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## 2 BACKGROUND

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### 2.1 Scope

The purpose of this section is to provide a brief background to the reasons why air pollution causes health concerns, and in broad terms the nature of the health effects.

### 2.2 *Health effects of air pollutants from motor vehicles*

It has been known for a long time that many of the substances that are referred to as air pollutants produce human health effects at high levels of exposure. This has been well documented in case studies of a series of air pollution episodes in the mid-1900s which showed dramatic effects on health, and in high dose toxicological studies in animals. Air pollution episodes in the Meuse Valley of Belgium in 1930, Donora in the United States of America in 1948 and London, England in 1952 were investigated in detail. In the 1952 London air pollution episode it was estimated that 4,000 extra deaths occurred as a result of the high concentrations of sulphur dioxide and particulate matter (Brimblecombe, 1987).

Emphasis on these severe episodes of air pollution may have distracted attention from the effects of long term exposure to air pollutants. Studies in London in the 1950s and 60s (Waller, 1971) showed that the self-reported state of health of a panel of patients suffering from chronic bronchitis varied with day-to-day levels of pollution. It was noted, however, using simple methods of analysis, that symptoms did not increase unless the concentrations of smoke (measured as "British Standard Smoke") and sulphur dioxide exceeded 250 and 500  $\mu\text{g m}^{-3}$ , respectively. It is likely that, had more searching methods of analysis been applied, effects would have been seen at lower concentrations. This is an early illustration of a feature of the effects of air pollution - known as the 'threshold effect'. The threshold, for any pollutant is the concentration below which no effect is observed (and it is different for different substances, sometimes zero).

Since the 1950s a great body of evidence has accumulated showing that air pollutants have a damaging effect on health. Two features of that body of work are the consistency of the results and that the effects occur at concentrations of air pollutants previously considered to be "safe".

Emissions from motor vehicles that can produce health effects are the gases carbon monoxide, nitrogen oxides, volatile organic compounds, and sulphur dioxide, as well as solid particulate matter (now commonly referred to as particles). Additionally, other gases (such as ozone) and particles (sulphates and nitrates) can form in the atmosphere from reactions involving some of those primary emissions. The health effects of carbon monoxide, nitrogen dioxide, ozone, particles and sulphur dioxide are reported elsewhere (Denison, Rolfe and Graham, 2000) and the following is a brief summary of that information.

#### **Carbon monoxide**

Carbon monoxide is an odourless gas formed as a result of incomplete combustion of carbon-containing fuels, including petrol and diesel. Carbon monoxide is readily absorbed from the lungs into the blood stream, which then reacts with haemoglobin molecules in the blood to

form carboxyhaemoglobin. This reduces the oxygen carrying capacity of blood, which in turn impairs oxygen release into tissue and adversely affects sensitive organs such as the brain and heart (Bascom et al, 1996).

Motor vehicles are the predominant sources of carbon monoxide in most urban areas. As a consequence of the age of the vehicle fleet, New Zealand has relatively high urban air concentrations of carbon monoxide. It has been reported (Ministry of Economic Development, 2001) that nearly 50% of the New Zealand car fleet is more than 10 years old, and only one in five is less than five years old. Furthermore, only about one-quarter of the car fleet have catalytic converters, even though they have been mandatory in countries from where vehicles have been sourced since the 1970s.

Long-standing international (and New Zealand) air quality guidelines/standards for carbon monoxide are based on keeping the carboxyhaemoglobin concentration in blood below a level of 2.5%, in order to protect people from an increased risk due to heart attacks. This has led to little variation in the guidelines/standards, being typically  $10 \text{ mg m}^{-3}$ , 8-hour average, and  $30 \text{ mg m}^{-3}$ , 1-hour average. That situation may soon change, because there is emerging research that indicates adverse health effects at carboxyhaemoglobin levels less than 2.5% (for example, Morris and Naumova, 1998). This new information is especially relevant to New Zealand, because of the relatively high urban air concentrations of carbon monoxide.

### **Nitrogen dioxide**

Nitrogen oxides (primarily nitric oxide and lesser quantities of nitrogen dioxide) are gases formed by oxidation of nitrogen in air at high combustion temperatures. Nitric oxide is oxidised to nitrogen dioxide in ambient air, which has a major role in atmospheric reactions that are associated with the formation of photochemical oxidants (such as ozone) and particles (such as nitrates).

Nitrogen dioxide is also a serious air pollutant in its own right. It contributes both to morbidity and mortality, especially in susceptible groups such as young children, asthmatics, and those with chronic bronchitis and related conditions (for example, Morris and Naumova, 1998). Nitrogen dioxide appears to exert its effects directly on the lung, leading to an inflammatory reaction on the surfaces of the lung (Streeton, 1997). Motor vehicles are usually the major sources of nitrogen oxides in urban areas.

Air quality guidelines/standards for nitrogen dioxide are set to minimise the occurrence of changes in lung function in susceptible groups. The lowest observed effect level in asthmatics for short-term exposures to nitrogen dioxide is about  $400 \text{ } \mu\text{g m}^{-3}$ . Although less data are available, there is increasing evidence that longer-term exposure to about  $80 \text{ } \mu\text{g m}^{-3}$  during early and middle childhood can lead to the development of recurrent upper and lower respiratory tract symptoms. A safety factor of 2 is usually applied to those lowest observed effect levels, giving air quality guidelines/standards for nitrogen dioxide of  $200 \text{ } \mu\text{g m}^{-3}$ , 1-hour average, and either  $40 \text{ } \mu\text{g m}^{-3}$ , annual average, or  $100 \text{ } \mu\text{g m}^{-3}$ , 24-hour average (these two longer-term exposure concentrations being roughly equivalent).

### **Hydrocarbons**

Volatile organic compounds are a range of hydrocarbons, the most important of which are benzene, toluene, and xylene, 1,3-butadiene, polycyclic aromatic hydrocarbons (PAHs), formaldehyde and acetaldehyde. The potential health impacts of these include carcinogenic and non-carcinogenic effects. Benzene and PAHs are definitely carcinogenic, 1,3-butadiene and formaldehyde are probably carcinogenic, and acetaldehyde is possibly carcinogenic.

Non-carcinogenic effects of toluene and xylene include damage to the central nervous system and skin irritation. Heavier volatile organic compounds are also responsible for much of the odour associated with diesel exhaust emissions.

Motor vehicles are the predominant sources of volatile organic compounds in urban areas. Benzene, toluene, xylene, and 1,3-butadiene are all largely associated with petrol vehicle emissions. The first three result from the benzene and aromatics contents of petrol, and 1,3-butadiene results from the olefins content. Evaporative emissions, as well as exhaust emissions, can also be significant, especially for benzene. Motor vehicles are major sources of formaldehyde and acetaldehyde. These carbonyls are very reactive and are important in atmospheric reactions, being products of most photochemical reactions. PAHs arise from the incomplete combustion of fuels, including diesel.

Of the volatile organic compounds, the most important in the New Zealand context is benzene. The benzene content of petrol is high, often exceeding 4% by volume, especially for the “premium” grade, whereas many overseas countries restrict the benzene content to less than 1% by volume. Health effects data and guidelines/standards for hazardous air pollutants have been reported elsewhere (Chiodo and Rolfe, 2000), and include recommended air quality guidelines for benzene of  $10 \mu\text{g m}^{-3}$  (now) and  $3.6 \mu\text{g m}^{-3}$  (when the benzene content of petrol is reduced), both guidelines being annual average concentrations. The implied cancer risks (leukaemia) corresponding to those air concentrations are, respectively, 44-75 per million population and 16-27 per million population, based on World Health Organization unit risk factors for benzene.

## **Sulphur dioxide**

Sulphur oxides (primarily sulphur dioxide and lesser quantities of sulphur trioxide) are gases formed by the oxidation of sulphur contaminants in fuel on combustion. Sulphur dioxide is a potent respiratory irritant, and has been associated with increased hospital admissions for respiratory and cardiovascular disease (Bascom et al, 1996), as well as mortality (Katsouyanni et al, 1997). Asthmatics are a particularly susceptible group. Although sulphur dioxide concentrations in New Zealand are relatively low, and motor vehicles are minor contributors to ambient sulphur dioxide, the measured levels in Auckland (for example) have increased in recent years, after many years of decline, as a result of the increasing number of diesel vehicles (and the relatively high sulphur content of diesel in New Zealand).

There appears to be a threshold concentration for adverse effects in asthmatics from short-term exposures to sulphur dioxide at a concentration of  $570 \mu\text{g m}^{-3}$ , for 15 minutes (Streton, 1997). Ambient air guidelines/standards are based on this figure, for example the guidelines for New Zealand are  $350 \mu\text{g m}^{-3}$ , 1-hour average, and  $120 \mu\text{g m}^{-3}$ , 24-hour average.

Sulphur oxides from fuel combustion are further oxidised to solid sulphates, to a certain extent within the engine and completely in the atmosphere. The former inhibits the performance of exhaust emission control equipment for nitrogen oxides and particles, and this is a major reason why the sulphur contents of petrol and diesel are being reduced internationally. New Zealand currently has a high sulphur content diesel (up to about 2,500 parts per million by volume). Many countries are moving to “sulphur-free” petrol and diesel (less than 10 ppm). It is an unfortunate reality that unless the sulphur content of diesel is less than about 120 ppm, vehicles with advanced emission control systems are actually net producers of additional fine particles, because of oxidation of the sulphur oxides to sulphates.

## Particulates

Fine particles such as sulphates cause increased morbidity and mortality, and there are no apparent threshold concentrations for those health effects. As a result the World Health Organization (WHO) has decided not to recommend air quality guidelines for particles, but most countries (including New Zealand) have been more pragmatic and have set guidelines (typically  $50 \mu\text{g m}^{-3}$  for  $\text{PM}_{10}$ , 24-hour average) aimed at minimising the occurrence of health effects. Recent preliminary research is showing that it is probably the finer particles causing greater effects ( $\text{PM}_{2.5}$ ), and particles from diesel emissions possibly having greater effects than those from other sources.

## Ozone

Ozone is a secondary air pollutant formed by reactions of nitrogen oxides and volatile organic compounds in the presence of sunlight. These primary emissions arise mainly from motor vehicles. Ozone is only one of a group of chemicals called photochemical oxidants (commonly called photochemical smog), but it is the predominant one. Also present in photochemical smog are formaldehyde, other aldehydes, and peroxyacetyl nitrate.

Ozone is another air pollutant that has respiratory tract impacts (Woodward et al, 1995). Its toxicity occurs in a continuum in which higher concentrations, longer exposure, and greater activity levels during exposure cause greater effects. It contributes both to morbidity and mortality, especially in susceptible groups such as those with asthma and chronic lung disease, healthy young adults undertaking active outdoor exercise over extended periods, and the elderly, especially those with cardiovascular disease. Substantial acute effects occur during exercise with one hour exposures to ozone concentrations of  $500 \mu\text{g m}^{-3}$  or higher.

Ozone, like particles, is an air pollutant for which there is no indication of a threshold concentration for health effects (Streeton, 1997). (However, unlike particles, the WHO has established air quality guidelines for ozone.) More than any other air pollutant, there is considerable variation in air quality guidelines/standards for ozone, because of complexities involved in reducing ambient concentrations of it. In New Zealand a relatively "pure" approach has been taken, and air quality guidelines for ozone of  $150 \mu\text{g m}^{-3}$ , 1-hour average, and  $100 \mu\text{g m}^{-3}$ , 8-hour average have been established.

## Summary

A large number of epidemiological studies have been carried out worldwide which has shown associations between ambient air pollution levels and adverse health effects. The nature of those studies is described in the next section of this report. What remains to be determined is definitive information on the biological mechanisms by which air pollution may cause increased morbidity and mortality. It would seem, however, that inflammation of the airways is a common pathway for several air pollutants. It is also apparent that there are groups within the population that are particularly susceptible to the effects of air pollution, including the elderly, people with existing respiratory and cardiovascular disease, asthmatics, and children.