APPENDIX D

ASSOCIATE PROFESSOR DAVID MCKENZIE'S RESPONSE TO HEALTH MATTERS

D

T4 PROJECT HEALTH ASSESSMENT

EXPERT REPORT PREPARED BY

ASSOCIATE PROFESSOR DAVID McKENZIE

1. INSTRUCTIONS

1.1. This report has been prepared on behalf of Minter Ellison.

1.2. This report has been prepared having regard to Division 2 of Part 31 of the *Uniform Civil Procedure Rules* 2005 and the Expert Witness Code of Conduct in Schedule 7 of the *Uniform Civil Procedure Rules* 2005. I have read these provisions and agree to be bound by them.

1.3. In preparing this report I have had regard to the following material:

(a) Environmental Assessment Prepared by EMM (February 2012)

(b) Air Quality Assessment for Modified Project, Prepared by Environ Australia P/L

(c) A review of the medical literature

1.4. In preparing this report I have been instructed to:

(a) review and provide an opinion on any health effects from exposure to suspended particulate matter with special reference to coal dust and the air quality projections contained in the Environmental Assessment prepared by EMM for the T4 Project and the Air Quality Assessment for Modified Project prepared by Environ Australia P/L;

(b) provide an opinion on any potential risks to health from the projected fugitive emissions from trains for residents along the rail corridors, especially of particulates in the respirable range (PM_{10} and smaller);

1.5. Some assistance with the literature searches for this document was provided by my colleague Dr Elizabeth Brown MB,BS FRACP, PhD, Respiratory and Sleep Physician and medical researcher. In selecting articles for citation every effort has been made to include the major relevant works and reviews from mainstream sources.

1.6. I am a Respiratory and Sleep Physician with 30 years clinical experience and considerable expertise in occupational lung disorders and airborne pollution. I am the Head of the Department of Respiratory and Sleep Medicine at the Prince of Wales Hospital, and the Director of the Cardiac and Respiratory Clinical Stream of the South Eastern Sydney Local Health District. I am also in private practice at the Prince of Wales Private Hospital and it is in the latter capacity that I have prepared this report. A copy of my Curriculum Vitae is attached.

2. SUMMARY

2.1. High levels of urban pollution are known to cause adverse health effects, especially in the frail elderly with cardiac and respiratory disease.

2.2. The urban pollutants most strongly associated with adverse outcomes are "black smoke" or "elemental carbon" which are products of combustion and most strongly associated with diesel traffic.

2.3. The types and proportions of suspended particulates (PM_{10} and $PM_{2.5}$) vary between urban, industrial, semi-rural, rural and coastal regions. Air quality criteria for PM_{10} and other specific pollutants (e.g. oxides of sulphur and nitrogen and ozone) have been established largely from studies of densely populated urban areas and applied to other settings. Given the difference in source of PM_{10} in an urban setting (i.e. predominantly vehicle combustion) compared to a rural or coastal setting (predominantly mechanical disturbance of crustal material, organic matter and sea salt) using the urban criteria in other settings is regarded as conservative. The T4 Project site is coastal and located on a river with some relatively low density urban areas and industrial sites nearby and no major roads such as freeways or highways.

2.4. The medical literature which indicates adverse health effects from suspended particulate matter (PM_{10} and $PM_{2.5}$) and other components of airborne pollution is based on studies of urban environments with varying levels of pollution. This literature is summarised below. I have also reviewed the literature on adverse health effects for the *workers* in coal mines who would have exposure levels to coal dust and other pollutants hundreds of times higher than individuals in communities near coal facilities.

2.5. Based on the data available to me from the Environmental Assessment, and the Air Quality Assessment for Modified Project, the levels of airborne dust, small particles, diesel exhaust emissions and specific components of combustion would be within the current standards for the residents of communities in the vicinity of the T4 Project.

2.6. Using the dispersion modelling data for TSP, PM_{10} and $PM_{2.5}$, dust levels would be at acceptable levels in terms of health risk in the surrounding residential areas even if the maximum 24-hour average PM_{10} values exceed the criteria from time to time. Infrequent exceedances of the 24-hour average PM_{10} criterion have been historically recorded in the region, generally due to regional events such as bush fires and dust storms. The contribution from the T4 Project is predicted to be a small percentage of the total and not cause any additional exceedances of the criterion. Fumes and gases from diesel exhausts and ships are likely to disperse further and more quickly in the atmosphere than particulate matter. These are not predicted to exceed criteria levels or provide a health risk for residents.

3. INTRODUCTION – AIR QUALITY

3.1. Inhalation of suspended particulate matter is a natural phenomenon that has occurred throughout evolution. The healthy lung can remove most forms of foreign matter using the mucus lining the airways and special scavenger cells (macrophages) in the air sacs (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 <10μ m in diameter (i.e. small enough to be inhaled into the lung, also known as PM_{10}) and the individual susceptibility of the person inhaling the dust. Many scientific studies are now concentrating on the smaller particles ($PM_{2.5}$), which are inhaled as far as the alveoli in the lungs, and specific components of pollution.

3.2. Most of the dust that we 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 inflammatory and fibrotic reactions in the lungs. Silica is contained in quartz, sand and many rocks such as granite. The percentage of silica in coal and the overburden (material above or around the coal seam) varies substantially from site to site but is usually a relatively small fraction. Atmospheric levels of silica have been measured in the vicinity of two open cast mines in the Hunter Valley and the worst case values (0.5 -1.8 $\mu g/m^3$) were well below the environmental standard for an annual average (3.0 $\mu g/m^3$, ACARP Study, C18026, June 2012). Most of the silica is removed from coal by washing prior to transport from the mine.

3.3. Dust we inhale also contains numerous other substances usually in very small quantities. In heavily populated urban areas the air contains particles from the exhaust systems of vehicles (especially diesel trucks and buses), and other processes which combust hydrocarbon fuels such as power stations, heavy industries, jet aeroplanes etc. The products of combustion are predominantly in the $PM_{2.5}$ fraction which may explain why smaller particles appear to be more harmful than larger. In rural communities, the dust may contain particles from wood fire smoke and dust released from earth moving, ploughing and the harvesting or processing of various grains such as wheat. There are also small concentrations of pollens and spores of fungi. In coastal regions the PM_{10} fraction includes fine particles of sea salt. Airborne particulate matter in rural and coastal regions generally has lower percentages of $PM_{2.5}$ than that of urban areas. Using urban criteria for other regions is regarded as conservative.

3.4. The PM_{10} (and $PM_{2.5}$) fraction of airborne dust will therefore vary in composition from place to place depending on the types of industry and the density of vehicular traffic. It also varies with the temperature, humidity, weather (including rain, wind speed and direction), and the time of year. A region with mixed urban and coastal (and/or rural) areas will have a mixture of the components of airborne particulate material described above. Motor vehicles are the major source of air pollution in urban areas, contributing to the photochemical smog or haze seen on still days[1,2,3]. The urban pollutants most strongly associated with adverse outcomes are "black smoke" or "elemental carbon" which are products of combustion and most strongly associated with diesel traffic. However, the air quality in Australian cities is better than in most other cities of similar size in Europe and the US, particularly in relation to the levels of $PM_{2.5}$.

3.5. High levels of particulates occur with high winds in dry seasons and bush fires. When bushfires occur around Sydney PM_{10} levels can rise to 300-500 $\mu g/m^3$ (ie up to 10 times the 24 hr criterion). There is weak evidence for increased admissions to hospital for respiratory complaints during bush fires around Australian cities. During the infamous East Coast dust storm of 23 September 2009 PM_{10} levels in Sydney and Newcastle were variously reported at 2,000 $\mu g/m^3$ and 15,500 $\mu g/m^3$ (the latter presumably an instantaneous peak level). Emergency services reported an increase in complaints of breathing difficulties. Children, elderly and those with heart and lung conditions were advised to remain indoors. I have been unable to find reports of increased admissions to hospital or deaths during the East Coast dust storm.

3.6. The environmental standards for PM_{10} (see below) have been set at conservative levels to allow for worst case situations with regard to the composition of the particles and the most vulnerable individuals. Most of the studies in the medical literature, which indicate a correlation between levels of PM_{10} (or $PM_{2.5}$) and adverse health outcomes have been conducted in urban environments with varying density of population. The studies have recorded various measures of health ranging from respiratory symptoms such as cough and wheeze through to daily mortality rates and correlated these with levels of PM_{10} or $PM_{2.5}$ (e.g. Ref 1). However, PM_{10} is just one component of urban pollution, which is relatively easy to measure. When PM_{10} rise, other components of urban pollution (see below) are also likely to rise. The medical literature is inconclusive as to whether particles per se are the cause of adverse health outcomes or whether other specific components of the pollution are involved. The PM_{10} of urban pollution definitely contains a wide range of known toxic and carcinogenic substances (predominantly in the $PM_{2.5}$ fraction). Nevertheless it is assumed that all particles are potentially harmful.

3.7. Urban pollution contains a large number of substances including solid particles of dust, pollens, fungal spores, vapours consisting of tiny droplets of unburnt hydrocarbons including benzene, toxic organic micropollutants, heavy metals and various gases including ozone, nitrogen oxides, carbon monoxide and sulphur dioxide. The toxic organic micro pollutants include polyaromatic hydrocarbons, polychlorinated biphenyls, dioxins and furans. The bulk of these come from burning fossil fuels including petroleum products and oil. Criteria levels (or standards) for pollutants have been set at levels which are believed to be thresholds below which there are acceptably low risks for adverse health effects. For example, the current levels for PM_{10} are 50$\mu g/m^3$ for any 24 hour period and 30$\mu g/m^3$ for an annual average. The data relied on to set these values are reviewed in this report to provide a context in which the projected levels from the environmental assessment can be interpreted.

3.8. Authorities such as the Environment Protection Authority and Governments set limits for urban pollution at conservative levels based on data from epidemiological studies, which are not necessarily generalisable to communities other than where the study was done. The thresholds are set at a point below which the statistical risk of adverse health consequences is considered acceptable. In some areas, the level might have to rise considerably before any increase in adverse health effects could be detected. The thresholds are intended to be at levels where the majority of *susceptible* individuals would not get symptoms. Thus, it is not a threshold for most individuals. These levels are substantially below the values which are allowable in the occupational health and safety standards for workers (known as threshold limit values, discussed below).

3.9. Long-term exposure to excess levels of urban pollution is thought to induce oxidative stress, inflammation, progression of atherosclerosis and, possibly, altered autonomic function of the heart. Days with high concentrations of airborne urban pollution are associated with increased cardiovascular and respiratory disease (particularly exacerbations of asthma) and increased mortality. A small proportion of the population is at risk of adverse outcome from short-term exposure to pollution - that is the elderly, those with cardiopulmonary disease, infants and asthmatics. Most studies conclude that, although a susceptible individual's risk when exposed to air pollution is small, the large populations exposed in cities on high pollution days result in a significant burden of illness[4,5].

4. HEALTH EFFECTS OF URBAN AIR POLLUTION

4.1. It is not certain how exposure to high levels of urban pollution contributes to excess mortality. Chen et al (2007)[4] state "Although the evidence linking inhalational exposure to particulates with adverse health events is strong, definitive conclusions about toxicological mechanisms and exposure response relations for specific types of ambient particulates have not been fully defined." Long-term exposure to excess levels of urban pollution is thought to induce oxidative stress and inflammation thereby aggravating bronchial conditions and leading to progression of atherosclerosis and, possibly, altered autonomic function of the heart.

4.2. Methodological problems with studies of air pollution and disease have been identified and discussed in the scientific literature. These include:

4.2.1. Many studies are retrospective, relying on the subjects' memories of exposure (recall bias). Misclassification of exposure may occur because of recall bias or because of sampling errors due to design faults.

4.2.2. Many studies have looked at only one city, or high and low pollution communities in one country, so the conclusions may not be able to be generalised to other cities or countries.

4.2.3. Outdoor air sample data collected by government agencies has been used in many studies. However, outdoor data has been shown not to correlate with a child's actual exposure when measured by personal sampling device because there are additional sources of indoor air pollution (cooking and heating fumes, mould, damp, pets and microbial organisms) which also contribute to respiratory disease and asthma.[6]

4.2.4. Studies showing adverse effects are more likely to be submitted to journals by researchers and more likely to be accepted for publication than studies showing no effect.

4.2.5. Climate and local factors including levels of sunshine and aeroallergens may have a confounding effect. Many studies do not take climate differences into account[7].

4.2.6. The observed effects can be biased by underlying associations – i.e. the exposure and outcome could be both affected by a confounding variable.[8] For example, smoking rates could be higher in an area with higher pollution.

4.3. Results from heavily polluted cities overseas may not be applicable to Australia. The hot, dry climate in Australia means that windborne dust from semiarid countryside will be included in urban PM_{10} measurements especially on days with high levels. Thus there are several reasons why the criteria derived from overseas studies may be conservative for Australian cities.

PM_{10} and other markers of pollution in cities in US and Europe

4.4. A substantial amount of research has provided evidence that breathing PM is harmful to human health. Studies using a variety of methodology have observed reasonably consistent associations between daily changes in PM and morbidity and mortality outcomes. These associations have been observed in single city studies and in large multi city studies. The evidence for long-term chronic health effects has been strengthened by reanalyses and extended analysis of several studies.

4.5. Samet et al[2] reported pollution levels in 20 US cities and found the average PM_{10} per day was 20–50 µg/m³, with their guidelines at that time allowing up to 150 µg/m³. They found that there was a 0.3–0.6% increase in the rate of deaths per day for each 10 µg/m³ increase above background in PM_{10}, which is similar to findings of other studies. Levels of CO, NO_2 and SO_2 (adjusted for ozone and PM_{10}) had no significant association with the relative rate of death.

4.6. Other studies have concluded that those with advanced heart or lung disease are most at risk from pollution.[5,9] Daniels et al (2000) [9] found a linear relationship between pollution and mortality from cardiorespiratory disease, but other causes of mortality did not increase until a threshold of 65µg/m³.

4.7. Gauderman et al[8] studied lung development in 10–18 year olds in Southern California and found decreases in lung function associated with NO_2, acid vapour (organic and inorganic acids) and elemental carbon. Loss of FEV_1 was around 100 mL for boys and girls when comparing the highest to lowest exposures (mean 5–35 ppb NO_2). They concluded that lung development was reduced in adolescents exposed to higher pollution, though to a lesser extent than exposure to cigarettes.

4.8. The APHEA (Air Pollution and Health, a European Approach) study[10] looked at hospital admissions in 5 European cities and concluded that ozone had the only convincing relationship with admissions. An increase of 50 µg/m³ of NO_2 was associated with a 2.6% increase in asthma admissions, but this was of borderline statistical significance. There was a 3% increase in admissions with increases in "black smoke" (predominantly in the $PM_{2.5}$ fraction) but total suspended particles had no effect. They looked at combinations of pollutants and did not find a significant effect. This study was widely quoted in the European media but was criticized for problems with standardisation of data collection, including admission data and collection of pollution data in different cities as well as possible sampling bias.[11]

$PM_{2.5}$ and health effects

4.9. $PM_{2.5}$ levels have been found to be more strongly associated than PM_{10} with death and adverse health outcomes[12]. The bulk of elemental carbon and black smoke from diesel exhausts are in the $PM_{2.5}$ fraction while the less toxic larger dust particles are in the PM_{10}. In one study, an increase of $PM_{2.5}$ levels of 50µg/m³ resulted in 1.7 extra deaths per day per 1 million population[13]. The risk of being hospitalized because of increases in $PM_{2.5}$ pollution was roughly the same. The risk is small because there is only a very small proportion of the population with such compromised health that they could be affected in this way. Based on data such as this, the loss of years from a lifetime of exposure to pollution in an urban area in the US has been estimated at 1.8-3 years[13]. However, life expectancy in developed nations continues to increase because of continually improving public health measures and medical care.

4.10. It is unclear to what extent pollution reduces life expectancy in the very frail, i.e. those with little remaining life expectancy. It may be that death is advanced only by days to weeks. However, it has also been suggested that some of the mortality from pollution occurs in people not close to death. If so, such individuals must have a vulnerability that has not been explained or determined[13].

4.11. Simpson and others (2005)[14] reported the results of a meta-analysis of urban air pollution data from four cities in Australia. No statistically significant mortality risk was observed for PM_{10} or $PM_{2.5}$ exposure. However, there was an increased risk of hospitalisation with increased levels of PM. Bennett et al (2007)[15] 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". In Melbourne the standard for air quality is 50µg/m³ for PM_{10} (i.e. the same as NSW). According to EPA Victoria data, Melbourne air quality met the standard on 99% of days for the year studied by Bennett et al.

4.12. Karr et al (2007)[16] studied $PM_{2.5}$ and admissions for infant bronchiolitis in a large study in California and found a small but significant association. For every 10µg/m³ increase in $PM_{2.5}$ there was a 9% increase in admissions. Bronchiolitis is an inflammation of the small airways in infants which has some similarities to asthma. When the study was broken down into particular components of pollution, CO and NO_2 had no effect once adjusted for confounding factors. There was no statistically significant effect of these pollutants in sick infants (particularly those with pre-existing cardiac abnormalities and respiratory disease) but there were only small numbers. Ozone, surprisingly, was associated with a significantly reduced risk of bronchiolitis in the sick infants. They concluded that increasing particulate matter, including vapour droplets generated by diesel engines (known as "black smoke") and elemental carbon, were associated with increased risk of bronchiolitis rather than the gaseous products of combustion.

Components of pollution

4.13. Oxides of nitrogen and sulphur (NO_2, SO_2), ozone and black smoke are the pollutants most frequently associated with respiratory symptoms and asthma in the medical literature.

4.14. It is thought airway damage and inflammation caused by pollutants can facilitate airway hyper-responsiveness to aeroallergens such as pollens. Most studies of asthma have looked at exposures from automotive sources, which are the most significant source of NO_2 in the urban environment.[7] NO_2 itself is thought to be less irritating to the airways than ozone, although it may produce increases in airway tone in predisposed subjects. Studies of asthma and NO_2 have found contradictory effects and there are multiple negative studies. However the evidence seems to suggest short term exposure to increased NO_2 may cause respiratory symptoms in healthy individuals and exacerbations in asthmatics, and long term exposure is a risk factor for asthma development.[17]

4.15. The PEACE (Pollution Effects on Asthmatic Children in Europe) study[18] found no association between levels of NO_2, SO_2 or other measures of pollution on asthmatic subjects exposed to low levels of pollution. The most polluted city studied was Athens with a PM_{10} of 98.8 µg/m³ (compared to suburban Norway having a level of 11.2 µg/m³, the lowest value reported). They also stratified for climatic differences in the cities and found no correlation. Some measures of lung function showed a trend towards improvement following higher pollution days. The only significant negative correlation was with peak expiratory air flow in areas with the highest concentrations of black smoke. They studied over 2000 children so their study was adequately powered to find a very small effect (they aimed to detect a reduction of 0.02% of lung function).

4.16. Chauhan et al (1999)[19] stated that the relationship between lung function test results and NO_2 exposure was inconsistent. Normal and asthmatic subjects showed no change in resting lung function when exposed to NO_2 concentrations of up to 1880 µg/m³ (the current NSW standard is a 1 hour maximum of 246 µg/m³). However, these studies may have been relatively insensitive to show changes in the peripheral airways[7]. Controlled studies are also controversial – studies that have found an effect have not been able to be replicated.[17]

4.17. The APHEA II study[20] looked for an association between SO_2 levels and emergency department visits and found that for every 10 µg/m³/day increase in SO_2 there was a 1.3% increase in asthma admissions. SO_2 levels in the cities studied were 6.8–32.5 µg/m³ (the NSW standard is 60 µg/m³ for an annual average). The original APHEA study[10] found no consistent difference in admissions with increased SO_2 except for a possible association between the rate of admissions of elderly patients with an increase of SO_2 of 50 µg/m³/day. The authors stated in this study that the effect of SO_2 was "small, inconsistent". In another study, workers in an aluminium smelting plant exposed to very high concentrations of SO_2 had no increase in respiratory symptoms[21]. However, asthmatics are often screened out of working in these plants.

4.18. There has been considerable interest in the effects of vehicular traffic on health because the main pollutants thought to cause respiratory symptoms are elemental carbon and black smoke (products of combustion). Pollutant levels are thought to be increased above background levels for up to150 metres around a main road[22] because exhaust fumes dissipate rapidly and are progressively diluted beyond the point source. Most studies have been in children living in proximity to main roads on the assumption that children would be more susceptible. Trucks and diesel fuel seems to have the most impact on respiratory health in these studies, in particular black smoke. Wjst et al[23] found respiratory symptoms increased by 5–10% per 25,000 vehicles per day on the nearby road in Munich.

4.19. Van Vliet et al[24] studied the effects of living close to a freeway on lung function in children in the Netherlands. The motorways studied at that time averaged 80,000-150,000 vehicles per day with 8,000–17,500 trucks. They found no difference in the levels of $PM_{2.5}$ and PM_{10} between 15 and 300 metres away from the freeway, but black smoke was 12.2-14.9 µg/m³ 15 metres from the freeway and NO_2 concentration was 40.4–47.8 µg/m³. The NO_2 levels correlated better with traffic density than black smoke, but their findings suggest that black smoke was primarily responsible for the increase in respiratory symptoms seen. They concluded black smoke came mainly from diesel engines, which suggests that truck traffic was the main reason for the increased respiratory symptoms they found. Other studies have also found that respiratory symptoms in children are related to the level of truck traffic/ diesel on the nearby major road.[25]

4.20. There has been some interest in the possible effects of NO_2 emissions from gas appliances in the home and their relationship to respiratory health [19,26]. Two studies[19,26] suggested that peak exposure to NO_2 may be more significant than average exposure and that this may account for the lack of positive findings to date. It has been reported that the exposure from a gas stove is around 30 µg/m³.[19] One retrospective study has suggested that NO_2 exposure in the home in the first year of life is associated with wheeze and asthma in older children, however actual exposures were not measured.[27]

4.21. Pilotto et al (2004)[28] studied the effects of removing unflued gas heaters in South Australian schools. They found that in the classrooms where the heater was removed there were fewer respiratory symptoms reported. There was no difference in tests of lung function between the two groups. NO_2 levels in classrooms without gas heaters were 15.5 ppb (parts per billion) and 47.0 ppb in those with them.

4.22. Garrett et al (1998)[26] measured NO_2 concentrations in homes in La Trobe Valley, Melbourne. The rationale for their study was that previous studies had been inconclusive. The median value in the houses studied was 11.6 µg/m³ with the highest value 246 µg/m³ recorded in the only house with an unflued gas heater. Gas stoves, but not other gas appliances or NO_2 sources were associated with increased respiratory symptoms and asthma. Bedroom levels of NO_2 were not significantly associated with an increase in asthma or respiratory symptoms. The risks of gas stoves remained significant even after adjusting for NO_2, suggesting that other factors are also involved in the increased risk associated with gas cooking. The NO_2 concentrations in the houses were low with only 10 houses exceeding the Victorian indoor standard of 115 µg/m³. They concluded that there was no significant association between NO_2 exposure and respiratory symptoms at the generally low levels in the houses studied.

5. COAL DUST, STANDARDS AND HEALTH EFFECTS

5.1. In open cut coal mining the major source of dust is removal of topsoil and overburden and 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 (http://www.edo.org.au/edonsw/site/factsh/fs04_7.php). The percentage of silica in the PM_{10} fraction is likely to be small but varies from site to site depending on the composition of the overburden. If the silica fraction of the overburden is relatively high additional control measures must be introduced to protect the workers. Atmospheric levels of silica have been measured in the vicinity of two mines in the Hunter Valley (ACARP Study, C18026, June 2012) and the worst case values (0-5-1.8 $\mu g/m^3$) were well below the standard for an annual average (3.0 $\mu g/m^3$, below which a working lifetime of exposure should not cause silicosis). There will also be some particulate matter emitted in the exhaust of various mining and earth moving equipment and trains powered by diesel engines. There will also be some dust and gases released by blasting (for details see below).

5.2. As mentioned above, the potential for these dusts to cause adverse health effects depends on the concentration in the air breathed. The concentration is highest at the point where the dust is liberated. As the dust disperses it occupies a larger and larger volume of air so that, in theory, the concentration of the most dispersible dust will decrease according to the cube of the distance from the source. Thus, if a worker standing 10m from the dust source was exposed to a concentration of $1000\mu g/m^3$, then a person standing 1,000m from the source would be exposed to a concentration of 0.001 $\mu g/m^3$. The real situation is more complicated and will vary with dust composition and physical nature, if there are multiple sources of dust spread over an area, with humidity and atmospheric conditions and if there is a wind blowing in a particular direction. The size of dust particles which fall to the ground varies as distance from the mine increases. Within the first 5km much of the visible dust settling on any exposed surface would not be in the respirable fraction and therefore harmless. The finer particles, which would compose the bulk of the respirable fraction (ie the PM_{10}) tend to carry much further (tens of kilometers) before they settle.

5.3. There are occupational health standards (eg the Australian Safety and Compensation Council, ASCC, which has been adopted by most states) that must be complied with for workers exposed to various kinds of dust. The level for inhalable dust in all coal operations is $10mg/m^3$ (or $10{,}000\mu g/m^3$). If the dust contains quartz, the exposure limit for the worker is reduced substantially. The current standard for quartz-containing dust is $100\mu g/m^3$ of respirable silica for open cut mines or $120\mu g/m^3$ for underground mines (but some organisations have recommended that the underground criterion should be the same as the open cut). Other inhalable dusts have a specified limit of 2.5 mg/m^3 ($2{,}500\mu g/m^3$). These are the levels that the 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. 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. At the levels quoted above a very low percentage of workers would develop radiographic changes or abnormalities of lung function and even fewer would develop symptoms.

5.4. Thus, the environmental standards for an annual average of less than $30\mu g/m^3$ of PM_{10} and a 24 hour average of less than 50 $\mu g/m^3$ are well below the occupational health standards for dust containing a high proportion of silica, one of the potentially most harmful particulates, but which comprises a relatively small fraction of suspended particulates in most areas including the vicinity of an open cut coal mine. The environmental standards are less than 1% of the specified level for workers exposed to dust in coal operations.

5.5. Workers are regularly monitored for exposure to respirable dust and quartz at underground mines. If exposures exceed the limits described above operations must be reviewed and dust levels reduced. Workers at open cut mines are not continuously monitored because the levels have been found in the past to be within the appropriate guidelines.

6. LUNG DISEASE IN COAL MINERS

6.1. The medical literature on lung disease in coal workers is dominated by reports from underground mines because exposures to high levels of dust are dramatically increased by poor ventilation, which can occur at the blind end of tunnels (For review see ref 29). The majority of inhaled dust is removed in mucus, which lines the bronchi and is removed from the lungs by cilia (tiny hair-like projections from the lining cells which push the mucus along) or is coughed up if the amounts are excessive. Exposure to high levels of dust causes an increase in the production of mucus, which is a natural defence mechanism. The dust which gets into the alveoli is taken up by cells in the lungs called macrophages. These are either transported out of the lungs by the mucus or into the lymph glands in the centre of the chest or in the lymph channels on the surface of the lung. Pure coal dust can remain in those sites for many years causing relatively mild tissue reaction whereas silica creates an inflammatory response resulting in fibrosis and progressive scarring of the lungs (for review see reference 29).

6.2. The lungs have a very large capacity to remove dust, but this capacity can be exceeded. As the dust accumulates, it becomes visible on a chest x-ray. In the past, a proportion of coal workers and quarry workers exposed to dust levels well in excess of those listed above as current standards, would develop radiographically apparent dust accumulation over a period of 10-20 years[29] and some would develop disabling pneumoconiosis (see below). There was considerable variation between workers possibly related to individual susceptibility, smoking and other factors. At the time the abnormal x-ray was detected, there was usually no loss of breathing capacity or any disability. Removal of the worker from excessive exposure at that time would usually prevent the development of disability.

6.3. Most research into the effects of coal dust on human health has investigated disease in underground coal miners and has shown that disease secondary to coal mining mainly results from inhalation of particulate matter during the mining process [30]. Coal workers are at risk of developing coal workers pneumoconiosis, progressive massive fibrosis, silicosis and chronic obstructive pulmonary disease due to coal dust exposure. The Australian standard recommends that miners are not exposed to > 3 mg/m^3 respirable coal mine dust over a 40hr week. The predicted loss of FEV_1 after 40 years working with exposure to 1.5mg/m^3 is 73ml [31]. This is a small fraction of the normal FEV1 which is between 3 and 6 litres depending on height, age and sex. Symptoms of breathlessness are not usually noted until the FEV1 has decreased 30-40% (ie more than a litre). In longwall mining in NSW, miners working closest to the shearing process, which was the highest exposure recorded, had a median exposure of 1.72mg/m^3 [31]. The risk of a miner developing the most severe complication of coal dust exposure, massive pulmonary fibrosis, at exposure levels found in these mines after a working lifetime was 1 – 3% [31]. The amount of quartz in the coal also has a significant impact on the likelihood of lung disease and recent increases in incidence of rapidly progressing lung disease in coal miners around Eastern Kentucky, US, may be due to increased exposure to quartz containing dusts [32]. Seixis et al[33]. studied a large number of miners across several underground mines in the US and found with 15mg/m^3.years (= 1mg/m^3 per year for 15 years) there were increased respiratory symptoms (cough, bronchitis, wheeze, breathlessness) adjusted for confounding factors and a 5.5 ml (0.6 – 10ml, 95% confidence interval) decrease in FEV1. The British Pneumoconiosis Field Research estimated the effect to be about 1 – 1.6 ml FEV1 per mg/m^3.year of dust exposure which falls in the range quoted above[34].

6.4. The relative toxicity of coal dust, silica, varying combinations of the two and diesel fumes has been studied in various animal species and in studies of inflammatory markers in humans (e.g. for review see ref. 34). Silica is far more toxic than coal dust and this is reflected in the threshold limit values (TLVs) allowed for workers exposed to these dusts. While coal dust elicits inflammatory responses in rats and mice the effects are less pronounced in monkeys and humans[34]. The levels of bioavailable iron vary from region to region and have been reported to predict the toxicity of coal dust in animal experiments. This has been proposed as a mechanism for the variable rates of pneumoconiosis at different US mines with higher rates in Pennsylvania. However, in an analysis of lung inflammatory cell counts from bronchoalveolar lavage in coal miners and non-miners, quartz dust was a significant predictor of pulmonary inflammation and radiographic category of simple CWP while cumulative coal dust exposure did not significantly add to those predictions[34]. It is now generally accepted that, although silica is a known carcinogen, coal miners do not have excess rates of cancer (in particular lung or stomach cancer). It has been hypothesised that the inhibiting effect of coal dust on cytochrome activity might, at least partly, mitigate the carcinogenic effects of silica and tobacco smoke (for review see ref. 34).

Open cut coal mines

6.5. There are relatively few studies that specifically deal with workers in open cut coal mines. This is presumably because the known concentrations of inhaled dust are much lower than in an underground mine. The available data indicate that open cut coal miners are less likely to develop respiratory disease[35] than underground workers but they are still at risk of developing coal workers pneumoconiosis if they have worked at the coal face[36]. However the studies date from the late 90s, reporting exposures from the 1970s and 80s, and reflect dust and fume exposures much higher than would be permitted today. This is because 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 processes were not generally undertaken.

6.6. One study published by Love R G et al[37] examined a large number of workers employed at a number of sites in the United Kingdom. In this study, exposure levels were high by comparison with those generally found in Australian mines. However, none of the dust samples collected from the workers exceeded the recommended exposure limits for total dust of 10mg/m^3 and 99% of all silica concentrations were below the (then) recommended exposure limit of 400μg/m^3 (ie higher than the current standard of 100μg/m^3). The prevalence of 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. 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.

Diesel fumes

6.7. The potential health effects of diesel emissions are of great interest for coal workers, particularly since high concentrations of exhaust fumes may be encountered in some poorly ventilated underground mines. Diesel emissions are also one of the main sources of PM_{10} and $PM_{2.5}$ in urban pollution. Numerous studies show that exposure to excessive concentrations of fine respirable particles from diesel exhausts increase respiratory symptoms, decrease lung function, increase rates of hospitalisation and might be associated with an increased risk of lung cancer in workers with prolonged heavy exposure[38] (see also detailed discussion below).

6.8. Gases such as carbon monoxide, sulphur dioxide and nitrogen dioxide are also contained in diesel exhaust fumes. Diesel fumes are irritating in high concentrations and can produce a range of upper and lower respiratory tract symptoms. They do not cause asthma, but they might aggravate or exacerbate pre-existing asthma[4]. Animal experiments appear to support the contention that particulate emission from diesel engines may induce cancer. The literature on this subject in humans is not conclusive with more than half of the studies to date showing no significant association[39]. Two studies with different methodologies showed that underground coal miners on average actually had a lower than normal risk of developing lung cancer[29,38] (for review also see ref. 34). This may be because coal miners have less opportunity to smoke than most other blue collar workers. Coal dust may also moderate the carcinogenic effect of silica[34] (also discussed above).

6.9. Because of the significant potential for producing respiratory symptoms, the United States EPA has invoked standards for the level of total particulate emission from diesel engines at a level of 5$\mu g/m^3$. A truck driver might be exposed to peak levels up to 4 $\mu g/m^3$ and a tractor operator might be exposed to higher concentrations. Levels increase when the engine is under load. Most of these particles would be in the $PM_{2.5}$ range. These values were introduced to protect workers in underground mine situations where exposures can be very heavy (levels > 1000 $\mu g/m^3$ have been recorded underground).

6.10. In the open cut situation, there are no mandatory levels or monitoring programmes for diesel emissions because it is believed that the fumes will disperse rapidly into the atmosphere and levels will become undetectable within a relatively short period of time or distance from the point source. In studies of health effects of living close to a motorway it has been reported that pollutants from exhausts are increased over a distance of up to 150m[22]. There has been a substantial improvement over the past 20 years in both diesel motors and in diesel fuel. The former burn the fuel more efficiently and have more appropriate filters and emission control devices. The fuel is being progressively purified to contain lower amounts of sulphur. The allowable sulphur in diesel fuel is required to decline further over the next few years.

7. SUSCEPTIBLE INDIVIDUALS MAY BE AT INCREASED RISK

7.1. It is possible that an individual may develop symptoms with exposures *below* the specified criteria due to increased susceptibility. This question has been assessed in underground coal miners, who were followed for a long period of time[40]. They were divided into workers whose lung function was declining rapidly (thought to be susceptible) and workers whose lung function was declining at the normal rate. The groups were matched for age, weight, smoking history and initial lung function. Those with the rapid decline were found to have been more likely to have left the mining industry because of respiratory symptoms. However, the age at leaving the mine was similar for those with accelerated loss of lung function and the reference groups (54 vs 53 years). There were more chest illnesses including chronic bronchitis and asthma among the group with rapid decline of lung function. There was also an increase in risk of death from lung disease and heart disease in the miners with accelerated loss of lung function. However, the age at death was identical between the two groups. In other words, the workers with increased rate of loss of lung function appeared more likely to die of lung or heart problems, while those with normal lung function were more likely to die of other conditions, with the age of death being similar for the two groups.

7.2. These results confirm the concept that some people are inherently susceptible to the harmful effects of exposure to dust and/or fumes. Postulated mechanisms to account for this increased susceptibility include the subject's immune mechanisms or structural features of the lung itself (eg history of asthma or chest infections). It might also relate to the ability of the lungs to process and remove dust and smoking history. Similar results have been found in asbestos workers, grain handlers and dairy farmers[29].

7.3. Asthmatic individuals represent a subset of the population who might be expected to be susceptible to the development of increased respiratory symptoms with exposure to a wide range of airborne substances. Asthmatics are particularly sensitive to allergens such as house dust, pollens and cat fur, smoke and a variety of non specific irritant substances such as paint thinners, bathroom cleaners, perfumes and insect sprays. Exhaust emissions from diesel engines are also likely to trigger episodes of bronchospasm in susceptible individuals as would exposure to exhaust fumes from a petrol driven car and a range of other substances. Any such effect is likely to be smaller in a well controlled asthmatic taking prophylactic or preventive medication.

7.4. Employers have a responsibility to ensure that the most susceptible members of their work force do not come to harm. Environmental health planners have a similar responsibility to residents in the community. Therefore, criteria levels are set conservatively. The susceptible individuals described above were exposed to very high dust and fume levels working underground in a British coal mine. It seems unlikely that even highly susceptible individuals living some distance from an open cut coal mine or coal loading facility would have a significantly increased risk of illness as a result of exposure to coal dust or diesel fumes.

8. HEALTH OF COMMUNITIES NEAR COAL OPERATIONS

8.1. The major pollutants from open cut mining are dust particles including total suspended particulate (TSP) and PM_{10} - particles with aerodynamic equivalent diameter < 10μM. PM_{10} particles in urban pollution are known to have a health impact (discussed above). PM_{10} probably constitutes 30 to 50% of the particulate matter produced by open cut coal mines[41,42]. A variable portion will be derived from coal and overburden depending on the operation at the time and the nature of the mine. The highest concentrations of dust are at the coal face and the pit with 50% less concentration out of the pit. Concentrations diminish with increasing distance from the mine in a power function [41,43]. The larger particles are mainly deposited within a few km of the mine area[43]. The $PM_{2.5}$ fraction, which is the most harmful component of urban pollution, may be carried tens of km before settling. Whereas $PM_{2.5}$, largely derived from combustion of fossil fuel including diesel, constitutes the majority of PM_{10} in urban pollution it is a relatively small fraction of the dust liberated by open cut coal mining, transport and handling. This is because it takes enormous energy to grind or smash quartz and coal to such a small size.

Health effects in communities exposed to coal dust

8.2. There have been relatively few epidemiological studies of exposures in communities surrounding operations likely to result in emissions of coal dust. Notwithstanding, there is 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 40km of unsealed roads[44] rather than from the mining and coal handling and transport.

8.3. Children exposed to coal dust from a coal loader at a sea port in Liverpool, England were compared with those in nearby districts and not exposed [45]. The port also handled grains and other materials. There was increased absenteeism from school in the exposed population but there was no increase in hospitalizations for asthma and 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. Average dust deposition in the area was 6 g/m^2/month, a high figure. The applicable OEH criteria are a maximum increase of 2 g/m^2/month and a total level of 4 g/m^2/month. The T4 project is predicted to produce dust deposition of 0.01 to 0.06 g/m^2/month at the various assessment locations. The total cumulative dust deposition, including background levels and those related to future developments, is predicted to range from 0.6 to 3.3 g/m^2/month. The T4 project will produce on average less 1% of the dust levels reported for the Liverpool facility so this data indicates that there should be no effect on the respiratory health of children in the vicinity of T4.

8.4. Pless-Mulloli et al[46] documented children's health in a region in northern England where there was agriculture prior to open cut coal mining in the 1990s. There was no other heavy industry in the area to confound the results[47]. The greatest difference in PM_{10} readings between the mining and control communities was 35$\mu g/m^3$ over a 30 minute period with a mean in open cast communities of 17$\mu g/m^3$ and control communities 14.9 $\mu g/m^3$ over 6 weeks. The predominant mineral in the PM_{10} was shale dust, presumably from the overburden. 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. A third report[48] on the same population looking at asthma showed 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 and rates of long term illness. Asthmatic children in open cast communities 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.

8.5. In recent years a number of articles have appeared reporting an association between a range of adverse health outcomes and residence near coal mining operations in the Appalachian mountains of West Virginia[49,50,51]. The range of disorders reported to be increased in areas with high levels of coal production include self reported poor health status, cardiopulmonary disease, chronic obstructive pulmonary disease, "black lung", kidney disease and hypertension[49]. Cardiovascular mortality was reported to be increased in regions with mountaintop mining but not in those with other methods of coal mining[50]. Low infant birth weight was also reported as an association even after adjusting for the known factors which had the strongest associations (i.e. maternal smoking, medical risk factors, low education and socio-economic status)[51]. Poor dental health has also been reported. The authors discussed the limitations of these studies and the 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.

8.6. These results, if confirmed, suggest there may be special problems related to the methods of mountain top mining and the geography and population health in the Appalachian mountains. Several reports have raised concerns about possible contamination of ground water from mining processes and the practice of filling valleys with the overburden from the mountain tops. In particular, there are concerns about levels of lead, iron, manganese and, possibly, selenium and arsenic in streams and ground water[52]. The Appalachians are one of the poorest regions in the US with high levels of poverty, inadequate health care and substandard housing[52]. The poorest live in the valleys near mountain top mines often squatting in abandoned houses or trailers without access to reticulated water and sewerage. The winters are harsh and many burn coal for heating in poorly vented stoves. Most of the adverse health effects reported in the studies cited above are also associated with poor socio-economic status and it would be difficult to completely adjust for this potential bias. However, it is possible that exposure to contaminated drinking water and smoke from burning biomass fuel could contribute to the observed associations. The T4 project cannot be compared with mountaintop coal mining in the Appalachians. First, it is not a mining operation so many of the postulated mechanisms for the observations are not relevant. Second, any water runoff will be captured and managed through the surface water management system and will not have any potential to contaminate drinking water supplies. Third, the surrounding population differs substantially from that in the Appelachians having access to adequate housing, utilities, public health and medical care.

8.7. Reynolds et al[53] exposed rats to a surrogate "dust" made to the same composition of dust from Welsh open pit coal mines. They found no acute lung toxicity from high concentrations of open cut coal dust, in contrast to the rats which they exposed to quartz dust, as a positive control of a known lung irritant. Surface workers in low exposure occupations in the coal mining industry have been shown to have no increase in mortality from silicosis or chronic obstructive pulmonary disease (COPD)[54], so it is unlikely that individuals in the community with even lower exposures than workers would develop silicosis or COPD.

Discrepancy between perceived and measured health effects

8.8. Moffatt et al[47] studied the perceptions of people from four communities in Northern England about the effect of mines on asthma status and parental concerns for their children's health before and after the mines had opened. At the same time epidemiological studies[46, 48] (see 7.3 above) were performed to assess whether there was a demonstrable health effect. The study found that most health fears anticipated prior to opening the mines were not realised, although there was a demonstrable and perceived increase in dust levels. Parents expressed uncertainty about health outcomes in the planning stage of the mine and did not believe that there would be no health impact prior to the mine opening. After the mining commenced, out of 31 interviews conducted only one parent felt that their family's health had been affected. After the land had been remediated the respondents felt that it was not the same as before, but felt that there had been some community benefit in those regions where the mining company had built parks or other community utilities. The study makes the point that people believe their experience rather than scientific knowledge even when science contradicts their attribution of causality. "At the heart of the expert-lay divide in environmental health research is the gulf between objective, generalisable knowledge and situated, subjective knowledge. Environmental health studies that utilise both these methods have a better chance of arriving at conclusions that are meaningful to the scientific and affected communities".

9. AIR QUALITY ASSESSMENT FOR THE T4 PROJECT

9.1. The air quality assessment for the T4 project was prepared by ENVIRON. Predictions for the impact of the project on ambient air quality were based on modelling of the dispersion of emissions from the project cumulative to background emissions and the predicted emissions from proposed future developments in the area. The background air quality data were taken from a number of air quality and weather monitoring stations in the greater Newcastle region surrounding the site of the project. Chemical analysis of $PM_{2.5}$ at Mayfield revealed the major components to be seaspray, soil, black carbon, ammonium sulphate and organic compounds. The black carbon is believed to be mainly generated from combustion processes but could also include carbon from coal dust. Between 1993 and 2010 there was a decline in the percentage of elemental carbon and soil while there was an increase in the percentage of ammonium sulphate and sea salt. This composition is expected to be typical of the areas surrounding the Project because similar compositions have been measured in the upper Hunter and in Sydney.

9.2. Microscopic analysis of dust deposited in Stockton and Fern Bay between 2006 and 2010 found that coal particles comprised between 5% and 16% of the total dust. Deposited dust is predominantly comprised of large particles so the percentage of coal dust in the PM_{10} and $PM_{2.5}$ fractions would be less than the above figures.

9.3. Predictions of emissions from the T4 project were undertaken at three time points including construction, construction combined with operation at a nominal throughput of 25 Mtpa, and operations at the nominal maximum throughput of 70 Mtpa.

9.4. Cumulative annual average TSP concentrations and dust deposition rates are predicted to remain below the applicable assessment criteria at all of the assessment locations surrounding the project. The cumulative TSP concentrations are predicted to be between 45 and 59% of the OEH criterion of 90 μg/m³. Cumulative dust deposition rates are predicted to be between 15 and 82% of the OEH criterion of 4 g/m²/month.

9.5. Annual average cumulative PM_{10} concentrations are predicted to be between 59% and 71% of the OEH criterion of 30 μg/m³. Infrequent exceedances of the 24-hour average PM_{10} criterion have been historically recorded in the region, generally due to regional events such as bushfires or dust storms. For the baseline dataset used (ie without the T4 Project's contribution), the concentrations are predicted to exceed the OEH criterion of 50 μg/m³ on one day over a one-year period at all the assessment locations, but by a relatively small amount. The National Environment Protection (Ambient Air Quality) Measure (NEPM) goal is no more than five exceedances per year. The T4 project is not predicted to cause any exceedances additional to those that would already be caused by background levels. It was estimated that the T4 project contribution on the baseline exceedances days would be less than 1.3 μg/m³ for all assessed scenarios. The total levels on the exceedance days are predicted to be in the range of 50 to 80 μg/m³ which represents a relatively small increase in risk to health. Mostly the levels would be below 25 μg/m³ and concentrations between 30 and 40 μg/m³ would occur less than 5% of the time.

9.6. Cumulative annual average $PM_{2.5}$ concentrations are predicted to be between 60 and 71% of the ambient air quality standard of 8 μg/m³. The maximum cumulative 24-hour average $PM_{2.5}$ concentrations are predicted to be between 76 and 84% of the ambient air quality standards of 25 μg/m³. Exceedances of this standard have been recorded in the region due to events such as bushfires and dust storms. The T4 project related increments are predicted to be small compared with background peaks and should not cause additional exceedances.

9.7. Although no additional exceedances were predicted for the T4 Project based on the air quality assessment, it is conceivable that the use of a different base case year might result in an additional exceedance(s). The magnitude of such an exceedance will however be small given the limited increment predicted due to the T4 Project. It is my opinion that any Project-related exceedances will be so small that the heath risk will be minimal. The population exposed would not be large enough to observe a measurable increase in morbidity or mortality.

9.8. Combustion related emissions are predicted to be below the applicable OEH criteria at all assessment locations and for all assessed scenarios. The modelling assumes worst-case scenarios and includes contributions from docked ships and idling trains in the T4 project area, and from future developments proposed in the region where applicable. It is my opinion that diesel emissions at the T4 site will be well within the EPA guidelines. Levels at distances of 150 m or more from the sources will be very low.

9.9. Fugitive emissions from coal trains were also modelled. Based on a screening analysis of PM_{10} concentrations due to dust entrained from coal trains on-route to the T4 project area, given a coal delivery rate of 70 Mtpa, and taking into account results from an ARTC pilot monitoring study (see below), dust entrainment was not projected to result in significant air quality impacts along the rail corridor. Peak 24-hour PM_{10} concentrations due to coal trains were estimated to range between 1.5 and 6.4 $\mu g/m^3$ at a distance of 20 m from the rail corridor assuming a coal delivery rate of 70 Mtpa. The high figure is a worst-case scenario which is unlikely to occur. Dust concentrations decrease rapidly with distance from the source of emission. There are relatively few residences within a distance of 20 m from the rail corridor. The impact of dust generated by passing trains on air quality will be heavily influenced by background levels which are likely to vary considerably along the corridor depending on proximity to roads, industry and urban areas.

9.10. The Australian Rail Track Corporation (ARTC) was required by the EPA to undertake a pollution reduction program. This required the ARTC to quantify the level of dust including fine particulates generated from rail transport including coal trains in the Newcastle area rail corridor. This study was completed in September 2012 and will be used by the EPA to determine whether mitigation strategies are required such as covering the loads or other measures to reduce fugitive dust emissions. Monitors were set up at a distance of 3m from the tracks in Metford and Mayfield and dust levels were measured and compared between different types of train and when no trains were passing. While there some differences between the two sites, passenger trains produced the highest *peak* levels of 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. The background (no train) levels were similar to those at other monitoring stations in the Hunter region. The increases in PM_{10} and $PM_{2.5}$ with trains were 2.2 – 4.8 $\mu g/m^3$ and 0.5 – 1.2 $\mu g/m^3$ respectively for Mayfield and Metford. These values are comparable to those derived from the modelling (see Section 9.9).

10. CONCLUSIONS

10.1. The Air Quality Assessment for the T4 Project indicates that maximum *baseline* 24-hour average PM_{10} concentrations (i.e. without any contribution from the T4 project) exceed the relevant OEH criteria on one day of the year. This is within the national criteria which allow up to 5 exceedances per year. The times when the criteria might be exceeded are likely to be when there are high background concentrations of PM_{10} (e.g. due to bush fires or dust storms). Predicted cumulative levels on the exceedance days are in the range of 50-80 $\mu g/m^3$ which represents a relatively small risk to health. The cumulative impact of the T4 project and other proposed developments was not predicted to cause any additional exceedances of the criterion; the T4 Project represents only a very small proportion of total emissions on the exceedance day.

10.2. The dispersion modelling data indicates that $PM_{2.5}$ levels would be at acceptable levels in the surrounding districts and residential areas even if the maximum 24-hour average PM_{10} values exceeded the criteria. Occasional exceedances of NEPM advisory reporting goals for $PM_{2.5}$ have been recorded in the past and it is likely that cumulative levels will occasionally exceed these goals in the future as a result of dust storms and bushfires.

10.3. TSP and dust deposition rates are predicted to be within the relevant criteria.

10.4. Using the dispersion modelling data for TSP, PM_{10} and $PM_{2.5}$, dust levels will be at acceptable levels in terms of health risk in the surrounding residential areas even if the PM_{10} values exceed the 24-hour average criteria from time to time. The contribution from the T4 Project is predicted to be a small percentage of the total and not be the cause of exceedances. In my opinion there will no measurable increase in morbidity or mortality as a result of the T4 project.

10.5. Fumes and gases from diesel exhausts and ships are likely to disperse further and more quickly in the atmosphere than particulate matter. These are not likely to exceed criteria levels or provide a health risk for residents.

10.6. Dust caused by coal trains was not predicted to result in significant air quality impacts along the rail corridor. Moreover, the impact is similar for passenger and freight trains of all types and the contribution of the trains to the cumulative level is relatively small. In my opinion it is unlikely that the type of dust predicted to be liberated from trains would cause adverse respiratory health effects in nearby residents, even if the OEH levels are exceeded intermittently. Fumes and gases from diesel exhausts are likely to disperse more quickly in the atmosphere than particulate matter. These are not likely to exceed criteria levels.

10.7. In addition, I understand that dust levels will be monitored and a range of other air quality and dust controls will be implemented.

David McKenzie BSc (Med) MB,BS PhD FRACP

REFERENCES

1. Kelsall JE, Samet JM, Zeger SL and Xu J. Air pollution and mortality in Philadelphia, 1974-1988. American Journal of Epidemiology 1997;146:750-762.

2. Samet J, Dominici F, Curriero F, Coursac I, Zeger S. Fine particulate air pollution and mortality in 20 US cities,1987-1994. N Engl J Med 2000;343:1742-9.

3. Brauer M, Hoek G, Van Vliet P, et al. Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children. Am J Respir Crit Care Med 2002;166:1092-8

4. Chen T, Gokhale J, Shofer S, Kuschner W. Outdoor Air Pollution: Particulate Matter Health Effects. American Journal of Medical Science 2007;333:235-43.

5. Pope III C. Air Pollution and Health - Good News and Bad. N Engl J Med 2004;351:1132-4.

6. Linaker C, Chauhan A, Inskip H, Holgate S, Coggon D. Personal exposures of children to nitrogen dioxide relative to concentrations in outdoor air. Occup Environ Med 2000;57:472-6.

7. D'Amato G. Outdoor air pollution, climate and allergic respiratory diseases: evidence of a link. Clin Exp Allergy 2002;32:1391-3.

8. Gauderman W, Avol E, Gilliland F, et al. The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age. N Engl J Med 2004;351:1057-67.

9. Daniels M, Dominici F, Samet J, Zeger S. Estimating Particulate Matter-Mortality Dose-Response Curves and Threshold Levels: An Analysis of Daily Time-Series for the 20 Largest US Cities. Am J Epidemiol 2000;152:397-406.

10. Spix C, Anderson H, Schwartz J, et al. Short-term effects of air pollution on hospital admissions of respiratory diseases in Europe : A quantitative summary of APHEA study results. Archives of Environmental Health 1998 53:54-64.

11. Hasford B, Fruhmann G. Air pollution and daily admissions for chronic obstructive pulmonary disease in six European cities: results from the APHEA project. Eur Respir J 1998;11:992-3.

12. Miller K, Siscovick D, Sheppard L, et al. Long-term exposure to air pollution and incidence of cardiovascular events in women. N Engl J Med 2007;356:447-58.

13. Pope C 3rd. Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? Environ Health Perspect 2000;108(suppl 4):713-23.

14. Simpson R, Williams G, Petroeschevsky A et al. The short term effects of of air pollution on daily mortality in four Australian cities. Aust NZ J Public Health 2005; 29:205-12.

15. Bennett C, Simpson P, Raven J, et al. Ambient fine particulate matter (PM2.5) and respiratory symptoms in Melbourne, 1998 - 2005. Respirology 2007;12(suppl 1):A36.

16. Karr C, Lumley T, Schreuder A, et al. Effects of Subchronic and Chronic Exposure to Ambient Air Pollutants on Infant Bronchiolitis. Am J Epidemiol 2007;165(5):553 - 60.

17. Chen T, Gokhale J, Shofer S, Kuschner W. Outdoor Air Pollution: Nitrogen Dioxide, Sulfur Dioxide, and Carbon Monoxide Health Effects. Am Med Sci 2007;333:249-56.

18. Roemer W, Hoek G, Brunekreef B, Haluszka J, Kalandidi A, Pekkanen J. Daily variations in air pollution and respiratory health in a multicentre study: the PEACE project. Eur Respir J 1998;12:1354-61.

19. Chauhan A. Gas cooking appliances and indoor pollution. Clinical and Experimental Allergy 1999;29:1009-13.

20. Sunyer J, Atkinson R, Ballester F, et al. Respiratory effects of sulphur dioxide: a hierarchical multicity analysis in the APHEA II study. In: Occup Environ Med, 2003.

21. Chan-Yeung M, Wong R, MacLean L. Epidemiologic health study of workers in an aluminum smelter in British Columbia: effects on the respiratory system. . Am Rev Respir Dis 1983;127:465-9.

22. Design Manual for Roads and Bridges. . In: Department of Transport, Scottish Office Industry Department, The Welsh Office a, Ireland. DotEfN, 3) EAs, eds. London: The Stationery Office; 1994.

23. Wjst M, Reitmeir P, Dold S, Nicolai T, Von Loeffelholz Colberg E, Von Mutius E. Road traffic and adverse effects on respiratory health in children. Br Med J 1993;307:596-600.

24. Van Vliet P, Knape M, De Hartog J, Janssen N, Harssema H, Brunekreef B. Motor Vehicle Exhaust and Chronic Respiratory Symptoms in Children Living near Freeways. Environmental Research 1997;74:122-32.

25. Brunekreef B, Janssen N, de Hartog J, Harssema H, Knape M, van Vliet P. Air Pollution from Truck Traffic and Lung Function in Children Living near Motorways. Epidemiology 1997;8:298-303.

26. Garrett M, Hooper M, Hooper B, Abramson M. Respiratory Symptoms in Children and Indoor Exposure to Nitrogen Dioxide and Gas Stoves. Am J Resp Crit Care Med 1998;158:891-5.

27. Phoa L, Toelle B, Ng K, Marks G. Effects of gas and other fume emitting heaters on the development of asthma during childhood. Thorax 2004;59:741-5.

28. Pilotto L, Nitschke M, Smith B, et al. Randomized controlled trial of unflued gas heater replacement on respiratory health of asthmatic schoolchildren. International Journal of Epidemiology 2003;33:208-14.

29. Parkes WR. Occupational Lung Disorders, 3rd edition 1994. Butterworth-Heinemann Oxford. ISBN 0 7506 1403 X.

30. Finkelman RB, Orem W, Castranova V, et al. Health impacts of coal and coal use: possible solutions. International Journal of Coal Geology 2002;50:425- 43.

31. Kizil G, Donoghue A. Coal dust exposures in the Longwall mines of New South Wales: a Respiratory Health risk assessment using joint coal board exposure data from 1985 - 1999. In: centre Tmisah, ed. Queensland mining industry health and safety conference; 2001.

32. Gamble JF, Reger RB, Glenn RE. Rapidly Progressing Coal Workers Pneumoconiosis as a Confounding Risk Factor in Assessing Coal Mine Dust Safe Exposure Levels. J Clinic Toxicol 2011:S1 - 003.

33. Seixas NS, Robins TG, Attfield MD, Moulton LH. Exposure-Response Relationships for Coal Mine Dust and Obstructive Lung Disease Following Enactment of the Federal Coal Mine Health and Safety Act of 1969. American Journal of Industrial Medicine 1992;21:715-34.

34. NIOSH (2011). Coal Mine Dust Exposures and Associated Health Outcomes: A review of information published since 1995. DHHS (NIOSH) Publication No. 2011 - 172.

35. Jacobsen M, Rae S, Walton WH. The relationship between dust exposure and pneumoconiosis in British coal mines. In: Walton WH, ed. Inhaled Particles Surrey: Unwin Brothers; 1970:903 -17.

36. Amandus HE, Petersen MR, Richards TB. Health status of anthracite surface coal miners. Arch Environ Health 1989;44:75-81.

37. Love RG, Miller BG, Groat SK et al. Respiratory health effects of open cast coal mining: a cross sectional study of current workers. Occupational and Environmental Medicine 1997;54:416-23.

38. Comstock ML. Diesel exhaust in the occupational setting. Current understanding of pulmonary health effects. Clinical and Laboratory Medicine 1998;18:767-79.

39. Morgan WK, Reger RB and Tucker DM. Health effects of diesel emissions. Annals of Occupational Hygiene 1998;42:63 65-6.

40. Beeckman LF, Wang ML, Petsonk EL and Wagner GR. Rapid declines in FEV1 and subsequent respiratory symptoms, illnesses and mortality in coal miners in the United States. American Journal of Respiratory and Critical Care Medicine 2001;163:633-39.

41. Trivedi R, Chakraborty MK, Sangode AG, Trewary BK. Study of air pollution load assessment around opencast coal project in India. IJEP 2009;30:198 - 206.

42. Ghose MK, Majee SR. Assessment of the status of work zone air environment due to opencast coal mining. Environmental Monitoring and Assessment 2002;71:51-60.

43. Jones T, Blackmore P, Leach M, Berbube K, Sexton K, Richards R. Characterisation of airborne particles collected within and proximal to an opencast coalmine: South Wales UK. Environmental Monitoring and Assessment 2002;75:293-312.

44. Huertas JI, Camacho DA, Huertas ME. Standardized emissions inventory methodology for open-pit mining areas. Environ Sci Pollut Res Int. 2012; 423:39-46.

45. Brabin B, Smith M, Milligan P, Benjamin C, Dunne E, Pearson M. Respiratory morbidity in Merseyside schoolchildren exposed to coal dust and air pollution. Archives of Disease in Childhood 1994; 70:305-12.

46. Pless-Mulloli T, HowelL D, King A, et al. Living near opencast coal mining sites and children's respiratory health. Occup Environ Med 2000; 57:145-51.

47. Moffatt S, Pless-Mulloli T. ''It wasn't the plague we expected.'' Parents' perceptions of the health and environmental impact of opencast coal mining. Social Science & Medicine 2003;57:437-51.

48. Pless-Mulloli T, Howell D, Prince H. Prevalence of asthma and other respiratory symptoms in children living near and away from open cast coal mining sites. International Journal of epidemiology 2001; 30:556-63.

49. Hendryx MH, Ahem MM. Relations Between Health Indicators and Residential Proximity to Coal Mining in West Virginia. Am J Public Health 2008; 98:669-71.

50. Esch L, Hendryx M. Chronic cardiovascular disease mortality in mountaintop mining areas of central Appalachian states. Journal of Rural Health 2011; 27: 350-357.

51. Ahern M, Mullett M, et al. Residence in coal-mining areas and low birth-weight outcomes. Maternal and Child Health Journal 2011; 15: 974-979.

52. Holtzman, DC. Mountain top removal mining: digging into community health concerns. Environ Health Perspect. 2011; 119:476-483.

53. Reynolds L, Jones TP, Berbube K, Wise H, Richards R. Toxicity of airborne dust generated by opencast coal mining. Mineralogical Magazin 2003; 67:141-52.

54. Tse LA, Yu ITS, Leung CC, Tam W, Wong TW. Mortality from non-malignant respiratory diseases among people with silicosis in Hong Kong: exposure-response analyses for exposure to silica dust. Occup Environ Med 2007; 64:87-92.