GLOUCESTER RESOURCES LTD

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Part 2B:
Health Risk Assessment

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COMMONLY USED ACRONYMS

AANEPM	Ambient Air National Environmental Protection Measure
ACS	American Cancer Society
AEGL	Acute exposure guideline level
AGV	Air guideline value
bsp	Back-scattering pattern (relates to dust measurement)
CI	Confidence interval
COPD	Chronic obstructive pulmonary disease
COMEAP	(UK) Committee on the Medical Effects of Air Pollutants
CRF	Concentration-response function
GLC	Ground level concentrations
GRL	Gloucester resources limited
HQ	Hazard quotient
HRA	Health risk assessment
LOEL	Lowest-observed-effect level
NEPC	National Environmental Protection Council
NEPM	National Environmental Protection Measure
NHC	National Health Council (of New Zealand)
NOAEL	No observed adverse effect level
NOx	Refers to Nitric Oxide (NO) and Nitrogen Dioxide (NO_2)
NSW	New South Wales
PM	Particulate matter
REL	Reference exposure level
RR	Relative risk
WHO	World Health Organization

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EXECUTIVE SUMMARY

Gloucester Resources Limited (GRL) proposes to develop and operate a small scale open cut coal mine, approximately 3.5km to 7km southeast of the Gloucester urban area, which is located approximately 120km north of Newcastle, New South Wales (NSW) in the Gloucester Basin (the Proposal).

GRL has contracted Toxikos Pty Ltd to perform an independent human health risk assessment (HRA) of the emissions to air from the Proposal. The HRA was conducted using methodology consistent with enHealth guidance; it evaluates the possibility of impacts on human health from acute (i.e. short term) and chronic (i.e. long term) inhalation exposure. Ground level concentrations (GLCs) of emission components were predicted at discrete locations around the Site by air dispersion modelling conducted by Pacific Environment Limited and provided to Toxikos. The emissions of concern addressed in the assessment were particulate matter (PM) and nitrogen dioxide (NO_2) (resulting from blasting).

The exposure assessment used estimates of the potential exposure concentrations (e.g. background plus modelled emissions from the Proposal) on an annual and daily basis due to emissions from the Proposal at the most affected receptor on the worst day. The use of these values adds to the conservatism of the HRA, as it overestimates the potential exposures to all the potential receptors.

The air quality impact assessment for the Proposal presents the dispersion modelling predictions for maximum 24-hour at a total of 6 private assessment locations and annual average $PM_{2.5}$ GLCs at a total of 150 private assessment locations in the vicinity of the Proposal. In examining the windroses, historically (based on 2010, 2011 data presented in **Figure 4-1**) the prevailing wind is from the north, northeast, however there is also a southerly wind pattern present. In the case of $PM_{2.5}$, the full dataset based on the 150 receptors examined in conjunction with background were examined as part of the HRA (see **Figure 4-2**). In examining the full data set, it is noted that based on the air modelling, over 90% of the time the cumulative $PM_{2.5}$ concentrations are less than 10$\mu g/m^3$, while approximately 65% of the time, the cumulative concentrations are less than 5$\mu g/m^3$. **Table 4-1** outlines the separate contributions of background and those of the Proposal based on various percentiles. It is noted that the background contribution represents 70 – 80% of the total cumulative concentrations of $PM_{2.5}$ (based on a 24-hour averaging period). Similarly, the background contribution represents approximately 90% of the total cumulative concentrations of the annual $PM_{2.5}$ concentrations. As such, the contribution of the emissions from the Proposal represents only a small proportion with respect to overall air quality in the area.

In further examining this data it should be noted that the 97.5th percentile incremental $PM_{2.5}$ concentration (24-hour average) is 5.3$\mu g/m^3$. This value was used in the HRA as a conservative measure to determine the potential risks to receptors in the area of the Proposal. In the case of the annual concentration the 97.5th percentile incremental $PM_{2.5}$ concentration (annual) is 0.51$\mu g/m^3$ and was used in the HRA to determine the potential risks to receptors in the area of the Proposal. In order to provide a conservative outcome for the HRA, these concentrations are used in the risk calculations.

For PM, the health end points assessed were short- and long-term mortality and daily hospitalisations. The general approach used to calculate the risks to health has drawn upon internationally recognised estimates of the impact of $PM_{2.5}$ on health in relation to the known health indicators for NSW. It involves estimating the change in the incidence of a health outcome resulting from a given change in $PM_{2.5}$ concentrations. Concentration-response functions (CRFs) for each of the health endpoints were sourced from the current epidemiological literature and utilised with exposure estimates to estimate increases in occurrences at the worst exposure receptors.

The critical health outcomes with acute and chronic exposure to NO_2 include respiratory disease and associated symptoms, and associated changes in lung function. The Australian NEPC Ambient Guideline Values, which are protective of these health outcomes in sensitive subpopulations, have been used to determine the potential for acute effects (246µg/m^3) and chronic effects (61.5µg/m^3) in association with exposure to NO_2 from the Proposal.

In examining the risk in the population due to the increased long-term exposure to $PM_{2.5}$ as a result of the Proposal, an increase in base incidence annual mortality from 658 to 660 per 100,000 was estimated, a resultant increase of 0.002%. This increased risk is considered to be negligible. Shorter term exposures to $PM_{2.5}$ are also not considered to pose an unacceptable risk as the estimated risk is orders of magnitude lower than that due to long-term exposure.

As NO_2 emissions for the worst affected receptor were less than the Australian AGVs, it is very unlikely the blast emissions will cause direct acute and/or chronic health effects. This outcome is reinforced by the use of a Blast Management Plan, which will avoid the modelled NO_2 concentrations that were utilised in this assessment.

Overall, it is concluded that air emissions from the proposed Rocky Hill Coal Project present little likelihood of causing adverse health effects to exposed individuals around the Site.

1. INTRODUCTION

1.1 GENERAL

Gloucester Resources Limited (GRL) proposes to develop and operate a small scale open cut coal mine, approximately 3.5km to 7km southeast of the Gloucester urban area, which is located approximately 120km north of Newcastle, New South Wales (NSW) in the Gloucester Basin (**Figure 1.1**).

The proposed Rocky Hill Coal Project comprises four principal components namely:

1. Four separate and/or contiguous open cut pits and a coal handling and preparation plant (CHPP) within the Mine Area.
2. An overland conveyor for transporting product coal to the Rail Load-out Facility. The overland conveyor is located within a 50m wide Overland Conveyor Corridor.
3. A Rail Load-out Facility (incorporating a rail loop and two coal storage bins).
4. Two Power Line Corridors incorporating a re-located 132kV power line and a new 11kV power line external to the Mine Area.

GRL has contracted Toxikos Pty Ltd to perform an independent human health risk assessment (HRA) of the emissions to air from the Proposal. The methodology adopted in the HRA is consistent with the protocols and guidelines recommended by the Australian enHealth Council (enHealth, 2012).

1.2 WHAT IS A HEALTH RISK ASSESSMENT?

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

The following are examples of determinants of health well-being (enHealth 2012, NHC 2004):

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

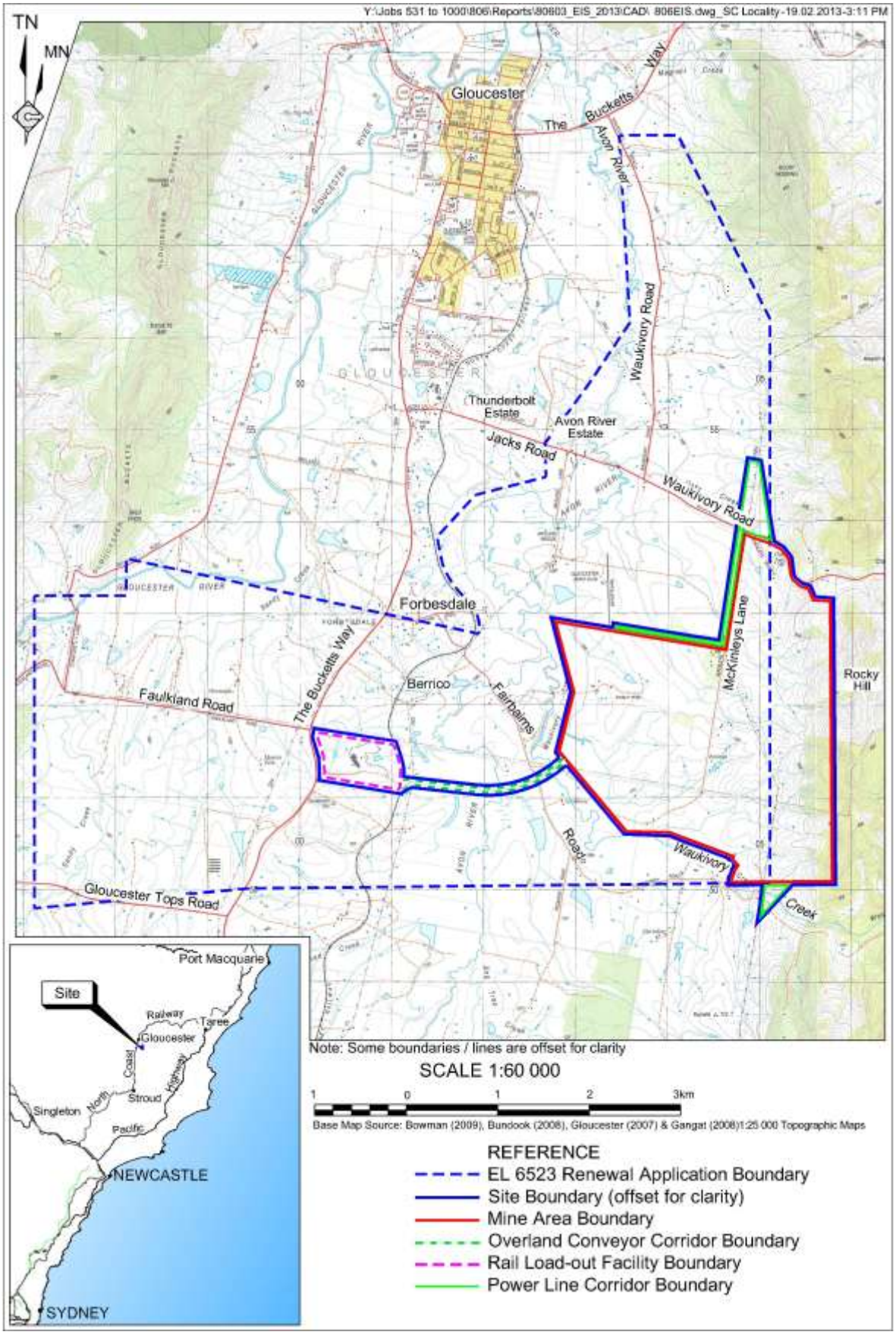


Figure 1.1 Locality plan

According to enHealth (2012), all developments have a potential impact on health. Some will have positive health impacts by providing jobs, attracting health services to an area, and improving overall economic well-being of a community etc. Other projects may have negative impacts such as increased risk of disease, social disruption, increased noise etc. Many developments will have both positive and negative aspects. It should be understood the potential influence of the Proposal on local area economic factors, social disruption and other such factors are not addressed in this document. Air quality is one of the many parameters influencing well-being. This HRA seeks to evaluate what the likelihood is for direct health effects when exposures to air emissions from the Proposal occur.

A health risk assessment is an analysis that uses information about hazardous pollutants to estimate a theoretical level of risk for people who might be exposed to defined levels of these substances. The information comes from scientific studies and measurement data of air emissions.

Risk assessments are often conducted by considering possible or theoretical community exposures predicted from air dispersion modelling. Conservative safety margins are built into a risk assessment analysis to ensure protection of the public. Therefore, people will not necessarily become unwell even if they are exposed to pollutants at higher concentration levels than those estimated by the risk assessment. During the risk assessment analysis, the most vulnerable people (e.g. children, the sick and elderly) are carefully considered to make sure that all members of the public will be protected.

The risk assessment helps answer the following common questions for people who might be exposed to hazardous pollutants in the environment, in this case components of the air emissions from the Proposal:

- Under what circumstances might I, my family and neighbours be exposed to hazardous pollutants from the Proposal?
- Is it possible we might be exposed to hazardous pollutants at levels higher than those determined to be safe?
- If the levels of hazardous pollutants are higher than regulatory standards, what are the health effects that might occur?

The HRA is a useful tool for estimating the likelihood and severity of risks to human health, safety and the environment and for informing decisions about how to manage those risks. It is a document that assembles and synthesizes scientific information to determine whether a potential hazard exists and/or the extent of possible risk to human health.

Although this report describes certain technical aspects of the risk assessment, it does not address the processes of risk management and risk communication.

1.3 SCOPE OF THE RISK ASSESSMENT

This HRA addresses likely impacts on community health from exposure to air emissions from the Proposal, considering the direct health effects from acute (i.e. short term) and chronic (i.e. long term) exposures. This HRA evaluates the potential of the emissions to cause direct effects on individuals who may be exposed either on a short term, infrequent basis or long term, i.e. assuming 24 hours per day for each day of the year for 70 years. Thus, from the aspect of chronic exposure assumptions, the HRA is conservative (i.e. errs on the side of safety), as the life of the mine is estimated to be between 16 and 21 years.

The risk assessment has been facilitated by provision of spread sheet results for the emission dispersion modelling undertaken by Pacific Environment Limited. These spread sheets contain predicted ground level concentrations (GLCs) of individual pollutants at sensitive receptor locations where people reside or gather.

2. AIR QUALITY HEALTH RISK ASSESSMENT METHODOLOGY

The methodology adopted in the conduct of this HRA is consistent with the protocols and guidelines recommended by the enHealth Council. These are detailed in the document "*Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazards*" (enHealth, 2012).

The development of a formalised HRA has resulted in the process being categorised into distinct stages. Some of the key factors and questions that are taken into consideration at each of these stages include the following.

1. **Hazard and Concentration – Dose-Response Assessment** – Identifies hazards and health endpoints associated with exposure to hazardous pollutants and provides a review of the current understanding of the toxicity and risk relationship of the exposure of humans to the hazards.
2. **Exposure Assessment** – This task identifies the groups of people who may be exposed to hazardous pollutants and provides an estimate as to the potential exposure concentrations.
3. **Risk Characterisation** – This task provides the qualitative/quantitative evaluation of potential risks to human health. The characterisation of risk is based on the review of the dose-response relationship and the assessment of the magnitude of exposure.

3. AIR QUALITY HAZARD AND CONCENTRATION – RESPONSE ASSESSMENT

3.1 IDENTIFICATION OF EMISSIONS OF HAZARDOUS POLLUTANTS

Mining activities generate fugitive dust emissions in the form of particulate matter (PM). Blasting is an activity undertaken in open cut mining operations that has the potential to result in emissions of oxides of nitrogen (NO_X). NO_X comprises both nitric oxide (NO) and nitrogen dioxide (NO_2). From the point of view of impacts on human health, it is NO_2 which is of greatest concern.

3.2 HEALTH ENDPOINTS

The adverse health effects resulting from exposure to ambient pollutants, such as PM and NO_2 range from the relatively mild sub clinical effects such as throat irritation, clinical effects of reduction in lung function or increased medication usage, through to seeking medical attention from a General Practitioner (GP), emergency department attendances, hospital admission and premature mortality due to various diseases. **Figure 3.1** illustrates the relationship between the frequency of an adverse health outcome and its severity. Mortality and hospital admissions are often studied in relation to ambient air pollutants, since they are clearly defined health outcomes that have a measurable impact on the community. Further discussion on the health endpoints of concern with respect to PM and NO_X are provided in Section 3.3 (Hazard Assessment).

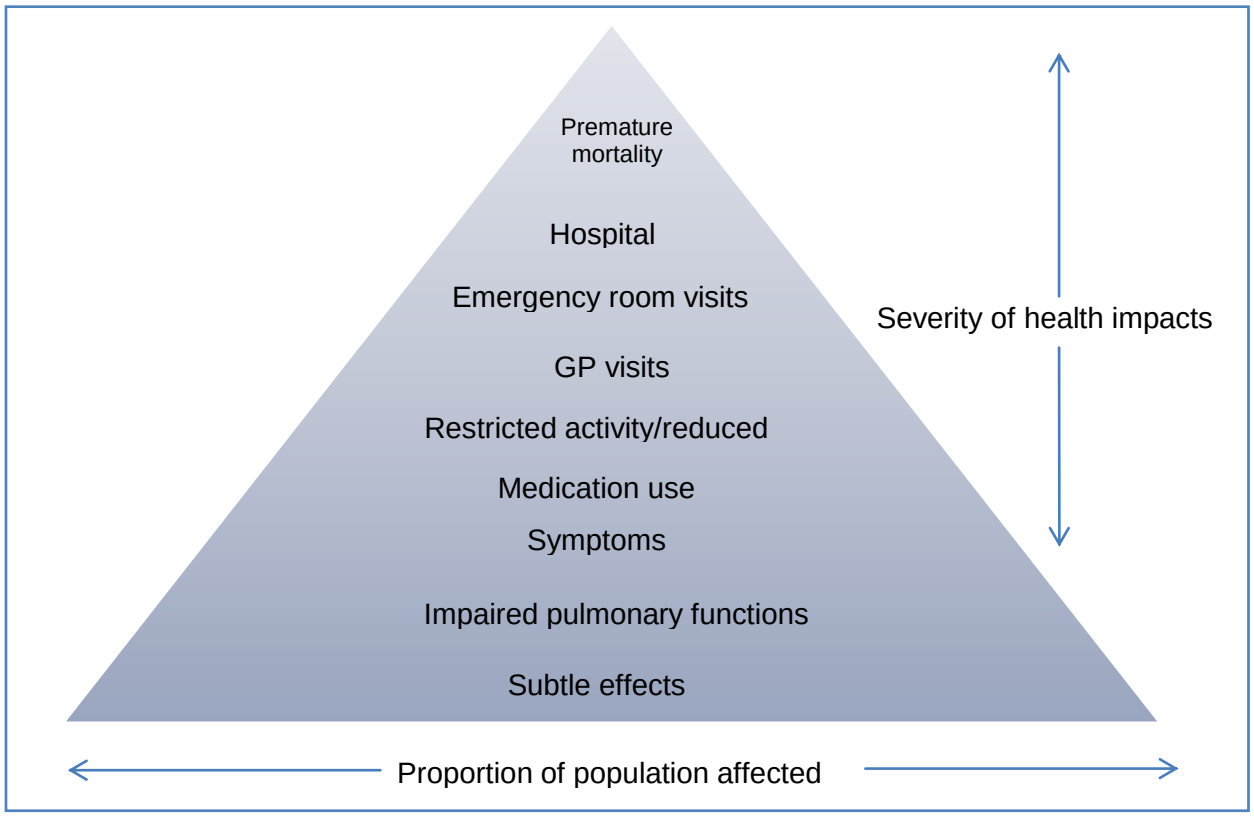


Figure 3.1 The severity and frequency of an adverse health impact as a result of ambient air pollution exposure (from WHO [2001])

3.3 HAZARD ASSESSMENT

3.3.1 Particulate Matter

Particulate matter is an air-suspended mixture of solid and liquid particles that vary in number, size, shape, surface area, chemical composition, solubility and origin. PM is classified by aerodynamic diameter, as size is a critical determinant of the likelihood and site of deposition within the respiratory tract.

- PM_{10} includes all inhalable particles less than 10 µm aerodynamic diameter. These are sufficiently small to penetrate to the thoracic region. Coarse particles consist of those between $PM_{2.5}$ and PM_{10} (i.e. $PM_{10-2.5}$) and sometimes may be referred to as 'thoracic' (coarse particles).
- $PM_{2.5}$ (fine particles) includes those inhalable particles less than 2.5 µm aerodynamic diameter. These have a high probability of deposition in the smaller conducting airways and alveoli.
- PM_{10} includes coarse, fine and ultrafine particles.
- $PM_{2.5}$ includes fine and ultrafine particles.

Both natural and anthropogenic processes contribute to the atmospheric load of PM. Coarse particles ($PM_{2.5-10}$) are derived primarily from mechanical processes resulting in the suspension of dust, soil, or other crustal[1] materials from roads, farming, mining, dust storms, and so forth. Coarse particles also include sea salts, pollen, mould, spores, and other plant parts. In general, mining dust is likely to be composed of predominantly coarse particulate matter (and larger).

$PM_{2.5}$ is derived primarily from combustion processes, such as vehicle emissions, wood burning, coal burning for power generation, and natural processes such as bush fires. Fine particles also consist of transformation products, including sulphate and nitrate particles, and secondary organic aerosol from volatile organic compound emissions.

All particles irrespective of their origin appear to cause adverse health impacts. In recent years, a significant amount of research has focused on the health effects of particles and an increasing body of literature reports associations between PM and adverse health effects. Epidemiological studies of the health effects of air pollution are usually classified as investigating *acute effects (*due to short-term exposures) or *chronic effects* (due to long-term exposures).

A range of health effects have been found for both PM_{10} and $PM_{2.5}$ and the majority of the information comes from population-based epidemiological studies.

3.3.2 PM Health effects and concentration-response relationships

Studies have demonstrated that for a wide range of PM concentrations, including concentrations below current assessment criteria, there are measurable associations between incremental increases in particle concentration and adverse health effects. That is, no threshold has been identified for particles and any increase in concentration may affect health.

[1] Crustal dust refers to dust generated from materials derived from the earth's crust.

As such, it can be deduced that ambient guidelines do not lead to complete protection against adverse health effects of PM.

However, the results of epidemiological studies can be used to make quantitative estimates concerning the health effects of air pollution on a population. A concentration response function (CRF) (reported by epidemiological studies) is the empirically estimated relationship between the concentration of PM and the observed health endpoints of interest (for example, hospital admissions for asthma) in a population.

There is a dose-response relationship with PM and many health outcomes where the health risk increases with exposure to both PM_{10} and $PM_{2.5}$.

Epidemiological studies estimating health outcomes in the population are often not available for a particular location, or the available results from local studies may not be considered as robust as the combined results of epidemiological studies from other locations/populations. Because the uncertainty in the precision of the risk estimate from an epidemiological study decreases with increasing sample size (for example, the population in the study), combining results from several studies may yield more robust estimates of effect (e.g., meta-analysis).

At present, Australian studies, in particular studies involving mining and extractive industries are currently considered insufficient to reliably establish specific Australian concentration-response relationships for relevant health outcomes and PM exposures. Therefore, overseas data has been be used to quantify health effects in this health risk assessment. Additionally, in line with WHO advice, all particles should be treated as equally harmful irrespective of source and chemical composition.

In order to generate sufficient data to adequately characterise the concentration-response relationship for compounds present in air, meta-analytic point estimates rather than estimates from one single study can enhance the value of the available information and deal with potential heterogeneity between studies. As a result, cities with large populations provide the best data upon which to study and characterise the concentration-response relationship for compounds present in air. Similarly, multi-city studies can also produce even more reliable effect estimates as the sample size is much larger than those in single city studies. While, the results are based on compounds and exposures typical to an urban setting and not those of a rural, mine-based setting, they nevertheless provide the best information upon which to characterise the concentration-response.

Over the last few decades, there has been a substantial amount of research that has added to the evidence that breathing PM is harmful to human health. Various lines of research have helped connect some of the important gaps in our knowledge. Different studies using alternative time series approaches and case crossover designs continue to observe reasonably consistent associations between morbidity and mortality outcomes and daily changes in PM. The associations are observed, not only in many single-city studies, but also in various large multicity studies (Pope and Dockery, 2006). The evidence of long-term chronic health effects has been strengthened by various reanalyses and extended analyses of the Harvard Six Cities study (Dockery *et al*. 1993), ACS cohorts (Pope *et al*. 1995) and by results from several other independent studies of long-term PM exposure.

The PM-mortality effect estimates from the studies of long-term exposure are substantially larger than those from the short-term time series or case-crossover studies that evaluate daily changes in exposure. Overall, the results suggest that PM health effects are dependent on both exposure concentrations and length of exposure and that the short-term studies only capture a small amount of the overall health effects of PM exposure. Long-term repeated exposures have larger, more persistent cumulative effects than short-term transient exposures.

It is important to note that the observed association between PM and health outcomes is statistical. As such, particles are not the primary cause of the observed increase in mortality, but are one of many environmental and other risk factors. More recently the statistical associations have been revised downwards based on a review of the statistical methods used, but the association remains (HEI, 2003).

3.3.3 Evidence of acute effects from short-term exposures to PM

A recent review examining PM exposure increments (predominantly examining increased incremental exposures of 10 & 20µg/m^3 sized particles) and its associated relative found that short-term exposure to PM is likely to be causally associated with mortality from cardiopulmonary diseases, hospitalisation and emergency department visits for cardiopulmonary diseases, increased respiratory symptoms, decreased lung function, and physiological changes or biomarkers for cardiac changes (Pope and Dockery, 2006).

The epidemiological data collected have undergone various meta-analyses and the most recent and comprehensive analysis of worldwide data was undertaken by the WHO (Anderson *et al*. 2004). The results of this analysis of short-term effects for PM_{10} and $PM_{2.5}$ are summarised in **Table 3.1**.

Table 3.1
Short term effects on health from 10µg/m^3 increases in PM concentration

Health outcome	**Estimated percentage increase in risk per 10µg/m^3 PM_{10} (95% confidence interval)**	**Estimated percentage increase in risk per 10µg/m^3 $PM_{2.5}$ (95% confidence interval)**
All-cause mortality	0.6 (0.4-0.8)	0.9 (0.6-1.3)
Mortality from respiratory diseases	1.3 (0.3-2.0)	1.1 (0.2-2.0)
Mortality from cardiovascular diseases	0.9 (0.5-1.3)	1.3 (0.5-2.2)
Hospital admissions for respiratory disease, people age 65 years and over	0.7 (0.2-1.3)	
Cough, children aged 5-15years with chronic symptoms	0.0 (-1.3-1.1)	
Medication use, children aged 5-15 years with chronic symptoms	0.5 (-1.9-2.9)	
Source: Anderson HR *et al*. Meta-analysis of time series studies and panel studies of particulate matter (PM) and ozone (O_3) (Report of a WHO task group). Copenhagen, WHO Regional Office for Europe, 2004 (http://www.euro.who.int/document/e82792.pdf)		

A recent meta-analysis in Australia was undertaken by Simpson and others using data from Sydney, Perth, Melbourne and Brisbane (Simpson, 2005a and 2005b). Data from mass measurements of PM_{10} and $PM_{2.5}$ were analysed along with measurements of light scattering using nephelometry and reported as back-scattering pattern (bsp). Nephelometry measures total suspended particulate (not just PM_{10} and $PM_{2.5}$) and reports the results as back-scattering pattern based on how much light is scattered as the source is passed through the stream and reflected due to the particle density. **Table 3.3** and **Table 3.4** summarise the increase in daily mortality and daily hospital admissions in Australia arising from increased exposure to particulate matter. It should be noted that the results based on exposure in an urban environment may not reflect those that are present in the area of the Proposal. Concentrations of other contributing factors to bsp and levels of compounds associated with PM are likely higher in an urban environment than those present in the area of the Proposal. Bsp data were available for the four cities and PM_{10} and $PM_{2.5}$ data were available for three of the cities. It is noted from the results presented in **Table 3.2** that no statistically significant mortality risk is present based on PM_{10} or $PM_{2.5}$ exposure, however a correlation is evident in terms of an increased risk in relation to bsp.

Table 3.2
Increases in daily mortality as a result of a 1 unit increase in 24-hour bsp or a 10µg/m^3 increase in 24-hour PM_{10} or $PM_{2.5}$ (Simpson, 2005a)

City	Cause of mortality	Estimated percentage increase in risk (95% confidence interval (CI))
Four cities (bsp)	All causes	2.84 (0.15-5.6)
	Cardiovascular	4.79 (0.76-8.98)
Three cities (PM_{10})	All causes	0.2 (-0.8-1.2)
Three cities ($PM_{2.5}$)	All causes	0.9 (-0.7-2.5)

Table 3.3
Increases in hospital admissions as a result of a 1 unit increase in bsp or a 10µg/m^3 increase in 24-hour PM_{10} or $PM_{2.5}$ (Simpson, 2005a)

City	Hospital admissions	Estimated percentage increase in risk (95% CI)
Four cities (bsp)	Cardiac all ages	8.56 (6.03-11.16)
	Respiratory 65+ years	5.52 (0.82-10.45)
	Asthma 15-64 years	8.93 (0.24-15.87)
Three cities (PM_{10})	Cardiac all ages	2.4 (1.5-3.4)
	Respiratory 65+ years	2.9 (1.3-4.4)
Three cities ($PM_{2.5}$)	Cardiac all ages	5.1 (3.5-6.7)

In the case of reported hospitalisations, it is noted from the results presented in **Table 3.3** that there is a correlation between PM_{10} or $PM_{2.5}$ exposure and hospitalisation, with an even larger correlation being present in relation to bsp.

Published by the US EPA (Abt Associates Inc. 2008), Moolgavkar reported an association between $PM_{2.5}$ and cardiovascular disease and respiratory hospital admissions of 1.6% and 0.7%, respectively, for a 10µg/m^3 increase in $PM_{2.5}$.

The daily excess mortality (defined as a premature death, or one that occurs before the average life expectancy for a person of a particular demographic category) for PM_{10} is lower for the Australian data (0.2% (i.e. Simpson 2005a, 2005b)) than the world wide data (0.6%). The daily excess mortality for $PM_{2.5}$ is the same (0.9%) for the Australian data and the world wide data.

In regards to hospital admissions for PM_{10}, comparisons between admissions for respiratory disease in people aged 65+ shows a higher level in the Australian data (2.9% increase in risk) compared to 0.7% increase in worldwide studies. Cardiovascular hospital admissions for $PM_{2.5}$ are lower for US data (1.6%) than for Australia (5.1%).

3.3.4 Evidence of chronic effects from long-term exposures to PM

Long-term exposure to PM is associated with all-cause mortality and deaths resulting from cardiopulmonary diseases and lung cancer, and effects on the respiratory system such as decreased lung function or the development of chronic respiratory disease. Long-term $PM_{2.5}$ exposure has been shown to be strongly associated with ischemic heart disease, dysrhythmias, heart failure and cardiac arrest mortality.

Long-term exposure to high levels of PM is linked to reductions in life expectancy of the average population by a year or more. It is generally thought that the public health significance of the long-term effects of PM clearly outweighs that of the short-term effects (World Health Organization, 2004).

The major cohort studies of long-term exposure to $PM_{2.5}$ and mortality are summarised in **Table 3.4**.

Table 3.4
Summary of percentage increase (and 95% CI) in relative risk of all cause and cause specific mortality associated with 10µg/m^3 increase in long-term particulate exposure to $PM_{2.5}$

Study cohort	Authors	Per cent increase in mortality (all-cause) (95% CI)
Harvard 6 cities, original	Dockery *et al* 1993	13 (4.2,2.3)
Harvard 6 cities HEI reanalysis	Krewski *et al* 2000	14 (4.5,23)
Harvard 6 cities, extended analysis	Laden *et al* 2006	16 (7,26)
ACS original	Pope *et al* 1995	6.5 (3.5,9.8)
ACS HEI analysis	Krewski *et al* 2000	7.0 (3.5,10)
ACS extended analysis	Pope *et al* 2002	6.2 (1.6,11)
Source: Modified from Pope and Dockery (Pope and Dockery, 2006)		

The Pope *et al.* (2002) analysis summarised in **Table 3.4** is a longitudinal cohort tracking. It uses the same American Cancer Society (ACS) cohort as the original Pope *et al*. (1995) study and Krewski *et al.* (2000) reanalysis. Pope *et al.* (2002) analysed the survival data for the cohort from 1982 through 1998, 9 years longer than the original Pope study. Pope *et al.* (2002) presented results for all-cause deaths, lung cancer and cardiopulmonary mortality, reporting a relative risk of 1.06 for all-cause mortality associated with a change in annual mean exposure of 10µg/m^3.

The UK report (COMEAP, 2009) recommends using a relative risk of 1.06 for all-cause mortality associated with a 10µg/m^3 increase in $PM_{2.5}$.

3.3.5 $PM_{2.5}$ concentration-response functions used in this assessment

Many studies have used PM_{10} as an indicator of PM. However, there is increasing evidence that the adverse health effects, particularly mortality, are more closely associated with $PM_{2.5}$ (Pope and Dockery, 2006). A recent UK report states that $PM_{2.5}$ is considered the best index of PM for quantitative assessments of the effects of PM (COMEAP, 2009). For this reason, $PM_{2.5}$ is considered the best index of PM air pollution for quantitative assessments of the associated health effects (COMEAP, 2009). In this assessment, $PM_{2.5}$ has been used as the metric to assess risks to health from exposure to PM.

Table 3.5 provides the health endpoints and concentration response functions used in this study. We did not use the risk estimates from the four cities study from Australia (5.1% increase for a 10µg/m^3 increase in $PM_{2.5}$) as the authors considered their results indicative only due to the small number of cities included in the analysis (Simpson, Williams *et al*. 2005).

Table 3.5
Health endpoints and concentration response function for 10µg/m^3 increase in $PM_{2.5}$

Health endpoint	Concentration response function for 10µg/m^3 increase in $PM_{2.5}$	Reference
Deaths		
Long-term deaths (age 30+ years)	1.06 (1.02-1.11)	(Pope, *et al*. 2002)
Short-term all non-trauma deaths (all ages)	1.009 (1.006-1.013)	(Anderson, *et al* 2004)
Hospitalisations		
Cardiovascular disease (age 65+ years)	1.0159 (1.0092-1.0227)	(Moolgavkar 2003)
All respiratory disease (all ages)	1.007 (1.002-1.013)	(Anderson, *et al* 2004)

As the incremental increases in $PM_{2.5}$ as a result of the Rocky Hill Coal Project are less than or equal to 10µg/m^3 (noted to be 1 – 11µg/m^3 for a 24-hour average and 0.1 – 1.8µg/m^3 for an annual average based on PEL, 2013) it is necessary to convert the relative risks (RR) above to RRs for the estimated increments from the Proposal. This is accomplished by converting the RR from an increment of 10µg/m^3 back to the beta coefficient derived from the statistical model and adjusting accordingly.

An example calculation for the long-term RR of 1.06 (6%), based on the excess mortality for $PM_{2.5}$ as outlined in **Table 3.5** is, provided below.

- Convert the RR back to the beta-coefficient - Natural log (1.06) = 0.05827 for 10µg/m^3 change in $PM_{2.5}$
- Convert the beta coefficient for a 1µg/m^3 change in $PM_{2.5}$ - 0.05827 / 10 = 0.005827.
- Reconvert beta coefficient to relative risk by calculating the exponent of the beta coefficient - RR for 1µg/m^3 change in $PM_{2.5}$ = exp (0.005827) = 1.005844.

Table 3.6 displays the resultant RRs for a 1µg/m^3 increase in $PM_{2.5}$ used for risk estimation in this assessment.

Table 3.6
Health endpoints and concentration response function for 1µg/m^3 increase in $PM_{2.5}$

Health endpoint	Concentration response function for 1µg/m^3 increase in $PM_{2.5}$ - Relative risk
Deaths[a]	
Long-term deaths (age 30+ years)	1.0058 (0.58%)
Short-term all non-trauma deaths (all ages)	1.0009 (0.09%)
Hospitalisations[b]	
Cardiovascular disease (age 65+ years)	1.0016 (0.16%)
All respiratory disease (all ages)	1.0007 (0.07%)
[A] Annual average $PM_{2.5}$ used for long-term deaths [b] 24-h average $PM_{2.5}$ used for hospitalisations	

3.3.6 Oxides of Nitrogen and Health Effects

Blasting activities at the Site have the potential to result in emissions of oxides of nitrogen (NO_X). NO_X comprises both nitric oxide (NO) and nitrogen dioxide (NO_2). From the point of view of impacts on human health, it is NO_2 which is of greatest concern.

In recent years, the health effects of NO_2 linked to ambient exposures have been well studied and reviewed by international agencies (USEPA, 2008a; WHO, 2006; California EPA). The main acute health outcomes identified in epidemiology studies are increased respiratory disease and symptoms. The evidence for the chronic effects of long-term exposure to NO_2 is limited. As with acute exposure, the critical health outcomes with chronic exposure include respiratory disease and associated symptoms, and associated changes in lung function. Individuals with asthma and other chronic lung disease and cardiovascular diseases are recognised as being particularly vulnerable. Other susceptible populations include infants, children and the elderly (>65 years of age) (NEPM 2010).

Only very high concentrations of NO_2 (approximately 2 000µg/m^3 (~1,050 ppb)) affect breathing in healthy people. However, small changes in lung function (< 5%) and changes in airway responsiveness have been reported in several studies of sensitive asthmatics or the elderly exposed to concentrations as low as 375-575µg/m^3 (~200-300 ppb) over a period of 20 minutes to 4h (Bauer *et al*., 1986; Bylin *et al*., 1988; Roger et al., 1990; Morrow *et al*., 1992; Strand *et al*., 1996, 1997, Streeton 1997). These levels represent a clear lowest-observed-effect level (LOEL) for NO_2 based on increased responsiveness in mild asthmatics to bronchoconstrictors or in subjects with chronic obstructive pulmonary disease (COPD). It is noted that the study by Bauer *et al*., (1986) did not find a significant change in pulmonary function when asthmatics were exposed to 560µg/m^3 NO_2 when resting, with decreases recorded only after the subjects exercised. Similarly, testing asthmatics the day after exposure to 490µg/m^3 NO_2 did not decrease lung function before allergen challenge (Strand *et al*., 1997), noting that the effects of NO_2 exposure are transient in nature.

The identification of an obvious no observed adverse effect level (NOAEL) is less clear, but it is suggested to be around 200µg/m^3 (approx 100 ppb). Studies have shown that effects can be detected in mild asthmatics after short-term exposure (20 minutes to 4 hours in duration) to 488-500µg/m^3 (260-240 ppb) NO_2 who are subsequently exposed to an inhalation challenge (Strand *et al*., 1996, 1997; Kraft *et al*., 2005). However, in a study where mild asthmatic subjects were exposed for 1h to 200µg/m^3 (~100 ppb) NO_2 and then immediately exposed to a house dust mite challenge, the late asthmatic response (as tested using forced expiratory volume in one second; FEV1) was found to be greater than when compared to air (NO_2 -7.76% vs. Air -2.85%), but the results were not found to be significant (Tunnicliffe *et al*., 1994). The current air guideline for acute exposure to NO_2 in the NEPM is 246µg/m^3 (0.12 ppm) measured as a 1h average.

According to Streeton (1997) there is an increasing body of evidence to suggest that longer term (years) ambient exposure to significantly lower concentrations of NO_2, of the order of 40 - 80 ppb (approx 75-150µg/m^3) during early and middle childhood years 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.

Similarly, more recent studies of self-reported asthmatic individuals living in homes with flue-less gas heaters have shown significant effects of NO_2 exposures to those aged ≤14 years with chest tightness, breathlessness on exertion and asthma attacks experienced either the same day or with one-day lag (Smith *et al*., 2000). The range of median indoor levels of NO_2 measured by positional passive samplers in homes during this study were indicated to be between 0-147 ppb (0-277µg/m^3) with time weighted average levels measured by personal passive sampler of 0-1 760 ppb (0-3 300µg/m^3). Subsequent investigations with flue-less space heaters in primary schools indicated that over the 12 week winter heating period asthma symptoms were significantly higher in children exposed to gas combustion products, with mean NO_2 levels of 47.0 ppb (88µg/m^3) versus children in schools where a replacement intervention programme had removed or replaced the flue-less gas heaters, leading to a mean NO_2 level of 15.5 ppb (29.3µg/m^3) (Pilotto *et al*., 2004).

Based upon a review of the literature, Streeton (1997) considered short-term ambient exposures to 200-300 ppb (375-565µg/m^3) NO_2 and chronic exposures between 40-80 ppb (75-150µg/m^3) capable of causing recurrent upper and lower respiratory tract symptoms, an increased incidence of respiratory infection and onset of symptoms in mild asthmatics. Streeton (1997) considered these effects as the lowest observed adverse effect levels (LOAEL), and has suggested that an uncertainty factor of 2 need apply to account for susceptible people within the population, therefore establishing a short-term guideline in the range 100-150 ppb (205-307.5µg/m^3) as a 1h average and a chronic guideline between 20-40 ppb (41-82µg/m^3) for longer term exposures as an annual average (Streeton, 1997).

The WHO (1997, 2000) took a different approach to reach a similar conclusion. Similar to Streeton (1997), the WHO noted the epidemiological studies suggesting human health effects associated with long-term NO_2 exposures; however, the WHO (1997) have stated that this is supported by animal toxicological findings showing increased susceptibility to respiratory infections and impairment of host defences as a result of subchronic or chronic exposures to NO_2 concentrations near ambient concentrations (i.e. 20-60µg/m^3; 11-32 ppb). On the basis of a background level of 15µg/m^3 (8 ppb) as determined in Finland during the 1980s (Jaakkola *et al*., 1991) and the fact that significant adverse health effects occur with an additional

concentration of 28.2µg/m^3 (15 ppb) or more, which is an estimate of an increased risk of about 20% for respiratory symptoms and disease (Hasselblad *et al*., 1992; WHO, 1997), an annual guideline value of 40µg/m^3 (22 ppb) was derived by the WHO (1997). The WHO considers the guideline value will be protective of most serious effects. The fact that a no-effect level for subchronic or chronic NO_2 exposure concentrations has not yet been determined was emphasised.

Since their publication, both the NEPM and the WHO air quality guidelines for particles, ozone, nitrogen dioxide and sulfur dioxide have been subject to review (NEPM, 2010; WHO, 2006). In both instances, review of the guidelines considered newly available information from various locations around the world, including Australia. The WHO concluded that the scientific literature has not accumulated sufficient evidence to justify revising the existing NO_2 guidelines. According to NEPM (2010), available epidemiological information indicates increased hospital admissions and emergency department attendance for respiratory symptoms, particularly in asthmatics and children, following short-term exposure to ambient concentrations. However, the available information remains under consideration by the NEPM and no changes to the standards have been made at this point in time (NEPM 2010).

In summary:

- Concentrations of around 2 000µg/m^3 (~1 000 ppb) are needed to affect respiration of healthy people based on acute exposure (less than 24 hours) to NO_2.
- The low effect level for increased bronchial reactivity in sensitive asthmatics is 375-575µg/m^3 (~200-300 ppb) for exposures from 20 minutes up to 4 hours.
- The no effect level for increased bronchial reactivity based on acute exposure is ~200 ppb.
- The increased bronchial reactivity may remain for up to 10 hours after cessation of NO_2 exposure.

Table 3.7 presents a summary of guideline values established relating to acute and chronic exposure to NO_2. For the estimation of risks, the NEPC (1998) Ambient Air National Environmental Protection Measure (AANEPM) has been used.

Table 3.7
Summary of national and international guideline values established relating to acute exposure to NO_2

Guideline µg / m^3 / ppb		Averaging period	Derivation	Reference
246	120	Ambient air quality guideline 1 hr average	The Australian National Environmental Protection Council ambient air quality standard. It is based on a low observed adverse effect level (LOAEL) of 0.2 to 0.3 ppm 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 2 to protect susceptible people (i.e. asthmatic children) was applied to the LOAEL.	NEPC (1998), Streeton (1997)
470	250	Inhalation reference exposure level (REL) 1 hr average	The REL is also the ambient air quality standard of California. It is the no observed adverse effect level in sensitive asthmatics for NO_2 mediated increased responsiveness to other bronchoconstrictors (e.g. exercising in cold air).	Cal EPA (1999a)
200	106	Ambient air quality guideline 1 hr average	Lowest concentration causing small (~5%) changes in lung function in mild asthmatics is 560µg/m^3. Some but not all studies show increased responsiveness to bronchoconstrictors at NO_2 levels as low as 376–560µg/m^3. In other studies, higher levels had no such effect. Allergen challenges showed no effects at 190µg/m^3. According to WHO there have been no studies of 1 hour exposures to NO_2 at 100µg/m^3.	WHO (2000b)
940	500	Acute Exposure Guideline Level (AEGL – 1) 10 min, 30 min, 1 hr average.	Based on a study that reported that 7/13 asthmatics experienced slight burning of the eyes, slight headache, and chest tightness or laboured breathing with exercise when exposed at 500 ppb for 2 h; at this concentration, the odour of NO_2 was perceptible but the subjects became unaware of it after about 15 min. No changes in any pulmonary function tests were found immediately following the chamber exposure. The effects are not disabling and are transient and reversible upon cessation of exposure.	USEPA (2001)

Based on the above, the study by Streeton (1997) provides the most scientifically defensible information with respect to the potential for both acute and chronic effects in association with exposure to NO_2. As a result, the Australian NEPC Air Guideline Value AGVs, which are protective of health outcomes in sensitive subpopulations, are used to determine the potential for acute effects (246µg/m^3) and chronic effects (61.5µg/m^3) in association with exposure to NO_2 from the Proposal.

4. EXPOSURE ASSESSMENT

The exposure assessment outlined below estimates the total cumulative exposure (e.g., background plus modelled emissions from the Proposal) on an annual and daily basis at the most affected receptor on the worst day. The use of these values adds to the conservative of the HRA, as it overestimates the potential exposures to all the potential receptors. In the case of evaluating exposure to air, there are other factors that must be considered as the concentrations are anticipated to vary according to the direction and strength of the wind, time of day, and how far away the receptor is from the source of the air emissions. Wind roses for the Site for the periods of July 2010 to June 2011 and July 2011 to June 2012, as previously shown in the associated Air Quality Assessment report (PEL, 2013), are shown in **Figure 4.1.** On an annual basis, the two years of wind roses show similar patterns with winds from the south and northeast. The annual percentage of calms (winds less than 0.5 m/s) is 6.1% and 7.0% respectively and the annual average wind speeds are again similar at 2.6 and 2.3 m/s respectively.

4.1 EXPOSURE TO $PM_{2.5}$

The air quality impact assessment for the Proposal (PEL, 2013) presents the dispersion modelling predictions for maximum 24-hour and annual average $PM_{2.5}$ GLCs at a total of 150 private assessment locations in the vicinity of the Proposal. In examining the windroses, historically (based on 2010, 2011 data presented in **Figure 4.1**) the prevailing wind is from the north, northeast, however there is also a southerly wind pattern present. In the case of $PM_{2.5}$, the full dataset based on the 150 private receptors examined in conjunction with background were examined as part of the HRA (**Figure 4.2**). In examining the full data set (as shown in **Figure 4.3** via the frequency distribution), it is noted that based on the air modelling over 90% of the time, the cumulative $PM_{2.5}$ concentrations are less than $10\mu g/m^3$, while approximately 65% of the time, the cumulative concentrations are less than $5\mu g/m^3$. **Table 4.1** outlines the separate contributions of background and those of the proposal based on various percentiles. It is noted that the background contribution represents 70 – 80% of the total cumulative concentrations of $PM_{2.5}$ (based on a 24-hour averaging period). Similarly, the background contribution represents approximately 90% of the total cumulative concentrations of the annual $PM_{2.5}$ concentrations. As such, the contribution of the emissions from the Proposal represent only a small proportion with respect to overall air quality in the area.

Table 4.1
Background and Incremental $PM_{2.5}$ concentrations (24-hour and annual) (in $\mu g/m^3$)

	50th	**95th**	**97.5th**
24-hour			
Background	3.0	10.4	14
Incremental	0.7	4.2	5.3
Annual			
Background	4.0		
Incremental	0.23	0.46	0.51

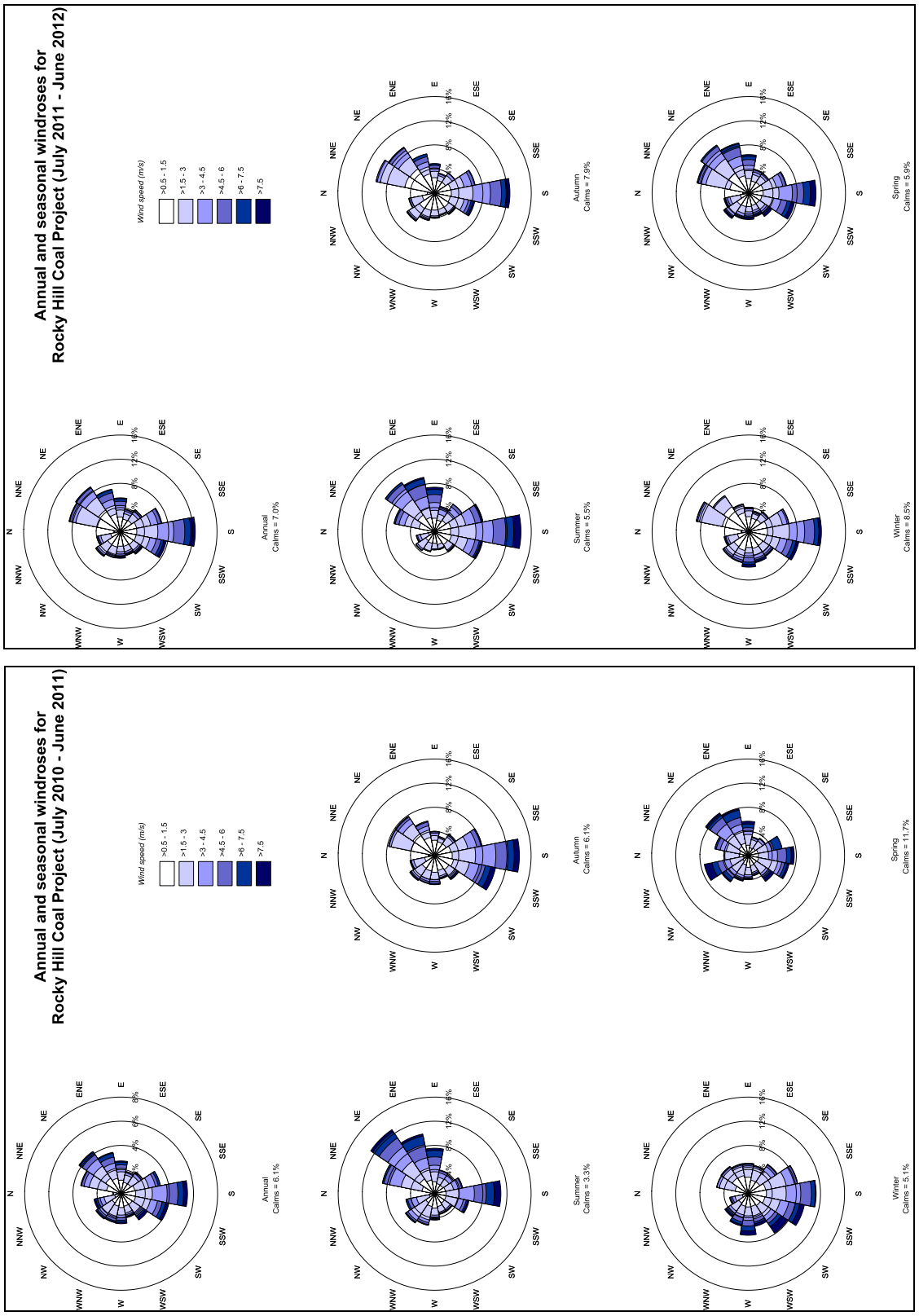
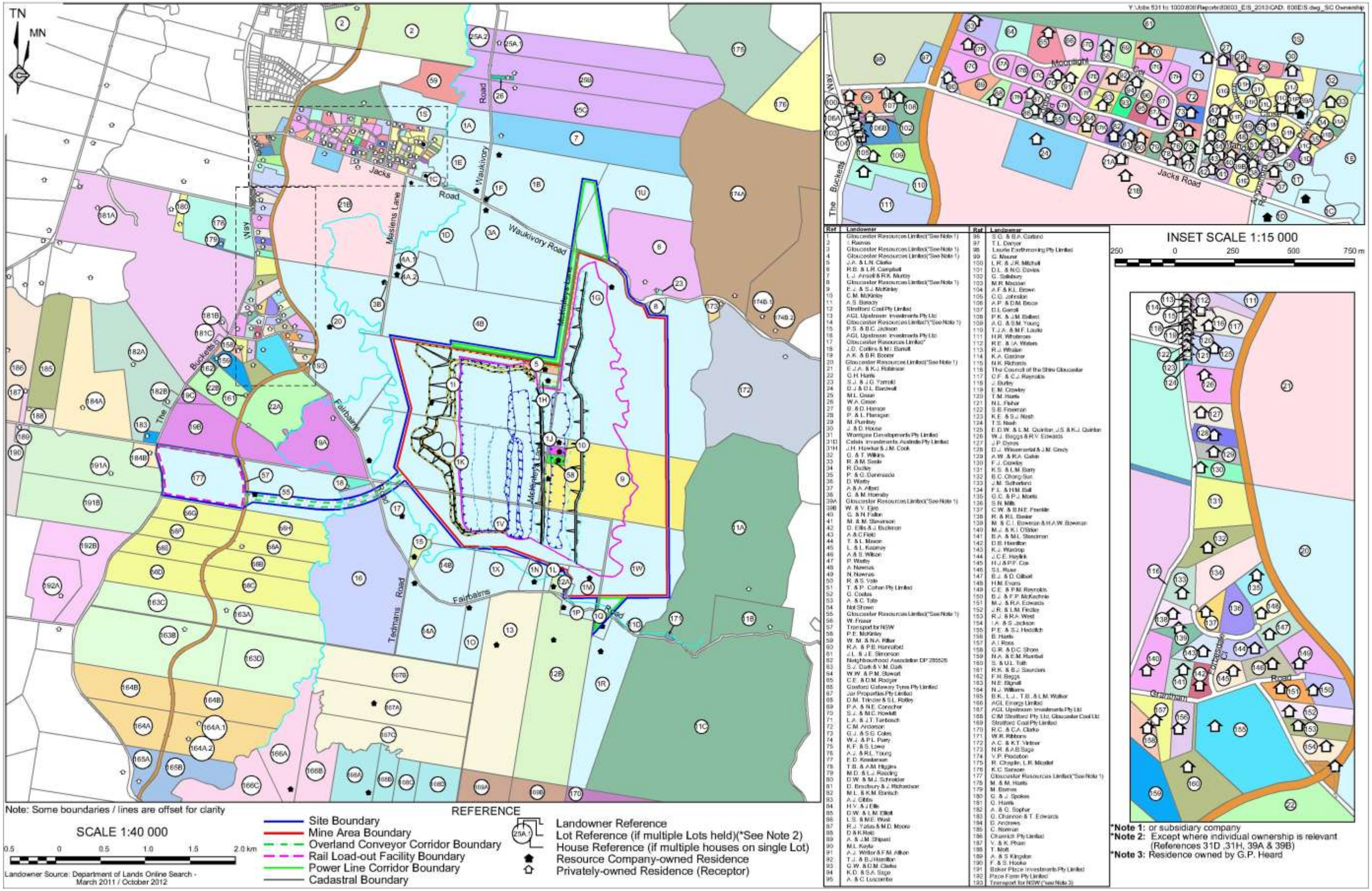


Figure 4.1 Annual and Seasonal Wind Roses for the Rocky Hill Coal Project (July 2010 – June 2011 and July 2011 – June 2012)



Ref	Landowner
1	Gloucester Resources Limited(*See Note 1)
2	J. Keenan
3	Gloucester Resources Limited(*See Note 1)
4	Gloucester Resources Limited(*See Note 1)
5	J.A. & L.N. Clarke
6	R.B. & L.R. Campbell
7	L.J. Ansell & R.K. Mundy
8	Gloucester Resources Limited(*See Note 1)
9	E.J. & S.J. McKinley
10	C.M. McKinley
11	A.S. Barclay
12	Stratford Coal Pty Limited
13	AGL Upstream Investments Pty Ltd
14	Gloucester Resources Limited(*See Note 1)
15	P.S. & D.C. Jackson
16	AGL Upstream Investments Pty Ltd
17	Gloucester Resources Limited*
18	J.D. Collins & M.J. Barrett
19	A.K. & B.R. Boorer
20	Gloucester Resources Limited(*See Note 1)
21	E.J.A. & K.J. Robinson
22	G.H. Harris
23	S.J. & J.G. Yarrold
24	D.J. & D.L. Bardwell
25	M.L. Dixon
26	W.A. Green
27	B. & D. Hanson
28	P. & L. Flanagan
29	M. Pumbuy
30	J. & D. House
31	Wombgee Developments Pty Limited
31D	Calala Investments Australia Pty Limited
31H	J.H. Hawker & J.M. Cook
32	G. & T. Wilkins
33	R. & M. Seale
34	R. Dudley
35	P. & G. Dominoccio
36	D. Watty
37	A & A. Allard
38	G. & M. Hornsby
39A	Gloucester Resources Limited(*See Note 1)
39B	W. & V. Ejlec
40	G. & N. Fallon
41	M. & M. Stevenson
42	D. Ellis & J. Buckman
43	A & C Field
44	T. & L. Mason
45	L. & L. Kearney
46	A & S. Wilson
47	P. Watty
48	A. Nownes
49	N. Nownes
50	R. & S. Vale
51	T. & P. Cohen Pty Limited
52	G. Coates
53	A. & C. Tate
54	Not Shown
55	Gloucester Resources Limited(*See Note 1)
56	W. Fraser
57	Transport for NSW
58	P.E. McKinley
59	W. M. & N.A. Ritter
60	R.A. & P.B. Hannaford
61	J.L. & J.E. Simonson
62	Neighbourhood Association DP 285576
63	S.J. Clark & V.M. Clark
64	W.W. & P.M. Stewart
65	C.E. & D.M. Rayner
66	Gosford Gateway Tyres Pty Limited
67	Jar Properties Pty Limited
68	D.M. Trihidir & S.L. Rolley
69	P.A. & N.E. Corscher
70	S.J. & M.C. Howlett
71	L.A. & J.T. Tanbonch
72	C.M. Anderson
73	G.J. & S.G. Coles
74	W.J. & P.L. Parry
75	K.F. & S. Lowe
76	A.J. & R.L. Young
77	F.D. Keelanson
78	T.B. & A.M. Higgins
79	M.D. & L.J. Reading
80	D.W. & M.J. Schneider
81	D. Brockbury & J. Richardson
82	M.L. & K.M. Bonisch
83	A.J. Gibbs
84	H.V. & J. Ellis
85	D.W. & L.M. Elliott
86	L.S. & M.E. West
87	R.J. Yallas & M.D. Moore
88	D & K Reid
89	A. & J.M. Shipard
90	M.L. Keyte
91	A.J. Writer & F.M. Aitken
92	T.J. & B.J Hamilton
93	G.W. & D.M. Clarke
94	K.D. & S.A. Sage
95	A. & C. Luscombe
96	S.G. & B.A. Carbone
97	T.L. Denyer
98	Laurie Earthmoving Pty Limited
99	G. Maurer
100	L.R. & J.R. Mitchell
101	D.L. & N.G. Davies
102	G. Salisbury
103	M.R. Maclean
104	A.F. & K.L. Brown
105	C.G. Johnston
106	A.P. & D.M. Brice
107	D.L Gerroll
108	P.K. & J.M. Bellert
109	A.G. & S.M. Young
110	T.J.A. & M.F. Lauriu
111	H.R. Whitemore
112	R.E. & J.A. Waters
113	R.J. Whalon
114	K.A. Gardner
115	N.K. Richards
116	The Council of the Shire Gloucester
117	C.F. & C.J. Reynolds
118	J. Burley
119	E.M. Crowley
120	T.M. Hunts
121	N.L. Fisher
122	S.B. Freeman
123	K.E. & S.J. Nash
124	T.S. Nash
125	E.D.W. & L.M. Quinlan, J.S & K.J. Quinlan
126	W.J. Beggs & R.V. Edwards
127	J.P. Dynes
128	D.J. Wilsonsmith & J.M. Grecy
129	A.W. & R.A. Calvin
130	F.J. Cowlips
131	K.S. & L.M. Barry
132	B.C. Chong-Sun
133	J.M. Sutherland
134	F.L. & H.M. Ball
135	G.C. & P.J. Morris
136	S.N. Mills
137	C.W. & B.N.E. Franklin
138	R. & R.L. Baxter
139	M. & C.I. Bowman & H.A.W. Bowman
140	M.J. & K.I. O'Brien
141	B.A. & M.L. Steadman
142	D.B. Hamilton
143	K.J. Wardrop
144	J.C.E. Heylink
145	H.J. & P.F. Cox
146	S.L. Rose
147	B.J. & D. Gilbert
148	H.M. Evans
149	C.E. & P.M. Reynolds
150	D.J. & F.P. McKechnie
151	M.J. & R.A. Edwards
152	J.R. & L.M. Findlay
153	R.J. & R.A. West
154	I.A. & S. Jackson
155	P.E. & S.J. Hezelton
156	B. Harris
157	A.I. Ross
158	G.R. & D.C. Shore
159	N.A. & E.M. Mumbel
160	S. & U.L. Tuth
161	R.K. & B.J. Saunders
162	F.H. Beggs
163	N.E. Bignall
164	N.J. Williams
165	B.K. L.J., T.B. & L.M. Walker
166	AGL Energy Limited
167	AGL Upstream Investments Pty Ltd
168	CIM Stratford Pty Ltd, Gloucester Coal Ltd
169	Stratford Coal Pty Limited
170	R.C. & C.A. Clarke
171	W.R. Ribbons
172	A.C. & K.T. Vintner
173	N.R. & A.B. Sage
174	V.P. Pearston
175	R. Chaplin, L.R. Micallef
176	K.C. Sansom
177	Gloucester Resources Limited(*See Note 1)
178	M. & M. Harris
179	M. Barnes
180	G. & J. Spokes
181	G. Harris
182	A. & G. Sophar
183	G. Channon & T. Edwards
184	D. Andrews
185	C. Norman
186	Chuenich Pty Limited
187	V. & K. Phan
188	T. Mott
189	A. & S Kingston
190	F. & S. Hooke
191	Baker Place Investments Pty Limited
192	Pace Farm Pty Limited
193	Transport for NSW(*see Note 3)

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In further examining this data it should be noted that the 97.5th percentile incremental $PM_{2.5}$ concentration (24-hour average) is 5.3μg/m^3. This value was used in the HRA as a conservative measure to determine the potential risks to receptors in the area of the Proposal. In the case of the annual concentration the 97.5th percentile incremental $PM_{2.5}$ concentration (annual) is 0.51μg/m^3 and was used in the HRA to determine the potential risks to receptors in the area of the Proposal. Additionally, the exposures of the potentially sensitive receptors of the Gloucester High School and Hospital, as well as the Captain Cook Park were qualitatively examined (see Tables 9.10 & 9.11 from PEL, 2013).

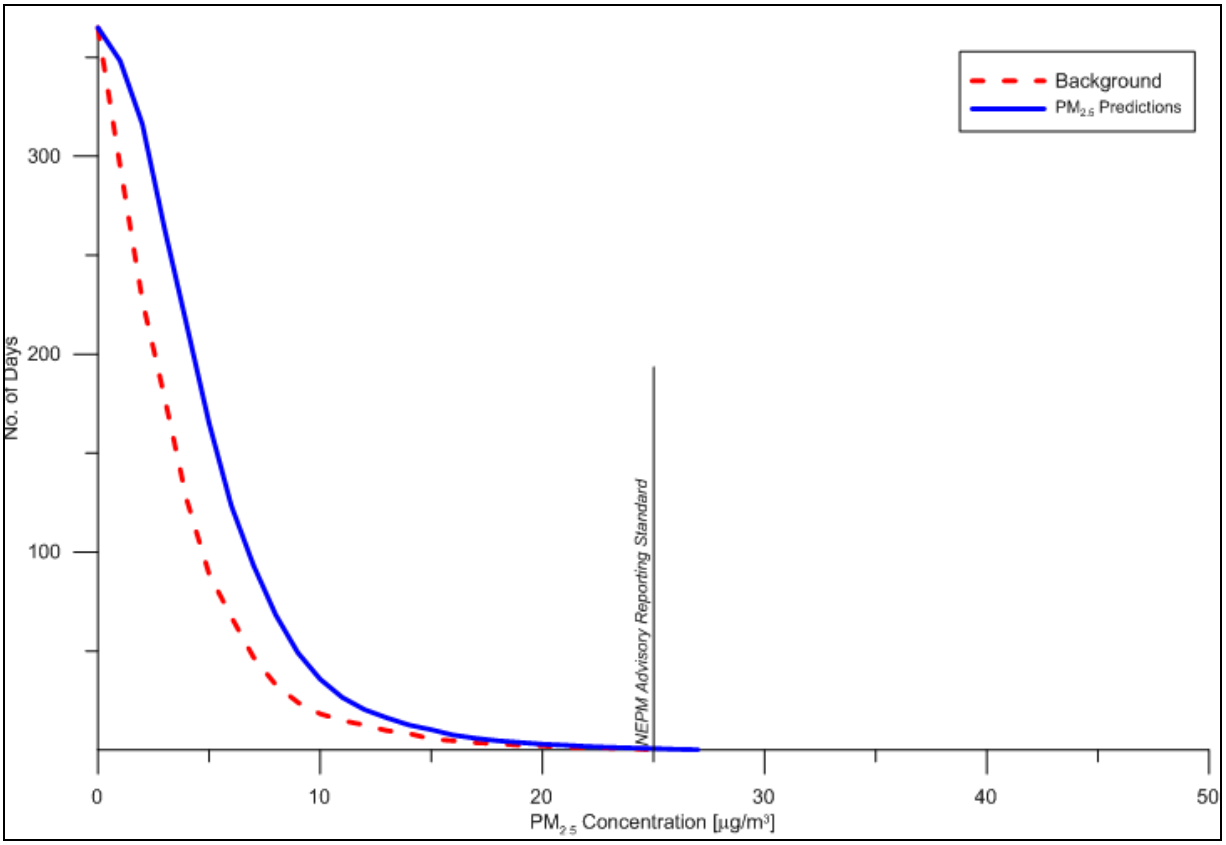

Figure 4.3 Frequency distribution of cumulative 24-hr $PM_{2.5}$ concentration using Monte Carlo Simulation

4.2 EXPOSURE TO NO_2

The air dispersion modelling for blasting conducted by PEL (2013) provided probability estimates for the GLCs at six selected residences that represented the highest impacts and spatial variety around the Site. Concentrations of NO_2 in the air from blasting are not constant; the concentrations are anticipated to vary according to the direction and strength of the wind, time of day, and how far away the location is from the blast.

The locations of the selected receptors are shown in **Figure 4.4.** Year 2.5 and Year 4.25 of the modelled years were chosen for blasting assessment as these years represent greatest impacts from blasting are expected.

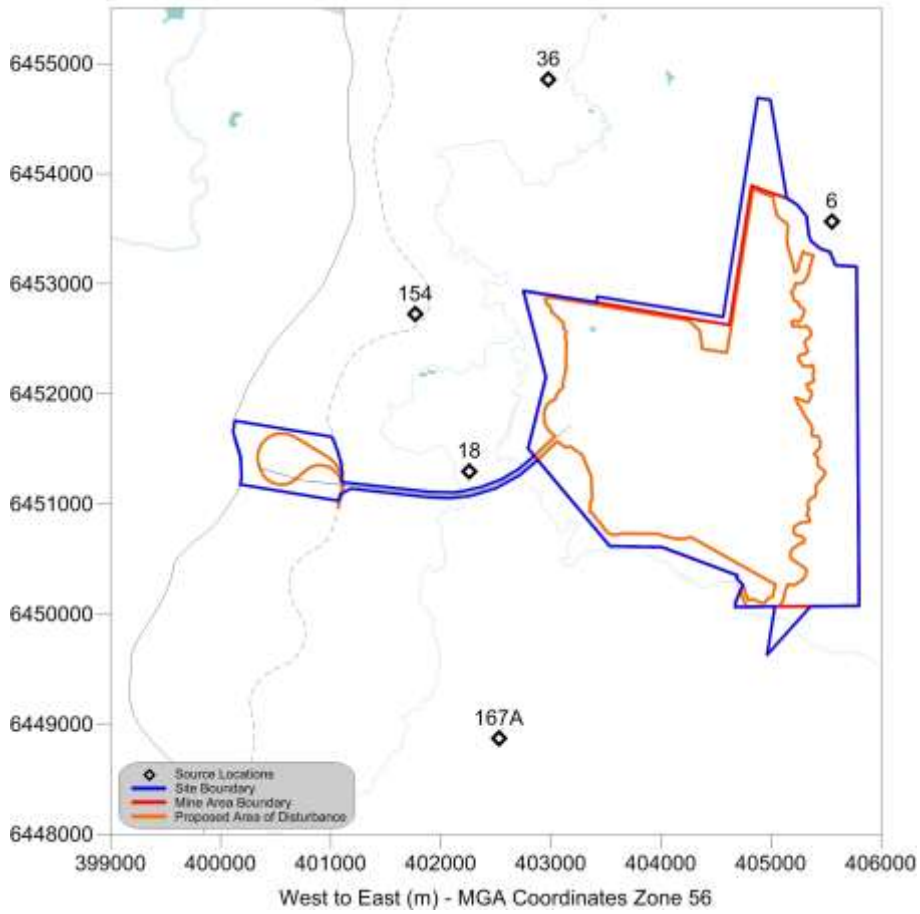

Figure 4.4 Location of receptors assessed for blast fumes

The modelling assumed a possible 2,920 hours per year when blasting could occur (8 hours a day, 365 days a year). In reality, blasting would only occur to a maximum of four events per week and a maximum of 100 per year, when meteorological conditions were favourable. Measures to minimise or avoid imperfect blasts and blasting in unfavourable conditions would be implemented and addressed in a Blast Management Plan.

In capturing a range of meteorological conditions, including unrealistic scenarios of blasting during unfavourable conditions, and assuming a worst case Level 4 fume category, this over-estimates the peak concentrations of NO_2 at the selected receptors. Thus, maximum peak concentrations have not be used for the characterisation of risk (as blasting would not occur in times of poor dispersion conditions). Instead, several percentile values at the most affected receptor have been used.

Percentiles can be pragmatically regarded as code for the number of times a concentration will occur during the year, or the number of times during the year a person is likely to be exposed to a certain concentration if they are at the same location at the same time the high concentration occurs. **Table 4.2** provides the key to the code.

Table 4.2
Frequency percentiles and approx. number of times they occur for a 1 hour averaging time.

	Frequency Percentile					
	95th	**99th**	**99.5th**	**99.7th**	**99.9th**	**Max**
Approx. number of times per year a 1 hour average concentration might occur at a given percentile.	438	88	44	27	9	1

This information shows how rapidly the concentration decreases from the maximum down to the 95th percentile. The number of times a percentile will occur has been rounded up to a whole number.

To factor a person's behaviour (i.e. average daily movements) into a risk assessment is quite challenging, and is rarely done. Instead, an assumption is made that throughout their entire life a person is in a situation where they could be exposed to the highest concentrations predicted to occur by the dispersion modelling. This assumption adds conservatism (i.e. safety) into the risk assessment. Based on this assumption, whether or not a person is affected by a pollutant compound in air requires them to be present at the location at the same time the high concentration occurs. However people do not spend all their time in one spot, for example an average adult only spends approximately 1.5 hours outdoors per day. Given that people also move around during the time they spend outdoors, the chance of being present in the unlikely event that NO_2 concentrations are elevated is therefore quite low.

Table 4.3 provides the GLCs for 1 hour average NO_2 at the worst affect receptor, Receptor 6. The 99.9th, 99.5th and 99th percentiles at the most affect receptor will be used in the risk calculations.

Table 4.3
Ground level concentration of 1 hour average NO_2 at various percentiles for Receptor 6

	Max	**99.9th**	**99.5th**	**99th**	**95th**
µg/m^3	1637	**155**	**114**	**103**	**66**
ppb	799	76	56	50	32

Given the extremely intermittent nature of the blasting activities, it was not possible to model a reliable annual average GLC for NO_2. Given the potential for adverse health effects in conjunction with long-term exposure to NO_2 as outlined in Section 3.3.7, a conservative annual average GLC of 4.1µg/m^3, based on the 10th percentile[2] of the 1 hour average has been used to estimate long-term exposures.

[2] PEL modelled a possible 2920 hours per year when blasting could occur to estimate a worst case hour. In reality, blasting activities are restricted to no more than 4 blasts per week. The maximum number of blasts on site will be 100 per year, as such the assumptions used both in the air quality assessment and the HRA are considered to be conservative, as the 10th percentile concentration is considered to overestimate the potential exposure and therefore risk.

5. RISK CHARACTERISATION

The NEPM considers an additional risk of '1 per 100,000' for adverse health outcomes to be sufficiently small and to be of no cause for concern. The recent enHealth update on environmental risk assessment has not prescribed a target risk level. Rather, it has noted that any target risk level does not:

"… *imply certainty that one person will get the disease if there are at least one million people exposed. It is simply a way of expressing risk, as a numerical expression of the likelihood of an event occurring under the defined conditions of exposure, based on extrapolation of dose–response data*."

As such, it is important to note that the risk values provided based on the CRFs for PM represent only probabilities, and not defined health outcomes.

In the case of NO_X, as noted previously, the study Streeton (1997) provides the most scientifically defensible information with respect to the potential for both acute and chronic effects in association with exposure to NO_2. As a result, the Australian NEPC Air Guideline Value AGVs, which are protective of health outcomes in sensitive subpopulations, are used to determine the potential for acute effects (246µg/m^3) and chronic effects (61.5µg/m^3) in association with exposure to NO_2 from the Proposal.

5.1 HEALTH RISKS DUE TO $PM_{2.5}$ EXPOSURE FROM THE PROPOSAL

The general approach used to calculate the risks to health has drawn upon internationally recognised estimates of the impact of $PM_{2.5}$ on health in relation to the known health indicators for NSW. It involves estimating the change in the incidence of a health outcome resulting from a given change in $PM_{2.5}$ concentrations. The RRs provide an estimate of the relationship between the health endpoint of interest and PM concentrations. As noted previously in Section 3.3.3 in relation to risks associated with exposure to PM, it should be stressed that no statistically significant mortality risk has been shown to be present based on PM_{10} or $PM_{2.5}$ exposure, however a correlation is evident in terms of an increased risk in relation to bsp.

The baseline health effect incidence rate provides the number of cases of the health effect per year, usually per 100,000 of the general population.

The risk factors in **Table 3.6** have been used to estimate the risks associated with exposure to the particulate emissions from the Proposal. Daily and annual mortality rates for the Hunter New England Local Health District for 2009-2010 and daily hospital admissions for all of NSW in 2006-2007 were obtained from the NSW Health website (http://www.healthstats.nsw.gov.au/ accessed September 2012).

An example of the calculation is shown below.

For a population size of 100,000 exposed to an increased 24-hour $PM_{2.5}$ concentration of 11μg/m^3 where the daily mortality is 1.74/100,000 people and the additional risk of death is 1.0026 (0.26%) per 1μg/m^3 increase in $PM_{2.5}$, the number of additional deaths per 100,000 would be:

Additional deaths per day = ((Base incidence x % increase) – Base incidence) x PM increase

= ((1.74 x 1.0009) – 1.74) x 11

= 0.02 per 100,000 or 2 per 100,000,000

Table 5.1 summarises the risks for the most exposed individual assuming a maximum daily incremental increase of 5.3μg/m^3 and an annual increase of 0.51μg/m^3.

Table 5.1
Risk calculations and estimated increase in risk for the worst case $PM_{2.5}$

Health outcome	Base (Background) Incidence (per 100,000)	Relative risk based on exposure to an increase of 1ug/m^3 of $PM_{2.5}$	$PM_{2.5}$ increase μg/m^3)	Increased risk (per 100,000)[a]
Annual mortality	658	1.0058	0.51	1.9
Daily mortality	1.80	1.0009	5.3	0.009
Daily hospital admissions for cardiovascular disease (all ages)	1.04	1.0016	5.3	0.009
Daily hospital admission respiratory (all ages)	4.67	1.0007	5.3	0.017
[a] Rounded-up				

In examining the increased risk in the population (based on annual mortality rates, all causes) due to the increased long-term exposure to $PM_{2.5}$ as a result of the Proposal, it is noted that it would increase the total from the base incidence of 658 to 660 per 100,000 or a resultant increase of 0.002%. This value is even smaller when you examine the case of daily exposures. In comparing the incremental increase in $PM_{2.5}$ concentrations due to the Proposal to the Air NEPM, the concentrations are well below the national ambient air quality standards of 25μg/m^3 (24-hour) and 8μg/m^3 (annual). In qualitatively evaluating the risks related to the incremental exposure to $PM_{2.5}$ at the Gloucester High School, Hospital and the Captain Cook Park, the concentrations based on a 24-hour average represent the 50th percentile of all data (0.7 - 0.9 μg/m^3 vs. 0.7 μg/m^3 for all receptors) and as such represent a negligible risk. Similarly, examining the annual average incremental concentration of $PM_{2.5}$ for these receptors is represented by approximately half of the 50th percentile (0.1 μg/m^3 vs. 0.23 μg/m^3 for all receptors) for all receptors and again represents a negligible risk.

In examining the context of these risk results, it should be noted that the dispersion modelling results are estimates only and the predicted annual average concentration for $PM_{2.5}$ represents a very low concentration and is subject to significant conservatism. The concentrations are unlikely to be higher than the reported annual average concentration and are more likely to be lower during operation of the Proposal. It is also important to note that $PM_{2.5}$ exposure alone does not result in mortality, but is one of many factors that can combine to contribute to a premature mortality in susceptible populations (e.g., elderly, persons with breathing problems – COPD, etc.).

The increased risks to the population (based on examining the full dataset – see Section 4.1) due to long-term exposure to $PM_{2.5}$ are considered to be negligible based on the 0.002% increase in the potential for an adverse health outcome at the closest potentially affected receptor. Additionally, the consideration of chronic (long-term) exposure and health impacts leads to uncertainty and over estimation, because the Pope *et al*. (2002) hazard rate of 6% per 10µg/m^3 $PM_{2.5}$ relates to a lifetime of PM exposure and the health impacts accrue over long periods of time, involving a mix of short- and long-term effects.

Shorter term exposures to $PM_{2.5}$ are also considered not to pose an unacceptable risk as the estimated risk is orders of magnitude lower than that due to long-term exposure.

Based on the foregoing, the weight of evidence notes that it is unlikely that an unacceptable risk due to $PM_{2.5}$ exposure will result from the Proposal.

5.2 HEALTH RISKS DUE TO NO_2

5.2.1 Introduction to hazard quotients and their interpretation

For assessing the potential acute and chronic health impacts of NO_2, predicted GLCs are compared to individual health-based ambient air guideline values (AGV) generated to protect public health. This comparison is performed by calculating a hazard quotient (HQ) which is the ratio of GLC to the ambient AGV (in this case NEPC Ambient Air NEPM of 246µg/m^3 (1-hour) and 61.5µg/m^3 (annual)).

The hazard quotient is calculated using the simple equation below.

HQ = Estimated ground concentration/Health based air guideline value …….**Equation 1**

An ‘unacceptable’ risk, as defined by regulatory standards and requirements, is often determined as the exposure being larger than the AGV used to calculate the hazard quotient, i.e. the HQ>1. This definition of unacceptable risk does not equate with imminent adverse health effects or even high risk of adverse health effects. It simply means that the health guideline level has been exceeded.

Notwithstanding their use in this risk assessment, HQs are relatively blunt tools used to assist in characterising and prioritising risks. Care must be taken as to the level of importance that is placed on the numerical value of the HQ. HQs should not be used in isolation of other pertinent data such as mechanistic information on the toxic mode of action and knowledge of the conservatism incorporated into the exposure assessment and the toxicity values.

The general rule of thumb for interpreting a HQ is that values less than 1 present no cause for concern; values greater than 1 but less than 10 generally also do not represent cause for concern because of the inherent conservatism embedded in the exposure portions of a risk assessment. However, it is usual to examine, and perhaps refine, the level of conservatism that has been assumed in the exposure assumptions. HQs that are around 10 present some concern regarding possible health risks, and in these circumstances it is usual to evaluate the extent to which the “safety margins” in the health guideline value used to compare estimated exposures may have been eroded in order to gauge whether concern is warranted. It is common that the risk assessment needs to be refined using site-specific exposure information or additional analytical data when HQs are greater than unity (greater than 1).

5.2.2 Risk calculations

Table 5.2 shows the acute HQs for the representative percentiles of predicted ground level concentrations of NO_2 from blast emissions at the worst affected receptor, Receptor 6. All the HQs are less than unity (less than 1) indicating acute direct health effects are very unlikely.

Table 5.2
Acute HQs for percentile ground level concentrations for Receptor 6

	99.9th	**99.5th**	**99th**	**95th**
GLC µg/m^3	155	114	103	66
AGV µg/m^3	246	246	246	246
HQ	**0.6**	**0.5**	**0.4**	**0.3**

In the case of the potential risks due to chronic exposure to NO_2, the resultant HQ is 0.07 based on a GLC of 4.1µg/m^3 and an annual AGV of 61.5µg/m^3 (0.02 ppm). As with acute exposures, the HQ is less than unity, and as such indicates that an adverse health effect due to exposure is very unlikely.

6. CONCLUSIONS

This health risk assessment has estimated the risks of an increased incidence of selected health outcomes due to increased exposures of $PM_{2.5}$ and NO_2.

The risk factors used in the analysis have been developed using research results from the last decade on the health effects of PM and NO_2 on human populations in urban areas.

The analysis provides estimates of the increase in annual and daily mortality due to emissions from the Proposal at the most affected receptor on the worst day. In addition, estimates are provided on the increase in daily hospital admissions that could be expected from the most exposed individual due to emissions from the Proposal on the worst day.

In examining the increased risk in the population (based on annual mortality rates, all causes) due to the increased long-term exposure to $PM_{2.5}$ as a result of the Proposal, it is noted that it would increase the total from the base incidence of 658 to 660 per 100,000 or a resultant increase of 0.002%. This value is even smaller when you examine the case of daily exposures. Additionally, in consideration of the ground level concentrations for $PM_{2.5}$ for all receptors, in comparing the concentration to the NEPM air quality guideline, the modelled concentrations (based on the 97.5th percentile) of 5.3µg/m^3 and 0.51µg/m^3 are well below the AGVs of 25µg/m^3 (24-hour) and 8µg/m^3 (annual), respectively.

In examining the full data set, it is noted that based on the air modelling over 90% of the time the cumulative $PM_{2.5}$ concentrations are less than 10µg/m^3, while approximately 65% of the time the cumulative concentrations are less than 5µg/m^3. **Table 4.1** outlined the separate contributions of background and those of the proposal based on various percentiles. It is noted that the background contribution represents 70 – 80% of the total cumulative concentrations of $PM_{2.5}$ (based on a 24-hour averaging period). Similarly, the background contribution represents approximately 90% of the total cumulative concentrations of the annual $PM_{2.5}$ concentrations. As such, the contribution of the emissions from the Proposal represents only a small proportion with respect to overall air quality in the area.

Additionally, it should be noted that the dispersion modelling results are estimates only and the predicted annual average concentration for $PM_{2.5}$ represents a very low concentration and is subject to significant uncertainty. The concentrations are unlikely to be higher than the reported annual average concentration and are more likely to be lower during operation of the Proposal. It is also important to note that $PM_{2.5}$ exposure alone does not result in mortality, but is one of many factors that can combine to contribute to a premature mortality in susceptible populations (e.g., elderly, persons with breathing problems – COPD, etc.).

Shorter term exposures to $PM_{2.5}$ are also not considered to pose an unacceptable risk as the estimated risk is orders of magnitude lower than that due to long-term exposure. Based on the foregoing, the weight of evidence notes that it is unlikely that an unacceptable risk due to $PM_{2.5}$ exposure will result from the Proposal.

Because all of HQs for blast NO_2 emissions for the worst affected receptor are all less than 1, it is very unlikely the blast emissions will cause direct acute and/or chronic health effects. This outcome is reinforced by the use of a Blast Management Plan, that will avoid the modelled NO_2 concentrations that were utilised in this assessment.

Overall, it is concluded that air emissions from the proposed Rocky Hill Coal Project present little likelihood of causing adverse health effects to exposed individuals privately-owned residences around the Site.

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