MOOLARBEN COAL PROJECT

APPENDIX 3A

Health Risk Assessment

HEALTH RISK ASSESSMENT
MOOLARBEN OPEN CUT COAL MINE

August 2006

Prepared for
Wells Environmental Services

by

Holmes Air Sciences
Suite 2B, 14 Glen St
Eastwood NSW 2122
Phone : (02) 9874 8644
Fax : (02) 9874 8904
Email : has@holmair.com.au

CONTENTS

TABLES

1. INTRODUCTION

This report has been prepared by Holmes Air Sciences on behalf of Wells Environmental Services who are preparing an environmental assessment for Moolarben Coal Pty Ltd (MCM). MCM propose to develop an open cut coal mine in the area to the south of the village of Ulan and the existing Ulan Coal Mine. The report provides a quantitative health risk assessment (HRA) of impacts associated with the air emissions from the mine.

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 (**Holmes Air Sciences, 2006**). 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 proposed mine.
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 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 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} ($PM_{2.5}$) showing a stronger correlation with health impacts than the total mass of PM_{10}.

It is likely that it may be the even finer particles (ultrafine, less than 0.1 microns) 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 sulphur 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 sulphur 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 refect 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

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 μg/m3 in the general urban environment, 4 μg/m3 for truck drivers and up to 1740 μg/m3 (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 μg/m3 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 μg/m3 over a lifetime. The most recent US EPA review (**US EPA, 2002** Page 6-32) concludes that exposure to "5 μg/m3 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/m3 for PM2.5, which of course includes all sources of fine particle, not just particles associated with diesel exhaust.

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

3. HEALTH RISKS ASSOCIATED WITH PARTICLE EXPOSURE

Population studies 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, apart 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/m³ increases in PM_{10} concentration

Health outcome	Estimated percentage increase in risk per 10 μg/m³ PM_{10} (95% confidence interval)	Estimated percentage increase in risk per 10 μg/m³ $PM_{2.5}$ (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/m³ increase in long-term $PM_{2.5}$ concentration (**Pope and others, 2002**).

The most 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 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.

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/m³ 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/m³ 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 cardiac PM_{10} data in **Table 3** with the $PM_{2.5}$ data, the hospital admission $PM_{2.5}$ risk for cardiac and respiratory cases combined can be inferred to be11.3 % (5.1 +2.9 x (5.1/2.4)

4. HEALTH RISKS DUE TO PARTICULATE EXPOSURE FROM MOOLARBEN MINING OPERATION

The data analysis summarised above was based 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 of predominantly crustal origin provides a conservative estimate of the risk associated with exposure to particulates from the Moolarbern mine operations. This would be particularly true for the PM_{10} risk factors, give that the $PM_{2.5}$ fraction in mining dust is much lower than experienced in cities.

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

Table 4 presents the predicted maximum 24-hour and annual average $PM_{2.5}$ concentrations at sensitive receptors (residences, the school and other community facilities) due to Moolarben mining operations in various years of operation. The locations of the sensitive receptors are shown on **Figure 1**. (Note receptors in the Ulan Village are not numbered). It should be noted that in the early years mine operations would need to be managed so that the 24-hour average PM_{10} concentrations due to emissions from the mine were always less than 50 μg/m³. The 24-hour $PM_{2.5}$ concentrations corresponding to PM_{10} concentration of 50 μg/m³ would be expected to be approximately 12% of 50 μg/m³ namely 6 μg/m³ and thus

any prediction above 6 μg/m³ would be expected to be lower in practice because the mining operations would have been modified to maintain the PM_{10} concentration at a level less than 50 μg/m³.

Table 4 Predicted maximum 24-hour and annual average $PM_{2.5}$ concentration at selected sensitive receptors

				Year-2		Year-5		Year-8		Year-10	
Easting (m)	Northing (m)	Residence Identification	Assumed Occupancy	24-hour PM2.5	Annual average PM2.5	24-hour PM2.5	Annual average PM2.5	24-hour PM2.5	Annual average	24-hour PM2.5	Annual average PM2.5
758171	6424610	46A	3	9.2	2.0	13.2	0.8	3.1	0.4	2.1	0.2
757370	6423594	49	3	9.7	1.3	12.6	0.5	2.9	0.4	2.0	0.2
757478	6422930	9	3	15.7	1.0	14.7	0.4	3.8	0.5	2.4	0.2
757342	6421298	22	3	8.6	0.4	5.8	0.2	4.7	0.8	1.6	0.2
759764	6420796	5	3	5.9	0.2	3.6	0.2	13.2	2.1	3.0	0.4
759147	6422220	46B	3	9.1	0.6	8.0	0.3	3.9	0.6	2.2	0.3
760008	6416123	31	3	1.9	0.1	1.6	0.1	1.9	0.2	12.1	0.9
760388	6416975	36	3	2.2	0.1	1.8	0.1	4.5	0.4	10.8	1.6
763590	6413194	32	3	1.0	0.0	1.3	0.0	2.2	0.1	4.2	0.2
760293	6413734	47	3	1.4	0.0	1.3	0.1	1.3	0.1	4.7	0.3
758435	6416631	30	3	1.6	0.1	1.6	0.1	2.6	0.2	8.5	0.7
757110	6421102	23	3	7.8	0.3	5.4	0.2	5.0	0.8	1.6	0.3
756926	6419919	58	3	4.6	0.2	3.9	0.1	4.3	0.6	2.4	0.3
758295	6421382	20	3	7.7	0.4	6.1	0.2	4.9	1.0	1.8	0.3
758351	6425038	160A	School 41	9.8	1.3	9.5	1.0	3.2	0.5	1.9	0.3
758427	6423790	25	3	21.9	3.8	20.9	0.7	4.8	0.5	3.8	0.3
765574	6412269	48	3	0.9	0.0	1.1	0.0	1.6	0.1	3.2	0.1
763756	6415963	29A	3	1.3	0.1	1.3	0.1	3.5	0.2	36.2	2.6
762840	6415591	29B	3	1.4	0.1	1.9	0.1	3.6	0.2	61.6	7.8
759316	6416451	28	3	1.5	0.1	1.6	0.1	2.0	0.2	10.7	0.8
763424	6421248	2	3	3.6	0.2	3.8	0.2	5.2	0.7	2.4	0.3
763220	6422900	8	3	4.9	0.3	6.5	0.4	2.9	0.5	1.7	0.2
761480	6422256	7	3	9.7	0.5	7.0	0.3	6.9	0.8	2.6	0.3
764184	6424816	16	3	3.8	0.3	9.6	0.6	1.5	0.3	1.2	0.2
763884	6423532	46C	3	4.9	0.3	9.2	0.4	2.2	0.4	1.4	0.2
763904	6424204	46D	3	4.1	0.3	9.8	0.5	1.7	0.3	1.2	0.2
764864	6425885	14	3	4.5	0.3	9.4	0.7	1.4	0.3	1.1	0.2
763608	6426397	12	3	4.7	0.6	23.6	1.8	2.0	0.4	1.6	0.2
763860	6426113	13	3	4.7	0.5	17.5	1.3	2.1	0.3	1.4	0.2
764496	6425257	15	3	4.1	0.3	9.7	0.7	1.5	0.3	1.1	0.2
762910	6431699	10A	3	2.2	0.2	5.0	0.4	1.6	0.1	1.0	0.1
762881	6431819	10B	3	2.2	0.2	4.9	0.4	1.5	0.1	1.0	0.1
765265	6431931	11	3	1.7	0.2	2.9	0.4	1.1	0.1	0.7	0.1
758663	6425526	46E	3	9.5	0.9	13.5	1.7	3.0	0.6	1.9	0.4
758339	6425214	162	Hotel	9.8	1.1	9.0	1.2	3.1	0.6	1.9	0.3
757430	6423706	26	3	9.6	1.5	12.6	0.5	2.9	0.4	2.0	0.2
758311	6425114	161	3	9.8	1.2	8.9	1.1	3.1	0.5	1.9	0.3
758279	6425022	160B	3	9.6	1.3	9.2	1.0	3.1	0.5	1.9	0.3
758431	6425114	148	3	10.0	1.2	9.5	1.1	3.3	0.6	1.9	0.3

				Year-2		Year-5		Year-8		Year-10	
Easting (m)	Northing (m)	Residence Identification	Assumed Occupancy	24-hour PM2.5	Annual average PM2.5	24-hour PM2.5	Annual average PM2.5	24-hour PM2.5	Annual average	24-hour PM2.5	Annual average PM2.5
758391	6425134	167	3	10.0	1.1	9.2	1.1	3.2	0.6	1.9	0.3
758271	6425286	165	3	9.5	1.0	9.0	1.2	3.0	0.5	1.9	0.3
758235	6425134	159	3	9.6	1.2	8.7	1.0	3.1	0.5	1.9	0.3
758243	6425158	41C	3	9.6	1.1	8.6	1.1	3.1	0.5	1.9	0.3
758199	6425198	157	3	9.5	1.1	8.5	1.1	3.0	0.5	1.9	0.3
758111	6425214	154	3	9.3	1.1	8.5	1.1	3.0	0.5	1.9	0.3
758095	6425122	155	3	9.3	1.2	8.5	1.0	3.0	0.5	1.9	0.3
758071	6425074	156	3	9.3	1.3	8.7	1.0	3.0	0.5	1.9	0.3
757999	6425110	153	3	9.2	1.3	8.4	1.0	2.9	0.5	1.9	0.3
757943	6425078	150	3	9.1	1.3	8.4	0.9	2.9	0.5	1.9	0.2
758371	6425110	168	Church	9.9	1.2	9.2	1.1	3.2	0.5	1.9	0.3
757979	6425062	151	Church	9.1	1.3	8.5	0.9	2.9	0.5	1.9	0.3
757835	6424894	158	3	9.0	1.5	8.8	0.8	2.8	0.4	1.9	0.2
757462	6425554	46F	3	8.3	1.1	7.4	1.0	2.5	0.4	1.8	0.2
756626	6424030	169	3	7.6	1.1	8.2	0.5	2.5	0.4	1.6	0.2
756822	6421246	41A	3	9.0	0.4	5.8	0.2	4.7	0.7	1.8	0.3
756822	6421246	57	3	9.0	0.4	5.8	0.2	4.7	0.7	1.8	0.3
758171	6424610	46A	3	9.2	2.0	13.2	0.8	3.1	0.4	2.1	0.2

The predicted concentrations in **Table 4** have been used in conjunction with the risk factors in **Table 2** and **Table 3** to estimate the risks associated with exposure to the particulate emissions from the mine. Daily and annual mortality rates for the Hunter and New England region for 2003 were obtained from the NSW Health website (http://www.health.nsw.gov.au/public-health/chorep/bod/bod_dth_ahs.htm. Accessed (28/04/2006)).

It was assumed that daily hospital admission rate for childhood asthma was 1 in 100,000.

An example of the calculation is shown below.

For a population of size 1000 exposed to a 24-hour $PM_{2.5}$ concentration of 1 $\mu g/m^3$ where the daily mortality is 2/100,000 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 1/10 x 2/100,000 x 1000
=0.000018

If we consider firstly the mine without any real-time controls it is found that for a hypothetical individual, located at the most-affected receptor on the worst day in the life of the mine (i.e. the day with the highest predicted 24-hour average $PM_{2.5}$ concentrations) the $PM_{2.5}$ (24-hour) concentration to which they are predicted to be exposed is 23.6 $\mu g/m^3$ (Residence 12 in Year 5, see **Table 4**). This would translate into an increased risk of mortality of 0.9/100 x 23.6/10 x 2/100,000 is 0.00000042 or 1 in 2,354,000.

However, as noted earlier, the mine would be managed so that the 24-hour PM_{10} concentrations due to emissions from the mine did not increase above 50 $\mu g/m^3$ which would be equivalent to limiting the increase in 24-hour average $PM_{2.5}$ concentrations to 6 $\mu g/m^3$. The associated increase in mortality risk for an individual would then be 1 in 9,260,000.

Table 5 summarises the risks for the population of Ulan in for the worst-day and in the worst year in the life of the mine based on the assumption that the mine does not practice real-time management of dust to minimise the effects on the Village. The estimates are based on the assumption that the population of Ulan Village is 140. The worst-day's $PM_{2.5}$ concentration due to emissions from the mine is taken to be 9.8 $\mu g/m^3$ (i.e. the same as predicted at Receptor 160a (the school see **Figure 1**) and the highest annual average $PM_{2.5}$ concentration due to the mine is 1.3 $\mu g/m^3$.

Table 5. Estimated increase in risk of indicated event for the worst-day and worst year

Event	Frequency of occurrence	Additional risk per 10 μg/m³ increase in $PM_{2.5}$	Additional risk due to exposure to particulate emissions from Moolarben Mine
	Per 100,000	(%)	At R160a taken to represent Ulan Village
			No of extra deaths or admissions to hospital
Daily mortality	2	0.9	0.000025
Annual mortality	724	6	0.0079
Daily hospital admissions for childhood asthma	1	11.3.	0.000155

It is important to recognise that the figures in the table above relate to the worst day in the life of the project and the worst year in the life of the project. Not the average day or average year. These results highlight the difficulty in applying risk assessments of this type to "transient" activities" such as mining. The increase in risk of the indicated event for a particular day has some validity but the impacts of the mine will change significantly over a period of a year or so and hence the risks based on annual average concentrations is hypothetical rather than real.

5. SUMMARY AND CONCLUSIONS

The impacts of the proposed Moolarben Mine 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.

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 the life of the mine. In addition estimates are provided on the increase in daily and annual mortality and daily and annual hospital admissions that could be expected from the residents in Ulan Village due to emissions from the project on the worst day and in the worst year.

The increase in number of deaths in Ulan on the worst day in the life of the mine is estimated to be 0.000025 and the increase in hospital admissions is estimated to be 0.000155.

The number of deaths in the worst year of the project is estimated to increase by 0.0079.

6. 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

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

Health Effects Institute (2003)
"Revised analyses of time-series studies of air pollution and health" Special Report May 2003

Holmes Air Sciences (2006)

"Air quality impact assessment – Moolarben Coal Mine" prepared for Wells Environmental Services, April 2006

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 2002; 287: 1132041

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)

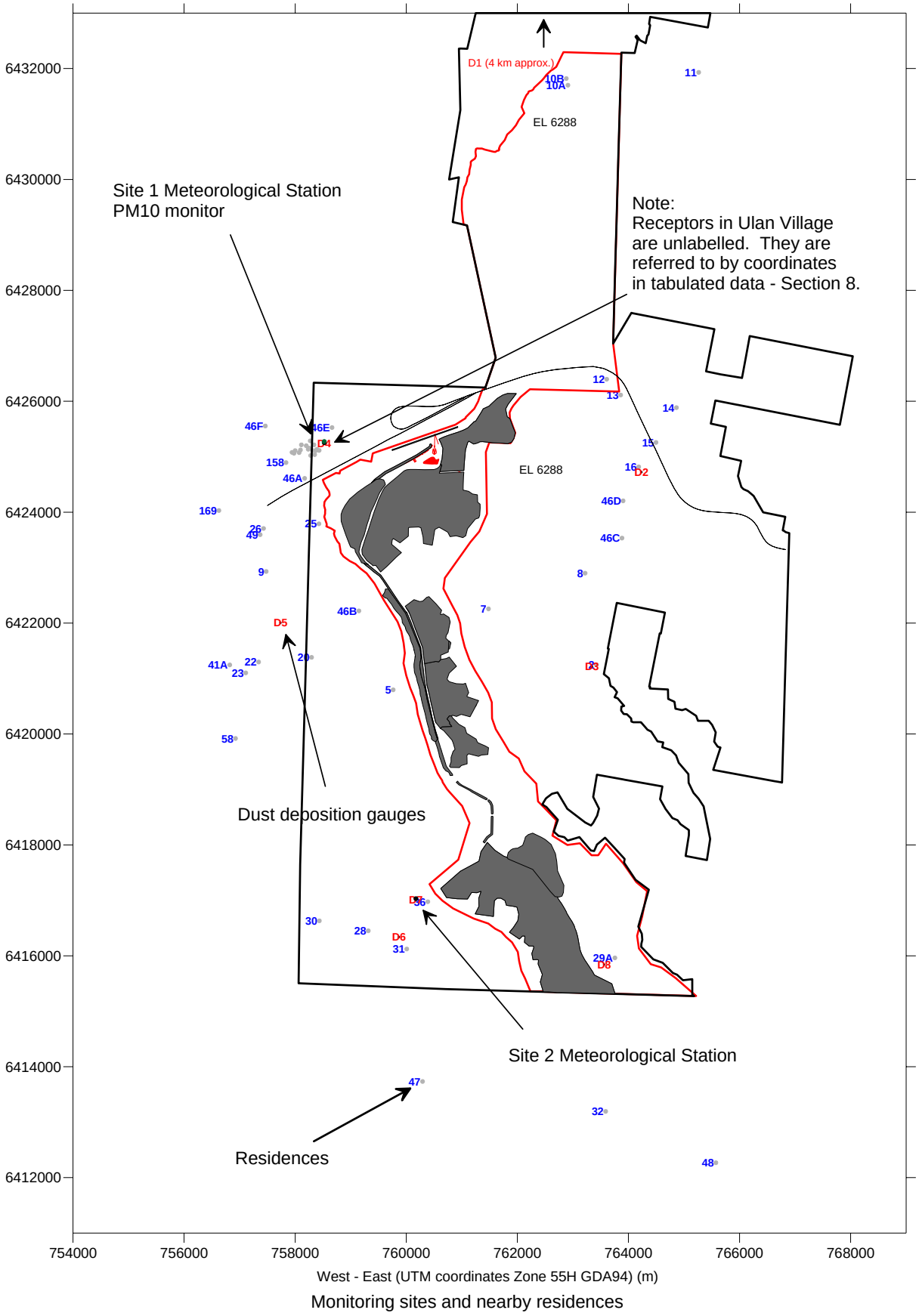
Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G, Neller A (2005a)
"The short-term effects of air pollution on daily mortality in four Australian cities" Aust N Z J Public Health 2005; 29: 205-12

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

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).

FIGURES



Monitoring sites and nearby residences

FIGURE 1