Appendix J

Health Impact Assessment Report

HEALTH IMPACTS OF THE PROPOSED HUME COAL PROJECT

Introduction

The Hume Coal Project (the project) will develop and operate an underground coal mine (the project) and associated mine infrastructure in the Southern Coalfield of New South Wales (NSW). Dr David McKenzie was engaged to review the air quality and the noise and vibration assessments and undertake a health risk assessment for the surrounding population. The risk assessment addresses likely impacts on community health from exposure to air emissions and noise from the project, considering the direct health effects from acute (short-term) and chronic (long-term) exposures.

This report summarises the epidemiological literature concerning associations between exposure to airborne pollution and ambient noise and various health outcomes. The literature on health effects of coal mining is also reviewed. The health risk assessment is presented in detail.

Airborne pollution: dust and particulates

People have inhaled suspended particulates throughout evolution, such that normal lungs are well adapted to remove most forms of foreign matter using a range of defence mechanisms, including the mucus lining the airways and special scavenger cells in the air sacs (or alveoli).

The potential for inhaled particulates to cause harm depends on: the chemical and physical nature of the particles; the concentration of the particles in the air; the percentage of particulate matter which is in the respirable size range of <10 microns (μ) in diameter, also known as PM_{10}, (in other words small enough to be inhaled into the lung and large enough to be deposited into the lung rather than to be exhaled); and the individual susceptibility of the person inhaling the dust.

Silica

Most of the dust that people inhale is harmless and sometimes referred to as 'nuisance' dust. Asbestos fibres and very fine particles of crystalline silica, on the other hand, may produce severe fibrotic reactions in the lungs. Fibrosis occurs as part of the repair process following injury or inflammation and connective tissue forms in an organ or tissue, resulting in scarring or thickening of the affected tissue.

Silica is contained in quartz, sand and many rocks, such as granite, which may be present in the overburden removed before open-cut coal mining begins, and to reach coal seams in underground mines. Atmospheric levels of silica have been measured in micrograms per cubic metre (μg/m³) near two open-cut mines in the Hunter Valley and the worst-case values (0.5–1.8 μg/m³) were well below the environmental standard for an annual average of 3.0 μg/m³ (ACARP Study C18026). Underground coal mining releases less silica into the atmosphere of the surrounding area than open-cut mining. Generally, most silica is removed from coal by processing and washing before it is transported from a mine.

PM_{10} in airborne dust varies in composition

Inhaled dust also contains numerous other substances usually in very small quantities. The main concern in heavily populated urban areas is particles from the exhaust systems of vehicles, especially diesel trucks and buses and other processes that combust hydrocarbon fuels, such as power stations, heavy industries and jet aircraft. In rural communities, the dust may contain particles from wood fire smoke and dust released from ploughing, vehicles moving along unsealed roads and harvesting or processing various grains such as wheat. There are also variable concentrations of pollens and spores of fungi. In coastal areas sea salt spray makes up a large part of respirable airborne particles.

The PM_{10} fraction of airborne dust thus varies markedly in composition from place to place. It also varies with the temperature, humidity, weather (including rain, wind speed and direction), and the time of year. The composition of fine airborne dust particles and pollution collected in an urban area such as Sydney would be different from the dust particles collected in a rural area such as the Hunter Valley. In rural areas it would vary with season, the type of farming and any other variable such as an open-cut coal mine, power station or smelter in the district. One major difference is that the bulk of PM_{10} in urban areas is in the $PM_{2.5}$ fraction whereas in rural areas the majority of the particles are larger than 2.5 µg. However, the air quality in Australian cities is better than in most other cities of similar size, particularly in relation to the levels of $PM_{2.5}$ (see below).

Effects of exposure to PM_{10} and $PM_{2.5}$ on human health

Numerous studies have reported increased risks of death from long-term exposure to urban pollution. A variety of markers and components of urban pollution have been studied, including PM_{10}, $PM_{2.5}$, black smoke, oxides of nitrogen and sulphur, and ozone. Arguably the best studies have followed cohorts, or groups, of individuals who had baseline data collected for up to 25 years. Increased long-term and short-term exposure to PM_{10} and $PM_{2.5}$ are correlated with deaths and hospitalisations due to heart and lung disease and also with asthma. However, there remains some uncertainty and conflict within the published literature, most of which is based on studies in urban settings in the US and Europe. Suspended particulates and respirable particles in rural settings are likely to be less harmful than those in urban areas, which have a much larger fraction of products of combustion.

Evidence suggests the health effects of airborne particles are mainly due to the $PM_{2.5}$ fraction, which includes the bulk of the products of combustion.

Long-term exposures

The PAARC survey from France (Filleul et al. 2005) enrolled over 14,000 adults in 1974, obtained baseline data and followed them for 25 years recording the cause of any deaths that occurred during this time. The authors also examined the levels of black smoke in the area where people were living at enrolment with annual mean (average) levels between 1974 and 1976 in different districts ranging from 21 to 152 µg/m³. Black smoke is most strongly related to traffic emissions, particularly diesel trucks, and is mainly within the $PM_{2.5}$ fraction. By the end of the study the levels had fallen to 10 to 14 µg/m³. The main result of the study was that for every 10 µg/m³ increase in black smoke there was a 1.07 increase in the adjusted risk ratio for non-accidental mortality after adjusting for varying confounders such as smoking, occupation and educational level. This means that the risk of death increased by 7% for those with higher exposures. The increased risks may have reflected the high levels at or before the study began. It is not clear whether it is valid to extrapolate results such as these to an Australian setting where air pollution concentrations are much lower, that is with annual average $PM_{2.5}$ levels around 6 µg/ m³.

Pope et al. (2002) reported on 1.2 million adults recruited in 1982 in the United States. They were able to examine the data for 500,000 and relate this to levels of air pollution at their homes at the beginning of the study and to their subsequent cause of death up until 1998. The mean, or average, level of $PM_{2.5}$ was 21.1 µg/m³ at the beginning of the study and 14.0 µg/m³ at the end (that is two to three times the Australian standard and three to four times the typical Australian levels). They found that for every 10 µg/m³ increase in $PM_{2.5}$ there was a 4% increase in all-cause mortality, a 6% increase in cardiopulmonary mortality and 8% increase in lung cancer. Importantly, the authors found no increased risk of mortality associated with either total suspended particulates or with the coarse fraction of PM_{10}, ie PM2.5–10. A number of other studies have also concluded that risk of death from airborne pollution is largely related to the $PM_{2.5}$ fraction (for review see Pope & Dockery 2006). The bulk of the dust from coal mining operations is in the coarse fraction.

A World Health Organisation Working Group (2003) reviewed a large amount of data and literature, most of which came from Europe, USA and Canada, and concluded that long-term exposure to airborne particulate matter (particularly $PM_{2.5}$) led to a reduction in life expectancy from cardiopulmonary conditions and lung cancer as well as increases in respiratory symptoms and reduced lung function. Life expectancy could be reduced by as much as three years, with a possible contribution to increased infant mortality in the most heavily polluted regions. The adults at greatest risk have been shown to be those who are elderly, are poor (of low socio-economic status), smokers, workers in industrial occupations and those who have pre-existing cardio-respiratory disease.

Short-term exposures

Many publications have linked PM concentrations averaged over 24 hours with various health outcomes over the ensuing several days, including daily mortality and hospital admissions due to all causes and cardio-respiratory illnesses. Recent studies have used more reliable methodologies including time-series analysis. The Health Effects Institute study (Katsouyanni et al. 2009) used the most rigorous contemporary methods and, because it was based on data from two continents (Europe and North America) involving large numbers of subjects, it provided the strongest available evidence concerning the short-term effects of PM_{10}. $PM_{2.5}$ was not measured, but it is probable that the effects attributed to PM_{10} were due to the $PM_{2.5}$ fraction.

In the European contribution to the study, 22 cities were examined with populations ranging from 200,000 to 7,000,000. The annual average PM_{10} values ranged from 13 to 65 µg/m³ with an average of 40 µg/m³. In the US there were 15 cities with daily PM data with populations ranging from 250,000 to 9 million. The annual average PM_{10} ranged from 14 to 43 µg/m³ with an average of 28 µg/m³. Canada had 12 cities with populations ranging from 100,000 to 2,000,000 and annual average PM_{10} values ranging from 11 to 28 µg/m³ with an average of 20 µg/m³. The overall average for Australia is about 15 µg/m³.

The results from Europe and the US were similar while the results from Canada were variable. For a 10 µg/m³ increase in 24-hour PM_{10} there was a significant increase in all-cause mortality and cardiovascular mortality in all three regions with the greatest effects seen in Canada. The size of the effect was about 0.2% for all-cause mortality and 0.4% for cardiovascular mortality. The effect on respiratory mortality was inconsistent with a -1.4% risk (that is, reduced mortality, but not statistically significant) found in the Canadian data.

The results for hospital admissions were also inconsistent. For cardiovascular admissions there was an increased risk of 0.6 to 0.7% in Europe and the US but not in Canada. The increased risk for respiratory admissions related to a 10 µg/m³ increase in PM_{10} was small and mostly insignificant.

This was a major study involving many millions of subjects on two continents. It illustrates how the effects of pollution on mortality and hospital admissions are very small and very large subject samples are required to obtain significant results. The inconsistent results obtained from the Canadian data (such as the inability to document an increased risk of cardiovascular admissions to hospital in spite of showing an increased risk of cardiovascular mortality and the negative effect for respiratory mortality) were attributed to the relatively small populations studied in Canada and that the pollution levels were relatively low in Canada compared with Europe and the United States. The failure to show an increased risk of respiratory admissions to hospital was also unexpected given the excellent methodology and statistical power of the study. PM_{10} values in Australian cities are similar to or less than those quoted for Canada.

The weather is also a factor

The mechanisms whereby an increase in PM_{10} or $PM_{2.5}$ or other components of airborne pollution can lead to more cardio-respiratory illness or death on that day or the days following an increase are not understood (for example, see WHO 2003). It is claimed that some components of urban pollution, particularly the fine ($PM_{2.5}$) and, possibly, ultrafine particles ($PM_{1.0}$), contribute to inflammation in blood vessels and airways. Some individuals are likely to be more susceptible than others and these may include those with advanced pre-existing cardio-pulmonary disease. It is likely the excess mortality (premature death) is among the very frail and their deaths may simply be advanced by weeks or months.

High pollution days tend to occur during heatwaves with inversions and during cold snaps. Temperature extremes are associated with increased mortality and hospital admissions for reasons other than airborne pollution, including heat stress, hypothermia and respiratory infections. During cold snaps many people are exposed to increased indoor air pollution as a result of burning wood or using gas or oil heating. Epidemiological studies use various methods in an attempt to control for these variables but it is often difficult to control for all confounders, some of which may be unknown.

Relevance to Australia

Whether these studies would apply to Australian populations is questionable because of the much lower density of population, and therefore less road traffic, in Australian cities, and the marked differences in weather patterns. Australian cities are among the cleanest in the world in relation to airborne pollution, particularly $PM_{2.5}$ (Bennett et al. 2007; NSW EPA 2013; WHO 2016). Australia is also a relatively dry continent and the PM_{10} contains a relatively high proportion of non-industrial dust and pollens. The commonest causes for increases in PM_{10} above the criterion of 50 µg/m³ in Australian towns and cities are dust storms and bushfires or controlled vegetation back-burns (discussed below).

High pollution days in Australian cities are usually associated with high temperatures, which place additional stresses on the frail elderly and infants. Li et al. (2012) showed the increase in mortality in Brisbane associated with a 10 µg/m³ increase in PM_{10} was higher (0.218%) with high temperature than low temperatures (0.062%) and the temperature effect was more marked in Brisbane than comparable figures in Beijing even though the pollution levels in Beijing were much higher than those in Brisbane. These results suggest an independent effect of high temperature on pollution or that the pollutants may differ in toxicity with high ambient temperature. In rural areas, high pollution days occur more often in winter during cold snaps due to burning wood so the risks may be lower than in urban settings.

The actual risk associated with a 10 µg/m³ increase in PM_{10} can be calculated using various assumptions. For example, if the average death rate is 500/100,000 a 0.2% increase in mortality equates to one extra death in 100,000 people each year.

Do the National Environment Protection Measure standards for PM_{10} and $PM_{2.5}$ protect health?

The National Environment Protection Measure (NEPM) standards for air quality reflect the medical literature and are set in response to health, social, environmental and economic impacts as well as feasibility (that is, not just health alone). They are further informed by cost-benefit analyses.

The current US standard for PM_{10} is a 24-hour average of 150 µg/m³ allowing one exceedance a year averaged over three years. The Australian 24-hour standard for PM_{10} at 50 µg/m³ is well below the US standard and is equal to the EU and World Health Organisation (WHO) guidelines. The standard allows five exceedances a year to account for dust storms and bushfires. In 2006, the USA EPA removed the then standard for an annual average PM_{10} of 50 µg/m³ because the authors concluded that $PM_{2.5}$ had been shown to be the component of concern for health and that 'available evidence does not show a definitive link between long-term exposure to current PM_{10} levels and health problems'. Australia retains an annual average standard for PM_{10} of 25 µg/m³, which is below the former US standard. Many countries set regulatory limits that allow greater emissions because that is all that can be achieved. Australian cities have better air quality than most others.

The Australian NEPM standards for $PM_{2.5}$ are a 24-hour average of 25 µg/m³ and an annual average of 8 µg/m³. These values are conservative compared with other developed nations. The US EPA standard is a 24-hour average of 35 µg/m³. There are two annual average standards: the secondary standard is 15 µg/m³ and the primary standard is 12 µg/m³, recently introduced to protect sensitive individuals such as asthmatics, children and the elderly. The EU standard is even higher, with an annual mean of 25 µg/m³. Between 2009 and 2011, only four EU countries had annual mean $PM_{2.5}$ levels below 10 µg/m³ (Finland, Sweden, Estonia and Portugal). The levels were greater than 20 µg/m³ in nine countries and greater than 25 µg/m³ in two countries. The WHO aspirational goal, or guideline, is an annual standard of 10 µg/m³, which is recommended for nations to work towards. The WHO has set three interim targets for annual mean $PM_{2.5}$ levels decreasing from 35 to 15 µg/m³. Thus, the Australian advisory standard of 8 µg/m³ is lower than even the WHO aspirational guideline.

Is there a threshold level for PM below which there are no health impacts?

Individuals vary in their susceptibility to the effects of air pollutants. There may be some highly susceptible individuals who will experience adverse effects at levels within the standards. It is likely the relationship between PM levels and health effects flattens at high and at low levels. For example, there is evidence the exposure response relationship at very high levels (bushfires, dust storms) is not linear (that is it flattens). It is also likely the response relationship flattens at low exposure levels (that is a threshold for effects exists) but most studies have not measured the effects of low levels such as those likely to be experienced in Australia and near the project.

The question whether there is a threshold level below which air pollution does not cause any discernible health impacts has been discussed widely in the medical literature. There is general agreement the current data does not provide evidence for a threshold. There is also agreement that the studies done to date were not designed or were inadequate to detect a threshold. WHO (2003) states 'if there is a threshold, it is within the lower band of currently observed PM concentrations in Europe'.

WHO studies

The WHO (2005) update suggested an annual average guideline value of 10 µg/m³ for $PM_{2.5}$ because this represented the lower end of the range of levels to which populations studied in the epidemiological literature had been exposed to and which had shown significant effects (10 to 35 µg/m³). In other words, health outcomes were compared between populations exposed over long periods to varying levels of $PM_{2.5}$ greater than 10 µg/m³ and increases above that level were found to have adverse effects. The authors stated that, although thresholds were not apparent, there was statistical uncertainty in risk estimates in the American Cancer Society study (Pope et al. 2004) at concentrations of around 13 µg/m³ (that is at the lower end of the range of levels studied). The authors also stated that, in the Harvard six cities study (Dockery et al. 1993) the health risks were similar in the two cities with the lowest long-term $PM_{2.5}$ concentrations of 11.0 and 12.5 µg/m³ but were higher in the next city with an average concentration of 14.9 µg/m³. This might suggest a levelling off of risk of health impacts at around 10 µg/m³. Therefore, the authors concluded a guideline for $PM_{2.5}$ of 10 µg/m³ 'would be below the mean of the most likely effects levels indicated in the available literature'.

The same WHO (2005) update concluded that a guideline limit for $PM_{2.5}$ annual average of 10 µg/m³ would also be applicable to the studies of short-term exposure, which were daily exposure time series studies examining relationships between $PM_{2.5}$ and acute adverse health outcomes. These studies were on populations exposed to long-term averages ranging from 13 to 18 µg/m³. The authors stated that 'although adverse effects on health cannot be entirely ruled out even below those levels, the annual average WHO AQG (air quality guideline) represent levels that have been shown to be achievable in large urban areas in highly developed countries, and attainment is expected to effectively reduce the health risks'.

WHO (2013) reviewed a range of studies with particular emphasis on cardiovascular mortality in an effort to determine the level of minimal or no risk from $PM_{2.5}$. The authors found no evidence for an adverse effect at the lowest levels but felt there was insufficient data to define a threshold.[1] It is generally believed the level of $PM_{2.5}$ could never approach zero. Estimates of a baseline or background level of $PM_{2.5}$ with no industrial activity range between 2 and 5 µg/m³. Levels of $PM_{2.5}$ at Cape Grim in western Tasmania are higher than this and yet it is regarded as a benchmark for clean air. Some individuals may be extremely susceptible to certain particulates.

For example, an asthmatic may react to a very low concentration of pollen granules. Some individuals may be extremely susceptible to the carcinogenic effects of the products of combustion, particularly from diesel fuel.

Another way of looking at this is to consider a range of thresholds for different groups within the population. The threshold for certain pollutants may be different between adults and children and between healthy individuals and those with chronic disease and frail, elderly individuals. It is possible, if not probable, that there is a threshold for health effects at a level of $PM_{2.5}$ around 8 µg/m³ that will apply to a very large proportion of the population. A small number of individuals might remain susceptible at levels below the standard and it may never be possible to reach a level that would completely protect them.

WHO (2003) stated that 'if there is a threshold, it is within the lower band of currently observed PM concentrations in Europe'. Levels of $PM_{2.5}$ in Australia, including the coal mining areas of the Hunter Valley, and in the Southern Tablelands where the project is located, are lower than most cities and regions in Europe and therefore would be within or below the EU 'lower band'. If there is a threshold it would mean the low levels experienced in Australian rural settings would have no discernible health impacts.

Bennett et al. (2007) studied the effect of $PM_{2.5}$ in Melbourne and found no significant association with most respiratory symptoms. Paradoxically they found a 'protective' effect with 'cough' and 'cough with phlegm' on higher pollution days. Part of their conclusion was that 'ambient $PM_{2.5}$ in Melbourne may currently be too low to have a detectable effect on respiratory symptoms'.

[1] The lack of reliable data for low levels of exposure was observed. The conclusion was: 'there is clearly no evidence of an association below the levels observed, and it is impractical to estimate shape of the curve at the extremes of the exposure distribution'. Based on a range of assumptions, it was suggested that, taking into account the scatter of data points, the minimal level was 5.8 µg/m³ and the 5th percentile 8.8 µg/m³ with a midpoint of 7.3 µg/m³. These results appear to confirm the setting of air quality guidelines for $PM_{2.5}$ at an annual average of 8 µg/m³, and to justify some recent recommendations to lower the standard in the future to 7 µg/m³.

Air quality in Australia

Recently, WHO (2016) stated that, in 2014, more than 80% of people living in urban areas that monitor air pollution were exposed to air quality levels that exceed the WHO guideline for annual average $PM_{2.5}$ of 10 µg/m³ (98% of cities in low and middle income countries and 56% of cities in high income countries). The database covers 3000 cities in 103 countries. The global annual average $PM_{2.5}$ for these cities was 38.4 µg/m³ with the range from less than 10 µg/m³ to well over 100 µg/m³. Australian cities were among the cleanest in the world with an annual average of 5.8 µg/m³ in 2014.

During the infamous east coast dust storm of 23 September 2009, 24-hour PM_{10} levels in Sydney reached 11,000 µg/m³ and 24-hour $PM_{2.5}$ reached 1,600 µg/m³ (60 times the NEPM standard of 8.0 µg/m³). Emergency services reported an increase in complaints of breathing difficulties. Children, the elderly and those with heart and lung conditions were advised to remain indoors. Merrifield et al. (2013) reported a 23% increase in emergency department visits for asthma and a 20% increase in respiratory visits. There was no significant increase in cardiovascular visits to hospital emergency departments or hospital admissions generally for any reason. Contrary to expectations, there was a 5% decrease in the risk of admission to hospital for respiratory illness. The authors stated that: 'While the dust storm was associated with a higher risk of acute respiratory problems, these may not have been severe enough to warrant admission to hospital.' A similar result was obtained by Barnett et al. (2012) who studied the effect of the same dust storm on emergency admissions to hospitals in Brisbane. There was a 39% increase in emergency presentations associated with the storm. However, there was no significant change in the characteristics of admissions during the storm and, specifically, there was no increase in respiratory admissions. The authors concluded that either the public took effective avoidance measures or that 'the dust was simply not toxic, being composed mainly of soil'. The authors quoted studies from Cyprus and Taiwan showing similar results with dust storms they had experienced.

When bushfires occur around Sydney PM_{10} levels can rise to between 100 and 500 µg/m³ (that is up to 10 times the 24-hour criterion). Morgan et al. (2010) looked for an association between increases in PM_{10} levels during major bushfire events around Sydney and daily mortality and hospital admissions between 1994 and 2002. Increases in PM_{10} levels due to bushfires were consistently associated with an overall increase in admissions for respiratory complaints of 1.24% increasing to 4–5% for elderly people with chronic obstructive pulmonary disease (COPD) and for adult asthma. In contrast to the effects of urban pollution there was no increase in cardiovascular admissions or in mortality.

These studies provide reasonable evidence that even extremely high levels of PM_{10} and $PM_{2.5}$ derived from ordinary dust from the earth's crust do not cause serious health impacts or a discernible increase in mortality in cities the size of Sydney and Brisbane. Crustal elements dominate the emissions from coal mining. Very high levels of PM_{10} and $PM_{2.5}$ from bushfires do produce measurable impacts on respiratory admissions but the effects are not as high as would be expected from studies of urban pollution (reviewed above).

The fraction of $PM_{2.5}$ contained within the PM_{10} varies considerably between locations depending on the sources of the dust. In densely populated US and EU urban areas the $PM_{2.5}/PM_{10}$ fraction is around 0.8 whereas in rural areas and developing nations it is usually below 0.5. This is because there are fewer products of combustion and more dust, pollen, salt spray and similar sources in rural areas.

The Upper Hunter Fine Particle Characterisation Study focused on $PM_{2.5}$ levels at two sites – Singleton and Muswellbrook – two major Hunter towns close to mines and power stations. It was commissioned by the NSW EPA and undertaken by the CSIRO and ANSTO. It found secondary sulphate (~20%, from sources such as power stations) and wood smoke (~30%, primarily from residential wood heaters) to be the largest contributing factors to $PM_{2.5}$ levels in Muswellbrook and Singleton respectively. Soil, which includes fugitive coal dust, accounted for 10–14% across the Upper Hunter (Hibberd et al. 2013).

Air quality at the Hume Coal Project

Air quality at the project is being monitored at sensitive receptors and other locations nearby. There are several potential sources of air pollution nearby including cement works, stock feed manufacture, Dux manufacturing, rail operations and sewage works. The EIS provides readings from particle monitors operated by Hume Coal and Boral Cement Works as well as several NSW OEH monitoring stations at Bargo, Camden and an ACT monitor at Monash. Between January 2010 and December 2014, most of the 24-hour values of PM_{10} were less than 50 µg/m³ and the majority were less than 30 µg/m³. The majority of the Hume Coal readings were below 20 µg/m³ (see EIS Appendix L Figure 5.3). The annual average for the monitors was in the range of 10 to 20 µg/ m³. The annual average for the Hume Coal monitor was 14.3 µg/m³. It can be seen from Figure 5.2 in the Air Quality Assessment that the recorded 24-hour average PM_{10} concentrations at all locations fluctuated throughout the period presented. Exceedances of the 24-hour average NSW EPA assessment criterion (50 µg/m³) occurred at all monitoring locations. The most notable grouping of recorded criteria exceedance was in late October/early November 2013, which was a period of extensive bushfires across NSW.

Monitoring $PM_{2.5}$ over the same period showed occasional exceedances of the 24-hour criterion of 25 µg/ m³ at all the monitors (Figure 5.5 in the Air Quality Assessment) but the only exceedances for the project area were during the bushfires of October 2013. Most of the Hume Coal measurements were below 10 µg/ m³, with an average of 6.3 µg/ m³ (Figure 5.7 in the Air Quality Assessment). Dust deposition values were well below criteria at all monitoring locations.

Thus the air quality near the proposed coal mine, in terms of respirable particulate matter, has been similar to or better than that in the rest of the Southern Highlands. Burning wood and other biomass fuels is a significant contributor to $PM_{2.5}$ levels, especially in winter and around larger population centres. This is a common problem in rural towns.

By way of contrast, the air quality in Tasmania is generally regarded among the best in the world. However, there have been concerns about air quality in Launceston and other towns largely due to burning wood for domestic heating, which contributes between 30% and 75% of the PM_{10} depending on the season. Other main contributors were dust from roads, especially unpaved roads, and motor vehicles with industrial facilities contributing 7%.

Between 1997 and 2005, there was a gradual improvement with exceedances of the NEPM standard of 50 µg/m³ decreasing from 51 to 14 a year (Department of Tourism, Arts and the Environment 2006). Even at Cape Grim at the north-western tip of Tasmania PM_{10} levels regularly exceeded 30 µg/m³ between 2007 and 2011 and exceeded 50 µg/m³ when forest fire smoke was blown in its direction. The 24-hour $PM_{2.5}$ levels were less than 10 µg/m³ except during a forest fire in 2008. Sea salt is a significant contributor to PM_{10} and $PM_{2.5}$ levels at Cape Grim because the monitor is on the coast facing the prevailing strong westerly winds from the Southern Ocean.

Hume Coal Project Air Quality Assessment: PM_{10} and $PM_{2.5}$

The air quality predictions for the project are well within the NEPM standards. For example, Table 9.1 in the Air Quality Assessment gives predicted incremental values for $PM_{2.5}$ and PM_{10} resulting from the project during construction and operations. During operations two levels of dust mitigation are modelled. The 24-hour average incremental values of $PM_{2.5}$ at the sensitive receptors are predicted to increase by 1.6–1.9 µg/m³ (NEPM standard 25 µg/m³) while the annual average values are predicted to increase by 0.2–0.3 µg/m³ (NEPM standard 8 µg/m³). As these predictions relate to receptors close to the mine area, the increments at towns further away would be lower. The 24-hour average incremental values for PM_{10} ranged from 2.1–4.6 µg/m³ (NEPM standard 50 µg/m³) while the annual average values are predicted to increase by 0.3–0.9 µg/m³ (NEPM standard 30 µg/m³). The higher values are for the construction phase due to transport on dirt roads and other works above ground. Such activities have less effect on $PM_{2.5}$ levels in the atmosphere.

Figures 9.7, 9.8 and Table 9.3 in the Air Quality Assessment give predicted maximal (that is highest) cumulative values for $PM_{2.5}$ and PM_{10} resulting from the project added to existing and predicted background levels and contributions from other sources of pollution during the construction phase and during operations. During operations, two levels of dust impact are modelled. The 24-hour average values of $PM_{2.5}$ at the sensitive receptors are predicted to mostly be below 10 µg/m³ (NEPM standard 25 µg/m³) with less than two exceedances per year while the annual average values are predicted to range from 6.8–6.9 µg/m³ (NEPM standard 8 µg/m³). As these predictions relate to receptors close to the mine area, the levels at towns further away would be lower. The exceedances are likely to relate to dust storms or fires rather than mine activity. The 24-hour average values for PM_{10} are predicted to mostly be below 20 µg/m³ (NEPM standard 50 µg/m³) while the annual average values are predicted to range from 17.4–17.5 µg/m³ (NEPM standard 30 µg/m³). Exceedances are predicted to occur less than one day per annum.

For each sensitive receptor location the contribution to cumulative annual PM_{10} and $PM_{2.5}$ concentrations, relative to neighbouring sources and ambient background levels, is given in figures 9-1, 9-2, 9-3, 9-4, 9-5 and 9-6 in the Air Quality Assessment. The predicted annual average PM_{10} and $PM_{2.5}$ concentrations from the project are low relative to the combination of existing neighbouring emission sources and ambient baseline levels at all selected receptor locations.

Gaseous pollutants

Maximum concentrations of nitrogen dioxide (NO_2) and volatile organic compounds (VOCs) are presented in Table 9-4 in the Air Quality Assessment for project only and other sources. The maximum cumulative predicted concentrations of NO_2 and VOCs are also presented (Hume Coal + neighbouring emissions sources + ambient background) across all selected sensitive receptor locations for the peak operations scenarios.

The data in Table 9-4 shows that all predicted concentrations are well below applicable air quality impact assessment criteria. In addition, the methodology for deriving NO_2 concentrations from predicted NO_x concentrations is regarded as conservative. That is, the actual levels are likely to be lower than those predicted. **Likely impact of the Hume Coal Project on the health of nearby residents**

The air quality modelling detailed above for the construction and operational phases of the project indicates predicted maximum and average concentrations and deposition rates of particulate matter, diesel combustion and sources of odour such as volatile organic compounds are well below applicable air quality impact assessment criteria, and small relative to existing ambient background conditions. The construction phase will generate higher impacts than during operations due to a greater proportion of surface-based material handling, and truck transportation, with the main impact being greater generation of crustal sources of PM_{10}.

When incremental concentrations from the project are combined with concentrations from neighbouring emission sources, the combined concentrations are well below applicable regulatory criteria. Predicted cumulative impacts, including contributions from the project and neighbouring emission sources added to ambient background levels, are unlikely to exceed applicable NSW EPA regulatory criteria. Exceedances are only likely to occur because of external and natural phenomena such as fires and dust storms.

The project's operation is predicted to result in a small increase in the levels of PM_{10} and a smaller increase in $PM_{2.5}$ in the area. Mining operations will have a small impact on $PM_{2.5}$ levels. The main source of $PM_{2.5}$ would be diesel equipment and machinery, which are present in relatively small numbers. Diesel exhaust fumes disperse rapidly in the atmosphere. Studies of school children and residents living near major roads in Europe suggest that a localised increase in exhaust emissions can be detected for about 200 m (Van Vliet et al. 1997). Emissions from diesel engines have been included in the predictions of particulate matter (PM) in the Air Quality Assessment. Any increase in PM levels from the mining operations will not exceed the NEPM $PM_{2.5}$ standard and the predicted levels of $PM_{2.5}$ are very low by world standards. Nevertheless, any increase in PM levels may result in a small increase in the statistical risk of health effects. Due to the small population potentially exposed, it would not be possible to measure health effects resulting from the predicted increase in atmospheric particles but it is possible to make some predictions (see Health Risk Assessment below).

Air quality impacts from blasting.
Concerns about fumes from blasting are mostly in relation to risks to underground mine workers where ventilation may be poor and elevated levels of oxides of nitrogen and sulphur may persist for prolonged periods. In the open, fumes from blasting are transient and dissipate rapidly (within 5–10 minutes). A range of procedures are in place so that blasts do no take place when high moisture could be present or when conditions are windy. The explosives are designed to reduce the possibility of absorption of moisture to avoid incomplete combustion and generating oxides of nitrogen and sulphur. If a blast is efficiently conducted (ie right mixture and conditions), it produces little in the way of fumes and gases such as nitrogen dioxide (NO_2) and CO. Table 9.4 in the Air Quality Assessment indicates that *peak* levels of NO_2 will be below the regulatory standards at all receptors and the contribution from the project will be small compared with ambient and other sources in the area. Therefore, there is no risk of adverse health consequences for residents from fumes from blasting.

Impact of fugitive emissions from coal trains along the rail corridor
A number of studies have measured dust levels, generated with the passage of coal and other goods and passenger trains, at a distance equivalent to or less than the nearest homes or schools (usually 15 or 20 m). Although there may be some brief periods of high dust levels, the 24-hour averages are well within the air quality standards.

The Australian Rail Track Corporation (ARTC) quantified fine particulates generated from rail transport including coal trains in the Newcastle area rail corridor (September 2012). Monitors were set up 3 m from the tracks in Metford and Mayfield. While there were some differences between the two sites, passenger trains produced the highest peak levels of total suspended particles (TSP), PM_{10} and $PM_{2.5}$ while the mean levels for PM_{10} and $PM_{2.5}$ were higher for coal and freight trains, with no significant difference between loaded and unloaded coal trains or between coal trains and other freight trains.

The differences between the types of train were small and thought to be due to speed (higher for passenger trains) and duration of passing (longer with coal and freight trains). The background (no train) levels were similar to those at other monitoring stations in the Hunter region. The transient increases in airborne particles with trains ranged from 2.2–4.8 µg/m³ for PM_{10} and 0.5–1.2 µg/m³ for $PM_{2.5}$ respectively for Mayfield and Metford. Most of the dust was entrained from the ground rather than fugitive from the trains, and only a small percentage was coal dust even when the dust appeared black.

The Queensland Department of Science, Information Technology, Innovation and the Arts (Neale D et al. 2013) monitored dust levels at six locations along the rail corridors of coal trains from the west to the Port of Brisbane in 2013 and produced similar results to the NSW study.

The 24-hour mean levels of PM_{10} and $PM_{2.5}$ did not exceed the Queensland EP(A)P criteria on any day with the highest $PM_{2.5}$ value less than the annual objective of 8 µg/m³. The levels were similar to those elsewhere in the region and related to regional emissions rather than the trains. The levels did not seem to be influenced by the number of trains each day, which varied considerably.

The Queensland Department of Health concluded there were unlikely to be any additional adverse health effects from dust particles for people living along the rail corridor. A similar conclusion was reached in the NSW report.

The coal trains from the project will be covered so the fugitive emissions will be less than those reported above from uncovered wagons.

Health effects of coal mining

Most concerns about potential population health effects from coal mining have been expressed in relation to open-cut mining rather than underground mining. In underground mines the health concerns are mainly related to the miners who are potentially exposed to high concentrations of dust and fumes due to the relatively small volume of air for dispersion and limited ventilation. In open-cut coal mining the major source of dust is from removing topsoil and overburden and the transport of this material. A large fraction of the dust is inert 'nuisance' dust, which is either composed of particles too large to be inhaled deep into the lungs or is non-fibrogenic dust (ie low concentration of crystalline silica). Coal dust typically contributes about 10% to the PM_{10} fraction of the dust (EDO 2010). In underground mines a proportion of airborne PM_{10} and $PM_{2.5}$ will settle below ground. Overall, much less dust is generated because there is limited overburden material being removed. Thus the main potential sources for PM_{10} and $PM_{2.5}$ for surrounding receptors are the ventilation shafts and the above ground operations.

There are occupational health standards (such as the Australian Safety and Compensation Council, ASCC, which have been adopted by most states) that must be complied with for workers exposed to various kinds of dust. The allowable level for inhalable dust in all coal operations is 10 mg/m³ (or 10,000 µg/m³). If the dust contains quartz, the exposure limit for the worker is reduced substantially. The current standard for quartz-containing dust is 100 µg/m³ of respirable silica for open-cut mines or 120 µg/m³ for underground mines (but some organisations have recommended that the underground criterion should be the same as for open cut). Other inhalable dusts have a specified limit of 2.5 mg/m³ (2,500 µg/m³). These are the levels the medical authorities consider an acceptable risk for a worker to be exposed to for 8 hours a day, 5 days a week for an average working life (eg 40 years). The levels are based on epidemiological studies of large numbers of workers in dusty industries where accurate sampling data of cumulative dust exposure can be related to incidence of silicosis and airway disease. For review see Parkes (1994) and the section below. At the levels quoted above, a low percentage of workers would develop radiographic changes or abnormalities of lung function and even fewer would develop respiratory symptoms.

It can be seen that the environmental standards for an annual average of less than 30 µg/m³ of PM_{10} and a 24-hour average of less than 50 µg/m³ are well below the occupational health standards even for dust containing a high proportion of silica. The environmental standards are less than 1% of the specified limit values for workers exposed to dust in coal mining operations.

Workers are regularly monitored for exposure to respirable dust and quartz in underground mines. If exposures exceed the limits described above operations must be reviewed and dust levels reduced. Miners in Australia also receive regular medical checks with radiographs and lung function tests.

Lung disease in coal miners

The medical literature on lung disease in coal workers is dominated by reports from underground mines. Exposures to high levels of dust are increased by poor ventilation. Underground miners are also exposed to high concentrations of diesel fumes that contribute to the higher risk of lung disease. Longwall miners in NSW who had coal dust exposure levels of 1,720 µg/m³, (below the standard of 3,000 µg/m³) had a 1–3% risk of developing pulmonary fibrosis or CWP (Kizil et al. 2001). CWP typically takes 10–20 years to become apparent on radiographs. The literature in this area indicates variation in risk of disease between workers (that is individual susceptibility varies; smoking, for example, markedly increases the effects of dust) and between mines (silica content and iron content). Thus it is still possible to find cases of CWP, as has been reported in Queensland over recent years. The aim of regular health checks is to detect CWP early before symptoms or impairment occur and to remove susceptible individuals from further exposure. It is now generally accepted that exposure to coal mining does not increase the risk of lung cancer (e.g. for review see Stayner and Graber, 2011).

Available data indicates that open-cut coal miners are less likely to develop respiratory disease than underground workers but coal workers' pneumoconiosis has been reported among those that have worked at the coal face (Jacobson et al. 1970; Amandus et al. 1989). However, the studies were reporting exposures from the 1970s and 80s, and reflect dust and fume exposures much higher than would be permitted throughout the industry today. Furthermore, loaders and dump trucks did not have air-conditioned cabs, diesel engines and fuel were not as refined as they are now and dust mitigation was generally not used.

Love et al. (1997) examined a large number of workers employed at a number of sites throughout the United Kingdom in a study where exposure levels were high by comparison with those generally found within the Australian mining industry. The proportion of miners with abnormal chest radiographs was low at 4%, considering that exposures were higher than allowed today and some of the men had worked in the industry for many years and some may have worked underground in the past. The frequency of chronic bronchitis was influenced by the number of years worked in the industry. Asthmatic symptoms were reported by 5% of the workers, which is similar to the proportion found in a non-exposed population. Lung function tests were normal and showed no association with the extent of exposure.

Health effects in communities exposed to coal dust

There have been relatively few epidemiological studies of exposures in communities living close to coal mining operations. Despite that, there is very little evidence of significant health effects from coal dust and air pollution for communities living near open-cut coal mines (see below). It is difficult to compare pollution data between various studies because of variation in mining and processing methods, size and output of the mine and regional mineral composition (particularly quartz and iron content). There are also variations in regulations for air quality, dust suppression measures, general health of the surrounding population and air pollution from other sources. Near a Colombian mine, the major factor causing pollution in the surrounding villages was dust from 40 km of unsealed roads rather than from the mining and coal handling and transport (Huertas et al. 2012).

England

Children exposed to coal dust from a coal loader at a sea port in Liverpool, England, were compared with those in nearby districts who were not exposed (Brabin et al. 1994). The port also handled grains and other materials. There was increased absenteeism from school in the exposed population but no increase in hospitalisations for asthma. Modelling showed that going to school in the exposed zone did not increase the odds of respiratory symptoms once confounding factors such as parental smoking were taken into account.

The absenteeism from school may have related to social factors. The average reading of dust measurements in the area for particles settling on the ground was 6 g/m^2/month, a high figure.

Pless-Mulloli et al. (2000; 2001) documented children's health in a region in northern England where there was agriculture before open-cut coal mining in the 1990s. There was a significant increase in GP consultations for respiratory, skin and eye conditions in the coal mining communities, but there was no difference in the total number of GP consultations between case and control communities. There was no increased prevalence of asthma or respiratory illness in children living near open cut coal mines, which were the only source of pollution in the area. There was no difference in the general health or rates of long-term illness. Asthmatic children in communities near open-cut coal mines did not have more frequent or more severe attacks than the children in control communities. There was no significant difference in the prevalence of asthma between communities.

United States

In recent years, Hendryx and colleagues have reported an association between a range of adverse health outcomes and living near coal mining operations in the Appalachian Mountains of West Virginia in the United States (Hendryx and Ahern 2008; Esch and Hendryx 2011; Ahern et al. 2011). The range of disorders included self-reported poor health status, cardiopulmonary disease, chronic obstructive pulmonary disease, 'black lung', kidney disease and hypertension. Cardiovascular mortality was reported to be increased within those regions with mountaintop mining but not in those regions with other methods of coal mining (underground mining). Low infant birth weight was also reported as an association even after adjusting for the known factors that had the strongest associations (that is maternal smoking, medical risk factors, low education and socio-economic status). Poor dental health was also reported. The authors discussed the limitations of these studies and their attempts to control for confounding factors. For example, the results may have been influenced by pollution from coal burning power stations within the coal mining regions; inclusion of miners in the study samples; and difficulty adjusting for smoking, health and socio-economic status between residents living near and far from mine operations.

Appalachian Mountain top mining involves pushing the overburden from the mountain top into the valleys. This may contaminate groundwater with a range of toxic elements (arsenic, lead, cadmium, mercury and radioactive isotopes). The areas are among the poorest in the US with poor health care and substandard housing. Many residents occupied abandoned mining camps with no services and used groundwater for drinking and reject coal for heating and cooking (Holtzman 2011).

Australia

In 2010, the NSW Department of Health studied respiratory and cardiovascular diseases and cancers among residents in the Hunter and New England health service areas and compared these with the rest of NSW (NSW Health 2010). Areas with and without coal mines within the Hunter region were also compared.

Overall the rates of respiratory admissions were lower than the rest of NSW. Respiratory presentations to hospitals in Muswellbrook and Singleton (major mining areas with coal-fired power stations) ranked below those of Tamworth and Gunnedah in all age groups. The rate for Muswellbrook was higher than the state average but that for Singleton was lower, indicating no consistent trend with mining activity.

For asthma presentations Singleton ranked 11th in New South Wales in 0 to 14-year-olds, 6th in 15 to 34-year-olds, 3rd in 35 to 64-year-olds and 25th in those aged over 65 (that is, no consistent trend). Overall the rate of hospital admissions for respiratory disease in the Hunter area is lower than that for the rest of NSW. The rate of hospital admissions for asthma was also lower than that for the rest of NSW for both adults and children. There was variation between areas with higher rates in rural areas thought to reflect higher allergen exposure. Overall, there were more emergency department presentations for all conditions in Hunter area compared with the rest of NSW, a finding thought to reflect social factors.

There was no consistent association between presentations to emergency and mining activity with some coal mining areas higher than average and some lower.

The rate of hospitalisation for cardiovascular disease in the Hunter and New England area as a whole was comparable to that for the rest of New South Wales. However, the rates were higher in areas with coal mines than other areas. The report cautioned that these areas were rural and had higher smoking rates.

The death rate from all causes was slightly higher compared with the rest of NSW in the lower Hunter but slightly lower in the upper Hunter. Deaths from cardiovascular disease were higher in the upper Hunter and lower in the lower Hunter, with the average for the two regions similar to that for the remainder of NSW. The incidence of cancer and the death rate from cancer in the Hunter and New England areas were higher than the average for the rest of NSW, with the highest rates found in Mehi, McIntyre and upper Hunter (that is, not associated with mining areas). The cancers that had higher rates included colorectal, prostate and melanoma: cancers not known to be related to coal mining or airborne pollution. The rate for lung cancer was not higher in the Hunter region than the rest of NSW and was not higher in areas with coal mining operations than other areas in the Hunter and New England regions.

The project cannot be compared with the Appalachian mines because the mining methods are very different and the general health and living standards of the surrounding population are substantially higher. Compared with the open-cut coal mining in the Hunter Valley, the project will produce substantially less dust and airborne particulate matter (PM_{10} and $PM_{2.5}$). Moreover, the Hunter Valley has significant sources of $PM_{2.5}$ derived from combustion, including an aluminium smelter, coal fired power stations, other industry and a much higher density of population with associated pollution from vehicles and wood burning for heat in winter. $PM_{2.5}$ levels in the Hunter would therefore comprise higher concentrations of the products of combustion than near the project area.

Health risk assessment for the Hume Coal Project

A health risk assessment uses information about potentially hazardous pollutants to estimate a theoretical level of risk for people who might be exposed to defined levels of these pollutants.

Risk assessments often consider possible or theoretical community exposures based on the outcomes of air dispersion modelling. Conservative safety margins are built into a risk assessment to protect the public so that people will not necessarily become unwell even if they are exposed to pollutants at higher concentration levels than those estimated by the risk assessment. During a risk assessment, the most vulnerable people (such as children, the elderly and the sick) are carefully considered to make sure all members of the public will be protected.

Assumptions used in this and other assessments relate to the population profile and health of those potentially exposed, existing air quality, incremental exposures related to the project, and known and possible health effects of the exposures. There are not enough data on health effects of living near a coal mine to derive estimates of the size of an adverse health response in relation to exposure to a particular concentration of the various components of pollution (that is, exposure–response functions). Therefore these functions are drawn from large overseas epidemiological studies of populations in cities exposed to air pollution dominated by products of combustion.

Another assumption is the health effects of urban pollution at higher concentrations can be extrapolated to (in other words, used to infer what is not known from what is) lower concentrations in a rural setting with a coal mine and other industries. This assumption is likely to overestimate the risk from a mine and is therefore conservative.

Specific effects (or endpoints) that may be evaluated in health assessments include: annual mortality all cause 30+ years; cardiopulmonary mortality 30+ years; ischaemic heart disease 30+ years; lung cancer mortality 30+ years; daily mortality all causes all ages; daily mortality cardiovascular disease all ages; hospital admissions respiratory disease 65+; hospital admissions cardiac disease 65+; hospital admissions pneumonia and bronchitis 65+; hospital admissions cardiovascular disease 65+ years; hospital admissions respiratory disease 15–64 years; and emergency department visits (asthma) 1–14 years. For each of these end points the potential effect of $PM_{2.5}$ or PM_{10} has been derived from the range of responses reported in the epidemiological literature and referred to as concentration response functions (CRFs).[2]

In summary, the estimated health impact in the population, due to the worst case annual average increased long-term exposure to $PM_{2.5}$ and PM_{10} as a result of the project and cumulative exposure for excess all-cause mortality would be substantially less than 1 extra death per 100,000 population and therefore considered to be "sufficiently small to be of no cause for concern" (NEPM AAQM).

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[2] For example, the reported increase in mortality (all causes, ages 30+) with long-term exposure to an increase of $PM_{2.5}$ of 10 µg/m³ ranged from 4–8%. Assuming a value for the CRF of 6% Jalaludin and Cowie's (2012) method gives a beta coefficient of 0.0058 for each 1 µg/m³ increase in $PM_{2.5}$.

For this assessment the following end-points have been used in relation to $PM_{2.5}$ as with Jalaludin and Cowie's method: annual mortality all cause 30+ years (CRF 6% per 10 µg/m³ increase), daily mortality all causes all ages (CRF 0.9% per 3.78 µg/m³ increase), and hospital admissions for respiratory disease 15–64 years (1.1% per 3.78 µg/m³ increase).

In relation to PM_{10} the following end-points have been used as with Jalaludin and Cowie's method: annual mortality all cause 30+ years (CRF 3.86% per 10 µg/m³ increase), daily mortality cardiovascular all ages (CRF 1.3% per 10 µg/m³ increase), and hospital admissions for respiratory disease 15–64 years (1.22% per 10 µg/m³ increase).

For baseline health statistics average rural values for NSW (incidence per annum per 100,000 population) have been used, namely:

- mortality all causes 30+ years: 353;
- cardiovascular mortality:136; and
- respiratory admissions 15–64 years: 299.

To calculate the excess mortality (30+ years) attributable to the exposure (Hume emissions of $PM_{2.5}$) per 100,000 population has been calculated as the beta coefficient for the Exposure Response Function (0.00582) x increment in $PM_{2.5}$ (0.3 µg/m³) x population exposed x death rate 353/100,000 = 0.02 additional cases assuming an exposed population in the sensitive receptor zone of 1,300. This represents an increase in annual mortality of approximately 0.005%.

Similar calculations for daily mortality from all causes and for all ages for increases in the 24-hour average of $PM_{2.5}$ yield 0.0001 attributable cases per 100,000 population.

Similar calculations for hospital respiratory admissions 15–64 years yield approximately 0.005 attributable cases per 100,000 population.

Calculations using the CRFs for PM_{10} provide smaller numbers for the attributable cases.

Exposures to daily increases of $PM_{2.5}$ and PM_{10} pose an insignificant risk as the predicted number of attributable cases of daily mortality and cardiovascular disease are substantially less than 1 per 100,000 population and orders of magnitude lower than that due to long-term exposure.

Exposures to gaseous emissions such as NO_2 and VOCs are so small that any health effects are likely to be undetectable. The likely concentrations would be below those believed to have no effect in asthmatics, the individuals most sensitive to NO_2.

Impact on human health due to noise and vibration impacts

Road, rail and aircraft noise are the main environmental noise pollutants that have been investigated and reported in the medical literature. In NSW night-time traffic noise at nearby homes should be below 55 dBA as an average but transient peaks of 65–70 dBA may occur (eg trucks, motor bikes and sports cars). Homes near train lines may be exposed to peaks of 80 dBA at night. Aircraft produce noise levels typically in the range of 70–85 dBA depending on the distance from an airport. The impact of noise varies according to a range of factors, including ambient noise level, frequency of the sound, wind direction and velocity, humidity and air temperature.

Noisy industries (mechanical hammers and presses, excavation machines, grinders and so on) can cause significant health effects on workers in addition to hearing loss, including increased risk of cardiovascular disease. Extraneous noise pollution in residential and other settings (e.g. schools) seems to have minimal health effects at levels below 55 dBA. Aircraft noise greater than 55 dBA may have a weak association with hypertension and has a relative risk 1.0–2.5, which is not significant statistically (Rosenlund et al. 2001) and cardiovascular relative risk of 1.1–1.5 (Stansfeld & Matheson 2003).

Babisch (2000) published a systematic review of studies relating traffic noise to cardiovascular disease and concluded there was no relationship between traffic noise exposure and blood pressure in adults. Noise-related increases in blood pressure were reported in several studies of children exposed to levels up to 70 dBA compared with those exposed to 53 to 60 dBA. He concluded that there was ‘little evidence that exposure to high traffic noise is associated with an increased risk’ of clinical hypertension. For ischaemic heart disease there was some evidence of an increased risk in people who live in areas with outdoor noise levels of greater than 65–70 dBA. Sudden intense exposure to noise has not been found to be associated with cardiac arrhythmia in patients with heart disease.

The participants in a Swiss study were not informed that airport and traffic noise was the focus of the research to reduce bias in an area where an extra runway was being constructed. The results showed no association between noise exposure and mental health status. Annoyance was found to be the most common psychological impact of noise (Babisch 2000). Annoyance was associated with frequent occurrence of noise, the intensity of noise, whether vibration was present, level of threat and whether there was a belief that authorities could control the noise. At noise levels of 55–60 dBA (indoors) 35–40% of office workers report being ‘highly annoyed’ while industrial workers have a similar reaction at levels greater than 85 dBA. Most studies have found no evidence of increased prevalence of mental illness in areas where noise levels are increased.

Significant noise pollution has been associated with attention deficit in primary school children and poorer auditory discrimination (hearing) and speech recognition with lower reading and school performance (Stansfeld & Matheson 2003). Children in a classroom close to a railway line (possibly exposed to transient train noise of 80 dBA, see above) were found to have a 3–4 month difference in reading ability compared with children at the same school in a quiet classroom further from the line (Bronzaft & McCarthy 1975). When an airport relocated in Munich, the children at the old airport site reversed their learning delay, while the children exposed to noise at the new airport became delayed (Evans et al. 1995). The elderly, with less cognitive reserve, may also be vulnerable to the effects of noise on cognition. Noise exposed children were less motivated at problem solving and may have had increased stress levels (Walinder et al. 2007).

Noise generated by the project: regulatory requirements

The regulatory requirements to operate the project are determined in relation to the intrusiveness criterion that limits additional noise to 5 dBA above background. The background level adopted (RBL) is 30 dBA for a quiet rural area, which results in an intrusiveness criterion of 35 dBA Leq, 15 min (see Table 3.2 in the Noise and Vibration Assessment). The Leq is a time average to allow for occasional louder noises such as vehicles driving by. This then results in an indoor sound level criterion of 25 dBA Leq, which equates to a level of 35 dBA Leq outdoors assuming a 10 dBA attrition due to the façade with partially open windows. The maximum night-time noise level (Lmax) criterion is 45 dBA. These limits are conservative in relation to noise levels that currently exist in most urban environments and reflect the mostly quiet rural conditions in the area.

Operational Noise

Predicted operational noise levels will be above applicable noise guidelines for 11 sensitive receptors near the project area (Project Specific Noise Level [PSNL] see Table 5.1, Noise and Vibration Assessment).

One of these locations will have negligible noise impact (1–2 dB above PSNL), eight locations will have moderate (3–5 dB above PSNLs) and two will have significant (>5 dB above PSNLs) noise impact. The significant noise levels at the two worst affected receptors (41–43 dBA) are orders of magnitude less than those described above which have been shown to cause health effects or learning problems. The three locations predicted to experience significant noise impacts are entitled to voluntary acquisition; the eight moderately affected locations are entitled to voluntary noise mitigation measures.

Real-time noise will be monitored and, if standards are likely to be exceeded due to adverse temperature, humidity and wind conditions, work will cease or be modified. Mitigation strategies will be used if monitoring reveals potential exceedances. Measures that will be available to complainants through the independent review process will include modifying plant and procedures, erecting noise barriers and fitting insulation and air conditioning to homes.

Construction Noise

During the construction phase of the project, some noise will be above relevant noise management levels (NMLs). This will be confined to properties to the north-west of the project area and will mostly be from 1 dB to 3 dB above NMLs. The ‘highly affected’ NML of 75 dB will not occur at any assessment location. NMLs are not acceptability criteria (as are operational noise limits), but a trigger for consideration of noise mitigation measures (e.g. limiting construction activities to standard working hours). However, the predicted maximum noise levels in standard working hours during the construction phase are below levels known to cause adverse health effects.

Drift and shaft construction, work inside enclosed buildings/structures, and the construction of the accommodation village, will occur 24 hours a day, 7 days a week. Construction noise levels from ventilation shaft construction and the use of a blind bore rig are predicted to be up to 3 dB above the evening and night NML. Noise levels will be monitored and localised noise barriers will be installed if noise levels are above the NMLs. Noise barriers could provide 5 to 10 dB of noise reduction and, therefore, with such mitigation in place, these activities are predicted to satisfy the NMLs at all assessment locations.

Further, maximum noise levels should not exceed the minimum sleep disturbance screening criterion of 45 dB, Lmax.

The vibration criteria for human response and cosmetic damage are predicted to be unlikely to be exceeded. It is also predicted that blast overpressure and vibration for construction of shafts and portals will satisfy the appropriate criteria.

Traffic Noise

All roads that will be used to access the project area where nearby residences exist will experience zero to negligible (1–2 dB) noise level increases and therefore satisfy the appropriate requirements.

Impact on human health due to noise impacts on sleep for nearby residents

Sleep is an important restorative function and most people report sleep disturbance from unexpected loud noises. It is known that sudden arousals from sleep are associated with increases in heart rate and blood pressure due to autonomic nerve discharge. However, the scientific literature indicates only a weak relationship between outdoor noise and sleep disturbance (Stansfeld & Matheson 2003). Sleep disturbance and cardiovascular/autonomic consequences of arousals secondary to noise have been shown in many studies to habituate over time (Townsend et al. 1973; Zaharna & Guilleminault 2010), although some individuals are thought to be more sensitive than others. People who live near railway lines or under flight paths adapt to the intermittent disturbances and their sleep returns to normal patterns.

Few studies have investigated the effects of long term chronic noise exposure, but one study gave subjects a short tone of 80–90 dB, (which is far higher than the 45 dBA Lmax limit of the rural criteria) every 22 seconds for 40 days and found at the end of the study no significant change in heart rate, time taken to go to sleep, amount of sleep, awakenings or time in each sleep stage.

The bulk of the evidence suggests that sleep quantity and patterns and objective measures of sleep quality, including daytime performance, are unaffected at indoor noise levels less than 40–55 dBA (Passier-Vermeer & Passchier 2000).

The regulatory requirements for the operation of the project vary with time of day and between suburban and rural. The sleep disturbance criteria are shown in Table 3.6 in the Noise and Vibration Assessment. The maximum night time noise level (Lmax) is 45 dB based on a limit of 15 dBA above background and assuming a background level of 30 dBA in the rural locations. Urban locations have higher limits.

Guidance about the potential for sleep disturbance is also provided in the NSW Road Noise Policy (RNP). The RNP relies upon a number of studies into the effect of maximum noise levels on sleep. The RNP acknowledges that, at the current level of understanding, it is not possible to establish absolute noise level criteria that would correlate to an acceptable level of sleep disturbance. However, the RNP provides the following conclusions from the research on sleep disturbance:

(i) maximum internal noise levels (Lmax) below 50 to 55 dBA are unlikely to awaken people from sleep; and

(ii) (ii) one or two noise events per night, with maximum internal noise levels (Lmax) of 65 to 70 dBA, are not likely to affect health and wellbeing significantly.

It is commonly accepted by acoustic practitioners and regulatory bodies that a facade of a residential building of standard construction including a partially open window will reduce external noise levels by 10 dB. Therefore, external noise levels in the order of 60 to 65 dBL_max calculated at the facade of a residence are unlikely to cause sleep disturbance effects.

Noise modelling (Table 5.4, Noise and Vibration Assessment) showed that Lmax external noise would be below the sleep disturbance screening guideline at most assessment locations.

Three locations are predicted to experience possible exceedances of the EPA's screening criterion for sleep disturbance in calm or adverse weather conditions of up to 1, 4 and 6 dB. This equates to maximal internal noise levels of 36, 38–41 and 42–43 dB for the three locations in calm and adverse conditions. These levels are well below those likely to cause sleep disturbances reported in the medical literature discussed above. The exceedances are predicted to be due to train movements, which would occur no more than twice a night. This number of arousals would not impair sleep quality and the literature indicates that adaptation to train noise occurs readily. Noise mitigation strategies (such as improved glazing and air conditioning to allow windows to be closed at night) could reduce the highest indoor noise levels to 31 dB or less at these receptor locations.

Based on the above discussion and the predicted noise levels in Section 5 of the Noise and Vibration Assessment, it is likely noise from the project will not result in significant sleep disturbances for nearby residents.

Low frequency (LF) noise or infra-noise

Low frequency noise is considered separately because it has some properties that differ from other frequencies of noise. It is barely audible but can vibrate windows and its intensity does not weaken with transmission distance as readily as higher frequencies. Examples include the low frequency components of the sound of jet aeroplanes, wind turbines and some heavy machinery. Some people find the low rumble distressing and there remains some anxiety about possible health effects. However, there is no evidence that low frequency noise within prescribed standards produces health effects (Leventhal et al. 2003). Experiments on astronauts indicated that, for 24-hour exposure, low frequency noises at a frequency of 20 Hz and very high pressure levels of 120–130 dB are tolerable and do not cause physiological damage (for review see Leventhall et al. 2003).

Prolonged exposure to audible low frequency sound may produce fatigue, poor concentration, headache, sleepiness, sleep disturbance and psychological distress (Leventhal et al. 2003). Except for annoyance and self-reported sleep disturbance, no consistent effects on health have been reported from wind turbines (Bolin et al. 2011). No medical condition has been reported in the literature to be associated with the perception of infrasound or associated sensations (Tierney et al. 2003).

The EIS (Appendix I, Section 5.4) indicates that predictions for low frequency noise impacts are the same as those reported to be impacted by operational noise with the same locations (12 and 13) with significant noise impacts exceeding the low frequency (LF) reference by >5 dB. Similarly 10 of the properties with moderate and negligible noise impact exceed the LF reference by up to 5 dB. Residents exposed to these levels of low frequency sound would not be expected to develop adverse health effects.

Conclusions

The predictions from the modelling for the highest levels of $PM_{2.5}$, PM_{10} and gaseous pollutants from the project, both during peak construction and peak operation, show the project's emissions will be small compared with background levels and that the cumulative levels will be well below the applicable regulatory criteria. The NEPM standards are unlikely to be exceeded other than when they are due to external events such as dust storms and bush fires. The health impact is predicted to be so small as to be immeasurable and well below the regulatory criteria.

Noise criteria are likely to be exceeded at a number of locations that are close by. The predicted worst case noise levels are not enough to cause measurable health impacts or sleep disturbance but breach the amenity criteria at three homes and therefore trigger acquisitions.

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