# Health effects due to motor vehicle air pollution in New Zealand

*Report to the Ministry of Transport*

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# EXECUTIVE SUMMARY

The Ministry of Transport has commissioned this study in order to assess the health effects due to air pollution emissions from vehicles on the population of New Zealand.

The study has been based on methodologies established overseas, in particular a recent study in Europe which showed that the number of pre-mature deaths due to vehicle related air pollution was greater than that due to the road toll.

Whilst health effects can be attributed to a wide range of contaminants from vehicles, the focus of this study has been on fine particulates ($PM_{10}$). These are shown to have the dominant effect, and can also be considered as a good 'indicator' of the combined exposure to the range of pollutants from motor vehicles

An analysis has been conducted of the relevance of overseas research to New Zealand, and concludes that the overseas results are applicable and the methodologies valid for making such an assessment in New Zealand.

The input data used includes all available and appropriate particulate monitoring data from around New Zealand, and the study is based on average annual exposures in each city and town with a population of over 5,000 people. This covers approximately 80% of the population, and includes most people who might be exposed to any significant air pollution. By far the greatest fraction of people exposed are in the major city areas with populations over 100,000. Results are given for (a) the whole of New Zealand, (b) separately for the four main centres, and (c) combined for smaller centres in the North and South Islands.

It must be emphasised that the amount of monitoring and exposure data available for New Zealand is relatively small, particularly in comparison to Europe. There is also considerable uncertainty over many aspects - such as the fraction of air pollution due to motor vehicles, the exposure rates in areas where no monitoring has been conducted, and the various risk levels and thresholds used to make mortality assessments. Nevertheless, this study has used whatever data are available, making realistic assumptions - which are all explained in detail - to arrive at the current best estimate for public health effects of vehicle related particulate emissions.

The authors and reviewers emphasise that this is a preliminary study. It should be considered as the first attempt in New Zealand to quantify health effects due to air pollution from vehicles - and as discussed throughout this report, is subject to many uncertainties and assumptions. It is likely these will be revised as planned research is completed. The results may be revised upwards - or downwards - but at present they are the best estimate based on available information.

The most likely estimate of the number of people above 30 years of age who experience pre-mature mortality in New Zealand due to exposure to emissions of $PM_{10}$ particulates from vehicles is 399 per year (with a 95% confidence range of 241-566 people). This compares with 970 people above age 30 experiencing pre-mature mortality due to particulate pollution from all sources (including burning for home heating), and with 502 people dying from road accidents (all ages).

Analysed on a regional basis, most of the increased mortality due to vehicle emissions (253 people, or 64% of the total) occurs in the greater Auckland region. Wellington and Christchurch experience somewhat lesser rates (56 and 41 people respectively, or 14% and 10%). The other cities and towns larger than 5000 people through New Zealand experience the remainder (46 people, or 12%).

For some purposes - such as a health cost analysis, or a comparison with the accident road toll - it may be appropriate to assess the traffic related air pollution mortality in terms of years of life lost, since air pollution mortality generally affects older people, resulting in fewer years of life lost than for other causes of death. This has been done by analysing causes of death, and results in an "adjusted" mortality due to $PM_{10}$ of 200 people per year (although there are still 399 pre-mature deaths per year).

Although confidence limits are given in the mortality estimates, there are other factors which may need to be taken into account, which may be different in different parts of the country. One of these is the variability in particulate pollution from year to year - this appears to be greater in areas more affected by weather factors, which can vary substantially between years. Another is the potential for other types of vehicle emissions to affect mortality - including confounding effects from gaseous pollutants and possible carcinogenic effects due to aromatics such as benzene. Another is the effects on under 30 year olds - particularly young children - which are likely to be less, but non-negligible. These factors have not been included in the present report.

The $PM_{10}$ exposure results are consistent with previous studies in New Zealand examining mortality due to all sources in Christchurch.

The results are also consistent with the European studies, which show that mortality due to vehicle related air pollution is of the order of twice the accident road toll. New Zealand has a relatively higher road toll per capita, and a relatively lower air pollution problem than many European countries - but the results still show that the public health impacts from vehicle related pollution emissions are not insignificant.

# 1 INTRODUCTION

Emissions of contaminants to the air from vehicles has been shown overseas to lead to a variety of health effects on the public. The Ministry of Transport has commissioned this report in order to assess and quantify the nature of such effects in New Zealand.

This is a preliminary study, conducted and reviewed by a number of the leading air quality and public health specialists in New Zealand. The work has involved:-

- Examining the overseas methodologies and results,
- Collating whatever relevant data are available in New Zealand,
- Assessing the relevance of overseas comparisons of the public health aspects of deaths due to air pollution effects and road crashes in the New Zealand situation,
- Making a preliminary assessment of the public exposure to both total particulate air pollution, as well as the vehicle related component,
- Assessing the public health impacts of this exposure,
- Reviewing of the state of information available, analysing the research gaps and providing recommendations for future, and more refined public health impact assessments.

# 2 BACKGROUND

## 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 $\mu g\ m^{-3}$, 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 mg $m^{-3}$, 8-hour average, and 30 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 µg $m^{-3}$. Although less data are available, there is increasing evidence that longer-term exposure to about 80 µg $m^{-3}$ 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 µg $m^{-3}$, 1-hour average, and either 40 µg $m^{-3}$, annual average, or 100 µg $m^{-3}$, 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 $\mu g\ m^{-3}$ (now) and 3.6 $\mu g\ m^{-3}$ (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 $\mu g\ m^{-3}$, for 15 minutes (Streeton, 1997). Ambient air guidelines/standards are based on this figure, for example the guidelines for New Zealand are 350 $\mu g\ m^{-3}$, 1-hour average, and 120 $\mu g\ m^{-3}$, 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 µg $m^{-3}$ for $PM_{10}$, 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 ($PM_{2.5}$), 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 µg $m^{-3}$ 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 µg $m^{-3}$, 1-hour average, and 100 µg $m^{-3}$, 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.

# 3 OVERSEAS RESEARCH

## 3.1 Scope

The purpose of this section is to summarise some of the overseas research conducted on health effects of air pollution, and present a brief overview of the results obtained.

## 3.2 Overseas research

As mentioned in the previous section of this report, since the 1950s a great body of evidence has accumulated showing that air pollutants have a damaging effect on health. Two principal approaches can be identified – first, studies on volunteers exposed to air pollutants under controlled conditions, and second, epidemiological studies. The latter include time-series studies, comparing daily occurrences of events such as deaths or admissions to hospitals with daily average concentrations of air pollutants.

Air quality guidelines/standards developed up until the 1980s (for example WHO, 1987) were derived mainly from the results of controlled studies. Where such studies demonstrated a lowest observed effect level, this was used as the starting point for determining the relevant air quality guideline/standard. The results of epidemiological studies that demonstrated a threshold effect were used in the same way. This approach is still used today (WHO, 2000) and is the basis of the air quality guidelines for carbon monoxide, nitrogen dioxide and sulphur dioxide.

A number of epidemiological studies were carried out in the late 1980s and the 1990s. These were mainly time-series studies first in the United States of America and later in Europe and elsewhere (Schwartz et al., 1996). The time-series approach takes the day as the unit of analysis and relates the daily occurrence of events, such as deaths or admissions to hospital, to daily average concentrations of air pollutants, whilst taking careful account of confounding factors such as season, temperature and day of the week (Zmirou et al., 1998). Powerful statistical techniques are applied, and coefficients relating daily average concentrations of pollutants to effects are produced. The results of these studies have been remarkably consistent and have withstood critical examination well (Samet et al., 1996).

Epidemiological studies evaluate the incidence of diseases or effects and risk factors, and associate them with air pollution data. They do not necessarily demonstrate causality or provide clear evidence of mechanisms. Therefore the database of epidemiological studies cannot always be expected to prove the possible or probable causal nature of the associations demonstrated. However, detailed examination of the data, and application of the usual tests for likelihood of causality, has convinced many of the strength of the relationships.

Associations have been demonstrated between daily average concentrations of carbon monoxide, nitrogen dioxide, ozone, particles and sulphur dioxide, and daily occurrences of deaths, hospital admissions, etc. These associations are reported in detail elsewhere (Denison, Rolfe and Graham, 2000). The associations for each of the pollutants are not significant in all studies though, taking the body of evidence as a whole, the consistency is striking. A particular outcome of the studies involving ozone and particles is that there is little indication

of any threshold of effect. (Similar conclusions have been reached regarding the lack of a threshold of effect at a population level for atmospheric concentrations of lead.)

Particles, in particular $PM_{10}$, have been the subject of many epidemiological studies and, in recent times, many reviews of those studies. The studies, in various parts of the world with differing climates, socio-economic status, pollution levels, etc, have consistently observed relationships between 24-hour average concentrations of $PM_{10}$ and daily mortality and daily hospital admissions. These studies have been critically assessed in some 15 reviews, and recently a "review of the reviews" was published (Dab et al., 2001). A total of 57 studies in 37 cities of 15 countries were considered. The conclusion reached is that the relationships are both valid and causal.

Time-series studies relate the concentrations of air pollutants to their effects on health; in fact they provide the slope of a regression line relating concentrations to health effects. The slope of the regression line is the relative risk estimates for particular health outcomes associated with, for example, a 10 $\mu g\ m^{-3}$ increase in $PM_{10}$ concentrations. The relative risk estimate is proportion by which the incidence of a particular factor changes due to the increase in $PM_{10}$. Recent World Health Organization guidelines (WHO, 2000) present such relative risk estimates, and 95% confidence intervals for the estimates. Although others could be quoted, the following are relative risk estimates used in the study for Austria, France and Switzerland published in The Lancet (Künzli et al., 2000), shown in Table 3.1.

***Table 3.1. Risk estimate used in Künzli et al (2000).***

| **Health outcome** | **Relative risk estimate associated with a 10 $\mu g\ m^{-3}$ increase in $PM_{10}$** | **95% confidence levels for the relative risk estimate** |
|---|---|---|
| Total mortality (adults >30 years, excluding violent deaths) | 1.043 | 1.026-1.061 |
| Respiratory hospital admissions (all ages) | 1.013 | 1.001-1.025 |
| Cardiovascular hospital admissions (all ages) | 1.013 | 1.007-1.019 |
| Chronic bronchitis incidence (adults >25 years) | 1.098 | 1.009-1.194 |
| Bronchitis episodes (children <15 years) | 1.306 | 1.135-1.502 |
| Restricted activity days (adults >20 years)* | 1.094 | 1.079-1.502 |
| Asthma attacks (children <15 years)+ | 1.044 | 1.027-1.062 |
| Asthma attacks (adults >15 years)+ | 1.039 | 1.019-1.059 |

* *Total person-days per year*

\+ *Total person-days per year with asthma attacks*

Some may consider that $PM_{10}$ is not a particularly good air pollutant to focus on when considering the health effects of motor vehicle air pollution. An air pollutant directly related to emissions from motor vehicles is benzene, and cancer risk data for a population can be

calculated from unit risk factors and benzene exposure data. This would be an especially useful exercise in the New Zealand context, because of the high benzene content of petrol and the need to come up with information to encourage reductions in the benzene content of petrol. Unfortunately, adequate benzene exposure data are not available at this time.

The cancer risk from exposure to benzene was mentioned in the previous section of this report. The World Health Organization calculate a range of unit risks for lifetime exposure to 1 µg $m^{-3}$ of benzene of 4.4 to 7.5 per million population, and propose that the geometric mean value of that range, 6.0 per million, be used (WHO, 2000). When sufficient benzene exposure data are available, cancer risk estimates for populations can be calculated.

An area of current focus in the United States of America, especially in California, is the cancer risk associated with diesel particulate. This is despite the United States having a relatively low proportion of diesel vehicles in its fleet. Estimates have been made of the national and individual metropolitan area cancer risks from diesel particulate (STAPPA and ALAPCO, 2000). The national estimate is 125,110 additional cases, and for the larger individual metropolitan areas: Los Angeles 16,250, New York 10,360 and Washington/Baltimore 3,750. The concern raised by those estimates has been a factor in recent decisions in the US to lower the sulphur content of diesel (that is, to introduce "sulphur-free" (<15 ppm) diesel in every state by 2005, and for its use to be mandatory from 2011) and for much enhanced programmes to retrofit emission control devices to diesel vehicles (both at the federal and state levels).

The methodology used to estimate the cancer risk in the US study is based on a unit risk for lifetime exposure to 1 µg $m^{-3}$ of diesel particulate of 300 per million population. This is considered a conservative value (that is the 'true' risk value in any given circumstance is likely to be at least this or higher). The diesel particulate air concentrations were assumed to be 1.04 times the elemental carbon concentrations. The latter were taken as 3.3 µg $m^{-3}$ for Los Angeles, 1.65 µg $m^{-3}$ for other metropolitan areas, and 0.33 µg $m^{-3}$ for non-metropolitan areas. When diesel particulate exposure data are available, cancer risk estimates for populations elsewhere can be calculated.

# 4 THE NEW ZEALAND SITUATION

## 4.1 Scope

The purpose of this section is to examine the specific elements of the New Zealand situation. It includes a discussion on the applicability of overseas results in New Zealand, an analysis of the assessment methodologies and comparison with road toll deaths, and some discussion on possible confounding effects.

## 4.2 Applicability of overseas research

One measure of the applicability of overseas research is to consider the results of studies in New Zealand. The only relevant studies to date are those carried out in Christchurch. These show an association between 24-hour concentrations of $PM_{10}$ and mortality (1-day lag) and hospital admissions. A 10 µg $m^{-3}$ increase in 24-hour $PM_{10}$ is associated with a 1% increase in all cause mortality and a 4% increase in respiratory mortality (Hales et al., 2000a), and a 3% increase in respiratory hospital admissions of adults and children and a 1% increase in cardiac hospital admissions of adults (McGowan et al., 2000). The results of these studies are consistent with studies elsewhere in the world, especially those for which the major sources of $PM_{10}$ are solid fuel combustion processes.

The Christchurch studies are related to the winter-time particles problem caused by wood and coal combustion for domestic heating. They may not be relevant to $PM_{10}$ concentrations associated with motor vehicles. New Zealand, like Europe, has a relatively high number of diesel vehicles – currently 430,000 registered, and increasing rapidly (Ministry of Economic Development, 2001). Also, as mentioned in previous sections of this report, the sulphur content of New Zealand diesel is high (up to 2,500 ppm, whereas in Europe the mandated maximum sulphur content of diesel is currently 350 ppm, reducing to 50 ppm in 2005, and in several urban areas it is already less than 10 ppm). It is likely therefore that the $PM_{10}$ in New Zealand associated with motor vehicles may be relatively high in sulphates. Although the database is limited, WHO regression lines for the relative risks for the health outcomes of mortality and hospital admissions show a steeper relationship (that is, a larger relative risk) for sulphates than for either total $PM_{10}$ or other particulate size fractions.

A major point of difference between New Zealand urban areas and most cities in developed countries overseas is the relatively high concentrations of carbon monoxide. The biological mechanism by which carbon monoxide affects health is that it reduces the oxygen transport capability of haemoglobin. It is worth considering what impact the impaired oxygen release to tissue, and the consequence effects on such sensitive organs as the brain and heart, has on the ability to be able to cope with exposures to other air pollutants, such as $PM_{10}$, which can cause inflammation of airways. The combined effects may well be synergistic.

Another air pollutant that may influence health responses to other forms of motor vehicle air pollution is nitrogen dioxide. There have been some relatively high concentrations of nitrogen oxides measured at inner city sites in Auckland and Christchurch close to major roads and busy intersections. Again, the impact of exposures to nitrogen dioxide, which affects the surface of the lungs, on the ability to cope with concentrations of $PM_{10}$ (for

example) is an area of research well worth considering further in the New Zealand context, especially given the particular fuels specifications which are different from many other places.

Overseas studies that are also particularly relevant and applicable to New Zealand are those that estimate the cancer risk associated with atmospheric exposures to benzene. As mentioned in previous sections of this report, New Zealand petrols have high benzene contents, especially the “premium” grade (often exceeding 4% by volume), and so considerations of the health effects of exposures to benzene are worthy of study. Unfortunately, adequate benzene exposure data are not available at this time. When it is, the cancer risk (leukaemia) can be estimated using the geometric mean of the World Health Organization unit risk (that is, for 1 µg $m^{-3}$ exposures) of 6.0 per million population (WHO, 2000).

## 4.3 Validity of comparisons between 'health effects' and 'road toll effects'

When comparing the "air pollution road toll" with the "traffic accident road toll" one could argue that a death is a death and should be considered equal in terms of its health, social and economic consequences.

However, the age at death has importance for the social and economic consequences. A person dying at age 30 - 60 is likely to have social and financial commitments of a different type than a 60 - 85 year old. In addition, the younger person may have a more direct impact on the monetary economy of the country. Traffic accidents tend to affect mainly young people, while the non-external cause mortality that is being used as the basis for the "air pollution road toll" calculation mainly affects older people. A comparison of traffic accident mortality and air pollution mortality may therefore be more valid if the numbers are weighted by the "years of life lost" due to each death.

Table 4.1 shows a comparison of New Zealand 1996 data for all causes of death, non-external causes and traffic accidents. There were 20,219 deaths over age 30, 19,334 of which were non-external and 222 traffic accidents in this age group (these numbers are fairly stable from year to year, but the traffic accident numbers have been decreasing in recent years).

The "person years of life lost", PYLL, is the numbers of years lost before a specified age (in this case 85 years of age). It is seen that a person dying in a traffic accident loses on average 33 years of life, while a person dying from non-external causes loses on average 14 years. Thus, from this perspective, each traffic accident death in this age range has twice the impact on public health of the non-external deaths that include the “traffic air pollution deaths”. This is used in the interpretation of calculated results.

***Table 4.1. Analysis of causes of death in New Zealand, 1996.***

| | Age | Deaths | Person years of life lost | |
|---|---|---|---|---|
| | | | PYLL(total) | PYLL/death |
| **All causes of deaths in 1996** | 30 - 64 | 5,447 | 172,464 | 31.7 |
| | 65 - 84 | 14,772 | 131,435 | 8.9 |
| | Total | 20,219 | 303,899 | 15.0 |
| **All deaths other than external causes** | 30 - 64 | 4,831 | 147,278 | 30.5 |
| | 65 - 84 | 14,503 | 128,928 | 8.9 |
| | Total | 19,334 | 276,206 | 14.3 |
| **Traffic accidents** | 30 - 64 | 168 | 6,805 | 40.5 |
| | 65 - 84 | 54 | 580 | 10.7 |
| | Total | 222 | 7,385 | 33.3 |

*1. Only deaths aged 30 to 84 years were included in calculation.*
*2. PYLL cut point is 85 years old.*

## 4.4 Possible confounding effects

Confounding effects occur when an association between two variables is explained by the action of another factor, which happens to be associated with the "exposure" and is in its own right a cause of the "outcome". The risk estimates used by Künzli et al. were derived from two US studies that compared mortality rates in cities with different average air quality measures. The investigators collected information on a wide range of potential confounding factors, such as age, socio-economic status and smoking, and the relation of mortality with particulate levels has been adjusted for these factors. The nature of the research means that there always remains the possibility that other, unmeasured factors may explain at least part of the difference between the cities. However, the consistency between the findings of the US studies and other research into the health effects of particulates suggests that uncontrolled confounding is not a major issue. For instance, studies of $PM_{10}$ levels and daily mortality within a city (such as that carried out in Christchurch by Hales et al., 2000a) also show a dose response relationship, with no evidence of a lower threshold. Time trend studies such as these are not subject to confounding in the same way as the cohort studies (since it is most unlikely that variables such as smoking rates and age structures will vary from day to day in the same way as air pollution).

## 4.5 Previous studies

### New Zealand studies linking air quality and health effects

The most significant published New Zealand study (Hales et al., 2000a) that analysed the mortality effect of $PM_{10}$ indicated that an increased total and respiratory mortality can indeed be measured. This study was designed to investigate the relationship between the daily number of deaths, weather and ambient air pollution. This involved using daily data for the city of Christchurch (population 300,000) from June 1988 to December 1993. Poisson regression models were used, controlled for season using a parametric method. The results showed that above the third quartile (20.5 degrees C) of maximum temperature, an increase of 1 degree C was associated with a 1% (95% CI: 0.4 to 2.1%) increase in all-cause mortality and a 3% (0.1 to 6.0%) increase in respiratory mortality. An increase in $PM_{10}$ of 10 $\mu g\ m^{-3}$ was associated (after a lag of one day) with a 1% (0.5 to 2.2%) increase in all-cause mortality

and a 4% (1.5 to 5.9%) increase in respiratory mortality. No evidence was found of interaction between the effects of temperature and particulate air pollution. The overall conclusion was that high temperatures and particulate air pollution are independently associated with increased daily mortality in Christchurch. The fact that these results are consistent with those of similar studies in other countries strengthens the argument that the associations are likely to be causal. These findings contribute to evidence of health consequences of fuel combustion, both in the short term (from local air pollution) and in the long term (from the global climatic effects of increased atmospheric $CO_2$).

A further study undertook an analysis of mortality among census areas in Christchurch (Hales et al., 2000b). The number of deaths following days with high particulate air pollution (defined as 24 hour average $PM_{10}$ > 50 $\mu g\ m^{-3}$) were compared with deaths on matched unpolluted days (defined as $PM_{10}$ < 50 $\mu g\ m^{-3}$). The possible role of population age structure, relative deprivation (estimated using the New Zealand Deprivation 1996 index) and local exposure to outdoor air pollution from household fires (estimated using a chimney density index) was explored. There was a statistically significant association between mortality and air pollution. Substantial variation in pollution-related mortality among census area units was found. Relative deprivation (but not the proportion of elderly people or chimney density) was found to be a statistically significant predictor of mortality patterns. There was also a positive association between chimney density and relative deprivation. These findings suggest that relative deprivation may increase vulnerability to the effects of particulate air pollution on daily mortality, independently of the effects of age and local variation in exposure.

A risk assessment, based on daily dose-response relationships and current air pollution levels in Christchurch (Foster, 1996) concluded that each year the days of high air pollution (due to all sources) possibly causes 29 extra deaths and 40 extra hospital admissions. In addition, it was estimated that air pollution causes 82,000 days of 'restricted activity', such as absence from school or work due to respiratory symptoms (CRC, 1997). These calculations were revised in 1999 following a more detailed study and an adoption of the 'no threshold' criterion to 40-70 deaths, around 75-100 hospitalisations per year, and 300,000 to 600,000 restricted activity days (Wilton, 1999). The method used was similar to that used by the British Columbia Ministry of Environment, Lands and Parks (BCMELP, 1995) to calculate the health impact of particulate air pollution in the province. For each 10 $\mu g\ m^{-3}$ "increment" of 24-hour particulate air pollution above 20 $\mu g\ m^{-3}$ a certain percentage increase of mortality or morbidity is assumed to occur. For instance, in Christchurch a 1% increase of total daily mortality was assumed to occur for each "increment". These calculations have been widely debated in Christchurch and some critics believe that the lack of local data supporting this risk assessment puts in question the regional air quality management policy.

It should be pointed out that 29 (or 40-70) extra deaths may seem small, as it is only 1% of all deaths in Christchurch during a year. However, these deaths are related to conditions during the 30 worst polluted days. Thus, 29 deaths is about 10% of the deaths during those days. In addition, not all deaths are truly preventable. People still die of 'old age' and many of the deaths during the worst polluted days have nothing to do with air pollution. The 29 extra deaths may therefore be a much larger proportion of the 'preventable' deaths during these days.

Another risk assessment of the health effects of air pollution has been produced for the Land Transport Pricing Study of the Ministry of Transport (MoT, 1996). The aim was to estimate the cost of health damage due to air pollution and other environmental impacts from motor vehicles on roads. Based on a review of a number of epidemiological studies it was

concluded that lifetime exposure to 10 $\mu g\ m^{-3}$ particulate air pollution would increase total mortality by 1.6% and that lifetime exposure to 1 $\mu g\ m^{-3}$ benzene would increase cancer mortality by 4 per million. The estimates were eventually expressed as the estimated cost in dollars per kilometre of road and the cost of particulate air pollution health damage was about 20 times greater than the cost of benzene health damage. These calculations are likely to be very approximate, but they indicate the importance of particulate air pollution when indicators are established to monitor health effects of air pollution.

A few other health effects of air pollution have been published. Dawson et al. (1983) studied the relationship between hospital attendance for acute asthma attacks and air pollution levels in Christchurch during the winter of 1981 and found a negative correlation. No explanation for this unexpected result was found, but the relatively small study size would have limited the statistical power of the study. Another study of asthma in Christchurch children (Wilkie et al., 1995) focussed on potential air pollution during the summer of 1993 around a fertilizer plant. No increase of asthma was found compared to a control group of children from the whole of Christchurch. The pollution situation was quite different from the winter smoke of major concern. The only other study is a panel study of 40 subjects with COPD (Harre et al., 1997), in which their reported prevalence of night time chest symptoms was increased during the day after a 24-hour period when the $PM_{10}$ levels increased by 35 $\mu g\ m^{-3}$ or more. Again, the small study size makes it difficult to draw definite conclusions.

# 5 AIR POLLUTION EXPOSURE

## 5.1 Scope

The purpose of this section is to provide a quantitative assessment of the exposure of the entire population of New Zealand to air pollution. The methodology and outputs follow closely those used in previous overseas studies (particularly Künzli et al., 2000), to allow for comparability of results.

## 5.2 Methodology

The exposure analysis requires the following information:-

**The annual average concentration of $PM_{10}$ to which the population of New Zealand is exposed.**

Since measurements are not made everywhere, all the time, and the population is highly mobile, certain assumptions have to be made, and data constraints taken account of:-

1. **YEAR:** The target analysis year used is 2001. Provisional 2001 census data have recently become available for use in population and emissions analysis. $PM_{10}$ data have been averaged over the last 5 years - where available. This has been done in order to reduce some of the variability in the data - in many cases, only shorter term records are available, often for a single year between 1996 and 2000. These have been used as an estimate in the absence of anything else.

2. **RESOLUTION:** The basic working unit of area is the Census Area Unit (CAU) as defined by Statistics New Zealand. These are convenient units, for which good statistical information is available. They are variable in size, with populations of a few tens of people - in remote rural areas, to a few thousand people - in dense urban areas.

3. **AREAS:** For the purposes of assessment, only CAUs having a population density exceeding 500 people per square kilometre are used. This covers all the main centres, including approximately 80% of New Zealand's population. The final calculations, and reporting, are on a 'city' basis. The choice of the density criteria has been made in order to only include 'cities' and urban areas that are likely to experience exposure to vehicle emissions. There will be many small communities for which the annual average $PM_{10}$ due to vehicles is insignificantly small. The CAUs have been aggregated to a more natural 'city' size, which includes most centres with more than 5000 residents.

4. **MEASURED DATA:** The primary source of $PM_{10}$ data is from local Council monitoring programmes. Results have to be used carefully, as many monitoring sites may not be truly representative of the areas being considered. For instance the Auckland Khyber Pass site is situated at a major intersection, and results are not necessarily representative for residential areas. In the analysis, a conservative

approach has been adopted, using all data, and assuming a general degree of representativeness.

5. **MODELLED DATA:** The secondary source of $PM_{10}$ data is from airshed modelling estimates. For some cities - Auckland, Christchurch and Hamilton - extensive airshed modelling has been conducted which gives a more detailed indication of $PM_{10}$ concentrations over the city. Model results have also been used to aggregate CAUs into larger units, in order to reduce the amount of data processing.

6. **VEHICLE COMPONENT:** Measured and modelled data are separated into two components - total $PM_{10}$, and $PM_{10}$ due to vehicle emissions - using emissions inventory information. The ratio of vehicle emissions to other emissions has been estimated for New Zealand, by Territorial Local Authority (TLA). For cities within these TLAs, this ratio can be used to estimate the fraction of $PM_{10}$ due to vehicles. This analysis has to also account for seasonal variations in emissions, as a large amount of $PM_{10}$ can be attributed to winter home heating in many cities. There are several potential problems with this method, discussed later.,

7. **DERIVED DATA:** Where neither monitoring nor modelling data are available, an estimation of $PM_{10}$ concentrations is made using Statistics New Zealand data on vehicle numbers and population density in the city. This requires a new model of the relationship between vehicle use/population density and the resulting $PM_{10}$ concentrations.

8. **EXPOSURE ASSUMPTION:** It is assumed that all of the people in the city area are exposed to the annual average $PM_{10}$ concentration calculated. This is a conservative assumption, which follows overseas methodology. In general, many people spend much of their time indoors, where $PM_{10}$ concentrations may not be the same as those outside - in many cases the exposure will be lower than average. Conversely, some people spend a significant amount of time in outdoor locations near major traffic routes, where their exposure is considerably greater than average.

9. **EXPOSURE CATEGORIES:** The following $PM_{10}$ exposure categories are used (consistent with Künzli et al., 2000). 0-5, >5-10, >10-15, >15-20, >20-25, >25-30, >30-35, >35-40, >40 $\mu g\ m^{-3}$. (These are referred to later as Categories 1 through to 9).

10. **OUTPUT:** The final output is the number of people exposed to each category, for each city. The basic working tables are by cities (being aggregated CAUs), and the final output is a single national table, and several regional breakdowns.

## *5.3 Data sources*

This project has used as input, the following data sources:-

1. All of the major sources of air pollution monitoring data available in New Zealand. These are summarised in Table 5.1.
2. Population data from Statistics New Zealand.
3. Emissions inventory data from the National Emissions Inventory (NIWA, 1997).
4. Airshed modelling results for Auckland and Christchurch. (Gimson, 2001: Scoggins et al, 2001).
5. Analysis of meteorological data affecting $PM_{10}$ concentrations.

***Table 5.1. Data sources for air pollution monitoring in New Zealand.***

| **Region** | **Particulate Monitoring** |
|---|---|
| Northland | Hi-Vol , various places, shorter term |
| Auckland | Hi-Vol, 1994-99, Penrose, Takapuna<br>Med-Vol, 1998-99, Khyber Pass, Mt Eden, Henderson<br>Med-Vol, 1999, Queen St<br>TEOM, 1996-99, Takapuna |
| Waikato | TEOM, 1999, Peachgrove Rd.<br>Beta Gauge, 1995-99, rural Huntly<br>Hi-Vol, 1995-96, Huntly |
| Bay of Plenty | Mini-Vol, 1996, Rotorua<br>TEOM, 1996, Kawerau |
| Gisborne | Surveys only |
| Hawke's Bay | Med-Vol, 1998, Heretaunga St |
| Taranaki | Surveys only |
| Manawatu /Wanganui | Surveys only |
| Wellington | H-Vol, 1998, Civic Square, Newtown, Avalon, Huia Pool |
| Nelson | Hi-Vol, 2001, City, Victory School, Hospital |
| Marlborough | Surveys only |
| Tasman | Surveys only |
| Canterbury | TEOM, 1994-99, St Albans<br>TEOM, 1995-98, Beckenham<br>TEOM, 1995-99, Hornby<br>Beta Gauge, 1998-99, Opawa<br>TEOM, 1998-99, Rangiora<br>TEOM, 1997-99, Timaru, Ashburton |
| West Coast | Surveys only |
| Otago | Hi-Vol, 2000, Dunedin City, Green Island, N.E. Valley<br>Hi-Vol, 2000, Mosgiel<br>Hi-Vol, 2000, Alexandra |
| Southland | Hi-Vol, 1998, Southland Dairy Co-op |

## Measurement methods

Measurements of $PM_{10}$ are made by several different techniques - Hi-Vol, Med-Vol, Mini-Vol, Beta Gauge and TEOM. There are some known differences between these methods, which have been assumed negligible for the purposes of this study. The one exception is the known underestimate of the TEOM method due to inlet heating. This applies only in the Canterbury region (which uses several TEOMs) and has been corrected using factors established by Environment Canterbury's studies. (Note that these corrections are of the order of 1.2 - 1.4 times the 'TEOM measured' value to obtain the 'standard' value).

TSP data have not been used, as the relationship between TSP and $PM_{10}$ is highly variable. Data using optical monitors - such as the Grimm - have similarly not been included, as the relationship to the Hi-Vol standard has not yet been fully investigated.

## Proportion due to vehicles

The measured data reflects concentrations due to all sources. The purpose of this study is to examine effects due to vehicle sources alone. $PM_{10}$ in New Zealand comes from four main source categories - vehicles, industrial emissions, domestic (or area) emissions, and natural sources (such as sea spray). In different parts of the country these occur in different proportions. For instance in many cities, particularly in the South Island, the burning of coal and wood for domestic heating is the predominant source on an annual basis. Home heating generally only occurs during the winter months (April/May to September/October). However even in the summer months, domestic sources can contribute non-negligible amounts, through various combustion sources and small business activities. In some areas - such as Taranaki - westerly winds bring sea salt inland as fine particulates, and this is probably the dominant component of the $PM_{10}$.

Thus a method is required to apportion the contribution of the ambient $PM_{10}$ concentration due to vehicles. This has been done by analysing the proportion of emissions using the emissions inventories. For areas where emissions inventories have been calculated this is done directly, and for areas where it has not, it is inferred from census data on population (as a surrogate for domestic sources) and vehicle numbers (as a surrogate for vehicle sources).

This methodology has been checked by using results from detailed urban airshed models in the two cities where these are available - Auckland and Christchurch.

The proportion of $PM_{10}$ due to vehicles varies from 80-90% in very dense central urban areas, to 60-70% in busy urban areas, to 40-50% in city suburbs, to 20-30% in smaller city areas, to 10% in rural areas.

The cases for South Island cities - particularly Christchurch - are highly variable throughout the year. For instance the ambient $PM_{10}$ due to vehicles, analysed on a monthly basis, shows only a 10% contribution in winter, but a 90% contribution in summer. In the winter case the emissions are dominated by domestic fires. In the summer case, the proportion is very similar to that found in Auckland, where vehicle emissions dominate. In the calculations here, these differences have been averaged out, and a figure of 40% used for Christchurch and most other South Island cities.

The application of these ratios to produce annual average exposures is somewhat subjective, both on the grounds that for some areas no confirming monitoring data are available, and for

some areas the seasonal variations are substantial. However the results of airshed modelling for Auckland and Christchurch confirm that the ratios used are realistic.

## *5.4 Concentration results*

### Data derivation

Tables in the Appendix summarise all the $PM_{10}$ data available, as both peak 24-hour concentrations and annual averages. The $PM_{10}$ concentration due to vehicles are also calculated using the emissions ratios discussed above.

These are the basic data used in this study.

Some derivations need to be made for areas where no data are available, and to assess the proportion due to vehicle emissions. The methods used are discussed below.

### City areas

Air quality is affected by the emissions over some natural 'airshed' region. These airsheds do not in general correspond to either a CAU, a TLA, nor other geopolitical area. They are usually a complex mix of geographical and weather related factors. These regions are sometimes relatively easy to establish - for instance in a relatively flat area, with light winds and a high frequency of calm conditions (such as Hamilton) - they will be closely aligned to the emissions area (which in turn is usually very closely aligned to the population density). However in other instances the airsheds are very complex - for instance in Auckland, with a highly variable meteorology and geography across the region.

The city areas used for this study have been defined using a combination of population density and geography.

For most smaller to medium city areas - such as Rotorua, New Plymouth, Timaru, etc - these are defined as the urban area (using the population density criteria noted previously).

For larger, or complex, city areas - such as Tauranga, Wellington, Christchurch and Dunedin - these have been refined by splitting the area into two or three natural airsheds. For Auckland the 'city' airshed have been determined using output from extensive numerical airshed modelling research which shows that each of the five areas chosen exhibits particular characteristics - both in emissions and resultant pollutant concentrations - which are different from adjoining areas. An example output of this process is shown in Figure 5.1, using NOx emissions (which in Auckland are closely correlated with $PM_{10}$).

(NB These Auckland 'city' areas are actually quite closely aligned with the TLA boundaries. This is probably due to the way the cities were set up and have developed - the airshed areas have been determined from airshed modelling and their correspondence with the existing city boundaries is coincidental).

The city areas used are illustrated in Figures 5.2 a, b, c, d, for the areas chosen for Auckland, Wellington, Christchurch and Dunedin.

*Figure 5.1* ***Results of airshed modelling analysis for Auckland, showing areas of similar 'air pollution risk'.***

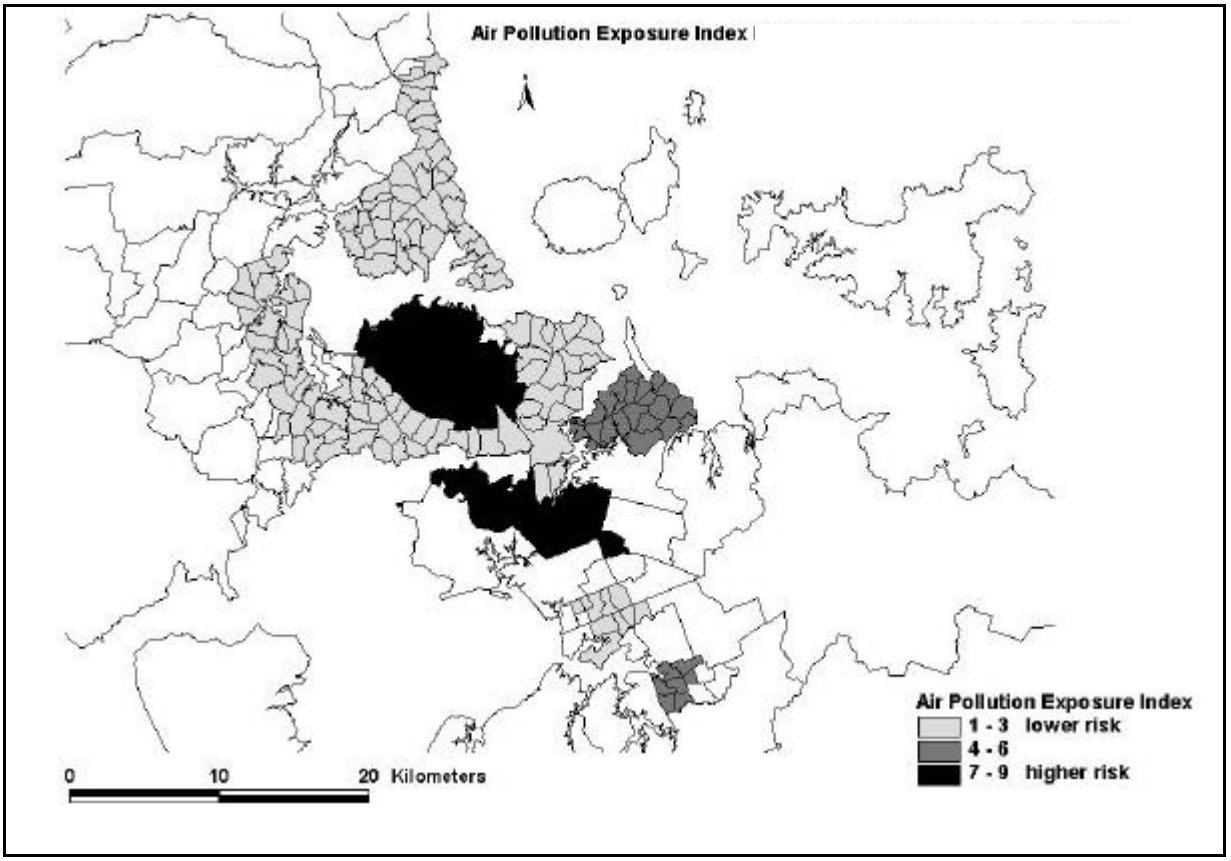
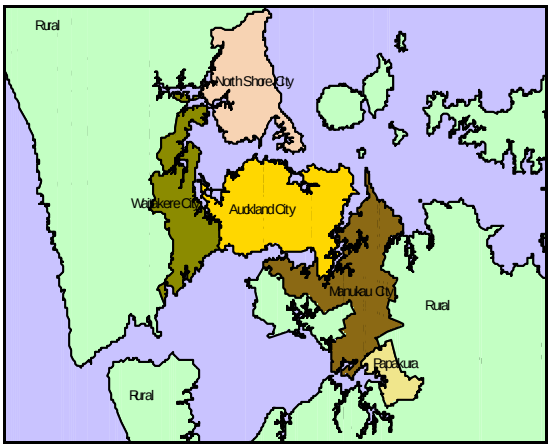
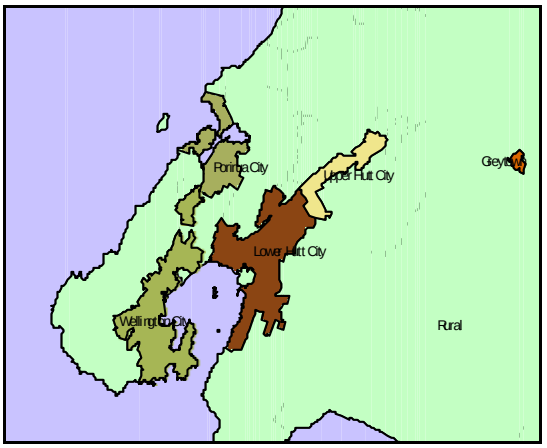



*Figure 5.2 a, b.* ***City areas used as basic working units, based on population and geographical characteristics - major urban areas, North Island.***

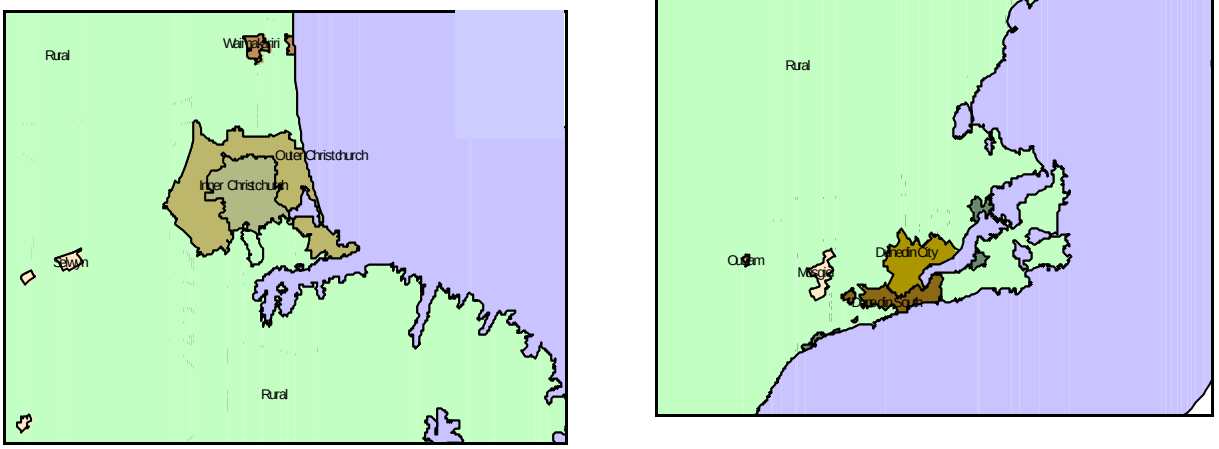



AUCKLAND




WELLINGTON

*Figure 5.2 c, d.* ***City areas used as basic working units, based on population and geographical characteristics - major urban areas, South Island.***




CHRISTCHURCH DUNEDIN

## Concentration estimates

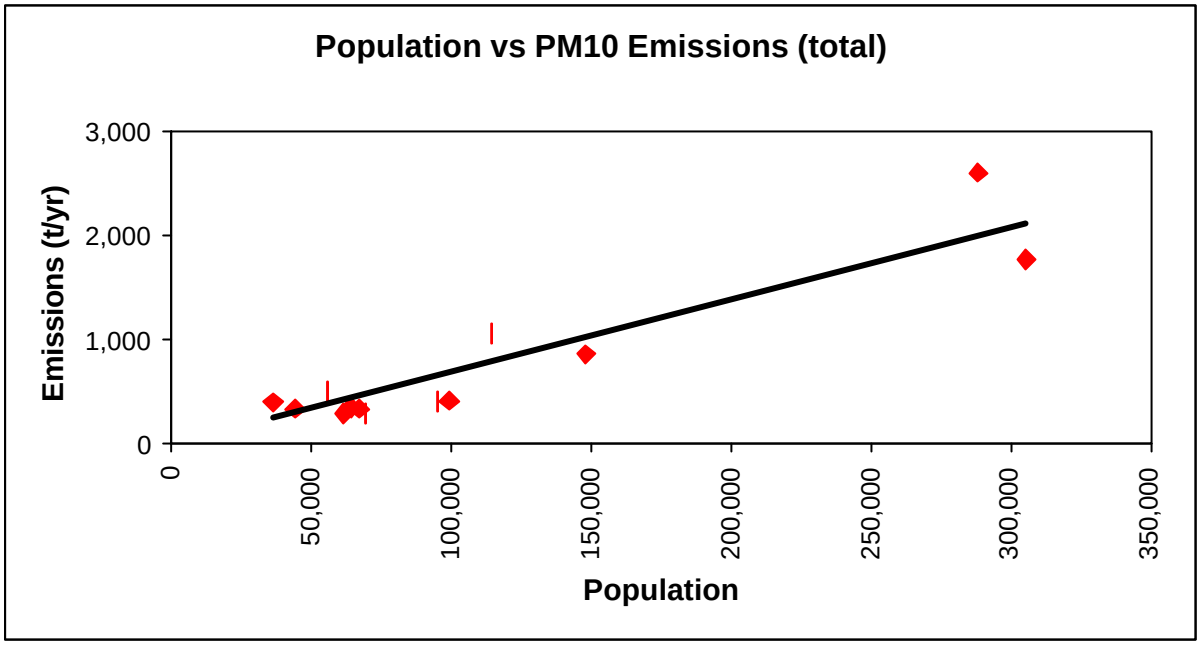
Tables in the Appendix show the concentration results for areas where some monitoring has occurred. An assessment needs to be made for areas where there has been no suitable monitoring. This has been done by using a simplified model, as follows.

It is postulated that there is a reasonably direct relationship between the emissions of $PM_{10}$ and the concentration in air. The nature of this relationship will vary according to site specific factors - mainly the geographical exposure and weather of the area. An indication of this relationship is derived using monitoring data (Tables in the Appendix), for a range of cities in New Zealand. It is further assumed that each area can be categorised by assuming a similarity with an appropriate similar area - for instance Taupo is assumed to be similar to Rotorua (because of broad scale weather and geographical exposure), so the emissions/concentration relationship from Rotorua (where there are measurements) can also be applied to Taupo (where there are no suitable measurements).

This is not necessarily true for each hour, of even each day, but is assumed valid for periods which are long enough to average out short term weather variations - such as over a year.

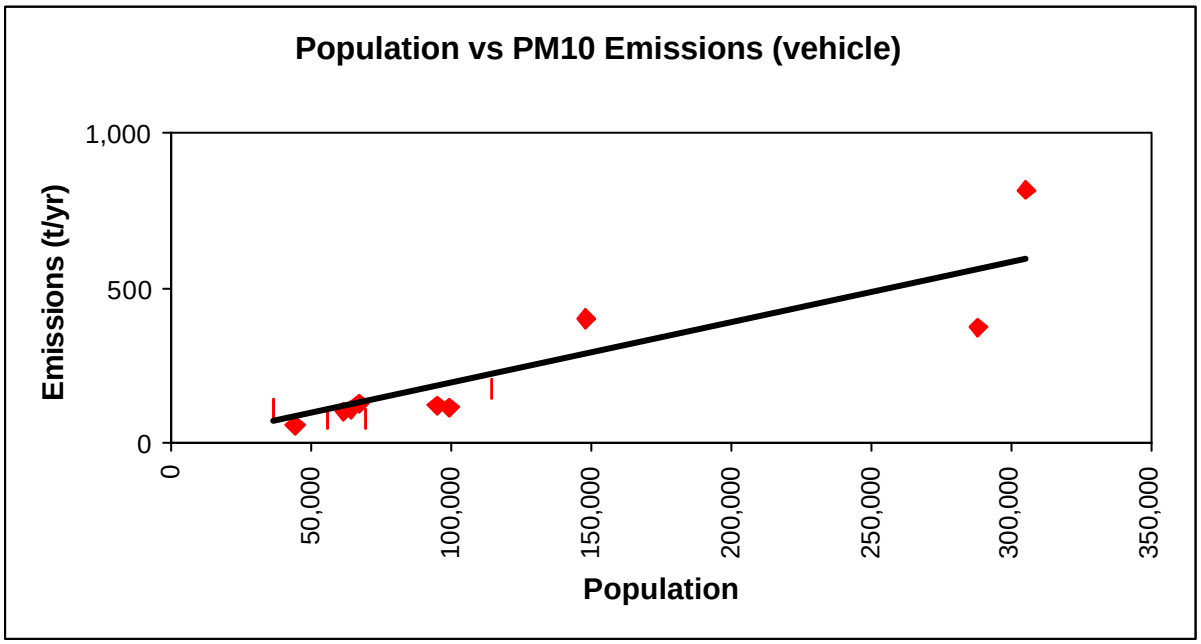
Firstly, there is a direct relationship between population and emissions - as illustrated in Figures 5.3 a and b, for total emissions and transport related emissions respectively.

*Figure 5.3 a. Relationship between population in a city area, and the total $PM_{10}$ emissions (in tonnes per year) for that area.*




Note that the use of 'population' as an indicator is subjective. However other relevant indicators show a very similar correlation - whether it is housing density, population density, or vehicle numbers. A similar relationship holds for vehicle related emissions (Figure 5.3 b).

*Figure 5.3 b. Relationship between population in a city area, and the transport related $PM_{10}$ emissions (in tonnes per year) for that area.*




The data shown in Figures 5.3 a and b are not sufficient to describe any relationship between population (or any other pressure indicator) and ambient $PM_{10}$ concentration. The weather factor can be important - i.e. for a given city size, with a given $PM_{10}$ emission total, the amount of windiness will have an effect on how much $PM_{10}$ can build up. A good example is

say the difference between two cities of similar size - New Plymouth (which is relatively exposed with a high frequency of strong winds) and Napier (which has fewer strong winds, and thus higher average $PM_{10}$ concentrations).

In order to account for this weather factor, an analysis was undertaken of the relationship between emissions/population and resultant annual average $PM_{10}$ concentration for each area where sufficient data were available.

Along with this, the "weather factor" for the areas was calculated using a measure of the number of hours of 'calm' periods in a year, where 'calm' is defined as an hourly average wind speed less than 2 m $s^{-1}$.

The detailed results are not presented here, but they show clear categories, according to whether the area is strongly influenced by 'calms' or not. It is shown that for those cities defined as having a high frequency of calms, a given level of $PM_{10}$ emissions can result in a higher ambient concentration due to the build up of $PM_{10}$.

These relationships are used to derive the expected annual average concentration of $PM_{10}$ in areas where insufficient data are available (shown in Appendix B).

## Uncertainty ranges

Concentrations, and hence exposures, of $PM_{10}$ vary greatly in space and time. Even the most comprehensive monitoring data will still be subject to uncertainties. The data used in this study is the best available, but some account must be made of the size on the inherent uncertainties.

Some concept of the uncertainties due to year to year variations can be obtained by comparing measurements for different years, at sites where this information available. There are several of these, particularly in Auckland and Christchurch. The data (shown in tables in the Appendix) generally show a variation of around +/- 10% for Auckland, and +/- 20% for Christchurch. The higher variability in Christchurch is probably due to the strong influence of home heating emissions that in turn depend on weather factors. Any trends in the data over the 4-6 year records are probably masked by this inherent variability.

Spatial variations are more difficult to deal with, since the required density of observation sites is not available anywhere in New Zealand. Some account of spatial variation is made in choosing the 'city areas' used, but there will be inevitable unresolved variations within these areas.

In order to quantify the uncertainty range, for the further analysis, the following assumption has been made. The exposure calculations are made on the 'best estimate' concentration - representing expected average annual $PM_{10}$ concentrations. To account for variations due to different conditions from year to year, the expected concentrations for 'low exposure' and 'high exposure' years have also been calculated . For the North Island, the low exposure and high exposure estimates are the best estimate exposure +/- 10%. For the South Island the range is +/- 20%. Whilst not perfect, these uncertainties are broadly consistent with the actual data record. Exposure categories are thus calculated using each of these three estimates.

## Final concentrations

The peak and annual average $PM_{10}$ ($\mu g\ m^{-3}$) exposures have been assessed for all cities, by total effects and by derived vehicle effects. The full tables are shown in Appendix B. These

combine the values obtained from monitoring (shown in Appendix A) and the values derived using the methods described above.

## *5.5 Discussion*

The determination of $PM_{10}$ concentrations, and the subsequent population exposure analysis, is a crucial component of this study. It is difficult, and as discussed throughout this section of the report, subject to many assumptions and uncertainties. How these have been handled may be subject of debate, and there are many viable alternative methods which could have been used. Some of these are discussed, and the choices made are further justified.

### Extreme days

Firstly, the whole of the current analysis has been conducted using annual average concentrations. As stated, this has been done to follow the methodology of the Künzli study. However it is possible that two areas having identical annual averages may not experience the same health impact, due to the way the concentrations occur. In one extreme the concentration may be the same every day (Auckland is a little like this), but at the other extreme the average may be due to most days being relatively clean, with a few very high concentration days (Christchurch is a little like this). Is it clear that the public health effects in these two cases are the same? Perhaps not. One can postulate (based on many studies, going as far back as the 1952 London event) that one 'extreme' day has a greater impact that a whole series of 'average' days. This is probably true - but the overall effect is likely to be worse that that identified here.

This is indeed the topic for current and future research, especially in Christchurch, where such extreme days do occur.

### Natural sources

It is likely that many of the measurements of $PM_{10}$ throughout New Zealand contain varying amounts of sea salt. There is an argument that this should be excluded from the exposure analysis, being a 'natural' contaminant. This is taken account of to some extent in the use of thresholds when analysing exposure - that is only considering the effects of $PM_{10}$ concentrations increments above 7.5 $\mu g\ m^{-3}$. However it is recognised that this is an aspect of the analysis which requires further investigation, once more information becomes available from current studies on source fractions of $PM_{10}$ in the major New Zealand cities.

### Seasonal variations

In a similar manner to the case discussed above for extreme days, there are obvious seasonal differences in almost all monitoring records. This is due to two main factors (a) differences in emissions - for instance home heating only occurs in winter, and to a greater extent in the South Island, and (b) differences is dispersion - for instance concentrations tend to be higher in winter because of a greater frequency of inversions and light winds.

These differences can be minor - of the order of few percent - in northern areas such as Auckland, but can be substantial in southern areas such as Christchurch.

There may also be significant seasonal variation in other confounding factors - such as peoples' general health - which tends to be worse in winter, the amount of time people spend

outdoors - which tend to be more in summer, and perhaps the occurrence of other contaminants such as ozone and nitrogen dioxide.

It is difficult to explicitly account for these factors and some reliance has been placed on the averaging out of effects over the whole period of one year.

## Vehicle proportion

Finally, the procedure for determining the fraction of the $PM_{10}$ concentration due to vehicle emissions is fraught with difficulties. This is impossible to measure directly, and so must be derived. But here are many issues to deal with:-

- Emissions from different source occur at different times of the day - vehicle contributions might dominate during rush-hours, but be negligible at night.
- Emissions vary during day of the week - vehicle emissions tend to be lower on Sundays, but perhaps population exposure is greater on the weekends as more people are outdoors.
- The fractions will definitely vary through the season, particularly in regions with home heating emissions.
- Vehicle emissions tend to occur near to the ground, so perhaps they have more of an effect than industrial emissions occurring well above the ground.
- Particles of soot from vehicles, potentially carrying traces of toxic substances, may have a greater effect that those from other sources.
- Some people spend a significant amount of time in or around vehicles, whereas others may spend almost no time in significantly exposed situations.

This list serves only to identify the issues, and it has not been possible to include any more detailed analysis of these factors in this study at this time. Some of these factors will serve to make the health effects worse, and some better. Only when substantial amounts of further research has been done can these effects be quantified.

However, it must be re-iterated that the existence of these uncertainties should not be used to undermine the implications of the results. As is shown in later sections, the results do have significant implications for the public health of New Zealanders, even if the uncertainties may seem large.

## 5.6 Exposure results

### Total NZ population

The results from the section above are applied to population data for each of the city areas used. The city areas cover all cities and towns in New Zealand with over 5,000 residents. The exposure analysis has been conducted using residents over the age of 30, to be consistent with the overseas research. Whilst full 2001 population data were available and used, the proportion of over 30s was not, and is taken from the 1996 census. It is assumed that all of the target population in the area is exposed to the same annual average. The annual $PM_{10}$ averages are assigned to one of the 9 exposure categories, and thus the total population exposed to this category is calculated, along with an uncertainty range as discussed above. The results are shown in Table 5.3 a for the total exposure to $PM_{10}$, and in Table 5.3 b for the vehicle related component.

***Table 5.3 a.*** ***<u>ALL NEW ZEALAND:</u> Number of people (over 30) exposed (000s), by category, for TOTAL annual average $PM_{10}$ (mg $m^{-3}$). The table includes all cities and towns with more than 5000 people, representing 78% of the total population. ('Best', 'Low', and 'High' estimates are based on the full range of expected particulate exposures - see text). 1,552,000 people.***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 320 | 248 | 373 | 297 | 314 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **282** | **168** | **326** | **322** | **340** | **114** | **0** | **0** |
| High exposure year | 0 | 178 | 190 | 276 | 266 | 215 | 313 | 114 | 0 |

***Table 5.3 b.*** ***<u>ALL NEW ZEALAND:</u> Number of people (over 30) exposed (000s), by category, for VEHICLE RELATED annual average $PM_{10}$ (mg $m^{-3}$). The table includes all cities and towns with more than 5000 people, representing 78% of the total population. ('Best', 'Low', and 'High' estimates are based on the full range of expected particulate exposures - see text). 1,552,000 people.***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 129 | 788 | 305 | 130 | 200 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **95** | **562** | **460** | **235** | **200** | **0** | **0** | **0** | **0** |
| High exposure year | 30 | 485 | 453 | 383 | 0 | 200 | 0 | 0 | 0 |

Tables 5.3 a and b cover 1,552,378 over-30 year olds in New Zealand, out of the total over-30 year population of 1,768,511. The total all-ages population is 2,884,684 according to the 2001 census.

*(Explanatory note on Tables 5.3 a and b: The distribution of people across categories is not smooth - for instance in Table 5.3 b, moving from the 'best estimate' to the 'high exposure' year estimate seems to shift 200,000 out of one category, into a higher one, leaving no people in the category. This seems unrealistic, as it is expected that the exposure functions would be smooth curves. However this eventuality is an artefact of having to work with discreet categories - as in the exposed concentration ranges.)*

These are the basic annual average $PM_{10}$ exposure figures for New Zealand that are used in the epidemiological analysis. (A detailed breakdown by city size is given in Appendix C).

## Regional breakdown

Further information can be gained by examining the breakdown between cities and regions. The exposure results have been calculated separately for Auckland, Wellington, Christchurch, Dunedin, all other North Island towns (>5000 people), and all other South Island towns (>5000 people).

The details are given in Appendix D.

# 6 HEALTH EFFECTS

## 6.1 Scope

The purpose of this section is to apply the air pollution exposure information derived in the previous section to assess the health effects on the population. It covers effects due to fine particulates, using the same methodologies as overseas research.

## 6.2 Calculation methods

This report focuses on the mortality effects estimated using the methodology used by Künzli et al., 2000 (Künzli, 2000). As described above a number of other health effects may occur due to air pollution, and the "mortality effect" may be considered just the "nose of the hippo" of the health impact of air pollution from traffic.

The method of calculating mortality effects uses the data on number of people exposed at different annual $PM_{10}$ levels as listed in tables in the previous section and applies "hockey-stick" dose-response relationships to each group. Different assumptions of the threshold for mortality effects occurring in these relationships are applied. Above the threshold a linear increase of mortality risk is assumed at 4.3% above the background mortality rate for New Zealand in 1996 for each 10 $\mu g\ m^{-3}$ annual average increase of $PM_{10}$ (Künzli et al., 2000). The threshold is assumed to be 7.5 $\mu g\ m^{-3}$ (as per Künzli et al., 2000) and for comparison - 10, and 5 and 0 $\mu g\ m^{-3}$.

The formula (Künzli et al., 1999) for calculation of air pollution associated mortality is:-

$$Po = \frac{Pe}{1 + [(RR - 1)(E - B) / 10]}$$

- Po = baseline mortality, after deducting the air pollution effect (this will depend on the other variables).
- Pe = the observed mortality in the population (age > 30) = 12.8 / 1000.
- E = observed average $PM_{10}$ exposure level in each calculation group (varies between groups as identified in exposure tables).
- B = threshold $PM_{10}$ exposure level for mortality effect. Four options are given: 7.5 $\mu g\ m^{-3}$ (as in the Künzli report), 5, 10 and 0 $\mu g\ m^{-3}$ (the latter only applied to vehicle-related $PM_{10}$, as this part of $PM_{10}$ is on top of a non-zero level due to other sources).
- RR = the epidemiologically derived relative risk for a 10 $\mu g\ m^{-3}$ increment of $PM_{10}$, assuming a liner dose-response relationship above the threshold (B). RR was concluded to be 1.043, with a 95% confidence interval of 1.026 – 1.061 (Künzli et al., 2000).

The increased mortality is then calculated:-

$$D_{10} = P_o * (RR - 1)$$

$D_{10}$ = the number of additional deaths per 1000 people to the baseline mortality for a 10 $\mu g/m^3$ increase in $PM_{10}$.

And then:-

$$N_c = D_{10} * P_c * (X_c - B) / 10.$$

$N_c$ = the number of deaths due to $PM_{10}$ for the population in category 'c' of exposure.
$P_c$ = the population ('000s) in category 'c' of exposure.
$X_c$ = the average exposure level in category 'c'.

The calculation is made here for the total number exposed and the two largest sizes of urban areas included applying the four different assumptions of exposure threshold for the start of mortality effects. Appendix D shows results with the detailed calculations.

## *6.3 Dose-response relationships*

As the Künzli study has such an important role in this analysis, it is described it in some detail below. The dose-response relationship used in the Künzli study was derived from two long-term studies in the USA, and these will also be described in some detail in order that the reader understands the basis for the calculations.

### The Künzli study

This study was published in September 2000 in the well-respected medical science journal The Lancet. It presented the results of an international collaborative study, which was funded by the National Science Foundation (USA), the Austrian Federal Ministry of Environment, Youth and Family Affairs (and other Austrian government agencies), the Agency for Environment and Energy Management, France, and the Federal Department of Environment, Transport, Energy and Communications, Switzerland.

A detailed methodological report was prepared for the 3rd WHO Ministerial Conference of Environment and Health, London, 1999 (Künzli et al., 1999). The study analysed the public health impact of outdoor and traffic-related air pollution in three countries: Austria, France and Switzerland. The conclusion was that outdoor air pollution caused 6% of total mortality, and half of this was related to air pollution from motorized traffic. The estimated number of traffic air pollution related deaths was about twice the number of traffic crash deaths, so the term "hidden road toll" seems apt for this type of air pollution public health impact.

The annual average outdoor air pollution exposure levels in 1 $km^2$ grid squares covering each of the countries was estimated using GIS methodology and a combination of air monitoring and emission inventory data. Previous research has related increased mortality to each of the major air pollutants (references). As the air concentrations of these pollutants are often

closely correlated it was decided to use $PM_{10}$ as a proxy for total air pollution and a "useful indicator of several sources of outdoor air pollution such as fossil-fuel combustion". The contribution by traffic to the estimated air pollution levels was calculated from Swiss emission-dispersion models. The traffic share of total outdoor $PM_{10}$ varied according to the $PM_{10}$ level. For concentrations of $PM_{10} < 15$ $\mu g\ m^{-3}$ the share was 28%, increasing to 58% for $PM_{10} > 40$ $\mu g\ m^{-3}$. (this relationship may be the opposite in New Zealand, as high $PM_{10}$ is likely to be due to wood and coal smoke from home fires).

Population data for the 1 $km^2$ grid squares was used to estimate the exposed population at different annual $PM_{10}$ levels. The $PM_{10}$ data by grid square were categorized into groups in 5 $\mu g\ m^{-3}$ steps (0-5, >5-10, >10-15, etc. $\mu g\ m^{-3}$ annual mean). Thus, for each country the population living in areas with different $PM_{10}$ exposure categories could be calculated.

In order to calculate the air pollution associated deaths, dose-response relationships reported from two long-term exposure epidemiology studies form the USA (Dockery et al., 1993; Pope et al., 1995) were used. It was assumed to have a "hockey-stick" shape, with no additional mortality risk below 10 $\mu g\ m^{-3}$ (thus, none below 7.5 $\mu g\ m^{-3}$ for the average of the category 5-10 $\mu g\ m^{-3}$). Above this threshold the mortality increased according to a linear function with a slope of 4.3% increase of mortality for each 10 $\mu g\ m^{-3}$ increase of annual $PM_{10}$. This estimate was derived from the two studies described below.

By accumulating the annual air pollution attributable deaths for all of the grid squares, the total air pollution impact and the impact from traffic air pollution was calculated. Uncertainties of the estimates were quantified and 95% confidence intervals reported, but the exact method for quantification of confidence intervals was not reported.

## Studies providing the dose-response relationship for the Künzli study

Two studies are the only published studies that have analysed the associations between longer-term (annual) average $PM_{10}$ levels and longer-term mortality. A large number of studies have demonstrated associations between daily $PM_{10}$ levels and daily mortality, including one study in Christchurch (Hales et al., 2000a). These studies have been reviewed in a number of reports (e.g. NRC, 1998) and will not be dealt with in detail here.

The so-called "six city study" (Dockery et al., 1993) was a prospective cohort study of 8111 white adults (aged 25 - 74 years) in six US cities (Portage, Topeka, Watertown, Harriman, St Louis and Steubenville) where the long-term average "inhalable particle" levels ($PM_{10}$) were 18, 24, 26, 31, 33 and 47 $\mu g\ m^{-3}$. Individual data on age, sex, weight, height, education level, complete smoking history, occupational history and medical history was available. Spirometric test results were also available. Much of the air pollution would be due to traffic. Power stations, industry and home heating could be other sources, but these were not identified. It was assumed that the people from each city were exposed to the average level of $PM_{10}$ in that city.

It was found that when all the potentially confounding variables in the study were taken into account, there was a significant increase of total mortality and cardio-pulmonary mortality when comparing the worst polluted and the least polluted city. The rate ratios (RR) were 1.26 (95% confidence interval = 1.08-1.47) and 1.37 (1.11 – 1.68) respectively. Lung cancer also had a tendency for increase (RR = 1.37), but it was not statistically significant. The combined mortality of all other causes of death was not increased (RR = 1.01). The difference of the mean long-term $PM_{10}$ levels was about 60 $\mu g\ m^{-3}$. As the total mortality was increased 26%

due to this difference the conclusion would be that the mortality increase per 10 $\mu g\ m^{-3}$ $PM_{10}$ is 4.3%. This is the logic behind the RR of 1.043 used in the Künzli report. The Dockery et al., (1993) report also provides data on "fine particle" exposures ($PM_{2.5}$). The difference between the worst and least polluted cities was 30 $\mu g\ m^{-3}$ for $PM_{2.5}$. Thus, a dose-response relationship for $PM_{2.5}$ would use an RR function of 8.3% increase per 10 $\mu g\ m^{-3}$ instead of the 4.3% for $PM_{10}$.

It is interesting to note that the Dockery et al., (1993) study also quantified the combined effect of smoking and air pollution. Non-smokers in the study had a non-significant RR of 1.19 for total mortality comparing the worst and least polluted cities, while current and former smokers had an RR of 1.33 (1.03 – 1.70).

The other long-term study quoted is the study by Pope et al. (1995). This study covered 552,138 volunteer adults included in a cancer prevention study. Individual information was collected about age, sex, weight, height, race (4.1% were black), smoking history, alcohol use, occupational exposures, and other characteristics. The participants' mortality was monitored over 7 years. Their air pollution exposure was based on the address at the time of entry into the study, and data on air pollution monitoring in the cities where they lived. Fine particle ($PM_{2.5}$) one-year average levels varied between 9 and 33.5 $\mu g\ m^{-3}$ (difference = 24.5 $\mu g\ m^{-3}$).

Multiple regression analysis showed an increase of all-cause mortality associated with air pollution level. The RR for the fine particle range of 24.5 $\mu g\ m^{-3}$ was 1.17 (1.09-1.26), equivalent to a 6.9% increase of mortality per 10 $\mu g\ m^{-3}$ $PM_{2.5}$. When converted to a risk function for $PM_{10}$, this would mean 3.5% increase of mortality per 10 $\mu g\ m^{-3}$, very similar to the findings in the Dockery et al., (1993) study. Again, cardio-pulmonary mortality was especially increased. However, the study by Pope et al. (1995) did not find a higher mortality increase among smokers.

A large number of time-series studies of the association of daily mortality and daily average $PM_{10}$ levels, including studies in Sydney (Morgan et al., 1998) and Christchurch (Hales et al., 2000a). These studies generally find an increase of total mortality of about 1% per 10 $\mu g\ m^{-3}$ $PM_{10}$. Based on these studies the WHO Air Quality Guidelines (WHO, 2000) recommended a linear dose-response function of 0.74% (0.62-0.86%) increase of mortality per 10 $\mu g\ m^{-3}$ $PM_{10}$ for calculations of daily mortality increases. WHO also recommends a linear dose-response function for long-term exposure and long-term increases of mortality at 10% (3-18%) per 10 $\mu g\ m^{-3}$ $PM_{10}$ (WHO, 2000). This guideline dose-response coefficient is similar to (but higher than) the results found in the two studies quoted above (Dockery et al., 1993; Pope et al., 1995). The use here of the Künzli study coefficient of 4.3% increase/$PM_{10}$ can be considered "conservative" as the use of the WHO guideline coefficient of 10% would more than double the estimates below. It should also be pointed out that the WHO guideline values assume no threshold for the start of the mortality effect, which can significantly increase the estimated number of deaths from air pollution when using these guideline values.

## 6.4 Results

### Absolute mortality

Detailed results for the increased mortality (for over 30 year olds) are given in the Tables in Appendix E. Tables 6.1 a and 6.1 b below highlight the main results, for total exposure and vehicle related exposure respectively. These results are for the absolute mortality increases, not adjusted for any 'years of lost life' effect (see next section).

***Table 6.1 a. Summary of key findings. Best estimates of exposed population deaths per annum in New Zealand city areas due to TOTAL $PM_{10}$ emissions.***

| City size | Threshold $PM_{10}$ for mortality effect | | | |
|---|---|---|---|---|
| | 0 mg $m^{-3}$ | 5 mg $m^{-3}$ | 7.5 mg $m^{-3}$ | 10 mg $m^{-3}$ |
| All cities >5,000 | na | 1160 | **970** | 812 |
| **Detail** | | | | |
| >100,000 | na | 788 | **693** | 593 |
| 30,000-100,000 | na | 300 | **239** | 190 |
| 5,000-30,000 | na | 72 | **38** | 29 |

***Table 6.1 b. Summary of key findings. Best estimates of exposed population deaths per annum in New Zealand city areas due to VEHICLE related $PM_{10}$ emissions.***

| City size | Threshold $PM_{10}$ for mortality effect | | | |
|---|---|---|---|---|
| | 0 mg $m^{-3}$ | 5 mg $m^{-3}$ | 7.5 mg $m^{-3}$ | 10 mg $m^{-3}$ |
| All cities >5,000 | 953 | 583 | **399** | 285 |
| **Detail** | | | | |
| >100,000 | 624 | 423 | **318** | 235 |
| 30,000-100,000 | 237 | 123 | **69** | 42 |
| 5,000-30,000 | 92 | 37 | **12** | 7 |

At an annual $PM_{10}$ threshold for mortality effects of 7.5 $\mu g\ m^{-3}$ (the one used by Künzli et al., 2000) the "best estimate" of the number of people in New Zealand dying in non-external causes that may be associated with traffic air pollution is 399 per year (all people in urban areas with more than 5,000 population). Most of these deaths occur in urban areas with more than 100,000 population (318 deaths) and most of the remainder occur in urban areas with

between 30,000 and 100,000 population (69 deaths). Only 12 traffic air pollution deaths are estimated to occur in the urban areas with population between 5,000 and 30,000.

Table 6.1 also shows that moving the assumption of the effect threshold changes the estimated air pollution mortality substantially. With no threshold at all for this fraction of the $PM_{10}$ exposure the total number of traffic air pollution deaths is 953 per year. The typical vehicle related exposure estimates for most areas are of the order of 5-15 $\mu g\ m^{-3}$ lower than the exposures to total $PM_{10}$. Thus the assumption of 'no threshold' for vehicle related $PM_{10}$ has a default threshold of about 10 $\mu g\ m^{-3}$. (This is also the reason that no figures are given in Table 6.1 a for 0 $\mu g\ m^{-3}$ threshold - it is not a valid quantity to calculate. Expressed another way - there is always some non-zero particulate concentration in the air, even in the cleanest possible natural conditions).

With a threshold at 5 $\mu g\ m^{-3}$ the number due to vehicle emissions is 583 deaths per year, and using a "conservative" assumption of a threshold at 10 $\mu g\ m^{-3}$ results in 285 deaths.

All of these numbers are higher than the traffic accident road toll in the same age range (that is taking the fraction of over 30-years olds shows 222 deaths per year - see Table 4.1).

In the Künzli et al. (2000) study it was concluded that the "traffic air pollution road toll" in Austria, France and Switzerland was about twice as high as the "traffic accident road toll". This is called the pollution/accident ratio in the total road toll. In these European countries it is about 2-3. In New Zealand it is about 0.8 using the number 399 above and the traffic accident road toll for all ages which is 502. The lower ratio in New Zealand is to be expected, as there is a higher traffic accident mortality rate than most European countries, and New Zealand's urban air pollution levels due to traffic are likely to be lower than in Europe.

This is shown in more detail in Table 6.2. The total mortality for both air pollution due to vehicles, and due to accidents, are shown for France, Austria, Switzerland and New Zealand.

***Table 6.2. Analysis of total mortality due to road toll and air pollution.***

| Country | Pop. (m) (1996) | Traffic accident deaths | Mortality due to traffic air pollution | Ratio |
|---|---|---|---|---|
| **France** | 58.3 | 8,919 | 17,629 | 1 : 2.0 |
| **Austria** | 8.1 | 963 | 2,411 | 1 : 2.5 |
| **Switzerland** | 7.1 | 597 | 1,762 | 1 : 3.0 |
| **New Zealand** | 3.7 | 502 | 399 | 1 : 0.8 |

## Rates per million people

Table 6.2 shows the totals for each country. An alternative comparison between New Zealand Europe can be made by considering the rates per million people, and taking into account that most of the air pollution related deaths affect adults over 30 years old, shown in Table 6.3.

Table 6.3 also shows that New Zealand has an all-ages accident death rate of 137 people per million. This is higher than Switzerland and Austria, but lower than France.

*Table 6.3. Analysis of rates of mortality due to road toll and air pollution affecting over 30 year olds, per million people.*

| Country | Pop. (m) (1996) | Mortality due to traffic accidents for all ages | Mortality due to traffic air pollution for adults > 30 | Ratio |
|---|---|---|---|---|
| **France** | 58.3 | 153 per million | 501 per million | 1 : 3.3 |
| **Austria** | 8.1 | 119 per million | 487 per million | 1 : 4.1 |
| **Switzerland** | 7.1 | 84 per million | 400 per million | 1 : 4.8 |
| **New Zealand** | 3.7 | 137 per million | 196 per million | 1 : 1.4 |

New Zealand has a total mortality rate of over 30 year olds due to vehicle related air pollution of 196 per million people. However, as might be expected, this rate is less than that found in European countries, which generally have more vehicles and suffer worse air pollution.

## Years of life lost

A more refined estimate of the health effects is obtained by including an analysis of the age of people affected, and the years of life lost by pre-mature mortality. This factor has been discussed in the previous study (Künzli et al., 2000), but not included in their results tables. Taking the 'years of life lost' into account results in an "adjusted" 'air pollution mortality effect' for New Zealand over 30 year olds of 200. (This is essentially a correction factor to make a comparison with the "accident road toll" more valid - see Section 4.3.).

## Regional breakdown

Mortality figures have also been calculated for the major centres and the North Island and South Island. The summary results are shown in Tables 6.4 a and b, for both mortality due to total $PM_{10}$ exposure, and for that due to vehicle related emissions.

*Table 6.4 a. Summary of key findings. Best estimates of exposed population deaths per annum in New Zealand regions due to TOTAL $PM_{10}$ emissions.*

| Region | Threshold $PM_{10}$ for mortality effect | | | |
|---|---|---|---|---|
| | 0 mg $m^{-3}$ | 5 mg $m^{-3}$ | 7.5 mg $m^{-3}$ | 10 mg $m^{-3}$ |
| **Auckland** | na | 503 | **436** | 368 |
| **Wellington** | na | 101 | **79** | 57 |
| **Christchurch** | na | 201 | **182** | 163 |
| **Dunedin** | na | 54 | **48** | 43 |
| **Rest of North Is** | na | 193 | **133** | 104 |
| **Rest of South Is** | na | 100 | **80** | 67 |

*Table 6.4 b. Summary of key findings. Best estimates of exposed population deaths per annum in New Zealand regions due to VEHICLE related $PM_{10}$ emissions.*

| Region | Threshold $PM_{10}$ for mortality effect | | | |
|---|---|---|---|---|
| | 0 mg $m^{-3}$ | 5 mg $m^{-3}$ | 7.5 mg $m^{-3}$ | 10 mg $m^{-3}$ |
| **Auckland** | 454 | 321 | **253** | 199 |
| **Wellington** | 121 | 78 | **56** | 40 |
| **Christchurch** | 99 | 61 | **41** | 21 |
| **Dunedin** | 22 | 11 | **6** | 3 |
| **Rest of North Is** | 177 | 71 | **21** | 11 |
| **Rest of South Is** | 75 | 38 | **19** | 10 |

*(NB In Tables 6.4a and 6.4b, the sum of the areas may not add to the totals presented earlier due to rounding in the calculations).*

The largest numbers occur, as expected, in the Auckland region where over a third of New Zealand's vehicles are registered, and which has by far the country's largest traffic volumes and densities.

## Summary

The total mortality for over 30 year olds in New Zealand due to traffic related air pollution is estimated to be 399 people per year. This figure accounts for all pre-mature deaths but does not take into account the age of death.

The years of lost life estimate provides an alternative comparison which may be more valid for assessing the health costs of the air pollution related mortality, or for comparison with the accident road toll. This parameter accounts for the fact that accident mortality generally affects younger people (more years lost), and air pollution mortality generally affects older people (fewer years lost). The adjusted estimate for traffic related air pollution mortality is 200 people per year.

The largest fraction of pre-mature mortality occurs in the Auckland region, affecting 253 people per year. In Christchurch vehicle emissions affect 41 people. In Wellington, 56 people, and in Dunedin, 6 people. For the rest of the North Island, and the rest of the South Island the effects are 21 people, and 19 people respectively.

There is also a strong regional difference in the fraction of total mortality to vehicle related mortality. In Auckland and Wellington, the fraction due to vehicles is 60-70% of the total, whereas in Christchurch and Dunedin it is only 10-20%. This is because the effects of winter-time air pollution from burning wood and coal for home heating are much greater in the South Island, and remain the dominant source of particulates affecting public health.

# 7 RESEARCH GAP ANALYSIS

## 7.1 Scope

The purpose of this section is to provide information and discussion on current gaps in knowledge in the sciences of air pollution and exposure effects, and how these have been accounted for in this study. The discussion covers both the fundamental gaps - which are being addressed by wide-scale international research, as well as New Zealand-specific gaps, which generally relate to either specific local factors, or gaps in local information sources.

## 7.2 Exposure information

### Data availability

While there is a reasonable amount of temporal data (data collected over a time period) for some cities in New Zealand (see Table 5.1), there are notable gaps in data availability (for example Palmerston North and New Plymouth only have data from short term studies, of less than a year duration). Without actual monitored data (or in some cases modelled data) pollution exposure values have been derived based on available data for vehicles numbers and population densities. These are based on simple models. To more accurately estimate exposure, monitoring needs to take place at more locations. In addition, research needs to be undertaken to establish the nature of any relationship between the census variables and monitored pollution values.

### Measurement methods

A further complication alluded to previously relates to the method of monitoring particulates. Measurements of $PM_{10}$ are made by several different techniques – Hi-Vol, Med-Vol, Mini-Vol, Beta Gauge and TEOM. There are some known differences between these methods. In this study, these differences have been assumed negligible (the one exception is the known underestimate of the TEOM method due to inlet heating which has been corrected using established factors). However ideally these assumptions should be tested and accurately quantified. This is particularly applicable in areas where the New Zealand pollution situation is significantly different to most overseas situations, especially in South Island towns and cities where domestic heating is a major $PM_{10}$ source (unlike most other overseas towns and cities).

### Representativeness of sampling sites

The exposure information used in this study and that of Künzli et al. (2000), and most other similar studies, links the health of individuals in a population to the quality of air as measured at a fixed sampling point (often related to a pre-defined areal unit such as census area units (CAUs)). This approach inherently makes the assumption that the individuals are exposed to a quantity of pollution as measured at the point. The validity of this assumption depends to some extent on the representativness of the sampling site as an indicator of personal pollution exposure. In most overseas studies the validity of using a fixed site as an indicator of personal exposure is a cause for some concern (Vostal, 1994). In New Zealand where there is a relatively limited number of fixed sampling sites, the location of them is of huge

significance. Currently there is limited evidence indicating how representative monitoring sites are of general population exposure and so more research is needed to ascertain this.

## Spatial variation

Spatial variation in particulate pollution has been examined in a variety of studies and is often seen to vary significantly across urban areas (Lam et al., 1999; Monn, 2001; Kingham et al., 2000; Fisher et al., 2000; Van der Wal and Janssen, 2000). There is limited spatial sampling in most New Zealand cities, with few studies examining spatial patterns across urban areas (Sturman, 1982). While something may be known about the level of pollution at a particular site and of how representative it is of exposure in the local vicinity, less is known about spatial variability across urban areas. More spatial sampling needs to be done to assess the validity of the current monitoring sites and to give a clearer picture of spatial patterns of pollution.

Additionally, there is evidence that there can be variation in particulate concentration with distance above the ground. This has implications for the height at which samplers are located, while it has been suggested that vertical variation can also lead to different height groups of the population being exposed to different pollutant concentrations (Colls and Micallef, 1997; Micallef and Colls, 1998). Most of such research has been done in areas where traffic is the main source of particulate matter. In many parts of New Zealand, there are significant other sources (such as domestic heating), which makes it more important to understand more of the vertical variation of pollution concentrations.

## Short term temporal variation

This study, the work of Künzli et al. (2000) and other similar studies have compared long term pollution averages (in this case annual averages) with health. There is some discussion in the literature about the health impacts of short term peak exposures compared to long term averages. While there is ample evidence that pollution varies temporally over long periods (years, months etc), there has been less research looking at variations over minutes and hours. In the case of New Zealand some monitoring at the daily time scale has been undertaken in Canterbury and in parts of Auckland. However, to more clearly understand the nature of peak exposures, detailed short term temporal sampling needs to be undertaken at more sites.

## Indoor air

A further aspect of personal exposure that Künzli et al. (2000) and other studies rarely account for is indoor air quality. There is evidence that people actually spend very little time outdoors (Koponen et al., 2000). A number of studies have concluded that in developed countries people spend between 87% and 89% of time their indoors, the majority at home (Ashmore, 1995; Jenkins et al., 1992; Monn et al., 1997; Robinson and Nelson, 1995). In addition it is worth noting that those groups most at health-risk from air pollution, the young, the old, women and those with existing health conditions (BMA, 1997), are also those potentially more likely to spend a greater amount of time indoors.

The relationships between indoor and outdoor particulate pollution have been researched in recent years as a result of association with traffic-related pollution. Ratios of indoor to outdoor pollution vary between studies from 0.5 to 2 and greater (Kim and Stock, 1986). In homes with indoor sources (such as domestic heating) indoor/outdoor ratios were generally greater than 1, showing that the exposure of subjects to particles can be greater than outdoors (Jones et al., 2000). In homes with no obvious indoor source, the indoor/outdoor ratios are likely to be less than or equal to one (Chao et al., 1998; Monn et al., 1997).

Limited research has been done on indoor air quality in New Zealand (Comins, 2001). Yet there are many features of the New Zealand situation that make it potentially unique. These include limited insulation in older homes (thus allowing more infiltration), high use of natural gas in un-flued heaters, and the use of wood and coal as common sources of home heating (thus providing indoor sources of pollution).

## Personal mobility

As previously discussed this study along with Künzli et al. (2000) and others inherently assumes that individuals are exposed to a quantity of pollution as measured at a point. While the validity of this assumption depends to some extent on the representativness of the sampling site, even if monitors were located at the home of every person in New Zealand, it would still not be possible to know each individuals personal pollution exposure. In reality this varies according to a wide variety of factors beyond merely home location. Most people spend large parts of their time away from their home area, often at a place of work or education. Most studies of this type are unable to account for this. However, research has been able to show that the activities people are involved in and the location of them will affect their personal pollution exposure.

As an example there is some European research that has identified differences in personal pollution exposure to particulates according to the individual's chosen mode of transport (Bevan et al., 1991; den Tonkelaar and van der Tuin, 1983; Kingham et al., 1998; Rank et al., 2001; Sitzmann et al., 1996). This research consistently shows that those people travelling by car are exposed to the highest levels of pollution.

There is substantial evidence that an individual's workplace may also have an effect on pollution exposure, especially for people working in polluted environments (for example Alderson, 1983; Pukkala, 1995). From the perspective of this study those working in close proximity to road traffic are of particular concern (Watt et al., 1995).

The EXPOLIS study has attempted to examine personal pollution exposure over various sites in Europe (Boudet et al., 2001; Edwards et al., 2001a; Edwards et al., 2001b; Helm et al., 2000; Koistinen et al., 2001; Koistinen et al., 1999; Kousa et al., 2001; Rotko et al., 2000a; Rotko et al., 2000b). Among the pollutants studied was $PM_{2.5}$. Boudet et al. (2001) discussed the validity of using fixed air monitors in order to assess a population exposure based on results from the European EXPOLIS study. They devised a weighting scheme that used available monitoring site data to account for population based time-activity patterns. Rotko et al. (2000) and Koistinen et al. (2001) suggest that some socio-economic, behavioural and environmental determinants can impact personal exposure. No research of this kind has been carried out in New Zealand.

## Pollution concentrations and emissions

Some Regional Councils collect data on emissions to aid in devising air quality management strategies (e.g. Environment Canterbury). However, relationships between emissions and measured pollution concentrations are relatively un-researched in the international literature, while in New Zealand little has been published, and current research using airshed modelling is not yet completed (Scoggins et al, 2001). Yet again, the situation of New Zealand with domestic heating as a significant source of particulates in many areas, makes applying international figures to the local situation rather difficult. More and better emissions data need to be collected, and research needs to be done to more fully understand the relationship between emissions and ambient concentrations.

## Pollution and meteorology

The severity of the air pollution problem in New Zealand urban areas is not merely a function of the emissions, as ambient concentrations are also dependent on local meteorology. It is known internationally that the state of the atmosphere determines its ability to disperse or remove the air pollution emitted into it (Elsom, 1992). Wind speed and the vertical temperature profile are major controls of measured air pollution levels, and these parameters are known to vary significantly between differing urban areas in New Zealand, as well as more locally. However, meteorological measurements are generally not available at a small enough time and space scale to allow clear relationships between atmospheric processes and air quality to be identified. Some research is currently underway, based on short periods of intensive measurements and computer modelling techniques, to understand more fully the local atmospheric controls on air pollution (Sturman et al. 2001). For example, these observations have shown that vertical mixing of air pollution in the Christchurch area is restricted to an unusually shallow surface layer compared to overseas experience (Spronken-Smith et al. 2001). These shallow layers are seen to trap high concentrations of pollutants, including carbon monoxide (Corsmeier et al. 2000). The long term installation of vertical wind and temperature profiling systems would significantly increase knowledge of the atmospheric processes that affect human exposure to air pollution.

Existing work is being used to inform regional councils about how the atmosphere contributes to the dynamic nature of the air pollution problem, and the implications for development of air quality strategies (Sturman and Zawar-Reza, 2001). Further work is required to apply international knowledge and modelling tools to answer local questions relating to such issues as the transport of pollutants from their sources across urban areas, the identification and maintenance of buffer zones of clean air around urban areas, and the appropriate location of monitoring sites so that they are representative of ambient air pollution concentrations. Where accurate emissions estimates are available, atmospheric models can be used to predict changes in ambient air pollution levels resulting from proposed future control strategies. They can also be applied to assessment of the environmental impact of new developments, such as highways, where discharges to the air are likely to be significant.

### Summary of 'exposure information' research gaps

**New Zealand priorities**

- Monitoring needs to take place in more urban areas.
- Establish the nature of relationships between census variables and monitored pollution values.
- Spatial sampling to assess the validity of the current monitoring sites and to give a clearer picture of spatial patterns of pollution.
- Provide a better understanding of vertical variations in pollution levels, especially in areas where there are significant other sources of particulates.
- Detailed short term temporal sampling needs to be undertaken to more clearly understand the nature of peak exposures.
- Identify the relationships between indoor and outdoor air, especially in areas of significant indoor pollution sources.
- Develop more refined and integrated models of both concentrations and population exposures, taking account of more detailed demographic and activity patterns.

**Generic**

- Differences between sampling methods should be tested and accurately quantified.
- Identify better, and model, the relationship between emissions and concentrations.

## 7.3 Causes of particulate health effects

Some scientists do not fully accept that the associations between fine particles in air and morbidity and mortality are causal because the biological mechanisms of what cause the health effects remain unclear. There is much effort worldwide to try to elucidate a biological mechanism that will explain the results observed in epidemiological studies. Many possible pathways have been postulated and been shown to be plausible. Indeed at this point in time, the question is no longer what is the biologic mechanism but rather which of several observed mechanisms is responsible for the observed epidemiologic associations (Dockery, 2001).

The biological effects of a particle are primarily determined by the physical and, probably also, the chemical, nature of the particle, the physics of particle deposition and distribution in the respiratory tract, and physiological events that occur in response to the presence of the particle. Particle size is the most important characteristic influencing deposition in the airways, causing airways constriction. Particles larger than 5 μm in diameter deposit in the upper airways or larger lower airways. Particles smaller than 5 μm in diameter deposit in the smaller, such as the bronchioles and alveoli.

Recent studies indicate that inflammation as well as airways constriction may be important in explaining the respiratory effects of particles, and that crustal particles as well as combustion particles can evoke the same response. These studies are reported in detail elsewhere (Denison, Rolfe and Graham, 2000). The role of the macrophage in the observed responses is emerging as a key issue. There are two types of macrophages in the lung – stimulatory and suppressant. The stimulatory macrophages cause the immune system to respond to the presence of a foreign material and try to remove it. An excess of stimulatory macrophages can cause damage to the lung in the process of removal of the foreign material. Suppressor macrophages are present to stop that happening. The alveolar macrophage is the principal lung cell in the regulation of the immune response to inhaled pathogens and the development of inflammation.

It has been shown that when suppressor macrophages are exposed to urban air particles, both crustal and combustion, they undergo apoptosis ("cell suicide"). This causes an imbalance in favour of the stimulatory macrophages, leading to an inflammatory response (Horlian et al., 1998). Asthmatics and people with other chronic lung diseases have only approximately one-third the suppressor macrophages of people with healthy lungs. It seems likely therefore that this imbalance would make these groups more susceptible to the inflammation responses observed in isolated cells.

Less well understood are the cardiovascular effects of particles. Recent studies report that episodes of particle air pollution are associated with indicators of autonomic nervous responses of the heart, such as increased heart rate and decreased heart variability. It has been suggested that particles may trigger an inflammatory response in the lung that causes the release of chemical mediators which affect the autonomic control of the heart (Stone and Godleski, 1999). An alternative hypothesis is that particle air pollution alters the coaguability of blood (Seaton, 1996).

Research on the chemical composition of the particles and the combined effects of exposures to particles in the presence of gaseous pollutants is also of importance, and can contribute to the mechanisms causing the effects. Of note is the different concentration of gaseous pollutants in New Zealand urban areas, which tend to be low in sulphur compounds and

relatively higher in nitrogen and chlorine compounds than many Northern hemisphere countries (Fisher, 2000).

**Summary of 'health effects' research gaps**

**New Zealand priorities**

- Identify the effects in the presence of other pollutants, particularly in New Zealand specific conditions.

**Generic**

- Identify the biological causes of the particulate effects on health.
- Identify the mechanisms of respiratory irritation.
- Determine the cellular response function.

## *7.4 Epidemiological information*

### Relating health effects to particular pollutants

Most epidemiological studies, including this one, have used $PM_{10}$ as an indicator of air pollution. $PM_{10}$ is likely to be a good indicator for these purposes, because fine particles are likely to be biologically significant, but in real life situations air pollution is a complex mixture of chemical species. At present not enough is understood about how the various components in this mixture affect health singly, and in combination. These questions concern air pollution researchers everywhere. Particular issues in New Zealand include the implications for health of size and source profiles of fine particles in local environments, and interactions between pollutants in specific hot spots. (For instance, little is known about the possible interaction between vehicle emissions and products of home fires in Christchurch.).

### High risk groups

It is unlikely that everyone in the population is affected to the same extent by air pollution, but relatively little is known about susceptibility. High risk groups are likely to include the elderly, infants, and people with chronic lung and heart conditions. Given the high prevalence of the disease in New Zealand, an important question is whether people with asthma are at increased risk of dying from air pollution. No information from local populations on this point is available. Work done in Christchurch has shown that areas of high social deprivation (measured by the New Zealand Deprivation Index) have greater increases in daily mortality for a given average $PM_{10}$ level than are observed in areas of low deprivation. (Hales et al., 2000b) This finding needs to be tested elsewhere, and the implications explored. No work has been carried out on the effects of air pollution by ethnicity. Given differences in socio-economic status, smoking rates and the prevalence of chronic diseases of the heart and lung, one would expect Maori and Pacific populations to be more severely affected by air pollution. However there may be other factors associated with ethnicity (such as, perhaps, quality and location of housing, access to health services) that are important, and these deserve closer attention.

### Mortality under 30 years

This report is concerned only with deaths in adults aged over 30. This was to replicate the calculations carried out by Künzli et al., and reflects the fact that by far the greatest number of deaths occur in middle age and amongst the elderly. However, in terms of years of life lost or premature mortality, deaths at earlier ages are particularly important, and warrant separate analysis. Research into indoor air pollution has shown that children are particularly vulnerable

in the first three years of life, and several overseas studies have reported an increase in infant mortality with elevated outdoor $PM_{10}$ levels (Bobak and Leon, 1999; Loomis et al., 1999).

### Morbidity

The number of deaths due to air pollution is likely to be very small compared with the number of non-fatal illnesses attributed to the same cause. Work done elsewhere has linked a large number of non-fatal health outcomes to particulates and other air pollutants. Examples include hospital admissions (all causes), episodes of wheezing, GP visits for respiratory ailments, asthma attacks and restricted activity days. (ref - Künzli's longer report). As was apparent in the literature review, very few studies have been carried out in New Zealand.

### Economic consequences

New Zealand studies of the economic consequences of air pollution (including the health impacts) are generally lacking, with the exception of the work done for the Land Transport Pricing Study (MoT, 1996).

### Integrated analysis

There is a lack of overall assessments, taking a big picture approach to incorporate all the health-relevant aspects of transport policy. These include, in addition to the direct health effects of vehicle emissions, physical activity, injuries and other indirect effects (such as those resulting long-term from climate change). Such an analysis would be used to evaluate the sustainability of various forms of transport and transport infrastructure.

### Summary of 'epidemiological' research gaps

**New Zealand priorities**

- Analyse the different effects on different risk groups.
- Further study the effects on infants and younger age groups.
- Integrated effects analysis of the sustainability of transport systems, including other health factors, social factors, and economic implications.

**Generic**

- Identify better the specific effects of particles of specific sizes and composition.
- Identify the overall mechanisms for health effects from particulates

## *7.5 Other contaminants*

Air pollutants from motor vehicle are inevitably closely correlated. This is also true of species such as ozone and aerosol sulphates and nitrates formed by secondary reactions in the atmosphere. The question *"….can particles cause effects in the absence of sulphur dioxide or sulphates?…"* has been well answered by showing that the health effects of particles occur in regions where these two pollutants are virtually absent (Bates, 2000). Yet this does not prove that where they co-exist, the presence of gaseous species may not enhance the effect of particles. Quite the reverse seems likely. In the case of cardiovascular mortality and morbidity, for example, the presence of carbon monoxide might aggravate the effects of particle pollution. This is an important issue to address, especially in the New Zealand context, with relatively high urban concentrations of carbon monoxide.

However there is another effect due to the presence of gaseous pollution - especially sulphates and nitrates. This is the formation 'secondary particulate'. Little research has been conducted in New Zealand, although some research programmes are currently addressing this issue

(Fisher, 2000b). It is likely that secondary particulate formation will be responsible for 10-20% of the $PM_{10}$ in urban areas, but it will vary with location. One of the contributors to this is the high sulphur content of diesel fuel used. This factor is fully account for in the current study (as the exposure data are based on monitoring which includes the secondary particulate), but it may have more relevance when considering mitigation options.

In accordance with the approach adopted by Künzli et al. (2000) this research has related levels of $PM_{10}$ to mortality. However, there is substantial evidence that other pollutants not considered in this study will also have an effect on mortality (WHO, 2000a). Many of these are traffic-related, such as carbon monoxide, nitrogen dioxide and benzene. Research needs to assess population exposure to other pollutants and quantify health risk.

Of particular concern, both internationally and in New Zealand, is the high concentrations of benzene in urban environments (Fisher, 2000a). Although extensive measurements are not available, it is likely that the proposed new annual average guideline for benzene in air will be exceeded in Christchurch, and perhaps parts of Auckland. The current benzene content of New Zealand fuels is high by international standards.

Other air pollutants are also coming under scrutiny, especially the potentially carcinogenic products of combustion such as polycyclic aromatic hydrocarbons (PAHs). Proposed new guidelines are likely to come into effect in New Zealand. Some preliminary monitoring programmes have shown that concentrations can be high. However the major sources of PAHs are more likely to be wood and waste burning, rather than from vehicles.

Finally, this study has focused on particulates as $PM_{10}$. Of increasing health concern are smaller particles, especially those less than 2.5 micrometers ($PM_{2.5}$), and even smaller ($PM_{1.0}$). While there is a growing quantity of literature looking at $PM_{2.5}$ (for example Koistinen et al., 1999, 2001; Rotko et al., 2000, Monn et al., 1997, Kingham et al., 2000, Fisher et al., 2000) little information has been collected in New Zealand. As well as a need to collect more information on $PM_{2.5}$, research needs to be carried out to ascertain relationships between levels of $PM_{10}$ and $PM_{2.5}$, and to quantify the health impact on New Zealanders of exposure to $PM_{2.5}$. On the longer term, some evidence is appearing that it is not the 'mass' of particulates that leads to an effects, but their 'number' or the total 'surface area'. These are much more difficult quantities to measure, and the process of assessing any implications is only just starting, with results several years away.

## Summary of 'other contaminant' research gaps

**New Zealand priorities**

- The population exposure to all other relevant pollutants needs to be assessed, and further prioritised for attention.
- Determine the concentrations, and hence population exposure and risks, due to benzene.
- Determine the fraction, and sources, of secondary particulates.
- Ambient concentrations of $PM_{2.5}$ need to be measured at more locations.

**Generic**

- Quantify the relationship between concentrations of $PM_{10}$ and $PM_{2.5}$.
- Quantify the health impacts of exposure to $PM_{2.5}$.
- Review the implications of new research on measuring particulate parameters.

# 8 SUMMARY

A study of the health effects due to air pollution emissions from vehicles in New Zealand has been conducted, based on available exposure data and following the methodology of a recent European study.

Firstly, the background issues of the health effects of vehicle emissions have been discussed, highlighting the prioritised focus on particulates. Secondly, an assessment has been made of the overseas research results, and their applicability to the New Zealand case. There is ample evidence to suggest that both the epidemiological results and the methodologies used overseas are directly applicable to New Zealand.

The New Zealand situation is analysed in detail, with summaries of previous research conducted on the public health effects of air pollution exposure.

Using all relevant and available particulate monitoring data from around New Zealand, exposure information is derived for all people living in cities and towns with populations over 5,000. The air quality indicator used for assessing health effect is the annual average ambient concentration of $PM_{10}$, following the methodology of the overseas studies. This includes exposures due to all sources (total exposure) and exposure due to emissions from motor vehicles alone (vehicle related exposure). This covers approximately 80% of the population, including all people living in potentially polluted urban areas (the excluded 20% are all in smaller and remote communities and are unlikely to experience any significant exposure).

The air pollution exposure data have been used to assess the number of people, over the age of 30, who risk experiencing early mortality. These results are shown in Table 8.1, compared to equivalent results for three European countries as published recently in The Lancet (Künzli et al, 2000). The European countries have higher populations and higher traffic numbers, and generally higher densities of both, so direct comparison with New Zealand is not totally valid.

***Table 8.1. Mortality due to total air pollution and the traffic related component.***

| Country | 1996 Long-term Mortality Estimates for Adults ³ 30 years; (95% Confidence Intervals) | | Population (1996)[2] | Road Toll (1998)[2] |
|---|---|---|---|---|
| | Total Air Pollution | Traffic Air Pollution | | |
| **France** | 31,692 (19,202 - 44,369) | 17,629 (10,681 - 24,680) | 58.26 mil | 8918 |
| **Austria** | 5,576 (3,370 - 7,813) | 2,411 (1,457 - 3,378) | 8.06 mil | 963 |
| **Switzerland** | 3,314 (1,986 - 4,651) | 1,762 (1,056 - 2,472) | 7.08 mil | 597 |
| **New Zealand**[1] | 970 (586 - 1,376) | 399 (241 - 566) | 3.66 mil | 502 |

*1. New Zealand exposure data calculated on 2001 census population figures.*
*2. Population and road toll data from the OECD International Road Traffic Accident Database 2001.*

Table 8.1 is a simplified summary of what are essentially very complex results presented and discussed throughout this report. This table is provided to show a particular comparison with the results of the European study quoted extensively in this report, although the figures given may not be the most appropriate to use in every application. For instance, the average years of life lost for a death caused by a traffic accident is about twice as many years as for an air pollution related death. Therefore, when comparing the 'air pollution road toll' with the 'traffic accident road toll' this difference should be taken into account. Two air pollution deaths may be equivalent to one traffic accident death in overall terms. This factor should be used for a more valid analysis of the health costs of air pollution related mortality. The "adjusted" equivalent mortality number is 200 deaths per year.

There are a number of assumptions made in deriving results in Table 8.1 and earlier Tables, and the final figures are those agreed as best estimates, following as closely as possible the assumptions made in the overseas studies. Firstly, the question of overall exposure to $PM_{10}$ has been made assuming that data from the monitoring stations are applicable to wide, representative areas. This is a reasonable first assumption, which could be revised, but only when much more extensive monitoring data are available. It is likely that some parts of the community will experience lower exposures, but there are also parts which will experience higher exposures - and the likely overall effect is a realistic average.

Secondly, some assumptions have been made to estimate $PM_{10}$ exposures in areas where no monitoring has been conducted. This has been based on a modelled relationship between emissions activities and ambient concentrations, taking account of geographical and weather influences.

Thirdly, the fraction of exposure due to vehicles has been assessed using somewhat limited emissions inventory data, and a knowledge of regional variations caused mainly by different uses of burning wood and coal for home heating. For instance the vehicle fraction in Auckland is much greater than those in the South Island cities which experience a high winter particulate loading from domestic heating using wood and coal.

Fourthly, the health effects have been calculated using a well established relationship derived by the World Health Organisation, and confirmed by one major New Zealand study. The final figures have been derived using the same assumptions as the European study, but within this there are options. For instance the WHO has stated that there is no threshold limit - that is, each incremental increase in $PM_{10}$ results in a consequent incremental increase in mortality - whether the increase is from 0 to 1 $\mu g\ m^{-3}$, or from 100 to 101 $\mu g\ m^{-3}$. Some researchers work on the basis that since there is always some non-zero background concentration of $PM_{10}$, the analysis should account for this, and only assess exposure above some threshold level. Calculations in this study have been made on various threshold levels - 5, 7.5 and 10 $\mu g\ m^{-3}$. The final results reported adopt the 7.5 $\mu g\ m^{-3}$ threshold, although valid arguments can be made for using another threshold (details of all results are given). In summary, using a higher threshold of 10 $\mu g\ m^{-3}$ reduces the 399 per annum mortality to 285 (71%), whereas using a lower threshold of 5 $\mu g\ m^{-3}$ increases the annual mortality to 583 (146%).

The final mortality estimates, although subject to a potentially wide uncertainty due to the assumptions made, have been reported as 'best estimates' based on all the relevant factors, fully discussed in this report. A key point is that even at the extremes, the absolute mortality in over 30 year olds due to vehicle related $PM_{10}$ emissions is of the same order, or only slightly less, than the mortality due to the total road toll.

# 9 ACKNOWLEDGMENTS

This report has been reviewed by:-

- **Caroline Austwick, Ministry for the Environment;**

*Comments:* "The portrayal of precise figures for mortality perhaps implies an accuracy which isn't real. We need to remember that there are a lot of assumptions and uncertainties behind these figure. I suspect the uncertainty range may be larger than that quoted. Overall, it is reasonable first attempt, and does point the way to future research needed. We may need greater certainty in the mortality figures before going on to further stages using these figures"

- **Dr Neil Gimson, NIWA;**

*Comments:* "All the relevant factors appear to have been covered, and it is a pity that not more data are available on the New Zealand specific conditions. However all the potential limitations and assumptions needed are explained in detail."

- **Kevin Mahon, Auckland Regional Council;**

*Comments:* "The results are reasonably consistent with our expectations, and broadly tie in with our own studies. There is very recent USA research (perhaps not yet published), showing higher rates of mortality from diesel particulates, and this should be addressed in any future work. We would like to see more specific and detailed analyses focussing on the Auckland region, which probably has a larger vehicle pollution issue than most other parts of the country. I would also like to see in future a more specific inclusion of 'other pollutants' - particularly toxics and carcinogens relating to the fuel used in the Auckland region. I suspect the mortality effects of these will also be significant - perhaps of the same order as the effects from $PM_{10}$ alone."

- **Bob Ayrey, Environment Canterbury;**

*Comments:* "This is a difficult project to complete, because of the uncertainties involved and the lack of enough New Zealand specific data on the detailed aspects of pollution exposure. For instance we really do not know well enough what fraction of $PM_{10}$ is due to motor vehicles, especially in cities such as Christchurch where home heating combustion emissions dominate the total exposure. We also do not know well enough the concentrations in many smaller centres, as not enough basic monitoring has been conducted. The models used to assess annual concentrations based on activity or emissions factors are subject to numerous influences - such as the weather - and to get a robust method is extremely difficult and probably needs more research and monitoring over several years. I would also emphasise the evolving nature of such work, and the different assessment methods being applied. For instance in evaluating the health effects in Christchurch - studies which have been on-going for several years - we have focused on using the Ministry for the Environment guideline criteria as a basis for deciding on effects, This approach is slightly different to that used here, and the detailed results may thus not be consistent."

# 10 REFERENCES


Alderson, M. (1983). Occupational Cancer. Butterworths. London.

Ashmore, M. (1995). Human exposure to air pollutants. *Clinical and Experimental Allergy* 25, Supplement 3, pp 12-22.

Bascom, R., Bromberg, P.A., Costa, D.A., Devlin, R., Dockery, D.W., Frampton, M.W., Lambert, W., Samet, J.M., Speizer, F.E., and Utel, M. (1996). Health effects of outdoor air pollution: parts I and II, *American Journal of Respiratory Critical Care Medicine*, Vol. 153, pp 477-498.

Bates, D.V. (2000). Interpreting epidemiological studies of the effects of air pollution. *Proceedings of the Clean Air and Environment Conference, Clean Air Society of Australia and New Zealand Inc.*, Sydney, Australia, 26-30 November 2000, pp 15-18.

Bevan, M.A.J., Proctor, C.J., Baker-Rogers, J. and Warren, N.D. (1991). Exposure to carbon monoxide, respirable suspended particulates and volatile organic compounds while commuting by bicycle. *Environmental Science Technology* 25, pp 788-791.

BCMELP (1995). Health effects of inhalable particles: Implications for British Columbia. Vancouver, British Columbia Ministry of Environment, Lands and Parks.

Boudet, C., Zmirou, D. and Vestri, V, (2001). Can one use ambient air concentration to estimate personal and population exposures to particles? An approach within the European EXPOLIS study. *The Science of the Total Environment* 267, pp 141-150.

Brimblecombe, P. (1987). The Big Smoke. Routledge, London, United Kingdom.

British Medical Association (1997). Road Transport and Health. BMA, London.

Burnett, T.B., et al.. (1998). The association between ambient carbon monoxide levels and daily mortality in Toronto, Canada. *J. Air and Waste Manage. Assoc.*, 48, pp 689-700.

Chan, L., Kwok, W., Lee, S. and Chan, C. (2001). Spatial variation of mass concentration of roadside suspended particulate matter in metropolitan Hong Kong. *Atmospheric Environment*, 5, pp 3167-3176.

Chao, C., Tung, T. and Burnett, J. (1998). Influences of different indoor activities on the indoor particulate levels in residential buildings. *Indoor and Built Environment*, 7, pp 110-121.

Chiodo, J. and Rolfe, K. (2000). Health effects of eleven hazardous air contaminants and recommended evaluation criteria. Ministry for the Environment Air Quality Technical Report No. 13, October 2000.

Colls, J.J. and Micallef, A. (1997). Towards better human exposure estimates for setting of air quality standards. *Atmospheric Environment*, 31, 24, pp 4253-4254.

Comins, P. 2001. Impact of home heating on indoor air quality in Christchurch. Unpublished MSc Thesis, Dept of Geography, University of Canterbury.

Corsmeier, U., Fiedler, F., Hübner, C., Kalthoff, N., Megerle, A. and Kossmann, M. (2000). Variation of vertical carbon monoxide profiles with atmospheric stability during wintersmog episodes in Christchurch. 'Fresh Perspectives' – A joint Conference of the New Zealand Hydrological Society, the New Zealand Meteorological Society and the New Zealand Limnological Society, 21-24 November 2000, Christchurch, New Zealand, pp 258-259.

CRC (1997). Take a deep breath. A discussion document about the improvement of Christchurch air quality. Christchurch, Canterbury Regional Council.

Dab, W., Segala, C., Dor, F., Festy, B., Lameloise, P., Le Moullec, Y., Le Tertre, A., Medina, S., Quenel, P., Wallaert, B., and Zmirou, D. (2001). Air pollution and health: Correlation or causality? The case of the relationship between exposure to particles and

cardiopulmonary mortality, *Jn Air and Waste Management Association,* Vol. 51, February 2001, pp 220-235.

Dawson, K.P., Allan, J., and Fergusson, D.M. (1983). Asthma, air pollution and climate: a Christchurch study. *NZ Med J,* 96, pp 165-167.

den Tonkelaar, W.A.M. and van der Tuin, J. (1983). In-vehicle air pollution. Investigation of the exposure of car drivers. Report G 995. IMG-TNO, Delft.

Denison, L., Rolfe, K., and Graham, B. (2000). Health effects of five common air contaminants and recommended protective ranges. Ministry for the Environment Air Quality Technical Report No. 12, October 2000.

Dockery, D.W., Pope, C.A., Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris, B.G. and Speizer, F.E. (1993). An association between air pollution and mortality in six US cities. *New England J of Med*., 329, pp 1753-1759.

Dockery, D.W. (2001). Fine particulate air pollution: Smoke and mirrors of the 90s or hazard of the new millennium?, *Clean Air*, Vol. 35, No. 1, February 2001, pp 31- 34.

Dora, C, and Phillips, M. (2000). Transport, Environment and Health. WHO Regional Publications, European Series, No. 89. WHO, Copenhagen.

Edwards, R., Jurvelin, J., Saarela, K. and Jantuen, M. (2001a). VOC concentrations measured in personal samples and residential indoor, outdoor and workplace microenvironments in EXPOLIS-Helsinki, Finland. *Atmospheric Environment*, 35, pp 4531-4543.

Edwards, R., Juvelin, J., Koistinen, K., Saarela, K. and Jantunen, M. (2001b). VOC source identification from personal and residential indoor, outdoor and workplace microenvironment samples in EXPOLIS-Helsinki, Finland. *Atmospheric Environment*, 35, pp 4829-4841.

Elsom, D. (1992). Atmospheric pollution - causes, effects and control policies. 2nd edition, Blackwell, Oxford, 256 pp.

Fischer, P, Hoek, G., Van Reeuwijk, H., Briggs, D., Lebret, E., Van Wijnen, J., Kingham, S., Elliott, P. (2000). Traffic-related differences in outdoor and indoor concentrations of particles and volatile organic compounds in Amsterdam. *Atmospheric Environment*, 34, 22, pp 3713–3722.

Fisher, G., Gimson, N.R. and Foster, E. (1998). Modelling urban fine particulate concentrations. In: *Proc. of the 11th World Clean Air and Environment Congress.* Durban, South Africa, pp 13-18.

Fisher, G.W. (2000a). Air quality in New Zealand: A 20 year review. *Clean Air.* 34 (2). pp 40-41.

Fisher, G.W. (2000b) The Christchurch Air Pollution Study. *Clean Air*. 34 (3). pp 28-29.

Foster, E. (1996). Health effects of suspended particulate. Technical Report No R96(2). Christchurch, Canterbury Regional Council.

Gimson, N.R. (2001). Developments in air pollution modelling - New Zealand issues and perspectives. Clean Air. 35. pp 32-34.

Hales, S., Salmond, C., Town, G., Kjellstrom, T., and Woodward, A. (2000a). Daily mortality in relation to weather and air pollution in Christchurch, *Australian and New Zealand Journal of Public Health*, Vol. 24, pp 89-91.

Hales, S., Salmond, C., Exeter, D., Purvis, M., Woodward, A., Kjellstrom, T. (2000b). Spatial patterns of mortality in relation to particulate air pollution in Christchurch, 1988-1997. Proceedings of the 12th Annual Colloquium of the Spatial Information Research Centre University of Otago, Dunedin, New Zealand: December 10-13th 2000

Harre, E.S.M. et al. (1997). Respiratory effects or air pollution in chronic obstructive lung disease: a three month prospective study. *Thorax*, 52, pp 1040-1044.

Helm, D., Jantuen, M. and Rotko, T. (2000). Reporting personal results to participants of exposure studies. *The Science of the Total Environment*, 262, pp 191-195.

HMPHS (1954). Mortality and morbidity during the London fog of December 1952. Reports on Public Health and Medical subjects, No 95. London, Her Majesty's Public Health Service.

Holian, A., Hamilton, R.F., Morandi, M.T., Brown, S.D., and Li, L. (1998). Urban particle-induced apoptosis and phenotype shifts in human alveolar macrophages, *Environmental Health Perspectives*, Vol. 106, No. 3, pp 127-132.

Jenkins, P., Phillips, T., Mulberj, J. and Hu,i S. (1992). Activity patterns of Californians: use of and proximity to indoor air pollutant sources. *Atmospheric Environment* 26A, pp 2141-2148.

Jones, N., Thornton, C., Mark, D. and Harrison, R. (2000). Indoor/Outdoor relationships of particulate matter in domestic home with roadside, urban and rural locations. *Atmospheric Environment*, 34, pp 2603-2612.

Katsouyanni, K., Touloumi, G., Spix, C., Schwartz, J, Balbucci, F., Medina, S, Rossi, G., Wojtyniak, B., Sunyer, J., Bacharova, L., Schouten, J.P., Ponka, A., and Anderson, H.R. (1997). Short-term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project, *British Medical Journal*, Vol. 314 (7095), pp 1658-1663.

Kim, Y and Stock, T, (1986). House specific characterisation if indoor and outdoor aerosols. *Environment International* 12, pp 75-92.

Kingham, S, Briggs, D, Elliott, P, Fischer, P and Lebret E, (2000), Differences in concentrations of traffic-related pollutants in indoor and outdoor air in relation to vehicle intensity in Huddersfield, England. *Atmospheric Environment*, 34, 6, pp 905-916.

Kingham, S., Meaton, J., Sheard, A. and Lawrenson, O. (1998). Assessment of exposure to traffic related fumes during the journey to work. Transportation Research D: *Transport and Environment*, 3, 4, pp 271-274.

Koistinen, K., Hänninen, O., Rotko, T., Edwards, D., Moschandreas, D. and Jantunen, M. (2001). Behavioural and environmental determinants of personal exposures to $PM_{2.5}$ in EXPOLIS - Helsinki, Finland. *Atmospheric Environment*, 35, pp 2473-2481.

Koistinen, K., Kousa, A., Tenhola, V., Hänninen, O. and Jantunen, M. (1999). Fine particle ($PM_{2.5}$) measurement methodology, quality assurance procedures, pilot results of the EXPOLIS study. *Jn Air and Waste Management Association*, 49, pp 1212-1220.

Koponen, K., Asmi, A., Keronen, P., Puhto, K. and Kulmala, M. (2001)., Indoor air measurement campaign in Helsinki, Finland 1999 – the effect of outdoor air pollution on indoor air. Atmospheric Environment 35, pp 1465-1477.

Kousa, A., Monn, C., Rotko, T., Alm, S., Oglesby, L. and Jantunen, M. (2001). Personal exposures to NO2 in the EXPOLIS-study: relation to residential indoor, outdoor and workplace concentrations in Basel, Helsinki and Prague. *Atmospheric Environment*, 35, pp 3405-3412.

Künzli, N., Kaiser, R., Medina, S., Studnicka, M., Oberfeld, G. and Horak, F. (1999). Health costs due to road traffic-related air pollution: an impact assessment project of Austria, France and Switzerland. Report to the 3rd Ministerial Conference of Environment and Health, London, 1999.

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., and Sommer, H. (2000). Public-health impact of outdoor and traffic-related air pollution: a European assessment, *The Lancet*, Vol 356, September 2000, pp 795-801.

Lam, G., Leung, D., Niewiadomski, M., Pang, S., Lee, A. and Louie, P. (1999). Street-level concentrations of nitrogen dioxide and suspended particulate matter in Hong Kong. *Atmospheric Environment*, 33, pp 1-11.

McGowan, J., Hider, P., Chacko, E., and Town, G. (2000). Particulate pollution and cardio-respiratory admissions in Christchurch. Paper presented at the Air Pollution and Health Conference, Christchurch, 3-6 August 2000.

Micallef, A. and Collis, J. (1998). Variation in airborne particulate matter concentration over the first three metres from ground in a street canyon: Implications for human exposure. *Atmospheric Environment*, 32 (21), pp 3795-3799.

Ministry of Transport (1996). Land transport pricing study. Environmental externalities. Discussion paper. Wellington, Ministry of Transport.

Ministry of Economic Development (2001). Petrol and Diesel – delivering quality: A review of the Petroleum Products Specifications Regulations, August 2001.

Monn, C., Fuchs, A., Hogger, D., Junker, M., Kogelshatz, T., Roth, N., and Wanner, H.U. (1997). Particulate matter less than 10 μm ($PM_{10}$) and fine particles less than 2.5 μm ($PM_{2.5}$): relationships between indoor, outdoor and personal concentrations *The Science of the Total Environmen*t 208, pp 15-21,

Monn, C. (2001). Exposure assessment of air pollutants: a review on spatial heterogeneity and indoor/outdoor/personal exposure to suspended particulate matter, nitrogen dioxide and ozone. *Atmospheric Environment*, 35, pp 1-32.

Morgan, G., Corbett, S., Wlodarczyk, J., and Lewis, P. (1998). Air pollution and daily mortality in Sydney, Australia, 1989 - 1993. *Am J of Public Health*, 88, pp 759-763.

Morris, R.D. and Naumova, E.N. (1998). Carbon monoxide and hospital admissions for congestive heart failure: evidence of an increased effect at low temperatures, *Environmental Health Perspectives*, Vol. 106 (10), pp 649-653.

NIWA. (1998). Total air emissions inventory for New Zealand. NIWA Report AK98064, prepared for the Ministry for the Environment.

NRC (1998). Research priorities for airborne particulate matter. Washington DC, National Academy Press.

Pope, C.A., Thun, M.J., Namboodiri, M.M., Dockery, D.W., Evans, J.S., Speizer, F.E. and Heath, C.W. (1995) Particulate air pollution as a predictor of mortality in a prospective study of US adults. *Am J Respir Crit Care Med*, 151, pp 669-674.

Pukkala, E. (1995). Cancer Risk by Social Class and Occupation. Karger.

Rank, J., Folke, J. and Jespersen, P.H. (2001). Differences in cyclists and car drivers exposure to air pollution from traffic in the city of Copenhagen. *Science of the Total Environment* 279, pp 131-136.

Rajkumar, T., Guesgen, H.W., Robinson, S., Fisher, G.W. (2000). A new dose model for assessment of health risk due to contamination in air. *Journal of the Air and Waste Management Association*. 50. pp 3 - 20.

Robinson, J. and Nelson, W. (1995). National Human Activity Pattern Survey Database. USEPA Research Triangle Park. North Carolina.

Rotko, T., Koistinen, K., Hänninen, O. and Jantunen, M. (2000a). Sociodemographic descriptors of personal exposure to fine particles ($PM_{2.5}$) in EXPOLIS Helsinki. *Exposure Analysis and Environmental Epidemiology*, 10, pp 385-393.

Rotko, T., Oglesby, L., Künzli, N. and Jantunen, M. (2000b). Population sampling in European air pollution exposure study, EXPOLIS: comparisons between the cities and representatives of the samples. *Exposure and Analysis and Environmental Epidemiology*, 10, pp 355-364.

Samet, J., Zeger, S., and Berhane, K. (1995). The association of mortality and particulate air pollution. In: Particulate air pollution and daily mortality, replication and validation of selected studies, the Phase I Report of the Particle Epidemiology Evaluation Project. Health Effects Institute, Washington DC.

Schwartz, J., Spix, C., Touloumi, G., Bacharova, L., Barumamdzadeh, T., le Tertre, A., Piekarksi, T., Ponce de Leon, A., Ponka, A., Rossi, G., Saez, M., and Schouten., J.P. (1996). Methodological issues in studies of air pollution and daily counts of death or hospital admissions, *Journal of Epidemiology and Community Health*, Vol. 50, Supplement. 1, pp S3-S11.

Scoggins, A., Fisher, G.W., Kjellstrom, T. (2001). Exposure mapping using air quality modelling. Accepted for the 13th Annual Colloquium of the Spatial Information Research Centre, University of Otago, Dunedin, 2-5 December 2001.

Seaton, A. (1996). Particles in the air: the enigma of urban air pollution, *Journal of the Royal Society of Medicine*, Vol. 89, pp 604-607.

Seethaler, R. (1999). Synthesis report of World Health Organisation reports on health costs due to road traffic-related air pollution. An impact assessment project of Austria, France and Switzerland. WHO Ministerial Conference on Environment and Health, June 1999. Fed. Depart. of Environment, Transport, Energy, Communications, Berne, Switzerland.

Sitzmann, B., Kendall, M., Watt, J. and Williams, I. (1996). Personal exposure study of cyclists to airborne particulate matter in London.

Spronken-Smith, R.A., Sturman, A.P. and Wilton, E. (2001). The air pollution problem in Christchurch, New Zealand – progress and prospects. Accepted by *Clean Air.*

STAPPA and ALAPCO (2000). Cancer Risk from Diesel Particulate: National and Metropolitan Area Estimates for the United States. Prepared by the State and Territorial Air Pollution Program Administrators and the Association of Local Air Pollution Control Officials, 15 March 2000.

Stone, P.H. and Godleski, J.J. (1999). First steps toward understanding the pathophysiologic link between air pollution and cardiac mortality, *American Heart Journal*, Vol. 138, pp 803-807.

Streeton, J.A. (1997). A Review of Existing Health Data on Six Air Pollutants. A report to the National Environment Protection Council, Australia.

Sturman, A. (1982). Statistical analysis of spatial patterns of smoke concentrations in Christchurch. *New Zealand Geographer*, 38(1), pp 9-18.

Sturman, A.P., Kossmann, M., Spronken-Smith, R.A. and Zawar-Reza, P. (2001). The Christchurch Air Pollution Study (CAPS) 2000 – An overview and preliminary results. *Proceedings of the Third Urban Air Quality Symposium: Measurement, Modelling and Management*, Loutraki, March 19-23, 2001, pp 68-72.

Sturman, A. and Zawar-Reza, P. (2001). Application of back-trajectory techniques to the delimitation of a Clean Air Zone for the Christchurch airshed. Technical Report U01/3, Environment Canterbury, Christchurch, 23p.

Van der Wal, J. and Janssen, L. (2000). Analysis of spatial and temporal variations of $PM_{10}$ concentrations using Kalman filtering. *Atmospheric Environment*, 34, pp 3675-3687.

Vostal, J.J. (1994), Physiologically based assessment of human exposure to urban air pollutants and its significance for public health risk evaluation. *Environmental Health Perspectives* 102 (Supplement 4), pp 101-106.

Waller, R. (1971). Air pollution and community health, *Journal of the Royal College of Physicians*, Vol. 5, pp 362-368.

Watt, M., Godden, D., Cherrie, J. and Seaton, A. (1995). Individual exposure to particulate air pollution and its relevance to thresholds for health effects: a study of traffic wardens. Occupational and Environmental Medicine, 52, pp 790-792.

WHO (1987). Air Quality Guidelines for Europe. WHO Regional Publications, European Series No. 23, Copenhagen.

WHO (1995). Update and revision of the air quality guidelines for Europe. Document EUR/ICP/EHAZ.94.05/PB01. Copenhagen, World Health Organization

WHO (1996). Evaluating the implementation of the strategy for health for all by the year 2000. Common framework, 3rd evaluation. Geneva, World Health Organization.

WHO (2000). Air Quality Guidelines for Europe, Second Edition. WHO Regional Publications, European Series No. 91, Copenhagen.

WHO/NILU (1996). Quantification of health effects related to $SO_2$, $NO_2$, $O_3$ and particulate matter exposure. WHO Regional Office for Europe and Norwegian Institute for Air Research, Kjeller, Norway. Report NILU – OR 63/96.

Wilkie, et al. (1995). Prevalence of childhood asthma symptoms in an industrial suburb of Christchurch. *NZ Med J*, 108, pp 188-190.

Wilton, E. (1999). Update: The health effects of suspended particulate. Canterbury Regional Council Report U99/51. Environment Canterbury.

Woodward, A., Guest, C., Steer, K., Harman, A., Scicchitano, R., Pisaniello, D., Calder, L. and McMichael, A. (1995). Tropospheric ozone: respiratory effects and Australian air quality goals, *Journal of Epidemiology and Community Health*, Vol. 49, pp 401-407.

Zmirou, D., Schwartz, J., Saez, M., Zanobetti, A., Wojtyniak, B., Touloumi, G., Spix, C., Ponce de Leon, A., Le Moullec, Y., Bacharova, L., Schouten, J., Ponka, A., and Katsouyanni, K. (1998). Time-series analysis of air pollution and cause specific mortality, *Epidemiology*, 9, pp 495-503.

# 11 APPENDICES

### *Appendix A1. BASIC MONITORING DATA*

The tables below summarise all the basic annual average $PM_{10}$ monitoring used in this study. Tables A1a and A1b show the available data from a range of sites, Tables A2a and A2b show the derived proportion due to vehicles.

**Notes on data tables A1 a and b.**

| | |
|---|---|
| Column 1. | City - common name for the area. |
| Column 2. | Location or area within the city. |
| Column 3. | Year of measurement used. Where multiple years are available, the average has been used. This does not account for trends, but the inter-annual variability is too large, and the total record period too small, to determine trends. |
| Column 4. | Peak 24-hour measured $PM_{10}$ concentration anywhere in the record, due to all sources. |
| Column 5. | Annual average measured $PM_{10}$ concentration, due to all sources. |
| Column 6. | Lower estimate of the site annual average $PM_{10}$.(Based on a 'low exposure' year). |
| Column 7. | Average 24-hour concentration at the site, averaging all available years. |
| Column 8. | Upper estimate of the site annual average $PM_{10}$.(Based on a 'high exposure' year). |
| Column 9. | Nominal annual exposure category, where:-<br>1 = 0 - 5 $\mu g\ m^{-3}$<br>2 = 5 - 10 $\mu g\ m^{-3}$<br>3 = 10 - 15 $\mu g\ m^{-3}$<br>4 = 15 - 20 $\mu g\ m^{-3}$<br>5 = 20 - 25 $\mu g\ m^{-3}$<br>6 = 25 - 30 $\mu g\ m^{-3}$<br>7 = 30 - 35 $\mu g\ m^{-3}$<br>7 = 30 - 35 $\mu g\ m^{-3}$<br>8 = 35 - 40 $\mu g\ m^{-3}$ |

(NB All figures are rounded to 1 $\mu g\ m^{-3}$ in each calculation)

*Appendix A1. BASIC MONITORING DATA - Continued*

*Table A1a. Peak 24-hour and annual average $PM_{10}$ (mg $m^{-3}$) assessed for sites where measurements are available, by Total Effects: North Island.*

| City North Island | Site | Year | Total Peak | Total Average | Lower Range | Site Average | Upper Range | Exposure Category |
|---|---|---|---|---|---|---|---|---|
| **Auckland** | Queen St | 1999 | 51.1 | 26.7 | 24 | **27** | 29 | **6** |
| | Khyber Pass | 1998 | 121.4 | 30.0 | | | | |
| | Khyber Pass | 1999 | 62.2 | 27.4 | 27 | **29** | 30 | **6** |
| | Penrose | 1994 | 101.1 | 23.7 | | | | |
| | Penrose | 1995 | 77.3 | 24.8 | | | | |
| | Penrose | 1996 | 48.4 | 25.0 | | | | |
| | Penrose | 1997 | 65.8 | 25.3 | | | | |
| | Penrose | 1998 | 48.1 | 23.4 | | | | |
| | Penrose | 1999 | 80.5 | 24.5 | 23 | **24** | 25 | **5** |
| | Takapuna | 1996 | 46.4 | 13.4 | | | | |
| | Takapuna | 1996 | 22.9 | 16.3 | | | | |
| | Takapuna | 1997 | 49.9 | 18.4 | | | | |
| | Takapuna | 1997 | 50.5 | 19.2 | | | | |
| | Takapuna | 1998 | 57.6 | 18.0 | | | | |
| | Takapuna | 1998 | 38.1 | 15.4 | | | | |
| | Takapuna | 1999 | 47.1 | 16.6 | | | | |
| | Takapuna | 1999 | 39.0 | 14.9 | 13 | **17** | 19 | **4** |
| | Mt Eden | 1997 | 42.0 | 16.1 | | | | |
| | Mt Eden | 1998 | 39.8 | 20.6 | | | | |
| | Mt Eden | 1999 | 45.8 | 20.3 | 16 | **19** | 21 | **4** |
| | Henderson | 1998 | 33.1 | 17.7 | | | | |
| | Henderson | 1999 | 55.4 | 21.4 | 18 | **20** | 21 | **4** |
| **Huntly** | Ralph Rd | 1995 | 29.9 | 7.0 | | | | |
| | Ralph Rd | 1996 | 19.2 | 8.2 | | | | |
| | Ralph Rd | 1997 | 13.4 | 3.6 | | | | |
| | Ralph Rd | 1998 | 10.3 | 4.4 | | | | |
| | Ralph Rd | 1999 | 15.6 | 5.5 | 4 | **6** | 8 | **2** |
| | Croft Tce | 1995 | 48.2 | 12.7 | | | | |
| | Croft Tce | 1996 | 30.1 | 13.0 | 13 | **13** | 13 | **3** |
| **Hamilton** | Peachgrove | 1999 | 43.8 | 19.5 | 18 | **20** | 21 | **4** |
| **Rotorua** | Town | 1996 | 30.1 | 21.9 | 20 | **22** | 24 | **5** |
| **Hastings** | Heretaunga | 1998 | 37.0 | 25.6 | 22 | **24** | 26 | **5** |
| **Kawerau** | Mill | 1996 | 132.0 | 14.0 | 13 | **14** | 15 | **3** |
| **Wellington** | Civic | 1998 | 42.7 | 20.7 | 19 | **21** | 23 | **5** |
| | Avalon | 1998 | 29.3 | 14.3 | 13 | **14** | 16 | **3** |
| | Newtown | 1998 | 17.9 | 11.3 | 10 | **11** | 12 | **3** |
| **Lower Hutt** | Huia Pool | 1998 | 37.8 | 12.1 | 11 | **12** | 13 | **3** |

*Appendix A1. BASIC MONITORING DATA - Continued*

*Table A1b. Peak 24-hour and annual average e $PM_{10}$ (mg $m^{-3}$) assessed for sites where measurements are available, by Total Effects: South Island.*

| City South Island | Site | Year | Total Peak | Total Average | Lower Range | Site Average | Upper Range | Exposure Category |
|---|---|---|---|---|---|---|---|---|
| **Nelson** | City | 2001 | 65.0 | 24.9 | 20 | **25** | 30 | **5** |
| | Victory | 2001 | 36.2 | 24.0 | 19 | **24** | 29 | **5** |
| | Hospital | 2001 | 27.9 | 16.8 | 13 | **17** | 20 | **4** |
| **Christchurch** | St Albans | 1995 | 255.0 | 33.6 | | | | |
| | St Albans | 1996 | 221.0 | 32.1 | | | | |
| | St Albans | 1997 | 335.0 | 35.1 | | | | |
| | St Albans | 1998 | 181.0 | 23.8 | | | | |
| | St Albans | 1999 | 222.0 | 31.4 | 24 | **31** | 35 | **7** |
| | Beckenham | 1995 | 127.0 | 25.2 | | | | |
| | Beckenham | 1996 | 168.0 | 22.8 | | | | |
| | Beckenham | 1997 | 51.0 | 18.3 | 18 | **22** | 25 | **5** |
| | Opawa | 1995 | 137.0 | 30.9 | | | | |
| | Opawa | 1996 | 148.0 | 33.3 | | | | |
| | Opawa | 1997 | 237.0 | 34.8 | | | | |
| | Opawa | 1998 | 229.0 | 23.9 | 24 | **31** | 35 | **7** |
| | Hornby | 1995 | 112.0 | 30.1 | | | | |
| | Hornby | 1996 | 118.0 | 15.7 | | | | |
| | Hornby | 1997 | 126.0 | 33.7 | | | | |
| | Hornby | 1998 | 85.0 | 22.0 | 16 | **25** | 30 | **6** |
| **Rangiora** | Town | 1998 | 19.0 | 12.4 | | | | |
| | Town | 1999 | 82.0 | 20.5 | 12 | **16** | 20 | **4** |
| **Ashburton** | Town | 1997 | 47.0 | 24.1 | | | | |
| | Town | 1998 | 50.0 | 17.7 | | | | |
| | Town | 1999 | 96.0 | 24.8 | 18 | **21** | 25 | **5** |
| **Timaru** | Town | 1997 | 150.0 | 36.6 | | | | |
| | Town | 1998 | 102.0 | 26.7 | | | | |
| | Town | 1999 | 157.0 | 31.6 | 27 | **32** | 37 | **7** |
| **Dunedin** | City | 2000 | 48.0 | 21.0 | 17 | **21** | 25 | **5** |
| | N E Valley | 2000 | 57.0 | 18.0 | 14 | **18** | 22 | **4** |
| | Green Island | 2000 | 107.0 | 32.0 | 26 | **32** | 38 | **7** |
| **Alexandra** | Town | 2000 | 108.0 | 30.0 | 24 | **30** | 36 | **6** |
| **Mosgiel** | Town | 2000 | 70.0 | 26.0 | 21 | **26** | 31 | **6** |
| **Southland** | Dairy Co-op | 1998 | 46.0 | 10.0 | 8 | **10** | 12 | **2** |

*Appendix A2. DERIVED VEHICLE DATA*

**Notes on data tables A2 a and b.**

| | |
|---|---|
| Column 1. | City - common name for the area. |
| Column 2. | Location or area within the city. |
| Column 3. | Year of measurement used. Where multiple years are available, the average has been used. This does not account for trends, but the inter-annual variability is too large, and the total record period too small, to determine trends. |
| Column 4. | Calculated annual average ratio of $PM_{10}$ emissions due to vehicles. This is the total over the year, and may vary on shorter time scales due to seasonal differences in emissions sources - for instance the average for central Christchurch is 0.4, which is a nominal average of approximately 0.1 in winter (when there are high home heating emission rates) and 0.9 in summer (when vehicles are the main source). |
| Column 5. | Peak 24-hour measured $PM_{10}$ concentration anywhere in the record, due to vehicle sources. |
| Column 6. | Annual average measured $PM_{10}$ concentration, due to vehicle sources. |
| Column 7. | Lower estimate of the site annual average $PM_{10}$. (Based on a 'low exposure' year). |
| Column 8. | Average 24-hour concentration at the site, due to vehicle sources, averaging all available years. |
| Column 9. | Upper estimate of the site annual average $PM_{10}$.(Based on a 'high exposure' year). |
| Column 10. | Nominal annual vehicle-attributable exposure category, where:- |

1 = 0 - 5 $\mu g\ m^{-3}$
2 = 5 - 10 $\mu g\ m^{-3}$
3 = 10 - 15 $\mu g\ m^{-3}$
4 = 15 - 20 $\mu g\ m^{-3}$
5 = 20 - 25 $\mu g\ m^{-3}$
6 = 25 - 30 $\mu g\ m^{-3}$
7 = 30 - 35 $\mu g\ m^{-3}$
7 = 30 - 35 $\mu g\ m^{-3}$
8 = 35 - 40 $\mu g\ m^{-3}$

(NB All figures are rounded to 1 $\mu g\ m^{-3}$ in each calculation)

*Appendix A2. DERIVED VEHICLE DATA - Continued*

*Table A2a. Peak 24-hour and annual average $PM_{10}$ (mg $m^{-3}$) assessed for sites where measurements are available, by derived Vehicle Effects: North Island.*

| City North Island | Site | Year | Ratio (due to vehicles) | Vehicle Peak | Vehicle Average | Lower Range | Site Average Vehicle | Upper Range | Exposure Category |
|---|---|---|---|---|---|---|---|---|---|
| **Auckland** | Queen St | 1999 | 0.9 | 46.0 | 24.0 | 22 | **24** | 26 | **5** |
| | Khyber Pass | 1998 | 0.8 | 97.1 | 24.0 | | | | |
| | Khyber Pass | 1999 | 0.8 | 49.8 | 21.9 | 22 | **23** | 24 | **5** |
| | Penrose | 1994 | 0.7 | 70.8 | 16.6 | | | | |
| | Penrose | 1995 | 0.7 | 54.1 | 17.4 | | | | |
| | Penrose | 1996 | 0.7 | 33.9 | 17.5 | | | | |
| | Penrose | 1997 | 0.7 | 46.1 | 17.7 | | | | |
| | Penrose | 1998 | 0.7 | 33.7 | 16.4 | | | | |
| | Penrose | 1999 | 0.7 | 56.4 | 17.2 | 16 | **17** | 18 | **4** |
| | Takapuna | 1996 | 0.5 | 23.2 | 6.7 | | | | |
| | Takapuna | 1996 | 0.5 | 11.5 | 8.2 | | | | |
| | Takapuna | 1997 | 0.5 | 25.0 | 9.2 | | | | |
| | Takapuna | 1997 | 0.5 | 25.3 | 9.6 | | | | |
| | Takapuna | 1998 | 0.5 | 28.8 | 9.0 | | | | |
| | Takapuna | 1998 | 0.5 | 19.1 | 7.7 | | | | |
| | Takapuna | 1999 | 0.5 | 23.6 | 8.3 | | | | |
| | Takapuna | 1999 | 0.5 | 19.5 | 7.5 | 7 | **8** | 10 | **2** |
| | Mt Eden | 1997 | 0.5 | 21.0 | 8.1 | | | | |
| | Mt Eden | 1998 | 0.5 | 19.9 | 10.3 | | | | |
| | Mt Eden | 1999 | 0.5 | 22.9 | 10.2 | 8 | **10** | 11 | **2** |
| | Henderson | 1998 | 0.5 | 16.6 | 8.9 | | | | |
| | Henderson | 1999 | 0.5 | 27.7 | 10.7 | 9 | **10** | 11 | **2** |
| **Huntly** | Ralph Rd | 1995 | 0.1 | 3.0 | 0.7 | | | | |
| | Ralph Rd | 1996 | 0.1 | 1.9 | 0.8 | | | | |
| | Ralph Rd | 1997 | 0.1 | 1.3 | 0.4 | | | | |
| | Ralph Rd | 1998 | 0.1 | 1.0 | 0.4 | | | | |
| | Ralph Rd | 1999 | 0.1 | 1.6 | 0.6 | 0 | **1** | 1 | **1** |
| | Croft Tce | 1995 | 0.3 | 14.5 | 3.8 | | | | |
| | Croft Tce | 1996 | 0.3 | 9.0 | 3.9 | 4 | **4** | 4 | **1** |
| **Hamilton** | Peachgrove | 1999 | 0.4 | 17.5 | 7.8 | 7 | **8** | 9 | **2** |
| **Rotorua** | Town | 1996 | 0.4 | 12.0 | 8.8 | 8 | **9** | 10 | **2** |
| **Hastings** | Heretaunga | 1998 | 0.4 | 14.8 | 10.2 | 9 | **10** | 11 | **3** |
| **Kawerau** | Mill | 1996 | 0.2 | 26.4 | 2.8 | 3 | **3** | 3 | **1** |
| **Wellington** | Civic | 1998 | 0.5 | 21.4 | 10.4 | 9 | **10** | 11 | **3** |
| | Avalon | 1998 | 0.5 | 14.7 | 7.2 | 6 | **7** | 8 | **2** |
| | Newtown | 1998 | 0.5 | 9.0 | 5.7 | 5 | **6** | 6 | **2** |
| **Lower Hutt** | Huia Pool | 1998 | 0.5 | 18.9 | 6.1 | 5 | **6** | 7 | **2** |

*Appendix A2. DERIVED VEHICLE DATA - Continued*

*Table A2b. Peak 24-hour and annual average $PM_{10}$ (mg $m^{-3}$) assessed for sites where measurements are available, by derived Vehicle Effects: South Island.*

| City South Island | Site | Year | Ratio (due to vehicles) | Vehicle Peak | Vehicle Average | Lower Range | Site Average Vehicle | Upper Range | Exposure Category |
|---|---|---|---|---|---|---|---|---|---|
| **Nelson** | City | 2001 | 0.4 | 26.0 | 10.0 | 8 | **10** | 12 | **2** |
| | Victory | 2001 | 0.4 | 14.5 | 9.6 | 8 | **10** | 12 | **2** |
| | Hospital | 2001 | 0.4 | 11.2 | 6.7 | 5 | **7** | 8 | **2** |
| **Christchurch** | St Albans | 1995 | 0.4 | 102.0 | 13.4 | | | | |
| | St Albans | 1996 | 0.4 | 88.4 | 12.8 | | | | |
| | St Albans | 1997 | 0.4 | 134.0 | 14.0 | | | | |
| | St Albans | 1998 | 0.4 | 72.4 | 9.5 | | | | |
| | St Albans | 1999 | 0.4 | 88.8 | 12.6 | 10 | **12** | 14 | **3** |
| | Beckenham | 1995 | 0.4 | 50.8 | 10.1 | | | | |
| | Beckenham | 1996 | 0.4 | 67.2 | 9.1 | | | | |
| | Beckenham | 1997 | 0.4 | 20.4 | 7.3 | 7 | **9** | 10 | **2** |
| | Opawa | 1995 | 0.4 | 54.8 | 12.4 | | | | |
| | Opawa | 1996 | 0.4 | 59.2 | 13.3 | | | | |
| | Opawa | 1997 | 0.4 | 94.8 | 13.9 | | | | |
| | Opawa | 1998 | 0.4 | 91.6 | 9.6 | 10 | **12** | 14 | **3** |
| | Hornby | 1995 | 0.4 | 44.8 | 12.0 | | | | |
| | Hornby | 1996 | 0.4 | 47.2 | 6.3 | | | | |
| | Hornby | 1997 | 0.4 | 50.4 | 13.5 | | | | |
| | Hornby | 1998 | 0.4 | 34.0 | 8.8 | 6 | **10** | 12 | **3** |
| **Rangiora** | Town | 1998 | 0.4 | 7.6 | 5.0 | | | | |
| | Town | 1999 | 0.4 | 32.8 | 8.2 | 5 | **7** | 8 | **2** |
| **Ashburton** | Town | 1997 | 0.4 | 18.8 | 9.6 | | | | |
| | Town | 1998 | 0.4 | 20.0 | 7.1 | | | | |
| | Town | 1999 | 0.4 | 38.4 | 9.9 | 7 | **9** | 10 | **2** |
| **Timaru** | Town | 1997 | 0.4 | 60.0 | 14.6 | | | | |
| | Town | 1998 | 0.4 | 40.8 | 10.7 | | | | |
| | Town | 1999 | 0.4 | 62.8 | 12.6 | 11 | **13** | 15 | **3** |
| **Dunedin** | City | 2000 | 0.4 | 19.2 | 8.4 | 7 | **8** | 10 | **2** |
| | N E Valley | 2000 | 0.4 | 22.8 | 7.2 | 6 | **7** | 9 | **2** |
| | Green Island | 2000 | 0.4 | 42.8 | 12.8 | 10 | **13** | 15 | **3** |
| **Alexandra** | Town | 2000 | 0.2 | 21.6 | 6.0 | 5 | **6** | 7 | **2** |
| **Mosgiel** | Town | 2000 | 0.2 | 14.0 | 5.2 | 4 | **5** | 6 | **2** |
| **Southland** | Dairy Co-op | 1998 | 0.1 | 4.6 | 1.0 | 1 | **1** | 1 | **1** |

## *Appendix B1. CALULATED FULL TOTAL EXPOSURE DATA*

The table below shows the full calculated data for each 'city area' in three size categories, for the total exposure.

| City | Population total | Population over 30 | Measured Total Average | Estimated Total Average | TOTAL | *Range* | *Range* | Exposure Category |
|---|---|---|---|---|---|---|---|---|
| | | | PM10 | PM10 | PM10 | *Lo* | *Hi* | |
| Auckland - Central | 368,508 | 199,813 | 29 | - | **29** | *26* | *32* | **6** |
| Auckland - Manukau | 266,061 | 130,366 | 24 | - | **24** | *22* | *26* | **5** |
| Auckland - North Shore | 181,260 | 102,835 | 17 | - | **17** | *15* | *19* | **4** |
| Wellington - City | 164,205 | 87,408 | - | 19 | **19** | *17* | *21* | **4** |
| Christchurch - Suburbs | 154,941 | 87,339 | 25 | - | **25** | *20* | *30* | **6** |
| Auckland - West | 151,872 | 78,559 | 20 | - | **20** | *18* | *22* | **5** |
| Christchurch - Central | 140,469 | 75,526 | 31 | - | **31** | *25* | *37* | **7** |
| Hamilton | 111,219 | 54,701 | 20 | - | **20** | *18* | *22* | **5** |
| Lower Hutt | 90,465 | 48,781 | 12 | - | **12** | *11* | *13* | **3** |
| Tauranga | 87,864 | 52,216 | - | 15 | **15** | *14* | *17* | **4** |
| Palmerston North | 65,526 | 32,602 | - | 9 | **9** | *8* | *10* | **2** |
| New Plymouth | 53,937 | 30,343 | - | 5 | **5** | *5* | *6* | **2** |
| Dunedin - City | 52,266 | 23,261 | 21 | - | **21** | *17* | *25* | **5** |
| Rotorua | 51,726 | 26,517 | 22 | - | **22** | *20* | *24* | **5** |
| Napier | 51,159 | 29,392 | - | 14 | **14** | *13* | *15* | **3** |
| Hastings | 48,870 | 26,415 | 24 | - | **24** | *22* | *26* | **5** |
| Whangarei | 42,000 | 22,777 | - | 10 | **10** | *9* | *11* | **3** |
| Invercargill | 41,502 | 22,810 | - | 16 | **16** | *13* | *19* | **4** |
| Wellington - Porirua | 41,283 | 20,333 | - | 15 | **15** | *14* | *17* | **4** |
| Wanganui | 37,389 | 21,184 | - | 6 | **6** | *5* | *7* | **2** |
| Auckland - Papakura | 35,112 | 17,622 | - | 19 | **19** | *17* | *21* | **4** |
| Nelson | 34,353 | 19,911 | 25 | - | **25** | *20* | *30* | **6** |
| Timaru | 32,319 | 19,475 | 32 | - | **32** | *26* | *38* | **7** |
| Rodney | 32,022 | 20,060 | - | 12 | **12** | *10* | *14* | **3** |
| Dunedin - South | 31,992 | 18,832 | 32 | - | **32** | *26* | *38* | **7** |
| Upper Hutt | 31,935 | 17,465 | - | 15 | **15** | *12* | *18* | **4** |
| Kapiti | 30,831 | 18,996 | - | 8 | **8** | *7* | *9* | **2** |
| Gisborne | 29,640 | 15,528 | - | 7 | **7** | *6* | *8* | **2** |
| Marlborough | 27,810 | 16,055 | - | 7 | **7** | *6* | *8* | **2** |
| Taupo | 23,319 | 12,452 | - | 7 | **7** | *6* | *8* | **2** |
| Waimakariri | 22,641 | 12,785 | - | 9 | **9** | *7* | *11* | **2** |
| Whakatane | 20,955 | 11,514 | - | 8 | **8** | *7* | *9* | **2** |
| Queenstown | 20,043 | 10,628 | - | 12 | **12** | *10* | *14* | **3** |
| Levin | 19,797 | 11,658 | - | 8 | **8** | *7* | *9* | **2** |
| South Waikato | 18,057 | 8,694 | - | 8 | **8** | *6* | *10* | **2** |
| Masterton | 17,646 | 10,045 | - | 9 | **9** | *8* | *10* | **2** |
| Ashburton | 15,084 | 8,800 | 21 | - | **21** | *17* | *25* | **5** |
| Fielding | 14,313 | 6,782 | - | 7 | **7** | *6* | *8* | **2** |
| Tasman | 13,998 | 8,531 | - | 9 | **9** | *7* | *11* | **2** |
| Hawera | 13,860 | 7,023 | - | 7 | **7** | *6* | *8* | **2** |
| Waipa | 13,224 | 7,498 | - | 7 | **7** | *6* | *8* | **2** |
| Pukekohe | 12,996 | 6,940 | - | 10 | **10** | *8* | *12* | **3** |
| Matamata | 12,462 | 7,339 | - | 8 | **8** | *6* | *10* | **2** |
| Waitaki | 12,033 | 7,585 | - | 7 | **7** | *6* | *8* | **2** |
| Southland | 10,710 | 4,181 | - | 7 | **7** | *6* | *8* | **2** |
| Mosgiel | 9,816 | 6,258 | 26 | - | **26** | *21* | *31* | **6** |
| Gore | 9,297 | 5,392 | - | 12 | **12** | *10* | *14* | **3** |
| Tararua | 9,090 | 5,096 | - | 7 | **7** | *6* | *8* | **2** |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Clutha | 8,808 | 3,543 | - | 8 | **8** | *6* | *10* | **2** |
| Rangitikei | 8,697 | 4,415 | - | 8 | **8** | *6* | *10* | **2** |
| Western Bay of Plenty | 8,430 | 5,571 | - | 8 | **8** | *6* | *10* | **2** |
| Hauraki | 8,178 | 4,653 | - | 8 | **8** | *6* | *10* | **2** |
| Thames | 8,157 | 5,236 | - | 8 | **8** | *6* | *10* | **2** |
| Central Otago | 7,971 | 4,413 | - | 14 | **14** | *11* | *17* | **3** |
| Greytown | 7,623 | 4,323 | - | 12 | **12** | *10* | *14* | **3** |
| Kawerau | 6,957 | 3,154 | - | 10 | **10** | *8* | *12* | **3** |
| Buller | 6,822 | 4,047 | - | 14 | **14** | *11* | *17* | **3** |
| Selwyn | 6,558 | 1,511 | - | 16 | **16** | *13* | *19* | **4** |
| Ruapehu | 6,252 | 2,768 | - | 10 | **10** | *8* | *12* | **3** |
| Leamington | 6,114 | 3,353 | - | 10 | **10** | *8* | *12* | **3** |
| Dunedin Outskirts | 5,592 | 3,241 | - | 16 | **16** | *13* | *19* | **4** |
| Waiuku | 5,352 | 2,854 | - | 12 | **12** | *10* | *14* | **3** |
| Stratford | 5,256 | 2,972 | - | 7 | **7** | *6* | *8* | **2** |

## *Appendix B2. CALULATED FULL VEHICLE EXPOSURE DATA*

The table below shows the full calculated data for each 'city area' in three size categories, for the exposure due to vehicle emissions.

| City | Population total | Population over 30 | Measured Total Average | Estimated Total Average | VEHICLE | *Range* | *Range* | Exposure Category |
|---|---|---|---|---|---|---|---|---|
| | | | **PM10** | **PM10** | **PM10** | ***Lo*** | ***Hi*** | |
| Auckland - Central | 368,508 | 199,813 | 23 | - | **23** | *21* | *25* | **5** |
| Auckland - Manukau | 266,061 | 130,366 | 17 | - | **17** | *15* | *19* | **4** |
| Auckland - North Shore | 181,260 | 102,835 | 8 | - | **8** | *7* | *9* | **2** |
| Wellington - City | 164,205 | 87,408 | - | 15 | **15** | *14* | *17* | **4** |
| Christchurch - Suburbs | 154,941 | 87,339 | 10 | - | **10** | *8* | *12* | **3** |
| Auckland - West | 151,872 | 78,559 | 10 | - | **10** | *9* | *11* | **3** |
| Christchurch - Central | 140,469 | 75,526 | 12 | - | **12** | *10* | *14* | **3** |
| Hamilton | 111,219 | 54,701 | 8 | - | **8** | *7* | *9* | **2** |
| Lower Hutt | 90,465 | 48,781 | 6 | - | **6** | *5* | *7* | **2** |
| Tauranga | 87,864 | 52,216 | - | 11 | **11** | *10* | *12* | **3** |
| Palmerston North | 65,526 | 32,602 | - | 5 | **5** | *5* | *6* | **2** |
| New Plymouth | 53,937 | 30,343 | - | 3 | **3** | *3* | *3* | **1** |
| Dunedin - City | 52,266 | 23,261 | 8 | - | **8** | *6* | *10* | **2** |
| Rotorua | 51,726 | 26,517 | 9 | - | **9** | *8* | *10* | **2** |
| Napier | 51,159 | 29,392 | - | 9 | **9** | *8* | *10* | **2** |
| Hastings | 48,870 | 26,415 | 10 | - | **10** | *9* | *11* | **3** |
| Whangarei | 42,000 | 22,777 | - | 6 | **6** | *5* | *7* | **2** |
| Invercargill | 41,502 | 22,810 | - | 12 | **12** | *10* | *14* | **3** |
| Wellington - Porirua | 41,283 | 20,333 | - | 10 | **10** | *9* | *11* | **3** |
| Wanganui | 37,389 | 21,184 | - | 4 | **4** | *4* | *4* | **1** |
| Auckland - Papakura | 35,112 | 17,622 | - | 15 | **15** | *14* | *17* | **4** |
| Nelson | 34,353 | 19,911 | 10 | - | **10** | *8* | *12* | **3** |
| Timaru | 32,319 | 19,475 | 13 | - | **13** | *10* | *16* | **3** |
| Rodney | 32,022 | 20,060 | - | 9 | **9** | *7* | *11* | **2** |
| Dunedin - South | 31,992 | 18,832 | 14 | - | **14** | *11* | *17* | **3** |
| Upper Hutt | 31,935 | 17,465 | - | 10 | **10** | *8* | *12* | **3** |
| Kapiti | 30,831 | 18,996 | - | 8 | **8** | *7* | *9* | **2** |
| Gisborne | 29,640 | 15,528 | - | 5 | **5** | *5* | *6* | **2** |
| Marlborough | 27,810 | 16,055 | - | 6 | **6** | *5* | *7* | **2** |
| Taupo | 23,319 | 12,452 | - | 4 | **4** | *4* | *4* | **1** |
| Waimakariri | 22,641 | 12,785 | - | 6 | **6** | *5* | *7* | **2** |
| Whakatane | 20,955 | 11,514 | - | 8 | **8** | *7* | *9* | **2** |
| Queenstown | 20,043 | 10,628 | - | 12 | **12** | *10* | *14* | **3** |
| Levin | 19,797 | 11,658 | - | 8 | **8** | *7* | *9* | **2** |
| South Waikato | 18,057 | 8,694 | - | 8 | **8** | *6* | *10* | **2** |
| Masterton | 17,646 | 10,045 | - | 9 | **9** | *8* | *10* | **2** |
| Ashburton | 15,084 | 8,800 | 9 | - | **9** | *7* | *11* | **2** |
| Fielding | 14,313 | 6,782 | - | 7 | **7** | *6* | *8* | **2** |
| Tasman | 13,998 | 8,531 | - | 9 | **9** | *7* | *11* | **2** |
| Hawera | 13,860 | 7,023 | - | 7 | **7** | *6* | *8* | **2** |
| Waipa | 13,224 | 7,498 | - | 7 | **7** | *6* | *8* | **2** |
| Pukekohe | 12,996 | 6,940 | - | 10 | **10** | *8* | *12* | **3** |
| Matamata | 12,462 | 7,339 | - | 5 | **5** | *4* | *6* | **2** |
| Waitaki | 12,033 | 7,585 | - | 4 | **4** | *3* | *5* | **1** |
| Southland | 10,710 | 4,181 | - | 4 | **4** | *3* | *5* | **1** |
| Mosgiel | 9,816 | 6,258 | 5 | - | **5** | *4* | *6* | **2** |
| Gore | 9,297 | 5,392 | - | 8 | **8** | *6* | *10* | **2** |
| Tararua | 9,090 | 5,096 | - | 7 | **7** | *6* | *8* | **2** |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Clutha | 8,808 | 3,543 | - | 5 | **5** | *4* | *6* | **2** |
| Rangitikei | 8,697 | 4,415 | - | 5 | **5** | *4* | *6* | **2** |
| Western Bay of Plenty | 8,430 | 5,571 | - | 4 | **4** | *3* | *5* | **1** |
| Hauraki | 8,178 | 4,653 | - | 4 | **4** | *3* | *5* | **1** |
| Thames | 8,157 | 5,236 | - | 5 | **5** | *4* | *6* | **2** |
| Central Otago | 7,971 | 4,413 | - | 6 | **6** | *5* | *7* | **2** |
| Greytown | 7,623 | 4,323 | - | 5 | **5** | *4* | *6* | **2** |
| Kawerau | 6,957 | 3,154 | - | 5 | **5** | *4* | *6* | **2** |
| Buller | 6,822 | 4,047 | - | 6 | **6** | *5* | *7* | **2** |
| Selwyn | 6,558 | 1,511 | - | 7 | **7** | *6* | *8* | **2** |
| Ruapehu | 6,252 | 2,768 | - | 4 | **4** | *3* | *5* | **1** |
| Leamington | 6,114 | 3,353 | - | 4 | **4** | *3* | *5* | **1** |
| Dunedin Outskirts | 5,592 | 3,241 | - | 10 | **10** | *8* | *12* | **3** |
| Waiuku | 5,352 | 2,854 | - | 7 | **7** | *6* | *8* | **2** |
| Stratford | 5,256 | 2,972 | - | 4 | **4** | *3* | *5* | **1** |

## *Appendix C. EXPOSURE NUMBERS BY CITY SIZE*

## FOR CITIES WITH OVER 100,000 PEOPLE

***Total Exposure***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 0 | 0 | 324 | 218 | 275 | 0 | 0 | 0 |
| **Best estimate** | **0** | **0** | **0** | **190** | **264** | **287** | **76** | **0** | **0** |
| High exposure year | 0 | 0 | 0 | 103 | 221 | 130 | 287 | 76 | 0 |

***Vehicle Related Exposure***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 236 | 175 | 206 | 200 | 0 | 0 | 0 | 0 |
| **Best estimate** | **0** | **158** | **79** | **381** | **200** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 158 | 79 | 305 | 76 | 200 | 0 | 0 | 0 |

## FOR CITIES WITH 30,000- 100,000 PEOPLE

***Total Exposure***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 126 | 211 | 41 | 73 | 38 | 0 | 0 | 0 |
| **Best estimate** | **0** | **103** | **121** | **130** | **50** | **46** | **38** | **0** | **0** |
| High exposure year | 0 | 71 | 104 | 139 | 40 | 76 | 20 | 38 | 0 |

***Vehicle Related Exposure***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 52 | 306 | 111 | 19 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate** | **52** | **222** | **126** | **89** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 30 | 144 | 183 | 111 | 19 | 0 | 0 | 0 | 0 |

## FOR CITIES WITH 10,000-30,000 PEOPLE

***Total Exposure***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 96 | 26 | 9 | 6 | 0 | 0 | 0 | 0 |
| **Best estimate** | **0** | **80** | **37** | **5** | **9** | **6** | **0** | **0** | **0** |
| High exposure year | 0 | 41 | 55 | 21 | 5 | 9 | 6 | 0 | 0 |

***Vehicle Related Exposure***

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 65 | 63 | 9 | 0 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate** | **31** | **87** | **19** | **0** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 104 | 24 | 9 | 0 | 0 | 0 | 0 | 0 |

## *Appendix D. EXPOSURE NUMBERS BY REGION*

***AUCKLAND*: Number of people (over 30) exposed (000s), by category, for TOTAL annual average $PM_{10}$ (mg $m^{-3}$). 557,000.**

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 7 | 20 | 199 | 130 | 200 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **0** | **27** | **120** | **209** | **200** | **0** | **0** | **0** |
| High exposure year | 0 | 0 | 7 | 123 | 96 | 130 | 200 | 0 | 0 |

***AUCKLAND*: Number of people (over 30) exposed (000s), by category, for VEHICLE RELATED annual average $PM_{10}$ (mg $m^{-3}$). 557,000.**

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 208 | 18 | 131 | 200 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **123** | **86** | **148** | **200** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 103 | 106 | 148 | 0 | 200 | 0 | 0 | 0 |

Auckland includes Auckland City, Manukau, North Shore, Waitakere, Papakura, Rodney and Pukekohe.

***WELLINGTON*: Number of people (over 30) exposed (000s), by category, for TOTAL annual average $PM_{10}$ (mg $m^{-3}$). 174,000.**

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 0 | 87 | 87 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **0** | **49** | **125** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 0 | 49 | 38 | 87 | 0 | 0 | 0 | 0 |

***WELLINGTON*: Number of people (over 30) exposed (000s), by category, for VEHICLE RELATED annual average $PM_{10}$ (mg $m^{-3}$). 174,000.**

| **Exposure** | **0-5** | **>5-10** | **>10-15** | **>15-20** | **>20-25** | **>25-30** | **>30-35** | **>35-40** | **>40** |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 86 | 88 | 0 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **49** | **38** | **87** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 49 | 38 | 87 | 0 | 0 | 0 | 0 | 0 |

Wellington includes Wellington City, Porirua, Lower Hutt and Upper Hutt.

***CHRISTCHURCH**: Number of people (over 30) exposed (000s), by category, for TOTAL annual average $PM_{10}$ (mg $m^{-3}$). 163,000.*

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 0 | 0 | 0 | 0 | 87 | 76 | 0 | 0 |
| **Best estimate - average year** | **0** | **0** | **0** | **0** | **0** | **87** | **76** | **0** | **0** |
| High exposure year | 0 | 0 | 0 | 0 | 0 | 87 | 76 | 0 | 0 |

***CHRISTCHURCH**: Number of people (over 30) exposed (000s), by category, for VEHICLE RELATED annual average $PM_{10}$ (mg $m^{-3}$). 163,000.*

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 87 | 76 | 0 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **0** | **163** | **0** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 0 | 87 | 76 | 0 | 0 | 0 | 0 | 0 |

Christchurch includes the greater Christchurch area.

***DUNEDIN**: Number of people (over 30) exposed (000s), by category, for TOTAL annual average $PM_{10}$ (mg $m^{-3}$). 46,000.*

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 0 | 3 | 23 | 0 | 19 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **0** | **0** | **3** | **0** | **23** | **19** | **0** | **0** |
| High exposure year | 0 | 0 | 0 | 0 | 3 | 23 | 0 | 19 | 0 |

***DUNEDIN**: Number of people (over 30) exposed (000s), by category, for VEHICLE RELATED annual average $PM_{10}$ (mg $m^{-3}$). 46,000.*

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 27 | 19 | 0 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **24** | **22** | **0** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 0 | 27 | 19 | 0 | 0 | 0 | 0 | 0 |

Dunedin includes the greater Dunedin area (excluding Mosgiel).

***REST OF NORTH ISLAND*: Number of people (over 30) exposed (000s), by category, for TOTAL annual average $PM_{10}$ (mg $m^{-3}$). 457,000.**

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 262 | 89 | 55 | 53 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **230** | **69** | **52** | **108** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 151 | 111 | 89 | 55 | 53 | 0 | 0 | 0 |

***REST OF NORTH ISLAND*: Number of people (over 30) exposed (000s), by category, for VEHICLE RELATED annual average $PM_{10}$ (mg $m^{-3}$). 457,000.**

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 108 | 298 | 51 | 0 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **82** | **296** | **79** | **0** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 30 | 274 | 153 | 0 | 0 | 0 | 0 | 0 | 0 |

Rest of North Island includes Hamilton, Tauranga, Palmerston North, New Plymouth, Rotorua, Napier, Hastings, Whangarei, Wanganui, Kapiti, Gisborne, Taupo, Whakatane, Levin, South Waikato, Masterton, Fielding, Hawera, Waipa, Matamata, Tararua, Rangitikei, Western Bay of Plenty, Hauraki, Thames, Greytown, Kawerau, Ruapehu, Leamington, Waiuku, Stratford.

***REST OF SOUTH ISLAND*: Number of people (over 30) exposed (000s), by category, for TOTAL annual average $PM_{10}$ (mg $m^{-3}$). 156,000.**

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 0 | 52 | 49 | 9 | 26 | 20 | 0 | 0 | 0 |
| **Best estimate - average year** | **0** | **52** | **25** | **24** | **9** | **26** | **20** | **0** | **0** |
| High exposure year | 0 | 27 | 25 | 25 | 24 | 9 | 26 | 20 | 0 |

***REST OF SOUTH ISLAND*: Number of people (over 30) exposed (000s), by category, for VEHICLE RELATED annual average $PM_{10}$ (mg $m^{-3}$). 156,000.**

| Exposure | 0-5 | >5-10 | >10-15 | >15-20 | >20-25 | >25-30 | >30-35 | >35-40 | >40 |
|---|---|---|---|---|---|---|---|---|---|
| **Category** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** |
| Low exposure year | 22 | 81 | 53 | 0 | 0 | 0 | 0 | 0 | 0 |
| **Best estimate - average year** | **12** | **71** | **73** | **0** | **0** | **0** | **0** | **0** | **0** |
| High exposure year | 0 | 60 | 43 | 53 | 0 | 0 | 0 | 0 | 0 |

Rest of South Island includes Invercargill, Nelson, Timaru, Marlborough, Waimakariri, Queenstown, Ashburton, Tasman, Waitaki, Southland, Mosgiel, Gore, Clutha, Central Otago, Buller, Selwyn.

## *Appendix E. MORTALITY WITH DIFFERENT ASSUMPTIONS*

*(NB Calculation of total $PM_{10}$ at 0 mg $m^{-3}$ threshold is not valid - see text. "Cl" = Confidence limit).*

***Best estimates of deaths (over 30) per annum due to $PM_{10}$ emissions, in all New Zealand cities with more than 5000 people***

| **$PM_{10}$ emissions** | **Threshold $PM_{10}$ for mortality effect** | | | |
|---|---|---|---|---|
| | **0 mg $m^{-3}$** | **5 mg $m^{-3}$** | **7.5 mg $m^{-3}$** | **10 mg $m^{-3}$** |
| Total $PM_{10}$ | NA | 1160 | 970 | 812 |
| Vehicle related $PM_{10}$ | 953 | 583 | 399 | 285 |

**Summary of all scenarios**

**Estimated attributable number of deaths for all New Zealand cities with more than 5000 people**

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 0 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | NA | NA | NA | 852 | 515 | 1208 |
| **Best estimate** | **NA** | **NA** | **NA** | **953** | **576** | **1351** |
| High exposure year | NA | NA | NA | 1093 | 661 | 1550 |

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 1012 | 612 | 1435 | 485 | 293 | 688 |
| **Best estimate** | **1160** | **702** | **1646** | **583** | **353** | **828** |
| High exposure year | 1401 | 847 | 1988 | 718 | 434 | 1018 |

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 7.5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 820 | 496 | 1163 | 304 | 184 | 431 |
| **Best estimate** | **970** | **586** | **1376** | **399** | **241** | **566** |
| High exposure year | 1213 | 734 | 1721 | 527 | 319 | 747 |

| | ***Due to TOTAL PM10*** | | | ***due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 10 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 666 | 402 | 944 | 223 | 135 | 317 |
| **Best estimate** | **812** | **491** | **1152** | **285** | **173** | **405** |
| High exposure year | 1045 | 632 | 1482 | 396 | 239 | 561 |

***Appendix E. MORTALITY WITH DIFFERENT ASSUMPTIONS - Ctd***

*(NB Calculation of total $PM_{10}$ at 0 mg $m^{-3}$ threshold is not valid - see text. "Cl" = Confidence limit).*

***Best estimates of deaths (over 30) per annum due to $PM_{10}$ emissions, in Auckland***

| **$PM_{10}$ emissions** | **Threshold $PM_{10}$ for mortality effect** | | | |
|---|---|---|---|---|
| | **0 mg $m^{-3}$** | **5 mg $m^{-3}$** | **7.5 mg $m^{-3}$** | **10 mg $m^{-3}$** |
| Total $PM_{10}$ | NA | 503 | 436 | 368 |
| Vehicle related $PM_{10}$ | 454 | 321 | 253 | 199 |

**Summary of all scenarios**

**Estimated attributable number of deaths for Auckland**

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 0 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | NA | NA | NA | 429 | 259 | 608 |
| **Best estimate** | **NA** | **NA** | **NA** | **454** | **275** | **644** |
| High exposure year | NA | NA | NA | 509 | 308 | 722 |

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 481 | 291 | 682 | 295 | 179 | 419 |
| **Best estimate** | **503** | **304** | **713** | **321** | **194** | **456** |
| High exposure year | 596 | 360 | 846 | 377 | 228 | 535 |

| | ***due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 7.5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 414 | 250 | 587 | 227 | 137 | 321 |
| **Best estimate** | **436** | **264** | **619** | **253** | **153** | **359** |
| High exposure year | 531 | 321 | 753 | 309 | 187 | 439 |

| | ***due to TOTAL PM10*** | | | ***due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 10 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 347 | 210 | 492 | 183 | 111 | 260 |
| **Best estimate** | **368** | **223** | **523** | **199** | **120** | **282** |
| High exposure year | 464 | 280 | 658 | 254 | 153 | 360 |

***Appendix E. MORTALITY WITH DIFFERENT ASSUMPTIONS - Ctd***

*(NB Calculation of total $PM_{10}$ at 0 mg $m^{-3}$ threshold is not valid - see text. "Cl" = Confidence limit).*

***Best estimates of deaths (over 30) per annum due to $PM_{10}$ emissions, in Wellington***

| **$PM_{10}$ emissions** | **Threshold $PM_{10}$ for mortality effect** | | | |
|---|---|---|---|---|
| | **0 mg $m^{-3}$** | **5 mg $m^{-3}$** | **7.5 mg $m^{-3}$** | **10 mg $m^{-3}$** |
| Total $PM_{10}$ | NA | 101 | 79 | 57 |
| Vehicle related $PM_{10}$ | 121 | 78 | 56 | 40 |

**Summary of all scenarios**

**Estimated attributable number of deaths for Wellington**

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 0 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | NA | NA | NA | 90 | 54 | 127 |
| **Best estimate** | **NA** | **NA** | **NA** | **121** | **73** | **172** |
| High exposure year | NA | NA | NA | 121 | 73 | 172 |

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 91 | 55 | 129 | 46 | 28 | 65 |
| **Best estimate** | **101** | **61** | **143** | **78** | **47** | **111** |
| High exposure year | 124 | 75 | 176 | 78 | 47 | 111 |

| | ***due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 7.5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 69 | 42 | 98 | 23 | 14 | 33 |
| **Best estimate** | **79** | **48** | **112** | **56** | **34** | **80** |
| High exposure year | 102 | 62 | 145 | 56 | 34 | 80 |

| | ***due to TOTAL PM10*** | | | ***due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 10 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 47 | 28 | 66 | 12 | 7 | 17 |
| **Best estimate** | **57** | **34** | **80** | **40** | **24** | **57** |
| High exposure year | 80 | 48 | 113 | 40 | 24 | 57 |

## *Appendix E. MORTALITY WITH DIFFERENT ASSUMPTIONS - Ctd*

*(NB Calculation of total $PM_{10}$ at 0 mg $m^{-3}$ threshold is not valid - see text. "Cl" = Confidence limit).*

***Best estimates of deaths (over 30) per annum due to $PM_{10}$ emissions, in Christchurch***

| **$PM_{10}$ emissions** | **Threshold $PM_{10}$ for mortality effect** | | | |
|---|---|---|---|---|
| | **0 mg $m^{-3}$** | **5 mg $m^{-3}$** | **7.5 mg $m^{-3}$** | **10 mg $m^{-3}$** |
| Total $PM_{10}$ | NA | 201 | 182 | 163 |
| Vehicle related $PM_{10}$ | 99 | 61 | 41 | 21 |

**Summary of all scenarios**

**Estimated attributable number of deaths for Christchurch**

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 0 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | NA | NA | NA | 78 | 47 | 111 |
| **Best estimate** | **NA** | **NA** | **NA** | **99** | **60** | **141** |
| High exposure year | NA | NA | NA | 118 | 71 | 167 |

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 201 | 121 | 285 | 39 | 24 | 55 |
| **Best estimate** | **201** | **121** | **285** | **61** | **37** | **86** |
| High exposure year | 201 | 121 | 285 | 79 | 48 | 113 |

| | ***due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 7.5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 182 | 110 | 259 | 19 | 12 | 27 |
| **Best estimate** | **182** | **110** | **259** | **41** | **25** | **58** |
| High exposure year | 182 | 110 | 259 | 60 | 36 | 85 |

| | ***due to TOTAL PM10*** | | | ***due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 10 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 163 | 99 | 232 | 10 | 6 | 14 |
| **Best estimate** | **163** | **99** | **232** | **21** | **12** | **29** |
| High exposure year | 163 | 99 | 232 | 40 | 24 | 56 |

***Appendix E. MORTALITY WITH DIFFERENT ASSUMPTIONS - Ctd***

*(NB Calculation of total $PM_{10}$ at 0 mg $m^{-3}$ threshold is not valid - see text. "Cl" = Confidence limit).*

***Best estimates of deaths (over 30) per annum due to $PM_{10}$ emissions, in Dunedin***

| **$PM_{10}$ emissions** | **Threshold $PM_{10}$ for mortality effect** | | | |
|---|---|---|---|---|
| | **0 mg $m^{-3}$** | **5 mg $m^{-3}$** | **7.5 mg $m^{-3}$** | **10 mg $m^{-3}$** |
| Total $PM_{10}$ | NA | 54 | 48 | 43 |
| Vehicle related $PM_{10}$ | 22 | 11 | 6 | 3 |

**Summary of all scenarios**

**Estimated attributable number of deaths for Dunedin**

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 0 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | NA | NA | NA | 21 | 13 | 30 |
| **Best estimate** | **NA** | **NA** | **NA** | **22** | **13** | **31** |
| High exposure year | NA | NA | NA | 33 | 20 | 46 |

| | ***Due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 37 | 22 | 52 | 10 | 6 | 15 |
| **Best estimate** | **54** | **32** | **76** | **11** | **7** | **16** |
| High exposure year | 59 | 36 | 84 | 22 | 13 | 31 |

| | ***due to TOTAL PM10*** | | | ***Due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 7.5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 31 | 19 | 44 | 5 | 3 | 7 |
| **Best estimate** | **48** | **29** | **69** | **6** | **3** | **8** |
| High exposure year | 54 | 33 | 77 | 16 | 10 | 23 |

| | ***due to TOTAL PM10*** | | | ***due to VEHICLE RELATED PM10*** | | |
|---|---|---|---|---|---|---|
| Threshold = 10 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 26 | 16 | 37 | 2 | 1 | 3 |
| **Best estimate** | **43** | **26** | **61** | **3** | **2** | **4** |
| High exposure year | 49 | 30 | 69 | 11 | 6 | 15 |

***Appendix E. MORTALITY WITH DIFFERENT ASSUMPTIONS - Ctd***

*(NB Calculation of total $PM_{10}$ at 0 mg $m^{-3}$ threshold is not valid - see text. "Cl" = Confidence limit).*

***Best estimates of deaths (over 30) per annum due to $PM_{10}$ emissions, in the rest of North Island***

| $PM_{10}$ emissions | Threshold $PM_{10}$ for mortality effect | | | |
|---|---|---|---|---|
| | **0 mg $m^{-3}$** | **5 mg $m^{-3}$** | **7.5 mg $m^{-3}$** | **10 mg $m^{-3}$** |
| Total $PM_{10}$ | NA | 193 | 133 | 104 |
| Vehicle related $PM_{10}$ | 177 | 71 | 21 | 11 |

**Summary of all scenarios**

**Estimated attributable number of deaths for the rest of North Island**

| | *Due to TOTAL PM10* | | | *Due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 0 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | NA | NA | NA | 163 | 99 | 232 |
| **Best estimate** | **NA** | **NA** | **NA** | **177** | **107** | **252** |
| High exposure year | NA | NA | NA | 210 | 127 | 298 |

| | *Due to TOTAL PM10* | | | *Due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 156 | 94 | 221 | 60 | 36 | 85 |
| **Best estimate** | **193** | **117** | **273** | **71** | **43** | **100** |
| High exposure year | 238 | 144 | 337 | 97 | 59 | 138 |

| | *due to TOTAL PM10* | | | *Due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 7.5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 96 | 58 | 136 | 14 | 8 | 19 |
| **Best estimate** | **133** | **81** | **189** | **21** | **13** | **30** |
| High exposure year | 179 | 108 | 253 | 41 | 25 | 58 |

| | *due to TOTAL PM10* | | | *due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 10 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 70 | 43 | 100 | 7 | 4 | 10 |
| **Best estimate** | **104** | **63** | **147** | **11** | **6** | **15** |
| High exposure year | 139 | 84 | 197 | 21 | 13 | 29 |

***Appendix E. MORTALITY WITH DIFFERENT ASSUMPTIONS - Ctd***

*(NB Calculation of total $PM_{10}$ at 0 mg $m^{-3}$ threshold is not valid - see text. "Cl" = Confidence limit).*

***Best estimates of deaths (over 30) per annum due to $PM_{10}$ emissions, in the rest of South Island***

| $PM_{10}$ emissions | Threshold $PM_{10}$ for mortality effect | | | |
|---|---|---|---|---|
| | **0 mg $m^{-3}$** | **5 mg $m^{-3}$** | **7.5 mg $m^{-3}$** | **10 mg $m^{-3}$** |
| Total $PM_{10}$ | NA | 100 | 80 | 67 |
| Vehicle related $PM_{10}$ | 75 | 38 | 19 | 10 |

**Summary of all scenarios**

**Estimated attributable number of deaths for the rest of South Island**

| | *Due to TOTAL PM10* | | | *Due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 0 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | NA | NA | NA | 68 | 41 | 96 |
| **Best estimate** | **NA** | **NA** | **NA** | **75** | **46** | **107** |
| High exposure year | NA | NA | NA | 98 | 59 | 139 |

| | *Due to TOTAL PM10* | | | *Due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 79 | 48 | 112 | 31 | 19 | 44 |
| **Best estimate** | **100** | **60** | **141** | **38** | **23** | **54** |
| High exposure year | 133 | 81 | 189 | 59 | 36 | 84 |

| | *due to TOTAL PM10* | | | *Due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 7.5 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 59 | 36 | 84 | 14 | 8 | 20 |
| **Best estimate** | **80** | **48** | **114** | **19** | **12** | **27** |
| High exposure year | 114 | 69 | 162 | 39 | 24 | 56 |

| | *due to TOTAL PM10* | | | *due to VEHICLE RELATED PM10* | | |
|---|---|---|---|---|---|---|
| Threshold = 10 µg $m^{-3}$ | ***Estimate*** | ***95% CI*** | | ***Estimate*** | ***95% CI*** | |
| Low exposure year | 46 | 28 | 65 | 7 | 4 | 10 |
| **Best estimate** | **67** | **41** | **95** | **10** | **6** | **14** |
| High exposure year | 98 | 59 | 139 | 27 | 16 | 38 |