GRIFFIN ENERGY PTY LTD

Collie B Power Station

Proponent's Response to Submissions

Attachment 12

Criteria Pollutants Health Effects

April 2005

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Sulphur Dioxide (SO2)

Sulphur dioxide (SO2) is a potent respiratory irritant when inhaled. Asthmatics are particularly susceptible. SO2 acts directly on the upper airways (nose, throat, trachea and major bronchi), producing rapid responses within minutes. It achieves maximum effect in 10 to 15 minutes, particularly in individuals with significant airway reactivity, such as asthmatics and those suffering similar bronchospastic conditions.

The symptoms of SO2 inhalation may include wheezing, chest tightness, shortness of breath or coughing, which are related to reductions in ventilatory capacity (for example, reduction in forced expiratory volume in one second, or FEV1), and increased specific airway resistance. If exposure occurs during exercise, the observed response may be accentuated because of an increased breathing rate associated with exercise. A wide range of sensitivity is evident in both healthy individuals and more susceptible people, such as asthmatics, the latter being the most sensitive to irritant.

Epidemiological studies have shown significant associations between daily average SO2 levels and mortality from respiratory and cardiovascular causes. Increases in hospital admissions and emergency room visits for asthma, COPD and respiratory disease have also been associated with ambient SO2 levels. These associations were observed with up to a two-day lag period. Long-term exposure to SO2 and fine particle sulphates (SO42-) has been associated with an increase in mortality from lung cancer and development of asthma and cardio-pulmonary obstructive disease. Increases in respiratory symptoms have also been associated with SO2 levels.

Nitrogen Oxides (NOx)

The inhalation of nitrogen dioxide (NO2) has been shown to cause reversible effects on airway responsiveness and lung function. Exposure may also cause an increase in the sensitivity to natural allergens. Inhalation by children will increase the risk of respiratory infection and may lead to poor lung function in later life. Association between ambient NO2 exposure and increases in daily mortality and hospital admissions for respiratory disease, has been shown in recent epidemiological studies. NO2 has also been shown to increase the effects of exposure to other known irritants, such as ozone and respirable particles.

There is some evidence that acute exposure to NO2 may cause an increase in airway responsiveness in asthmatic individuals. This response has been observed only at relatively low NO2 concentrations, mostly in the range of 400–600 µg/m3. However, the findings of both clinical and epidemiological studies do not provide any clear quantitative conclusions about the health effects of short-term exposures to NO2. The adverse health effects at low levels of NO2 remain uncertain, with conflicting patterns of results obtained in both controlled exposure studies and in epidemiological studies. The contribution of NO2 as one of a mixture of pollutants in the ambient environment has yet to be clearly defined.

Particulate Matter < 10μm (PM10)

The major health effects from airborne particles are:

- increased mortality;
- aggravation of existing respiratory and cardiovascular disease;
- hospital admissions and emergency department visits;
- school absences;
- lost work days; and
- restricted activity days.

People most susceptible to the effects of particles include: the elderly; those with existing respiratory disease such as asthma, chronic obstructive pulmonary disease and bronchitis; those with cardiovascular disease; those with infections such as pneumonia; and children. The results of epidemiological studies have provided no evidence for the existence of a threshold value below which no adverse health effects are observed.

Carbon Monoxide (CO)

The health effects of carbon monoxide are well understood. When inhaled, CO combines with haemoglobin (Hb), the blood's oxygen-carrying protein, to form COHb. In this state the Hb is unable to carry oxygen (O2). It takes about 4 to 12 hours for CO concentrations in the blood to reach equilibrium with the CO concentration in air, so any fluctuations in the ambient CO concentrations are only slowly reflected in the COHb levels in humans.

High exposures to CO can cause acute poisoning, with coma and collapse occurring at COHb levels of over 40%. Ambient exposures to CO are several orders of magnitude lower than those associated with acute poisoning. However, some exposures in urban settings have been shown to adversely affect the heart, brain and central nervous system.

Adverse cardiovascular effects of CO inhalation include decreased O2 uptake and decreased work capacity. Those with angina may suffer decreased exercise capacity at onset of angina, and increased duration of angina. Adverse neurobehavioural effects of CO include a decrease in vigilance, visual perception, manual dexterity, ability to learn and perform complex sensorimotor tasks in healthy individuals, and reduced birth weight in non-smoking mothers.

Ozone (O3)

Epidemiological evidence indicates that a wide variety of health outcomes are possible from exposure to O3, including short-term effects on mortality, hospital admissions and emergency room attendances, respiratory symptoms and lung function. Experimental evidence has demonstrated short-term physiological and pathological changes in the respiratory system of humans. Although potentially more important, there is little

evidence of long-term effects. Recently, ozone has been found to cause asthma, particularly in young children exercising in areas with higher ozone levels.

The health effects associated with exposure to ozone can be summarised as follows:

- increase in daily mortality, respiratory and cardiovascular disease;
- increase in hospital admissions and emergency room visits;
- increase in respiratory and cardiovascular disease;
- decrease in lung function;
- increase in symptoms of respiratory illness such as cough, phlegm and wheeze; and
- increase in bronchodilator usage.

These effects are observed in sensitive sub-populations, although effects on lung function have also been observed in the healthy normal population.

Lead (Pb)

The health effects of lead are related to the level of lead in human blood. Although there are some differences in the bio-availability of different lead compounds, the health effects caused by increased blood lead levels are the same, regardless of the lead compounds causing the exposure.

One of the most widely recognised effects of lead exposure is a decrease in intelligence and general academic performance in children, especially when exposed to lead within the first two to three years of life. The sub-groups most vulnerable to lead are young children and developing foetuses. There is now clear epidemiological evidence of a close causal relationship between prenatal exposure to lead and early mental development indices, and it has not been possible to identify a clear threshold for its effects.

Where there is the likelihood of ingestion from deposited lead, this must be taken into account in conjunction with inhalation exposure when considering the total body burden. This is especially so when assessing potential health effects on children living in an area where lead may be inhaled and/or ingested.

Mercury (Hg)

The effects of chronic exposure to elemental mercury (Hg) include central nervous system (CNS) effects (such as erethism, irritability, insomnia), severe salivation, gingivitis and tremor, kidney effects (including proteinuria), and acrodynia in children. The primary effect of chronic exposure to methyl mercury is CNS damage, while chronic exposure to inorganic mercury induces kidney damage (US EPA, 1998). Acute inhalation exposure to high levels of elemental mercury in humans results in CNS effects such as hallucinations, delirium and suicidal tendencies; gastrointestinal effects; and respiratory effects such as chest pains, dyspnoea, cough, pulmonary function

impairment, and interstitial pneumonitis. Acute exposure to high levels of methyl mercury also results in CNS effects, including blindness, deafness, impaired level of consciousness and death.

Studies of the effects on human reproduction and development from exposure to inorganic mercury are ambivalent. There is no information on reproductive and developmental effects on humans, but animal studies have reported effects including testicular changes and developmental abnormalities. Studies on the carcinogenic effects of elemental mercury on humans are inconclusive. No studies are available on the carcinogenic effects of methyl mercury on humans.

The US EPA has classified inorganic and methyl mercury as Group C carcinogens, and elemental mercury as Group D (unclassifiable). IARC has classified methyl mercury compounds as a Group 2B carcinogen, and mercury and inorganic compounds as Group 3 (unclassifiable) (IARC, 1998).

No unit risk factors are available for mercury and mercury compounds. Their status as carcinogens is ambivalent. WHO recommends a guideline for inorganic mercury of 1 µg/m3 as an annual average. This is based on a lowest observable adverse effects level for renal tubular effects on humans of 20 µg/m3 and an uncertainty factor of 20.

The US EPA RfC for elemental mercury is 0.3 µg/m3, and the reference dose (RfD) for methyl mercury is 0.3 µg/kg/day (US EPA, 1993). The California Air Resources Board (CARB) RELs are as follows:

- elemental mercury 0.3 µg/m3 (chronic REL);
- inorganic mercury and mercury compounds 30 µg/m3 (acute REL); and
- methyl mercury 1 µg/m3 (chronic REL).

The acute REL for inorganic mercury is under review, and a draft value of 1.8 µg/m3 is to be reviewed by the Scientific Review Panel on Toxic Air Contaminants.