REPORT

HEALTH RISK ASSESSMENT: PROPOSED WALLARAH 2 UNDERGROUND COAL MINE, NSW

International Environmental Consultants

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1 INTRODUCTION

This report has been prepared by PAEHolmes on behalf of International Environmental Consultants (IEC).who are preparing an Environmental Assessment for the proposed Wallarah 2 Coal Project (W2CP) to be developed by the Wyong Areas Coal Joint Venture (WACJV). The report provides a quantitative health risk assessment (HRA) of impacts associated with the air emissions from the project.

An air quality assessment has been prepared for this project and is presented in a separate document which also includes a description of the project and the local setting (**PAEHolmes, 2009**). The assessment is based on dispersion modelling of air emissions from the proposed mine and the results of the modelling have been carried forward into the HRA.

This report contains the following information:

1. A discussion of potential health impacts of particulate matter, the most significant air emission from the Project;
2. A review of existing health effects studies with a summary of the most recent knowledge of the risks associated with exposure to particulate matter;
3. A review of the health risks associated with exposure to silica which is a component of coal dust; and
4. A summary of the exposure levels of the local community to emissions from the proposed mine and an estimate of health risks associated with this exposure.

2 HEALTH EFFECTS OF PARTICLES

The human respiratory system has in-built defensive systems that prevent particles larger than approximately 10 microns (μm) from reaching the more sensitive parts of the respiratory system. Respirable particles (PM_{10}) are a health concern because they are easily inhaled and retained in the lung. The epidemiological evidence for the health impacts of particles is based on the mass of particles in the atmosphere with the fine fraction of PM_{10} (i.e. those particles smaller than $PM_{2.5}$) showing a stronger correlation with health impacts than the total mass of PM_{10}. Particles in the range between 2.5 μm and 10 μm ($PM_{10-2.5}$) are referred to as coarse thoracic particles. These particles which are produced primarily from mechanical processes, deposit preferentially in the upper and larger airways. The fine particles are predominantly the products of combustion and these can enter the smaller airways and the alveoli. This is discussed in more detail below.

It is likely that it may be the even finer particles (ultrafine, less than 0.1 μm) which are the main contributor to health impacts and it is also possible that it is the number of particles rather than the mass which is important. At this stage however, the total mass of PM_{10} and $PM_{2.5}$ provides a reasonable surrogate for measuring the "healthiness" or otherwise of the ambient air in urban environments. Furthermore there are no agreed methods for the routine measurement of ultrafine particles or particle numbers and no ambient goals for these measures of particle pollution. The World Health Organisation (**WHO, 2005**) notes that while there is considerable toxicological evidence of the potential detrimental effects of ultrafine particles on human health, the epidemiological evidence is insufficient to reach a conclusion on the exposure/response relationship to ultrafine particles. On that basis, no recommendation has been provided by the WHO at this stage as to guidelines for ultrafine particles. There is also no goal or guideline in Australia for exposure to ultrafine particles.

Much of the recent concern for the health effects of fine particles followed from the investigations carried out in the US, with the view to quantifying the health risks associated with both long-term and short-term exposure to airborne particulate matter. The study is

colloquially referred to as "The Six Cities Study" from the original work by **Dockery et al. (1993)**, which determined a relationship between fine particulate matter ($PM_{2.5}$) in the air and mortality in six US cities.

The basic findings of the Six Cities Study are that there is an increase in mortality with increasing concentrations of fine particulate matter. The conclusions appear to be robust and have been supported by subsequent studies and as far as can be determined are not confounded by other known variables. It is important to note that the observed association between fine particles and mortality 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.

Particles found in the atmosphere can be from numerous sources and include a very broad range of substances, unlike gases which are usually a specific chemical compound (such as sulfur dioxide, nitrogen dioxide, etc).

Simple measures of particulate concentration do not identify the size distribution, number or source of the particles involved. For example, a single measurement of PM_{10} concentration or load does not reveal whether 90% of the particles are in the 0 to 1 μm size range (fine, submicron particles), or if 90% are in the 2.5 μm to 10 μm range (coarse particles). These issues are important because evidence indicates that differences in particle size and composition are important in the health effects that arise.

Coarse particles come from sources such as windblown dust from the desert or agricultural fields and dust kicked up on unpaved roads by vehicle traffic. Dust from mining and quarrying fall into this category due to the large quantities of geological materials handled by mechanical methods and by vehicular use on unpaved roads.

Fine particles are generally emitted from activities such as industrial and residential combustion and from vehicular exhaust. Fine particles are also formed in the atmosphere when gases such as sulfur dioxide, nitrogen oxides, and volatile organic compounds, emitted by combustion activities, are transformed by chemical reactions in the air (forming secondary particles, which can agglomerate and grow in size).

Approximately 90% of particles released into the atmosphere come from natural sources, such as windblown dust, sea-salt, volcanic emissions, forest fires, pollen and other biological debris. Industrial dusts account for some 3% of all aerosol particles (equivalent to about half of that contributed by sea salt particles), and only a small fraction of this is related to dust from mining activities.

The air quality goals for particulate matter do not reflect their chemical composition, however it is recognised that not all particles are the same in terms of their health impacts. It is very likely that particles generated from windblown dust and mining activities are intrinsically less toxic than particle emissions from combustion processes which may contain heavy metals and carcinogenic material, including polycyclic aromatic hydrocarbons. Particulate diesel emissions fall into this category and they are discussed in more details below.

Effects of diesel emissions

Diesel emissions from theW2CP are expected to be minor, however a discussion of their health impacts is included for completeness.

In 1999 **Cohen and Nikula (1999)** published a substantial review of the health effects of diesel exhaust and in 2002 the US EPA completed a major review, including contributions from over 30 authors, of the effects of diesel engine exhaust (**US EPA 2002**). The information in these two publications is very similar and both have been used to prepare the following summary.

Based on a review of these two publications it can be concluded that the chemical composition of diesel exhaust is reasonably well known and that diesel exhaust contains substances that are known to be harmful to health, both because of the form in which they occur (fine particles as well as gases) and their composition. From a health perspective diesel exhaust (DE) is a complex mixture of gases and PM with the PM having a mass median diameter in the range 0.05 to 1 μm. These particles are capable of reaching the deepest parts of the respiratory system. Exposure to diesel exhaust is usually expressed in terms of exposure to the mass of the particulate phase.

Harmful effects are believed to include an increase in the incidence of cancer and other effects such as the exacerbation of asthma symptoms and irritation and inflammation symptoms. This brief review focuses more on the incidence of cancer as this is far better studied.

Actual human exposures are difficult to determine accurately and dose-response relationships are less well known. Dose response relationships can be determined in a number of ways. Laboratory experiments in which rats, hamsters and other laboratory animals are exposed to various controlled levels of the pollutant provide some insight into heath effects. Deliberate exposure of human populations is generally not done for obvious reasons, but studies of the effects caused by occupational exposures provide valuable information.

For diesel exhaust the most exposed populations are underground mine workers followed by workers in the transport industry such as truck and bus drivers and those that work in places where diesel equipment is widely used. The major difficulty in using such data is in compensating for confounding effects such as smoking and other socio-economic factors that affect health and in making accurate estimates of exposure.

Information from **Cohen and Nikula (1999)** (Page 710) provides data indicating that the range of concentrations that human populations are exposed to spans three orders of magnitude and ranges from 1 to 10 $\mu g/m^3$ in the general urban environment, 4 $\mu g/m^3$ for truck drivers and up to 1740 $\mu g/m^3$ (over a working shift) for underground mine workers.

The studies cited in the review show a wide range of effects ranging from no significant change in lung cancer rates to significant changes. The study with the largest population was one covering 18,000 British coalminers (Page 714), controlled for smoking and allowing a latency period of 15 years, indicated that the relative risk for lung cancer was 1.16 per gram-hour of exposure to diesel exhaust (95% confidence interval 0.90 to 1.49). Thus the overall conclusion from the data reviewed is that occupational exposures are likely to cause an increase in the relative risk of cancer. This is consistent with the IARC judgement (Page 725) that classes diesel exhaust as a "probable carcinogen".

The WHO has also reached a similar conclusion (Page 726), but has added that no data existed that would allow human risk factors to be developed. Other organisations have attempted to assign a quantitative relative risk factor to exposure. The US EPA has used animal studies to estimate that a lifetime exposure to 1 $\mu g/m^3$ would produce 1 excess cancer per 100,000 people. The Californian EPA has used US rail worker data to estimate the risk to be 2 excess cancers per 1,000 people exposed to 1 $\mu g/m^3$ over a lifetime. The most recent US EPA review (**US EPA, 2002** Page 6-32) concludes that exposure to "5 $\mu g/m^3$ of diesel PM is a chronic

exposure level likely to be without an appreciable risk of adverse human health effects". They consider that this is consistent with their annual average standard of 15 μg/m^3 for $PM_{2.5}$, which of course includes all sources of fine particles, not just particles associated with diesel exhaust.

This level therefore provides some benchmark for assessing the impacts of diesel emissions from mining activities.

3 HEALTH RISKS ASSOCIATED WITH PARTICLE EXPOSURE

Much of the evidence for the health effects of particulate matter is drawn from population studies. These can either be time series studies, where routinely collected population outcomes such as deaths, hospitalisation, etc are modelled against fluctuations in routinely collected ambient pollutant levels. These are a powerful way to examine the influence of pollutants upon the community however they are confounded by a large range of factors such as influenza epidemics, weather, and the difficulty of determining individual exposure to ambient pollutants. However if they are carried out on a sufficiently large population they can provide useful correlations.

All of these studies, in both Australia and overseas, have demonstrated that levels of particulate matter that are within ambient air quality goals can have an adverse affect on mortality and hospital admissions. The type of effect is related predominantly to respiratory illness including childhood asthma but also includes elderly heart disease and elderly chronic obstructive pulmonary disease (COPD).

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 and others 2004**). The results of the analysis of short-term effects for PM_{10} and $PM_{2.5}$ are summarised in **Table 1** below.

Table 1. Short term effects on health from 10 μg/m^3 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 (O_3). Report of a WHO task group. Copenhagen, WHO Regional Office for Europe, 2004 (http://www.euro.who.int/document/e82792.pdf)

Long-term exposure to particulate matter also affects life expectancy. Pope and others reported a 6% increase in the risk of death from all causes per 10 μg/m^3 increase in long-term $PM_{2.5}$ concentration (**Pope and others, 2002**).

The **WHO (2006)** has recently released a global update which provides a summary of the risk assessment underpinning their guidelines for $PM_{2.5}$ and PM_{10}. The WHO guideline for PM_{10} 24-hour mean is the same as the NEPM and NSW Department of Environment, Climate Change and Water (DECCW) goal, that is 50 $\mu g/m^3$. The WHO 24-hour goal for $PM_{2.5}$ is 25 $\mu g/m^3$.

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 (10^{-4} m^{-1})). **Table 2** and **Table 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 PM_{10} and $PM_{2.5}$ data were available for three of the cities.

Table 2. Increases in daily mortality as a result of a 1 unit increase in 24-hour bsp ($10^{-4}m^{-1}$) or a 10 µg/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)
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. Increases in hospital admissions as a result of a 1 unit increase in bsp ($10^{-4}m^{-1}$) or a 10 µg/m^3 increase in 24-hour PM_{10} or $PM_{2.5}$ (Simpson, 2005b)

City	Hospital admissions	Estimated percentage increase in risk (95% confidence interval)
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)

The daily excess mortality for $PM_{2.5}$ is therefore the same (0.9%) for the Australian data and the world wide data.

Only cardiac hospital admissions data are available for $PM_{2.5}$ but by comparing the bsp data in **Table 3** with the $PM_{2.5}$ data, the hospital admission risk for respiratory and asthma cases can be inferred to be similar to the risk for cardiac cases from exposure to a 10 $\mu g/m^3$ increase in 24-hour $PM_{2.5}$.

There is more limited information available on mortality and morbidity associated with coarse particles, particularly in rural areas. This has been reviewed by **Brunekreef and Forsberg, 2005**. While the review showed that there were associated risks of morbidity with increased exposure to coarse particles, the majority of the studies were in urban environments, rather than in rural environments.

In the few studies where dust storms or wind-blown dust were examined, the following findings were made:

- A study in Anchorage Alaska showed some effects of dust-storms on outpatient visits for asthma, bronchitis and upper respiratory tract infections (**Gordian and others, 1996**);
- A study from Washington State showed a small increase in respiratory hospital admissions after dust storms where maximum PM_{10} concentrations exceeded 1000 µg/m^3 (**Hefflin and others, 1994**);
- A time series study of mortality from Spokane, Washington showed that in high dust storm days, with an average PM_{10} concentration of 263 µg/m^3 there was no increase in mortality compared with control days which had an average PM_{10} concentration of 42 µg/m^3 (**Schwartz et al, 1999a**); and
- Studies of respiratory and cardiovascular hospital admissions from the same city found an association with high PM_{10} (**Schwartz, 1996** and Schwartz**, 1999b**). However the later 1999a report by Schwartz commented that these studies excluded dust storm days and were focused on days where much of the PM_{10} in Spokane was from combustion sources.

A recent large study from Johns Hopkins Bloomberg School of Public Health of the effect of exposure to coarse particulate matter on hospital admissions in approximately 12 million Medicare enrolees over a six year period (**Peng and others, 2008**) concluded that after adjustment for the effect of $PM_{2.5}$ there were no statistically significant associations between coarse particulate matter and hospital admission for cardiovascular and respiratory diseases.

In view of these findings, no quantitative health risk assessment has been undertaken for exposure to coarse particulate matter. The assessment has focussed rather on the small fraction of fine particulate matter present in the mine dust. That is not to say that the coarse particles have no effect, but the air quality guidelines for PM_{10}.are set to protect against such health effects. It should be noted that the US EPA have revoked their long-term goal for PM_{10} on the basis that the available evidence did not suggest a link between long-term exposure to coarse particles and health problems. However they have retained their annual $PM_{2.5}$ goal of 15 µg/m^3 NSW has no long-term goal for $PM_{2.5}$ although the NEPM goal of 8 µg/m^3 can be referred to.

4 HEALTH RISKS ASSOCIATED WITH EXPOSURE TO SILICA

Silica (SiO_2) is a naturally occurring mineral composed of silicon and oxygen. It is a component of coal and in the case of the W2CP it is estimated to be approximately 7%. It exists in crystalline and amorphous forms depending on the structural arrangement of the oxygen and silicon atoms. Only the crystalline forms are known to be fibrogenic[a] and only the respirable particles (those which are capable of reaching the gas exchange region of the lungs) are considered in determining health effects of crystalline silica.

There are a number of size-based descriptors used in describing particulate matter, particularly with respect to occupational exposure, and it is useful to define these for the following discussion. The descriptors are:

- PM_{10} - particles with an equivalent aerodynamic diameter of less than approximately 10µm (as discussed above) – the largest of these are inhalable;

[a] Fibrogenic dust is a dust which causes increase of fibrotic (scar) tissue after deposition in the gas exchange region of the lung.

- PM_7 - particles with an equivalent aerodynamic diameter of less than approximately 7μm – the largest of these can reach the thoracic region of the respiratory system;
- PM_4 - particles with an equivalent aerodynamic diameter of less than approximately 4μm – the largest of these can reach the alveoli; and
- $PM_{2.5}$ - particles with an equivalent aerodynamic diameter of less than approximately 2.5μm (as discussed above) – fine particle fraction.

Particles described as PM_7 are a sub-component of PM_{10}, PM_4 particles are a sub-component of PM_7 and PM_{10}, and $PM_{2.5}$ particles are a sub-component of PM_4, PM_7 and PM_{10}.

The three most common types of crystalline silica are quartz, tridymite and cristobalite. Human exposure to crystalline silica occurs most often during occupational activities that involve the working of materials containing crystalline silica products (e.g. masonry, concrete, sandstone) or use or manufacture of crystalline silica-containing products. Activities that involve cutting, grinding or breaking of these materials can result in the liberation of particles in PM_{10}, PM_7, PM_4 and $PM_{2.5}$ size ranges. Ambient crystalline silica dust can occur due to natural, industrial and agricultural activities.

Repeated and prolonged exposure to relatively high concentrations of crystalline silica can cause the disease known as silicosis. This respiratory disease is characterised by scarring and hardening of the lung tissue and it reduces the ability of the lungs to extract oxygen from the air. Occurrences of silicosis are highly correlated to occupations where particles of crystalline silica are released to the atmosphere (e.g. mining, quarrying, foundries, sandblasting). Silicosis is becoming less common in industrialised nations due to the stringent controls on airborne dust and the use of protective devices.

General community (non-occupational) exposure to respirable crystalline silica is typically well below the national exposure standards and consequently 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*".

4.1 Assessment Criteria

Occupational Goals

In Australia, the occupational exposure standards for respirable crystalline silica are defined by the National Occupational Health and Safety Commission (NOHSC). The national exposure standard for respirable crystalline silica is 100μg/m^3 (Time Weighted Average (TWA))[b]. Although the occupational standard is not applicable to the assessment of the ambient air quality, the risk of silicosis among people living in areas surrounding activities such as quarrying would generally be very small provided the concentration of respirable particles at the source was acceptable in terms of occupational safety.

[b] TWA - the average airborne concentration of a particular substance when calculated over a normal eight-hour working day, for a five-day working week

Ambient Goals

NSW has not set any impact assessment criteria for crystalline silica. The Victorian EPA has adopted an ambient assessment criterion for mining and extractive industries of 3μg/m^3 (annual average as $PM_{2.5}$) (**VEPA, 2007**). This has been derived from the Reference Exposure Level (REL)[c] set by the California EPA Office of Environmental Health Hazard Assessment of 3μg/m^3 (annual average as PM_4) (**OEHHA, 2005**).

US EPA Silicosis Potency Estimates

The **US EPA (1996)** examined the non-cancer epidemiological literature on crystalline silica induced diseases. From the extensive data available, which examined the medical histories of thousands of miners, they concluded that the cumulative risk of developing silicosis is zero for cumulative exposures of less than 1000μg/m^3.years.

Cumulative exposure is the average respirable crystalline silica concentration a person is exposed to over a period of time, multiplied by the number of years exposed. For example, an exposure of 1000μg/m^3.years, would be experienced by an individual exposed to 14.3 μg/m^3 per year for 70 years. For cumulative exposures less than 1000μg/m^3.years, the US EPA concludes that the risk of developing silicosis is zero.

The methodology defined by the US EPA has been applied to the predicted impacts at the nearby residences to determine the cumulative risk

5 HEALTH RISKS DUE TO PARTICULATE EXPOSURE FROM THE PROJECT

5.1 Exposure to $PM_{2.5}$

The data analysis summarised above in relation to particulate matter was based predominantly on studies in urban environments, where, as discussed above, combustion sources make a significant contribution to total particulate concentrations. The chemical composition of particulates in urban environments is therefore substantially different from the particulates associated with mining. Further, 40 to 60% of ambient PM_{10} levels in an urban environment are typically $PM_{2.5}$, whereas on average the $PM_{2.5}$ fraction of the PM_{10} emitted from mining operations is typically about 12%.

It is reasonable to assume therefore that applying the risk factors developed for urban environments to emissions which are a mixture of crustal origin and coal dust with some contribution from diesel vehicles provides a conservative estimate of the risk associated with exposure to particulates from the Project.

A more realistic, but still conservative approach, given the difference in chemical composition, is to estimate the risk associated with exposure to $PM_{2.5}$ emissions from the Project.

Table 4 presents the predicted maximum 24-hour and annual average PM_{10} and $PM_{2.5}$ concentrations at sensitive receptors potentially arising from the Project operations. The

[c] RELs are used by the California Environmental Protection Agency as indicators of potential adverse health effects. A REL is a concentration level (g/m3) or dose (mg/kg/day) at (or below) which no adverse health effects are anticipated for a specified time period. RELs are generally based on the most sensitive adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety.

locations of the sensitive receptors are shown on **Figure 1**. The 24-hour predictions corresponded to the busiest day.

Table 4. Predicted maximum 24-hour and annual average PM_{10} and $PM_{2.5}$ concentrations due to the Project alone at selected sensitive receptors for the Project operations

	24-hour maximum PM_{10} ($\mu g/m^3$)	24-hour maximum $PM_{2.5}$ ($\mu g/m^3$)	Annual average PM_{10} ($\mu g/m^3$)	Annual average $PM_{2.5}$ ($\mu g/m^3$)
Receptor				
P1	8.0	1.3	0.2	0.04
P2	10.9	1.8	0.3	0.05
P3	28.7	4.3	0.6	0.09
P4	15.7	2.6	0.3	0.04
P5	7.4	1.1	0.2	0.02
P6	6.9	1.1	0.3	0.04
P7	14.7	2.1	0.5	0.07
P8	12.6	1.9	0.7	0.09
P9	17.2	2.7	0.7	0.11
P10	10.1	1.6	0.5	0.07
R1	4.2	0.7	0.1	0.01
R2	4.4	0.7	0.1	0.01
R3	3.3	0.6	0.1	0.01
R4	3.1	0.5	0.1	0.01
R5 (Blue Haven)	8.6	1.7	0.3	0.04

The risk factors in **Table 2** and **Table 3** have been used to estimate the risks associated with exposure to the particulate emissions from the mine. Daily and annual mortality rates for the Northern Sydney and Central Coast region for 2002 to 2006 and daily hospital admissions for all of NSW in 2004-2005 were obtained from the NSW Health website (http://www.health.nsw.gov.au/publichealth/chorep/ Accessed (November/2009)).

An example of the calculation is shown below.

For a population size of 1000 exposed to a 24-hour $PM_{2.5}$ concentration of 4 $\mu g/m^3$ where the daily mortality is 1.9/100,000 people and the additional risk of death is 0.9% per 10 $\mu g/m^3$ increase in $PM_{2.5}$, the number of additional deaths would be:

Additional deaths per day =0.9/100 x 3/10 x 1.9/100,000 x 1000

=0.0000513

Table 5 summarises the risks for the most exposed individual assuming a maximum daily exposure to 4.3 $\mu g/m^3$ of $PM_{2.5}$ and an annual average exposure of 0.7 $\mu g/m^3$

Table 5. Estimated increase in risk of indicated event for the worst-day – assumed to be exposure to 6 μg/m³ of $PM_{2.5}$

Event	Frequency of occurrence	Additional risk per 10 μg/m³ increase in $PM_{2.5}$	Additional risk per daily 4.3 μg/m³ increase in $PM_{2.5}$ and annual 0.7 μg/m³ increase in $PM_{2.5}$ due to exposure to particulate emissions from the Project.
	Per 100,000	(%)	At most exposed individual
			Risk of death or admission to hospital
Daily mortality	1.6	0.9	1 in 16.4million (6.10E-08)
Daily hospital admissions for cardiovascular disease (all ages)	5.8	5.1.	1 in 0.802million (1.25E-06)
Daily hospital admissions for respiratory disease (65 + years)	1.7	5.1 (assumed to be same as cardiovascular)	1 in 2.74million (3.66E-07)
Daily hospital admissions for asthma 15-64 years	0.5	5.1 (assumed to be same as cardiovascular)	1 in 9.3 million (1.08E-07)
Annual mortality	575	6	1 in 0.041million (2.42E-05)

5.2 Exposure to silica

In order to compare the predicted concentrations of crystalline silica in the ambient air due to the project with the relevant assessment criteria, it is necessary to know the crystalline silica content of the particulate emissions from the proposed operations.

The ratio of PM_4/PM_{10} is 0.3 (**SPCC, 1986**), therefore, the predicted annual average PM_{10} concentrations have been converted to annual average PM_4 concentrations by multiplying by the ratio of PM_4/PM_{10}, i.e. 0.3. The crystalline silica content of the PM_4 emissions has been assumed to be the same as the silica content of the coal, that is 7%.

At the most affected residence, the predicted annual average PM_{10} concentration was 0.7$\mu g/m^3$. Therefore, the estimated annual average respirable (PM_4) concentration of crystalline silica was 0.015 $\mu g/m^3$ [0.7 $\mu g/m^3$ x 0.3 x 0.07]. This is less than the Californian REL of 3 $\mu g/m^3$.

5.2.1 Consideration of the US EPA Risk Estimate

As exposure to crystalline silica is primarily an occupational health hazard, very little monitoring has been conducted to measure ambient concentrations.

Data collected in Victoria estimated the background concentration to be 0.7$\mu g/m^3$ (**Toxikos, 2005**). This is comparable to the ambient level in a Californian rural area of 0.6$\mu g/m^3$ (**OEHHA, 2005**). In the absence of any local data, it has been assumed the background level of 0.7 $\mu g/m^3$ as measured in Victoria applies to the Project Site. The cumulative risk related to the exposure of crystalline silica is therefore as presented below.

Existing background respirable crystalline silica	=	0.7 $\mu g/m^3$
Increase in respirable crystalline silica at most affected receptor	=	0.015 $\mu g/m^3$
Total annual respirable crystalline silica	=	0.715 $\mu g/m^3$

Based on the assumption that the project has a proposed life of 40 years, the respirable crystalline silica exposure during the operation of the quarry is 0.715$\mu g/m^3$ x 40 years = 28.6 $\mu g/m^3$.years (most of which is due to the assumed background concentration).

If an individual were to live for the remaining 30 years of their assumed 70 year lifetime in the same area, the exposure to respirable crystalline silica would be 0.7 $\mu g/m^3$ x 30 years = 21.0 $\mu g/m^3$.years.

The total lifetime exposure from background concentrations and the proposed Project would therefore be 28.6$\mu g/m^3$.years + 21.0$\mu g/m^3$.years = 49.6 $\mu g/m^3$.years.

As discussed above, the US EPA concluded that the cumulative risk of developing silicosis is zero for cumulative exposures of less than 1000$\mu g/m^3$.years. The estimated total lifetime cumulative exposure for the proposed Project is approximately 20 times lower than this.

6 SUMMARY AND CONCLUSIONS

The impacts of the proposed mine project have been assessed in terms of the likely risks to various health outcomes. The risk factors used in the analysis have been developed using research results from the last decade on the health effects of particulate matter on human populations in urban areas. Risks of exposure to silica have also been considered.

The analysis provides estimates of the increase in daily mortality due to emissions from the mine 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 mine is estimated to be 1 in 16.4 million. This is a small risk. Increase in risk for hospital admission is also low.

The health risk from exposure to silica is also extremely low.

7 REFERENCES

Anderson H R, Atkinson R W, Peacock J L, Marston L, Konstantinou K (2004)

"Meta-analysis of time-series studies and panel studies of Particulate Matter (PM) and Ozone (O_3)" Report of WHO Task Group

Brunekreef B and Forsberg B (2005)

"Epidemiological evidence of effects of coarse airborne particles on health" European Respiratory Journal 26, 309-318

CICAD (2000)

"Concise International Chemical Assessment Document 24. Crystalline Silica, Quartz" Published by the World Health Organization, Geneva, 2000.

Cohen A J and Nikula K (1999)

"The Health Effects of Diesel Exhaust" Published in "Air Pollution and Health" Academic Press, 24-48 Oval Road, London, NW1 7DX Editors Holgate S T, Samet J M, Kpren H K and Maynard R L.

Dockery D W, Pope C A, Xiping Xu, Spengler J D, Ware J H, Fay M E, Ferris B G and Speizer F E (1993)

"An association between air pollution and mortality in six US cities" The New England Journal of Medicine, Volume 329, Number 24,1753-1759

Gordian ME, Ozkaynak, H Xue J, Morris SS, Spengler JD (1996)

"Particulate air pollution and respiratory disease in Anchorage, Alaska" Environmental Health Perspectives, 104 290-297

Health Effects Institute (2003)

"Revised analyses of time-series studies of air pollution and health" Special Report May 2003

Hefflin BJ, Jalaludin B, Mclure E et al (1994)

"Surveillance for dust storms and respiratory diseases in Washington" Archives of Environmental Health, 49, 170-174

Peng R D, Chang, H H, Bell M L, McDermott A, Zeger S L, Samet J M, Dominici F (2008)

"Coarse particulate matter air pollution and hospital admission for cardiovascular and respiratory disease among Medicare patients" Journal of the American Medical Association, 299:2172-2179 (2008)

Pope C A 3rd, Burnett R T, Thun M J, Calle E E, Krewski D, Ito K, et al. (2002)

"Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution". Journal of the American Medical Association 287: 1132041 (2002)

Pope C A 3rd, Burnett R T, Thurston G D, Thun M J, Calle E E, Krewski D, Godleski J J (2004)

"Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution" Circulation, 109: 71-77 (2004)

Schwartz J (1996)

"Air pollution and hospital admissions for respiratory disease" Epidemiology 7:20-28 (1996)

Schwartz J, Norris G, Larson T, Sheppard L, Claiborne C, Koenig J (1999a)

"Episodes of high coarse particle concentrations are not associated with increased mortality" Environmental Health Perspectives Volume 107 Number 5 May 1999

Schwartz J (1999b)

"Air pollution and hospital admissions for heart disease in eight US Counties." Epidemiology 10:17-22 (1999)

Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G, Neller A (2005a)

"The short-term effects of air pollution on daily mortality in four Australian cities" Aust N Z J Public Health 2005; 29: 205-12

Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G (2005b)

"The short-term effects of air pollution on hospital admissions in four Australian cities" Aust N Z J Public Health 2005; 29:213-21

Toxikos (2005)

"Health Risk Assessment of Crystalline Silica from Alex Fraser's proposed Recycling/Transfer Station of Construction Waste, Clarinda", Prepared by Toxicos Pty Ltd, February 2005, TR080105-RF.

US EPA (2002)

"Health Assessment Document for Diesel Engine Exhaust" Published by National Centre for Environmental Assessment, Office of Research and Development, US EPA, Washington DC, Report Number EPA/600/8-90/057F

WHO (2005)

"WHO air quality guidelines global update 2005 Report on a Working Group meeting, Bonn, Germany, 18-20 October 2005" Published by WHO Regional Office for Europe, Scherfigsvej 8, DK-2100 Copenhagen O Denmark (also available from WHO/Europe web site http://www.euro.who.int/pubrequest).

WHO (2006)

Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: Global update 2005, Summary of risk assessment

FIGURES

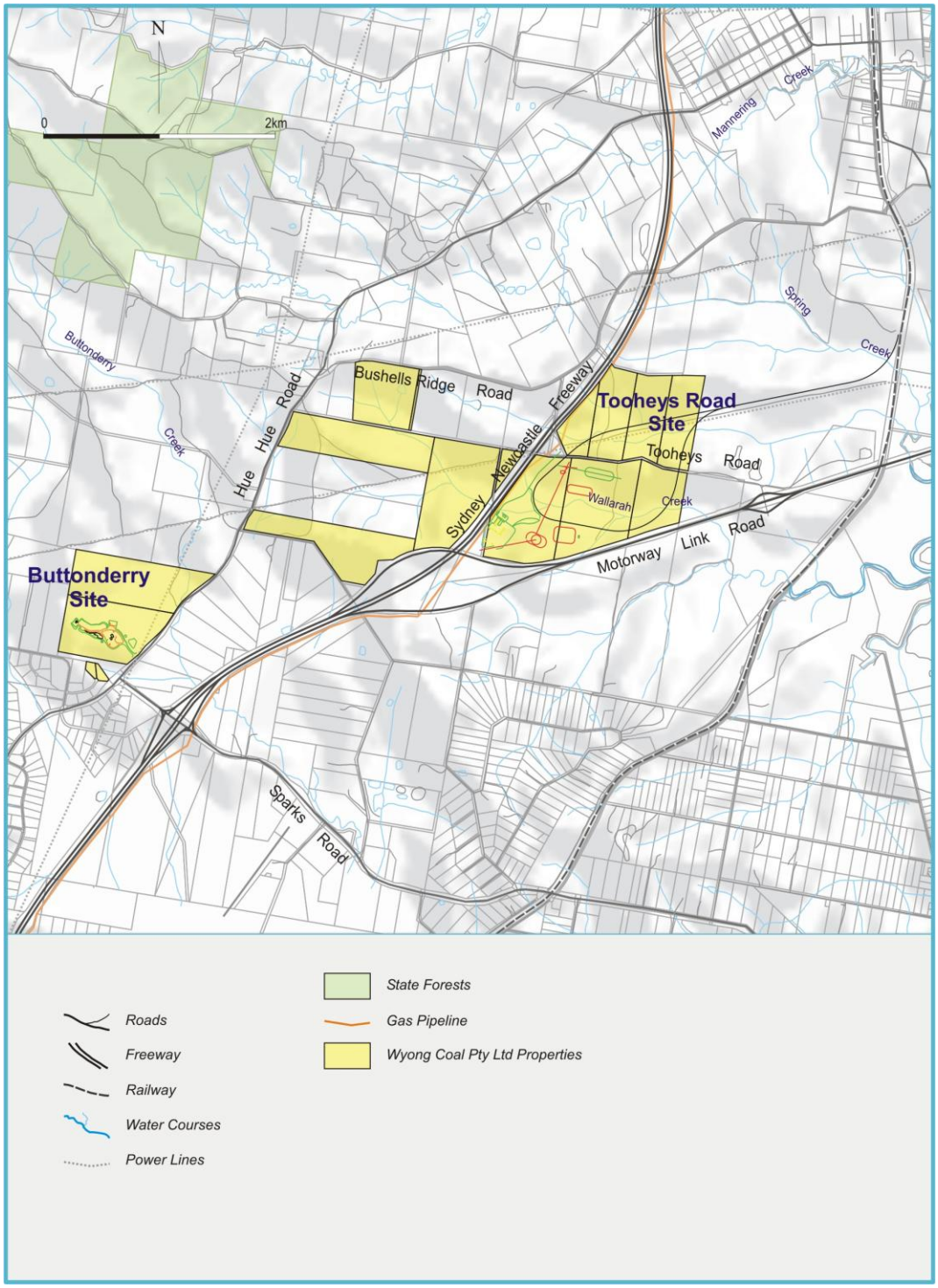

Figure 1 Location of Project