

Health effects of CO, NO<sub>2</sub>, SO<sub>2</sub>,  
ozone, benzene and  
benzo(a)pyrene in New Zealand

Air Quality Technical Report No. 43

Environet Limited

November 2003

Published in January 2004 by the  
Ministry for the Environment  
Manatū Mō Te Taiao  
PO Box 10-362, Wellington, New Zealand

Air Quality Technical Report 43

This document is available on the Ministry for the Environment's website:  
[www.mfe.govt.nz](http://www.mfe.govt.nz)



*Ministry for the*  
**Environment**  
*Manatū Mō Te Taiao*

## Foreword

We know that fine particles (PM<sub>10</sub>) contained in smoke emissions from domestic fires, vehicles and industry cause significant adverse health effects in New Zealand. But what about other air pollutants? Are concentrations of **carbon monoxide (CO)**, **nitrogen dioxide (NO<sub>2</sub>)**, **sulphur dioxide (SO<sub>2</sub>)**, **ozone (O<sub>3</sub>)**, **benzene and benzo(a)pyrene** also high enough to affect people's health and well-being?

This report discusses the potential health effects of these contaminants based on current monitoring information. Understanding health effects caused by these contaminants will enable us to develop appropriate national standards and to identify where action is required to reduce emissions.

For most of the time concentrations of these contaminants are generally low and therefore so are their potential health effects. However there are some places where nitrogen dioxide, ozone, benzene, benzo(a)pyrene and carbon monoxide concentrations occasionally exceed guideline values set to protect people's health. Unfortunately, there is often insufficient information to quantify the nature of health effects where these exceedences occur. Some estimates of the number of premature deaths relating to ozone have been made, however, for the other contaminants potential health effects are described in qualitative terms.

The information contained in this report is not government policy. It is one of three technical reports on these contaminants, with the other two covering emission inventory results and monitoring results.

I would like to thank all those councils and others who have contributed data, comments and information for this report.



Barry Carbon  
**Chief Executive**  
**Ministry for the Environment**

## Acknowledgements

The Ministry would like to thank the following for contributing data to assist with the preparation of this report and for commenting on the draft version:

- Paul Baynham, Northland Regional Council
- Karen Roberts, Hawkes Bay Regional Council
- Perry Davy, Wellington Regional Council
- Teresa Aberkane and Angie Scott, Environment Canterbury
- Richard Chilton and Kevin Mahon, Auckland Regional Council
- Chris McLay, Environment Waikato
- Gary Bedford, Taranaki Regional Council
- Trevor James, West Coast Regional Council
- Leif Pigott, Otago Regional Council
- Paul Sheldon, Nelson City Council
- Shane Ironmonger, Environment Bay of Plenty.

# Contents

Foreword	iii
Acknowledgements	iv
Executive Summary	vii
1 Introduction	1
2 Carbon Monoxide	2
2.1 Guideline values	2
2.2 Concentrations	2
2.3 Health effects of carbon monoxide	3
2.3.1 Implications for New Zealand	4
3 Nitrogen Dioxide	7
3.1 Guideline values	7
3.2 Concentrations	7
3.3 Health effects of nitrogen dioxide	7
3.3.1 Implications for New Zealand	8
4 Sulphur Dioxide	9
4.1 Guideline values	9
4.2 Concentrations	9
4.3 Health effects of sulphur dioxide	9
4.3.1 Implications for New Zealand	10
5 Ozone	11
5.1 Guideline values	11
5.2 Concentrations	11
5.3 Health effects of ozone	12
5.3.1 Implications for New Zealand	12
6 Benzene	14
6.1 Guideline values	14
6.2 Concentrations	14
6.3 Health effects of benzene	14
6.3.1 Implications for New Zealand	15
7 Benzo(a)pyrene	16
7.1 Guideline values	16
7.2 Concentrations	16
7.3 Health effects of benzo(a)pyrene	16
7.3.1 Implications for New Zealand	17

References	18
About the Ministry for the Environment	20

## List of Tables

Table 2.1: Adverse health effects from exposure to carbon monoxide	3
Table 2.2: Summary of epidemiological studies for carbon monoxide	4
Table 2.3: Estimate COHb% based on 99.9 percentile eight-hour average CO concentrations measured in urban centres of New Zealand	5
Table 5.1: Mortality estimates for different estimates of ozone exposure in Auckland	13
Table 6.1: Estimates of benzene risk to populations in Auckland, Christchurch, Hamilton and Dunedin	15
Table 7.1: Estimates of the potential impact of BaP concentrations in Christchurch	17

## Executive Summary

This report considers health effects literature in the context of concentrations of carbon monoxide (CO), nitrogen oxides (NO<sub>x</sub>), sulphur dioxide (SO<sub>2</sub>), ozone, benzene and benzo(a)pyrene (BaP) currently measured in New Zealand. The purpose of this report is to provide an indication of the extent to which existing concentrations of air contaminants in New Zealand may compromise health. This data will be subsequently used to evaluate the health benefits of improving air quality in New Zealand through the implementation of national environmental standards. While this report also describes benzene and benzo(a)pyrene, these contaminants have not yet been selected for standards development. They may be considered in the future.

In most areas of New Zealand, concentrations of CO, NO<sub>2</sub>, SO<sub>2</sub>, and benzene are below their respective ambient air quality guideline values (Ministry for the Environment, 2002). The main exceptions are concentrations of CO in the ambient air in Christchurch as well as roadside concentrations in Auckland, Christchurch, Wellington and Dunedin and roadside concentrations of NO<sub>2</sub> in Auckland.

There may be some health impacts as a result of exposure to CO concentrations in Christchurch and near to roadsides in Auckland and Wellington. Potential health impacts include a significant decrease in work capacity in healthy adults, decreased exercise capacity at onset of angina and increased duration of angina in people with ischaemic heart disease. Similarly prolonged exposure to concentrations measured at these sites and other sites such as those measured at Dominion Road and Khyber Pass could impact on developing foeti resulting in reduced birth weight in non-smokers.

The guideline values for NO<sub>2</sub> for New Zealand are based on a safety factor of 50% applied to the lowest observable adverse effect level for the protection of sensitive groups including children and asthmatics and people with chronic respiratory and cardiac disorders (Ministry for the Environment, 2002). Because the maximum one-hour average NO<sub>2</sub> concentrations measured at Khyber Pass Road are in excess of twice the guideline value, it is possible that sensitive individuals in this area will suffer health impacts as a result of NO<sub>2</sub> exposure. It is also possible that adverse health effects may occur as a result of NO<sub>2</sub> exposure close to other roadsides within Auckland, e.g. Dominion Road. In other areas of New Zealand, ambient air concentrations of NO<sub>2</sub> do not breach the guideline values and is unlikely to be causing adverse health effects.

Ozone concentrations have been measured in Auckland and on the outskirts of Christchurch. An estimate of the impact of ozone concentrations on mortality in Auckland indicates that over 100 deaths per year may be attributable to exposure to ozone concentrations. Concentrations of ozone in Auckland were in excess of the ambient air quality guideline values at one monitoring site. No estimates were made for Christchurch because of the large uncertainties surrounding exposure.

Although ambient air benzene concentrations are generally within existing guideline values, there is no threshold of effects for this contaminant. Estimates of health implications for New Zealand were made based on approximate concentrations of benzene in the major urban areas. This indicated that of the existing population in each area, leukaemia as a result of ambient air exposure to benzene was likely to affect less than 10 people in Hamilton and Dunedin, less than 40 people in Christchurch and less than 70 people in Auckland. This was based on the assumption of lifetime exposure to current benzene concentrations.

The potential health impacts of exposure to ambient air BaP in New Zealand could only be assessed for Christchurch because of the limited amount of monitoring that has been carried out for this contaminant. The annual average concentrations of BaP in Christchurch were over 10 times the ambient air quality guideline value. Around 70–100 people in Christchurch could be affected by exposure to this probable carcinogen, compared to around seven people if the ambient air quality guideline value for BaP was met.

# 1 Introduction

The Ministry for the Environment has prepared a number of reports reviewing the health implications of concentrations of air contaminants. These include technical reports for the 2002 ambient air quality guideline values (Dennison et al, 2002) and updates on the health effects literature currently being prepared. The latter have been prepared in two stages, the first being impacts of particles (PM<sub>10</sub> and PM<sub>2.5</sub>) and secondly the health effects of other contaminants including carbon monoxide (CO), nitrogen dioxide (NO<sub>2</sub>), sulphur dioxide (SO<sub>2</sub>), ozone (O<sub>3</sub>), benzene and benzo(a)pyrene (BaP). This report considers the health effects literature in the context of concentrations of the latter contaminants currently measured in New Zealand. The purpose of this report is to provide an indication of the extent to which existing concentrations of air contaminants in New Zealand may compromise health. This data will be used to evaluate the health benefits of improving air quality in New Zealand through the implementation of national environmental standards.

Based on existing concentrations and health data, fine particles (PM<sub>10</sub>) have the greatest impact on people's health in New Zealand. An estimate of their potential impacts is outlined in a previous report (Environet, 2003c). That report contains mortality estimates from Fisher et al (2002) calculated using the relationship derived by Kunzli (2000), which is based on concentrations of PM<sub>2.5</sub> but describes the impact as being for the pollution mix. Although the studies upon which the equations are derived are also based on concentrations of particles, results are presented this way because of difficulties in controlling for concentrations of other contaminants.

This highlights a difficulty in estimating the impact of different contaminants on health particularly for dose-response relationships derived from epidemiological studies, because the relationships may be based on the pollutant mix rather than one individual contaminant. Although some estimates of the potential impact on health are made for specific contaminants in this report, these results should be treated with caution because of these uncertainties. They should not necessarily be considered additive to those already estimated to be caused by PM<sub>10</sub>.

## 2 Carbon Monoxide

Carbon monoxide (CO) is a colourless, odourless and tasteless gas that is a product of the incomplete combustion of solid, liquid and gaseous carbon-based fuels. These include wood, coal, petrol, diesel, LPG, CNG, kerosene and oil.

Sources of carbon monoxide concentrations in ambient air in New Zealand are typically motor vehicle emissions and domestic home heating in most urban areas. Concentrations of carbon monoxide in the indoor environment from indoor sources can also pose a major health threat. High concentrations of CO indoors can occur as a result of emissions from non-vented gas cookers and heaters. Other common indoor sources of CO include solid fuel burning and smoking.

Carbon monoxide impacts on health by reducing the oxygen carrying capacity of the blood. This occurs because CO binds more readily to haemoglobin than does oxygen and results in the formation of carboxyhaemoglobin (COHb), which leaves less haemoglobin available for transferring oxygen around the body.

### 2.1 Guideline values

The ambient air quality guideline values (Ministry for the Environment, 2002) for CO are:

- 30 mg m<sup>-3</sup> (one-hour average)
- 10 mg m<sup>-3</sup> (eight-hour average).

These revised guideline values for CO are the same as those adopted in 1994 (Ministry for the Environment, 1994). The aim of the guideline values is to prevent exposure to levels of ambient CO that would result in blood COHb levels greater than 2.5%, at any level of physical activity.

### 2.2 Concentrations

Carbon monoxide concentrations have been measured in many urban centres of New Zealand. In most locations, concentrations are within the guideline values at ambient air quality monitoring sites. The main exception is Christchurch, where the eight-hour average guideline value is sometimes exceeded during the winter months at the central St Albans ambient air quality monitoring site. CO concentrations have also exceeded the ambient air quality guideline values at a number of 'traffic peak' sites that are primarily in Auckland, Wellington and Christchurch.

The proportion of the relative populations likely to be affected by guideline value exceedences at the 'traffic peak' monitoring sites is less than for the ambient 'residential neighbourhood' sites. This is because the elevated concentrations of CO will be localised at the roadside and will disperse with increasing distance from the source. Thus exposure to elevated concentrations will depend on proximity to the road. Those most likely to be affected by roadside CO concentrations include shop workers and those residing in other dwellings located near to high-density traffic areas, and drivers.

## 2.3 Health effects of carbon monoxide

The formation of COHb reduces the amount of haemoglobin available for the transportation of oxygen around the body. This can impact on the brain, nervous tissues, heart muscle and other specialised tissues that require large amounts of oxygen to function. As a result of oxygen deprivation, these organs and tissues may suffer temporary or permanent damage.

Those most susceptible to the health effects of ambient air exposure to CO include those with ischaemic heart disease, other forms of cardiac disease including cyanotic heart disease, hypoxaemic lung disease, cerebrovascular disease, peripheral vascular disease, those with anaemia and haemoglobin abnormalities, children, and developing foeti.

Dennison (2002) provides a comprehensive summary of the literature on health effects of carbon monoxide. This indicates the results of toxicological studies and the consequent evaluation of the lowest observed adverse effect levels (LOAEL) and no observed adverse effect levels (NOAEL) (Table 2.1) as well as recent epidemiological studies, which suggest that effects may occur at concentrations lower than indicated by toxicology. The latter suggest that health effects of CO concentrations may occur at concentrations lower than measured in many urban areas in New Zealand (Table 2.2).

**Table 2.1: Adverse health effects from exposure to carbon monoxide**

	LOAEL (% COHb)	NOAEL (% COHb)
<b>Cardiovascular effects</b>		
<i>Healthy adults</i>		
Decreased O <sub>2</sub> uptake, decreased work capacity (maximal exercise)	5.0–5.5%	< 5.0%
Significant decrease in work capacity	3.3–4.2%	< 3.0%
Strenuous exercise – maximal O <sub>2</sub> consumption	7–20%	
<i>People with ischaemic heart disease</i>		
Decreased exercise capacity at onset of angina, increased duration of angina	2.9–4.5%	2.5%
<b>Neurobehavioural effects</b>		
<i>Healthy adults</i>		
Statistically significant vigilance decrements	5.0–7.6%	< 5.0%
Statistically significant diminution of visual perception, manual dexterity, ability to learn, performance of complex sensorimotor tasks	5.0–17%	< 5.0%
<i>Foetal effects</i>		
Reduced birth weight (non-smoking mothers)	2.0–7.0%	< 2.0%

Source: Dennison et al, 2002.

**Table 2.2: Summary of epidemiological studies for carbon monoxide**

Location and period	Averaging time	Mean CO concentration (mg/m <sup>3</sup> )	Percent increase in health outcome (%)	Reference
<b>Mortality</b> 11 Canadian cities	24-hour average	1.2	2.5% (all-cause mortality)	Burnett et al, 1998
<b>Hospital admissions</b> Seattle, Washington, DC, 1987–94	1-hour maximum, 3-day lag	2.2	6% (95% CI: 3–9%) per 1.2 mg/m <sup>3</sup> (asthma admissions – non-elderly)	Sheppard et al, 1999
Reno Nevada, 1989–94	1-hour maximum	3.9	1.5% per 1 mg/m <sup>3</sup> (cardiovascular disease – elderly) 3.5% (ischaemic heart disease – elderly)	Yang et al, 1998
Tuscon, Arizona, (1988–90)	1-hour maximum	4.2	2.8% (95% CI: 0.5–5.4%) per 2.1 mg/m <sup>3</sup> (cardiovascular disease – elderly)	Schwartz, 1997
Eight US counties 1988–90	1-hour maximum	2.5–5.9	2.8% (95% CI: 1.89–3.68%) per 2.2 mg/m <sup>3</sup> (cardiovascular disease – elderly)	Schwartz, 1999
Chicago, Illinois, 1986–89	1-hour maximum		9% (95% CI: 6–12%) (congestive heart failure)	Morris and Naumova, 1998
10 Canadian cities, 1981–91	1-hour maximum 8-hour maximum	2.9 2.0	6.5% (95% CI: 3.10%) per 2 mg/m <sup>3</sup> one-hour CO (congestive heart failure – elderly)	Burnett et al, 1997
Seven US cities, 1986–89	1-hour maximum	2.2–5.0	10% (95% CI: 3–18%) to 36% (95% CI: 28–46%) per 10 mg/m <sup>3</sup> (congestive heart failure)	Morris et al, 1995
London, UK	24-hour average	1.1	2.1% (95% CI: 0.7–3.5%) (acute myocardial infarction)	Polniecki et al, 1997

Source: Dennison et al, 2002.

### 2.3.1 Implications for New Zealand

Table 2.3 gives an indication of approximate COHb blood levels (percentage) associated with the 99.9 percentile eight-hour average CO concentrations measured in each urban area in New Zealand. This is based on information presented in Dennison et al (2002) which indicates that the relationship between CO and haemoglobin is linear at CO concentrations of up to 250 mg/m<sup>3</sup> at sea level, and that COHb% at equilibrium can be reasonably approximated by the following relationship when exposure is continuous:

$$\text{COHb\%} = \text{CO}(\text{mg/m}^3) \times 0.16$$

(equation presented in Dennison et al, 2002, as adapted from Bascom et al, 1996).

**Table 2.3: Estimate COHb% based on 99.9 percentile eight-hour average CO concentrations measured in urban centres of New Zealand**

Location	Site classification	99.9 percentile eight-hour average CO (mg m <sup>-3</sup> )	COHb %
Western Hills Drive, Whangarei	Residential neighbourhood	2.5	0.4
Bank Street, Whangarei	Traffic dense	15.5	2.5
Queen Street (Tisdalls), Auckland	Traffic peak	23	3.7
Queen Street (carpark), Auckland	Traffic peak	17	2.7
Takapuna, Auckland	Residential peak	8	1.3
Khyber Pass, Auckland	Traffic peak	13	2.1
Hobson Street, Auckland	Traffic peak	9	1.4
Dominion Road, Auckland	Traffic peak	15	2.4
Henderson, Auckland	Residential peak	7	1.1
Manurewa, Auckland	Residential neighbourhood	6	1.0
Pakuranga, Auckland	Residential peak	8	1.3
Manukau, Auckland	Residential neighbourhood	6	1.0
Civic Centre, Wellington		25	4.0
Huia Pool, Wellington	Residential neighbourhood	25	4.0
Vivian Street, Wellington	Traffic peak	9	1.4
Maby Road, Lower Hutt	Residential neighbourhood	6	1.0
Birch Street, Lower Hutt	Residential neighbourhood	2	0.3
San Marino, Wellington		1	0.2
Upper Hutt	Residential neighbourhood	2	0.3
Masterton	Residential neighbourhood	2	0.3
Peachgrove Road, Hamilton	Residential peak	7	1.1
Napier	Residential neighbourhood	5	0.8
Hastings	Residential neighbourhood	2	0.3
Pereika Street, Rotorua	Residential neighbourhood	4	0.6
Marsh Street, Tauranga	Traffic peak	4	0.6
Otumoetai, Tauranga	Residential neighbourhood	2	0.3
Fenton Street, Rotorua	Traffic peak	5	0.8
Opotiki		1	0.2
St Albans, Christchurch	Residential neighbourhood	24	3.8
Beckenham, Christchurch	Residential neighbourhood	8	1.3
Hornby, Christchurch	Residential neighbourhood	7	1.1
Ashburton	Residential neighbourhood	3	0.5
Rangiora	Residential neighbourhood	4	0.6
Kaiapoi	Residential neighbourhood	5	0.8
Victory School, Nelson	Residential neighbourhood	5	0.8
Hospital, Nelson	Residential peak	5	0.8
Dunedin		10	1.6
Mosgiel	Residential neighbourhood	9	1.4

A comparison of the estimated COHb% levels associated with exposure to CO concentrations at the 99.9 percentile level in each monitoring site suggests that CO concentrations in some areas of New Zealand could have impacts on health. In the areas of Christchurch (St Albans), Wellington (Civic Centre and Huia Pool), and Auckland (Queen Street), exposure to CO concentrations could have resulted in a significant decrease in work capacity in healthy adults, a decreased exercise capacity at the onset of angina and an increased duration of angina in people with ischaemic heart disease. Similarly, prolonged exposure to concentrations measured at these sites and other sites such as those measured at Dominion Road and Khyber Pass in Auckland could impact on the developing foetus resulting in reduced birth weight in non-smokers.

The degree of these impacts, however, will depend on the extent of exposure. In areas such as Christchurch (St Albans), the monitoring is likely to reflect concentrations to which a reasonable proportion of the population is exposed for a reasonable duration. In comparison, elevated concentrations at some of the traffic peak sites may be limited to an area close to the roadside. The population exposed and the duration of the exposure could therefore be limited.

While results of the epidemiological studies presented in Table 2.2 suggest that health effects of CO may occur at COHb levels lower than 2.5%, Dennison et al (2002) concludes that there is still some question as to whether these effects are due to CO or whether CO is acting as an indicator for pollution from combustion sources. Consequently no additional calculations of the potential impact of CO based on these relationships have been carried out.

## 3 Nitrogen Dioxide

Nitrogen dioxide (NO<sub>2</sub>) is a red-brown pungent gas that is typically formed as a result of combustion processes. It is heavier than air with a vapour density of 1.58 compared to 1.0 for standard air. The odour threshold for NO<sub>2</sub>, between 1-6 parts per million (1,880–11,280 µgm<sup>-3</sup>), is much greater than concentrations measured in ambient air in New Zealand. Nitrogen dioxide gas is highly reactive, is corrosive to metals and is a strong oxidising agent. It combines with water to form nitric acid (HNO<sub>3</sub>) and nitric oxide (NO).

Emissions of NO<sub>2</sub> can occur directly from combustion processes and as a result of the conversion of NO gas in the atmosphere. In New Zealand, motor vehicle emissions are the main source of NO<sub>2</sub> in urban areas.

### 3.1 Guideline values

The ambient air quality guideline values (Ministry for the Environment, 2002) for NO<sub>2</sub> are:

- 200 µgm<sup>-3</sup> (one-hour average)
- 100 µgm<sup>-3</sup> (24-hour average).

The one-hour average guideline value was lowered from the 1994 value of 300 µgm<sup>-3</sup> (Ministry for the Environment, 1994) as a result of increased awareness of the impacts of this contaminant.

### 3.2 Concentrations

In most parts of New Zealand, concentrations of NO<sub>2</sub> are much less than the air quality guideline values for both the one-hour and 24-hour averages. The main exception is roadside monitoring in Auckland, in particular along Khyber Pass Road. At this site, NO<sub>2</sub> concentrations regularly exceed both ambient air guideline values with maximum one-hour concentrations per year ranging from around 240–440 µgm<sup>-3</sup>. Annual maximum 24-hour average NO<sub>2</sub> concentrations at the site range from around 115–135 µgm<sup>-3</sup>. The one-hour average guideline value has also been exceeded at an air quality monitoring site along Dominion Road in Auckland.

### 3.3 Health effects of nitrogen dioxide

Nitrogen dioxide is toxic to various animals as well as to humans. Its toxicity relates to its ability to form nitric acid with water in the eye, lung, mucus membrane and skin. Studies of the health impacts of NO<sub>2</sub> include experimental studies on animals, controlled laboratory studies on humans and observational studies.

In animals, long-term exposure to nitrogen oxides increases susceptibility to respiratory infections lowering their resistance to such diseases as pneumonia and influenza. Laboratory studies show susceptible humans, such as asthmatics, exposed to high concentrations of NO<sub>2</sub> can suffer lung irritation and potentially, lung damage.

Epidemiological studies have also shown associations between NO<sub>2</sub> concentrations and daily mortality from respiratory and cardiovascular causes and with hospital admissions for respiratory conditions. While results from these types of studies are not consistent, some suggest adverse effects may be associated with NO<sub>2</sub> exposure at levels below existing guideline values. For example, Burnett et al (1998) found a 4.3% increase in all-cause mortality for an increase in 24-hour average NO<sub>2</sub> levels of 47 µgm<sup>-3</sup>. Daily NO<sub>2</sub> levels during the study ranged from 29 to 56 µgm<sup>-3</sup>, much lower than the existing 24-hour average NO<sub>2</sub> guideline value for New Zealand of 100 µgm<sup>-3</sup>.

An evaluation of the health impacts of NO<sub>2</sub> concentrations carried out by the Ministry for the Environment in support of the derivation of ambient air quality guideline values concluded that the health impacts associated with low level exposure to NO<sub>2</sub> were equivocal and that the contribution of NO<sub>2</sub> as one of a mixture of pollutants in the ambient environment was yet to be clearly defined (Dennison et al, 2002).

### 3.3.1 Implications for New Zealand

The Ministry for the Environment indicates that the guideline values for NO<sub>2</sub> for New Zealand are based on a safety factor of 50% applied to the lowest observable adverse effect level for the protection of sensitive groups including children, asthmatics and people with chronic respiratory and cardiac disorders (Ministry for the Environment, 2002). Because the maximum one-hour average NO<sub>2</sub> concentrations measured at Khyber Pass Road are in excess of twice the guideline value, it is possible that sensitive individuals in this area will suffer health impacts as a result of NO<sub>2</sub> exposure. It is possible that adverse health effects might also occur as a result of NO<sub>2</sub> exposure close to other roadsides within Auckland, e.g. Dominion Road.

In other areas of New Zealand, ambient air concentrations of NO<sub>2</sub> do not breach the guideline values. If the existing guideline values provide adequate protection to sensitive individuals then adverse health effects associated with NO<sub>2</sub> exposure are unlikely to occur.

## 4 Sulphur Dioxide

Sulphur dioxide (SO<sub>2</sub>) is a colourless, water-soluble gas that is reactive and has a pungent odour. Sulphur dioxide is detectable to the human nose at concentrations of around 0.5–0.8 parts per million (1400–2240 µgm<sup>-3</sup>). Concentrations of SO<sub>2</sub> in ambient air typically occur as a result of combustion processes, in particular the burning of high sulphur fuels, although specific industries such as manufacturing fertiliser also discharge SO<sub>2</sub>. Sulphur dioxide is subject to a series of transformation processes in the atmosphere, which can result in, sulphurous and sulphuric acids, sulphites and sulphates being formed.

### 4.1 Guideline values

The ambient air quality guideline values (Ministry for the Environment, 2002) for SO<sub>2</sub> are:

- 350 µgm<sup>-3</sup> (one-hour average)
- 120 µgm<sup>-3</sup> (24-hour average).

Previously, the 1994 ambient air quality guideline values for New Zealand (Ministry for the Environment, 1994) included a 10-minute average SO<sub>2</sub> guideline value of 500 µgm<sup>-3</sup>.

### 4.2 Concentrations

Concentrations of SO<sub>2</sub> measured at ambient air quality monitoring sites in New Zealand indicate compliance with both 24-hour and one-hour average guideline values. It is possible that some locations within New Zealand have breached the short-term (10-minute) 1994 guideline value for SO<sub>2</sub>. For example, this value has been exceeded at an ambient air quality monitoring site in Hornby, Christchurch on a few occasions.

### 4.3 Health effects of sulphur dioxide

Sulphur dioxide causes its irritant effects by stimulating nerves in the lining of the nose and throat and the lung's airways. This causes a reflex cough, irritation, and a feeling of chest tightness, which may lead to narrowing of the airways. This latter effect is particularly likely to occur in people suffering from asthma and chronic lung disease, whose airways are often inflamed and easily irritated (Department of Environment, 1995).

Asthmatics are generally considered the most sensitive group in the community to concentrations of SO<sub>2</sub>. Other sensitive groups include those exercising. This is because SO<sub>2</sub> is very reactive and consequently the distribution of SO<sub>2</sub> along the conductive airways of the respiratory tract is non-uniform, depending on breathing volumes and types. For nasal breathing with low to moderate volumes the penetration into the lungs is negligible. For oral inhalation and larger volumes, doses may reach the segmental bronchi (World Health Organisation, 2000).

The health effects of concentrations of SO<sub>2</sub> have been studied in a number of ways including exposure of volunteers to sulphur dioxide in the air they are breathing in a laboratory situation and by examination of the effects on members of the population who have been exposed to episodes of atmospheric pollution. In the controlled laboratory situation, acute responses occur within the first few minutes of exposure and further inhalation does not increase effects.

Short-term (less than 24-hour exposure) guideline values for SO<sub>2</sub> have been developed based on the minimum concentrations associated with adverse effects in asthmatic patients exercising in a laboratory situation (World Health Organisation, 2000). Thus the guideline values represent a protective level for vulnerable groups within the community.

Information on the effects of exposure for longer periods (e.g. 24-hour) is obtained from epidemiological studies, which show associations between contaminants such as SO<sub>2</sub> and health impacts in communities and selected panels. In evaluating the health evidence relating to SO<sub>2</sub> exposure for the New Zealand ambient air quality guideline values, Dennison et al (2002) concludes that because of the correlations between SO<sub>2</sub> and other contaminants in the air it is difficult to confidently attribute the observed effects in the epidemiological studies to SO<sub>2</sub> alone. Experimental studies were therefore used to derive the dose-response relationships underpinning the ambient air quality guideline values for SO<sub>2</sub> for New Zealand.

#### **4.3.1 Implications for New Zealand**

Results of air quality monitoring would suggest that there are unlikely to be major health impacts associated with SO<sub>2</sub> exposure in New Zealand as concentrations in most areas are well within the ambient air quality guideline values. Some exceptions may occur on occasion in localised areas if significant industrial SO<sub>2</sub> concentrations exceed around 500 µg m<sup>-3</sup> (10-minute average). These effects should be considered and addressed as a part of the resource consent process for industrial discharges.

## 5 Ozone

Ozone (O<sub>3</sub>) is a colourless, pungent, highly reactive gas that is formed as a result of chemical reactions between primary pollutants. It is made up of three oxygen atoms. In the lower atmosphere, ozone is formed through photochemical reactions involving the action of ultraviolet light on the precursor pollutants oxides of nitrogen (NO<sub>x</sub>) and volatile organic compounds (VOC). Motor vehicles are the main source of NO<sub>x</sub> in New Zealand and contribute to VOC emissions. Other sources of VOCs include domestic home heating and industrial processes.

### 5.1 Guideline values

The ambient air quality guideline values (Ministry for the Environment, 2002) for O<sub>3</sub> are:

- 150 µg m<sup>-3</sup> (one-hour average)
- 100 µg m<sup>-3</sup> (eight-hour average).

The 2002 ambient air quality guideline values for O<sub>3</sub> are the same as those adopted in 1994 (Ministry for the Environment, 1994).

### 5.2 Concentrations

Concentrations of ozone have been measured in Auckland and on the outskirts of Christchurch. The location of sampling for the latter study, downwind of the main urban area, was to allow for the chemical reactions of the precursor pollutants to take place prior to measurement.

In Auckland concentrations of ozone have been measured at Pukekohe, Musick Point, Whangaparoa, Mangere and the Skytower. While the eight-hour guideline value for O<sub>3</sub> has only been exceeded at one site Musick Point (in 2002), all sites except Mangere have recorded concentrations within the 'alert' air quality category. That is, greater than 66% of the guideline value.

Concentrations of ozone on the outskirts of Christchurch were also within the guideline values with maximum one-hour and eight-hour averages of 97 µg m<sup>-3</sup> and 76 µg m<sup>-3</sup> at Lincoln, and 93 µg m<sup>-3</sup> and 75 µg m<sup>-3</sup> at Kainga.

Although monitoring of ozone has not been carried out in other urban centres of New Zealand, a report by McKendry (1996) indicates that Auckland, Christchurch and Hamilton have the greatest potential for elevated ozone concentrations in New Zealand.

## 5.3 Health effects of ozone

The health impacts of exposure to ozone concentrations have been widely studied using both epidemiological methods and laboratory studies. Dennison et al (2002) summarises the health effects associated with exposure to ozone as:

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

At low concentrations ozone can cause tissue injuries in the lungs and can result in significant impairment of pulmonary function. The impact of ozone on health depends on a number of factors including magnitude of concentration, duration of exposure, climate, individual sensitivity and pre-existing conditions. Those most susceptible to concentrations of ozone include children, people with pre-existing diseases, the elderly and healthy adults exercising in the outdoors.

Although some studies have indicated the potential for a no effects threshold, the overall interpretation of the health effects literature is that there is no threshold of exposure, below which effects do not occur (Dennison et al, 2002). A dose response relationship of 0.6% per  $10 \mu\text{g}\text{m}^{-3}$  (eight-hour mean) for mortality and 0.7% per  $10 \mu\text{g}\text{m}^{-3}$  (eight-hour mean) for hospital admissions was used to estimate the impact of ozone concentrations in the *Quantification of the Effects of Air Pollution on Health in the United Kingdom* study (Department of Health, 1998). This dose response relationship was based on studies carried out in the urban and rural areas of the United Kingdom during the summer months, prior to 1998.

### 5.3.1 Implications for New Zealand

The health implications of ozone in New Zealand are difficult to determine, as monitoring data are limited to the outskirts of Christchurch and a number of sites within Auckland. An estimate of the order of magnitude impact of ozone concentrations in Auckland on mortality based on the relationship used in the UK Department of Health report (1998) is shown in Table 5.1. Although monitoring for ozone has been carried out at a number of monitoring sites in Auckland the population exposure is uncertain. Estimates of health have been made based on three scenarios to provide a range of health estimates. The population exposure scenarios from which estimates were made were:

- 1) population exposure approximately equal to the average concentration across all sites
- 2) population exposure based on the annual average concentration at the worst case site
- 3) population exposure based on the annual average concentration at the best case site.

To calculate the estimated number of deaths per year in Auckland that may be associated with exposure to ozone, the annual average ozone concentration for each scenario was divided by ten to be consistent with the units for the dose response relationship. This value was multiplied by the annual mortality rate for Auckland, which was estimated at 7118 based on the average for 1998 and 1999. This value was multiplied by the dose-response relationship described by UK Department of Health (1998) to give an estimate of the annual mortality. Equation 5.1 shows the calculation for the estimate based on average exposure of 39 µgm<sup>-3</sup>.

$$\text{Equation 5.1: } 3.9 \times 7118 \times 0.007 = 195$$

Estimates are based on the assumptions that the dose-response relationships established for the United Kingdom in the COMEAP (1998) study are applicable to ozone exposure in Auckland and that the measured ozone concentrations in Auckland provide a reasonable indication of exposure in the city. The validity of these assumptions are uncertain and results should therefore be treated with caution.

These calculations indicate that the potential mortality (deaths brought forward) for Auckland may be over 100 per year.

**Table 5.1: Mortality estimates for different estimates of ozone exposure in Auckland**

	O <sub>3</sub> (average) µg m <sup>-3</sup>	Total mortality/year	O <sub>3</sub> related mortality/year
Average (all sites)	39		195
Maximum (Skytower)	48		239
Minimum (Mangere)	27		135
Total mortality (average 1998 and 1999)		7118	

## 6 Benzene

Benzene is an aromatic hydrocarbon that is produced by combustion and as a result of evaporative emissions. Benzene is used in the manufacture of plastics, detergents, pesticides, and other chemicals. In its most common form, benzene is a liquid that is clear, sweet smelling and highly combustible. Benzene evaporates quickly in the air and is partially soluble in water.

Sources of benzene in ambient air include motor vehicle emissions, domestic home heating, outdoor burning and industry. The main sources of benzene in ambient air in New Zealand are motor vehicle emissions, both tailpipe and petrol evaporation, and domestic home heating emissions. Other combustion processes may also contribute to ambient air benzene concentrations. In indoor environments, smoking can be a major contributor to benzene concentrations.

### 6.1 Guideline values

The ambient air quality guideline values (Ministry for the Environment, 2002) for benzene are:

- 10  $\mu\text{g m}^{-3}$  (annual average) existing
- 3.6  $\mu\text{g m}^{-3}$  (annual average) from 2010.

Previously, the 1994 ambient air quality guideline values for New Zealand (Ministry for the Environment, 1994) did not include an ambient air quality guideline value for benzene.

### 6.2 Concentrations

Concentrations of benzene in New Zealand have been measured in Auckland, Christchurch, Hamilton, Dunedin and Nelson. The monitoring has been carried out at the ambient 'residential neighbourhood' sites in all areas as well as 'traffic peak' sites in both Christchurch and Auckland. At the ambient air quality monitoring sites, annual average benzene concentrations have ranged from around 1  $\mu\text{g m}^{-3}$  to around 5  $\mu\text{g m}^{-3}$ . Annual average benzene concentrations in excess of 16  $\mu\text{g m}^{-3}$  have been measured at 'traffic peak' monitoring sites at Khyber Pass Road (Auckland) and Riccarton Road (Christchurch).

### 6.3 Health effects of benzene

Benzene is a known carcinogen and has been classified as a Group A carcinogen of medium potency by the US EPA, and a Group 1 carcinogen by the International Agency for Research on Cancer (IARC). Benzene also has haematological effects and is mutagenic. The major effect of long-term exposure to benzene is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease in red blood cells leading to anaemia. With exposures from less than five years to more than 30 years, individuals have developed, and died from, leukaemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection. Short-term exposure to high levels of benzene can cause drowsiness, dizziness, unconsciousness and death.

An overview of international studies on animal and human exposures to benzene is summarised in the Ministry for the Environment’s *Health effects of Eleven Hazardous Air Contaminants and Recommended Evaluation Criteria* (Chiodo and Rolfe, 2002).

Because benzene is a carcinogen, it has an inferred effects threshold of zero. Guideline values for contaminants for which there is no safe level are normally based on the evaluation of ‘acceptable risk’. A number of different risk models have been used to derive the risk of developing leukaemia from benzene exposure. The World Health Organisation (1996) indicates a range of  $4.4 \times 10^{-6}$  to  $7.5 \times 10^{-6}$  for estimates of excess lifetime risk of leukaemia at an ambient air concentration of  $1 \mu\text{g}/\text{m}^3$  (unit risk). Other risk estimates for benzene inhalation include the US EPA unit risk estimate of  $8.3 \times 10^{-6}$ , and the California Air Resources Board value of  $29 \times 10^{-6}$  (Chiodo et al, 2002).

### 6.3.1 Implications for New Zealand

Although ambient air quality guideline values for benzene are complied with in most areas of New Zealand, even concentrations of benzene below ambient air quality guideline values will have some impact. The unit risk estimates provided by World Health Organization (1996), the United States Environmental Protection Agency and the California Air Resources Board can be used in conjunction with air quality monitoring data for benzene to estimate the potential impacts of existing benzene concentrations for the main urban areas of New Zealand.

Table 6.1 shows an estimate of annual average benzene concentrations in Auckland, Hamilton and Dunedin. Auckland data are based on average concentrations measured in the Penrose, Henderson and Mt Eden for 2002 and do not include data for roadside monitoring sites, e.g. Khyber Pass. Hamilton and Dunedin data are based on the Ministry of Health 1996/1997 benzene monitoring (Stevenson and Narsey, 1998). These estimates are based on an average across the ambient air quality monitoring sites used in the study and do not include data for roadside monitoring sites. For Christchurch, the estimated annual average is based on more recent monitoring (Gunatilaka, 2002), which suggests lower average concentrations (average of ambient sites =  $3.3 \mu\text{g}/\text{m}^3$ ) than those measured during the 1996/1997 study (average of ambient sites =  $4.2 \mu\text{g}/\text{m}^3$ ). Note that estimates are not based on exposure modelling or detailed spatial modelling and are likely to be subject to a reasonable level of uncertainty.

Table 6.1 also shows the leukaemia risk for the existing population in each area based on the assumption of an average exposure to the estimated benzene concentrations throughout the study area. Some adjustments have been made to population data in some areas to better reflect likely exposure. For example, the Port Hills and outer suburbs of Christchurch were not included in the population exposure estimates.

**Table 6.1: Estimates of benzene risk to populations in Auckland, Christchurch, Hamilton and Dunedin**

	Estimated annual average benzene $\mu\text{g}/\text{m}^3$	Population	Estimated no. of people at $4.4 \times 10^{-6}$	Estimated no. of people at $7.5 \times 10^{-6}$	Estimated no. of people at $8.3 \times 10^{-6}$	Estimated no. of people at $29 \times 10^{-6}$	Percent of population effected based on $7.5 \times 10^{-6}$
Auckland	2.8	791,000	10	17	18	64	0.002%
Christchurch	3.3	280,000	4	7	8	27	0.002%
Hamilton	2.5	115,000	1	2	2	8	0.002%
Dunedin	1.9	90,000	1	1	1	5	0.001%

## 7 Benzo(a)pyrene

Benzo(a)pyrene (BaP) is a five ring polycyclic aromatic hydrocarbon (PAH) and is found in combustion generated fine particulate matter. The International Agency for Research on Cancer (IARC) considers BaP a known animal carcinogen and probable human carcinogen (Group 2A).

The main source of BaP in New Zealand is domestic home heating, in particular wood burning. Other potential sources include mobile sources, outdoor burning and rubber tyre wear. Indoor sources of BaP include smoking and open fires and wood burners.

In addition to inhalation of benzene, exposure can occur through dermal absorption and ingestion of food and water.

### 7.1 Guideline values

The ambient air quality guideline value (Ministry for the Environment, 2002) for benzo(a)pyrene is  $0.0003 \mu\text{g m}^{-3}$  (annual average). Previously, the 1994 ambient air quality guideline values for New Zealand (Ministry for the Environment, 1994) did not include an ambient air quality guideline value for BaP.

### 7.2 Concentrations

Monitoring of BaP concentrations in New Zealand is limited to a small amount of sampling carried out in Auckland during 1997-1998 and a study period in Christchurch 1999. An estimate of annual average BaP concentrations from the former study was not possible, owing to the poor detection limits of the analysis method. Results from the Christchurch study suggest annual average concentrations of BaP of at least  $0.004 \mu\text{g m}^{-3}$ , over 10 times higher than the ambient air quality guideline value. A strong correlation between BaP and  $\text{PM}_{10}$  was found (Gunatilaka, 2002). It is likely that concentrations of BaP are also of concern in other urban areas of New Zealand where  $\text{PM}_{10}$  concentrations exceed ambient air quality guideline values as a result of solid fuel burning for domestic heating.

### 7.3 Health effects of benzo(a)pyrene

While laboratory studies show that BaP is a known carcinogen in animals, epidemiological studies have only been able to assess the effect of a mixture of PAHs, including BaP found in soots, tars and oils. Mutagenicity refers to the ability of a chemical to induce mutations in DNA and in living cells. Benzo(a)pyrene is a promutagen, which means it needs to be metabolised before it can induce mutation. Benzo(a)pyrene can also react with ozone to produce strong mutagens such as benzo(a)pyrene-4,5 oxide (Californian Air Resources Board, 1994).

The IARC classification for BaP is Group 2A, probable human carcinogen. The US EPA has classified BaP as a Group B2 carcinogen of medium potency.

Like benzene, BaP has an inferred effects threshold of zero and the evaluation of ‘acceptable risk’ is used in determining appropriate ambient air guideline value concentrations. The World Health Organisation (1996) has determined an inhalation unit risk of  $8.7 \times 10^{-2}$  per  $1 \mu\text{g}\text{m}^{-3}$  BaP. This was based on interpolation from risk estimates for PAHs in coke oven emissions (Chiodo et al, 2002). In addition, WHO has also determined an inhalation unit risk from studies of animals exposed to complex mixtures of PAHs. However, in assessing ambient air quality guideline values for New Zealand, Chiodo et al (2002) conclude that the human based risk of  $8.7 \times 10^{-2}$  per  $1 \mu\text{g}\text{m}^{-3}$  BaP is appropriate.

### 7.3.1 Implications for New Zealand

It is difficult to assess the health implications of existing BaP concentrations in New Zealand because existing ambient air monitoring is limited to Auckland and Christchurch. It was not possible to establish an estimate of annual average BaP concentrations from the Auckland data because of the limitations of the analytical method. In Christchurch, annual average BaP concentrations of around 0.004 have been estimated based on monitoring in the St Albans area.

Table 7.1 shows an estimate of impact of BaP concentrations on the Christchurch population for two separate assumptions. The first (exposure 1) assumes that the concentrations measured in St Albans represent average exposure for the flat urban areas of the city. The second (exposure 2) is based on the assumption that average exposure across the city is 70% of the concentrations measured at St Albans. Both estimates are based on the assumption that all of the existing population of Christchurch City, around 280,000 will be exposed to elevated ambient air concentrations of contaminants.

**Table 7.1: Estimates of the potential impact of BaP concentrations in Christchurch**

	Estimated annual average BaP $\mu\text{g}\text{m}^{-3}$	Population exposed	Estimated no. of people affected at risk of $8.7 \times 10^{-2}$
Christchurch (exposure 1) – as measured at St Albans	0.004	280,000	97
Christchurch (exposure 2) – 70% of the St Albans concentrations	0.0028	280,000	68
If the guideline values were met	0.0003	280,000	7

## References

- Burnett RT, Dales RE, Brook JR, Razienne ME, Krewski D. 1997. Association between ambient carbon monoxide levels and hospitalisations for congestive heart failure in the elderly in 10 Canadian cities. *Epidemiol* 8: 162–7.
- Burnett RT, Cakmak S, Brook JR. 1998. The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities. *Canadian Journal of Public Health* 89(3):152–6.
- California Air Resources Board, The Office of Environmental Health Hazard Assessment. 1994. *Benzo(a)pyrene as a toxic air contaminant*. California Air Resources Board. <http://www.arb.ca.gov/toxics/summary/bap.pdf>.
- Chiodo J, Rolfe K. 2002. *Review of the Ambient Air Quality Guidelines – Health effects of eleven hazardous air contaminants and recommended evaluation criteria*. Ministry for the Environment Technical report no. 13.
- Department of the Environment. 1995. *Expert Panel on Air Quality Standards: Sulphur Dioxide*. London: Her Majesty's Stationery Office.
- Department of Health. 1998. *Quantification of the Effects of Air Pollution on Health in the United Kingdom*. Committee on the Medical Effects of Air Pollutants. London: The Stationery Office.
- Dennison L, Rolfe K, Graham B. 2000. *Review of the Ambient Air Quality Guidelines – Health effects of five common air contaminants and recommended protective ranges*. Ministry for the Environment Technical report no. 12.
- Chiodo J, Rolfe K. 2000. *Health Effects of Eleven Hazardous Air Pollutants and Recommended Evaluation Criteria*. Report prepared for the Ministry for the Environment. Air Quality Technical Report 13. Wellington, New Zealand.
- Environet Limited. 2003a. *Monitoring of PM<sub>10</sub> in New Zealand*. Wellington: Ministry for the Environment.
- Environet Limited. 2003b. *Amenity Effects of PM<sub>10</sub> and TSP Concentrations in New Zealand*. Wellington: Ministry for the Environment.
- Environet Limited. 2003c. *Health Effects of PM<sub>10</sub> in New Zealand*. Wellington: Ministry for the Environment.
- Environet Limited. 2003d. *Emission Inventories for PM<sub>10</sub> in New Zealand*. Wellington: Ministry for the Environment.
- Fisher GW, Rolfe KA, Kjellstrom T, Woodward A, Hales S, Sturman AP, Kingham S, Petersen J, Shrestha R, King D. 2002. *Health Effects Due to Motor Vehicle Air Pollution in New Zealand*. Report for the Ministry of Transport, Wellington.
- Gunatilaka M. 2002. *Hazardous Air Pollutant Monitoring in Christchurch*. Environment Canterbury Report No. R01/31.
- Gunatilaka M. 2003. *Hazardous Air Pollutants Concentrations of BTEX (Benzene, Toluene, Ethylbenzene and Xylene) in Christchurch (2001/2002)*. Environment Canterbury Report No. R03/9.
- Künzli N, Kaiser R, Medina S, Studnicka M, Chanel O, Filliger P, Henry M, Horak F, Puybonnieux-Texier V, Quenel P, Schneider J, Seethaler R, Vergnaud J-C, Sommer H. 2000. Public-health impact of outdoor and traffic-related air pollution: a European assessment. *The Lancet* 356(September): 795–801.
- McKendry IG. 1996. *A Study of Photochemical Pollution Potential in New Zealand's Major Cities*. Report prepared for the Sustainable Management Fund. Wellington: Ministry for the Environment.
- Ministry for the Environment. 1994. *Ambient Air Quality Guidelines*. Wellington: Ministry for the Environment.
- Ministry for the Environment. 2002. *Ambient Air Quality Guidelines – 2002 Update*. Wellington: Ministry for the Environment.

- Morris RD, Naumova EN, Munasinghe RL. 1995. Ambient air pollution and hospitalisation for congestive heart failure among elderly people in seven large US cities. *Am J Public Health* 85: 1361–5.
- Morris RD, Naumova EN. 1998. Carbon monoxide and hospital admissions for congestive heart failure: evidence of an increased effect at low temperatures. *Environ Health Perspect* 106(10): 649–53.
- Poloniecki JD, Atkinson RW, Ponce de Leon A, Anderson HR. 1997. Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occup Environ Med* 54: 535–40.
- Schwartz J. 1997. Air pollution and hospital admissions for cardiovascular disease in Tucson. *Epidemiol* 8: 371–7.
- Schwartz J. 1999. Air pollution and hospital admissions for heart disease in eight US counties. *Epidemiol* 10: 17–22.
- Sheppard L, Levy D, Norris G, Larson TV, Koenig JQ. 1999. Effects of ambient pollution on non-elderly asthma hospital admissions in Seattle, Washington, 1987–94. *Epidemiol* 10: 23–30.
- Stevenson C, Narsey H. 1998. *Benzene and Other Toxic Organic Compounds in Air July 1996–June 1997*. A report for the Ministry of Health, Wellington, August.
- World Health Organization. 1996. *Air Quality Guidelines for Europe, Second Edition*. WHO Regional Publications, European Series No. 91, Copenhagen.
- World Health Organization. 2000. *Quantification of Health Effects Related to SO<sub>2</sub>, NO<sub>2</sub>, O<sub>3</sub> and Particulate Matter Exposure*. Kjeller, Norway: WHO Regional Office for Europe and Norwegian Institute for Air Research. Report NILU – OR 63/96.
- Yang W, Jennison B, Omaye ST. 1998. Cardiovascular disease hospitalisation and ambient levels of carbon monoxide. *J Toxicol Environ Health* 55: 185–96.

## About the Ministry for the Environment

The Ministry for the Environment works with others to identify New Zealand's environmental problems and get action on solutions. Our focus is on the effects people's everyday activities have on the environment, so our work programmes cover both the natural world and the places where people live and work.

We advise the Government on New Zealand's environmental laws, policies, standards and guidelines, monitor how they are working in practice, and take any action needed to improve them. Through reporting on the state of our environment, we help raise community awareness and provide the information needed by decision makers. We also play our part in international action on global environmental issues.

On behalf of the Minister for the Environment, who has duties under various laws, we report on local government performance on environmental matters and on the work of the Environmental Risk Management Authority and the Energy Efficiency and Conservation Authority.

Besides the Environment Act 1986 under which it was set up, the Ministry is responsible for administering the Soil Conservation and Rivers Control Act 1941, the Resource Management Act 1991, the Ozone Layer Protection Act 1996, and the Hazardous Substances and New Organisms Act 1996.

### Head Office

Grand Annexe Building  
84 Boulcott Street  
PO Box 10-362  
Wellington, New Zealand  
Phone (04) 917 7400, fax (04) 917 7523  
Internet [www.mfe.govt.nz](http://www.mfe.govt.nz)

### Northern Regions Office

8–10 Whitaker Place  
PO Box 8270  
Auckland  
Phone (09) 913 1640, fax (09) 913 1649

### South Island Office

Level 4  
Price Waterhouse Centre  
119 Armagh Street  
PO Box 1345  
Christchurch  
Phone (03) 963 0940, fax (03) 963 2050