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AGL Leafs Gully Power Project Environmental Assessment

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Appendix G

Health Impact Assessment

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Switched on Business.

Natural Gas Power Stations, Air Quality and Respiratory Disease

Authors

A/Professor David McKenzie, BSc (Hons), MB BS (Hons), FRACP, PhD
Head, Respiratory and Sleep Medicine, Prince of Wales Hospital

Dr Elizabeth Brown, BSc, MB BS, FRACP
Research Fellow, Prince of Wales Medical Research Institute

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1. Background

This report was prepared at the request of Minter Ellison on behalf of AGL Power Generation (NSW) Pty Ltd to assess any possible health consequences arising from the proposed Leafs Gully natural gas peaking power station. We were provided with a brief including a letter of instruction dated 19 June 2008 and the updated "*Local Air Quality Assessment for Proposed Gas Turbine Power Station, Appin, NSW"* prepared by URS dated 9 August 2007. The proposal involves the construction of two turbines run on natural gas as a peaking plant with a total capacity of approximately 300 MW. It is anticipated the plant will operate for only 400 hrs per annum at times of peak demand for electricity but the potential air quality impact calculations have been based on the conservative assumption of continuous operation.

The Government sets standards for ambient air quality including criteria levels for common pollutants including oxides of nitrogen (NOx, e.g. NO and NO_2), carbon monoxide (CO), photochemical oxidants (ozone), sulphur dioxide (SO_2) and particulate matter (e.g. PM10). The Government also sets air impact assessment criteria designed to determine whether new or proposed sources of polluting emissions are likely to have a deleterious impact on ambient air quality. Depending on the pollutant several criteria levels have been set to allow for short term peaks which are higher than the annual average criteria. Thus there may be different criteria allowed for various time periods such as 10 minutes, 1 hour, 24 hours and annual average concentrations.

The Air Quality Assessment includes meteorological data relevant to the location such as prevailing wind directions and speeds, temperatures and other factors which will influence the dispersion and concentration of any potential airborne pollutant. It also provides data on the likely concentrations of common pollutants emitted from the plant at various locations in the vicinity, especially at the nearest communities, and data from observations of background levels of common pollutants at sites near the proposed location. The predicted impact from the plant for each of the potential pollutants has been added to the background levels and compared with the criteria. This data appears in Table 7.1. The assessment has used conservative assumptions at each step in the modeling process as discussed below.

In calculations of the impact of the plant it has been assumed the plant will operate continuously rather than for the current estimate of 400 hrs and that both turbines would always be started up simultaneously rather than one at a time. Separate estimates were derived for the conditions of start up of the generators when operating temperatures are low and emissions relatively high and normal operation. There were no data available for the levels of background pollution at the proposed site or in the vicinity of neighbouring communities. As these are semi-rural the levels are assumed to be low. The values used in the report were from Macarthur Station which is in close proximity to heavy vehicle traffic and likely to be substantially higher than in Leafs Gully or Appin. In calculating the impact the highest values were used corresponding to unusual weather events such as a temperature inversion. The worst case scenario would be simultaneous startup of both turbines on a high pollution day and these values are shown in Table 7.1 and have been used in this report to assess potential health effects.

2. Introduction: air pollution and respiratory disease

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 health effects from exposure to particulate matter suspended in the air depend on the inherent toxicity of the particle, its concentration and the proportion of particles in the size range which can be inhaled into the lungs (1-10μ) and the susceptibility of the individual inhaling the material.

The NSW Government monitors air quality and, among other substances, measures the concentrations of particulates less than 10μ in diameter and those less than 2.5μ (commonly reported as PM10s and PM2.5s). The Government sets criteria (or standards) for pollutants which are believed to be levels below which there are acceptably low risks for adverse health effects. For example, the levels for PM10 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 and other values are reviewed in this report.

The standards set are conservative levels based on epidemiological data. They are not necessarily generalisable to communities other than where the study was done. The standards are not thresholds beyond which the statistical risk of adverse health consequences is significant. In some cases, the level might have to rise considerably before any increase in adverse health effects could be reliably demonstrated. The standard is intended to be at a level where the most susceptible individuals would be unlikely to be harmed although they might develop symptoms. Thus, it is not a threshold for symptoms for most individuals. These standards are substantially below the threshold limit values, which have been mandated as allowable exposures for workers in occupational health and safety standards.

There can be no doubt that very high concentrations of airborne urban pollution are associated with increased cardiovascular and respiratory disease (particularly asthma) and increased mortality on high pollution days. The elderly and patients with coexisting severe cardiovascular or respiratory disease are most at risk of dying.

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]. Long-term exposure to excess levels of such pollution is thought to induce oxidative stress, inflammation, thrombosis, progression of atherosclerosis and, possibly, altered autonomic function of the heart. Only a small proportion of the population is believed to be at risk of adverse outcome from short-term exposure to pollution: e.g. the elderly, those with cardiopulmonary disease, infants and asthmatics. Most studies conclude that, although an individual's risk when exposed to air pollution is small, the large populations exposed in cities result in a significant burden of illness[3, 4].

In rural areas the bulk of airborne particulates is likely to be dust released from farming operations or road works and would include mineral dust, grain dust, pollens and, in winter, smoke from wood fires. There would be much lower concentrations of the toxic gases and fumes found in the petrochemical smog of cities.

It is not certain how exposure to high levels of urban pollution contributes to excess mortality. Chen *et al* (2007)[3] 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."

Studies of air pollution and disease are often beset with methodological problems which have been identified and discussed in the scientific literature. These include:

- 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.
- There is a strong negative bias inherent in studies of pollution. The public and researchers intuitively feel pollution is a bad thing and, although this does not mean that they are not right, it means that study design and interpretation of data can be biased.
- Most studies have looked at one city only, so the conclusions may not be able to be generalised to other cities.
- Outdoor air sample data collected by government agencies have been used in many studies which have sought correlations between the levels of various pollutants and symptoms, respiratory illness and lung function in children. However, outdoor data has been shown not to correlate with a child's actual exposure, which can be measured accurately by a personal sampling device, because there are additional sources of air pollution indoors such as allergens from house dust mites, pets and mould and fumes from heating and cooking appliances.[5]
- Publication bias means that positive studies are more likely to be submitted to journals by researchers and more likely to be accepted for publication.
- Climate and local factors including levels of sunshine and aeroallergens may have a confounding effect. Most studies do not take climate differences into account[6]. Thus, results of studies in European or North American cities may not be applicable to a hot, dry and dusty nation like Australia.
- Epidemiological studies have an inherent problem in that the observed effects can be biased by underlying associations – the exposure and outcome could be both affected by a confounding variable.[7] For example, smoking rates could be higher in an area with higher pollution.

In order to overcome some of these potential problems this report focuses on larger studies published in reputable journals.

3. Literature review of potential health impacts caused by emissions from gas fired power stations.

A search of the medical data bases failed to locate any reports of adverse health outcomes specifically attributed to gas fired power stations even though many such stations are in use in relatively close proximity to populated areas in Australia and elsewhere, in some instances less than 500 m from the nearest residence.

In the section below the levels of urban pollution and types of exposure which are believed to cause disease are discussed, along with sources of exposure and possible confounding factors. The review then examines the possible health effects of the proposed Leafs Gully natural gas peaking power station relating the potential average and worst case emissions to the published studies of urban pollution. The predicted emissions are added to measurements of background pollution in the region. It should be emphasized that the nature of particulate pollution in a rural setting would be likely to be very different from urban pollution which is dominated by emissions from vehicle exhausts (see introduction, above).

3.1 Health impacts caused by urban pollution.

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 (NO and NO_2), carbon monoxide (CO) and sulphur dioxide (SO_2). The health effects from exposure to particulate matter suspended in the air depend on the inherent toxicity of the particle, its concentration and the proportion of particles in the size range which can be inhaled into the lungs (1-10μ) and the susceptibility of the individual inhaling the material.

The NSW Government monitors air quality and, among other substances, measures the concentrations of particulates less than 10μ in diameter and those less than 2.5μ (commonly reported as PM10s and PM2.5s).

PM2.5 and mortality

PM2.5 levels have been found to be more strongly associated with adverse health outcomes than PM10.[8] In one study, the authors reported that an increase of PM2.5 levels of 50μg/m³ above background levels resulted in 1.7 extra deaths per day per 1 million population.[9] The risk of being hospitalized because of PM2.5 pollution was roughly the same. The risk is considered to be small because there is only a very small proportion of the population with such compromised health that they could be affected in this way. 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. However, life expectancy in developed nations continues to increase in spite of increases in some forms of pollution because of continually improving public health measures and medical care.

It is unclear to what extent pollution reduces life expectancy in the very frail; that is those with little remaining life expectancy. It may be that death is advanced only by days to weeks, although some very frail individuals have been known to survive longer than expected in the absence of a final insult (which is often an infection such as pneumonia). By contrast, 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 is not obvious or able to be quantified in medical terms.[9, 10]

PM 2.5 and respiratory illness

Bennett *et al* (2007)[11] studied the effect of PM2.5 in Melbourne in adults and found no significant association with most respiratory symptoms. Paradoxically they found a "protective" effect for cough and cough with phlegm on the morning of higher pollution days. Part of their conclusion was that "ambient PM2.5 in Melbourne may currently be too low to have a detectable effect on respiratory symptoms". In Melbourne the standard for air quality is set at 50μg/m³ for PM10 particles (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 Bennet *et al*.

Karr *et al* (2007)[12] studied PM2.5 and admissions for infant bronchiolitis in a large study in California and found a small but statistically significant association; for every 10μg/m³ increase in PM2.5 above background there was an increased odds ratio of 1.09 (i.e. 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 and ozone was found to be "protective", which was the opposite of the study hypothesis. The authors also found 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 so it is difficult to make a conclusion on these findings. Ozone, surprisingly, was associated with a significantly reduced risk of bronchiolitis in the sick infants. Thus, the authors concluded that increasing particulate matter is associated with increased risk of bronchiolitis rather than gaseous products of combustion. One possible explanation for the apparent effect of particulates but not other measures of pollution is that the relevant particles were aeroallergens rather than products of combustion.

PM10 and cities in the US and Europe

Samet *et al*[1] examined pollution levels in 20 US cities between 1987 and 1994 and found the average PM10 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 PM10, which is similar to findings of other studies. However they also looked at CO, NO_2 and SO_2 (adjusted for ozone and PM10) and found that there was little evidence these gases had a significant effect on the relative rate of death.

Other studies have concluded that those with advanced heart or lung disease are most at risk from pollution.[4,13] Daniels *et al* (2000)[13] 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. They suggested an estimate threshold of safety for patients with advanced cardiopulmonary disease at around 15μg/m³.

Pollution and lung development and age related decline

Gauderman *et al*[7] 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 forced expiratory volume in one second (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.

Downs et al[14] correlated the decline in lung function in adults with pollution exposure and found a 6% annual decrease in FEV1 for every 10 μg/m³ increase in PM10. However, this equated to a trivial 3.1ml decrease per year extra from pollution, which is unlikely to be clinically significant.

PM10 and asthma

The APHEA (Air Pollution and Health, a European Approach) study[15] examined hospital admissions in 5 cities in Europe and concluded that, among the pollutants studied, only ozone had a 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 significance. There was a 3% increase in admissions with a 50 μg/m³ increase in black smoke (which is measured by quantifying the reflectance of all particles captured using a standard filter) but total suspended particles appeared to have no effect. Black smoke is predominantly generated by diesel engines. At the time of this study PM2.5 and PM10 were rarely measured in Europe. The study also examined 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.[16]

Specific pollutants and Asthma

NO_2, SO_2, ozone and black smoke are the pollutants most frequently reported to be associated with respiratory symptoms and asthma in the medical literature. McCreanor[17] et al (2007) studied the effect on asthmatics of two hours exposure to high traffic (Oxford street) or low traffic (Hyde park) in London. They concluded that ultrafine particles and elemental carbon in diesel exhaust were the pollutants most strongly associated with reduced lung function and increased markers of inflammation. There was a less pronounced effect from NO_2.

NO_2

It has been proposed that inflammation and airway damage caused by pollutants can facilitate airway responsiveness to aeroallergens such as pollens. Most studies of asthma have examined exposures to pollution from automotive sources, which are the most significant source of NO_2 in the urban environment.[6] 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. Overall the evidence seems to suggest short term exposure to increased concentrations of NO_2 cause respiratory symptoms in healthy individuals and exacerbations in asthmatics, and long term exposure may be a risk factor for asthma development.[18]

The PEACE (Pollution Effects on Asthmatic Children in Europe) study[19] found no association between levels of NO_2, SO_2 or other measures of pollution and symptoms in asthmatic subjects exposed to low levels of pollution. The most polluted city studied was Athens with a PM10 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 the measurement of peak expiratory flow on day 1 post peak pollution days in areas with high concentrations of black smoke. The study included over 2000 children so it was adequately powered to find a very small effect (they aimed to detect a reduction of 0.02% in lung function).

Chauhan *et al* (1999)[20] reported that the relationship between lung function test results and NO_2 exposure was inconsistent in their review of the scientific literature. 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[12]. Controlled studies are also controversial – studies that have found an effect have not been able to be replicated.[18]

In the study by McCreanor[17] et al (2007) asthmatics were instructed to walk at a leisurely pace for two hours in either Oxford street or Hyde park in London. There was a recovery period of at least 2 weeks between each exposure. The mean level of NO2 was 142 μg/m³ in Oxford street London compared to 22 μg/m³ in Hyde park. They recorded small differences in lung function and in some inflammatory markers but there were no significant differences in clinical symptoms between the two exposures or in the use of asthma puffers in the following week.

SO_2

The APHEA II study[21] examined ambient 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. Average 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[15] found no consistent association of admissions with increased SO_2 except for a borderline significant increase in admissions of elderly patients with an increase of 50 μg/m³/day SO_2. The authors stated that the effect of SO_2 was "small, inconsistent". Workers in an aluminium smelting plant exposed to very high concentrations of SO_2 had no increase in respiratory symptoms.[22] However, asthmatics are often assessed as medically ineligible to work in these plants.

3.2 Other exposures and symptoms and lung disease

Freeways, motorways and pollution

Pollutant levels are thought to be increased above background levels for only 150 metres around a main road.[26] Most studies have examined children living in proximity to main roads because they are thought to be more susceptible to any harmful effects. Combustion products from trucks (i.e. from diesel fuel) seem to have the most impact on respiratory health in these studies, in particular black smoke. Wjst *et al*[27] found respiratory symptoms increased by 5–10% per 25,000 cars on the nearby road per day in Munich.

Van Vliet *et al*[28] studied the effects of living close to a freeway on lung function in children in the Netherlands. Their motorways 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 PM2.5 and PM10 between 15 and 300 metres away from the freeway, but black smoke was 12.2-14.9 μg/m³ 15 metres from the freeway decreasing to levels under 10 μg/m³ 300m away and NO_2 concentration was 40.4–47.8 μg/m³ decreasing to levels around 30 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 or diesel-powered vehicles on the nearby major road.[29]

Natural gas cooking, indoor air pollution and respiratory health

The levels of pollutants in the indoor environment depend on a number of factors: the rate, efficiency and duration of combustion of stoves and heaters, the efficiency of removal mechanisms (i.e. flues) and contamination of the indoor environment from outdoors. The major pollutants of concern from indoor combustion are NO_2 and CO. Most of the SO_2 and ozone in the indoor environment is derived from outdoors[24]. Particulate matter is not usually considered in studies from the developed world, presumably because the levels are relatively low. Particulates may be higher in places where wood or coal is burned.

NO_2

Many authors have concluded that the evidence regarding NO_2 and gas appliances in the home and their relationship to respiratory health is inconsistent.[20, 30] It has been 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 (peak exposure has not been studied to date) [20, 30]. The NO_2 exposure from a gas stove is around 30 μg/m³.[20] 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.[31]

Pilotto *et al* (2004)[32] reported on the removal of unflued gas heaters from South Australian schools. Fewer respiratory symptoms were reported in the classrooms where the heater had been removed, but there was no change in tests of lung function between the two groups.

The NO_2 levels in classrooms without gas heaters were 15.5 ppb and 47.0 ppb in those with them.

Garrett *et al* (1998)[30] measured NO_2 concentrations in homes in La Trobe Valley, an industrial area near Melbourne. The valley's brown coal power stations produce 90% of Victorias electricity and there are often significant levels of visible pollution. 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 a 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 may be responsible for the increased risk associated with gas stoves. The NO_2 concentrations in the houses were low with only 10 houses exceeding the Victorian indoor standard of 115 μg/m³. The authors concluded that there was no significant association between NO_2 exposure and respiratory symptoms at the generally low levels in the houses studied.

CO

No studies were found that investigated indoor CO levels and health effects in the population. Most studies of indoor levels of CO reported either the short term effects of exposure in experimental conditions or the long term effects after CO poisoning. Levels recorded indoors of CO from gas heating and cooking ranged from 5 – 50ppm[24]. The higher levels were found in older houses and those with inadequate ventilation.

Case study – Wagerup Alumina Refinery WA[23]

Aluminium smelters are known to have the potential to cause health and environmental impacts including exacerbations of asthma[24]. They are not comparable to a natural gas power station. This study is included in this review because it comes from a rural area in Australia and illustrates some interesting points about the response of a nearby community to a potentially polluting industry.

Wagerup is 120km south of Perth. Because the bauxite refined is close to the surface, organic matter is processed in the refining, emitting volatile gases when incinerated. The closest towns are 4 and 5km away, one to the north and one to the south. This is comparable with the distance of Appin (4km NW) from the proposed Leafs Gully Project. In 1984 a liquor burner was commissioned giving rise to community and employee complaints due to odour, irritation of mucosal membranes and other health issues including a rise in the diagnosis of multiple chemical sensitivity. Engineering measures were taken to substantially reduce emissions from the smelter. After these measures were taken community complaints were logged and air pollution as well as wind direction was measured. Air dispersion modeling, health risk assessment and air quality monitoring have been reported by Donoghue and Cullen[23] (2007). In addition to the commonly measured pollutants they sampled for 274 volatile organic compounds relevant to an aluminium smelter, including acrolein, aldehydes, ketones and metals and metal oxides.

Health complaints

Once emission reduction was instituted, no health based complaints were recorded from employees of the plant. Complaints from the local population were recorded from 2000 – 2004. 3124 complaints were recorded from 250 locals in this time. 376 (12%) of complaints were related to health, with many complainants registering more than one health problem. 74% of these related to upper respiratory tract symptoms, with 34% of complaints relating to cough. 55% of complainants complained of other symptoms including headache, gastrointestinal symptoms, fatigue, pain, insomnia. One case of multiple chemical sensitivity was reported after emission reduction was undertaken. Asthma rates were not recorded. Rates of complaint increased dramatically when there was discussion of land management by the refinery with the community.

Health effects

Acute and Chronic Hazard Indexes were calculated. These were the ground level concentration divided by the relevant health based guideline from the National Environmental Protection Measure. They found that the risk of acute or chronic hazard was negligible in the residential areas surrounding the refinery.

The maximum recorded 3 minute average NOx was 54µg/m³ing when the wind was from the refinery. The maximum 6 minute average PM10 was 406µg/m³; the maximum PM2.5 was 150µg/m³. SO_2 maximum 10minute average was 40µg/m³. Higher levels were recorded when the wind direction was not from the refinery, suggesting that there were other sources of pollution in the area

Complaints and air quality monitoring

The authors looked for a correlation between frequency of complaints and pollution levels. Pollution levels were recorded 2km from the plant, closer than the nearest towns. 16 locals made approximately half the complaints recorded, so the authors divided the complainants into two groups. Low frequency complainants (< 50 complaints recorded per person) were statistically more likely to complain on days when the wind was from the direction of the refinery when the highest levels of NOx were recorded. There was no correlation with other pollutants. High frequency complainants were more likely to complain when the wind was from the refinery but there was no correlation between the likelihood of a complaint and the level of pollutants measured on those days. It may be that odour was a cause for complaint. Visible pollution was not recorded. Previous studies have suggested that odour is related to perceived health effects[25].

The study authors concluded using predictive modeling based on national and international health guidelines that the health impact of the plant at current levels of emission is negligible.

4. Assessment of potential health impacts caused by predicted emissions from the power station project

Appendix D of the environmental assessment dealing with local air quality provides historical measurements of background levels of common pollutants at several sites in the vicinity of the proposed power station. The document also includes estimates of predicted levels of emissions based on data provided by the manufacturers of two different generators. To consider the potential health impacts from the project the predicted emissions have been added to the historical measurements of background pollution. The worst case situation is assumed to occur if the two generators were started up simultaneously during an unusual weather event such as a temperature inversion on a still day (emissions are higher during start up than during normal running). This will give the short term peak levels. For the annual and 24 hour average values it was assumed that the plant would be operating continuously throughout the year, whereas the operators predict that the plant would only operate during periods of peak demand, estimated at 15% of the time.

It can be seen from these and other tables that the short term maximum *background* concentrations of a number of the pollutants have been measured at relatively high levels. However, these levels are mostly well below the criteria set by the NSW Government. For example, the maximum background level of NO_2 during the observation period reached almost 50% of the air quality criterion while the 24 hour background level of PM10 exceeded 80% of the air quality criterion on a number of days during the observation period. The annual average was acceptably low at 12.9 $\mu g/m^3$.

When the predicted worst case air quality impacts were added to the highest background levels, none of the cumulative values exceeded the air quality criteria. The 24 hour maximum for PM10 approached the criterion. However the background pollution would be the main contributor to the high PM10 levels in the worst case scenario. The annual average for NO_2 was well below the criterion level. All the other potential pollutants are predicted to be at quite low levels and well below the criteria.

The potential health effects of the predicted cumulative levels, both the maximal and annual averages, are considered below. It should be noted that the discussion below is based on the assumption that the power station would comprise 2 generators, giving a total of 300 MW as described in the most recent project application of May 2007.

4.1 Respiratory symptoms and asthma

The most likely potential health impacts from relatively low levels of the types of pollutants emitted from a gas power station would be respiratory symptoms. Moreover the most vulnerable or susceptible individuals would be children with asthma and the frail elderly with advanced cardiopulmonary disease.

PM10

The PM10 levels are estimated as a worst case scenario to be less than 50 μg/m³. This would be below the criterion level for NSW and comparable to the levels reported in a Melbourne study which showed these levels are too low to cause a discernable increase in respiratory symptoms.[11] PM10 levels have not been conclusively linked to asthma exacerbations (discussed above).

NO_2

The predicted annual operating average of 23.3 μg/m³ of NO_2 is in the very low range and is comparable to the lowest range in most studies examined.[1, 13, 19] An increase of 0.3 μg/m³ to the background annual average is unlikely over the long term to affect lung development in children or to significantly increase the risk of developing asthma.[7]

If two generators are started up the 1 hour maximum NO_2 would increase by 33.2 μg/m³ in a worst case situation. It is unclear whether an increase of this magnitude would cause increased respiratory symptoms or wheeze in even the most susceptible exposed individuals, as results reported in the literature are inconsistent[18]. The maximum level of NO_2 predicted is 168 μg/m³ in the worst case situation during start up (when the background level is assumed to be 134.7 μg/m³). This resultant cumulative level would be unlikely to cause a detectable increase in symptoms or admissions. It is similar to the mean level of NO_2 (142 μg/m³) found in the McCreanor[17] study from London which showed no increase in symptoms or use of puffers in the week following 2 hours exposure.

SO_2

The maximum daily increase in SO_2 is predicted to be 0.6 μg/m³ with the 1 hour cumulative impact of 30.6 μg/m³ . The APHEA study[15] found a borderline effect with an increase in SO_2 of 50 μg/m³. Other studies have found either contradictory or no effects. The APHEA II study[19] found an increase in asthma admissions of 1.3% with a daily increase of 10 μg/m³. On the basis of these results, an increase of 0.6 μg/m³ due to the power station would be expected to have a negligible effect. However, on the days of the highest background pollution asthma admissions would be expected to increase, but the additional SO_2 emitted by the project would not result in any additional admissions.

4.2 Mortality

PM10 and PM2.5 are the pollutants reported to be associated with mortality in the literature. A large well designed study has shown that other commonly measured pollutants were not related to mortality rate, although the authors concluded that ozone may have a very weak effect in summer only[1]. Using the worst case scenario data for predicting the 24 hour maximum during startup, PM10 is estimated to increase by 1.12 μg/m³ but still remain below the criteria level of 50 μg/m³. Mortality rates are known to have a small (0.3-0.6%) increase for every 10 μg/m³ increase but it is possible that there is a threshold for this effect at the standard of 50 μg/m³. An increase of 1.12 μg/m³ is therefore unlikely to have any impact on mortality in the area even if the background level was approaching the criteria standard.

5. Comparison between potential impacts of predicted emissions and other common exposure sources

A search of the medical data bases did not reveal any reports of adverse health consequences associated with natural gas power stations, either for workers at the plants or for the neighbouring population. Therefore we have attempted to compare the possible risks from the predicted worst case emissions, added to the worst case background pollution levels, with the health impacts reported from commonly encountered sources of pollution.

Indoor natural gas combustion

NO_2 is the major indoor pollutant associated with indoor natural gas combustion discussed in the scientific literature. This is mainly due to the fact that natural gas is relatively clean burning. It is also suggested that the main source of other pollutants in the indoor environment is contamination from outdoor atmospheric pollution. It is difficult to compare indoor exposures to outdoor exposures due to other elements in the indoor environment causing respiratory disease, in particular indoor allergens such as house dust mite, moulds and animal dander[30].

The predicted maximum increase in NO_2 from start up of the the GE LMS 100 generator is well below the indoor concentrations produced by an unflued gas heater,[30] but would be slightly more than the increase associated with a gas stove (33 $\mu g/m^3$ is the predicted maximum level for the generator, compared to 30 $\mu g/m^3$ average indoor increase from a gas stove).[20]

The health impacts from NO_2 concentrations at such levels are probably negligible. As discussed above the medical literature is inconsistent. Those studies which reported an impact from gas stoves suggested that the effect was related to some exposure other than NO_2.

Comparison with pollution from motorways

It is also difficult to compare a natural gas plant exposure to pollution from motorways as most of the pollution causing respiratory disease from motorways is due to diesel fuel producing black smoke in addition to a number of other pollutants including oxides of nitrogen, carbon and sulphur.[28, 29] Natural gas is a relatively clean fuel without many of the toxic aromatic hydrocarbons produced when burning coal, diesel, petrol or wood. The annual daily average exposure to NO_2 would be far lower than expected exposure 15 meters from a busy motorway. The worst case predicted peak levels of NO_2 are far below those recorded adjacent to Oxford St, London which produced small changes in lung function of asthmatic subjects but no changes in symptoms or medication use over the following week[17]. The values are similar to those recorded 300m from a Dutch motorway with heavy truck traffic. In the latter report the authors concluded that respiratory symptoms were correlated with levels of black smoke rather than NO_2[28]. Similarly, the symptoms appeared to be unrelated to levels of PM10 or PM2.5.

Thus, any potential health impacts from the proposed project would be less than that of living between 150 and 300m from a freeway. The medical literature indicates that pollution levels are similar to background urban concentrations at distances greater than 150m from a freeway and that there are therefore no additional adverse health impacts.

Comparison with suburban environment

Mean pollution levels in the area in question are at present very low by international standards. The estimated potential annual averages in the air quality impact report after construction of a 300MW natural gas plant are comparable to the least polluted suburban areas in Europe and the United States in the studies quoted above and well below average suburban reported levels. Both the average and peak levels of all the potential pollutants are predicted to remain below the criteria standards set by the NSW Government.

Comparison with WA alumina refinery[23]

The study is included as a case study of community perceptions and health impacts in an Australian context. No studies were found of the health impacts of Natural Gas Plants after an extensive literature review. Wagerup is a refinery in a similar rural area with two townships of comparable distance of Appin from Leafs Gully. The main differences between the plants are that the Leafs Gully project is closer to a major metropolitan area and an alumina refinery emits a far greater concentration and number of known pollutants and irritants. Aluminium refining is also known to cause occupational asthma. Thus at Leafs Gully the major source of most pollutants is predicted to be the background levels whereas at Wagerup the smelter has a greater proportional contribution to the total. The Wagerup maximum averages measured were generally greater than the maximum predicted averages of Leaf Gully. In the Wagerup report the authors predicted minimal health impacts at the levels they recorded. Their data suggested a weak link between NOx and frequency of complaints, however most of the complaints related to odour. The highest levels of pollutants recorded were on days when the wind direction was inconsistent with the pollutants arising from the plant.

The results of this study suggest strongly that the Leafs Gully project with lower emissions and fewer known pollutants would be very unlikely to increase the rate of respiratory symptoms or illness either in the short or the long term.

6. Summary

This report was prepared at the request of Minter Ellison on behalf of AGL Power Generation (NSW) Pty Ltd to assess any possible health consequences arising from the proposed Leafs Gully natural gas peaking power station with a total capacity of approximately 300 MW. We were provided the updated "*Local Air Quality Assessment for Proposed Gas Turbine Power Station, Appin, NSW"* prepared by URS. The potential air quality impact calculations have been based on conservative assumptions.

The Government sets standards for ambient air quality including criteria levels for common pollutants and air impact assessment criteria designed to determine whether proposed developments are likely to have a deleterious impact on ambient air quality. The Air Quality Assessment includes the "worst-case" predicted impact from the plant for each of the potential pollutants, compared with the criteria.

A search of the medical data bases failed to locate any reports of adverse health outcomes specifically attributed to gas fired power stations even though many such stations are in use in relatively close proximity to populated areas in Australia and elsewhere, in some instances less than 500 m from the nearest residence. Therefore we reviewed the literature concerning health effects of urban pollution and other sources of pollution and compared the levels of specific pollutants reported in those studies with the potential worst case impact of the proposed Leafs Gully project.

Urban air pollution includes 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. 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]. Many of the toxic components of urban pollution are not found in emissions from natural gas combustion.

There are a number of problems with epidemiological studies in this area regarding safe levels and the potential impact of pollution on respiratory symptoms and disease and many studies have shown conflicting results.

It is believed that only a small proportion of the population is at risk from short-term exposure to pollution: e.g. the elderly, those with severe cardiopulmonary disease, frail infants and asthmatics.

Several studies have shown small increases in respiratory symptoms, asthma episodes, hospital admissions and deaths on high pollution days and some correlations with levels of common pollutants have been reported. These results have informed the setting of the criteria levels for common pollutants. However, there are some inconsistencies: for example some authors have found that black smoke (mostly derived from diesel vehicle emissions) is more important than particulate levels as a whole.

Observations of historical pollutant levels in the vicinity of the proposed plant revealed occasional peak and 24 hour average levels that were relatively high, but almost always below the criteria levels. The highest values occurred when there were bushfires in the area. The annual averages were comparable with the lowest values reported in the literature.

Of the potential emissions from the proposed plant the highest impact on ambient air quality is likely to be from nitrogen oxide (NO_2) because natural gas is relatively clean burning with low particulate and sulphur dioxide emissions. In the worst case situation for the proposed 300 MW natural gas power station (during simultaneous startup of both turbines), maximal emissions of NO_2 added to the highest ambient pollution levels (during an unusual weather event) are predicted to result in levels below the NSW criteria. At these levels any health impact would be difficult to detect. At the projected levels of the potential pollutants considered in the air quality impact document there is unlikely to be any adverse effect on mortality, lung development in children and adolescents, decline in lung function or rates of asthma development.

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