

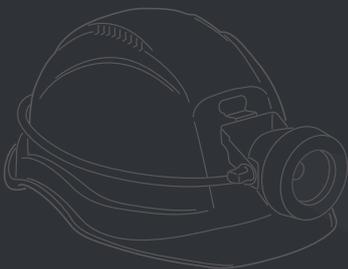
Wallarrah 2 Coal Project

Environmental Impact Statement

April 2013

Appendix M

Health Risk Assessment





REPORT

WALLARAH 2 COAL PROJECT – HEALTH RISK ASSESSMENT

Hansen Bailey Environmental Consultants

Job No: 6514A

7 December 2012



A PEL Company

PROJECT TITLE: WALLARAH 2 COAL PROJECT – HEALTH RISK ASSESSMENT

JOB NUMBER: 6514A

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DOCUMENT CONTROL

VERSION	DATE	PREPARED BY	REVIEWED BY
D1	14/08/2012	N. Aust	R. Kellaghan
D2	17/08/2012	N. Aust	R. Kellaghan
D3	29/08/2102	N. Aust	R. Kellaghan
Final	7/12/2012	N. Aust	R. Kellaghan

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1 INTRODUCTION

The Wyong Areas Coal Joint Venture (WACJV) seeks a Development Consent under Division 4.1 in Part 4 of the *Environmental Planning and Assessment Act 1979* (EP&A Act) for the Wallarah 2 Coal Project (the Project). This Health Risk Assessment (HRA) supports 'The Wallarah 2 Coal Project Environmental Impact Statement' (Walarah 2 EIS) prepared by Hansen Bailey Environmental Consultants to support the application.

This HRA has been prepared in accordance with the Director-General's Environmental Assessment Requirements (DGRs) for the Project issued 12 January 2012 in accordance with the requirements in Part 2 in Schedule 2 to the *Environmental Planning & Assessment Regulation 2000* (EP&A Regs).

Development Consent is sought to mine coal within the Extraction Area for a period of 28 years. The majority of this resource lies beneath the Wyong State Forest and surrounding ranges (including the Jiliby State Conservation Area (SCA)) while a proportion, to be extracted first, lies beneath a section of the Dooralong Valley and the Hue Hue area.

Key features of the Project include:

- The construction and operation of an underground mining operation extracting up to 5.0 Mtpa of export quality thermal coal by longwall methods at a depth of between 350 m and 690 m below the surface within the underground Extraction Area.
- Mining and related activities will occur 24 hours a day 7 days a week for a Project period of 28 years.
- Tooheys Road Site surface facilities on company owned and third party land (subject to a mining lease) between the Motorway Link Road and the F3 Freeway which will include (at least) a rail loop and spur, stockpiles, water and gas management facilities, workshop and offices.
- Buttonderry Site Surface Facilities on company owned land at Hue Hue Road between Sparks Road and the Wyong Shire Council's (WSC) Buttonderry Waste Management Facility. This facility will include (at least) the main personnel access to the mine, main ventilation facilities, offices and employee amenities.
- An inclined tunnel (or "drift") constructed from the coal seam beneath the Buttonderry Site to the surface at the Tooheys Road Site.
- Construction and use of various mining related infrastructure including water management structures, water treatment plant (reverse osmosis or similar), generator, second air intake ventilation shaft, boreholes, communications, water discharge point, powerlines, and easements to facilitate connection to the WSC (after July 2013, the Central Coast Water Corporation) water supply and sewerage system.
- Capture of methane for treatment initially involving flaring as practicable for greenhouse emission management and ultimately for beneficial use of methane such as electricity generation at the Tooheys Road Site.
- Transport of coal by rail to either the Newcastle port for export or to domestic power stations.
- A workforce of approximately 300 full-time company employees (plus an additional 30 contractors).
- Rehabilitation and closure of the site at cessation of mining operations.

1.1 Study Requirements

DGRs for the Project were issued 12 January 2012 in accordance with the requirements in Part 2 in Schedule 2 of the EP&A Regs.

Table 1.1 outlines the DGRs relevant to this HRA. Detailed agency comments have also been provided for inclusion within the Wallarah EIS and the agency comments relevant to this HRA are outlined in **Table 1.2**.

This HRA has been prepared in accordance with the DGRs and other relevant agency comments.

Table 1.1: Director-General's environmental assessment requirements

Discipline	Requirement	Section
Human Health	A detailed Human Health Risk Assessment addressing how the project's environmental impacts (particularly in relation to air quality, noise and drinking water quality) may impact on the health of the local community. The assessment should address both direct and indirect impacts, such as may result from additional rail and road movements;	Addressed throughout the report

Table 1.2: Agency Comments

Agency Comments	How comment is addressed
NSW EPA	
Assess the risk associated with potential discharges of fugitive and point source emissions for all stages of the proposal. Assessment of risk relates to environmental harm, risk to human health and amenity.	Addressed throughout the report
TRANSPORT FOR NSW - AIR	
Include a quantitative assessment of the potential air quality impacts of the project that explicitly includes consideration of both potential PM ₁₀ , PM _{2.5} and silica emissions of the project and measures to mitigate dust from loaded wagons.	Health risk from exposure to particulate matter and silica addressed in Section 3 of this HRA
NSW HEALTH CENTRAL COAST LOCAL HEALTH NETWORK	
<p>The Wallarah 2 Coal Project Background Document (October 2011) provides useful information that helped identify the main health impacts that may arise from the development. They relate primarily to air quality, drinking water, noise, greenhouse gases and road traffic.</p> <p>The proponent should provide a detailed Human Health Risk Assessment (HHRA) addressing how the proposed development will impact on these factors, and in turn, the health of the local community. It is anticipated that the EIS may use parts of the previous Environmental Assessment (EA), though, in many cases, there are more data available that need to be incorporated into the HHRA that may influence the outcomes. The HHRA should address the highlighted issues below, though not be limited to these issues.</p>	Addressed throughout the report
<p>The scientific evidence clearly demonstrates health effects of particulate pollution at levels below our current NSW guidelines. It is noted that an environmental indicator, dust deposition, has an absolute limit, and also has an incremental limit that should not be exceeded. At times, this can result in a dust deposition level below the absolute level being in excess of the incremental limit, and requiring investigation and action. Over the last several decades, there has been a trend of decreasing guideline levels for particulate air pollution.</p> <p>With this in mind, it is important that the air quality impact assessment addresses current guidelines and assesses the impact of any incremental increases in particulate air pollution. The HHRA should address both construction and operational stages.</p>	Addressed throughout the report
<p>As already noted, the Background Document provides greater detail to the proposal than was previously given in the EA from 2009. In addition, there are more air quality and meteorological data to be considered. There have also been changes to the local environment e.g. the new water pipeline and a DA for the Warnervale Town Centre.</p> <p>The implications of all this new information flows through to health impacts for air quality (increased truck movements increasing air pollution, more ambient air quality monitoring and meteorological data), water, noise, road traffic and greenhouse gases. They will require an EIS that is comprehensive, providing the relevant information in one document.</p>	Addressed throughout the report

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, 2002).

The development of 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 - Response Assessment** – Identifies hazards and health endpoints associated with exposure to hazardous agents and provides a review of the current understanding of the toxicity and risk relationship of the exposure of humans to the hazards.
2. **Exposure Assessment** – This task identifies the groups of people who may be exposed to hazardous agents and quantifies the exposure concentrations.
3. **Risk Characterisation** – This task provides the qualitative evaluation of potential risks to human health. The characterisation of risk is based on the review of concentration response relationship and the assessment of the magnitude of exposure.

3 AIR QUALITY HAZARD AND CONCENTRATION – RESPONSE ASSESSMENT

3.1 Health Endpoints

The adverse health effects resulting from exposure to ambient pollutant 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 disease.

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.

This assessment has focused on mortality and hospitalisation outcomes for receptor exposure to particulate matter (PM) and NO₂. For crystalline silica exposure, silicosis is the prime health outcome of interest.

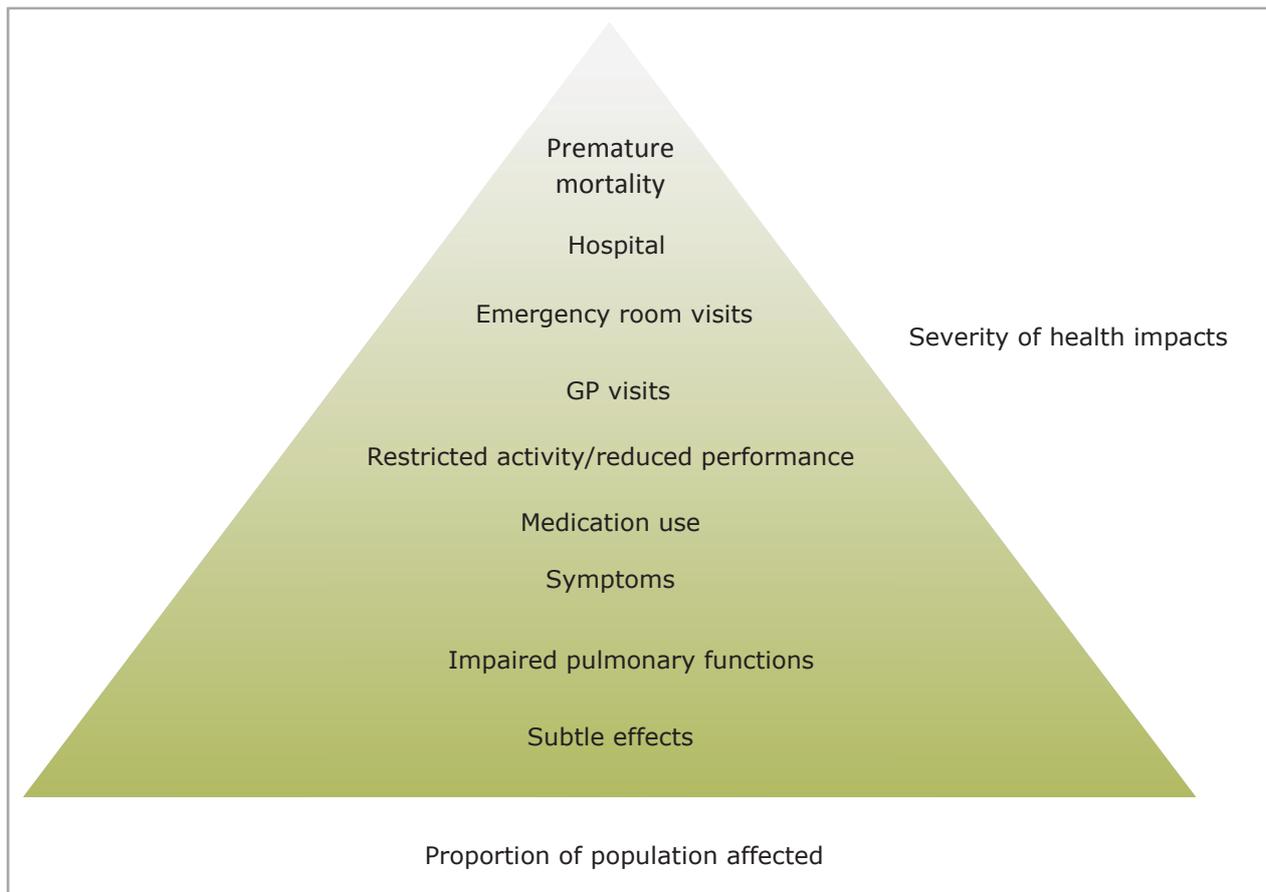


Figure 3-1: The severity and frequency of an adverse health impact as a result of ambient air pollution exposure (from WHO [2001])

3.2 Hazard Assessment

3.2.1 Particulate Matter

Particulate matter (PM) 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.

- PM₁₀ includes all inhalable particles less than 10 µm aerodynamic diameter that are sufficiently small to penetrate to the thoracic region. They are usually generated from solids by mechanical processes such as construction activities, road dust re-suspension and wind. Coarse particles consist of those between PM_{2.5} and PM₁₀ (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 that have a high probability of deposition in the smaller conducting airways and alveoli. These particles are usually generated through combustion processes.
- PM_{0.1} (ultrafine particles) includes those inhalable particles less than 0.1 µm in aerodynamic diameter that may activate irritant receptors and other mechanisms. These particles are usually generated by nucleation and condensation, largely from combustion emissions.
- PM₁₀ 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^a 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).

Fine particles or PM_{2.5} are 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 appeared 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 PM₁₀ and PM_{2.5} and the majority of the information comes from population-based epidemiological studies.

3.2.2 PM Health effects and concentration-response relationships

The results of epidemiological studies can be used to quantify 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 concentration-response relationship with PM and many health outcomes where the health risk increases with exposure to both PM₁₀ 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 of 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. 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. Similarly, multi-city studies can also produce more reliable effect estimates as the sample size is much larger than those in single city studies.

Over the last few decades, there has been a substantial amount of research that 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**), and ACS cohorts (**Pope et al. 1995**) and by results from several other independent studies of long-term PM exposure.

^a Crustal dust refers to dust generated from materials derived from the earth's crust.

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 evaluated 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. The particles are not the primary cause of death, 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**). However the current Australian air quality goals for particulate matter are still based on the more conservative associations.

3.2.3 Evidence of acute effects from short-term exposures

A recent review of exposures to PM 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 PM₁₀ and PM_{2.5} are summarised in **Table 3.1**.

Table 3.1: Short term effects on health from 10 µg/m³ increases in PM concentration

Health outcome	Estimated percentage increase in risk per 10 µg/m ³ PM ₁₀ (95% confidence interval)	Estimated percentage increase in risk per 10 µg/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 (O₃) (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 PM₁₀ and PM_{2.5} were analysed along with measurements of light scattering using nephelometry and reported as back-scattering pattern (bsp). **Table 3.2** and **Table 3.3** summarise the increase in daily mortality and daily hospital admissions in Australia arising from increased exposure to particulate matter. Bsp data were available for the four cities and PM₁₀ and PM_{2.5} data were available for three of the cities.

Table 3.2: Increases in daily mortality as a result of a 1 unit increase in 24-hour bsp (10-4m-1) or a 10 µg/m³ increase in 24-hour PM₁₀ 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 (10-4m-1) or a 10 µg/m³ increase in 24-hour PM₁₀ or PM_{2.5} (Simpson, 2005b)

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)

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 10 µg/m³ increase in PM_{2.5}.

The daily excess mortality for PM₁₀ is lower for the Australian data (0.2%) 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 PM₁₀, comparisons between admissions for respiratory disease in people aged 65+ show a higher level in the Australian data (2.9% increase) compared to 0.7% worldwide. Cardiovascular hospital admissions for PM_{2.5} are lower for US data (1.6%) than for Australia (5.1%).

3.2.4 Evidence of chronic effects from long-term exposures

Long-term exposure to PM is associated with mortality 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** below.

Table 3.4: Summary of percentage increase (and 95% CI) in relative risk of all cause and cause specific mortality associated with 10 µg/m³ 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 (4.2,23)
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 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 1.062 associated with a change in annual mean exposure of 10 µg/m³.

The UK report (**COMEAP, 2009**) recommends using a relative risk of 1.06 for all-cause mortality associated with a 10 µg/m³ increase in PM_{2.5}.

3.2.5 PM_{2.5} Concentration response functions used in this assessment

Many studies have used PM₁₀ 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 particulate air pollution for quantitative assessments of the associate 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 10 µg/m³ 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 et al. 2005a**).

Table 3.5: Health endpoints and concentration response function for 10 µg/m³ increase in PM_{2.5}

Health endpoint	Concentration response function for 10 µg/m ³ increase in PM _{2.5} Relative risk (95% CI)
Deaths	
Long-term deaths (age 30+ years) ^b	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^c	
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 Project are less than 10 µg/m³ (**PAEHolmes, 2012**) it is necessary to convert the relative risks (RR) above to RRs for the estimated increments from the Project. This accomplished by converting the RR from an increment of 10 µg/m³ 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%) is provided below.

- Convert the RR back to the beta-coefficient - Natural log (1.06) = 0.05827 for 10 µg/m³ change in PM_{2.5}.
- Convert the beta coefficient for a 1 µg/m³ 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 1 µg/m³ change in PM_{2.5} = exp (0.005827) = 1.005844.

Table 3.6 displays the resultant RRs for a 1 µg/m³ increase in PM_{2.5} used for risk estimation in this assessment.

Table 3.6: Health endpoints and concentration response function for 1 µg/m³ increase in PM_{2.5}

Health endpoint	Concentration response function for 1 µg/m ³ increase in PM _{2.5} - Relative risk
Deaths	
Long-term deaths (age 30+ years)	1.0058 (0.58%)
Short-term all non-trauma deaths (all ages)	1.0009 (0.09%)
Hospitalisations	
Cardiovascular disease (age 65+ years)	1.0016 (0.16%)
All respiratory disease (all ages)	1.0007 (0.07%)

^b Annual average PM_{2.5} used for long-term deaths

^c 24-h average PM_{2.5} and PM₁₀ used for hospitalisations

3.2.6 Respirable crystalline silica and health effects

Respirable crystalline silica (RCS) is emitted into the ambient air as a fractional component of particulate emissions or dust. Once entrained in the air, RCS may be inhaled and deposited in the lungs, where it could possibly cause disease.

Although human exposure to RCS occurs primarily in occupational environments, the general public can also be exposed to lower levels of RCS emitted from other sources such as sand blasting and entrained particles from surface soil.

Silica occurs in a crystalline or non-crystalline form and is often referred to as quartz. Epidemiological studies indicate that occupational exposure to RCS is associated with adverse health effects and general community (non-occupational) exposure to RCS is unlikely to present significant risks to public health. The World Health Organization's Concise International Chemical Assessment Document on Crystalline Silica, Quartz (**CICAD, 2000**) states that "*there are no known adverse health effects associated with the non-occupational exposure to quartz*".

As crystalline silica results in fibrogenic effects in the deeper areas of the respiratory system, it is the particles which are able to penetrate into the alveoli which are of prime concern (**NIOSH, 2002; US EPA, 1996**). It is generally considered that particles less than 2.5µm (PM_{2.5}) have a greater potential to reach the alveolar region (**US EPA, 2004 (a); IARC, 1997; CICAD, 2000**).

In the occupational setting chronic exposure to RCS is associated with increased incidences of tuberculosis, bronchitis, emphysema, chronic obstructive pulmonary disease, renal disease, silicosis and lung cancer. Of these potential health effects, silicosis and lung cancer are the effects of most concern associated with occupational exposure to RCS. Silicosis is considered to be the most sensitive health end point for which health risks from exposure to RCS should be assessed. The prevention of silicosis is considered to provide protection against the other possible health effects, including lung cancer, that may be associated with exposure to high levels of airborne crystalline silica (**HSE, 2002; OEHHA, 2005; CICAD, 2000; US EPA, 1996**).

Both toxicological and epidemiological data indicate there are levels of exposure of RCS below which there is zero risk of developing silicosis and lung cancer. Crystalline silica toxicity has been extensively investigated leading to a widely accepted toxicological mechanism involving chronic inflammation and oxidative stress. These toxicological mechanisms are consistent with a threshold exposure for both silicosis and lung cancer (**HSE, 2003**).

Silicosis and lung cancer are distinct disease conditions involving different cell types, but both stem from a common background of chronic inflammatory lung damage. Evidence implies that exposures to RCS insufficient to cause silicosis would be unlikely to lead to a significant increase in the risk of lung cancer over and above background levels, however this is not definitive (**CICAD, 2000; HSE, 2003**).

Evidence from occupational studies indicates that the severity and incidence of silicosis and silica-induced lung cancer increases with the intensity of dust exposure; increased cumulative duration of exposure; increasing peak concentrations of silica; and the increased percentage of silica within the respirable dust.

Early symptoms of silicosis are the first observable chronic health effects from inhalation of crystalline silica and silicosis is also regarded as the critical effect for hazard identification and exposure response assessment for crystalline silica (**CICAD, 2000; HSE, 2004; USEPA, 1996**).

In the occupational setting, acute exposure to very high peak concentrations of crystalline silica is also associated with silicosis (**EPA, 1996; NIOSH, 2002**).

However, in the absence of such high exposures, silicosis can result from longer term exposure to relatively low concentrations and the consequent accumulation of crystalline silica in the lungs with exposure is estimated as being cumulative over a number of years (for example $\mu\text{g}/\text{m}^3$ X number of years exposed) (**USEPA, 1996; HSE, 2002**). Therefore, for the purpose of estimating risks to sensitive receptors for exposures to low ambient concentrations of crystalline silica it is suitable to use modelling forecasts for annual concentrations rather than 24-hour estimates.

As such, in this assessment, we have used silicosis as the main health outcomes for annual average exposure to crystalline silica.

3.2.7 US EPA silicosis potency estimates

The US EPA (1996) has examined the non-cancer epidemiological literature on silica induced diseases and concluded that several studies of miners provide good quality data for risk assessment. The US EPA concluded that a thorough analysis of the most extensive occupational studies available, each of which examined the medical histories of thousands of miners, suggests that the cumulative risk of silicosis at or below $1,000 \mu\text{g}/\text{m}^3$.years (crystalline silica) is close to 0% (**US EPA, 1996**).

Cumulative exposure is an estimate of the average respirable crystalline silica concentration to which a person is exposed over the course of a year multiplied by the number of years exposed, using an assumed lifetime of 70 years.

This dose metric is to be interpreted as meaning a total exposure of $1,000 \mu\text{g}/\text{m}^3$ accumulated over a lifetime of 70 years, or $14.3 \mu\text{g}/\text{m}^3$ per year for 70 years ($1,000/70 = \sim 14.3$). If the total exposure of $1,000 \mu\text{g}/\text{m}^3$ was accumulated over 28 years (time estimate for life of the project) the risk of silicosis is the same, i.e. close to zero.

To characterise risks of silicosis associated with the silica content of dust exposure, in the vicinity of the Project, this risk assessment chooses the US EPA (**1996**) cumulative exposure estimate for zero risk of silicosis, i.e. $1,000 \mu\text{g}/\text{m}^3$ X years exposed respirable crystalline silica.

3.2.8 Comparison with the Office of Environmental Health Hazard Assessment Air Standard

In California, the Office of Environmental Health Hazard Assessment (OEHHA) has adopted a chronic Reference Exposure Level (REL) of $3 \mu\text{g}/\text{m}^3$ for RCS. A chronic REL is an airborne level of a substance at or below which no adverse health effects are anticipated in individuals indefinitely exposed to that level. RELs are developed from the best available published scientific data, based solely on health considerations.

While OEHHA does not promulgate environmental regulations directly, it is responsible for developing and providing risk managers in US state and local government agencies with toxicological and medical information relevant to decisions involving public health. Victoria has adopted the OEHHA REL, but measures the crystalline silica concentration in the $\text{PM}_{2.5}$ fraction of the dust.

3.2.9 Oxides of Nitrogen and Health Effects

The key pollutant released from flaring of methane or the use of gas engines will be oxides of nitrogen (NO_x). NO_x is comprised of nitric oxide (NO) and nitrogen dioxide (NO₂), however NO is not generally considered harmful to human health and not considered an air pollutant at the concentrations that are typically found in ambient environments.

In recent years the health effects of NO₂ linked to ambient exposures have been well studied and reviewed by international agencies (**USEPA, 2008a; WHO, 2006**). 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 NO₂ is limited.

There can be a high correlation between NO₂ levels and airborne particles, both are generated from the same combustion sources, and NO₂ is converted to nitrates and contributes to fine particle mass. Thus it is very difficult to differentiate the effects of NO₂ from those of other pollutants in epidemiological studies (**WHO, 2006**).

Studies of the health impacts of NO₂ include experimental studies on animals, controlled laboratory studies on humans and observational studies which demonstrated associations between NO₂ concentrations and daily mortality from respiratory and cardiovascular causes and with hospital admissions for respiratory conditions.

Nitric oxide is considerably less toxic to humans than nitrogen dioxide, but depending on conditions, in particular the presence of oxidizing agents such as ozone which is commonly found in urban air, oxidation from NO to NO₂ takes place in a matter of minutes. For example, WHO cite a figure of 50% conversion of NO to NO₂ in one minute in the presence of 0.1 ppm ozone.

Controlled human exposure studies have presented mixed results (**WHO, 2000**), however normal healthy people exposed at rest or with light exercise for less than 2 hours to concentrations of more than 4700 µg/m³ experience pronounced decrements in pulmonary function; generally, such people are not affected at concentrations less than 1880 µg/m³.

3.2.10 Evidence of acute effects from short-term exposure

A meta-analysis of 109 time-series studies of air pollution and mortality from around the world reported consistent associations with NO₂ (**Stieb et al. 2002**). A 2.8% (95% CI, 2.1-3.5%) increase in all-cause mortality was reported for 24.0 parts per billion (ppb) increase in NO₂, in a single-pollutant model.

In a meta-analysis of results from five Australian and two New Zealand cities Barnett et al. (**2005**) analysed hospital admissions for three age groups of children. Significant increases in hospital admissions for respiratory disease (1-4, 5-14 years) and asthma (5-14 years) were associated with interquartile range increases in either 1-hr or 24-hr NO₂. The largest association reported was a 6.0% increase in asthma admissions with a 5.1 ppb increase in 24-hr NO₂. The effect was not reduced by inclusion of PM₁₀ in the analysis.

3.2.11 Evidence of chronic effects from long-term exposures

Results of cohort studies in the United States and Europe examining the relationship between long-term exposure to NO₂ and mortality have been inconsistent. Further, when associations were suggested, they were not specific to NO₂ but also implicated particles and other traffic indicators.

In a recent review, **WHO (2006)** stated that it was not exposure to NO₂ associated with chronic effects, but rather particulate pollution especially from traffic sources, seemed to be responsible for the observed associations. In the most recent follow-up of the ACS cohort, no association between urban background concentrations of NO₂ and all-cause mortality was observed.

3.2.12 Nitrogen dioxide concentration response functions

Table 3.7 provides the health endpoints and CRF used in this study for estimation of impacts to health from exposure to NO₂. Original CRF were transformed to a 1 ppb increase in NO₂ concentrations using the same method for PM_{2.5} in **Section 3.2.5**.

Table 3.7: Health endpoints and concentration response function for increases in NO₂

Health endpoint	Concentration response function for 1 ppb increases in NO ₂
Deaths	
Short-term all non-trauma deaths (all ages)	1.001 (Stieb et al., 2002)
Hospitalisations	
Asthma admissions (5 – 14 years)	1.011 (Barnet et al. 2005)

3.3 Exposure Assessment

3.3.1 Exposure to PM_{2.5}

The air quality impact assessment for the Project (**PAEHolmes, 2012**) presents the dispersion modelling predictions direct from the Project for maximum 24-hour and annual average PM_{2.5} ground level concentrations (glcs) at assessment locations in the vicinity of the Project. Dust emissions associated with train loading have been included as part of the modelling assessment of mining operations. The limited additional employee road movements were deemed negligible in terms of air quality impacts, especially when compared to F3 Freeway traffic volumes.

The highest predictions were made at an assessment location to the north of the Tooheys Road Site P11 as shown on **Figure 3-2**, with a predicted incremental increase in 24-hour PM_{2.5} concentration of 5.0 µg/m³ and a predicted incremental increase in annual average PM_{2.5} concentration of 0.3 µg/m³. These concentrations are used in the risk calculations.

Additionally, the Project will result in an increase in rail movements from Wallarah to Newcastle potentially increasing fugitive coal dust emissions along the rail corridor. However, predicted PM concentration increases were not included as part of the assessment scope. Queensland Rail recently commissioned a study (QR Study) into fugitive coal dust emission and management along selected Queensland coal rail systems. A number of different approaches were used in the QR Study for the quantification and assessment of coal dust emissions from wagons. Key components of the QR Study were:

- Literature Review.
- Ambient Air Quality Monitoring (TSP, PM₁₀ and PM_{2.5} in residential areas and within the rail corridors across a number of locations throughout Central Queensland).
- Material characteristics wind tunnel testing.
- Dispersion modelling.
- Wagon and load profiling wind tunnel testing and CFD analysis.

The QR study concluded there is a low risk of health impacts from coal dust, either within or outside the rail corridor (**Connell Hatch, 2008**).

3.3.2 Nitrogen Dioxide

A worst case assessment of NO₂ emissions from the capture and use of methane (flaring and / or power generation) was presented in the air quality assessment (**PAEHolmes, 2012**). Predicted glcs of NO₂ were made based on the conservative assumption of 100% transformation of NO_x to NO₂. In reality, conversion is more likely to be 10%-20% for shorter averaging periods and the predicted glcs are conservatively high. Emissions from the existing road network, including the freeway would contribute to ambient levels of NO_x in the local area.

This assumption is retained for this HRA and which results in over-estimates of the risks to health from exposure to NO₂.

The most affected assessment location, P6 (as shown on **Figure 3-2**) has a predicted 1-hour NO_x concentration of 35 µg/m³ (18.6 ppb) and is used in the risk calculations.

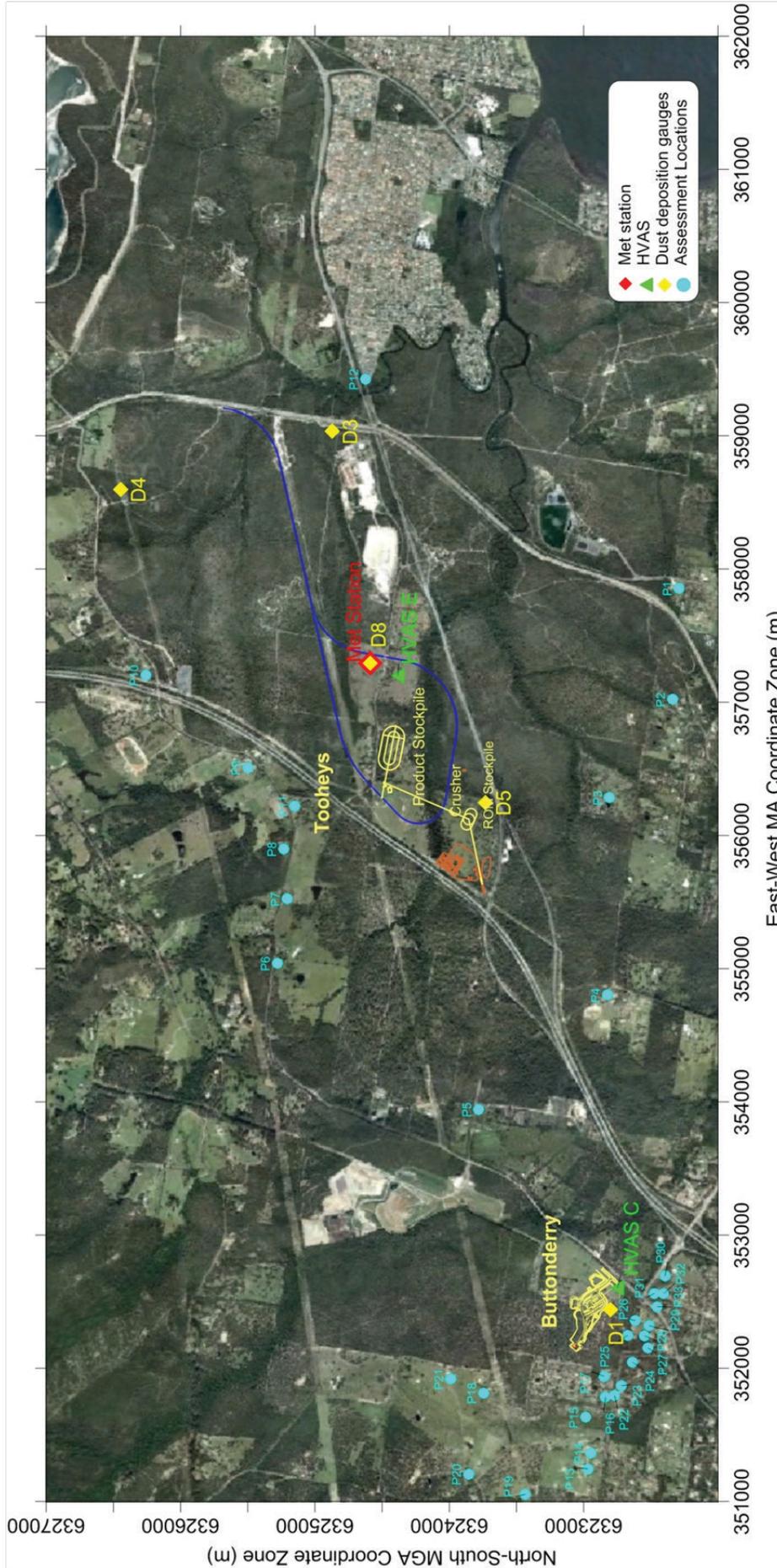


Figure 3-2: Local Setting, Relevant Receptor Locations and Monitoring Sites

3.3.3 Exposure to respirable crystalline silica

Exposure to RCS is assessed using the estimated total annual concentration, which is combination of existing background and increased concentrations due to the Project. Existing background concentrations of airborne crystalline silica are a result of quartz being emitted into the air as a component of particulate emissions produced by natural, industrial, and farming activities (**US EPA, 1996**). These activities include dust from travel on paved and unpaved roads, agricultural activities, bushfires, wind erosion, local construction and demolition and extractive industries (**US EPA, 1996; IARC, 1997**). Coal is composed primarily of carbon along with variable quantities of other elements, chiefly hydrogen, sulfur, oxygen, and nitrogen. It may contain variable but amounts of other mineral matter, of which silica is thought to be a minor component.

In underground mining, silica dust is created from several sources which generally include cutting, drilling, or milling of rock material. For coal mining this rock material may be found as an inherent constituent of the coal, although this is usually not a significant source of silica. The majority of the silica is found in the immediate overlaying or underlaying rock strata which are frequently removed during the open cut coal mining process but generally avoided during coal extraction in underground mines. Also, significant rock inclusions known as partings can sometimes occur within the coal seam itself and these are often removed with the coal. This is not the case in regard to the coal seam properties at Wallarah 2 Coal Project and also because only a portion of the coal seam will be extracted.

The crystalline silica component of ambient emissions has been observed to be higher within larger particle size fractions ($>10\mu\text{m}$) than those fractions less than $10\mu\text{m}$. It is suggested that this unequal distribution may be due to quartz, which is harder than most minerals, resisting reduction in size to finer particle sizes (**US EPA, 1996**). Background ambient respirable silica is not measured in area of the Project. As such, background concentrations are estimated based on existing studies both internationally and in Australia. Data collected in Victoria estimated the background concentration to be $0.7 \mu\text{g}/\text{m}^3$ (**Toxikos, 2005**). This is comparable to the ambient level in a Californian rural area of $0.6 \mu\text{g}/\text{m}^3$ (**OEHHA, 2005**). In the absence of any local data, a background level of $0.7 \mu\text{g}/\text{m}^3$ has been assumed for the Project Boundary.

In a report to the Monterey Bay Unified Air Pollution Control District of California, Goldsmith (**1991**) reported the PM_{10} and crystalline silica levels measured at two sites adjacent to a quarry in central coastal California. The mean PM_{10} concentrations for each site were 18.9 and $18.2 \mu\text{g}/\text{m}^3$, with mean crystalline silica concentrations of 1.33 and $1.10 \mu\text{g}/\text{m}^3$, respectively. The data show that 6 to 7% of the site-specific PM_{10} was crystalline silica. In this assessment we have used a proportion of 10% RCS in annual PM_{10} . The background annual average PM_{10} was reported by PAEHolmes is $18 \mu\text{g}/\text{m}^3$. The highest predicted incremental increase of PM_{10} as a result of the Project is $2.1 \mu\text{g}/\text{m}^3$, and therefore the increase in RCS is $0.16 \mu\text{g}/\text{m}^3$ ($1.6 \mu\text{g}/\text{m}^3 \times 10\% = 0.21 \mu\text{g}/\text{m}^3$). The cumulative exposure of crystalline silica for the worst affected receptor is presented in **Table 3.8**.

Table 3.8: Respirable crystalline silica concentrations (background and incremental increase)

	PM_{10}	%	RCS
Background	$18 \mu\text{g}/\text{m}^3$	10	$1.8 \mu\text{g}/\text{m}^3$
Incremental increase	$1.6 \mu\text{g}/\text{m}^3$	10	$0.16 \mu\text{g}/\text{m}^3$
Cumulative total	$19.6 \mu\text{g}/\text{m}^3$	10	$1.96 \mu\text{g}/\text{m}^3$

3.4 Risk Characterisation

The National Environment Protection Measures (NEPMs) consider an additional risk of '1 per 100,000' for adverse health outcomes to be sufficiently small and to be of no cause for concern. This is likely to be reflected in the upcoming enHealth criteria. The WHO has used a lifetime excess cancer risk of '1 per 100,000' as the basis for the derivation WHO drinking water guidelines and in New Zealand a risk level of '1 per 100,000' is also used for Contaminated Land Management. This risk reference is used for the characterisation of risk from exposure to PM_{2.5} and NO₂ from the Project.

The risk of health effects due to exposure to respirable crystalline silica has been evaluated using the US EPA (1996) estimates of crystalline silica's potency for zero risk of silicosis and by comparison with the chronic ambient air guideline standard from the Californian Office of Environmental Health Hazard Assessment.

3.4.1 Health risks due to PM_{2.5} exposure from the Project

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 relation between the health endpoint of interest and PM concentrations.

The baseline health effect incidence rate provides a number of cases of the health effect per year, usually per 100,000 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 Project. Daily and annual mortality rates for the Central Coast 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 June 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 5.0 µg/m³ where the daily mortality is 1.74/100,000 people and the additional risk of death is 1.0026 (0.26%) per 1 µg/m³ increase in PM_{2.5}, the number of additional deaths per 100,000 would be:

$$\begin{aligned}
 \text{Additional deaths per day} &= ((\text{Base incidence} \times \% \text{ increase}) - \text{Base incidence}) \times \text{PM increase} \\
 &= ((1.74 \times 1.0009) - 1.74) \times 5.0 \\
 &= 0.008 \text{ per } 100,000
 \end{aligned}$$

Table 3.9 summarises the risks for the most exposed individual assuming a daily incremental increase of 5.0 µg/m³ and an annual increase of 0.3 µg/m³.

Table 3.9: Estimated increase in risk of indicated event for the worst case PM_{2.5}

Health outcome	Base Incidence (per 100,000)	Relative risk	PM _{2.5} increase (µg/m ³)	Increased risk (per 100,000)
Annual mortality	635	1.0058	0.3	1.1
Daily mortality	1.74	1.0009	5.0	0.008
Daily hospital admissions for cardiovascular disease (all ages)	1.04	1.0016	5.0	0.008
Daily hospital admission respiratory (all ages)	4.67	1.0007	5.0	0.016

3.4.2 Health risks due to respirable crystalline silica

3.4.2.1 Using US EPA silicosis potency estimates

The US EPA (**1996**) has examined the non-cancer epidemiological literature on silica induced diseases and concluded that several studies of miners provide good quality data for risk assessment. The US EPA concluded that a thorough analysis of the most extensive occupational studies available, each of which examined the medical histories of thousands of miners, suggests that the cumulative risk of silicosis at or below 1,000 µg/m³.years (crystalline silica) is close to 0% (**US EPA, 1996**).

Cumulative exposure is an estimate of the average respirable crystalline silica concentration to which a person is exposed over the course of a year multiplied by the number of years exposed, using an assumed lifetime of 70 years.

This dose metric is to be interpreted as meaning a total exposure of 1,000 µg/m³ accumulated over a lifetime of 70 years, or 14.3 µg/m³ per year for 70 years. If the total exposure of 1,000µg/m³ x years was accumulated over 28 years (time estimate for life of the Project) the risk of silicosis is the same, i.e. close to zero.

Calculations:

- Background respirable crystalline silica is assumed to be 1.8 µg/m³ (10% of annual average PM₁₀ of 18 µg/m³).
- Maximum (annual average) incremental increase of respirable crystalline silica at the most affected receptor is estimated to be 0.16 µg/m³ (10% of annual average incremental increase PM₁₀ of 1.6 µg/m³).
- Total annual respirable crystalline silica concentration is 1.96 µg/m³.
- If it is assumed the Project has an operating life of 28 years then the cumulative silica exposure is 1.96 µg/m³ x 28 years = 54.9 µg/m³.years.
- If an individual lived for the remaining 42 years of their 70 year lifetime in the same area and was exposed to background levels of respirable crystalline silica the cumulative exposure would be 1.6 µg/m³ x 42 years = 82.3 µg/m³.years.
- Total lifetime exposure to respirable crystalline silica from background and the proposed Project is therefore 82.3 µg/m³.yrs + 54.9 µg/m³.years = 137.2 µg/m³.years.

The estimated total lifetime cumulative exposure is approximately 7 times lower than the cumulative exposure of 1,000 $\mu\text{g}/\text{m}^3$.years associated with close to zero risk of silicosis in workers.

3.4.2.2 Comparison with the Office of Environmental Health Hazard Assessment air standard

The total cumulative RCS annual concentration of 1.96 $\mu\text{g}/\text{m}^3$ is considerably less than the REL of 3 $\mu\text{g}/\text{m}^3$ set by the OEHHA and adopted by the Victorian Environmental Protection Agency.

3.4.3 Health risks due to nitrogen dioxide

The approach used to calculate the risks to health from NO_2 emissions has used the same method for estimating risks from $\text{PM}_{2.5}$ on health in **Section 3.4.1**.

The equation used is as follows.

Additional deaths per day = ((Base incidence x % increase) – Base incidence) x NO_2 increase.

Table 3.10 summarises the risks for the most exposed individual assuming an incremental increase of 18.6 ppb.

Table 3.10: Estimated increase in risk of indicated event for the worst case NO_2 exposure scenarios

Health outcome	Base Incidence (per 100,000)	Relative risk	NO_2 increase (ppb)	Increased risk (per 100,000)
Daily mortality	1.74	1.001	18.6	0.05
Daily hospital admissions for asthma (5-14 years)	0.35	1.011	18.6	0.07

Baseline incidence rates were obtained from the NSW Health website (<http://www.healthstats.nsw.gov.au/> Accessed June 2012).

3.5 Summary of Air Quality Risk Assessment

In addition to the traditional impact assessment criteria, this health risk assessment has estimated risks of increased incidence of selected health outcomes due to increased exposures of PM_{2.5}, respirable crystalline silica and NO₂.

The risk factors used in the analysis have been developed using research results from the last decade on the health effects of particulate matter and nitrogen dioxide on human populations in urban areas. Risks of exposure to silica have also been considered.

The analysis provides conservative estimates of the increase in annual and daily mortality due to emissions from the Project 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 Project on the worst day.

The increase in risk of daily mortality on the worst day in the life of the Project is estimated to be approximately 1 in 100,000. All other health outcomes risks are less than 1 in 100,000. This is a small risk.

In regards to risks from rail movements, the QR Study concluded that there appears to be minimal risk of adverse human health and amenity impacts due to fugitive coal emissions from trains, based on results of monitoring and modelling predictions. To ensure fugitive dust emissions are kept to a minimum during the relatively short journey to port, the Project proponents will commit to water spraying the coal surface during train loading. In summary, the rail load out facility will be designed such that:

- Surface spraying of product coal for transportation.
- Load size is limited to ensure coal is below wagon sidewalls.
- Loading is such that a consistent profile is maintained.

Risk of silicosis as a result of operations of the Project is negligible. This conclusion is agreeable with the statement made by the World Health Organisation that *"to date, there are no known adverse health effects associated with non-occupational exposure to quartz dust"* (CICAD, 2000).

4 NOISE

The following section is a qualitative assessment of the potential health impacts from exposure to noise. This assessment does not quantitatively estimate the burden of disease on the local community, but draws on the current literature to discuss the potential risks to health of those exposed to environmental noise.

Firstly, there is an overview of health effects and noise, followed by a discussion of potential health impacts based on the conclusions and recommendations provided in the report “*Walarah No.2 Coal Project Noise and Vibration Impact Assessment*” (**Atkins Acoustics, 2012**) which assessed the predicted noise and vibration levels at sensitive receivers generated by Project related activities.

4.1 Overview of environmental noise and health impacts

There is now sufficient evidence internationally that environmental noise may pose a general public health risk. Groups most exposed to this noise (by virtue of where they live, work and recreate) and those most sensitive to its impact, may face even greater risks. They include infants and school children, shift workers, the elderly, the blind, and those suffering hearing impairment, sleep disorders, and physical and mental health conditions. Australian surveys have found respondents were concerned about environmental noise from a wide range of transportation and other sources, as well as noise generated by neighbours’ loud voices, loud appliances and pets (indoors and outdoors).

According to WHO (1946), health is “*a state of complete physical, mental and social wellbeing and merely the absence of disease or infirmity*”. The WHO Community Noise Guidelines (**Berglund et al 1999**) note the following effects of noise and provides comprehensive summaries of each of:

- Noise-induced hearing loss.
- Interference with speech communication.
- Sleep disturbance.
- Cardiovascular and physiological effects.
- Mental health effects.
- The effects of noise on performance.
- The effects of noise on residential behaviour and annoyance.

The complexity of the effects of noise exposure on humans is illustrated in Babisch’s (**2002**) noise effects reaction scheme shown in **Figure 4-1**.

Figure 4-1 illustrates that noise can effect humans directly through a physiological pathway causing hearing loss, and indirectly through disturbance to normal human activities such as sleep, communication or concentration, and by generating an emotional response (i.e. annoyance). The combination of these effects can elevate the stress of the individual and this, along with other stressors and risk factors for the individual, may manifest in other disorders, for example cardiovascular diseases.

All aspects of Babisch’s diagram have been the focus of research over many decades, and this is continuing, with more emphasis on large epidemiological studies that are necessary to test for links between noise and objective measures of human health.

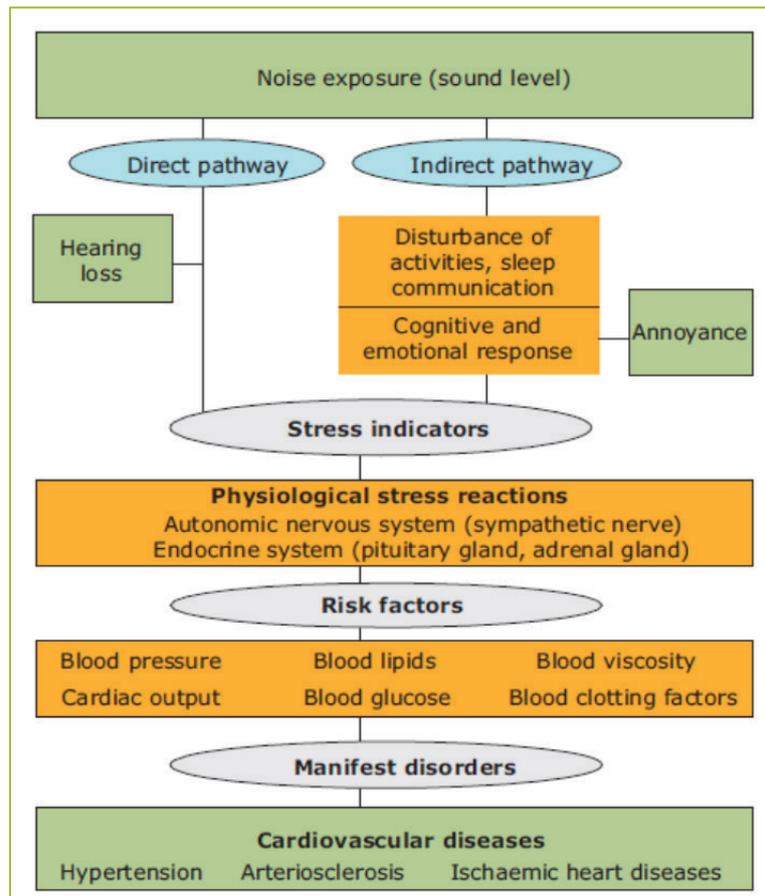


Figure 4-1: Noise stress relationship

The severity of the health outcome, its prevalence in the general population, the frequency of exposure considered relevant for health, and the magnitude of effect are important issues in risk assessment (Neus H. and Biokat U., 2000). The term "adverse" is essential in this context of environmental standard setting.

Risk management should ensure that "adverse" health effects do not occur. Decisions on whether or not any effect is adverse, requires expert judgement. The WHO defines an "adverse effect" as follows (WHO, 1994): "Change in morphology, physiology, growth, development or life span of an organism, which results in impairment of the functional capacity to compensate for additional stress, or increase in susceptibility to the harmful effect of other environmental influences". It is obvious that the relevance of a noise effect increases with increasing severity and the high prevalence of the considered health outcome. Ischemic heart diseases are one of the major causes of premature death in modern societies (Doll R., 1992; WHO Regional Office Europe, 1999). For this reason cardiovascular disorders including hypertension and myocardial infarction have been in the primary focus of recent epidemiological noise research.

The general stress theory is the rationale for the hypothesis that noise affects the autonomic nervous system and the endocrine system, which in turn affects the homeostasis of the human organism. In regards to the noise/stress concept, researchers have looked at endogenous response variables which are related to the natural stress mechanism, however, these indicators may not have pathological significance. Furthermore, due to improvements of measuring techniques, thresholds tend to decline to levels without clinical relevance. It is sometimes suggested to refer to exceedances of "normal values" of physiological factors as a criterion of effect. However, even such exceedances are not necessarily associated with an increased risk. Babisch (2002) argues that all responses to noise are not necessarily adverse.

Figure 4-2, adapted from Babisch (2002), displays the severity of effects of noise. The first outcome is often used along with weighting factors to develop environmental objectives for noise sources (NSW EPA, 1999). Effects regarding the top three outcomes of the triangle may be attributed "adverse" according to the WHO (1994) criteria.

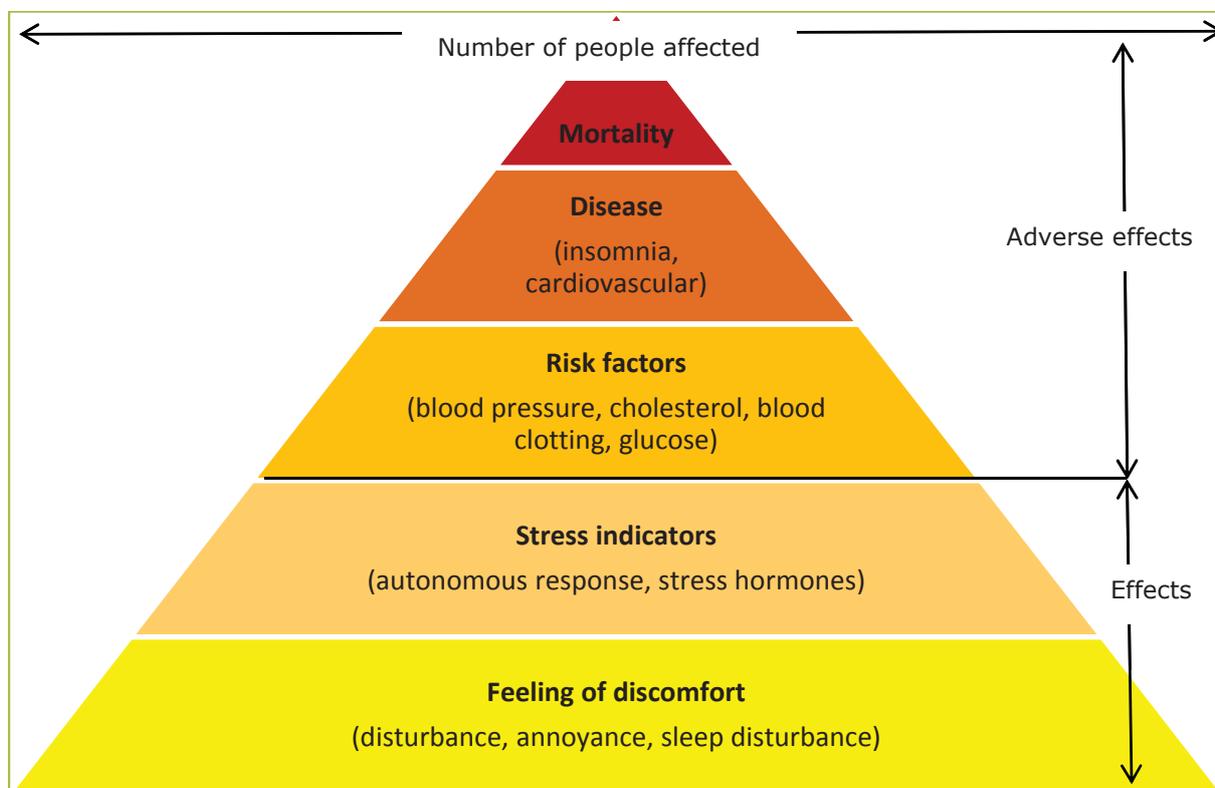


Figure 4-2: Severity of noise effects

Source: Babisch, W. 2002

The strength of current scientific evidence across the different components of human reaction to noise is variable, but there is sufficient evidence now with respect to intermediate effects in the model - annoyance and sleep for example. Some effects are measured by self-report of those affected (e.g. subjective assessments of annoyance) while others by objective measurement on those affected (e.g. sleep motility measurements).

Noise levels have been derived from reviews of the environmental noise and health literature, below which health effects would not be expected. Although not all reviews agree on the safe levels for each specific health effect, there is a reasonable degree of consensus.

The literature demonstrates that the shorter term effects of excessive noise exposure on sleep (causing sleep disturbance) include fatigue, moodiness, irritability, headaches, nervous stomach, lack of concentration and reduced work ability. These effects are observed during the daytime period following nightly exposure and do not appear to be reduced through repeated exposure.

Longer-term effects on health are more difficult to quantify, although tentative links have been drawn between noise exposure and heart rate, immune response, hypertension, blood pressure, occurrence of ischemic heart disease, cardiovascular disease and myocardial infarction. The above links are often difficult to identify and quantify due to the presence of other environmental and other factors.

In the assessment of health effects from noise exposure, it should be noted that, because of variability in human response, effects will still be experienced by some people at lower levels the bounds of the noise metric used in the assessment, and effects may not be experienced by some people at higher levels.

4.2 Summary of Health Risks associated with Noise

Conclusions of the “*Wallarah No.2 Coal Project Noise and Vibration Impact Assessment*” (**Atkins Acoustics, 2012**) indicate that for Operational and Construction scenarios there are no predicted exceedances at residences. However, impacts have been predicted over 25% of two vacant blocks, albeit with no houses.

In regards to existing background noise, site attended audits confirmed that the local acoustic environments are influenced by road traffic, natural sources and localised domestic activities. In regards to Operational Traffic and rail generated noise levels, criteria were satisfied or marginal increases (1-2dBA) were detected.

The management of noise, like many other environmental and occupational health hazards, involves three main options:

- Elimination or reduction of noise at the source.
- Elimination or disruption of the transmission path.
- Isolation or insulation of the receiver from the noise.

Combinations of these three options represent much of international ‘best practice’. In response to potential criteria exceedances Atkins Acoustics have recommended a set of operational management strategies that should be adopted to assist in controlling noise emissions from the Project. The strategies consist of a combination of the above international best practice options and when applied, they are assumed to effectively control noise emissions from the Project and consequently deeming any risk of adverse health effects as negligible.

5 WATER

5.1 Overview of water management

The Project is located within the Macquarie Tuggerah Lakes Basin, which has a total catchment area of approximately 1,830 km² including several water supply storages, including Mardi Dam and Mangrove Creek Dam which are used for irrigation and domestic water storage for urban centres in the lower sections of the Tuggerah Lakes catchment. During operation, the Project will not discharge groundwater or mine water within the water supply catchment area (**WRM Water & Environment, 2012**).

Some land disturbance, apart from subsidence, in the water supply catchment area may occur during the construction of the proposed ventilation shaft within the Wyong State Forest. Land disturbance associated with the construction of the shaft will be managed through the implementation of best practice erosion and sediment control measures, which will ensure that surface runoff from any disturbed area will meet appropriate water quality standards prior to discharge.

It must be noted that the water supply catchment already contains extensive areas of land-disturbing activities, including agriculture and residential development. The construction of the ventilation shaft, with appropriate control measures in place, will have no measurable adverse effect on downstream water quality.

Impacts on low-flow water quality due to subsidence, such as reduced pH and increased metals concentrations due to fracturing of rock within drainage lines in the upper catchment, are not anticipated to occur. However, subsidence and water quality monitoring programs will be implemented to identify and manage any unexpected impacts.

The main components utilised for water management and its related infrastructure include (**WRM Water & Environment, 2012**):

- A Stockpile Dam which collects runoff from the product coal stockpile.
- A Portal Dam which collects runoff from the raw coal stockpile, offices and workshop area.
- A Mine Operations Dam to store water pumped out of the underground. The Mine Operations Dam will also store runoff water pumped from the Portal Dam and Stockpile Dam.
- A reverse osmosis (RO), or similar, Water Treatment Plant to treat excess mine water from the Mine Operations Dam and supply treated water to the site surface and underground demands.
- A Treated Water Storage to store raffinate from the Water Treatment Plant for reuse. This storage is likely to be a cell in the Mine Operations Dam (separate from the higher salinity mine water).
- Sediment traps and drainage channels to collect and treat runoff from the rail loop and access road.
- Clean water drains to divert runoff from undisturbed catchments around areas disturbed by mining/infrastructure.
- Discharge infrastructure at Tooheys Road Site for treated water releases to Wallarah Creek.
- An Entrance Dam to store water to augment the Buttonderry site demands.
- A sediment dam to collect and treat runoff from the Buttonderry buildings, paved and hardstand areas.

- An underground mine water storage sump.

Monitoring of surface water quality both within and external to the Project boundary will form a key component of the surface water management system. Monitoring of upstream, on site and downstream water quality will assist in demonstrating that the site water management system is effective in meeting its objective of no adverse impact on receiving water quality and will allow for early detection of any impacts and appropriate corrective action (**WRM Water & Environment, 2012**).

5.2 Summary of Health Risk associated with Water

The proposed water management system will ensure the separation of clean and mine water on the site and no uncontrolled discharges from the Mine Water System under all but extreme weather conditions. Given that site surface facilities are strategically located outside of the drinking water catchment, no discharges from site surface facilities will occur into that catchment. The quality of treated water discharged into other catchment areas will be controlled through the operating parameters of the water treatment plant and these parameters will be set to ensure that the quality of water discharged was of similar or better quality than the receiving water.

Given this, there is no likelihood of increases in risks to health from water discharge.

6 OVERALL CONCLUSIONS

The HRA was conducted using methodology consistent with enHealth recommendations, evaluating the possibility of direct and indirect health effects on the local community, resulting from potential environmental impacts (particularly in relation to air quality, noise and drinking water quality). Although this risk assessment is primarily quantitative for air emission exposures, noise and water impacts were assessed qualitatively.

Overall, it is concluded that air emissions, noise and water impacts from the Project present little likelihood of causing adverse health effects as to people living in the nearby areas.

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