during the day and night in Years 3 and 4. The exceedances of the adopted noise guideline (trigger level) have been further evaluated to determine the level of exceedance.

Marginal exceedances (3 to 5 dBA) were identified at 4 receptors (402, 403, 411 and 538), with an additional 10 receptors (390, 398, 400, 418, 419, 420, 421, 423, 424 and 539) identified as having negligible exceedances (1 to 2 dBA, which is not considered to be discernible). These impacts are considered to sit within a Noise Management Zone. There are no receptors where noise impacts from the Project result in greater than 5 dBA increase in noise and hence no higher level of noise impacts have been identified. For the Noise Management Zone, noise management procedures are recommended. The Noise Impact Assessment has outlined a number of other management measures that could be considered to further manage noise in this area (Wilkinson Murray 2019).

Predicted $L_{Aeq,15\text{-}minute}$ noise predictions in the Noise Impact Assessment have been converted to represent $LA_{eq,day}$, $LA_{eq,evening}$ or $LA_{eq,night}$[14]. Using these noise levels, all predicted noise levels during the day (maximum $LA_{eq,day}$ = 40 dBA) and night (maxim $LA_{eq,night}$ = 38 dBA) (taken to be outdoor noise predictions at each receptor) are below health based noise guidelines.

Even in the absence of the proposed implementation of noise mitigation measures at some receptors to the north of the Project, from a health perspective, there are no noise impacts identified that would be considered to be of concern to community health.

Cumulative noise impacts, from the Project and the Mt Arthur Mine, were also considered in the Noise Impact Assessment. Impacts from both these sources more specifically relate to selected receptors to the north of the Project. The focus of the cumulative noise impact assessment was night-time noise as this is the most sensitive period for noise impacts. No cumulative noise impacts exceeded the adopted noise amenity levels relevant to the assessment of cumulative impacts. As these noise criteria are protective of health, no health impacts of concern have been identified.

There were no exceedances of the maximum noise level, at any of the receptors, for any of the Project years and time periods evaluated. As these noise criteria are protective of health, there would be no health impacts of concern in relation to the maximum noise levels related to the Project.

[14] Conversion of $LA_{eq,15\text{-}minute}$ to $LA_{eq,period}$ is outlined in the NPfI NSW EPA 2017, Noise Policy for Industry (and in the Noise Impact Assessment), where $LA_{eq,period}$ = $LA_{eq,15\text{-}minute}$ - 3 dB.

Road and rail noise

Assessment of road noise impacts considered expected road traffic volumes relevant to the Project, on Thomas Mitchell Drive. The assessment determined that noise at all receptors along this road will comply with the relevant noise guideline, with a predicted increase in noise of 0.4-0.5 $LA_{eq,15\ hour}$ (day and evening) and 0.3 dBA as $LA_{eq,9\ hour}$ (night). These changes in noise levels are very small, would not be discernible and would not be of concern to the health of receptors located near Thomas Mitchell Drive.

Assessment of rail noise impacts considered the location of the rail activity and the proposed number of rail movements each day (including the size of the train and time periods relevant to rail activity). It is noted that there are a number of other existing rail movements as a result of the use of the rail line by a number of other mining operations, as well as general freight and passenger trains. All noise impacts predicted at all receptors as a result of the Project were below the relevant rail noise criteria. As these noise criteria are protective of health, there would be no health impacts of concern in relation to rail noise.

Overall

Based on the available information, the potential for noise impacts to result in adverse health impacts within the community is considered to be negligible.

6.5 Uncertainties

The assessment of presented in relation to potential noise impacts, and the potential for impacts on community health as a result of changes in noise as a result of the Project are considered to be conservative. There are a number of areas within the noise impact assessment where conservative assumptions and approaches have been adopted. This includes the selection of RBLs relevant to the off-site areas, consideration of the worst-case meteorological conditions and assuming these occur on a regular basis, use of the upper end of noise impacts for comparison with relevant guidelines and approach adopted for the assessment of rail noise.

On the basis of the above, conclusions in relation to potential impacts on community health are expected to be conservative.

6.6 Outcomes of health risk assessment: noise

Table 6.1 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in noise, associated with the Project, on community health.

Table 6.1: Summary of health risks - noise

Noise emissions	
Impacts	Based on the predicted noise levels and potential mitigation measures, the potential for adverse health impacts within the off-site community associated with noise generated during construction and operations is considered to be negligible
Mitigation	Development of a Noise Management Plan prior to commencement of operations at the Project that would outline the measures to manage noise, including real-time and attended noise monitoring, use of meteorological forecasting and other general noise mitigation measures (consistent with the Noise Impact Assessment).

Section 7. Health impact assessment: Water

7.1 Approach

Health impacts associated with potential impacts of the Project on water access and quality relevant to the local community have been evaluated on the basis of information provided in the following reports:

- HydroSimulations, 2019. Maxwell Project, Groundwater Assessment
- WRM Water & Environment Pty Ltd (WRM), 2019. Maxwell Project, Surface Water Assessment.

The assessment undertaken in relation to water, has involved a qualitative review of the available information to determine if there is the potential for the Project to result in changes to surface water of groundwater quality or quantity, and where such changes may occur, if these may adversely affect the health of the community who may access and use these water resources.

7.2 Existing surface water and groundwater

The Project is located in the Hunter River catchment, which has an area of approximately 13,400 km^2 to Jerrys Plains. The catchment extends some 110 km to the north and 140 km to the west and includes the major tributaries of the Pages River, Dart Brook and the Goulburn River.

The Maxwell Infrastructure is located in the upper headwaters of the following tributaries of the Hunter River:

- Ramrod Creek;
- Bayswater Creek;
- Saltwater Creek; and
- Saddlers Creek.

The main drainage feature in the vicinity of the Maxwell Underground is Saddlers Creek which flows intermittently (being dry approximately 45% of the time). The eastern side of the Maxwell Underground area drains to Plashett Reservoir or directly to Saltwater Creek downstream of Plashett Reservoir. The Plashett Reservoir serves as an off-river water storage for the Bayswater Power Station, along with providing water supply to the Jerrys Plains township.

In relation to groundwater, the Project area and surrounds are characterised by the presence of three key groundwater systems:

- alluvium associated with the Hunter River;
- alluvium associated with Saddlers Creek and regolith; and
- Permian strata that host the target coal measures (which are within the Wittingham Coal Measures).

Most of the groundwater usage in the area is from the Hunter River alluvium. The Saddlers Creek alluvium is not commonly used, and comparatively few registered bores exist in the Permian porous rock aquifer, likely due to its lower yield and poorer water quality.

7.3 Project management and use of water

The Project objectives include a water management system to:

- protect the integrity of local and regional water resources;
- separate runoff from undisturbed, rehabilitated and mining-affected areas;
- design and manage the system to operate reliably throughout the life of the Project in all seasonal conditions, including both extended wet and dry periods;
- provide water for use in mining operations that is of sufficient volume and quality;
- maximise the re-use of water on-site; and
- manage groundwater inflows and CHPP process water on-site.

In addition to the above, storage and usage procedures for potentially hazardous materials (e.g. fuels, oils, greases) would be developed in accordance with Australian Standards and relevant legislation.

The main water sources for the operation are:

- groundwater inflows to underground workings and existing mine voids;
- recovery from CHPP rejects (through dewatering and/or decant return water);
- catchment runoff and infiltration; and
- small volumes of potable water imported to site.

Water would be required for: CHPP operation; underground mining operations (e.g. for cooling and underground dust suppression); stockpile dust suppression; washdown usage; and other minor non-potable uses.

7.4 Review of Project impacts on surface water and groundwater

7.4.1 Surface water

A simulated site water balance based on 129 years of climatic data has been prepared by WRM (2019) to simulate the performance of the water management system over the life of the Project (incorporating the Maxwell Infrastructure and the Maxwell Underground). The site water balance modelling demonstrates the proposed water management system has sufficient capacity and flexibility to accommodate a wide range of groundwater inflows and climate scenarios while (WRM 2019):

- providing security of supply for mine operations;
- minimising the risk of uncontrolled off-site release; and
- avoiding controlled release of water to the Hunter River.

In consideration of the above, WRM (2019) concluded that the Project would have negligible impacts on surface water quality in the Hunter River, Saddlers Creek or Ramrod Creek.

Where there is a negligible impact on surface water quality in these waterways, there are no health risk issues of concern related to impacts from the Project, regardless of the likely use of surface water in the local area.

7.4.2 Groundwater

Numerical modelling of potential drawdown due to the Project has been undertaken by HydroSimulations (2019) for the Groundwater Assessment. The results of the modelling indicate the Project would result in a small increase in leakage of water from the alluvium to the underlying hard rock aquifers. Accordingly, the Project would have negligible adverse impact on groundwater quality in the alluvium (HydroSimulations 2019).

Where there is a negligible impact on groundwater quality in the alluvium (the aquifer most likely to be extracted and used), there are no health risk issues of concern related to impacts from the Project, regardless of the likely use of groundwater in the local area.

Numerical modelling of the long-term recovery of the groundwater system has also been undertaken by HydroSimulations (2019) for the Groundwater Assessment. The results of this modelling indicate:

- the Maxwell Underground would remain a sink towards which groundwater would flow during mining, and well into the long-term post-mining period; and
- water levels in the voids at the Maxwell Infrastructure would equilibrate and remain as groundwater sinks in perpetuity.

The quality of water within the Permian hard rock aquifers is typically moderately saline (HydroSimulations 2019). Therefore, it is considered highly unlikely that mining-induced mixing of groundwater will result in changes to groundwater quality, in terms of beneficial uses of the Permian hard rock aquifers in or around the Project area during or following mining (HydroSimulations 2019).

On the basis of the groundwater assessment undertaken, there are no health risk issues of concern related to impacts from the Project on the deeper Permian rock aquifer, regardless whether this aquifer is accessed and used.

The Project would not materially affect the availability of groundwater for irrigation, domestic (e.g. garden watering) or stock use. Under the NSW Aquifer Interference Policy (NOW 2012), if more than minimal impact occurs at any groundwater supply work (more than 2 m decline cumulatively), then Malabar would be required to implement “make good” provisions (i.e. including an alternative long term supply of water that is equivalent to the loss attributable to the Project, if required).

A Groundwater Management Plan would be developed and implemented for the Project, and would define a groundwater monitoring strategy, groundwater level triggers and a trigger action response plan.

7.5 Uncertainties

The assessment presented in relation to potential surface water and groundwater impacts, and the potential for impacts on community health as a result of surface water and groundwater impacts as a result of the Project are considered to be conservative. There are a number of areas within the surface water and groundwater assessments where conservative assumptions and approaches have been adopted. The conclusions of these assessments have also been informed by sensitivity and uncertainty analysis.

On the basis of the above, conclusions in relation to potential impacts on community health are expected to be conservative.

7.6 Outcomes of health risk assessment: water

Table 7.1 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in surface water and groundwater, associated with the Project, on community health.

Table 7.1: Summary of health risks - water

Water	
Impacts	Based on the assessments undertaken, the potential for adverse health impacts within the off-site community associated with impacts to surface water and groundwater as a result of the Project is considered to be negligible.
Mitigation	Implementation of the water management system.

Section 8. Conclusions

The HHRA presented in this report has considered potential impacts on community health in relation to air quality, noise, vibration and water.

Based on the available information, and with consideration of the uncertainties identified no health risk issues of concern have been identified for the off-site community. More specifically, **Table 8.1** presents a summary of the health impact assessment and mitigation measures relevant to ensuring impacts are minimised or mitigated.

Table 8.1: Summary of health risks

Air emissions	
Impacts	Based on the available data and information in relation to emissions of dust and nitrogen dioxide from the Project, potential impacts on the health of the community have been assessed. The impact assessment has concluded there are no health risk issues of concern relevant to the Project (including construction and operational phases).
Mitigation	Development of an Air Quality Management Plan prior to commencement of operations at the Project that would outline the measures to manage air emissions (consistent with the AQGGA) and include aspects such as key performance indicators, monitoring methods, response mechanisms, compliance reporting and complaints management.
Noise emissions	
Impacts	Based on the predicted noise levels and potential mitigation measures, the potential for adverse health impacts within the off-site community associated with noise generated during construction and operations is considered to be negligible.
Mitigation	Development of a Noise Management Plan prior to commencement of operations at the Project that would outline the measures to manage noise, including real-time and attended noise monitoring, use of meteorological forecasting and other general noise mitigation measures (consistent with the Noise Impact Assessment).
Water	
Impacts	Based on the assessments undertaken, the potential for adverse health impacts within the off-site community associated with impacts to surface water and groundwater as a result of the Project is considered to be negligible.
Mitigation	Implementation of the water management system.

Section 9. References

Project specific reference:

Todoroski Air Sciences Pty Ltd, 2019. Maxwell Project, Air Quality and Greenhouse Gas Assessment (AQGGA).

Wilkinson Murray Pty Limited (Wilkinson Murray), 2019. Maxwell Project, Noise Impact Assessment.

HydroSimulations, 2019. Maxwell Project, Groundwater Assessment.

WRM Water & Environment Pty Ltd (WRM), 2019. Maxwell Project, Surface Water Assessment.

Other references:

ABS 2016, *2016 Census, including Community Profiles (accessed online)*, Australian Bureau of Statistics. <https://www.abs.gov.au/websitedbs/censushome.nsf/home/2016; https://quickstats.censusdata.abs.gov.au/census_services/getproduct/census/2016/communityprofile/036?opendocument>.

AIHW 2010, *Monitoring the impact of air pollution on asthma in Australia, A methods paper*, Australian Institute of Health and Welfare, Canberra.

Anderson, CH, Atkinson, RW, Peacock, JL, Marston, L & Konstantinou, K 2004, *Meta-analysis of time-series studies and panel studies of Particulate Matter (PM) and Ozone (O3), Report of a WHO task group*, World Health Organisation.

ANZG 2018, *Australian and New Zealand Guidelines for Fresh and Marine Water Quality*, A joint initiative of the Australian and New Zealand Governments in partnership with the Australian state and territory governments, Online. viewed August 2018, <http://www.waterquality.gov.au/anz-guidelines>.

Australian Government 2018, *LGA Data tables — Small Area Labour Markets, Data to December 2018 (online data)*, Department of Employment, Skills, Small and Family Business <https://docs.jobs.gov.au/documents/lga-data-tables-small-area-labour-markets-december-quarter-2018>.

Bell, ML, Ebisu, K, Peng, RD, Walker, J, Samet, JM, Zeger, SL & Dominici, F 2008, 'Seasonal and Regional Short-term Effects of Fine Particles on Hospital Admissions in 202 US Counties, 1999–2005', *American Journal of Epidemiology,* vol. 168, no. 11, December 1, 2008, pp. 1301-10.

Bell, ML 2012, 'Assessment of the health impacts of particulate matter characteristics', *Research report*, no. 161, Jan, pp. 5-38.

Burgers, M & Walsh, S 2002, *Exposure Assessment and Risk Characterisation for the Development of a PM2.5 Standard*, NEPC. <http://www.nepc.gov.au/system/files/resources/9947318f-af8c-0b24-d928-04e4d3a4b25c/files/aaq-pm25-rpt-exposure-assessment-and-risk-characterisation-final-200209.pdf>.

COMEAP 2015, *Statement on the Evidence for the Effects of Nitrogen Dioxide on Health*, Committee on the Medical Effects of Air Pollutants. <https://www.gov.uk/government/publications/nitrogen-dioxide-health-effects-of-exposure>.

EC 2011, *Final report on risk functions used in the case studies*, Health and Environment Integrated Methodology and Toolbox for Scenario Development (HEIMTSA).

enHealth 2012, *Environmental Health Risk Assessment, Guidelines for assessing human health risks from environmental hazards*, Commonwealth of Australia, Canberra. <http://www.health.gov.au/internet/main/publishing.nsf/content/804F8795BABFB1C7CA256F1900045479/$File/DoHA-EHRA-120910.pdf >.

EPHC 2010, *Expansion of the multi-city mortality and morbidity study, Final Report*, Environment Protection and Heritage Council.

EU 2015, *Directive 2008/50/EC of the European Parliament and of the Council of 21 May 2008 on ambient air quality and cleaner air for Europe*, European Parliament, Council of the European Union <https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:32008L0050>.

Golder 2013, *Exposure Assessment and Risk Characterisation to Inform Recommendations for Updating Ambient Air Quality Standards for PM2.5, PMN10, O3, NO2, SO2*, Golder Associates for National Environment Protection Council Service Corporation. <https://www.environment.gov.au/system/files/pages/dfe7ed5d-1eaf-4ff2-bfe7-dbb7ebaf21a9/files/exposure-assessment-risk-characterisation.pdf>.

Health 2010, *Respiratory and cardiovascular diseases and cancer among residents in the Hunter New England Area Health Service*, NSW Department of Health. <https://www.health.nsw.gov.au/environment/Pages/hne-respi-cardio.aspx>.

Hibberd, M, Selleck, P, Keywood, M & Cohen, D 2013, *Upper Hunter Valley Particle Characterization Study*, CSIRO Marine & Atmospheric Research and ANSTO, perpated for NSW Office of Environment and Heritage and NSW Department of Health.

I-INCE 2011, *Guidelines for Community Noise Impact Assessment and Mitigation, I-INCE Publication Number: 11-1*, International Institute of Noise Control Engineering (I-INCE) Technical Study Group on Community Noise: Environmental Noise Impact Assessment and Mitigation.

IARC 2013a, *Outdoor air pollution a leading environmental cause of cancer deaths*, International Agency for Research on Cancer. <http://www.iarc.fr/en/media-centre/iarcnews/pdf/ pr221_E.pdf>.

IARC 2013b, *Air Pollution and Cancer, IARC Scientific Publication No. 161*, International Agency for Research on Cancer. <http://publications.iarc.fr/Book-And-Report-Series/Iarc-Scientific-Publications/Air-Pollution-And-Cancer-2013>.

Jalaludin, B, Khalaj, B, Sheppeard, V & Morgan, G 2008, 'Air pollution and ED visits for asthma in Australian children: a case-crossover analysis', *Int Arch Occup Environ Health,* vol. 81, no. 8, Aug, pp. 967-74.

Krewski, D, Jerrett, M, Burnett, RT, Ma, R, Hughes, E, Shi, Y, Turner, MC, Pope, CA, 3rd, Thurston, G, Calle, EE, Thun, MJ, Beckerman, B, DeLuca, P, Finkelstein, N, Ito, K, Moore, DK, Newbold, KB, Ramsay, T, Ross, Z, Shin, H & Tempalski, B 2009, 'Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality', *Research report*, no. 140, May, pp. 5-114; discussion 15-36.

Lucas, SS, Coombes, PJ, Planner, J & Welschman, S 2009, 'Rainfall Harvesting and Coal Dust: The Potential Health Impacts of Trace Elements in Coal Dust in Rainwater', *Air Quality and Climate Change,* vol. 43, no. 2, pp. 23-30.

Martuzzi, M, Galasso, C, Ostro, B, Forastiere, F & Bertollini, R 2002, *Health Impact Assessment of Air Pollution in the Eight Major Italian Cities*, World Health Organisation, Europe.

Morawska, L, Moore, MR & Ristovski, ZD 2004, *Health Impacts of Ultrafine Particles, Desktop Literature Review and Analysis*, Australian Government, Department of the Environment and Heritage.

Morgan, G, Broom, R & Jalaludin, B 2013, *Summary for Policy Makers of the Health Risk Assessment on Air Pollution in Australia*, Prepared for National Environment Protection Council by the University Centre for Rural Health, North Coast, Education Research Workforce, A collaboration between The University of Sydney, Southern Cross University, The University of Western Sydney, The University of Wollongong, Canberra.

NEPC 1998, *National Environment Protection (Ambient Air Quality) Measure - Revised Impact Statement*, National Environment Protection Council.

NEPC 1999 amended 2013a, *Schedule B1, Guideline on Investigation Levels For Soil and Groundwater, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013b, *Schedule B4, Guideline on Health Risk Assessment Methodology, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013c, *Schedule B8 Guideline on Community Engagement and Risk Communication, National Environment Protection (Assessment of Site Contamination) Measure* National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013d, *Schedule B7, Guideline on Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013e, *Schedule B6 Guideline on Risk Based Assessment of Groundwater Contamination, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 2002, *National Environment Protection (Ambient Air Quality) Measure, Impact Statement for PM2.5 Variation Setting a PM2.5 Standard in Australia*, National Environment Protection Council.

NEPC 2003, *National Environment Protection (Ambient Air Quality) Measure*, National Environment Protection Council.

NEPC 2010, *Review of the National Environment Protection (Ambient Air Quality) Measure, Discussion Paper, Air Quality Standards*, National Environmental Protection Council.

NEPC 2014, *Draft Variation to the National Environment, protection (Ambient Air Quality) Measure, Impact Statement*, National Environment Protection Council.

NEPC 2016, *National Environment Protection (Ambient Air Quality) Measure*, Federal Register of Legislative Instruments F2016C00215.

NHMRC 2011 updated 2018, *Australian Drinking Water Guidelines 6, Version 3.5 Updated August 2018, National Water Quality Management Strategy*, National Health and Medical Research Council, National Resource Management Ministerial Council, Canberra.

NOW 2012, *NSW Aquifer Interference Policy*, Office of Water, NSW Department of Primary Industries. <http://www.water.nsw.gov.au/__data/assets/pdf_file/0004/549175/nsw_aquifer_interference_policy.pdf>.

NSW DEC 2006, *Assessing vibration: a technical guideline*, NSW Department of Environment and Conservation. <http://epa.nsw.gov.au/noise/vibrationguide.htm>.

NSW DECC 2009, *Interim Construction Noise Guideline*, NSW Department of Environment and Climate Change. <www.environment.nsw.gov.au/resources/stormwater/0801soilsconststorm2a.pdf>.

NSW DECCW 2011, *NSW Road Noise Policy*, NSW Department of Environment, Climate Change and Water, Sydney.

NSW EPA 2013, *Rail Infrastructure Noise Guideline* NSW Environment Protection Authority, Sydney. <https://www.epa.nsw.gov.au/-/media/epa/corporate-site/resources/noise/20130018eparing.pdf>.

NSW EPA 2016a, *Lower Hunter Dust Deposition Study*, Report prepared by AECOM for the New South Wales Environment Proection Authority. <https://www.epa.nsw.gov.au/~/media/EPA/Corporate%20Site/resources/air/lower-hunter-dust-study-report.ashx>.

NSW EPA 2016b, *Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales*, State of NSW and Environment Protection Authority, Sydney.

NSW EPA 2017, *Noise Policy for Industry*, NSW Environment Protection Authority, Sydney. <https://www.epa.nsw.gov.au/your-environment/noise/industrial-noise/noise-policy-for-industry-(2017)>.

NSW Government 2014, *State Environmental Planning Policy No 33—Hazardous and Offensive Development*, NSW Government under Environmental Planning and Assessment Act 1979. <http://www.legislation.nsw.gov.au/inforce/e5ebfcd2-5ebc-11dd-8fae-00144f4fe975/1992-129.pdf>.

OEHHA 2002, *Staff Report: Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates*, Office of Environmental Health Hazard Assessment.

Ostro, B 2004, *Outdoor Air Pollution: Assessing the environmental burden of disease at national and local levels.*, World Health Organisation.

Ostro, B, Broadwin, R, Green, S, Feng, WY & Lipsett, M 2006, 'Fine particulate air pollution and mortality in nine California counties: results from CALFINE', *Environmental health perspectives,* vol. 114, no. 1, Jan, pp. 29-33.

Pope, CA, 3rd, Burnett, RT, Thun, MJ, Calle, EE, Krewski, D, Ito, K & Thurston, GD 2002, 'Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution', *JAMA : the journal of the American Medical Association,* vol. 287, no. 9, Mar 6, pp. 1132-41.

Sjoberg, K, Haeger-Eugensson, M, Forsberg, B, Astrom, S, Hellsten, S, Larsson, K, Bjork, A & Blomgren, H 2009, *Quantification of population exposure to PM2.5 and PM10 in Sweden 2005*, Swedish Environmental Research Institute.

USEPA 2005, *Particulate Matter Health Risk Assessment For Selected Urban Areas*, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards.

USEPA 2009, *Integrated Science Assessment for Particulate Matter*, United States Environmental Protection Agency. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546#Download>.

USEPA 2010, *Quantitative Health Risk Assessment for Particulate Matter*, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency.

USEPA 2012, *Provisional Assessment of Recent Studies on Health Effects of Particulate Matter Exposure*, National Center for Environmental Assessment RTP Division, Office of Research and Development, U.S. Environmental Protection Agency.

USEPA 2015, *Integrated Science Assessment for Oxides of Nitrogen–Health Criteria, Second External Review Draft*, National Center for Environmental Assessment-RTP Division, Office of Research and Development, U.S. Environmental Protection Agency.

WHO 1999, *Guidelines for Community Noise*, World Health Organisation, Geneva.

WHO 2000, *WHO air quality guidelines for Europe, 2nd edition, 2000 (CD ROM version)*, World Health Organisation.

WHO 2003, *Health Aspects of Air Pollution with Particulate Matter, Ozone and Nitrogen Dioxide, Report on a WHO Working Group*, World Health Organisation.

WHO 2005, *WHO air quality guidelines global update 2005, Report on a Working Group meeting, Bonn, Germany, 18-20 October 2005*, World Health Organisation.

WHO 2006, *Health risks or particulate matter from long-range transboundary air pollution*, World Health Organisation Regional Office for Europe.

WHO 2009, *Night Noise Guidelines for Europe* World Health Organisation Regional Office for Europe.

WHO 2011, *Burden of disease from environmental noise, Quantification of healthy life years lost in Europe*, World Health Organisation and JRC European Commission.

WHO 2013a, *Review of evidence on health aspects of air pollution - REVIHAAP Project, Technical Report*, World Health Organization, Regional Office for Europe.

WHO 2013b, *Health Effects of Particulate Matter, Policy implications for countries in eastern Europe, Caucasus and central Asia*, WHO Regional Office for Europe.

WHO 2018, *Environmental Noise Guidelines for the European Region*, World Health Organization Regional Office for Europe. <http://www.euro.who.int/en/publications/abstracts/environmental-noise-guidelines-for-the-european-region-2018>.

Appendix A Calculation of risks from particulates and nitrogen dioxide

A1 Mortality and morbidity health endpoints

Quantitative assessment of risk for mortality and morbidity health endpoints uses a mathematical relationship between an exposure concentration (i.e. concentration in air) and a response (namely a health effect). This relationship is termed an exposure-response relationship and is relevant to the range of health effects (or endpoints) identified as relevant (to the nature of the emissions assessed) and robust (as identified in the main document). An exposure-response relationship can have a threshold, where there is a safe level of exposure, below which there are no adverse effects; or the relationship can have no threshold (and is regarded as linear) where there is some potential for adverse effects at any level of exposure.

In relation to the health effects associated with exposure to particulate matter and nitrogen dioxide, no threshold has been identified. Non-threshold exposure-response relationships have been identified for the health endpoints considered in this assessment.

A2 Quantification of impact and risk

The assessment of health impacts for a particular population associated with exposure to particulate matter or nitrogen dioxide has been undertaken utilising the methodology presented by the WHO (Ostro 2004)[15] where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

The calculation of changes in health endpoints associated with exposure to particulate matter or nitrogen dioxide as outlined by the WHO (Ostro 2004) has considered the following four elements:

- Estimates of the changes in particulate matter exposure levels or nitrogen dioxide levels (i.e. incremental impacts) due to the Project for the relevant modelled scenarios.
- Estimates of the number of people exposed to particulate matter or nitrogen dioxide at a given location.
- Baseline incidence of the key health endpoints that are relevant to the population exposed.
- Exposure-response relationships expressed as a percentage change in health endpoint per $\mu g/m^3$ change in particulate matter or nitrogen dioxide exposure, where a relative risk (RR) is determined.

From the above, the increased incidence of a health endpoint corresponding to a particular change in exposure has been calculated using the approach outlined below.

15 For regional guidance, such as that provided for Europe by the WHO 2006, Health risks or particulate matter from long-range transboundary air pollution, regional background incidence data for relevant health endpoints are combined with exposure-response functions to present an impact function, which is expressed as the number/change in incidence/new cases per 100,000 population exposed per microgram per cubic metre change in particulate matter exposure. These impact functions are simpler to use than the approach adopted in this assessment, however in utilising this approach it is assumed that the baseline incidence of the health effects is consistent throughout the whole population (as used in the studies) and is specifically applicable to the sub-population group being evaluated. For the assessment of exposures in the areas evaluated surrounding the Project it is more relevant to utilise local data in relation to baseline incidence rather than assume that the population is similar to that in Europe (where these relationships are derived).

The attributable fraction/portion (AF) of health effects from air pollution can be calculated from the relative risk as:

Equation 1 $$AF_{air} = \frac{RR-1}{RR}$$

The assessment of potential risks associated with these exposures involves the calculation of a relative risk (RR). For the purpose of this assessment, the shape of the exposure-response function used to calculate the relative risk (RR) is assumed to be linear[16]. The calculation of a RR based on the change in relative risk exposure concentration from baseline/existing (i.e. based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

Equation 2 $$RR = \exp[\beta(X-X0)]$$

Where:
X-X0 = the change in particulate matter concentration to which the population is exposed (µg/m³)
β = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 µg/m³ increase in particulate matter exposure

Based on this equation, where the published studies have derived relative risk values that are associated with a 10 µg/m^3 increase in exposure, the β coefficient can be calculated using the following equation:

Equation 3 $$\beta = \frac{\ln(RR)}{10}$$

Where:
RR = relative risk for the relevant health endpoint as published (µg/m³)
10 = increase in particulate matter concentration or noise level associated with the RR (where the RR is associated with a 10 µg/m³ increase in concentration

The total number of cases attributable to the change in exposure (where a linear dose-response is assumed) can be calculated as:

Equation 4 $$E = AF \times B \times P$$

Where:
B = baseline incidence of a given health effect (e.g. mortality rate per person per year)
P = relevant exposed population

16 Some reviews have identified that a log-linear exposure response function may be more relevant for some of the health endpoints considered in this assessment. Review of outcomes where a log-linear exposure-response function has been adopted (Ostro 2004) for $PM_{2.5}$ identified that the log-linear relationship calculated slightly higher relative risks compared with the linear relationship within the range 10–30 micrograms per cubic metre,(relevant for evaluating potential impacts associated with air quality goals or guidelines) but lower relative risks below and above this range. For this assessment (where impacts from a particular project are being evaluated) the impacts assessed relate to concentrations of $PM_{2.5}$ that are well below 10 micrograms per cubic metre and hence use of the linear relationship is expected to provide a more conservative estimate of relative risk.

The above approach (while presented slightly differently) is consistent with that presented in Australia (Burgers & Walsh 2002), the USA (OEHHA 2002; USEPA 2005, 2010) and Europe (Martuzzi et al. 2002; Sjoberg et al. 2009).

The calculation of an increased incidence (i.e. number of cases) of a particular health endpoint is not relevant to a specific individual, rather this is relevant to a statistically relevant population. This calculation has been undertaken for populations within the areas surrounding the Project.

When considering the potential impact of the Project on the population for changes in air quality, the calculation has been undertaken using the following:

- The relative risk has been calculated for a population weighted annual average incremental increase in concentrations. The population weighted average has been calculated on the basis of an average concentration relevant to the study area.
- The attributable fraction has then been calculated.
- Equation 4 has been used to calculate the increased number of cases associated with the incremental impact evaluated. The calculation is undertaken utilising the baseline incidence data relevant for the endpoint considered and the population (for the relevant age groups) present the area evaluated.

The above approach can be simplified (mathematically, where the incremental change in particulate concentration is low, in the order of one microgram per cubic metre or less) as follows:

Equation 5 $E=\beta \times B \times \sum_{mesh}(\Delta X_{mesh} \times P_{mesh})$

Where:
β = slope coefficient relevant to the per cent change in response to a 1 µg/m³ change in exposure concentration
B = baseline incidence of a given health effect per person (e.g. annual mortality rate)
ΔX_{mesh} = change (increment) in exposure concentration in µg/m³ as an average within a small area defined as a mesh block (from the ABS – where many mesh blocks make up a suburb)
P_{mesh} = population (residential – based on data from the ABS) within each small mesh block

An additional risk is calculated as:

Equation 6 $Risk=\beta \times \Delta X \times B$

Where:
β = slope coefficient relevant to the per cent change in response to a 1 µg/m³ change in exposure
ΔX = change (increment) in exposure concentration in µg/m³ relevant to the project at the point of exposure
B = baseline incidence of a given health effect per person (eg annual mortality rate)

This calculation provides an annual risk for individuals exposed to changes in air quality from the Project at specific locations (such as the maximum, or at specific sensitive receiver locations). The calculated risk does not take into account the duration of exposure at any one location and so is considered to be representative of a population risk.

A3 Quantification of short-and long-term effects

The concentration-response functions adopted for the assessment of exposure are derived from long and short-term studies and relate to short or long-term effects endpoints (e.g. change in incidence from daily changes in nitrogen dioxide or particulate matter, or chronic incidence from long-term exposures to particulate matter).

Long-term or chronic effects are assessed on the basis of the identified exposure-response function and annual average concentrations. These then allow the calculation of a chronic incidence of the assessed health endpoint.

Short-term effects are also assessed on the basis of an exposure-response function that is expressed as a percentage change in endpoint per microgram per cubic metre change in concentration. For short-term effects, daily changes in nitrogen dioxide and particulate matter exposures are used to calculate changes in daily effects endpoints. While it may be possible to measure daily incidence of the evaluated health endpoints in a large population study specifically designed to include such data, it is not common to collect such data in hospitals nor are effects measurable in smaller communities. Instead these calculations relate to a parameter that is measurable, such as annual incidence of hospitalisations, mortality or lung cancer risks. The calculation of an annual incidence or additional risk can be undertaken using two approaches (Ostro 2004; USEPA 2010):

- Calculate the daily incidence or risk at each receiver location over every 24-hour period of the year (based on the modelled incremental 24-hour average concentration for each day of the year and daily baseline incidence data) and then sum the daily incidence/risk to get the annual risk.
- Calculate the annual incidence/risk based on the incremental annual average concentration at each receiver (and using annual baseline incidence data).

In the absence of a threshold, and assuming a linear concentration-response function (as is the case in this assessment), these two approaches result in the same outcome mathematically (calculated incidence or risk). Given that it is much simpler computationally to calculate the incidence (for each receiver) based on the incremental annual average, compared with calculating effects on each day of the year and then summing, this is the preferred calculation method. It is the recommended method outlined by the WHO (Ostro 2004).

The use of the simpler approach, based on annual average concentrations should not be taken as implying or suggesting that the calculation is quantifying the effects of long-term exposure.

For the calculations presented in this assessment **-** for long-term and short-term effects **-** annual average concentrations of nitrogen dioxide and particulate matter have thus been utilised.

Appendix B Risk calculations – $PM_{2.5}$ and PM_{10}

Quantification of Effects - $PM_{2.5}$ and PM_{10}
Maxwell Project

Air quality indicator:	$PM_{2.5}$	$PM_{2.5}$	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Endpoint:	**Mortality - All Causes**	**Hospitalisations - Cardiovascular**	**Hospitalisations - Respiratory**	**Mortality - All Causes**	**Morbidity - Asthma ED Admissions**
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
β (change in effect per 1 µg/m^3) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
Annual baseline incidence (per 100,000)	1026	9235	4168	643.2	2284
Baseline Incidence (per person per year)	0.01026	0.09235	0.04168	0.006432	0.02284

Sensitive Receptors	Change in Annual Average PM10 Concentration (µg/m^3)	Change in Annual Average PM2.5 Concentration (µg/m^3)	Risk	Risk	Risk	Risk	Risk
Maximum predicted anywhere off-site							
Scenario 1	0.36	0.0735	4E-06	5E-06	1E-06	1E-06	2E-06
Scenario 2	0.35	0.0775	5E-06	6E-06	1E-06	1E-06	3E-06
Scenario 3	0.79	0.0923	5E-06	7E-06	2E-06	3E-06	3E-06

Assessment of Increased Incidence - $PM_{2.5}$ and PM10
Scenarios 1-3

	Primary Indicators			Secondary Indicators	
Health Endpoint:	**Mortality - All Causes, Long-term**	**Hospitalisations - Cardiovascular, Short-term**	**Hospitalisations Respiratory, Short-term**	**PM10 Mortality - All Causes, Short-term**	**Morbidity - Asthma ED Admissions - Short-term**
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
β (change in effect per 1 µg/m³ PM) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
Scenario 1 - Year 1					
Total Population in study area:	394.2	394.2	394.2	394.2	394.2
% population in assessment age-group:	57%	12%	12%	100%	23%
Average change Δx (µg/m³):	0.014	0.014	0.014	0.055	0.014
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	4168	643.2	2284.0
Baseline Incidence (per person)	0.01026	0.09235	0.04168	0.00643	0.02284
Relative Risk:	1.000084	1.000012	1.000006	1.000033	1.000021
Attributable fraction (AF):	8.4E-05	1.2E-05	5.9E-06	3.3E-05	2.1E-05
Increased number of cases in population:	0.00019	0.000051	0.000012	0.000084	0.000044
Risk:	8.6E-07	1.1E-06	2.5E-07	2.1E-07	4.9E-07
Scenario 2 - Year 3					
Total Population in study area:	394.2	394.2	394.2	394.2	394.2
% population in assessment age-group:	57%	12%	12%	100%	23%
Average change Δx (µg/m³):	0.011	0.011	0.011	0.048	0.011
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	4168	643.2	2284.0
Baseline Incidence (per person)	0.01026	0.09235	0.04168	0.00643	0.02284
Relative Risk:	1.000062	1.000009	1.000004	1.000029	1.000016
Attributable fraction (AF):	6.2E-05	8.5E-06	4.4E-06	2.9E-05	1.6E-05
Increased number of cases in population:	0.00014	0.000037	0.0000086	0.000074	0.000032
Risk:	6.3E-07	7.9E-07	1.8E-07	1.9E-07	3.6E-07
Scenario 3 - Year 4					
Total Population in study area:	394.2	394.2	394.2	394.2	394.2
% population in assessment age-group:	57%	12%	12%	100%	23%
Average change Δx (µg/m³):	0.0089	0.0089	0.0089	0.073	0.0089
Baseline Incidence (per 100,000) (as per Table 4.4)	1026	9235	4168	643.2	2284.0
Baseline Incidence (per person)	0.01026	0.09235	0.04168	0.00643	0.02284
Relative Risk:	1.000052	1.000007	1.000004	1.000044	1.000013
Attributable fraction (AF):	5.2E-05	7.1E-06	3.7E-06	4.4E-05	1.3E-05
Increased number of cases in population:	0.00012	0.000031	0.0000072	0.00011	0.000027
Risk:	5.3E-07	6.6E-07	1.5E-07	2.8E-07	3.0E-07

Quantification of Effects - $PM_{2.5}$ and PM_{10}
Maxwell Project: Scenario 1 (Year 1)

Air quality indicator:	$PM_{2.5}$	$PM_{2.5}$	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Endpoint:	**Mortality - All Causes**	**Hospitalisations - Cardiovascular**	**Hospitalisations - Respiratory**	**Mortality - All Causes**	**Morbidity - Asthma ED Admissions**
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
β (change in effect per 1 µg/m³) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
Annual baseline incidence (per 100,000	1026	9235	4168	643.2	2284
Baseline Incidence (per person per year)	0.01026	0.09235	0.04168	0.006432	0.02284

Sensitive Receptors	Change in Annual Average PM10 Concentration (µg/m³)	Change in Annual Average PM2.5 Concentration (µg/m³)	Risk	Risk	Risk	Risk	Risk
Assessment of impacts are each receptor							
287	1.8E-02	4.5E-03	3E-07	3E-07	8E-08	7E-08	2E-07
279	1.7E-02	4.3E-03	3E-07	3E-07	7E-08	7E-08	1E-07
306	2.1E-02	5.3E-03	3E-07	4E-07	9E-08	8E-08	2E-07
451	6.4E-02	1.9E-02	1E-06	1E-06	3E-07	2E-07	6E-07
455	6.6E-02	2.0E-02	1E-06	1E-06	3E-07	3E-07	7E-07
255	1.9E-02	4.7E-03	3E-07	4E-07	8E-08	7E-08	2E-07
421	1.1E-01	3.1E-02	2E-06	2E-06	5E-07	4E-07	1E-06
441b	6.7E-02	2.0E-02	1E-06	1E-06	3E-07	3E-07	7E-07
441a	6.7E-02	2.0E-02	1E-06	1E-06	3E-07	3E-07	7E-07
219c	2.9E-02	7.4E-03	4E-07	5E-07	1E-07	1E-07	3E-07
219a	2.9E-02	7.4E-03	4E-07	5E-07	1E-07	1E-07	2E-07
219b	3.0E-02	7.6E-03	5E-07	6E-07	1E-07	1E-07	3E-07
219d	2.9E-02	7.4E-03	4E-07	5E-07	1E-07	1E-07	2E-07
230a	1.4E-02	3.6E-03	2E-07	3E-07	6E-08	5E-08	1E-07
228k	1.8E-02	4.6E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228g	1.8E-02	4.6E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228e	1.7E-02	4.5E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228c	1.7E-02	4.5E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228b	1.7E-02	4.5E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228a	1.7E-02	4.5E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228j	1.6E-02	4.0E-03	2E-07	3E-07	7E-08	6E-08	1E-07
228l	2.0E-02	5.3E-03	3E-07	4E-07	9E-08	8E-08	2E-07
228m	2.1E-02	5.5E-03	3E-07	4E-07	9E-08	8E-08	2E-07
226c	4.7E-02	1.2E-02	7E-07	9E-07	2E-07	2E-07	4E-07
226a	4.5E-02	1.1E-02	7E-07	8E-07	2E-07	2E-07	4E-07
226d	3.8E-02	9.4E-03	6E-07	7E-07	2E-07	1E-07	3E-07
227f	3.1E-02	7.8E-03	5E-07	6E-07	1E-07	1E-07	3E-07
228n	2.4E-02	6.0E-03	4E-07	4E-07	1E-07	9E-08	2E-07
227e	3.0E-02	7.6E-03	5E-07	6E-07	1E-07	1E-07	3E-07
227d	3.1E-02	7.8E-03	5E-07	6E-07	1E-07	1E-07	3E-07
227c	3.2E-02	8.2E-03	5E-07	6E-07	1E-07	1E-07	3E-07
227b	3.4E-02	8.5E-03	5E-07	6E-07	1E-07	1E-07	3E-07
227a	3.5E-02	8.9E-03	5E-07	7E-07	2E-07	1E-07	3E-07
226b	4.6E-02	1.1E-02	7E-07	8E-07	2E-07	2E-07	4E-07
228i	1.8E-02	4.6E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228h	1.8E-02	4.6E-03	3E-07	3E-07	8E-08	7E-08	2E-07
228f	1.8E-02	4.5E-03	3E-07	3E-07	8E-08	7E-08	2E-07
230b	1.5E-02	3.8E-03	2E-07	3E-07	7E-08	6E-08	1E-07
228o	2.1E-02	5.4E-03	3E-07	4E-07	9E-08	8E-08	2E-07
217c	3.3E-02	8.3E-03	5E-07	6E-07	1E-07	1E-07	3E-07
217d	3.2E-02	7.9E-03	5E-07	6E-07	1E-07	1E-07	3E-07
217e	3.0E-02	7.5E-03	4E-07	6E-07	1E-07	1E-07	3E-07
527	2.3E-02	5.8E-03	3E-07	4E-07	1E-07	9E-08	2E-07
219e	2.7E-02	6.9E-03	4E-07	5E-07	1E-07	1E-07	2E-07
228p	1.9E-02	4.8E-03	3E-07	4E-07	8E-08	7E-08	2E-07
217f	2.9E-02	7.4E-03	4E-07	5E-07	1E-07	1E-07	3E-07
398	1.4E-01	3.8E-02	2E-06	3E-06	6E-07	5E-07	1E-06
500	3.3E-02	9.9E-03	6E-07	7E-07	2E-07	1E-07	3E-07
211a	3.2E-02	8.0E-03	5E-07	6E-07	1E-07	1E-07	3E-07
528	3.1E-02	7.9E-03	5E-07	6E-07	1E-07	1E-07	3E-07
211b	3.3E-02	8.1E-03	5E-07	6E-07	1E-07	1E-07	3E-07
211c	3.2E-02	8.1E-03	5E-07	6E-07	1E-07	1E-07	3E-07
508	3.8E-02	1.1E-02	7E-07	8E-07	2E-07	1E-07	4E-07
238a	1.4E-02	3.8E-03	2E-07	3E-07	6E-08	6E-08	1E-07
238b	1.4E-02	3.8E-03	2E-07	3E-07	6E-08	5E-08	1E-07
238c	1.4E-02	3.8E-03	2E-07	3E-07	6E-08	6E-08	1E-07
238d	1.5E-02	3.8E-03	2E-07	3E-07	7E-08	6E-08	1E-07
238e	1.5E-02	3.8E-03	2E-07	3E-07	7E-08	6E-08	1E-07
238f	1.5E-02	3.8E-03	2E-07	3E-07	7E-08	6E-08	1E-07
239g	1.6E-02	4.2E-03	2E-07	3E-07	7E-08	6E-08	1E-07
239a	1.5E-02	4.1E-03	2E-07	3E-07	7E-08	6E-08	1E-07
239f	1.5E-02	4.1E-03	2E-07	3E-07	7E-08	6E-08	1E-07
239e	1.6E-02	4.2E-03	3E-07	3E-07	7E-08	6E-08	1E-07
239d	1.6E-02	4.2E-03	3E-07	3E-07	7E-08	6E-08	1E-07
239c	1.6E-02	4.3E-03	3E-07	3E-07	7E-08	6E-08	1E-07
239b	1.6E-02	4.3E-03	3E-07	3E-07	7E-08	6E-08	1E-07
239h	1.6E-02	4.3E-03	3E-07	3E-07	7E-08	6E-08	1E-07
239i	1.7E-02	4.4E-03	3E-07	3E-07	8E-08	6E-08	1E-07
240d	2.5E-02	6.5E-03	4E-07	5E-07	1E-07	1E-07	2E-07
240c	2.5E-02	6.5E-03	4E-07	5E-07	1E-07	1E-07	2E-07
240b	2.5E-02	6.4E-03	4E-07	5E-07	1E-07	1E-07	2E-07
240e	2.5E-02	6.3E-03	4E-07	5E-07	1E-07	1E-07	2E-07
240a	2.2E-02	5.7E-03	3E-07	4E-07	1E-07	8E-08	2E-07
239j	1.7E-02	4.5E-03	3E-07	3E-07	8E-08	7E-08	2E-07
239k	1.7E-02	4.4E-03	3E-07	3E-07	8E-08	7E-08	2E-07
238g	1.4E-02	3.7E-03	2E-07	3E-07	6E-08	5E-08	1E-07
238h	1.4E-02	3.7E-03	2E-07	3E-07	6E-08	5E-08	1E-07
207	2.9E-02	7.3E-03	4E-07	5E-07	1E-07	1E-07	2E-07
419	1.7E-01	4.9E-02	3E-06	4E-06	8E-07	7E-07	2E-06
424	8.7E-02	2.5E-02	2E-06	2E-06	4E-07	3E-07	9E-07
532	1.8E-02	4.6E-03	3E-07	3E-07	8E-08	7E-08	2E-07
60a	2.9E-01	5.9E-02	4E-06	4E-06	1E-06	1E-06	2E-06
60b	3.6E-01	7.3E-02	4E-06	5E-06	1E-06	1E-06	2E-06
60c	3.4E-01	6.9E-02	4E-06	5E-06	1E-06	1E-06	2E-06
60d	3.2E-01	6.5E-02	4E-06	5E-06	1E-06	1E-06	2E-06
254a	1.9E-02	4.9E-03	3E-07	4E-07	8E-08	7E-08	2E-07
254b	1.9E-02	4.9E-03	3E-07	4E-07	8E-08	7E-08	2E-07
254c	1.9E-02	4.9E-03	3E-07	4E-07	8E-08	7E-08	2E-07
250b	2.9E-02	7.1E-03	4E-07	5E-07	1E-07	1E-07	2E-07
250a	2.8E-02	7.0E-03	4E-07	5E-07	1E-07	1E-07	2E-07
432	5.0E-02	1.5E-02	9E-07	1E-06	2E-07	2E-07	5E-07
443	8.7E-02	2.6E-02	2E-06	2E-06	4E-07	3E-07	9E-07
298a	2.5E-02	6.1E-03	4E-07	5E-07	1E-07	9E-08	2E-07
298b	2.4E-02	6.0E-03	4E-07	4E-07	1E-07	9E-08	2E-07
400	6.7E-02	1.9E-02	1E-06	1E-06	3E-07	3E-07	7E-07

			Air quality indicator:	$PM_{2.5}$	$PM_{2.5}$	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
			Endpoint:	Mortality - All Causes	Hospitalisations - Cardiovascular	Hospitalisations - Respiratory	Mortality - All Causes	Morbidity - Asthma ED Admissions
			Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term
			Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
			β (change in effect per 1 μg/m^3) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
			Annual baseline incidence (per 100,000	1026	9235	4168	643.2	2284
			Baseline Incidence (per person per year)	0.01026	0.09235	0.04168	0.006432	0.02284
384	8.9E-02	2.5E-02		2E-06	2E-06	4E-07	3E-07	9E-07
444	1.1E-01	3.2E-02		2E-06	2E-06	5E-07	4E-07	1E-06
399	9.4E-02	2.6E-02		2E-06	2E-06	4E-07	4E-07	9E-07
420	1.4E-01	3.9E-02		2E-06	3E-06	7E-07	5E-07	1E-06
410	2.1E-01	6.0E-02		4E-06	4E-06	1E-06	8E-07	2E-06
404	8.8E-02	2.6E-02		2E-06	2E-06	4E-07	3E-07	9E-07
387	1.3E-01	3.5E-02		2E-06	3E-06	6E-07	5E-07	1E-06
145a	6.1E-02	1.4E-02		9E-07	1E-06	2E-07	2E-07	5E-07
145b	5.1E-02	1.2E-02		7E-07	9E-07	2E-07	2E-07	4E-07
145c	4.5E-02	1.1E-02		6E-07	8E-07	2E-07	2E-07	4E-07
58b	3.5E-02	8.8E-03		5E-07	6E-07	2E-07	1E-07	3E-07
57	4.8E-02	1.2E-02		7E-07	9E-07	2E-07	2E-07	4E-07
536	7.6E-02	1.8E-02		1E-06	1E-06	3E-07	3E-07	6E-07
507	4.0E-02	1.2E-02		7E-07	9E-07	2E-07	2E-07	4E-07
390	2.2E-01	5.7E-02		3E-06	4E-06	1E-06	8E-07	2E-06
460	5.0E-02	1.5E-02		9E-07	1E-06	3E-07	2E-07	5E-07
440	5.7E-02	1.7E-02		1E-06	1E-06	3E-07	2E-07	6E-07
435a	4.2E-02	1.2E-02		7E-07	9E-07	2E-07	2E-07	4E-07
435b	5.3E-02	1.5E-02		9E-07	1E-06	3E-07	2E-07	5E-07
433b	4.0E-02	1.2E-02		7E-07	9E-07	2E-07	2E-07	4E-07
433a	5.7E-02	1.7E-02		1E-06	1E-06	3E-07	2E-07	6E-07
253	2.0E-02	5.0E-03		3E-07	4E-07	9E-08	8E-08	2E-07
411	1.9E-01	5.4E-02		3E-06	4E-06	9E-07	7E-07	2E-06
423	1.0E-01	2.9E-02		2E-06	2E-06	5E-07	4E-07	1E-06
425	8.7E-02	2.5E-02		2E-06	2E-06	4E-07	3E-07	9E-07
209	3.4E-02	8.3E-03		5E-07	6E-07	1E-07	1E-07	3E-07
418	1.9E-01	5.3E-02		3E-06	4E-06	9E-07	7E-07	2E-06
509	3.2E-02	9.5E-03		6E-07	7E-07	2E-07	1E-07	3E-07
25	2.6E-02	6.8E-03		4E-07	5E-07	1E-07	1E-07	2E-07
24b	2.5E-02	6.5E-03		4E-07	5E-07	1E-07	1E-07	2E-07
24a	2.5E-02	6.5E-03		4E-07	5E-07	1E-07	1E-07	2E-07
386	1.1E-01	3.0E-02		2E-06	2E-06	5E-07	4E-07	1E-06
284	1.8E-02	4.6E-03		3E-07	3E-07	8E-08	7E-08	2E-07
403	1.2E-01	3.3E-02		2E-06	2E-06	6E-07	4E-07	1E-06
427	7.5E-02	2.2E-02		1E-06	2E-06	4E-07	3E-07	7E-07
402	9.5E-02	2.7E-02		2E-06	2E-06	5E-07	4E-07	9E-07
429	6.0E-02	1.8E-02		1E-06	1E-06	3E-07	2E-07	6E-07
285	1.7E-02	4.4E-03		3E-07	3E-07	7E-08	7E-08	1E-07
172	3.0E-02	7.6E-03		5E-07	6E-07	1E-07	1E-07	3E-07
456	6.0E-02	1.8E-02		1E-06	1E-06	3E-07	2E-07	6E-07
385	6.4E-02	1.9E-02		1E-06	1E-06	3E-07	2E-07	6E-07
446a	1.2E-01	3.5E-02		2E-06	3E-06	6E-07	5E-07	1E-06
438	2.7E-02	8.1E-03		5E-07	6E-07	1E-07	1E-07	3E-07
299	2.3E-02	5.8E-03		3E-07	4E-07	1E-07	9E-08	2E-07
58a	3.6E-02	9.0E-03		5E-07	7E-07	2E-07	1E-07	3E-07
389	2.9E-01	7.3E-02		4E-06	5E-06	1E-06	1E-06	2E-06
228q	2.0E-02	5.2E-03		3E-07	4E-07	9E-08	8E-08	2E-07
228r	2.1E-02	5.5E-03		3E-07	4E-07	9E-08	8E-08	2E-07
537	9.0E-02	2.4E-02		1E-06	2E-06	4E-07	3E-07	8E-07
538	7.8E-02	2.3E-02		1E-06	2E-06	4E-07	3E-07	8E-07
539	1.2E-01	3.5E-02		2E-06	3E-06	6E-07	5E-07	1E-06

Quantification of Effects - $PM_{2.5}$ and PM_{10}
Maxwell Project: Scenario 2 (Year 3)

Air quality indicator:	$PM_{2.5}$	$PM_{2.5}$	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Endpoint:	**Mortality - All Causes**	**Hospitalisations - Cardiovascular**	**Hospitalisations - Respiratory**	**Mortality - All Causes**	**Morbidity - Asthma ED Admissions**
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
β (change in effect per 1 µg/m³) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
Annual baseline incidence (per 100,000	1026	9235	4168	643.2	2284
Baseline Incidence (per person per year)	0.01026	0.09235	0.04168	0.006432	0.02284

Sensitive Receptors	Change in Annual Average PM10 Concentration (µg/m³)	Change in Annual Average PM2.5 Concentration (µg/m³)	Risk	Risk	Risk	Risk	Risk
Assessment of impacts are each receptor							
287	1.3E-02	2.5E-03	1E-07	2E-07	4E-08	5E-08	8E-08
279	1.3E-02	2.4E-03	1E-07	2E-07	4E-08	5E-08	8E-08
306	1.5E-02	2.8E-03	2E-07	2E-07	5E-08	6E-08	1E-07
451	6.1E-02	1.6E-02	9E-07	1E-06	3E-07	2E-07	5E-07
455	5.7E-02	1.5E-02	9E-07	1E-06	3E-07	2E-07	5E-07
255	1.3E-02	2.5E-03	2E-07	2E-07	4E-08	5E-08	9E-08
421	1.0E-01	2.7E-02	2E-06	2E-06	5E-07	4E-07	9E-07
441b	5.8E-02	1.5E-02	9E-07	1E-06	3E-07	2E-07	5E-07
441a	6.0E-02	1.6E-02	9E-07	1E-06	3E-07	2E-07	5E-07
219c	2.1E-02	3.9E-03	2E-07	3E-07	7E-08	8E-08	1E-07
219a	2.1E-02	3.9E-03	2E-07	3E-07	7E-08	8E-08	1E-07
219b	2.2E-02	3.9E-03	2E-07	3E-07	7E-08	8E-08	1E-07
219d	2.1E-02	3.9E-03	2E-07	3E-07	7E-08	8E-08	1E-07
230a	9.9E-03	1.9E-03	1E-07	1E-07	3E-08	4E-08	6E-08
228k	1.2E-02	2.4E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228g	1.2E-02	2.4E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228e	1.2E-02	2.4E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228c	1.2E-02	2.3E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228b	1.2E-02	2.3E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228a	1.2E-02	2.3E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228j	1.1E-02	2.1E-03	1E-07	2E-07	4E-08	4E-08	7E-08
228l	1.4E-02	2.7E-03	2E-07	2E-07	5E-08	6E-08	9E-08
228m	1.5E-02	2.9E-03	2E-07	2E-07	5E-08	6E-08	1E-07
226c	3.6E-02	6.3E-03	4E-07	5E-07	1E-07	1E-07	2E-07
226a	3.5E-02	6.0E-03	4E-07	4E-07	1E-07	1E-07	2E-07
226d	2.9E-02	5.1E-03	3E-07	4E-07	9E-08	1E-07	2E-07
227f	2.3E-02	4.1E-03	2E-07	3E-07	7E-08	9E-08	1E-07
228n	1.7E-02	3.2E-03	2E-07	2E-07	5E-08	7E-08	1E-07
227e	2.2E-02	4.0E-03	2E-07	3E-07	7E-08	9E-08	1E-07
227d	2.3E-02	4.2E-03	2E-07	3E-07	7E-08	9E-08	1E-07
227c	2.4E-02	4.4E-03	3E-07	3E-07	7E-08	9E-08	1E-07
227b	2.5E-02	4.5E-03	3E-07	3E-07	8E-08	1E-07	2E-07
227a	2.6E-02	4.7E-03	3E-07	4E-07	8E-08	1E-07	2E-07
226b	3.5E-02	6.1E-03	4E-07	5E-07	1E-07	1E-07	2E-07
228i	1.2E-02	2.4E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228h	1.2E-02	2.4E-03	1E-07	2E-07	4E-08	5E-08	8E-08
228f	1.2E-02	2.4E-03	1E-07	2E-07	4E-08	5E-08	8E-08
230b	1.1E-02	2.0E-03	1E-07	1E-07	3E-08	4E-08	7E-08
228o	1.5E-02	2.8E-03	2E-07	2E-07	5E-08	6E-08	1E-07
217c	2.3E-02	4.2E-03	3E-07	3E-07	7E-08	9E-08	1E-07
217d	2.2E-02	4.1E-03	2E-07	3E-07	7E-08	9E-08	1E-07
217e	2.1E-02	3.8E-03	2E-07	3E-07	7E-08	8E-08	1E-07
527	1.6E-02	3.0E-03	2E-07	2E-07	5E-08	6E-08	1E-07
219e	2.0E-02	3.6E-03	2E-07	3E-07	6E-08	8E-08	1E-07
228p	1.3E-02	2.5E-03	1E-07	2E-07	4E-08	5E-08	8E-08
217f	2.1E-02	3.8E-03	2E-07	3E-07	6E-08	8E-08	1E-07
398	1.4E-01	3.5E-02	2E-06	3E-06	6E-07	5E-07	1E-06
500	2.7E-02	7.4E-03	4E-07	5E-07	1E-07	1E-07	3E-07
211a	2.2E-02	4.0E-03	2E-07	3E-07	7E-08	8E-08	1E-07
528	2.1E-02	3.9E-03	2E-07	3E-07	7E-08	8E-08	1E-07
211b	2.2E-02	4.1E-03	2E-07	3E-07	7E-08	9E-08	1E-07
211c	2.2E-02	4.1E-03	2E-07	3E-07	7E-08	9E-08	1E-07
508	3.1E-02	8.1E-03	5E-07	6E-07	1E-07	1E-07	3E-07
238a	1.0E-02	2.0E-03	1E-07	1E-07	3E-08	4E-08	7E-08
238b	9.9E-03	1.9E-03	1E-07	1E-07	3E-08	4E-08	7E-08
238c	9.9E-03	2.0E-03	1E-07	1E-07	3E-08	4E-08	7E-08
238d	1.0E-02	2.0E-03	1E-07	1E-07	3E-08	4E-08	7E-08
238e	1.0E-02	2.0E-03	1E-07	1E-07	3E-08	4E-08	7E-08
238f	1.0E-02	2.0E-03	1E-07	1E-07	3E-08	4E-08	7E-08
239g	1.1E-02	2.2E-03	1E-07	2E-07	4E-08	4E-08	7E-08
239a	1.1E-02	2.1E-03	1E-07	2E-07	4E-08	4E-08	7E-08
239f	1.1E-02	2.1E-03	1E-07	2E-07	4E-08	4E-08	7E-08
239e	1.1E-02	2.2E-03	1E-07	2E-07	4E-08	4E-08	7E-08
239d	1.1E-02	2.2E-03	1E-07	2E-07	4E-08	4E-08	8E-08
239c	1.1E-02	2.2E-03	1E-07	2E-07	4E-08	4E-08	8E-08
239b	1.1E-02	2.2E-03	1E-07	2E-07	4E-08	4E-08	8E-08
239h	1.1E-02	2.2E-03	1E-07	2E-07	4E-08	4E-08	8E-08
239i	1.2E-02	2.3E-03	1E-07	2E-07	4E-08	5E-08	8E-08
240d	1.8E-02	3.4E-03	2E-07	3E-07	6E-08	7E-08	1E-07
240c	1.8E-02	3.4E-03	2E-07	3E-07	6E-08	7E-08	1E-07
240b	1.8E-02	3.4E-03	2E-07	2E-07	6E-08	7E-08	1E-07
240e	1.8E-02	3.3E-03	2E-07	2E-07	6E-08	7E-08	1E-07
240a	1.6E-02	3.0E-03	2E-07	2E-07	5E-08	6E-08	1E-07
239j	1.2E-02	2.3E-03	1E-07	2E-07	4E-08	5E-08	8E-08
239k	1.2E-02	2.3E-03	1E-07	2E-07	4E-08	5E-08	8E-08
238g	9.7E-03	1.9E-03	1E-07	1E-07	3E-08	4E-08	6E-08
238h	9.8E-03	1.9E-03	1E-07	1E-07	3E-08	4E-08	7E-08
207	2.0E-02	3.7E-03	2E-07	3E-07	6E-08	8E-08	1E-07
419	1.8E-01	4.5E-02	3E-06	3E-06	8E-07	7E-07	2E-06
424	8.1E-02	2.1E-02	1E-06	2E-06	4E-07	3E-07	7E-07
532	1.3E-02	2.5E-03	1E-07	2E-07	4E-08	5E-08	8E-08
60a	2.3E-01	3.2E-02	2E-06	2E-06	5E-07	9E-07	1E-06
60b	2.8E-01	3.9E-02	2E-06	3E-06	7E-07	1E-06	1E-06
60c	2.6E-01	3.7E-02	2E-06	3E-06	6E-07	1E-06	1E-06
60d	2.5E-01	3.5E-02	2E-06	3E-06	6E-07	1E-06	1E-06
254a	1.4E-02	2.6E-03	2E-07	2E-07	4E-08	5E-08	9E-08
254b	1.4E-02	2.6E-03	2E-07	2E-07	4E-08	5E-08	9E-08
254c	1.4E-02	2.6E-03	2E-07	2E-07	4E-08	5E-08	9E-08
250b	2.0E-02	3.8E-03	2E-07	3E-07	6E-08	8E-08	1E-07
250a	2.0E-02	3.7E-03	2E-07	3E-07	6E-08	8E-08	1E-07
432	4.2E-02	1.1E-02	7E-07	8E-07	2E-07	2E-07	4E-07
443	8.1E-02	2.1E-02	1E-06	2E-06	4E-07	3E-07	7E-07
298a	1.7E-02	3.3E-03	2E-07	2E-07	6E-08	7E-08	1E-07
298b	1.7E-02	3.2E-03	2E-07	2E-07	5E-08	6E-08	1E-07
400	6.0E-02	1.6E-02	1E-06	1E-06	3E-07	2E-07	5E-07

			Air quality indicator:	$PM_{2.5}$	$PM_{2.5}$	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
			Endpoint:	Mortality - All Causes	Hospitalisations - Cardiovascular	Hospitalisations - Respiratory	Mortality - All Causes	Morbidity - Asthma ED Admissions
			Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term
			Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
			β (change in effect per 1 μg/m^3) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
			Annual baseline incidence (per 100,000	1026	9235	4168	643.2	2284
			Baseline Incidence (per person per year)	0.01026	0.09235	0.04168	0.006432	0.02284
384	8.1E-02	2.0E-02		1E-06	1E-06	3E-07	3E-07	7E-07
444	1.1E-01	2.8E-02		2E-06	2E-06	5E-07	4E-07	9E-07
399	9.1E-02	2.4E-02		1E-06	2E-06	4E-07	4E-07	8E-07
420	1.4E-01	3.6E-02		2E-06	3E-06	6E-07	6E-07	1E-06
410	2.4E-01	5.9E-02		4E-06	4E-06	1E-06	9E-07	2E-06
404	8.1E-02	2.2E-02		1E-06	2E-06	4E-07	3E-07	7E-07
387	1.3E-01	3.3E-02		2E-06	2E-06	6E-07	5E-07	1E-06
145a	4.7E-02	7.8E-03		5E-07	6E-07	1E-07	2E-07	3E-07
145b	3.8E-02	6.5E-03		4E-07	5E-07	1E-07	1E-07	2E-07
145c	3.4E-02	5.8E-03		3E-07	4E-07	1E-07	1E-07	2E-07
58b	2.6E-02	4.7E-03		3E-07	3E-07	8E-08	1E-07	2E-07
57	3.5E-02	6.1E-03		4E-07	5E-07	1E-07	1E-07	2E-07
536	6.0E-02	9.8E-03		6E-07	7E-07	2E-07	2E-07	3E-07
507	3.3E-02	8.7E-03		5E-07	6E-07	1E-07	1E-07	3E-07
390	2.5E-01	5.7E-02		3E-06	4E-06	1E-06	1E-06	2E-06
460	4.2E-02	1.1E-02		7E-07	8E-07	2E-07	2E-07	4E-07
440	5.0E-02	1.3E-02		8E-07	1E-06	2E-07	2E-07	5E-07
435a	3.5E-02	9.2E-03		5E-07	7E-07	2E-07	1E-07	3E-07
435b	4.6E-02	1.2E-02		7E-07	9E-07	2E-07	2E-07	4E-07
433b	3.3E-02	8.8E-03		5E-07	6E-07	2E-07	1E-07	3E-07
433a	5.0E-02	1.3E-02		8E-07	1E-06	2E-07	2E-07	4E-07
253	1.4E-02	2.7E-03		2E-07	2E-07	5E-08	5E-08	9E-08
411	2.1E-01	5.2E-02		3E-06	4E-06	9E-07	8E-07	2E-06
423	9.9E-02	2.6E-02		2E-06	2E-06	4E-07	4E-07	9E-07
425	8.3E-02	2.2E-02		1E-06	2E-06	4E-07	3E-07	7E-07
209	2.3E-02	4.2E-03		3E-07	3E-07	7E-08	9E-08	1E-07
418	2.0E-01	5.1E-02		3E-06	4E-06	9E-07	8E-07	2E-06
509	2.6E-02	6.9E-03		4E-07	5E-07	1E-07	1E-07	2E-07
25	1.8E-02	3.5E-03		2E-07	3E-07	6E-08	7E-08	1E-07
24b	1.7E-02	3.3E-03		2E-07	2E-07	6E-08	7E-08	1E-07
24a	1.7E-02	3.3E-03		2E-07	2E-07	6E-08	7E-08	1E-07
386	1.1E-01	2.6E-02		2E-06	2E-06	4E-07	4E-07	9E-07
284	1.3E-02	2.5E-03		1E-07	2E-07	4E-08	5E-08	8E-08
403	1.1E-01	2.9E-02		2E-06	2E-06	5E-07	4E-07	1E-06
427	6.8E-02	1.8E-02		1E-06	1E-06	3E-07	3E-07	6E-07
402	8.9E-02	2.4E-02		1E-06	2E-06	4E-07	3E-07	8E-07
429	5.2E-02	1.4E-02		8E-07	1E-06	2E-07	2E-07	5E-07
285	1.3E-02	2.4E-03		1E-07	2E-07	4E-08	5E-08	8E-08
172	2.0E-02	3.8E-03		2E-07	3E-07	7E-08	8E-08	1E-07
456	5.2E-02	1.4E-02		8E-07	1E-06	2E-07	2E-07	5E-07
385	5.6E-02	1.5E-02		9E-07	1E-06	3E-07	2E-07	5E-07
446a	1.1E-01	2.8E-02		2E-06	2E-06	5E-07	4E-07	9E-07
438	2.1E-02	5.9E-03		3E-07	4E-07	1E-07	8E-08	2E-07
299	1.6E-02	3.1E-03		2E-07	2E-07	5E-08	6E-08	1E-07
58a	2.7E-02	4.8E-03		3E-07	4E-07	8E-08	1E-07	2E-07
389	3.5E-01	7.7E-02		5E-06	6E-06	1E-06	1E-06	3E-06
228q	1.4E-02	2.7E-03		2E-07	2E-07	5E-08	5E-08	9E-08
228r	1.5E-02	2.9E-03		2E-07	2E-07	5E-08	6E-08	1E-07
537	9.3E-02	2.1E-02		1E-06	2E-06	4E-07	4E-07	7E-07
538	7.0E-02	1.9E-02		1E-06	1E-06	3E-07	3E-07	6E-07
539	1.3E-01	3.2E-02		2E-06	2E-06	5E-07	5E-07	1E-06

Quantification of Effects - $PM_{2.5}$ and PM_{10}
Maxwell Project: Scenario 3 (Year 4)

Air quality indicator:	$PM_{2.5}$	$PM_{2.5}$	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Endpoint:	**Mortality - All Causes**	**Hospitalisations - Cardiovascular**	**Hospitalisations - Respiratory**	**Mortality - All Causes**	**Morbidity - Asthma ED Admissions**
Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term
Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
β (change in effect per 1 µg/m³) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
Annual baseline incidence (per 100,000	1026	9235	4168	643.2	2284
Baseline Incidence (per person per year)	0.01026	0.09235	0.04168	0.006432	0.02284

Sensitive Receptors	Change in Annual Average PM10 Concentration (µg/m³)	Change in Annual Average PM2.5 Concentration (µg/m³)	Risk	Risk	Risk	Risk	Risk
Assessment of impacts are each receptor							
278	1.2E-02	1.6E-03	9E-08	1E-07	3E-08	5E-08	5E-08
279	1.2E-02	1.5E-03	9E-08	1E-07	3E-08	5E-08	5E-08
306	1.3E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
451	1.2E-01	1.6E-02	9E-07	1E-06	3E-07	5E-07	5E-07
455	1.3E-01	1.7E-02	1E-06	1E-06	3E-07	5E-07	6E-07
255	1.1E-02	1.5E-03	9E-08	1E-07	3E-08	4E-08	5E-08
421	2.0E-01	2.5E-02	1E-06	2E-06	4E-07	8E-07	8E-07
441b	1.3E-01	1.7E-02	1E-06	1E-06	3E-07	5E-07	6E-07
441a	1.3E-01	1.7E-02	1E-06	1E-06	3E-07	5E-07	6E-07
219c	1.4E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
219a	1.4E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
219b	1.4E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
219d	1.4E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
230a	8.4E-03	1.1E-03	6E-08	8E-08	2E-08	3E-08	4E-08
228k	9.3E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228g	9.3E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228e	9.2E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228c	9.2E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228b	9.2E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228a	9.2E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228j	8.7E-03	1.1E-03	7E-08	8E-08	2E-08	3E-08	4E-08
228l	1.0E-02	1.3E-03	8E-08	1E-07	2E-08	4E-08	4E-08
228m	1.1E-02	1.4E-03	8E-08	1E-07	2E-08	4E-08	5E-08
226c	2.3E-02	2.8E-03	2E-07	2E-07	5E-08	9E-08	1E-07
226a	2.2E-02	2.7E-03	2E-07	2E-07	5E-08	9E-08	9E-08
226d	1.9E-02	2.3E-03	1E-07	2E-07	4E-08	7E-08	8E-08
227f	1.5E-02	1.9E-03	1E-07	1E-07	3E-08	6E-08	6E-08
228n	1.2E-02	1.5E-03	9E-08	1E-07	3E-08	5E-08	5E-08
227e	1.5E-02	1.9E-03	1E-07	1E-07	3E-08	6E-08	6E-08
227d	1.5E-02	1.9E-03	1E-07	1E-07	3E-08	6E-08	7E-08
227c	1.6E-02	2.0E-03	1E-07	1E-07	3E-08	6E-08	7E-08
227b	1.7E-02	2.1E-03	1E-07	2E-07	4E-08	6E-08	7E-08
227a	1.7E-02	2.2E-03	1E-07	2E-07	4E-08	7E-08	7E-08
226b	2.2E-02	2.8E-03	2E-07	2E-07	5E-08	9E-08	9E-08
228i	9.3E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228h	9.3E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
228f	9.3E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
230b	8.9E-03	1.1E-03	7E-08	8E-08	2E-08	3E-08	4E-08
228o	1.1E-02	1.4E-03	8E-08	1E-07	2E-08	4E-08	5E-08
217c	1.5E-02	1.9E-03	1E-07	1E-07	3E-08	6E-08	6E-08
217d	1.4E-02	1.8E-03	1E-07	1E-07	3E-08	6E-08	6E-08
217e	1.4E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
527	1.1E-02	1.4E-03	8E-08	1E-07	2E-08	4E-08	5E-08
219e	1.3E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
228p	9.6E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
217f	1.3E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
398	2.6E-01	3.1E-02	2E-06	2E-06	5E-07	1E-06	1E-06
500	4.2E-02	5.3E-03	3E-07	4E-07	9E-08	2E-07	2E-07
211a	1.4E-02	1.8E-03	1E-07	1E-07	3E-08	6E-08	6E-08
528	1.4E-02	1.8E-03	1E-07	1E-07	3E-08	5E-08	6E-08
211b	1.5E-02	1.8E-03	1E-07	1E-07	3E-08	6E-08	6E-08
211c	1.5E-02	1.8E-03	1E-07	1E-07	3E-08	6E-08	6E-08
508	5.4E-02	6.7E-03	4E-07	5E-07	1E-07	2E-07	2E-07
238a	7.8E-03	1.0E-03	6E-08	8E-08	2E-08	3E-08	3E-08
238b	7.7E-03	1.0E-03	6E-08	7E-08	2E-08	3E-08	3E-08
238c	7.8E-03	1.0E-03	6E-08	8E-08	2E-08	3E-08	3E-08
238d	7.8E-03	1.0E-03	6E-08	8E-08	2E-08	3E-08	3E-08
238e	7.8E-03	1.0E-03	6E-08	8E-08	2E-08	3E-08	3E-08
238f	7.9E-03	1.0E-03	6E-08	8E-08	2E-08	3E-08	3E-08
239g	8.8E-03	1.2E-03	7E-08	9E-08	2E-08	3E-08	4E-08
239a	8.6E-03	1.1E-03	7E-08	8E-08	2E-08	3E-08	4E-08
239f	8.6E-03	1.1E-03	7E-08	8E-08	2E-08	3E-08	4E-08
239e	8.9E-03	1.2E-03	7E-08	9E-08	2E-08	3E-08	4E-08
239d	8.9E-03	1.2E-03	7E-08	9E-08	2E-08	3E-08	4E-08
239c	9.0E-03	1.2E-03	7E-08	9E-08	2E-08	3E-08	4E-08
239b	9.1E-03	1.2E-03	7E-08	9E-08	2E-08	3E-08	4E-08
239h	9.0E-03	1.2E-03	7E-08	9E-08	2E-08	3E-08	4E-08
239i	9.3E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
240d	1.3E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
240c	1.3E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
240b	1.3E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
240e	1.3E-02	1.6E-03	1E-07	1E-07	3E-08	5E-08	6E-08
240a	1.2E-02	1.5E-03	9E-08	1E-07	3E-08	4E-08	5E-08
239j	9.5E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
239k	9.4E-03	1.2E-03	7E-08	9E-08	2E-08	4E-08	4E-08
238g	7.6E-03	1.0E-03	6E-08	7E-08	2E-08	3E-08	3E-08
238h	7.7E-03	1.0E-03	6E-08	7E-08	2E-08	3E-08	3E-08
207	1.3E-02	1.7E-03	1E-07	1E-07	3E-08	5E-08	6E-08
419	4.4E-01	5.4E-02	3E-06	4E-06	9E-07	2E-06	2E-06
424	1.4E-01	1.8E-02	1E-06	1E-06	3E-07	6E-07	6E-07
532	1.2E-02	1.6E-03	9E-08	1E-07	3E-08	5E-08	5E-08
60a	1.4E-01	1.5E-02	9E-07	1E-06	3E-07	5E-07	5E-07
60b	1.7E-01	1.9E-02	1E-06	1E-06	3E-07	7E-07	6E-07
60c	1.6E-01	1.8E-02	1E-06	1E-06	3E-07	6E-07	6E-07
60d	1.5E-01	1.7E-02	1E-06	1E-06	3E-07	6E-07	6E-07
254a	1.1E-02	1.5E-03	9E-08	1E-07	3E-08	4E-08	5E-08
254b	1.1E-02	1.5E-03	9E-08	1E-07	3E-08	4E-08	5E-08
254c	1.1E-02	1.5E-03	9E-08	1E-07	3E-08	4E-08	5E-08
250b	1.6E-02	2.1E-03	1E-07	2E-07	4E-08	6E-08	7E-08
250a	1.6E-02	2.1E-03	1E-07	2E-07	4E-08	6E-08	7E-08
432	7.2E-02	8.9E-03	5E-07	7E-07	2E-07	3E-07	3E-07
443	1.9E-01	2.4E-02	1E-06	2E-06	4E-07	7E-07	8E-07
298a	1.5E-02	1.9E-03	1E-07	1E-07	3E-08	6E-08	6E-08
298b	1.4E-02	1.8E-03	1E-07	1E-07	3E-08	5E-08	6E-08
400	9.8E-02	1.2E-02	7E-07	9E-07	2E-07	4E-07	4E-07

			Air quality indicator:	$PM_{2.5}$	$PM_{2.5}$	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
			Endpoint:	Mortality - All Causes	Hospitalisations - Cardiovascular	Hospitalisations - Respiratory	Mortality - All Causes	Morbidity - Asthma ED Admissions
			Effect Exposure Duration:	Long-term	Short-term	Short-term	Short-Term	Short-Term
			Age Group:	≥ 30 years	≥ 65 years	≥ 65 years	All ages	1-14 years
			β (change in effect per 1 µg/m^3) (as per Table 5.3)	0.0058	0.0008	0.00041	0.0006	0.00148
			Annual baseline incidence (per 100,000	1026	9235	4168	643.2	2284
			Baseline Incidence (per person per year)	0.01026	0.09235	0.04168	0.006432	0.02284
384	1.8E-01	2.2E-02		1E-06	2E-06	4E-07	7E-07	8E-07
444	2.5E-01	3.1E-02		2E-06	2E-06	5E-07	1E-06	1E-06
399	1.6E-01	1.9E-02		1E-06	1E-06	3E-07	6E-07	6E-07
420	3.2E-01	3.9E-02		2E-06	3E-06	7E-07	1E-06	1E-06
410	5.3E-01	6.4E-02		4E-06	5E-06	1E-06	2E-06	2E-06
404	1.3E-01	1.6E-02		1E-06	1E-06	3E-07	5E-07	5E-07
387	2.6E-01	3.0E-02		2E-06	2E-06	5E-07	1E-06	1E-06
145a	2.8E-02	3.4E-03		2E-07	2E-07	6E-08	1E-07	1E-07
145b	2.3E-02	2.8E-03		2E-07	2E-07	5E-08	9E-08	1E-07
145c	2.1E-02	2.5E-03		2E-07	2E-07	4E-08	8E-08	9E-08
58b	1.7E-02	2.2E-03		1E-07	2E-07	4E-08	7E-08	7E-08
57	2.3E-02	2.9E-03		2E-07	2E-07	5E-08	9E-08	1E-07
536	3.6E-02	4.2E-03		3E-07	3E-07	7E-08	1E-07	1E-07
507	5.7E-02	7.2E-03		4E-07	5E-07	1E-07	2E-07	2E-07
390	5.2E-01	6.0E-02		4E-06	4E-06	1E-06	2E-06	2E-06
460	7.2E-02	9.1E-03		5E-07	7E-07	2E-07	3E-07	3E-07
440	8.9E-02	1.1E-02		7E-07	8E-07	2E-07	3E-07	4E-07
435a	6.1E-02	7.6E-03		5E-07	6E-07	1E-07	2E-07	3E-07
435b	8.6E-02	1.1E-02		6E-07	8E-07	2E-07	3E-07	4E-07
433b	5.4E-02	6.7E-03		4E-07	5E-07	1E-07	2E-07	2E-07
433a	9.4E-02	1.2E-02		7E-07	9E-07	2E-07	4E-07	4E-07
253	1.2E-02	1.6E-03		9E-08	1E-07	3E-08	5E-08	5E-08
411	4.7E-01	5.7E-02		3E-06	4E-06	1E-06	2E-06	2E-06
423	1.8E-01	2.2E-02		1E-06	2E-06	4E-07	7E-07	8E-07
425	1.5E-01	1.9E-02		1E-06	1E-06	3E-07	6E-07	6E-07
227c	1.6E-02	2.0E-03		1E-07	1E-07	3E-08	6E-08	7E-08
227b	1.7E-02	2.1E-03		1E-07	2E-07	4E-08	6E-08	7E-08
227a	1.7E-02	2.2E-03		1E-07	2E-07	4E-08	7E-08	7E-08
226b	2.2E-02	2.8E-03		2E-07	2E-07	5E-08	9E-08	9E-08
228i	9.3E-03	1.2E-03		7E-08	9E-08	2E-08	4E-08	4E-08
228h	9.3E-03	1.2E-03		7E-08	9E-08	2E-08	4E-08	4E-08
228f	9.3E-03	1.2E-03		7E-08	9E-08	2E-08	4E-08	4E-08
230b	8.9E-03	1.1E-03		7E-08	8E-08	2E-08	3E-08	4E-08
228o	1.1E-02	1.4E-03		8E-08	1E-07	2E-08	4E-08	5E-08
217c	1.5E-02	1.9E-03		1E-07	1E-07	3E-08	6E-08	6E-08
217d	1.4E-02	1.8E-03		1E-07	1E-07	3E-08	6E-08	6E-08
217e	1.4E-02	1.7E-03		1E-07	1E-07	3E-08	5E-08	6E-08
527	1.1E-02	1.4E-03		8E-08	1E-07	2E-08	4E-08	5E-08
219e	1.3E-02	1.7E-03		1E-07	1E-07	3E-08	5E-08	6E-08
228p	9.6E-03	1.2E-03		7E-08	9E-08	2E-08	4E-08	4E-08
217f	1.3E-02	1.7E-03		1E-07	1E-07	3E-08	5E-08	6E-08
398	2.6E-01	3.1E-02		2E-06	2E-06	5E-07	1E-06	1E-06
500	4.2E-02	5.3E-03		3E-07	4E-07	9E-08	2E-07	2E-07
211a	1.4E-02	1.8E-03		1E-07	1E-07	3E-08	6E-08	6E-08
528	1.4E-02	1.8E-03		1E-07	1E-07	3E-08	5E-08	6E-08
211b	1.5E-02	1.8E-03		1E-07	1E-07	3E-08	6E-08	6E-08
211c	1.5E-02	1.8E-03		1E-07	1E-07	3E-08	6E-08	6E-08
508	5.4E-02	6.7E-03		4E-07	5E-07	1E-07	2E-07	2E-07
238a	7.8E-03	1.0E-03		6E-08	8E-08	2E-08	3E-08	3E-08
238b	7.7E-03	1.0E-03		6E-08	7E-08	2E-08	3E-08	3E-08
238c	7.8E-03	1.0E-03		6E-08	8E-08	2E-08	3E-08	3E-08

Appendix C Risk calculations – NO_2

Quantification of Effects - NO_2
Maxwell Project

	Constant emissions at licence limits		
Air quality indicator:	**NO2**	**NO2**	**NO2**
Endpoint:	**Mortality - All Causes (non-trauma)**	**Mortality - Respiratory**	**Asthma - ED Hospital admissions**
Effect Exposure Duration:	Short-term	Short-term	Short-term
Age Group:	All ages	All ages	1-14 years
β (change in effect per 1 µg/m^3 NO2) (as per Table 5.6)	0.00188	0.00426	0.00115
Annual baseline incidence (per 100,000)	643.2	51.9	2284
Baseline Incidence (per person per year)	0.006432	0.000519	0.02284

Sensitive Receptors	**Change in Annual Average NO2 Concentration (µg/m^3)**	**Risk**	**Risk**	**Risk**
Maximum predicted anywhere off-site				
Scenario 1	0.40	5E-06	9E-07	1E-05
Scenario 2	0.10	1E-06	2E-07	3E-06
Scenario 3	0.03	4E-07	7E-08	8E-07