



Ministry for the
Environment
Manatū Mō Te Taiao

Health effects of PM₁₀ in New Zealand

Prepared by Environet Limited for the
Ministry for the Environment

August 2003

Air Quality Technical Report No. 39

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Foreword

Thanks to growing council air quality monitoring programmes we now have a good picture of PM₁₀ concentrations in many New Zealand towns and cities. Unfortunately this monitoring has found that PM₁₀ concentrations frequently breach the Ministry for the Environment's ambient air quality guideline values of 50 ug/m³ (24 hour average) and 20 ug/m³ (annual average). Such concentrations are known to adversely affect people's health and wellbeing.

This technical report on the **health effects of PM₁₀ in New Zealand** (particles less than 10 microns in diameter) describes the potential health effects caused by inhaling PM₁₀ in New Zealand towns and cities. Health effects range from minor effects, such as nose and throat irritation, to more serious effects such as aggravation of existing respiratory and cardiovascular disease, increased hospital admissions and school absences, and premature death.

The report will assist with the development of national environmental standards for air quality under the Resource Management Act 1991. Knowledge about health effects will help to determine appropriate standards and to estimate the benefits of achieving them.

This report is released for information only. It forms part of the section 32 analysis required for standards development. Four other background reports on particles are available covering: emission inventory results, amenity issues and monitoring results.

Thank you to those councils and others who assisted with the preparation of this report.



Barry Carbon
Chief Executive

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Executive Summary

This technical report comprises a review of the health impacts of particles less than 10 microns in diameter (PM₁₀) although the implications of other size fractions and indicators is considered. The health impacts associated with concentrations of suspended particles include effects such as coughs, asthma symptoms, bronchitis, respiratory illness and mortality.

The science underpinning our understanding of the health impacts of particles is epidemiology. These observational studies on the relationship between concentrations of particles and health effects have been conducted in numerous locations throughout the world. Results show increases in mortality and other health effects are associated with increases in 24-hour average PM₁₀ concentrations. The consistency and coherency of the studies have lead researchers to conclude that the effect is causal. The relationship appears to be linear and it appears that there is no threshold below which effects do not occur.

The impact of PM₁₀ concentrations in areas of New Zealand on the health of the residents has been estimated in a number of different studies. The most extensive study was carried out by Fisher et al (2002) and estimates the number of deaths associated with PM₁₀ concentrations from all sources and from motor vehicles based on relationships described in a study by Kunzli et al (2000). Results are reported for the four major cities (Auckland, Christchurch, Wellington and Dunedin) with other areas being collated for both the North and South Island. The estimated annual mortality rates were: 440 for Auckland, 180 for Christchurch, 80 for Wellington, 50 for Dunedin, 40 for Hamilton and 20 for Nelson (see Table 6.1 on page 16 for further data).

Risk assessments of the impact of PM₁₀ concentrations in Christchurch and Nelson have also been carried out based on the results of studies of the health impacts of PM₁₀ in Christchurch (Hales et al, 1999; McGowan et al, 2002). These include mortality estimates as well as hospitalisations and restricted activity days. The mortality estimates for the latter risk assessments underestimate mortality by around 4–5 times compared to Fisher et al, (2002). This is attributed to the time-series methodology of the Hales (1999) study, which associates only those deaths that occur a relatively short time after the pollution episode to PM₁₀ concentrations. Thus they are limited to a selection of the acute impacts but do not estimate the reduced life expectancy due to long-term morbidity enhanced by air pollution.

This report estimates hospitalisations and restricted activity days for other areas within New Zealand based on relationships from McGowan (2002) for Christchurch and overseas relationships for restricted activity days (RADs). Results indicate a range in annual average hospitalisations per year in the larger cities, from around 25 in Dunedin to 200 in Auckland. Estimates of RADs in New Zealand cities range from around 90,000 per year in Dunedin to around 750,000 in Auckland. Table 6.1 on page 16 has further details.

1 Introduction

Concentrations of particles in urban areas of New Zealand pose a major air quality issue. Regional monitoring programmes have increased and become more extensive during the 1990s and show that PM₁₀ concentrations breach the air quality guideline values in many urban areas. The impacts of these concentrations are significant (Fisher et al, 2002).

This technical report comprises a review of the health implications of PM₁₀ concentrations in New Zealand and provides an overview of the existing knowledge on the health impacts of particle pollution.

Particles in the air can exist across a range of different sizes and chemical compositions. In New Zealand, historical monitoring of particles was based on measurements of total suspended particulate (TSP), which by definition includes all particles suspended in the air, although measurement methods have tended to capture those less than 20 microns in diameter. During the 1990s size selective inlets and monitoring methods were introduced to capture the PM₁₀ size fraction, encompassing particles in the air less than 10 microns in diameter. An alternative methodology also used in some locations was smoke monitoring. However, this method was disregarded in most areas by the late 1990s as it is biased towards the measurement of elemental carbon. More recently, some monitoring of particles less than 2.5 microns (PM_{2.5}) has been carried out in some locations.

The focus of much of the particle monitoring carried out in New Zealand has therefore been the PM₁₀ size fraction. Similarly, the majority of the particle health effects studies both in New Zealand and overseas have also been based on measurements of PM₁₀. The PM₁₀ size fraction includes particles referred to as coarse and fine particles. These classifications were originally intended to separate particles based on the nature of their source, with coarse particles representing those formed through abrasive type mechanisms and fine particles those formed through combustion processes and chemical reactions. However, the terms are more commonly used now to represent particles in the size ranges less than 2.5 microns (fine) and between 2.5 and 10 microns (coarse). Fine-mode particles are formed through nucleation¹ and grow by coagulation² and the condensation of other gases on the particle nuclei. Coarse particles also include many natural sources such as pollens, spores and sea spray.

From a health perspective, the size of the particles is important as it affects their ability to penetrate into the lungs and cause adverse health effects. The larger particles (e.g. those greater than 10 microns) tend to settle in the nose and mouth and are unlikely to pose a health risk. Finer particles can penetrate further into the lung and alveoli and therefore may be associated with more severe health impacts. The mechanisms of formation (e.g. nucleation, coagulation and condensation for the finer fraction or the more abrasive formation systems of the coarser fraction) may also have implications for respiratory related health effects.

¹ Molecules of complementary substances combine to form a condensation nucleus.

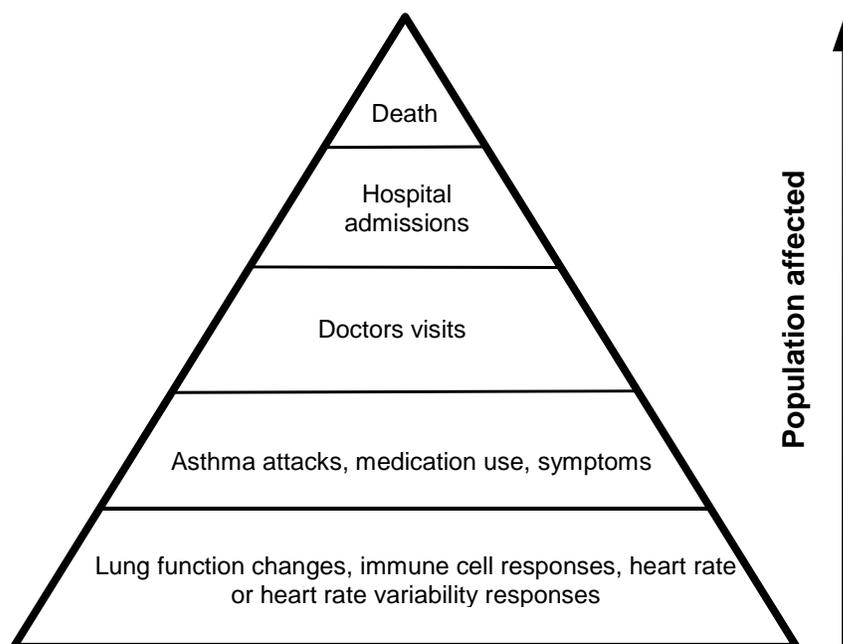
² The combination of existing particles.

2 Health Impacts of Particles

Our understanding of the health impacts of particles in the air is based largely on the results of epidemiological studies carried out in numerous locations throughout the world. Historically, much of these data came from studies carried out in the United States. However, in more recent years health effects studies have also been carried out in other countries. These studies range from extensive research in areas such as Europe to single city time-series studies in smaller urban areas such as Christchurch.

The health effects associated with particles in the air range from minor nose and throat irritations to more severe impacts such as hospital admissions and premature mortality. The population base susceptible to the different impacts varies by effect (Figure 2.1) with a smaller subsection comprising of the elderly and those with pre-existing conditions most at risk of impacts such as premature mortality.

Figure 2.1: Pyramid of the impacts of particle pollution



There are limitations associated with using epidemiological studies to determine health impacts, particularly in understanding the extent of impact in assessing causality and in determining biological mechanisms. The most common methodology for assessing the relationships has been the standard time series epidemiological studies. Historically these have shown increases in mortality of around 0–1% per $10 \mu\text{g m}^{-3}$ increase in the 24-hour average PM_{10} concentration. However, recent reanalysis of the National Morbidity, Mortality and Air Pollution Study (NMAPS) suggests that the size of the effect has been overestimated in a number of the US studies owing to the use of the default convergence criteria in the S-Plus, which were inadequate to produce optimal estimates and could result in an upward bias (Samet et al, 2000). The statistical technique is associated with the general additive model (GAM) method of estimating an effect of one or more variables on an outcome. Many other studies, including those carried out in Australia, have used the GAMs models but have not encountered the same issues as those

observed in the US studies (NEPC, 2002). The implications for New Zealand studies are minimal, as risk assessments have typically been based on studies such as Hales et al (1999) and Kunzli et al (2000), which incorporated different software and methodologies respectively.

Although the most common method of study, the time series studies provide only an indication of the acute health impacts that are documented immediately after exposure. This is because they are based on the temporal relationship between pollution episodes and the recording of health endpoints. An alternative study methodology, which is not subject to these limitations or potential underestimates associated with the GAMs models, is the prospective cohort design used in two US studies, the Harvard Six Cities study (Dockery et al, 1993) and the American Cancer Society study (Pope et al, 1995). These studies followed groups of 8111 and 552,138 adult subjects for 14–16 and seven years respectively. Higher concentrations of particles were found to be associated with increased mortality in both studies. In response to criticisms of the health impacts assessments, these studies were thoroughly reviewed for the United States Health Effects Institute (Krewski, 2000). The review was in support of the original findings.

Kunzli et al (2000) used the results from these studies to estimate the impact of PM₁₀ concentrations on mortality in Switzerland, France and Austria. This assessment was based on an increase of 4.3% in mortality per 10 µgm⁻³ increase in annual average PM₁₀ concentrations assuming a no effects threshold of 7.5 µgm⁻³. The basis was primarily the Dockery et al (1993) study which indicated an increase in mortality of around 26% from the least polluted to the most polluted city, giving a 4.3% increase per 10 µgm⁻³ increase in annual average PM₁₀. A higher ratio of 8.3% was found in for PM_{2.5}. Similar results were found in the Pope et al (1995) study, which indicated a 6.9% increase in mortality per 10 µgm⁻³ increase in annual average PM_{2.5}. The dose-response estimates used of 4.3% are conservative relative to the WHO (2002) recommendations, which indicate a 10% increase in mortality per 10 µgm⁻³ increase in PM₁₀ for the impacts of long-term exposure.

Although a significant study methodology, cohort studies of the impact of particle pollution are limited. In addition to the Dockery et al (1993) and Pope et al (1995) studies, a study was conducted by Abbey et al (1999) and more recently a cohort design study was carried out outside of the USA, in the Netherlands. This latter study examined the relationship between cardiopulmonary mortality and living near to a major road (Hoek et al, 2002). Results indicated increased relative risk of cardiopulmonary mortality and all cause mortality for those living near a major road but no increase risk for non-cardiopulmonary and non-lung cancer deaths.

Another recent publication of significance demonstrates the improvement in health impacts associated with regulations prohibiting the burning of coal in Dublin (Clancy et al, 2002). In the years following the ban, black smoke concentrations declined by 35.6 µgm⁻³, a 70% reduction, and there were about 116 fewer respiratory deaths and 243 fewer cardiovascular deaths. These equated to reductions of around 4% in respiratory mortality and around 3% reduction in cardiovascular mortality per 10 µgm⁻³ reduction in 24-hour average black smoke concentrations.

2.1 Current issues

Historically, one of the main concerns about the epidemiological studies has been establishing the causality of the relationship between particle pollution and the health impacts. That is, whether or not it was the particles themselves or some other factor that resulted in the observed relationship. Many researchers, policy makers and health experts in recent years have examined the case for and against causality. Generally the assessment is based on the weight of evidence approach and the causality of the association is considered using a set of established criteria, such as those proposed by Bradford Hill (1971). Of particular significance is a review by Dab et al (2001) which evaluated 15 assessments of causality conducted by leading researchers and concluded that the observed relationships were both valid and causal. Also significant in terms of causality are studies such as Clancy et al (2002) and Pope et al (1992) which demonstrate improvements in health indicators associated with reductions in particle concentrations.

This conclusion appears to be generally accepted and the focus of the PM₁₀ health endpoint relationship is now on better characterising relationships and establishing biological mechanisms.

2.2 Biological mechanisms

The biological mechanisms responsible for the health impacts associated with particle concentrations have been considered in detail in recent years. A number of mechanisms have been demonstrated for the respiratory related impacts that focus on mechanisms of inflammation, tissue damage and repair (Brunekreef and Holgate, 2002). However, less is known about the cardiovascular impacts. Brunekreef and Holgate identify a number of factors such as blood clotting, increased fibrinogen and platelets and sequestration of red blood cells in the lung mass that appear to contribute but conclude that their significance remains to be established. Peters and Pope (2002) suggest that evidence points towards particle-induced pulmonary and systemic inflammation, accelerated atherosclerosis and altered cardiac autonomic function as possible pathways linking particle pollution and cardiovascular mortality.

Most toxicological studies investigating biological mechanisms have used either experimental animals or isolated cell systems and have generally been used to test hypothesis generated through epidemiological studies. Toxicity studies place emphasis on the composition of the particles. A number of mechanisms are proposed for different compositions, including a strong focus on transition metals (EPAQS, 2000). However, impacts are demonstrated for highly variable compositions, including pollen and fungal spore derived particles and non-transition metal particles. One of the complicating factors when considering toxicology is that it is unlikely that any one toxic fraction will be the cause of all types of impacts.

In explaining the biological mechanisms, present thinking focuses on there being something associated with the particle surface, most likely adsorbed transition metals but also possibly some other physical or chemical property that is able to initiate oxidative stress when it comes in contact with lung cells. This results in inflammation, which may have different consequences depending on individual susceptibility (EPAQS, 2000).

2.3 Issues of mortality advancement

The extent to which the associations between PM₁₀ concentrations and mortality result in the advancement of death is not well addressed by most study designs. However, it is an important point in establishing the real life impact of the particle and mortality relationships.

The best information on the extent of mortality advancement stems from the cohort study designs (e.g. Dockery et al, 1993; Pope et al, 1995). These studies indicate life shortening by 1–2 years and that these effects may also depend on factors such as education and antioxidant vitamin status indicating a greater reduction in life expectancy for disadvantaged population groups (Brunekreef and Holgate, 2002).

The issue of life shortening is regularly raised in economic assessments, which attempt to put a cost on the value of the life lost. In that capacity, deaths are often characterised as acute harvest, acute non-harvest and chronic, with the acute harvest deaths referring to those that would have occurred within a period of a few weeks irrespective of the PM₁₀ concentrations. Although the cohort designs provide some indication of the extent of advancement for the non-harvest effects, they do not assist in determining the proportion of deaths that fall within each category. The impact in terms of costs associated with the harvest versus non-harvest deaths is significant. For example Bicknell (2001) places a value of \$14,195 on harvested deaths in New Zealand based on a one-month mortality advancement compared to a value of \$172,000 per year of reduced life for non-harvest deaths.

2.4 Health indicators

One of the more current questions regarding health effects of particles is the selection of an appropriate indicator of impact. For the past decade monitoring has focused on the PM₁₀ size fraction and consequently the majority of the epidemiology relates to this indicator. However, in more recent years there has been a tendency to focus on the smaller PM_{2.5} size fraction, because these particles penetrate deeper into the lung. Increased monitoring of PM_{2.5} has occurred within the last five years in both New Zealand and overseas with an additional focus on particle speciation to assist in studies on biological mechanisms and the impact of composition.

The relative impact of the PM_{2.5} size fraction versus the particles in the coarse PM₁₀–PM_{2.5} size fraction has been considered in a number of ways. Based on the epidemiology, the variation in risk across a range of ambient concentrations has generally been similar for PM₁₀ and PM_{2.5}. While the PM_{10-2.5} size fraction has tended to show less consistent associations, associations between the coarse size fraction and adverse health effects are shown to occur in a few studies. In particular the Expert Panel on Air Quality Standards (EPAQS, 2000) report examples of associations between the coarse size fraction and a number of health endpoints including:

- all cause mortality (from Dockery 1992, Mar et al, 2000, Castillejos et al, 2000 and Schwartz et al, 1996)
- cardiovascular mortality (from Mar et al, 2000)
- cough without other symptoms (from Schwartz and Neas, 2000)
- cough symptoms in children with chronic respiratory symptoms (from Tiittanen et al, 1999)
- hospital admissions for cardio-respiratory disease (from Burnett et al, 1997).

Other researchers (e.g. Brunekreef and Holgate, 2002) indicate that the bulk of the evidence suggests that mortality is associated with the finer $PM_{2.5}$ size fraction, although the coarse fraction may be responsible for other effects such as hospital admissions for asthma.

Studies of the specific impact of the coarse fraction are clearly varied. For example, Schwartz et al (1999) found no association between coarse particles of crustal origin on mortality in the city of Spokane, WA, whereas Ostro et al (1999) found an association between PM_{10} and daily mortality in a desert area of California where coarse particles comprise the larger part of PM_{10} .

Of particular interest in areas of New Zealand are the health implications of particles derived from sea spray emissions. While these particles typically reside in the PM_{10} size fraction, measurements in the finer $PM_{2.5}$ size range have been measured in New Zealand. The extent to which studies indicating effects associated with coarse particles apply to sea spray is uncertain. It could be argued that particles from sea spray would dissolve in the lungs and therefore that health effects are unlikely. However, in the absence of literature specific to the health implications of sea spray particle emissions, the precise health implications remain uncertain.

While these data suggest some health impacts are associated with the coarser particle fraction, it is possible that another measure (e.g. a smaller size fraction, compositional analysis or particle count data) may provide a better indicator for assessing health effects and understanding biological mechanisms. Consideration of an alternative indicator was assessed for the United Kingdom by the Expert Panel on Air Quality Standards (EPAQS, 2000). The report concluded that based on the present evidence, the measurement of PM_{10} provided the most appropriate basis for an air quality standard for the United Kingdom. However, they also indicated that further research may lead to alternative metrics such as $PM_{2.5}$ or counts of ultra fine particles but that current data on these measures was insufficient to derive standards.

In the United States a revised particle standard based on the $PM_{2.5}$ size fraction was introduced in 1997. Other countries such as Australia and Canada have also moved towards standards for the $PM_{2.5}$ size fraction. Some researchers argue that an even smaller size fraction (e.g. PM_1) or particle count measurements may be a more suitable indicator of health impacts. It is unlikely, however, that standards for these alternative indicators would be adopted without further data on the relationships between such measurements and health impacts.

3 Health Effects Studies for New Zealand

Epidemiological studies of the relationships between concentrations of particles and health in New Zealand are limited to two studies carried out in Christchurch. The first study was a time-series design that considered the impact of both PM₁₀ concentrations and temperature on mortality (Hales et al, 1999). Results indicated an increase in both all cause mortality and respiratory mortality associated with a one-day lag in PM₁₀ concentrations measured at the St Albans monitoring site in central Christchurch. The size of the effect, 1% (0.5–2.2%) for all cause mortality was reasonably consistent with similar studies carried out in other countries (e.g. Dockery et al, 1994). An association between respiratory mortality and PM₁₀ concentrations of 4% (1.5–5.9%) per 10 µgm⁻³ increase in daily PM₁₀ concentrations was also recorded. Unlike some overseas studies, no relationship between deaths from cardiovascular disease and PM₁₀ concentrations was observed.

In addition to the Hales et al (1999) mortality study, a study of the relationship between hospital admissions and particle pollution was carried out in Christchurch by McGowan et al (2002). Results indicated a 3.37% increase in respiratory hospital admissions and a 1.26% rise in cardiac admissions for each interquartile rise in PM₁₀ concentrations (interquartile value 14.8 µgm⁻³). The size of the effect was generally consistent with overseas studies (e.g. Schwartz and Morris, 1995).

4 High Risk Areas in New Zealand

Monitoring of PM₁₀ concentrations has been carried out in most of the main urban areas within New Zealand. Summary information on PM₁₀ concentrations relative to the air quality categories are shown in Figure 4.1. While results are generally comparable, measurements in some areas (e.g. Richmond) were made predominantly during the winter when PM₁₀ concentrations are highest, thus biasing results to the worst case. A more detailed discussion of the monitoring period, methods and results is presented in the report *Monitoring of PM₁₀ in New Zealand* (MfE, 2003).

In many urban areas within the South Island of New Zealand, PM₁₀ concentrations regularly exceed the 24-hour average ambient air quality guideline value of 50 µgm⁻³ (MfE and MoH, 2002). In many areas meteorological conditions are particularly conducive to elevated pollution levels, with temperature inversions and low wind speeds restricting vertical and horizontal mixing and pollution dispersion. Some of the worst areas, as indicated by maximum 24-hour average PM₁₀ concentrations, are Christchurch, Timaru, Nelson, Alexandra and Kaiapoi (Figure 4.2).

While maximum 24-hour average PM₁₀ concentrations are not generally as high in the North Island, many areas experience concentrations in excess of the ambient air quality guideline value. In particular, 24-hour average PM₁₀ concentrations in specific urban areas within the Wellington and Waikato regions are of concern. Also of concern in the North Island are annual average PM₁₀ concentrations measured in Auckland of around 28 µgm⁻³, which are approximately 40% above the annual guideline value of 20 µgm⁻³ (MfE and MoH, 2002). Unlike most urban areas in New Zealand, PM₁₀ concentrations in Auckland do not show strong seasonal variations. This may indicate the dominance of less season specific sources such as motor vehicles and sea spray. Other areas where elevated PM₁₀ concentrations are common during the summer months include Whangarei and Otaki. Possible sources in these areas include local industry, and sea spray and dusts respectively.

The sources of PM₁₀ may have some importance when considering the health implications. Although the role of factors such as particle size and composition are not yet fully understood, it is likely that PM₁₀ measurements in New Zealand can be used to provide a good indication of the extent of health impact. Some exceptions may occur in locations such as Otaki and the Christchurch suburb of Sumner, where sea spray is thought to be a significant contributor to measured PM₁₀ concentrations. As indicated previously, it is possible that some health effects associated with particles do not apply to sources such as sea spray. Similarly estimates of health impacts based on extrapolation of studies from other areas may be a bit speculative in areas where dusts or other sources of coarser PM₁₀ (e.g. industrial processes) are a dominant contributor.

Figure 4.1: The percentage of measured 24-hour average PM₁₀ concentrations within air quality categories in New Zealand

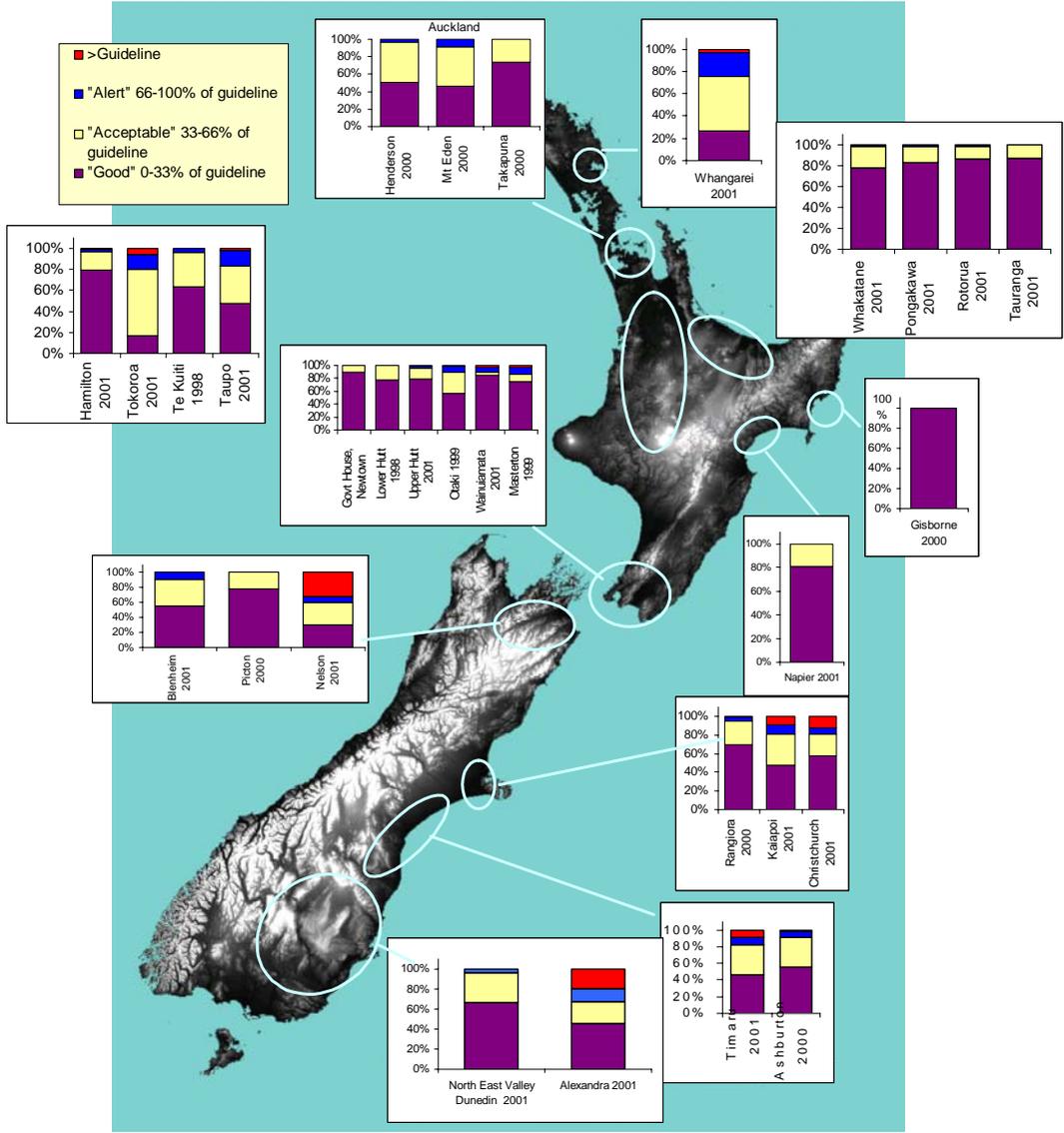
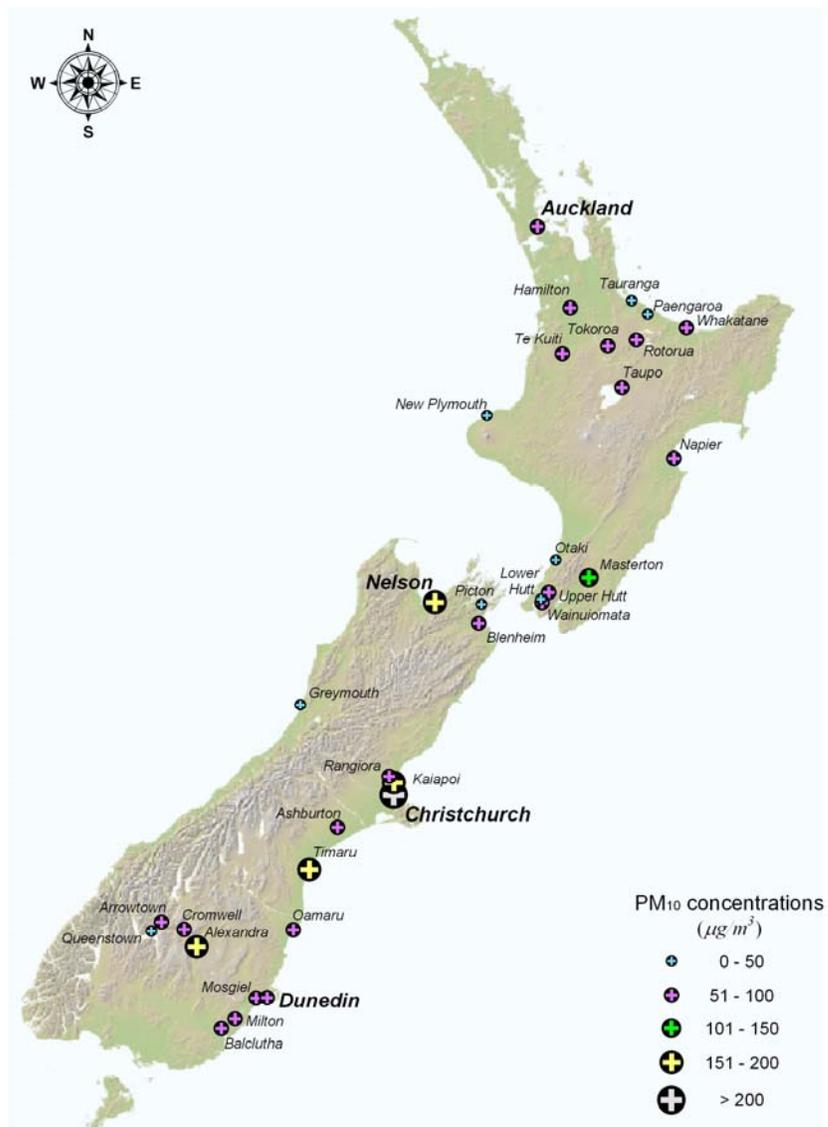


Figure 4.2: Maximum 24-hour average PM₁₀ concentrations in New Zealand measured between 1997 and 2001



Source: Aberkane and Wilton (2002).

5 Risk Assessments for New Zealand

A limited number of risk assessments have been carried out in New Zealand to estimate the impact of concentrations of PM₁₀ on mortality and morbidity. The largest of these, focuses on the number of deaths per year throughout New Zealand that may be associated with PM₁₀ concentrations from motor vehicle emissions, but also includes estimates of the impacts of PM₁₀ from other sources. The other two studies are risk assessments for Christchurch and Nelson, and are based on the relationships observed in the Hales study (1999), local mortality and morbidity data and measured PM₁₀ concentrations in those areas.

Differences in methodology between the New Zealand motor vehicle impact study (Fisher et al, 2002) and the two risk assessments carried out in Christchurch and Nelson include the following.

- The New Zealand motor vehicle study is based on Kunzli et al (2000) and applies a 4.3% increase in mortality for every 10 µgm⁻³ increase in annual average PM₁₀ concentrations above 7.5 µgm⁻³, whereas the Nelson and Christchurch studies are based on a 1% increase in daily mortality for every 10 µgm⁻³ increase in 24-hour average PM₁₀.
- The Christchurch and Nelson risk assessments include estimates of morbidity effects. While estimates of morbidity effects are included in Kunzli et al (2000), no estimates for New Zealand were made in Fisher et al (2002).
- The New Zealand motor vehicle study attempts to apportion the measured and estimated PM₁₀ concentrations and hence deaths to different sources (e.g. motor vehicles and domestic home heating).

5.1 The New Zealand motor vehicle impact study

The New Zealand motor vehicle impact study was a risk assessment carried out by Fisher et al (2002) to quantify the impact of PM₁₀ concentrations from motor vehicles in New Zealand on mortality. The three main components of the study were:

- the relationship between PM₁₀ concentrations and premature mortality
- estimates of exposure to PM₁₀ concentrations in New Zealand
- the contribution of motor vehicle emissions to PM₁₀ concentrations and exposure in New Zealand.

The methodology for assessing the impact of PM₁₀ concentrations on mortality was taken from the Kunzli et al (2000) risk assessment for Austria, France and Switzerland. The studies use a relationship of a 4.3% increase in mortality for every 10 µgm⁻³ increase in annual average PM₁₀ concentrations above 7.5 µgm⁻³ and are based on the longitudinal cohort studies of Dockery et al (1993) and Pope et al (1995). This impact estimate is about 4–5 times higher than the relationships typically observed in the standard times series epidemiological studies. This underestimate is attributed to the time-series methodology, which associates only those deaths that occur a relatively short time after the pollution episode to PM₁₀ concentrations. Thus they are limited to a selection of the acute impacts but do not estimate the reduced life expectancy due to long-term morbidity enhanced by air pollution (Kunzli et al, 2000).

Estimates of exposure to PM₁₀ concentrations for the New Zealand motor vehicle impacts study were based on measured PM₁₀ concentrations, where available and estimated exposure in other areas. The study was limited to areas containing at least 500 people per square kilometre, which included about 80% of the New Zealand population. In some of the larger cities, dispersion modelling was combined with monitored concentrations to estimate exposure. The main limitations in estimating exposure is lack of monitoring data in some areas as well as uncertainties associated with the accuracy of modelling in predicting exposure in the larger cities. The uncertainties associated with lack of monitoring data are unlikely to be significant owing to the relatively small number of people residing in the locations where data are lacking.

The third component of the New Zealand motor vehicles impact study was the attribution of measured or estimated PM₁₀ concentrations to motor vehicle sources. This was generally based on emission inventory studies where available and general estimates based on population, or similarities between locations in the absence of such studies. Uncertainties include absence of the quantification of natural sources including sea spray and dusts in many of the inventories and broad assumptions on similarities between areas.

Table 5.1 presents estimates of mortality for people over 30 exposed to PM₁₀ concentrations from all sources and just from motor vehicles. This data is based on all towns and cities with a population of greater than 5000 people, encompassing 78% of the population, using annual average PM₁₀ data for an average year assuming a no effects threshold of 7.5 µgm⁻³.

Table 5.1: Mortality estimates for PM₁₀ in New Zealand

	Total mortality*	Vehicle related mortality*
Auckland	436	253
Wellington	79	56
Christchurch	182	41
Dunedin	48	6
Rest of North Island	133	21
Rest of South Island	80	19
Total New Zealand	970**	399**

Source: Fisher et al (2002).

* Based on a 4.3% increase in mortality, in population over 30, per 10 µgm⁻³ increase in annual average PM_{2.5} concentrations above 7.5 µgm⁻³.

** Note: Some differences in totals occur as a result of rounding in calculations.

A number of limitations with the New Zealand motor vehicle impact study identified by the reviewers are presented as an Appendix to the Fisher et al (2002) report. These include focus on a number of uncertainties associated with the estimates of impact including issues of population exposure as well as apportionment of sources to motor vehicles and other sources. There are clearly a number of uncertainties, particularly with the latter apportionment assessment. However, these are acknowledged in the report and further studies to improve on these data and subsequent impact estimates will be undertaken using funding provided by the Health Research Council, the Ministry of Transport and the Ministry for the Environment.

5.2 Risk assessment for Nelson

A risk assessment of the impact of PM₁₀ concentrations in Nelson was estimated based on the number of 10 µgm⁻³ increments in 24-hour average PM₁₀ concentrations measured in Nelson, the baseline annual mortality and morbidity data for Nelson based on the years 1997 and 1998, and the dose-response relationships from epidemiological studies (Wilton, 2001).

The baseline mortality and morbidity data for Nelson were obtained from the New Zealand Health Institute Statistics (NZHIS). Data were not available for years beyond 1998 so statistics for 1997 and 1998 were used. Health statistics data for dose-response relationships observed in Christchurch were used in preference to the WHO statistics because of the similarities in particle sources between Christchurch and Nelson.

The assessment was based on the following information and assumptions:

- a 1% increase in all cause mortality for every 10 µgm⁻³ increase in 24-hour average ambient PM₁₀ concentrations (as reported in Hales, 1999)
- a 3% increase in respiratory mortality for every 10 µgm⁻³ increase in 24-hour average ambient PM₁₀ concentrations (as reported in Hales, 1999)
- a 2.3% increase in respiratory admissions (including asthma) per 10 µgm⁻³ increase in 24-hour average PM₁₀ concentrations (as reported in McGowan et al, 2000)
- a 1.25% increase in asthma admissions per 10 µgm⁻³ increase in 24-hour average PM₁₀ concentrations (as reported in McGowan et al, 2000)
- a 0.85% increase in cardiac admissions per 10 µgm⁻³ increase in 24-hour average PM₁₀ concentrations (as reported in McGowan et al, 2000)
- no threshold exists below which effects do not occur
- a total of 830 10 µgm⁻³ increments in Nelson based on estimated average exposure, per year
- baseline mortality and morbidity data from 1997 and 1998
- classification of illnesses as per the International Classification Codes described in appendices one and two
- the study area for the assessment including the following Nelson Census Area Units: Clifton, Atawhai, The Wood, Britannia Heights, Trafalgar, Kirks, Bronte, Atmore, Tahunanui, Tahuna Hills, Toi Toi, Broads, Grampians, The Brook, Nelson Airport, Nayland, Waimea Inlet East, Saxton, Langbein, Maitlands, Isel Park, Enner Glynn and Ngawhatu.

The risk assessment indicates that around eight deaths per year are likely to occur as a result of existing particle concentrations. In addition, six respiratory hospital admissions are likely to occur each year as a result of PM₁₀ concentrations, including one to two asthma admissions. Around eight cardiac admissions could be attributed to particle concentrations, giving a total of around 14 hospitalisations per year that are likely to occur as a result of existing particle concentrations. These estimates include only a selection of acute health impacts, namely those that would occur near to the pollution event. The analysis is therefore highly conservative and estimates of both mortality and hospitalisations are likely to underestimate actual impacts. Because of the assumptions associated with the analyses these data should be treated as indicative of order of magnitude only for the minimum impact.

The assessment also estimates the number of PM₁₀ related restricted activity days (RAD), which are defined by Ostro (1987) as days spent in bed, days missed from work and days when activities are partially restricted due to illness. Ostro examined the association between PM_{2.5} concentrations and RAD in the adult population during a two-week period in 49 United States metropolitan areas. In an assessment of the costs associated with concentrations of particles, the American Lung Association extrapolated the results of the Ostro study to population-based statistic. They indicated an increase in RAD of approximately 91,200 RAD each year per million of population for every 1 µgm⁻³ increase in annual average PM_{2.5}. Assuming a ratio of PM_{2.5} to PM₁₀ of 0.6:1 and an annual average PM₁₀ concentration of 26 µgm⁻³, these data indicate that around 58,000 RAD could be expected to occur each year as a result of concentrations of particles in Nelson.

5.3 Risk assessment for Christchurch

A similar risk assessment was carried out for Christchurch in 1999, based on health statistics for 1992 and 1993, air quality monitoring data from a central monitoring site and the relationships between PM₁₀ and mortality and morbidity from the Hales (1999) and McGowan (2002) studies. These include a 1% increase in all cause mortality for every 10 µgm⁻³ increase in 24-hour average ambient PM₁₀ concentrations, a 3% increase in respiratory mortality for every 10 µgm⁻³ increase in 24-hour average ambient PM₁₀ concentrations, a 2.3% increase in respiratory admissions (including asthma) per 10 µgm⁻³ increase in PM₁₀ concentrations, a 1.25% increase in asthma admissions per 10 µgm⁻³ increase in PM₁₀ concentrations and a 0.85% increase in cardiac admissions per 10 µgm⁻³ increase in 24-hour average PM₁₀ concentrations.

The assessment indicates that existing PM₁₀ concentrations are associated with around 40–70 deaths per year and around 75–100 hospitalisations (Wilton, 1999). An estimate of restricted activity days was also carried out based on the American Lung Association's extrapolation of the Ostro (1987) paper. Results indicated around 300,000 to 600,000 days per year when people's activities are restricted as a result of particle pollution (Wilton, 2001).

6 Health Effects of Particles in New Zealand

An estimate of the impact of existing PM₁₀ concentrations on total mortality in New Zealand has been made by Fisher et al (2002) based on the results of the Kunzli (1999) study. This includes estimates of effects not detected using the standard time series epidemiological studies. Thus the number of deaths estimated for Christchurch is considerably greater than the 40–70 estimated by Wilton (1999) for the acute impacts associated with the standard time series study design. Similarly estimates of the impact of PM₁₀ concentrations on mortality in Nelson will be about four times greater than those estimated in Wilton (2001) if the chronic and other acute effects are included.

Although there are some uncertainties with the mortality estimates of Fisher et al (2002) associated with the exposure estimates and allocation to sources, these are based on the best available information. Further improvements in these estimates without considerable investment in PM₁₀ monitoring and exposure assessment are unlikely.

Table 6.1 collates the existing health impact estimates for PM₁₀ in New Zealand and includes additional estimates of morbidity impacts in the main urban areas. These estimates are based on the standard time series studies and therefore include only the acute health impacts that occur shortly after the pollution event. The mortality estimates in this risk assessment are much higher than the hospitalisation because they also include estimates of impacts that do not occur directly following the pollution event. Thus the hospitalisation estimates are likely to be extremely conservative. Estimates of health endpoints from this study have been rounded to the nearest five deaths and hospitalisations and the nearest 10,000 for RAD. These data are based on the following assumptions and should be treated as indicative only.

- Measured PM₁₀ concentrations in these areas are indicative of average exposure across the urban areas.
- Increases in hospitalisations associated with PM₁₀ concentrations as per the McGowan et al (2002) study for Christchurch.
- Increase in RAD of approximately 91,200 RAD each year per million of population for every 1 µgm⁻³ increase in annual average PM_{2.5} (American Lung Association, 1995).
- Estimates of the relationships between PM₁₀ and PM_{2.5} in areas where these are not known.
- No threshold below which effects do not occur.

Table 6.1: Estimates of health impacts of particle concentrations in New Zealand

	Estimated annual mortality	Estimated hospitalisations per year	Estimated restricted activity days per year
Auckland	436 ⁽³⁾	200	750,000
Wellington	79 ⁽³⁾	30	100,000
Christchurch	182 ⁽³⁾	80	300,000 ⁽¹⁾
Dunedin	48 ⁽³⁾	20	80,000
Nelson	20 ⁽⁴⁾	14 ⁽²⁾	58,000 ⁽²⁾
Hamilton	40 ⁽⁴⁾	30	90,000
Timaru	20 ⁽⁴⁾	10	30,000
Lower Hutt	10 ⁽⁴⁾	20	60,000
Upper Hutt	20	10	30,000
Alexandra	5	<5	10,000
Tokoroa	10	5	20,000

1 Lower limit estimate from Wilton (2001).

2 From Wilton (2002).

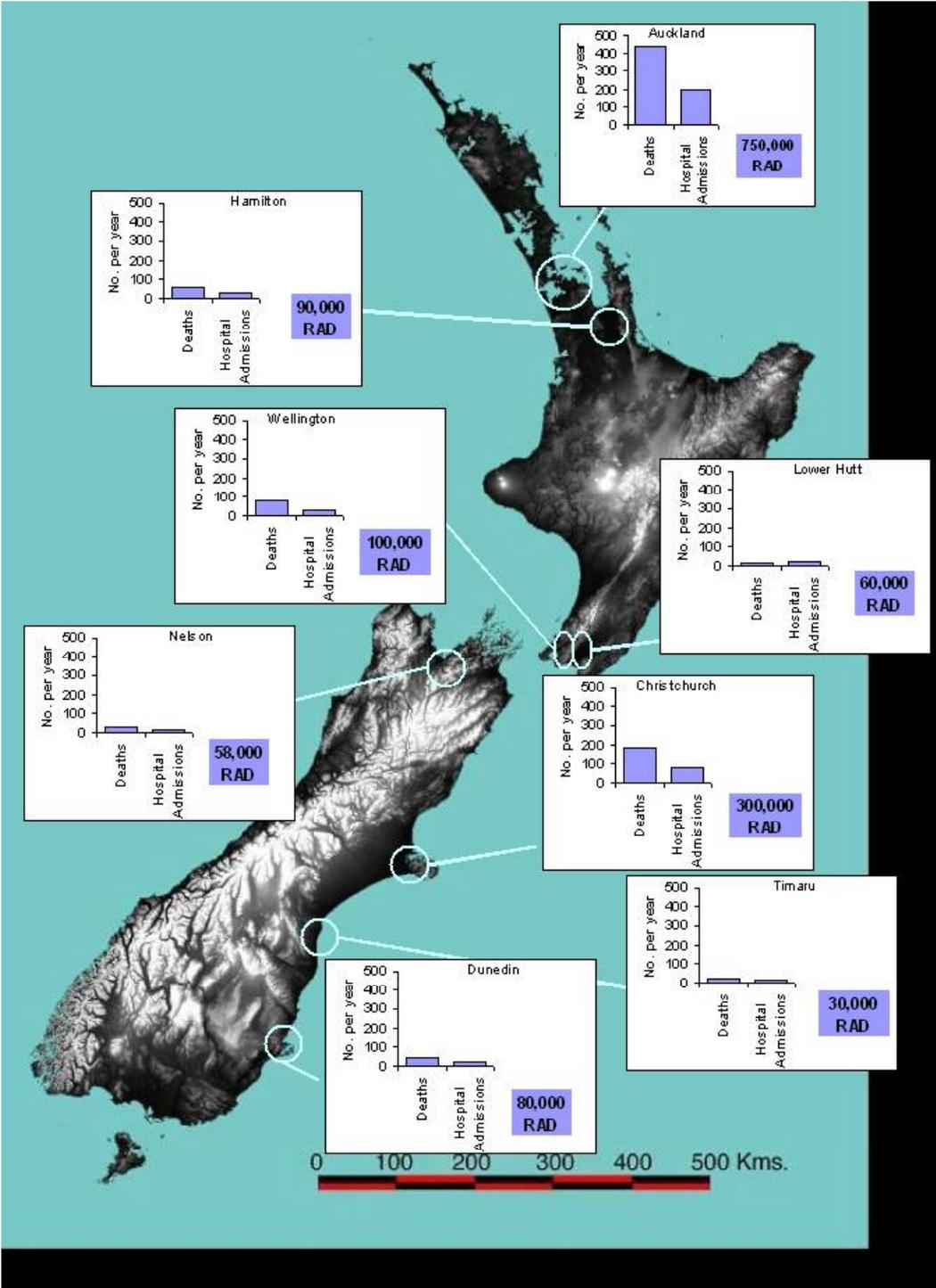
3 From Fisher et al (2002) based on a 4.3% increase in mortality, in population over 30, per 10 $\mu\text{g m}^{-3}$ increase in annual average $\text{PM}_{2.5}$ concentrations above 7.5 $\mu\text{g m}^{-3}$.

4 Rupendra Shresthra from Fisher et al (2002) analysis based on a 4.3% increase in mortality, in population over 30, per 10 $\mu\text{g m}^{-3}$ increase in annual average $\text{PM}_{2.5}$ concentrations above 7.5 $\mu\text{g m}^{-3}$.

The estimates of mortality for Christchurch and Nelson shown in Table 6.1 are greater than the values indicated in Wilton (1999) and (2002) of 40–70 and 14 respectively. This is because the former estimates were based on the time series methodology, which is unlikely to estimate the reduced life expectancy associated with long-term morbidity enhanced by air pollution. Estimates included in this study are based on the relationships described by Kunzli et al (2000).

Figure 6.1 provides an illustration of estimates of the impacts of existing PM_{10} concentrations on mortality, hospital admissions and restricted activity days in the main urban areas of New Zealand. These data, from Table 6.1, are based on assumptions relating to existing PM_{10} and $\text{PM}_{2.5}$ exposure and health endpoints and should be treated as indicative only.

Figure 6.1: Estimate of the impact of existing PM₁₀ concentrations on health in the main urban areas of New Zealand



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About the Ministry

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

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

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

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

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