

Air Pollution and Mortality in Christchurch, New Zealand

Rupendra Narashingh Shrestha

**A thesis submitted for the degree of Doctor of Philosophy
of The Australian National University**

March 2009

Unless otherwise stated, this thesis is my own work undertaken through National Centre for Epidemiology and Population Health of the Australian National University.

A handwritten signature in black ink, written in a cursive style, reading "Rupendra". The signature is positioned above a horizontal line that extends to the right.

Rupendra Narashingh Shrestha

Acknowledgements

I would like to thank my supervisors, Professor Tord Kjellstrom, Dr. Keith Dear and Associate Professor Geoff Morgan for their supervision, guidance, support and encouragement during my study at NCEPH. A special thanks to Tord for introducing me to environmental health research, and for his supervision and support not only during my study at NCEPH but also during my work at the University of Auckland, and for all his enthusiastic ideas which made a valuable contribution to the content and direction of this thesis.

Thanks to Environment Canterbury and Ms. Teresa Aberkane, in particular, for providing weather and air pollution data, and for clarifying my numerous data related queries. Thanks also to the HAPiNZ team and Dr. Simon Kingham, in particular, for sharing spatial PM₁₀ exposure data for this thesis.

I would like to thank Dr. Simon Hales from Wellington School of Medicine and Health Sciences, and Dr. Patricia Metcalf from the University of Auckland for their advice on estimating the missing weather and air pollution data. Thank you Amanda Scoggins and Ivan Hanigan for showing me how to make maps in GIS. Thank you Susie Fletcher and Shaun Paisley for proof reading my thesis.

I would like to express my thanks to fellow students and all at NCEPH for a friendly, supportive and great working environment. I would like to specially thank the administrative and IT staff at NCEPH, who were very helpful at all times. My colleagues Milton, Annie, Vasoontara and Saifur deserve special thanks for providing a great support. I learnt a lot about epidemiology from the numerous discussions I had with Milton.

I thank Dr. Deborah Schofield and the Northern Rivers University Department of Rural Health for providing me an office space and IT resources to work on my thesis towards the end of my PhD programme.

Nepalese friends, Sunil, Santosh, Rajani, Krishna, Amarottam, Pramod, and their families, and other members of Nepalese community in Canberra provided a great

support. Thanks for your support and occasional free dinner whenever I did not feel like cooking at the hostel.

I would like to thank my mother, Radheswori, brothers (Dipu and Manish) and their wives, and cousin Jay for their encouragement and support. My father always encouraged me to pursue higher education, while he was alive. Finally, I am grateful to my wife, Juni, for being so understanding. Without her love, care, support and encouragement, this thesis would not have been possible.

Abstract

Although numerous time series epidemiological studies have reported an association between daily mortality and air pollution, there is uncertainty about the extent of life shortening due to air pollution. It is unclear whether the increase in daily deaths as demonstrated by time series studies is just an indicator of the short-term displacement of the time of death of frail individuals by a few days, a phenomenon known as “harvesting”. This is an important question for the public health task of estimating the impact of air pollution on mortality. The impact will be much lower if the mortality displacement is very short-term i.e. just by a couple of days, rather than a substantial reduction of life expectancy by months or years. While several time series studies have provided evidence of the acute effects of air pollution on mortality, there is limited research analysing the extended effects of air pollution over a few weeks and the effects of long-term exposure to air pollution on mortality.

Christchurch has amongst the worst air quality in New Zealand. The frequent occurrence of calm periods and temperature inversions on cold winter nights, in combinations with emissions from the burning of coal and wood as domestic heating, exacerbate winter air pollution levels in Christchurch. For several decades, air pollution from the burning of coal and wood in the winter has been a major concern, primarily as an environmental nuisance. In addition to the emissions from domestic heating, air pollution from other sources, mostly vehicle emissions, is also a growing concern. Because of mounting evidence of the association between air pollution and mortality, air pollution has been increasingly recognised as a public health threat in Christchurch. This study examines whether or not particulate matter is associated with an increase in mortality in Christchurch. The four main objectives are: 1) to estimate the acute effects of daily air pollution on daily mortality, up to a few days after exposure; 2) to test whether an association between short-term exposure to particulate matter and mortality can be attributed to “harvesting”; 3) to estimate the extended effects of particulate matter on daily mortality up to a few weeks after exposure; and 4) to quantify the association between long-term exposure to particulate matter and annual mortality. Time series studies were carried out for the first three objectives and an ecological cross-sectional study was conducted for the fourth objective.

Mortality data for the years 1988 – 1999 were obtained from the New Zealand Health Information Service. Environment Canterbury provided routinely monitored air

pollution and weather data. For the first three objectives, daily mortality, air pollution and weather data for the period from June 1988 to December 1999 were analysed. For the fourth objective of estimating the effect of long-term exposure to PM₁₀ on mortality at annual level, census area unit based mortality data from 1996 to 1999 were associated with spatial annual average PM₁₀ exposure estimates provided by the University of Canterbury.

Modelling techniques included Poisson regression, polynomial distributed lag, time series analysis over the mid-scale variations of daily data and logistic regression across spatial units. Poisson regression models were used to associate daily air pollutant concentrations with daily mortality controlling for a long-term trend, seasonal variation in daily mortality and the confounding effects of weather variables. Polynomial distributed lag modelling was used to analyse the extended effects of PM₁₀ on mortality. The mid-scale variations of daily data were analysed to examine the presence of harvesting. Logistic regression across spatial units was used to associate annual average PM₁₀ concentrations with annual mortality.

A significant positive association was observed between daily PM₁₀ and daily non-external mortality. An increase of 10 µg/m³ in daily PM₁₀ was associated with a 1.5% increase (95% CI: 0.6, 2.5%) in the same day non-external deaths in the population aged 65+ years. When deaths up to several weeks were considered using polynomial distributed lag models, the total estimated effects of PM₁₀ on non-external cause mortality, circulatory mortality and respiratory mortality were found to be much larger. There was a total estimated increase of 14.9% (95% CI: 6.9, 23.4%) in non-external cause mortality in the population aged 65+ years during 41 days after exposure (including the day of exposure) for each 10 µg/m³ increase in daily PM₁₀. The analysis of long-term exposure to PM₁₀ estimated an increase of 17% (95% CI: 9, 26%) in annual non-external deaths in the population aged 65+ years for each 10 µg/m³ increase in annual average PM₁₀. The estimated PM₁₀ associated risk of mortality was higher for respiratory mortality than for other causes.

This study provides evidence of both short-term and long-term effects of PM₁₀ on mortality in Christchurch and suggests that studies focussing only on the effects of PM₁₀ on mortality on the same day or a couple of days after exposure underestimate the total effect of PM₁₀. It also shows that the short-term association between PM₁₀ and mortality can not be entirely attributed to harvesting. These findings contribute to the scientific evidence of mortality effects of PM₁₀ both in the short-term and long-term,

and suggest that air pollution may be a greater threat to public health than what has been thought until now.

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List of Abbreviations

AIC	Akaike's information criterion
APHEA	Air Pollution and Health: A European Approach Project
BS	Black smoke
CAU	Census area unit
CO	Carbon monoxide
GAM	Generalised additive model
GLM	Generalised linear model
HAPiNZ	Health and Air Pollution in New Zealand Project
HDOM91	1991 Health Domicile Code
HDOM96	1996 Health Domicile Code
ICD-10	International Classification of Diseases, Version 10
ICD-9	International Classification of Diseases, Version 9
NEOH	New Zealand Environmental and Occupational Health Research Centre
NMMAPS	The National Morbidity, Mortality and Air Pollution Study
NO ₂	Nitrogen dioxide
NZHIS	New Zealand Health Information Service
PM	Particulate matter
PM ₁₀	Particulate matter with an aerodynamic diameter of 10 µm or less
PM _{10-2.5}	Coarse particles: particulate matter with an aerodynamic diameter of less than 10 µm but greater than 2.5 µm
PM _{2.5}	Fine particles: particulate matter with an aerodynamic diameter of 2.5 µm or less
SO ₂	Sulphur dioxide
TAPM	The Air Pollution Model
TEOM	Tapered elemental oscillating microbalance particulate matter monitor
TSP	Total suspended particles

Chapter 1: Introduction

1.1 Background

Air pollution has been a part of human life since before the days of civilisation, as people used fire, a major source of air pollution, for cooking and warmth. Prehistoric evidence of human exposure to air pollution from the burning of solid fuels can be found in mummified lung tissues (Brimblecombe, 1999). With urbanisation, the use of fossil fuels such as coal, oil, petrol, diesel and natural gas for transport, power generation and other human activities increased air pollution. The major sources of air pollution in modern cities are vehicle emissions, emissions from domestic home heating using solid fuels such as wood and coal, and industrial emissions.

Air pollution has long been recognised as one of the major threats to public health. Extreme air pollution episodes, such as those occurred in the Meuse Valley, Belgium in December 1930 (Roholm, 1937), Donora, Pennsylvania in October 1948 (Ciocco and Thompson, 1961), and London in December 1952 (UKMoH, 1954), have been associated with increases in mortality and morbidity. The risk to health from air pollution was readily apparent during these extreme air pollution episodes. Increases in mortality and morbidity were reported during and after the episodes, and such events led to actions to reduce air pollution. Although ambient air pollution levels have decreased substantially in the major cities of the developed countries, the problems persist today in both developed and developing countries.

As ambient air pollution concentrations during subsequent episodes became significantly lower than in the earlier extreme episodes, it became harder to detect health effects during the short episodes (Brunekreef and Holgate, 2002). Research on the health effects of air pollution thus shifted from a focus upon the health effects during the episodes to time series methods which examine daily variations in air pollution over a longer period of time as determinants of daily variations in mortality. With advancement in analytical methods, time series methods have emerged as one of the most important statistical methods to analyse the associations between air pollution and mortality (and morbidity). Many of these time series studies have reported associations at ambient concentrations below existing health guidelines.

Time series epidemiological studies have documented short-term associations between air pollution and daily mortality in a number of cities, including Australian and New Zealand cities, using a wide range of modelling techniques (Schwartz, 1993;

Schwartz, Dockery and Neas, 1996; Katsouyanni *et al.*, 1997; Kelsall *et al.*, 1997; Simpson *et al.*, 1997; Michelozzi *et al.*, 1998; Morgan *et al.*, 1998; Ostro *et al.*, 1999; Hales *et al.*, 2000; Lee *et al.*, 2000; Samet *et al.*, 2000c; Denison *et al.*, 2001a; Zanobetti *et al.*, 2002). Time series studies associate day-to-day variations in mortality with day-to-day variations in air pollution concentrations after controlling for the effects of confounding variables that vary from day-to-day such as weather variables. These studies evaluate an increased risk of mortality due to an increase in air pollution over very short intervals of time, usually one day to a few days.

Air pollution can increase deaths on the same day as well as on several subsequent days. The effect of air pollution on mortality is likely to be distributed over several days (Zanobetti *et al.*, 2000), so only models that analyse the extended effects of air pollution on mortality by combining the effects of air pollution on mortality occurring on the same day and on subsequent days can estimate the overall effect of air pollution on mortality.

Although numerous time series studies have reported that an increase in daily mortality is associated with an increase in daily ambient air pollution concentrations, it is unclear whether most or even all the excess daily deaths would have occurred in a few days regardless of air pollution levels. It is unclear whether air pollution only displaces the time of deaths by a few days, usually known as “harvesting” or substantially shortens the life by months or years. If all air pollution related deaths occur only among those frail persons who would have died within a few days, their lifespan are not greatly shortened and air pollution will be less of a public health concern. In order to assess the public health significance of air pollution, it is important to know by how many days, months or years the time of deaths is advanced by air pollution. The public health impact will be much more if air pollution brings forward the time of death by months or years rather than just a few days (McMichael *et al.*, 1998; Brunekreef and Holgate, 2002). Any evidence that suggests that “harvesting” (i.e. short-term displacement of time of deaths by a few days) is not the dominating cause of increased mortality associated with air pollution will have significant public health implication.

Because of study design, daily time series studies can only demonstrate the short-term associations between air pollution exposure and mortality (and morbidity). They are designed to pick up only the short-term increase in the number of air pollution related deaths (and hospital admissions) against the background long-term mortality (and morbidity) and provide no information on the association between long-term exposure to air pollution and the background long-term mortality (and morbidity). The

relationship between long-term exposure and mortality (and morbidity) has been evaluated using both cross-sectional ecological studies (Chappie and Lave, 1982; Ozkaynak and Thurston, 1987; Scoggins *et al.*, 2004) and prospective cohort studies (Dockery *et al.*, 1993; Finkelstein *et al.*, 2003; Pope *et al.*, 2004; Filleul *et al.*, 2005; Krewski *et al.*, 2005c; Gauderman *et al.*, 2007). While time series studies estimate the acute effects of air pollution on mortality (and morbidity), cross-sectional ecological studies and cohort studies estimate the chronic effects of air pollution on mortality, and the effects of accumulated air pollution exposure on mortality. From a health point of view, all of these effects are equally important.

A few overseas studies, mainly North American and European, have examined the role of “harvesting” in the association of short-term exposure to particulate matter with mortality (Zeger, Dominici and Samet, 1999; Schwartz, 2000c; 2001; Dominici *et al.*, 2003c) and others have analysed the extended effects of particulate matter on mortality spread over several days (Schwartz, 2000b; Braga, Zanobetti and Schwartz, 2001; Zanobetti *et al.*, 2002; Goodman, Dockery and Clancy, 2004). These studies have provided evidence against “harvesting”. The studies analysing the extended effects of particulate matter on mortality showed that the total effect of particulate matter spread over several days were much larger than the effects on a single day mortality; either on the same day or on the mortality occurring a few days after exposure. To date, there has not been any research analysing these issues in detail in Australian and New Zealand cities.

Australian and New Zealand cities are very different from the North American and European cities in terms of air pollution (for example composition of air pollution mixtures), weather conditions, population structure, lifestyle which could affect exposure to air pollution and access to health services. For example, air pollution in Christchurch is mainly a winter time problem because of the burning of solid fuels for domestic heating in the winter. This is most likely to be very different from air pollution in most North American and European cities where vehicle and industry emissions are the dominant sources of air pollution. The climatic conditions in Christchurch are also considerably different from many other cities. Lengthy calm periods and temperature inversions frequently occur, especially in cold winter nights in Christchurch. Because of these differences, the findings in Christchurch may be different from the results of the US and European studies.

1.2 Air pollution and mortality in Christchurch

Although New Zealand, in general, has a clean and green image, and has clean air relative to major North American and European cities, some parts of New Zealand, most notably the city of Christchurch, have significant air pollution problems. For several decades, air pollution from the burning of solid fuels for domestic heating in the winter has been a major concern in Christchurch, primarily as an environmental nuisance. Domestic heating contributes about 82% of particulate matter of less than 10 micrometers in diameter (PM₁₀) in the winter months (Scott and Gunatilaka, 2004). Because of the topography and meteorology of Christchurch, lengthy calm periods and temperature inversions frequently occur, especially in cold winter nights, when emissions from domestic heating are most likely to be at the highest level. The frequent occurrences of such meteorological conditions, which are conducive to high air pollution, make the air pollution problem worse in the winter months in Christchurch. As a result, the twenty-four hour average PM₁₀ concentrations exceeds the Ministry for the Environment ambient air quality guideline of 50 µg/m³, on average, for 30 days each year in the winter (Canterbury Regional Council, 1997; Ministry for the Environment and Ministry of Health, 2002). In addition to the emissions from domestic heating, air pollution from other sources, particularly vehicle emissions, is also becoming a growing concern (Kjellstrom, Shrestha and Metcalf, 2002).

Because of growing evidence of the associations between air pollution and mortality provided by overseas studies, air pollution has been increasingly recognised as a public health concern in Christchurch. A few studies have been carried out analysing the association between air pollution and mortality (and morbidity) in Christchurch (Dawson, Allan and Fergusson, 1983; Wilkie *et al.*, 1995; Harre *et al.*, 1997; Hewat *et al.*, 1998; Hales *et al.*, 2000; McGowan *et al.*, 2002). While some studies showed statistically significant positive associations of daily particulate matter with daily mortality (Hales *et al.*, 2000) and with daily cardio-respiratory hospital admissions (McGowan *et al.*, 2002) in Christchurch, others did not find evidence of an association between the city's air pollution levels and health outcomes (Dawson, Allan and Fergusson, 1983; Wilkie *et al.*, 1995; Hewat *et al.*, 1998). Inconclusive evidence could have resulted from small sample sizes and the short duration of these studies, which may have limited their statistical power to detect any association between air pollution and health outcomes.

A time series analysis of the association of short-term exposure to PM₁₀ with daily mortality in Christchurch reported a 1% increase in all-cause mortality and a 4%

increase in respiratory mortality for each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} on the day prior to death (Hales *et al.*, 2000). While Hales *et al.* (2000) estimated the short-term association between PM_{10} and mortality, a national risk assessment, commissioned by the Ministry of Transport, based on the exposure-response relationship from long-term exposure studies, estimated 182 deaths per year due to particulate matter from all sources in the population aged 30+ years in Christchurch (Fisher *et al.*, 2002). This risk assessment followed the methodology used by Kunzli *et al.* (2000), which assumed a 4.3% increase in mortality for each $10 \mu\text{g}/\text{m}^3$ increase in annual average PM_{10} concentrations above the threshold level of $7.5 \mu\text{g}/\text{m}^3$ (Kunzli *et al.*, 2000). The exposure-response relationship was based on the exposure-response relationships from the two US cohort studies (Dockery *et al.*, 1993; Pope *et al.*, 1995).

The need for the detailed analysis of the associations between particulate matter and mortality in Christchurch became evident when I was working with Professor Tord Kjellstrom in the research projects on health effects of air pollution at the New Zealand Environmental and Occupational Health Research Centre, University of Auckland. We were involved in the national risk assessment, commissioned by the Ministry of Transport, which estimated the number of premature deaths in the population aged 30+ years in New Zealand due to exposure to PM_{10} from vehicle emissions and from all sources (Fisher *et al.*, 2002). The Health and Air Pollution in New Zealand (HAPiNZ) project, jointly funded by Health Research Council, the Ministry for the Environment, and Ministry of Transport, has recently been undertaken to determine the environmental, health, social, and economic costs of air pollution in New Zealand (Fisher *et al.*, 2005; Fisher *et al.*, 2007). My PhD thesis has made a major contribution to the HAPiNZ project, especially in the areas of epidemiological analysis of PM_{10} and mortality in Christchurch.

1.3 Aim and objectives

The main aims of this thesis are to examine whether or not particulate matter is associated with mortality and to analyse the associations over different time scales in Christchurch, New Zealand. Under these aims, the main objectives of this research are:

1. To quantify the effects of short-term exposure to particulate matter on daily mortality.
2. To test whether or not the association between short-term exposure to particulate matter and daily mortality is due to mortality displacement by a few days.

3. To quantify the extended effects of particulate matter on daily mortality using distributed lag models.
4. To explore the shape of the distribution of effects of particulate matter on daily mortality over lag days.
5. To study the association between long-term exposure to particulate matter and annual mortality.

1.4 Significance of the study

Although previous Australian and New Zealand based time series studies have documented positive associations between short-term exposure to air pollution, including particulate matter, and daily mortality and morbidity (Simpson *et al.*, 1997; Morgan, Corbett and Wlodarczyk, 1998; Morgan *et al.*, 1998; Hales *et al.*, 2000; Denison *et al.*, 2001a; Denison *et al.*, 2001b; McGowan *et al.*, 2002; Simpson *et al.*, 2005a; Simpson *et al.*, 2005b), there is very limited knowledge about the extended effects of air pollution on mortality and whether or not the short-term association between daily air pollution and mortality is due to “harvesting”. This is the first study to analyse these issues in Christchurch, and perhaps the first in this region. Analysis of the associations between exposure to particulate matter and mortality over different time scales, ranging from the very short-term to the long-term at annual level, in the same city, provides the overall knowledge about the effects of particulate matter on mortality. This study adds to what we know about the health effects of particulate matter in Christchurch in particular, and to scientific knowledge of the mortality effects of PM₁₀.

1.5 Structure of the thesis

This chapter provides the rationale for this study and states its main aim and objectives.

Chapter 2 is a literature review, which provides a context for the development of the thesis. This chapter reviews the literature relevant to the association between particulate matter and mortality with a major focus on time series studies. The review covers major issues associated with time series studies including statistical modelling, the role of short-term harvesting in the association between air pollution and mortality, the concentration-response relationship, the threshold levels and whether any air pollution effects are modified by certain factors.

Chapter 3 describes the study region, the sources of data, and the preparation of data for analysis. This chapter explains the preparation of exposure data and discusses

various issues related to the data, including how they were collected by Environment Canterbury and the estimation of missing data using regression techniques.

The descriptive analyses of data are presented in Chapter 4 and Chapter 5. Chapter 4 provides descriptive analyses of population and mortality data, and Chapter 5 provides descriptive analyses of weather and air pollution data. Annual trends and seasonal variations in mortality, weather, and air pollution data are examined in these chapters. The correlations between weather and air pollution data are also analysed in Chapter 5.

Chapters 6, 7, 8 and 9 are the main sections of this thesis presenting the findings of the research. Chapters 6, 7 and 8 present the analyses of the temporal data with time series regression models as the main analysis technique. Chapter 6 analyses the first objective, to quantify the effects of short-term exposure to particulate matter on daily mortality. This chapter analyses the effect on daily mortality of single day exposure to particulate matter using various modelling strategies. These models are adjusted for long-term trends and seasonal variations in daily mortality and the confounding effects of weather on mortality.

Chapter 7 analyses the second objective of this research. This chapter explains the concept of short-term mortality displacement and the method used to analyse it. It examines if any association between short-term exposure to air pollution and daily mortality is due to mortality displacement by a few days.

Building on the methods used in Chapter 6, the extended effects of particulate matter on daily mortality are analysed in Chapter 8, the third and fourth objectives of this research. The estimates of the total effects of particulate matter distributed over several days, using polynomial distributed lag models are presented and the shape of the distribution of effects of particulate matter on daily mortality over lag days is explored.

While Chapters 6, 7 and 8 describe the analysis of the associations between particulate matter and mortality at short to mid-term time scale, Chapter 9 provides the analysis of the association between annual average particulate matter and annual mortality, which is the fifth objective of this research. Instead of the regularly monitored temporal data used in Chapters 6, 7 and 8, spatial data are used in Chapter 9. Annual average spatial air pollution exposure estimates are derived from TAPM (The Air Pollution Model).

In Chapter 10, the findings of this study and their implications are discussed. Various issues related to data, method and the study's limitations are also discussed in this chapter. Chapter 11 summarises the main findings of this research.

Chapter 2: Literature review

2.1 Introduction

Striking increases in mortality following a series of high air pollution episodes in the United States and Europe in the mid-twentieth century have shown that air pollution at high concentrations causes excess deaths (Roholm, 1937; UKMoH, 1954; Ciocco and Thompson, 1961; Nemery, Hoet and Nemmar, 2001). As high air pollution episodes became less frequent and air pollution concentrations during the episodes became relatively lower than in the earlier episodes, the focus has shifted from air pollution episodes to time series studies. Several time series studies conducted both at a single city and across multiple cities have provided evidence of positive associations between daily air pollution levels and daily mortality/morbidity (Schwartz, 1993; Schwartz, Dockery and Neas, 1996; Katsouyanni *et al.*, 1997; Kelsall *et al.*, 1997; Michelozzi *et al.*, 1998; Morgan *et al.*, 1998; Ostro *et al.*, 1999; Lee *et al.*, 2000; Samet *et al.*, 2000c; Zanobetti *et al.*, 2002). Besides time series studies, which only demonstrate the effect of short-term exposure to air pollution, cohort studies, which can document the longer term effect of air pollution on mortality, have also shown that air pollution causes excess deaths in the long run (Dockery *et al.*, 1993; Pope *et al.*, 1995; Pope *et al.*, 2002; Nafstad *et al.*, 2004).

Although it is well understood that there is an association between air pollution and daily mortality, there are still a number of issues and uncertainties associated with air pollution epidemiological studies such as the choice of statistical modelling, the role of short-term harvesting in the association between air pollution and mortality, the concentration-response relationship, the threshold levels, and whether air pollution is modified by certain factors. Many studies have attempted to address one or more of the above issues. This literature review summarises the literature relevant to the association between air pollution and mortality including those studies, which have discussed the above issues. A major focus of this literature review is the time series studies of the effect of air pollution on mortality.

2.2 Statistical modelling in time series studies of particulate matter and health

Time series studies are the most commonly used epidemiological studies to study the association between air pollution and mortality/morbidity. These studies evaluate the

effects of exposure to air pollution over a short period of time typically one day to several days. They relate the change in health outcomes, usually the daily counts of mortality or hospital admissions, to the change in air pollution exposure levels on the same day or over the last few days.

In time series study design, the relationship between air pollution and health outcomes is confounded by other variables that vary on short time scale. As population characteristics like age, gender, smoking habit, socioeconomic class do not vary from day-to-day, these variables do not confound the short-term association between air pollution and health. Variables like temperature, relative humidity and epidemics, which vary on short time scale, are most likely to be confounders in time series studies. For example, number of deaths generally peaks during the winter months when temperature falls. PM₁₀ levels in a region like Christchurch are higher in the winter months than the rest of the year. Although PM₁₀ is not the only risk factor contributing to higher deaths in the winter months, the increased mortality may be wrongly linked only to PM₁₀, which will result in an overestimation of the effect of PM₁₀ on mortality if the confounders such as season and temperature are not adequately adjusted for. The pollutant variables other than the pollutant variable of interest might also confound the true association between pollutant and health outcomes. For example, PM₁₀ and CO levels are highly correlated and peak during the winter months. Thus, the likely effect of CO on mortality may contribute to the association between the variable of interest PM₁₀ and mortality resulting in an exaggerated effect of PM₁₀ if the confounding effect of CO is not adequately adjusted for.

Time series analysis estimates an increase in health outcomes, usually a number of deaths or hospital admissions, associated with a unit increase in air pollution concentrations controlling for confounders such as season, long-term trend, weather variables, influenza epidemics, etc. The typical unit of analysis in time series analysis are days. Regression models are fitted with the number of daily health outcomes as the response variable and daily air pollution levels and weather data as explanatory variables. These models can also include additional terms for other confounders like other pollutants.

Early time series air pollution and health effects studies assumed that the response variable (daily number of deaths or hospital admissions) was approximately distributed as a normal variable (or could be transformed to an approximately normally distributed variable with a log or a square root transformation) and used linear regression methods for analysis (Martin, 1964; Schimmel and Greenburg, 1972;

Mazumdar, Schimmel and Higgins, 1982; Ostro, 1984; Schwartz and Marcus, 1990; Kinney and Ozkaynak, 1991).

Long-term and seasonal trends were generally adjusted for by filtering out the long-term variation in data before analysis and fitting the models for filtered data rather than the original time series data. A few earlier studies filtered out the long-term variation in data by calculating the deviations in daily data from its 15-day centred moving average (Martin, 1964; Schimmel and Murawski, 1976; Ostro, 1984). An air pollution and mortality study in New York City regressed the time series data on a series of harmonic waves (sine and cosine curves) with different frequencies and used the residuals from this model as filtered data series in the subsequent analysis (Schimmel and Greenburg, 1972). The filtered data series represented only short-term fluctuations in the original series and hence were used to study the association between short-term changes in daily deaths and short-term changes in air pollution and weather variables. Schwartz & Marcus (1990) further refined the analysis to control for the short-term autocorrelations in the data by fitting autoregressive models instead of linear regression models to analyse air pollution and mortality in London during the winters of 1958 to 1972 (Schwartz and Marcus, 1990).

Since a very small proportion of the population dies or are admitted to hospital in any given day, daily mortality / hospital admission data are rare events. They are count data as they only take non-negative values. The counts of rare events are usually modelled as a Poisson variable. Hence, the time series studies of air pollution and mortality have later recognised that it is more appropriate to model the daily mortality/hospital admission data as a Poisson variable than a Gaussian variable. Studies then started using Poisson regression models instead of linear regression (Schwartz, 1991; Dockery, Schwartz and Spengler, 1992; Schwartz *et al.*, 1996). Schwartz *et al.* (1996) supported the use of linear regression in the earlier studies of the London winters arguing that the mean number of deaths in London winters was so high that it allowed a Gaussian approximation to a Poisson process. However, they also suggested that one should use Poisson regression for the studies with a fewer number of daily deaths (Schwartz *et al.*, 1996).

When Gaussian data are linearly filtered, the resulting data series is still Gaussian which allows fitting linear regression models for the filtered data series. But the filtered Poisson data is not Poisson. Hence, pre-filtering the data to remove a long-term variation before fitting models is not possible if the outcome variable is to be modelled as a Poisson variable (Schwartz *et al.*, 1996). In order to solve this problem,

studies added filters in the regression models to control for the long-term trend and seasonal variations instead of pre-filtering data series. This is similar to adding variables to control for confounding variables in the regression models.

Studies often used calendar time or one or more functions of calendar time or their combinations as predictors in regression models to control for the long-term trend and seasonal variations in daily mortality / morbidity. Calendar time was often used as a linear predictor of daily mortality / morbidity in regression models (Schwartz, 1991). However, a study of air pollution and daily mortality in Cincinnati, Ohio showed that a quadratic term of calendar time along with a linear term might be more appropriate (Schwartz, 1994c). These studies treated the calendar time as a continuous variable. The other approach is to divide calendar time into categorical variables representing months, years and seasons and use them in the model. Dummy variables for months, years and seasons are created and included as predictors in the regression models. This approach models daily mortality / morbidity as a step function of calendar time.

Schwartz (1991) created dummy variables for each year of the study and used them in the Poisson regression model in addition to a linear term for the calendar time (Schwartz, 1991). In some studies, dummy variables for month of year have been used to control for seasonal variations in daily mortality (Ostro *et al.*, 1996). As the seasonal pattern may vary from year to year, some studies created dummy variables for each month of the study (Schwartz, 1994c; 1994b; Morgan *et al.*, 1998). Some studies created dummy variables for seasons instead of months and put them in the regression models to control for seasonal variations (Schwartz and Dockery, 1992; Ostro *et al.*, 1999).

Harmonic waves or trigonometric filtering were used in some studies to remove the long-term seasonal and cyclical patterns in data (Kinney and Ozkaynak, 1991; Schwartz, 1993). A sequence of sine and cosine functions of time of year was added in regression models to model data as harmonic waves. In order to control for the complex seasonal patterns in data, studies used the sum of harmonic waves of increasing frequencies in the models. The Air Pollution and Health: A European Approach (APHEA) project, supported by the European Commission to study the short-term health effects of air pollution in 15 European cities, used this protocol to control for seasonal variations in daily mortality / morbidity. The protocol was to include up to six sinusoidal terms for periods of 12 months, 6 months, 4 months, 3 months, 73 days, and 2 months in the regression model so that it could pick up the patterns of up to two months and only the short-term variation of less than 2 months in mortality and air

pollution data series would be used to estimate the association between air pollution and mortality. The maximum number of sinusoidal terms varied for individual cities which was based on goodness of fit test, inspection of time series plots and residual periodograms (Katsouyanni *et al.*, 1995; Katsouyanni *et al.*, 1996; Spix and Wichmann, 1996; Sunyer *et al.*, 1996; Touloumi, Samoli and Katsouyanni, 1996; Wojtyniak and Piekarski, 1996; Zmirou *et al.*, 1996; Katsouyanni *et al.*, 1997; Touloumi *et al.*, 1997). In addition to the APHEA cities studies, a few other studies including studies in Australia and New Zealand also followed the same protocol to control for seasonal variations in daily mortality and hospital admissions (Simpson *et al.*, 1997; Hales *et al.*, 2000; Denison *et al.*, 2001a; Petroschevsky *et al.*, 2001).

Generalised linear models with parametric splines, such as natural cubic splines, and generalised additive models with non-parametric splines, such as smoothing splines or lowess smoothers, are the most commonly used statistical modelling techniques in the recent time series studies. These models are more flexible than earlier approaches in their assumptions about a long-term trend and seasonal variations in daily health outcomes, and about the associations of weather variables with health outcomes (Bell, Samet and Dominici, 2004).

2.3 Time series studies of particulate matter and mortality

The association between particulate matter and mortality has been reported by numerous time series studies in a number of cities in the USA, Canada, Europe, Australia, and other parts of the world (Schwartz, 1993; Ostro *et al.*, 1996; Kelsall *et al.*, 1997; Simpson *et al.*, 1997; Burnett *et al.*, 1998; Morgan *et al.*, 1998; Prescott *et al.*, 1998; Ostro *et al.*, 1999; Hales *et al.*, 2000; Hoek *et al.*, 2000). The modelling techniques of these time series analyses are not restricted to any one particular technique. With new methodology development, the statistical modelling approach for time series analysis shifted from simpler to more advanced methods as discussed in Section 2.2. Irrespective of the statistical analysis methods, almost all of these studies have provided evidence of an association between PM_{10} and mortality.

One earlier time series study conducted in New York City using data from 1963 to 1968 (Schimmel and Greenburg, 1972) and the extension of this study covering the data up to 1972 (Schimmel and Murawski, 1976) examined the association between daily premature deaths and daily ambient levels of SO_2 and smoke shade, a measure of particulate pollution, using an ordinary linear regression modelling method. They reported associations between air pollution levels and total mortality, respiratory

mortality and cardiac mortality. The percentage of premature deaths attributed to air pollution was reported to be about 3% after adjusting for temperature.

There were only a few studies using time series approach before the 1980s. A new wave of time series studies have begun with advances in statistical methods and software in late 1980s and early 1990s (Bell, Samet and Dominici, 2004). Using a wide variety of statistical approaches, these studies have reported an association between daily particulate matter and daily mortality in different geographic areas such as the US, Europe and other parts of the world. The association is generally positive and statistically significant in the majority of the air pollution and mortality studies. There are, however, a few studies which have found a negative, but inconclusive, association between daily particulate matter and daily mortality (Lee, Shin and Chung, 1999; Laden *et al.*, 2000; Anderson *et al.*, 2001).

The United States Environmental Protection Agency (USEPA) undertook two major reviews, one in 1996 and another in 2004, of epidemiological studies of human health effects associated with ambient particulate matter to issue air quality criteria for particulate matter (US Environmental Protection Agency, 1996; 2004). The 1996 Air Quality Criteria Document for Particulate Matter reviewed 35 PM₁₀ and mortality time series studies published between 1988 and 1996, and the 2004 Air Quality Criteria Document for Particulate Matter reviewed the studies, whose results became available (published or accepted for publication) between 1996 and 2003. The 1996 report concluded that the total acute non-accidental mortality relative risk estimate associated with an increase of 50 µg/m³ in daily PM₁₀ concentrations was in the order of 1.025 to 1.05 in the general population (US Environmental Protection Agency, 1996). This is same as an increase of 0.5% to 1% in total acute non-external deaths for a 10 µg/m³ increase in daily PM₁₀ concentrations. The PM₁₀ effect size estimates for total non-external deaths reported in the studies assessed in the 2004 review were generally in the range of 0.4% to 0.7% for a 10 µg/m³ increase in daily PM₁₀ concentrations, which were within but towards the lower end of the range of PM₁₀ effect estimates reported in the 1996 Air Quality Criteria Document for Particulate Matter (US Environmental Protection Agency, 2004).

In addition to the reviews carried out by the USEPA, a number of publications have critically reviewed air pollution epidemiological studies (Dockery and Pope, 1994; Schwartz, 1994a; Pope, Dockery and Schwartz, 1995). A review of reviews, which critically assessed some 15 reviews of the published studies studying the short-term effects of air pollution on mortality and morbidity reached the conclusion that the short-

term relation between air pollution and mortality and/or morbidity reported by many studies were valid and causal (Dab *et al.*, 2001). These studies generally reported an increase of 1% in total non-external deaths in the range of 0.7% to 1.6% for an increase of each 10 $\mu\text{g}/\text{m}^3$ in daily PM_{10} levels (Pope, Dockery and Schwartz, 1995).

2.4 Multicity studies

A wide variety of statistical models have been used to estimate the association between air pollution and health. Statistical models used in single city studies are not consistent. The choice of statistical modelling and controlling for confounders depends upon researchers' preference. Because of the heterogeneity of the statistical approach used, the validity of the findings from single city studies has been questioned. Critics found that the findings among single city studies were not consistent, and even analyses in the same city gave inconsistent results when the data were reanalysed independently (Lipfert and Wyzga, 1995). They questioned whether models that biased the effect estimate upwards had been selected in reporting the results (Dominici, 2002). Multicity studies addressed these criticisms of single city studies (Katsouyanni *et al.*, 1997; Samet *et al.*, 2000c).

The idea of multicity studies is to analyse data under the same framework in individual participating cities so that the results are comparable across different geographic locations. The multicity study follows a similar protocol for data handling and analysis, which is one of the major advantages over meta-analysis of independent studies. Due to a wide variety of exposure levels and different geographic locations of different cities in multicity studies, the results from these studies can provide evidence of consistency or heterogeneity in the effects of PM_{10} on mortality in individual cities. Further analysis can identify potential effect modifiers of the PM_{10} mortality association across different geographic locations. In addition, multicity studies suffer less from publication bias as these studies report the results from all cities participated in the study, irrespective of whether the results are statistically significant or not in the individual cities.

The APHEA project is a multicity study of the short-term effects of air pollution on mortality and hospital admissions in European cities. The first study included 15 European cities, and the second stage of the project (APHEA2 project) included 29 cities and a more recent study period (Katsouyanni *et al.*, 1995). City specific analysis showed associations between air pollution and non-external mortality, respiratory mortality in Paris and Lyon, France (Dab *et al.*, 1996; Zmirou *et al.*, 1996), Koln,

Germany (Spix and Wichmann, 1996), Barcelona, Spain (Sunyer *et al.*, 1996), and Athens, Greece (Touloumi, Samoli and Katsouyanni, 1996).

Katsouyanni *et al.* (1997) analysed data from 12 APHEA cities and pooled together the results from individual cities to get an overall estimate of the short-term effects of air pollution on daily non-external mortality across the cities. They reported a difference in the effects between the western European cities and the central eastern European cities, with higher effects in the western European cities. In the western European cities, they found a 3% increase (95% CI: 2%, 4%) for each 50 $\mu\text{g}/\text{m}^3$ increase in daily SO_2 or black smoke (BS) and a 2% increase (95% CI: 1%, 3%) for each 50 $\mu\text{g}/\text{m}^3$ increase in daily PM_{10} . In the central eastern European cities, the increases in daily mortality for 50 $\mu\text{g}/\text{m}^3$ increase in daily SO_2 and BS were respectively 0.8% (95% CI: 0.1%, 2.4%) and 0.6% (95% CI: 0.1%, 1.1%). They argued that the inconsistency in the results between the two groups of European cities could be due to the different pollutant toxicity or mix because of sources of pollutants, differences in sensitive sub-populations and differences in exposure levels (Katsouyanni *et al.*, 1997).

Samoli *et al.* (2001) reanalysed the APHEA data using generalised additive models (GAM) with LOESS smoothing terms for seasonal trend and weather variables instead of using sine/cosine used in the APHEA protocol in order to investigate the regional differences in the short-term association between air pollution and mortality. They found higher relative risks than those reported by Katsouyanni *et al.* (1997) for the central and eastern European cities but a similar result for the western European cities. When they restricted the analysis to the days with BS levels less than 150 $\mu\text{g}/\text{m}^3$, the differences in the effects between the two regions were further reduced. They argued that the statistical approach used in the previous study and the inclusion of days with higher pollutant levels in the analysis caused part of the heterogeneity in the estimates of air pollution effects in the two European regions (Samoli *et al.*, 2001). Data were further analysed using generalised additive models with more stringent convergence criteria and generalised linear models with natural splines smoothing (Samoli *et al.*, 2003). They found that the difference in the air pollution effects in mortality between western and central, and eastern European cities was less clear.

As part of the APHEA2 project, Katsouyanni *et al.* (2001) analysed data from 29 European cities using GAM with non-parametric LOESS smoother to control for seasonal trend and weather variables, and further reanalysed using GAM with more stringent convergence criteria, and with two parametric approaches (natural splines and penalized splines smoothing) to control for seasonal trend and weather variables

(Katsouyanni *et al.*, 2001; Katsouyanni *et al.*, 2003). They used a hierarchical modelling approach. First the regression models were fitted in individual cities and then the results were pooled together in the second stage analysis to estimate an overall relative risk across all 29 cities, and to investigate potential effect modifiers. The second stage analysis adjusted for potential effect modifiers like air pollution level and mix; climatic variables in different cities; health status of the population and geographic area (Katsouyanni *et al.*, 2001; Katsouyanni *et al.*, 2003). They reported a 0.62% (95% CI: 0.4%, 0.8%), 0.59% (95% CI: 0.4%, 0.8%), 0.41% (95% CI: 0.2%, 0.6%), and 0.55% (95% CI: 0.4%, 0.7%) increase in daily mortality for 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} using GAM default criteria, stringent criteria, natural spline and penalized spline respectively.

Another multicity study, the National Morbidity, Mortality and Air Pollution Study (NMMAPS) funded by the Health Effects Institute, studied the effects of short-term exposure to PM_{10} on mortality and hospital admissions in the 90 largest US cities during 1987-1994 (Samet *et al.*, 2000b; Samet *et al.*, 2000c). As part of this study, data were initially analysed for the 20 largest US cities (Samet *et al.*, 2000a). The estimated increase in the relative rate of non-external causes mortality for each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} level was 0.51% (95% posterior interval: 0.07%, 0.93%). Analysis was first conducted in individual cities and the overall estimate of the relative rates of mortality associated with pollutants was calculated using hierarchical regression models.

Dominici *et al.* (2000) analysed the NMMAPS data for the 20 largest US cities in more detail using a Markov Chain Monte Carlo (MCMC) algorithm with a block Gibbs sampler to approximate the posterior distribution in the second stage analysis of pooling the city specific estimate together. They also considered spatial models in which the relative risks in closer cities were assumed to be more correlated (Dominici, Samet and Zeger, 2000).

The methods developed for the analysis of the 20 largest US cities were applied in the analysis of 90 largest US cities. In second stage analysis, the heterogeneity in the effect estimates in individual cities was evaluated with city or region specific explanatory variables. The analysis used five types of city specific variables: mean pollution and weather levels; crude mortality rate; % not graduating from high school and median household income (socio-demographic variables); % of public transport (urbanization); and variables related to measurement error (Samet *et al.*, 2000c; Dominici *et al.*, 2002a).

Both the original and reanalysis of the 90 largest US cities showed significant evidence of the combined effect of short-term exposure to PM_{10} on mortality at all lags

(0, 1 and 2 day lags were examined). However, the effect of 1-day lagged PM₁₀ was the largest. When the data were reanalysed using a stricter convergence criteria in GAM function, the combined effect across the 90 cities at lag 1 dropped to a 0.27% increase from a 0.41% increase (original analysis) in total non-external mortality for every 10 µg/m³ increase in PM₁₀. The updated increase in the relative rate of non-external mortality for each 10 µg/m³ increase in PM₁₀ using GLM with natural cubic spline was 0.21% (95% posterior interval: 0.1%, 0.3%) (Samet *et al.*, 2000c; Dominici *et al.*, 2003b).

Besides the APHEA and NMMAPS, several other studies have analysed the effects of daily levels of particulate matter on mortality across multiple cities and found positive associations between particulate matter and mortality (Laden *et al.*, 2000; Moolgavkar, 2000; Schwartz, 2000a; 2000b; Schwartz and Zanobetti, 2000; Zanobetti and Schwartz, 2000; Braga, Zanobetti and Schwartz, 2001; Schwartz *et al.*, 2001; Burnett and Goldberg, 2003; Schwartz, 2003a; 2003b).

2.5 Problem with generalised additive model

The generalised additive model (GAM) has been the most widely used method in both single city and multicity time series studies of air pollution and mortality / morbidity as it allows for the adjustment of the non-linear confounding effects of trends, seasonality and weather variables non-parametrically (Samet *et al.*, 2000c; Samoli *et al.*, 2001). The most appealing feature of GAM is that it does not require a strong assumption about the functional relationship of mortality with the confounders such as temperature and relative humidity, which gives a greater degree of confidence against model misspecification (Lumley and Sheppard, 2003).

Most studies including the NMMAPS and APHEA2 applied GAM using the Splus function **gam** with its default convergence criteria (Samet *et al.*, 2000c; Simpson *et al.*, 2000; Katsouyanni *et al.*, 2001). It was later found that with the default convergence criteria, Splus (Version 3.4) function **gam** produced a biased estimate of the relative risk of mortality for air pollution. Dominici *et al.* (2002c) showed with a simulation that when the size of risk estimates are small and confounding variables are controlled using at least two non-parametric smoothers in the model, Splus (Version 3.4) function **gam** with default convergence criteria overestimates the risk estimates. They also reported that the **gam** function with stricter convergence criteria and the generalised linear model with parametric nonlinear adjustment (natural spline smoother) gave similar relative risk estimates (Dominici *et al.*, 2002c).

In addition, Ramsay et al. (2003) showed with simulations that if there is concavity (the non-parametric analogue of multicollinearity) in the data, GAM fitting could underestimate the standard error of the parameter estimates (the relative rate estimate), which may cause type 1 error and the parameter estimates may become statistically significant (Ramsay, Burnett and Krewski, 2003). For a more robust assessment of uncertainty of parameter estimates, a Splus function **gam.exact** has been developed which asymptotically computes the exact standard errors for each linear term in the model (except for the intercept) (McDermott, 2003a; 2003b).

Due to the problems observed in **gam** function and the dependence of the results on the choice of model (**gam** or **glm**), researchers were cautioned against choosing any particular model as a correct model and advised to explore the sensitivity of findings to model specification and to the degree of adjustment for confounding variables (Samet *et al.*, 2003).

Following the findings of Dominici et al. (2002b) that the **gam** function in Splus (Version 3.4) software with default convergence criteria overestimated the relative risk estimates for air pollution, NMMAPS data were reanalysed using **gam** with default convergence criteria; **gam** with stricter convergence criteria and the Poisson regression model with parametric non-linear adjustments for confounding factors (**glm** with natural cubic splines). When the default convergence criteria was implemented, the national average excess risk estimate for non-external mortality across 90 cities per 10 $\mu\text{g}/\text{m}^3$ in PM_{10} at lag 1 was 0.41%, which dropped to 0.27% when the stricter convergence criteria was used. Use of **glm** with natural cubic splines further reduced this estimate to 0.21% (Dominici *et al.*, 2002b).

The reanalysis of more than 35 published time series studies of air pollution and mortality and morbidity, which had earlier used **gam** with default convergence criteria, using **gam** with stricter convergence criteria and **glm** with parametric smoother reported new relative risk estimates for air pollution which were lower than what had been reported earlier. Although the new relative risk estimates were lower, the reanalysis did not qualitatively change the original findings that there was a positive association between air pollution and mortality and morbidity (Health Effects Institute, 2003).

2.6 Case-crossover study design

Time series studies have analysed the short-term association between daily PM_{10} and mortality/morbidity using analytical techniques ranging from simple to very sophisticated to control for long-term trends and seasonal variations in daily

mortality/morbidity and the confounding effects of weather variables and gaseous air pollutants (Schimmel and Greenburg, 1972; Ostro, 1984; Schwartz and Marcus, 1990; Schwartz, 1991; 1994c; Katsouyanni *et al.*, 1995; Katsouyanni *et al.*, 1996; Schwartz *et al.*, 1996; Morgan *et al.*, 1998; Ostro *et al.*, 1999; Dominici, Samet and Zeger, 2000; Samet *et al.*, 2000c; Katsouyanni *et al.*, 2001; Samoli *et al.*, 2001; Katsouyanni *et al.*, 2003). These methods generally involve fitting functional relationships of daily mortality/morbidity with the confounders and may require assumptions about the relationships. For example, in multi-pollutant models, the confounding effects of co-pollutants are usually adjusted for by assuming a linear relationship of mortality with each pollutant (Morgan *et al.*, 1998; Fairley, 1999; Chock, Winkler and Chen, 2000; Samet *et al.*, 2000a). If the association of mortality with any one pollutant is non-linear, confounding by co-pollutants may not have been adequately controlled for in the models and thus the models may not correctly estimate the independent mortality effects of pollutants (Schwartz, 2004).

One of the traditional approaches to control for potential confounding in epidemiology is matching (Szklo and Nieto, 2000). If, in a case-control study, the cases and controls are matched on a potential confounder, the analysis will not have to adjust for that confounder. The study design will itself control for the confounder. The case-crossover study design, introduced by Maclure (1991), is an adaptation of the case-control design in which each person who had an event (case) serves as his/her own control (Maclure, 1991). Using time series data, this study design can be used to investigate the acute effects of exposure. Each person who had an event at a certain time (case day) is matched with him/herself on a nearby time when he/she did not have the event (control days) and their personal covariates and environmental exposures on a case day are compared with those of control days (Neas, Schwartz and Dockery, 1999). All personal attributes such as age, smoking habit, usual diet, socioeconomic status etc. that change slowly over time can be adjusted for by selecting nearby control days.

The study design controls for a long-term trend and seasonal variations in daily mortality by selecting control days close to the case day, i.e. within a few days apart when using daily time series data (Bateson and Schwartz, 1999). This is one of the major advantages of case-crossover design over time series studies which use smooth functions such as natural cubic splines or lowess smoothers or other complex functions to control for them. Once all potential control days close enough to each case day to control for seasonal confounding are identified, the subset of control days that also matches on the level of environmental exposures such as temperature, relative humidity

and co-pollutants, and epidemics can be selected for each case day. This controls for the confounding effects of weather and co-pollutants by the study design without requiring complex modelling of the relationships between confounders and the health outcomes.

However, as the number of confounders increases, the number of potential control days that can be used to control for confounders by matching will reduce and thus, the power to detect any association will be reduced. This limits the applicability of this study design in single city studies (Bateson and Schwartz, 2001; Schwartz, 2004).

A number of studies have adopted this study design to analyse the acute effects of air pollution and have shown positive associations between air pollution and mortality and morbidity (Lee and Schwartz, 1999; Neas, Schwartz and Dockery, 1999; Sunyer *et al.*, 2000; Schwartz, 2004). This study design has also been used by two recent Australian and New Zealand multicity studies to analyse the associations of outdoor air pollution with hospital admissions. They have reported a positive association between outdoor air pollution and cardiovascular hospital admissions for the elderly (Barnett *et al.*, 2006) and a positive association between outdoor air pollution and respiratory hospital admissions for children (Barnett *et al.*, 2005).

2.7 Shape of dose–response relationship

The dose-response relationship is the relationship between the dose and the proportion of individuals in an exposed group that develop a specific effect due to exposure (Yassi *et al.*, 2001). The dose-response relationship is of particular importance in public health as it provides the foundation for setting safety standards.

The study of the shape of the concentration-response relationship between daily mortality and daily levels of particulate matter (PM) has been motivated by the findings of many studies that there is a positive association between daily mortality and daily PM levels even at the lower levels of concentrations (Ostro, 1984; Brunekreef, Dockery and Krzyzanowski, 1995; Schwartz, 2000a). The findings of a higher relative risk of mortality for unit change in daily PM levels at the lower levels than at the higher levels have generated more interest in the shape of the relationship between daily mortality and daily PM levels (Schwartz and Marcus, 1990; Schwartz, 2000a).

Although it is very common to assume a no threshold log-linear relation between daily mortality and daily PM concentrations in time series studies of the short-term effects of particulate matter on daily mortality, a few single city studies have explored the shape of the dose-response relationship between particulate matter and mortality by replacing a linear term for particulate matter with a non-parametric smooth function in

the log-linear Poisson regression models (Schwartz, 1993; 1994c; Pope and Kalkstein, 1996; Burnett *et al.*, 1998). These studies have found the shapes of the estimated concentration-response relationship approximately linear without well-defined thresholds.

The shape of exposure-response relationship has also been explored using data from multicity studies which has enhanced the statistical power to analyse the shape and its generalisability (Pope, 2000). Daniels *et al.* (2000) (reanalysis Dominici *et al.* (2003a)) analysed NMMAPS data for the 20 largest US cities to study the shape of the concentration-response relationship between daily PM₁₀ and daily mortality. They fitted several log-linear Poisson regression models in each city using (a) a linear term for PM₁₀; (b) a natural cubic spline with knots at 30 and 60 $\mu\text{g}/\text{m}^3$ which are approximately the 25th and 75th percentile of the distributions of PM₁₀ for many of the 20 cities; (c) a threshold model with possible thresholds between 5 $\mu\text{g}/\text{m}^3$ and 200 $\mu\text{g}/\text{m}^3$ with an increment of 5 $\mu\text{g}/\text{m}^3$, and combined the results across the cities. Comparison of the models within each city and over all cities based on the Akaike's Information Criteria (AIC) showed that the model with a linear term for PM₁₀ better fitted the data than the threshold and the natural spline models for all cause mortality and for cardiovascular and respiratory mortality combined. The results using the natural spline model showed that for total and cardiorespiratory mortality, the spline curves were nearly linear down to the lowest PM₁₀ level (Daniels *et al.*, 2000; Dominici *et al.*, 2003a).

Schwartz & Zanobetti (2000) conducted what they called a "meta-smoothing" analysis using daily mortality and pollution data from 10 US cities. They fitted generalised additive Poisson regression models using a smoothed function of PM₁₀ controlling for other confounders in each city and computed the predicted values of the log relative risk of daily mortality in each city for 2 $\mu\text{g}/\text{m}^3$ increments of PM₁₀. These predicted values at each increment were then combined across the 10 cities using inverse variance weighting. The estimated combined 10-city concentration-response relation was approximately linear down to the lowest PM₁₀ level observed suggesting a log-linear relationship without any threshold between daily PM₁₀ level and daily mortality (Schwartz and Zanobetti, 2000). Similar concentration-response relationships have also been reported between daily concentrations of black smoke and daily deaths in the eight Spanish cities study and between daily deaths and total PM_{2.5} and traffic related PM_{2.5} in the Harvard Six Cities study (Schwartz *et al.*, 2001; Schwartz, Laden and Zanobetti, 2002).

A recently published Cook County study (1987-1994) explored the concentration-response function between daily PM₁₀ and daily mortality by constraining the relationship to be biologically plausible i.e. the relative risk of mortality as a non-decreasing function of PM₁₀ concentrations. The study used piecewise linear regression with one and two change points to constrain the shape of the concentration-response relationship. By simulation, it first showed that constraining the concentration-response relationship to the biologically plausible gave a higher statistical precision of the estimates. Application of this method to real Cook County data did not provide evidence against a linear dose-response relation (Roberts, 2004). A study in Birmingham, Alabama (1985-1988) modelled the data using piecewise linear regression models and B-spline models to model non-linear effects of PM₁₀ on mortality and to find a threshold in the relationship between PM₁₀ and mortality. None of the modelling results provided statistically significant evidence either for a nonlinear concentration-response relationship or for a threshold effect (Smith *et al.*, 2000a).

Although a study in Phoenix, Arizona (1995-1998) did not find any evidence against a linear concentration-response relationship between daily coarse particles and mortality, it provided evidence of a non-linear relationship between daily fine particles and mortality, with a change of slope occurring at somewhere in the range of 20 to 25 µg/m³ (Smith *et al.*, 2000b). Unlike other studies which compared the effects of fine and coarse particles on daily mortality, this study found a statistically significant association between coarse particles and mortality but not between fine particles and mortality when a linear concentration-response relationship was assumed (Smith *et al.*, 2000b). Being based on a single city study for a relatively short-time period, the generalisability of these results is very limited.

Except for a few single city studies, a number of studies including large multicity studies provided no evidence against a linear dose-response relationship between daily particulate matter and mortality. As such, in most studies, the exposure-response relationship has been approximated to a straight-line relationship.

2.8 Threshold

A threshold is a dose level below which no effect on individual health occurs (or is observed). For many environmental hazards, the dose needs to reach a specific level before the effects on people start (Yassi *et al.*, 2001). The question of whether a threshold level exists below which air pollution has no effect in the population is of particular concern in air pollution studies. Identifying a threshold level has major policy

implications. It would be expected that there will be no additional public-health benefit from bringing air pollution far below this level (Brunekreef and Holgate, 2002).

It would be technically difficult to detect the threshold concentrations for mortality effect, even when they exist, from epidemiological studies because of inter-individual differences in susceptibility as well as intra-individual variability over time. The concentration level that might kill an individual at one time would not do so at another time as the individual's susceptibility to particulate matter is likely to change over time due to disease state and other physiological conditions and environmental stresses (US Environmental Protection Agency, 1996). Similarly, the individual's differences in threshold level for serious effects from particulate matter due to individual differences in pre-existing disease conditions and genetic factors makes it difficult to detect threshold concentrations at the population level (Schwartz and Zanobetti, 2000). Despite the difficulty in identifying a threshold level, a few studies have analysed whether thresholds exist for health effects of particulate matter.

In one earlier time series analysis, Ostro (1984) analysed London data for 14 winters from 1958 to 1972 to test for the existence of a threshold level in the relationship between BS and mortality. There was a statistically significant effect of BS on mortality below the hypothesized threshold level of $150 \mu\text{g}/\text{m}^3$ for BS, which showed no evidence of threshold level at $150 \mu\text{g}/\text{m}^3$ (Ostro, 1984). By presenting the same data graphically, Schwartz & Marcus (1990) showed a curvilinear relation between BS and mortality with no threshold. They also observed steeper slopes at lower air pollution levels than at the higher levels (Schwartz and Marcus, 1990).

In his attempt to detect a threshold level for PM_{10} effect, Schwartz (2000a) analysed the data from 10 US cities that had approximately daily PM_{10} levels by limiting the analysis to the days with PM_{10} below $50 \mu\text{g}/\text{m}^3$ and combined the results of 10 cities. If the threshold level was above $50 \mu\text{g}/\text{m}^3$, the slope of PM_{10} on daily deaths would be expected to be approximately zero in the restricted analysis. If the threshold level was below $50 \mu\text{g}/\text{m}^3$, the effect size would be expected to be smaller than the effect size from the analysis with all days as a large fraction of the days would be below the threshold levels in the restricted analysis. He reported a greater relative risk of mortality for $10 \mu\text{g}/\text{m}^3$ change in daily PM_{10} concentrations in the restricted analysis than in the analysis with all days. This showed no evidence of threshold in the relationship between PM_{10} and daily mortality (Schwartz, 2000a).

Daniels et al. (2000) (reanalysis Dominici et al. (2003a)) analysed NMMAPS data for the 20 largest US cities using regression splines to examine the presence of a

threshold for PM₁₀. They found no evidence of a threshold in the association between daily PM₁₀ and daily total and cardiorespiratory causes of deaths. However, for non-cardiorespiratory mortality, a threshold of 50 µg/m³ was reported (Daniels *et al.*, 2000; Dominici *et al.*, 2003a). Schwartz and Zanobetti (2000) also found no evidence of threshold in their analysis of 10 US cities, which had daily measurements of PM₁₀. They analysed the concentration-response relationship between PM₁₀ and daily deaths by modelling the logarithm of daily deaths as a smooth functions of PM₁₀ after adjusting for other confounders in each city and combined the results across the cities. They found an association between PM₁₀ and daily mortality in the entire range of PM₁₀ levels observed in the study (Schwartz and Zanobetti, 2000). Using a similar method, the eight Spanish cities study also reported no evidence of threshold for black smoke (Schwartz *et al.*, 2001). The analysis of Harvard Six Cities data also showed no evidence of threshold level for either total PM_{2.5} or traffic related PM_{2.5} (Schwartz, Laden and Zanobetti, 2002).

Although a number of large multicity studies suggested there was no threshold in the relationship between particulate matter and mortality, one study in Phoenix, Arizona (1995-1998) found a threshold most likely to be in the range of 20 to 25 µg/m³ for the effect of fine particles (PM_{2.5}) on mortality. The study analysed data using a piecewise linear model in which several possible thresholds were specified and using a B-spline model with 4 knots. Both methods provided an evidence of a threshold for fine particles. However the study did not find any evidence of a threshold for coarse particles (PM_{10-2.5}). Using a no threshold model, the study only found a statistically significant association between coarse particles and mortality but not between fine particles and mortality (Smith *et al.*, 2000b).

The threshold level, if it exists, along with the exposure-response relation and the distribution of exposure can be used to estimate the public health impact of air pollution. Kunzli *et al.* (2000) applied a threshold level of 7.5 µg/m³ for PM₁₀ effect in their calculation of public health impact of outdoor and traffic related air pollution in three European countries: France, Switzerland and Austria (Kunzli *et al.*, 2000). In a similar calculation of the number of deaths attributed to air pollution in New Zealand, Fisher *et al.* (2002) used no threshold and 5, 7.5 and 10 µg/m³ threshold levels for the effect of long-term exposure to PM₁₀ on mortality as different scenarios (Fisher *et al.*, 2002).

2.9 Effects of source oriented particulate matter

Particles from different sources may have different sizes and chemical compositions. The toxicities of particles depend on their size and chemical compositions. Fine particles which are mostly produced as a result of combustion, associated with diesel exhaust, power plants and other forms of rapid, hot combustion are considered more toxic than larger particles which are often the result of blowing dust or soot as the result of open combustion (Yassi *et al.*, 2001). Some studies have shown that mortality is more strongly associated with fine particles (PM_{2.5}) than with PM₁₀ and the coarse particles (PM_{2.5-10}) (Schwartz, Dockery and Neas, 1996; Smith *et al.*, 2000b; Schwartz, 2003b).

A few studies have analysed which components of particulate matter are actually responsible for its effect on mortality in order to examine the association between the source-specific particulate matter with mortality. An analysis of Harvard Six Cities data (1979 – 1988) used the elemental composition of fine particles (PM_{2.5}) to identify five distinct source-related fractions of fine particles for each city. The five sources of PM_{2.5} identified were motor vehicle emissions, coal combustion, soil and crustal material, fuel oil combustion and salt (Laden *et al.*, 2000). The study (reanalysis by Schwartz (2003b)) reported that fine particles from motor vehicle emissions and coal combustion source but not from soil and crustal factor were associated with increased mortality. The increase in mortality associated with fine particles from motor vehicle source was the highest among all sources. The reanalysis of the data from Laden *et al.* (2000) by Schwartz (2003b) showed a 3.6% increase in mortality associated with a 10 µg/m³ increase in motor vehicle emissions related PM_{2.5} and a 0.8% increase in mortality associated with a 10 µg/m³ increase in PM_{2.5} from coal combustion in the combined analysis of six cities (Schwartz, 2003b).

Mar *et al.* (2000) (reanalysis by Mar *et al.* (2003)) found in their analysis of Phoenix, Arizona data for 1995-1997 that each of motor vehicle factor (1-day lag), vegetative burning factor (3-day lag) and regional sulphate factor (same day) of fine particles (PM_{2.5}) had significant positive associations with cardiovascular mortality (Mar *et al.*, 2000; Mar *et al.*, 2003). Using factor analysis, another study in three New Jersey cities (Camden, Newark and Elizabeth) from 1981 to 1983 identified several major source components of PM including oil burning, industrial, geological, motor vehicle and sulfate/secondary aerosols. The study reported statistically significant associations between each of oil burning, industrial sulfate aerosol and motor vehicle related PM with mortality (Tsai, Apte and Daisey, 2000).

Although a few epidemiological studies have compared the effects of particulate matter of different sources on mortality/morbidity, none of the studies has specifically compared the effects of motor vehicle smoke and wood smoke on mortality. One approach could be to compare the results between the cities which have PM dominated by a single source. For example, the association between particulate matter and daily mortality in Santa Clara County in California could indicate the association between wood smoke related particulate matter and daily mortality as wood smoke is major source of particulate matter in Santa Clara County contributing about 40% of the wintertime PM_{2.5} (Fairley, 1990; 1999). Similarly the findings from a time series study in Amsterdam that individuals who lived on the main roads had much higher relative risks of death than people who lived away from the main roads, when analysed with data from the same background air pollution monitoring stations, suggested that traffic-related particulate matter was involved in increasing the mortality during high pollution days (Roemer and van Wijnen, 2001).

Since the multicity study follows the same protocol for data handling and analysis in each participant city, city specific results in multicity studies are the best to compare the effects of particulate matter of different sources on mortality if the cities have particulate matter dominated by a single source and the dominant source varies from one city to another (Katsouyanni *et al.*, 1997; Laden *et al.*, 2000; Samet *et al.*, 2000c). The APHEA analysis showed that the relative risks of mortality for particulate matter were higher in areas with high nitrogen dioxide (i.e. traffic density) suggesting that motor vehicle related PM might have a higher effect than particles from other sources (Katsouyanni *et al.*, 2001).

2.10 Distributed lag models

Daily mortality is likely to be affected not only by the same day's air pollution but also by the air pollution on a number of preceding days. In the same way, air pollution can increase the deaths occurring on the same day as well as on several subsequent days. The effect of air pollution on mortality is likely to be distributed over time (Zanobetti *et al.*, 2000). Therefore, models with single day's air pollution concentrations are likely to underestimate an effect of short-term exposure to air pollution on daily mortality. In order to measure the real effect of air pollution on mortality, we will need a model that combines the effects of air pollution levels on same day mortality and on subsequent days mortality.

One such modelling method is to develop a regression model for daily deaths with PM₁₀ concentrations of the same day and of previous days as independent variables. The overall effect of a unit increase in PM₁₀ will be the increase in mortality due to a unit increase in PM₁₀ levels on the same day plus the increases in mortality due to a unit increase in PM₁₀ levels on previous days. These models are generally termed “unconstrained distributed lag models”, because PM₁₀ effects on mortality/morbidity in this type of modelling are not constrained (Schwartz, 2000b). In unconstrained distributed lag models, if there is a serial correlation between PM₁₀ levels, there will be multicollinearity between lagged PM₁₀ variables and thus the regression models will have a collinearity problem. This will result in unstable estimates of PM₁₀ effects (Pope and Schwartz, 1996; Schwartz, 2000b).

In order to reduce the noise in PM₁₀ effects estimated by unconstrained distributed lag models, the set of PM₁₀ effects over time can be constrained to some shape. One method is to calculate the weighted average of lagged PM₁₀ concentrations with weights that reflect the relative effects of the same day and lagged PM₁₀ concentrations and use the weighted average as an exposure variable. These models put a constraint in the relative effects of lagged PM₁₀ variables and thus are referred to as constrained distributed lag models (Schwartz, 2000b). Some studies have used a moving average of daily PM₁₀ levels as an exposure variable. This approach assigns equal weightings to the effects of every days air pollution level, which is also likely to not reflect the real health risk.

Instead of assigning equal weights to the effects of the same day and all lagged air pollution levels, a few studies have analysed the shape of distribution of air pollution effects at different lags and assigned the weights accordingly. The most common approach is to constrain the shape to fit some polynomial function.

Schwartz (2000b) used a quadratic polynomial distributed lag model with 5 lags to study the effect of PM₁₀ on daily mortality of persons 65 years of age and over in 10 US cities. He compared the results with the results from the unconstrained distributed lag models, and the models with the same day’s PM₁₀ levels and the 2-day moving average of PM₁₀ levels (lag 0 and lag 1) as air pollution exposures and found that the overall effects from the distributed lag models were higher than the effects using one or 2-day moving average of PM₁₀ levels. The study reported an increase of 1.41% in daily deaths for a 10 µg/m³ increase in PM₁₀ when lag structure was considered as opposed to an increase of 1.05% in daily deaths for a 10 µg/m³ increase in 2-day moving average PM₁₀ levels. (Schwartz, 2000b).

Similar results were reported by a study conducted as part of the APHEA-2 study in its ten largest study cities. This study estimated the combined effects of PM₁₀ on the same day and the lagged effects up to 40 days using 3rd and 4th degree polynomial distributed lag models. It was found that the estimated effects of PM₁₀ were more than doubled in many cities when lagged effects were considered as compared to the 2-day moving average (same day and the day before) of PM₁₀ levels. The results were consistent for all distributed lag models including unconstrained distributed lag models (Zanobetti *et al.*, 2002).

Another study of the association between daily air pollution and daily mortality in Milan between 1980 and 1989 examined the effect of air pollution distributed over the same day and the following 45 days. Instead of polynomial distributed lag models, this study used a non-parametric smoothed distributed lag models which constrained the estimated air pollution effects to vary smoothly with the number of days lag between air pollution exposure and mortality. The study reported that the effect of cumulative exposure to air pollution was higher than the effect of air pollution on the same day (Zanobetti *et al.*, 2000).

Distributed lag modelling has also been used to study the extended effects of cold and air pollution simultaneously. Goodman *et al.* (2004) analysed Dublin data from April 1980 to December 1996 to assess the cumulative net effects of daily minimum temperature and Black Smoke (BS) particulate air pollution exposure over the following 40 days using polynomial distributed lag models. As in other studies, this study also reported higher effects of the extended exposure to air pollution on mortality. The study estimated a 1.1% increase in total non-trauma mortality associated with an increase of 10 µg/m³ in daily mean BS over the succeeding 40 days whereas the effect of each 10 µg/m³ increase in a 3-day mean BS was only 0.4% increase in total non-trauma mortality (Goodman, Dockery and Clancy, 2004).

2.11 Harvesting (mortality displacement)

The other key concern associated with the interpretation of the short-term association between air pollution and mortality as demonstrated by time series studies is that it is unclear whether the association is due to the short-term displacement of the deaths of people who would have died in a few days regardless of air pollution condition. Despite providing evidence of the short-term association between air pollution and mortality, time series studies can not substantiate that there will be an increase in longer term mortality due to long-term exposure to air pollution (McMichael *et al.*, 1998). The key

question in assessing the public health impact of air pollution is to understand whether higher air pollution levels increase mortality in the longer term or just bring forward the event of deaths by a few days known as mortality displacement (short-term “harvesting” of deaths). The public health significance will be considerably larger if life expectancy is reduced by months or years compared with deaths only brought forward by a few days (McMichael *et al.*, 1998; Brunekreef and Holgate, 2002).

Two papers have used conceptually similar approaches to test whether the short-term association between air pollution and mortality is due to short-term mortality displacement (Zeger, Dominici and Samet, 1999; Schwartz, 2000c). Both approaches assumed that under the short-term harvesting hypothesis, the increase in mortality during higher air pollution days would be immediately followed by lower than expected mortality, which persists until the mortality level comes back to the expected level. If there is a substantial time between the increases in mortality and lower than expected mortality, then the mortality is being displaced by a substantial amount of time. If there is short-term harvesting, then an association would only be detected at shorter time scales, but not on longer time scales (Bell, Samet and Dominici, 2004). One of the above approaches focussed on time scale to test the hypothesis of short-term harvesting and examined the associations between daily air pollution levels and daily mortality at different time scales (Schwartz, 2000c). The other approach tested the hypothesis in the frequency domain and examined the associations between daily air pollution levels and daily mortality at different frequency ranges (Kelsall, Zeger and Samet, 1999; Zeger, Dominici and Samet, 1999).

Schwartz (2000c) (reanalysed by Schwarz (2003b)) tested the hypothesis of short-term harvesting using Boston, Massachusetts data (1979–1986) by decomposing data into three independent time series data representing long time trends and seasonal variations, intermediate variations, and the shortest-term variations. By varying the smoothing window sizes (15, 30, 45 and 60 days), several time series data, with seasonal and shortest-term variation removed, representing intermediate variations of different time scales were generated and the associations between $PM_{2.5}$ and mortality were examined on these mid-scale components separately (Schwartz, 2000c; 2003b). Another study in Chicago (1988–1993) used the same approach to examine short-term harvesting in the association between PM_{10} and mortality and hospital admissions (Schwartz, 2001). Both studies used the STL algorithm, the seasonal and trend decomposition program introduced by Cleveland *et al.* (1990), which fits LOESS

smooths with different smoothing window sizes to a time series data to decompose it into different time scale components (Cleveland, 1979; Cleveland *et al.*, 1990).

Both studies reported associations between particulate matter and mortality and hospital admissions at different time scales. The effect of PM_{2.5} on mortality due to all causes, pneumonia and heart attacks increased with increase in time scale except for the mortality due to chronic obstructive pulmonary disease (COPD). The study reported a reduction in the relative risk of COPD deaths due to PM_{2.5} for the longer time scale suggesting the deaths due to COPD were brought forward by a few weeks or few months (Schwartz, 2000c; 2003b). However, the Chicago study showed that the effect of PM₁₀ on hospital admissions due to COPD increased with longer time scales and the effect size estimates were more than doubled for daily deaths and for COPD admissions (Schwartz, 2001).

Zeger *et al.* (1999) (reanalysis by Dominici *et al.* (2003a)) used a frequency domain log-linear regression approach (Kelsall, Zeger and Samet, 1999) to estimate the effects of total suspended particles (TSP) on mortality in Philadelphia data (1974-1988) that is resistant to short-term harvesting. The assumption was that short-term harvesting creates the association only at shorter time scales. They decomposed the time series data at different characteristic frequency ranges using Fourier series decomposition and estimated the effects at each characteristic frequency range separately. Under the short-term harvesting hypothesis, which is that the association between TSP and mortality is only due to short-term harvesting, the effects would be expected to be near zero at lower frequencies (longer time scales) and to increase towards higher frequencies (shorter time scales). However, the study found the results, which were opposite to what would have been expected under the short-term harvesting hypothesis. The relative increase in mortality associated with the relative change in TSP levels was significantly different from zero at lower frequencies and decreased towards higher frequencies (Zeger, Dominici and Samet, 1999; Dominici *et al.*, 2003a). Hence they argued that the short-term harvesting hypothesis was inconsistent with the Philadelphia data.

Another study in four US cities (Pittsburgh, Minneapolis, Chicago and Seattle) using data from 1987-1994 also found a larger relative risk for mortality associated with PM₁₀ at longer time scale than at shorter time scale (Dominici *et al.*, 2003c). The major difference between their method and the method proposed by Schwartz (2000c) and Zeger *et al.* (1999) is that Schwartz (2000c) and Zeger *et al.* (1999) decomposed all time series data including mortality, PM₁₀ and weather into three distinct time series representing long time trends and seasonal variations, intermediate variations, and the

shortest-term variations whereas Dominici et al. (2003c) decomposed only PM₁₀ data series into distinct time scale component series. They applied the discrete Fourier transformation with the frequencies of different cycles to the PM₁₀ series to decompose the daily air pollution data. They decomposed the daily time series data into the six series of independent time series ranging from the very smooth series, which fluctuates with a very low frequency (more than a 2 month cycle) to the less smooth series, which fluctuates with very high frequency (less than a 3.5 day cycle). They fitted the model for daily mortality with all six components series of air pollution data as exposure variables adjusting for temporal trend, days of the week and weather variables. This model estimated the relative risk for increasing daily mortality from an increase in air pollution levels at different time scales adjusting for weather variables. The analysis first calculated the city specific relative risks of mortality and then the results were pooled together to calculate the overall relative risk of mortality across four cities (Dominici *et al.*, 2003c).

Under the short-term harvesting hypothesis that air pollution mortality association is only due to short-term mortality displacement, mortality is associated only with the short-term time scale component of air pollution but not with the mid to long-term time scale component of air pollution.

In a study in Milan, Italy (1980 – 1989), Zanobetti et al. (2000) used generalised additive distributed lag models to quantify mortality displacement (Zanobetti *et al.*, 2000). Zanobetti et al. (2002) (reanalysis by Zanobetti & Schwartz (2003)) applied the same concept (distributed lag modelling) in a multicity analysis of the 10 European study cities of APHEA2. All these studies reported that the PM₁₀ risk estimates obtained from the distributed lag models with up to lag of 40 days was higher than the PM₁₀ risk estimates obtained for the average of lag 0 and lag 1 suggesting a lack of mortality displacement up to 40-45 days (Zanobetti *et al.*, 2002; Zanobetti and Schwartz, 2003).

Spix et al. (1993) investigated short-term mortality displacement due to SO₂ in Erfurt, Germany (1980 – 1989) using an interaction term between pollution and the mean number of deaths in the previous days. The study tried the interaction terms of pollution with the last 2 to 21 days mean mortality in the model and found the best fit with 15 days mean mortality. The assumption was if there was mortality displacement then there would be fewer than average deaths if more than average deaths occurred in the past 15 days. This would result in a significant interaction effect. However, they found that the interaction term was not statistically significant showing a lack of evidence for the short-term mortality displacement (Spix *et al.*, 1993).

A Philadelphia study for the period of 1973 to 1990 used state space modelling to estimate the relationship between air quality and mortality (Murray and Nelson, 2000). The model, which assumes harvesting effects, allows estimation of the size of the at risk population; life expectancy of individuals in that population and the effect of changes in air pollution on that life expectancy. Murray & Nelson (2000) first verified the model by simulation and then applied it to Philadelphia data. They estimated that TSP caused a difference of about 2.5 days on average in the life expectancy of the roughly 500 at risk population in Philadelphia. These results are in contrast with the findings of Zeger et al. (1999), Schwartz (2000c), Dominici et al. (2003c) and Zanobetti et al. (2002) which showed evidence of mortality displaced by air pollution by more than a few weeks. However, due to the nature of time series studies, these studies could not identify exactly by how many weeks or months the time of death is brought forward by particulate matter. These studies have found that the effect size estimates from the existing time series studies are smaller than the effect size when people are exposed to air pollution for a longer time period. However in order to draw any valid conclusions, these analyses need to be replicated in different populations (Brunekreef and Hoek, 2000).

2.12 Summary

This chapter reviewed the studies of the association between air pollution and mortality with a focus on time series studies. A large number of time series studies, both single city studies and multicity studies, carried out worldwide have shown positive associations between particulate matter and mortality. Despite the use of variety statistical methods, all studies provided similar conclusion.

There are a number of issues and uncertainties associated with air pollution epidemiological studies, which include concentration-response relationship, the threshold levels, the role of harvesting, extended effects of air pollution, and the differences in the effects of PM₁₀ from different sources. This review summarised some of the literatures, which have analysed one or more of the above issues. The conclusions from the review of several literatures are that there is a log-linear relationship between daily PM₁₀ and mortality, and there is no safe threshold level for mortality effect of PM₁₀. The positive association between daily PM₁₀ and mortality can not be attributed to harvesting. The effects of PM₁₀ on mortality are not limited to the first few days. Increase in PM₁₀ concentrations can be associated with an increase in mortality of several weeks after exposure.

Chapter 3: Study design and data preparation

3.1 Introduction

This chapter describes the study design, study area, sources of data, and measurements of air pollutants, mortality and population data. In terms of design, this study can be broadly divided into two parts. The first part (analysis of research objectives 1 to 4) is a time series study. The second part (analysis of research objective 5) is an ecological cross-sectional study. This chapter describes the preparation of air pollution, mortality and population data for the analysis of the first part of the study. The measurement of air pollutant and database preparation for the second part of the study is described in Chapter 9.

3.2 Study design

The first part of this study (analysis of research objectives 1 to 4) is a time series study with “day” as the unit of analysis. Time series study design associates daily variation in mortality with daily variation in air pollutant concentration after controlling for the variables that vary over time such as a long-term trend and seasonal variations in daily mortality, weather variables and other pollutants. This study analyses routinely collected mortality, weather and air pollution data. The second part (analysis of research objective 5) is an ecological cross-sectional study, which is described in detail in Chapter 9.

3.3 Study period

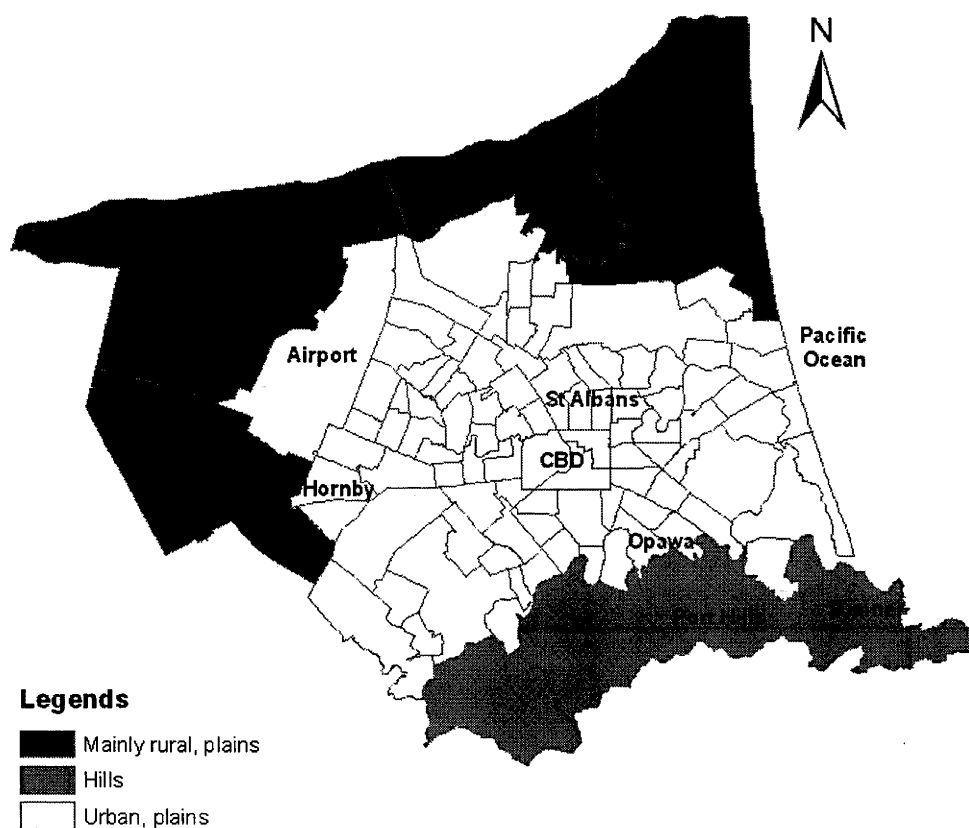
The first part of this study (Chapters 6, 7 and 8), which is a time series study, analyses the data from June 1988 to December 1999. The second part (Chapter 9), which is an ecological cross-sectional study, analyses mortality data from 1996 to 1999.

3.4 Study area

Christchurch is a coastal city with a population of about 316,000, located on the east coast of the South Island of New Zealand. The city lies at latitude 43° South and longitude 172° East, on the Canterbury Plains, bounded by the Pacific Ocean in the east, the Canterbury plains extending as far as the Southern Alps in the north and west, and the Port Hills in the south. Apart from the suburbs in the foothills of the Port Hills on the southern edge, Christchurch is almost flat, sloping gently downwards from the airport in the west to the Pacific Ocean in the east. Figure 3-1 shows a map of Christchurch with census area unit (CAU) boundaries. The CAUs are the basis for the

resident location of routinely collected health data, including mortality data. These areas are the second smallest geographic units defined by Statistics New Zealand for statistical purposes and are the most commonly used geographic units to disseminate the census data. Meshblocks are the smallest geographic units for which the census data are collected and processed. There are 106 CAUs in Christchurch, with the CAU population ranging from about 250 to 6,000.

Figure 3-1. Map of Christchurch showing census area unit boundaries



Christchurch is ideally situated for the frequent occurrence of calm weather and temperature inversions which are conducive to high air pollution on cold winter nights. Westerly cold air drainage from the Southern Alps converges with the drainage winds down the slopes of Bank Peninsula (including the Port Hills) over Christchurch forming zones of stagnant air which enhance the strength of temperature inversions (Kossmann and Sturman, 2004). Local meteorological conditions such as calm weather and temperature inversions, in combination with emissions from the burning of coal or wood as domestic heating on cold winter nights, exacerbate particulate air pollution levels in Christchurch. Twenty-four hour average PM_{10} concentrations exceeds the Ministry for the Environment ambient air quality guideline of $50 \mu\text{g}/\text{m}^3$, on average, for 30 days each year in the winter (Canterbury Regional Council, 1997; Ministry for the

Environment and Ministry of Health, 2002). Domestic heating is the major source of winter time particulate air pollution contributing about 82% of PM₁₀ in the winter months (Scott and Gunatilaka, 2004) and thus has been a major concern in winter. In addition to the emissions from domestic heating, air pollution from other sources, particularly vehicle emissions, is also becoming a growing concern in Christchurch (Kjellstrom, Shrestha and Metcalf, 2002).

3.5 Air pollutant measurements

3.5.1 Data source

Environment Canterbury provided hourly air pollution and meteorological data, monitored at the St. Albans air quality monitoring site, for the May 1988 to December 1999 period. This site was established between Packer Street and Madras Street at St. Albans in 1988 by the former Department of Health. The Ministry of Health continued to operate the site as part of its contribution to an international air quality monitoring programme Global Environmental Monitoring Systems (GEMS). The Ministry for the Environment took over this responsibility in 1999. From 1993 to September 2000, the Institute of Environmental Science and Research Limited (ESR) operated the site on behalf of the Ministry of Health, and then on behalf of the Ministry for the Environment. The site was operated in accordance with Environment Canterbury's quality assurance procedures for ambient air quality monitoring. ESR maintained the monitoring site and was responsible for the calibration of monitoring equipment, data collection and quality assurance of data.

ESR supplied data to Environment Canterbury after validating it according to Environment Canterbury's quality assurance procedures (Aberkane *et al.*, 2001) and Environment Canterbury archived them. Any invalid data that were not representative of ambient air quality and weather were removed from the database before the data were archived (Aberkane *et al.*, 2001). Environment Canterbury provided the quality assured archived hourly data for the following air pollutants and meteorological parameters for the St. Albans monitoring site:

- Air pollutants
 - a. Particulate matter of less than 10 µm diameter (PM₁₀) (µg/m³)
 - b. Carbon monoxide (CO) (mg/m³)
 - c. Nitrogen dioxide (NO₂) (µg/m³)

- Meteorological parameters
 - a. Wind speed (m/s)
 - b. Wind direction (degrees from North)
 - c. Temperature at 1 metre ($^{\circ}\text{C}$)
 - d. Temperature at 10 metres ($^{\circ}\text{C}$)
 - e. Relative humidity (%)

Hourly data for the following meteorological parameters collected at Christchurch Airport were also obtained for the years from 1988 to 1999.

- a. Wind direction (degree from North)
- b. Wind speed (m/s)
- c. Dry bulb temperature ($^{\circ}\text{C}$)
- d. Wet bulb temperature ($^{\circ}\text{C}$)
- e. Relative humidity (%)

3.5.2 Monitoring methods for air pollutants

Particulate matter (PM₁₀)

A number of different instruments have been used to measure PM₁₀ concentrations at the St. Albans monitoring site at Packer St. since its establishment. Monitoring was begun with an MPSI beta gauge in May 1988, which was used until 13 June 1994. For a short period, from 18 June to 7 August 1994, a high volume sampler was used to monitor PM₁₀ concentrations. Since 17 August 1994, they were monitored using the Rupprecht and Patashnick Co., Inc. Tapered Elemental Oscillating Microbalance (TEOM) particulate monitor. PM₁₀ concentrations were not monitored between 8 August and 16 August 1994. With the installation of a Wedding & Associates (W&A) beta gauge in May 1996, parallel monitoring of PM₁₀ concentrations using both the TEOM and the beta attenuation methods began at this site. Measurement of PM₁₀ concentrations depend on types of instruments used and the temperature settings of those instruments.

Carbon monoxide (CO)

Carbon monoxide was monitored using infrared spectrophotometry. Using this method, CO can be monitored by two different instrumental methods, the non-dispersive method and the gas filter correlation method. CO was monitored using a non-dispersive Uras 3G/20310 until 4 April 1997; thereafter it was monitored by an API 300 instrument, which uses the gas filter correlation method.

Nitrogen dioxide (NO₂)

Nitrogen dioxide concentrations was calculated by measuring nitrogen oxides using a technique called ozone chemiluminescence, which is a chemical reaction that emits energy in the form of light. NO₂ was monitored via single channel measurement. In this method, ambient airflow is alternately passed through two processes. One diverts the ambient airflow through a catalytic converter which reduces nitrogen dioxide (NO₂) to nitric oxide (NO). This adds NO to already present NO in the sample. The measurement of NO gives the level of total oxides of nitrogen (NO_x). The other process bypasses the converter and as such, the measurement of NO gives only the level of NO present in the sample. NO₂ is calculated based on the differences between NO_x levels (from the diverted air flow) and NO levels (from the undiverted air flow). Monitoring was carried out using an API 200A NO_x analyser.

3.5.3 Monitoring methods of PM₁₀

PM₁₀ concentrations were monitored using different methods over the twelve year period since monitoring began in May 1988 (Table 3-1) (Foster, 1998).

Table 3-1. Methods used to monitor PM₁₀ at St. Albans site

Method	Time period
Beta gauge analyser	28 May 1988 to 13 June 1994
Beta gauge analyser	May 1996 to present
High volume sampler	18 June 1994 to 7 August 1994
TEOM analyser at 50 ⁰ C	17 August 1994 to December 1997
TEOM analyser at 30 ⁰ C	January 1998 to December 1998
TEOM analyser at 40 ⁰ C	January 1999 to December 1999

A high volume sampler was used to measure PM₁₀ concentrations for a very short period from June to August in 1994. Unlike the TEOM and the beta gauge analyser, it is not possible to get results immediately from a high volume sampler and measurements for periods of less than 24 hours are not practicable using the high volume sampling method (Foster, 1998). Thus the variations of hourly concentrations over a day were not available during this period. Since the high volume sampler was used for a very short period and the method was not consistent with the other methods (TEOM and beta attenuation), the monitored PM₁₀ levels from the high volume sampler were not used in this study. Only the beta gauge and the TEOM measured PM₁₀ concentrations were used in this study. Parallel monitoring using both methods in the later years of the study

made it possible to compare the methods, and to convert the PM₁₀ concentrations measured by the beta gauge method in the earlier years into the equivalent PM₁₀ concentrations measured by the TEOM method.

Of the three methods used at the St. Albans site, the TEOM method is the most technologically advanced and the most accurate over short time periods. This method is used extensively in a number of countries including Canada, France, the United Kingdom and Australia. In this method, the TEOM sensing system is heated to evaporate water and to minimise the effects of thermal expansion and contraction of the mass sensor. This heating also causes the volatilisation of some volatile materials, including nitrates and low molecular weight organic compounds. The amount of material volatilised depends on the temperature settings of the TEOM analyser and the composition of particulate matter. At higher temperature settings, the degree of volatilisation is more likely to be higher, resulting in a greater loss of volatiles (Environet Limited, 2003). The standard sample temperature setting for the TEOM analyser in Canada is 40°C, and the UK authorities operate at the setting of 50°C (Foster, 1998).

There was no recommended national standard regarding the temperature setting for TEOM analysers in New Zealand when Canterbury Regional Council (in 2000, Canterbury Regional Council became Environment Canterbury) started using them, replacing the high volume sampler and the beta gauge analyser. The sample temperature on the TEOM analyser was initially set at 50°C. In order to minimise the loss of particulate matter, the sample temperature setting was changed to 30°C in January 1998. In September 1998, a temperature of 40°C was proposed as a national standard operating temperature for TEOM equipment (Foster, 1998). From January 1999, the temperature of the TEOM equipment at St. Albans monitoring site was set to the national standard operating temperature of 40°C.

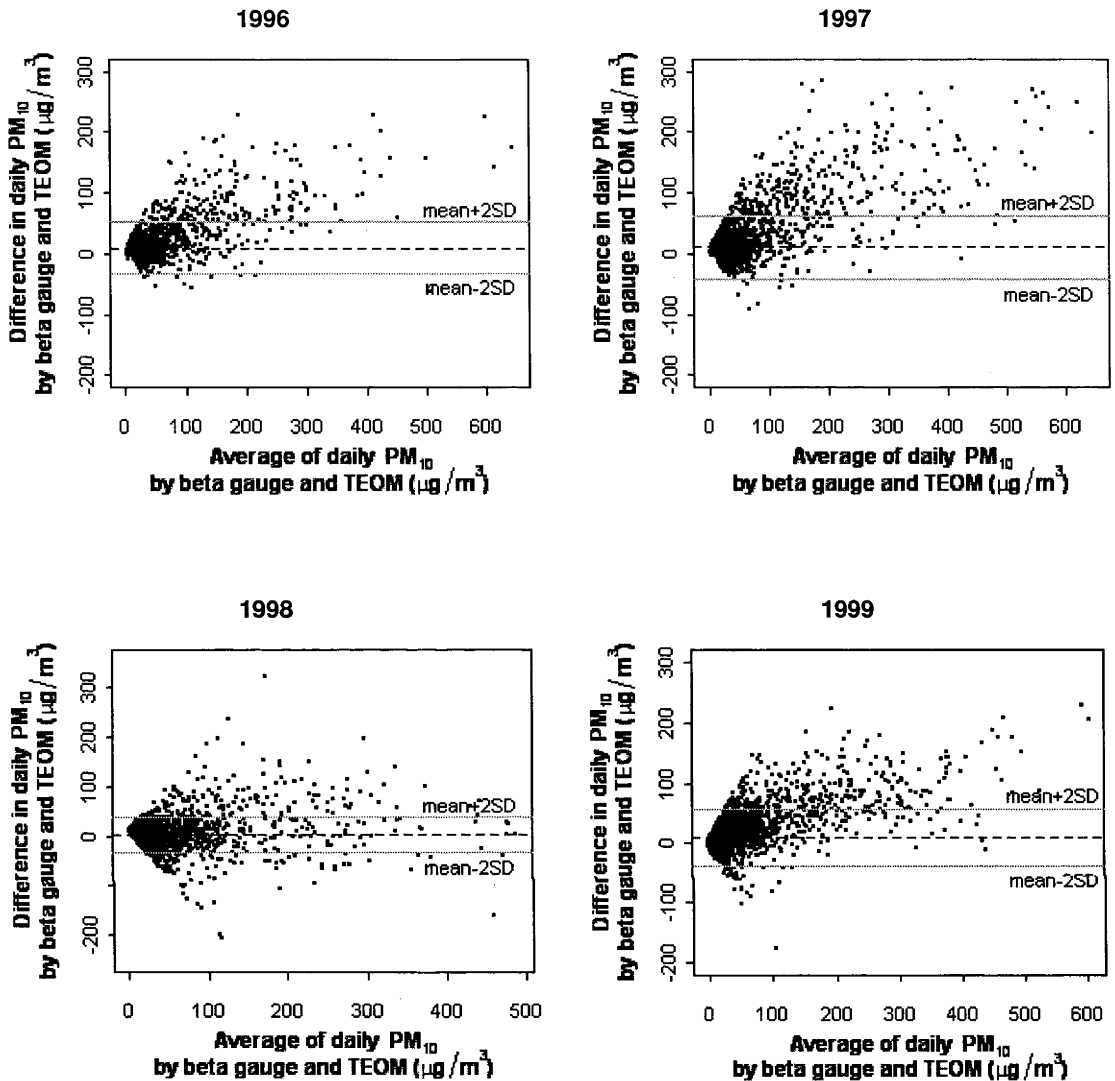
3.5.4 Comparison of different methods of measuring PM₁₀

From 1996, there was parallel monitoring of PM₁₀ concentrations using both the TEOM and the beta attenuation methods at St. Albans site (Table 3-1). Whether or not the PM₁₀ concentrations measured by the two methods were comparable was analysed using the Bland-Altman method for assessing the agreement between two methods of measurement (Bland and Altman, 1986). Figure 3-2 shows the Bland-Altman plots for each year from 1996 to 1999, when PM₁₀ data from both the beta attenuation method and the TEOM methods were available. Differences between PM₁₀ concentrations

measured by the two methods were plotted against the average of PM_{10} concentrations measured by the two methods. The dotted line in the plot represents the mean difference in the concentrations measured by the two methods. For 1996 and 1997, the beta gauge method was compared with the TEOM method with sample temperature setting of $50^{\circ}C$. For 1998 and 1999, it was compared with the TEOM method with sample temperature settings of $30^{\circ}C$ and $40^{\circ}C$ respectively.

The plots show that the two methods did not give the same concentrations and suggest that PM_{10} concentrations measured by the TEOM methods were on average lower than the concentrations measured by the beta gauge method. The differences in PM_{10} concentrations measured by the two methods increased with increase in PM_{10} concentrations. Differences were larger when higher sample temperature settings were used in the TEOM analysers. This was more likely due to the lower levels of PM_{10} concentrations measured by the TEOM analysers with higher sample temperature settings due to a greater loss of volatile materials in particulate matter.

Figure 3-2. Bland-Altman plots to assess the agreement between the beta attenuation method and the TEOM methods of measuring PM₁₀ concentrations



Note: Scales are different

3.5.5 Missing data

Because of instrument malfunction and data logger malfunction, the data measured by the instruments did not, at times, represent the actual ambient air quality and weather conditions. Data were validated according to Environment Canterbury's quality assurance procedures and any invalid data that were not representative of ambient air quality and weather were removed from the database before the data were archived by Environment Canterbury. This resulted in some data loss. In addition, other factors such as power failure of the instruments and discontinuity of remote access to the data also resulted, at times, in the loss of data. Missing data were interpolated or extrapolated by Environment Canterbury as per their Quality Assurance Procedures Manual by using

their in-house software TRANSFER (Aberkane *et al.*, 2001). Hourly data which did not satisfy the guidelines of the manual were left as gaps in the quality assured hourly datasets provided by Environment Canterbury.

A large portion of meteorological and pollutant data was missing for the years from 1991 to 1995. Table 3-2 shows the percentage of missing hourly data for both sets of variables by years. The years 1997 and 1998 had the least missing data for all variables. Relative humidity data was completely missing for 1993 and 1994.

Table 3-2. Percentage of missing hourly data for meteorological and pollutant variables for twelve year study period (1988 – 1999)

Year	Relative humidity	Temperature	Wind speed	PM ₁₀	CO	NO ₂
1988	4.5	4.4	4.5	6.7	5	4.6
1989	11.5	13.7	11.5	24	12.6	22.4
1990	1.1	1.1	1.1	4.1	2.8	7.4
1991	5.6	5.5	5.6	6.7	26.9	14.7
1992	69.7	18.2	18.8	20.3	19.3	32.3
1993	100	7.9	5.2	46.6	21.2	35
1994	100	16.1	18.3	58.6	7.8	43.1
1995	46.9	38.7	60.4	3.4	8.6	16.3
1996	12.2	1.2	38.7	3.3	1.4	2.4
1997	0.2	0.2	0.2	3.9	5.3	1.9
1998	0.8	0.8	0.9	0.8	0.9	6.1
1999	1.6	1.6	1.6	1.6	1.7	8.2

3.5.6 Estimating missing air pollutant concentrations

Missing hourly air pollutant data were estimated in two stages. First, missing hourly meteorological data were estimated and then the missing hourly pollution concentrations were estimated by fitting regression models based on meteorological variables.

Stage 1: Estimation of missing hourly meteorological data

There were strong correlations between the weather data measured at the St. Albans monitoring site and at the airport. For hourly temperature, the correlation coefficient between the airport data and the St. Albans monitoring station data in 1997 was 0.95. For hourly wind speed and hourly relative humidity, the correlation coefficients were 0.79 and 0.90 respectively.

Because of highly positive correlations between hourly weather data at the St. Albans monitoring site and hourly weather data at Christchurch Airport, the Christchurch Airport data can be used to predict the missing hourly meteorological data for the St Albans monitoring site. Linear regression models were fitted for each of three meteorological variables, with hourly data at St. Albans as a dependent variable and hourly data at Christchurch Airport as a predictor. Separate models were developed for each year. Based on these linear regression models, hourly levels for meteorological variables were predicted for the St. Albans site. The predictions were used as the estimated values for the missing hourly weather data at St. Albans. A complete hourly meteorological data set for St. Albans was prepared by replacing the missing hourly meteorological data with the corresponding predicted hourly levels. This data set was used to develop the regression models for pollutants, based on which hourly pollutant concentrations were predicted, which were then used to replace the missing hourly pollutant concentrations.

There was no relative humidity data at the St. Albans monitoring site for 1993 or 1994. No model was fitted for relative humidity for those two years. Instead, hourly relative humidity data at Christchurch Airport were used as the estimated hourly relative humidity at St. Albans for 1993 and 1994. Linear regression models for relative humidity for other years showed that there was very little difference between relative humidity at the airport and at St. Albans. The slopes of annual linear regression models with hourly data at the St. Albans monitoring site as a dependent variable and hourly data at Christchurch Airport as a predictor ranged from 0.89 to 1.03. It was thus assumed that relative humidity at St. Albans would be almost the same as relative humidity at Christchurch Airport.

Stage 2: Estimation of missing hourly air pollution data

Air pollution levels depend on emissions and meteorological conditions. Emissions of air pollutants from home heating are higher in the winter than in the summer because of home heating to keep houses warm in the cold weather of winter. In winter, the emissions from home heating in a 24 hour period are likely to vary, with higher emissions in the evenings and the early mornings when people burn coal and wood to keep houses warm. Vehicle emissions are higher during the hours when there are high volumes of traffic on the road. These emissions are higher on weekdays than on weekends because of a higher volume of traffic on weekdays. Thus, the emissions of air pollutants vary throughout the day and also depend on days of the week and seasons.

Meteorological conditions are strongly associated with the dispersion of air pollutants. Low wind speed and temperature inversions cause poor air circulation, restricting the dispersion of air pollutants. Temperature inversion limits the vertical dispersion of air pollutants and low wind speed limits their horizontal dispersion. In addition, temperature also plays a major role in the emissions of air pollutants from home heating as people heat their houses in cold weather.

In addition to the above mentioned meteorological conditions (i.e. wind speed and temperature inversion), the concentrations of one of the major air pollutants; ozone also depends upon sunshine. Sunshine is vital to the production of ozone. However, ozone levels were not monitored in Christchurch during the study period and thus were not analysed in this study.

The high dependence of air pollution levels upon meteorological conditions and hourly and seasonal emission patterns allowed the estimation of missing hourly air pollution data using regression models. In order to explain the variations in hourly emissions, indicator variables for each hour of a day were created. Similarly an indicator variable was created to explain the differences in emissions on weekdays and weekends. These indicator variables were used in the regression models as proxy variables for explaining the variations in the emissions of air pollutants. Regression models were fitted for hourly air pollution levels, with the indicator variables and meteorological variables as predictors. Regression models also had the interaction terms between meteorological variables as predictors if adding them improved the model fit.

Because of the time series nature of air pollution and meteorological data, serially correlated residuals were expected from the ordinary least squares regression models for hourly air pollutants. The Durbin-Watson test statistic confirmed that the residuals were serially correlated (Draper and Smith, 1998). The autoregressive models controlling for the autocorrelation in the residuals were fitted using the SAS AUTOREG procedure. The autoregressive models satisfied the standard assumptions of linear regression models such as the normality of residuals, the constant variance and the independence assumption of the residuals (Draper and Smith, 1998). In addition to the explanatory variables, these models included new autoregressive parameters to explain any serial correlations left in the pollutants, not captured by the explanatory variables. The inclusions of new parameters that explain serial correlations added explanatory power to the models, which helped to better estimate the missing hourly air pollution levels. The autoregressive parameters (lag numbers for the autocorrelated errors) in the model were chosen based on their significance level. Details of model development for

hourly air pollutant levels and model validation were reported elsewhere (Shrestha, Kjellstrom and Metcalf, 2002).

Due to the differences in the methods used to measure air pollutants over the twelve year period, modelling was carried out separately for each year. Models were separately fitted for the winter and the summer periods each year because of the substantial differences in air pollution levels during those periods. For modelling purposes, four months from May to August, when the emissions from home heating were the dominant source of air pollutants, were grouped as the winter period. The remaining eight months, from January to April and September to December, when the contributions of emissions from home heating to the ambient air pollution were very low, were grouped as the summer period. When air pollution concentrations were very low, fluctuating around zero, the instruments sometimes provided below zero readings. These negative readings should be considered as very low levels, close to zero (Personal communication with Teresa Aberkane, Environment Canterbury). The negative readings were converted to zero before fitting the models. Models were separately fitted for all three pollutants (PM₁₀, CO and NO₂).

Hourly air pollution levels for all three pollutants were predicted based on the fitted models for the respective pollutants. The predictions were used as the estimated levels for the missing hourly air pollution data at St. Albans. A complete hourly air pollution data set was prepared for St. Albans by replacing the missing hourly air pollution data with the corresponding predicted hourly levels in this thesis. The complete hourly air pollution and meteorological database with the missing data replaced by the corresponding model predictions was used as an exposure database in this study.

3.5.7 Conversion of measured hourly PM₁₀ concentrations from one method to another

From 1996, there was parallel monitoring of PM₁₀ concentrations using both the TEOM and the beta attenuation methods at the St. Albans site. There was a highly positive correlation between the PM₁₀ concentrations measured by the two analysers. Table 3-3 shows the correlation coefficients (ρ) between hourly PM₁₀ concentrations measured by the TEOM analyser and the beta gauge analyser and the regression equations showing the relationships between them for different years. The PM₁₀ concentrations measured by the TEOM analyser were on average lower than concentrations measured by the beta gauge monitor.

Table 3-3. Linear relationships between hourly PM₁₀ concentrations measured using a TEOM monitor and a beta gauge analyser for different years (1996 – 1999)

Year	Analysers	N	ρ	R ²	Regression equation
1996	TEOM at 50 ⁰ C and Beta gauge	5186	0.94	0.88	T ₁ = 0.68*BG + 0.27
1997	TEOM at 50 ⁰ C and Beta gauge	8308	0.93	0.87	T ₁ = 0.64*BG + 1.48
1998	TEOM at 30 ⁰ C and Beta gauge	8151	0.89	0.80	T ₃ = 0.83*BG + 1.40
1999	TEOM at 40 ⁰ C and Beta gauge	6584	0.94	0.88	T ₂ = 0.69*BG + 0.47
T ₁	TEOM at 50 ⁰ C	N			No. of hourly observations used in regression
T ₂	TEOM at 40 ⁰ C	ρ			Correlation coefficients
T ₃	TEOM at 30 ⁰ C	R ²			R ² value for the linear regression model fitting
BG	Beta gauge				

PM₁₀ concentrations measured by the TEOM analyser were dependent upon the sample temperature setting of the TEOM equipment. The monitored PM₁₀ concentrations were lower when the sample temperature settings were higher. This was due to a greater loss of volatile materials in particulate matter at higher temperature settings. The amount of material volatilised depends on the composition of particulate matter. The TEOM analyser operating at 40⁰C gave PM₁₀ readings approximately 31% less than the PM₁₀ levels measured by the beta gauge analyser. PM₁₀ levels measured by the TEOM analysers with sample temperature settings of 50⁰C and 30⁰C were approximately 36% and 17% lower, respectively, than the levels measured by the beta gauge analyser.

In order to make PM₁₀ levels comparable over the whole study period (June 1988 – December 1999), it was necessary to convert both the original and the model estimated hourly PM₁₀ concentrations, which replaced the missing hourly PM₁₀ concentrations, to a common measurement method. The TEOM method with a sample temperature setting of 40⁰C was chosen as the standard method to convert all hourly PM₁₀ concentrations as Environment Canterbury is currently using this method to monitor PM₁₀ concentrations for reporting purposes in Christchurch in the recent years.

Based on the relationship between the TEOM measured PM₁₀ and the beta gauge measured PM₁₀ (Table 3-3), equations between the hourly PM₁₀ concentrations measured by the TEOM methods at different sample temperature settings were developed (Table 3-4). The linear relationship between the TEOM and the beta gauge

measured PM₁₀ levels for 1997 was used to develop the equations between PM₁₀ concentrations measured by the TEOM method at sample temperature settings of 40°C and 50°C for the years from 1994 to 1997. The relationship based on the 1996 data was not used as PM₁₀ concentrations measured by the beta gauge method were available for relatively a short period of time in 1996 compared to those measured in 1997. The PM₁₀ data measured by the beta gauge method were not available for the four months from January to April in 1996.

Using equations in Table 3-4, all hourly PM₁₀ concentrations were converted to the equivalent reading of PM₁₀ concentrations measured by the TEOM method with a sample temperature setting of 40°C. PM₁₀ concentrations measured using the beta gauge method were adjusted downwards. PM₁₀ concentrations measured by the TEOM analyser with a sample temperature setting of 50°C were adjusted upwards whereas the readings from the TEOM analyser with a sample temperature setting of 30°C were adjusted downwards.

Table 3-4. Relationships between PM₁₀ concentrations measured by different methods (Table of adjustments)

Year	Analysers	Equations
1988 – 1993	TEOM at 40°C and Beta gauge	$T_2 = 0.69 \cdot BG + 0.47$
1994 – 1997	TEOM at 40°C and TEOM at 50°C	$T_2 = 1.09 \cdot T_1 - 1.14$
1998	TEOM at 40°C and TEOM at 30°C	$T_2 = 0.84 \cdot T_3 - 0.69$
T ₁	TEOM at 50°C	
T ₂	TEOM at 40°C	
T ₃	TEOM at 30°C	
BG	Beta gauge	

3.5.8 Exposure calculation

From the full hourly air pollution and meteorological database prepared for Christchurch, with the missing data replaced by the corresponding model predictions (Section 3.5.6), the following daily exposure variables for air pollutants and weather variables for each 24-hour period from midnight to midnight were computed:

- 24-hour average for all three pollutants (PM₁₀, CO and NO₂)
- Maximum one-hour concentration for all three pollutants
- 24-hour average of temperature
- Maximum one-hour temperature
- Minimum one-hour temperature

- 24-hour average of relative humidity

3.6 Population data

Populations of Christchurch census area units (CAU) were obtained for four census years (1986, 1991, 1996 and 2001) from the Statistics New Zealand Census of Population and Dwellings conducted in those years. For the 1991, 1996 and 2001 censuses, Statistics New Zealand provided population data for each CAU by sex, age group (<1, 1-14, 15-24, 25-44, 45-64, 65-84 and 85+ years) and ethnicity (European, NZ Maori, Pacific people, Asian, Others and 'not specified'). These census data were based on 2001 CAU boundaries. The 1996 CAU and 2001 CAU boundaries match each other in Christchurch. A few of 1991 CAU boundaries did not match the 1996 and 2001 boundaries, and as such were mapped to 2001 CAU boundaries by Statistics New Zealand before releasing the census data.

For the 1986 census, total population data for each CAU were obtained from Statistics New Zealand publication (Statistics New Zealand, 1998). These data were based on 1996 CAU boundaries, which match the 2001 boundaries for Christchurch. This ensures that the study area did not change during the study period. CAU populations were summed together to get the total population of Christchurch for 1986, 1991, 1996 and 2001.

Population data used in this analysis refer to the "usually resident population". The census counts people where they are on census night, but also asks about "the address of the person's usual residence". The "usually resident population" is based on the address of a person's usual residence where the person considers himself or herself to live (Statistics New Zealand, 1997). The population data used in this analysis refers to the population which usually lived in Christchurch, irrespective of where they were on the census night.

In order to assure the confidentiality of data, Statistics New Zealand has adopted a policy of randomly rounding all the figures in the published statistical tables and other aggregated census statistics to base three. This is to protect the confidentiality of the information about individual people and to ensure that no person can be identified from the published data. While rounding to base three, the probabilities of rounding up or down are set so that the expected value equals the original count in the long run (Statistics New Zealand, 2002). Rounding data in each cell of a table to base three makes the numbers in each cell the multiples of three.

Population estimates for non-census years

Total Christchurch populations for non-census years were estimated by linear interpolation from the populations of two census years. This assumed an equal increase in total Christchurch population each year between the two census years.

3.7 Mortality data

Mortality data were extracted from the NEOH (New Zealand Environmental and Occupation Health Research Centre) New Zealand mortality database for the period from 1988 to 1998. The NEOH New Zealand mortality database was prepared by combining New Zealand mortality datasets (provided by the New Zealand Health Information Service) with a uniform format of data for all years (Shrestha and Smartt, 2001). Mortality data for 1999 were provided by the New Zealand Health Information Service (NZHIS).

3.7.1 Health domicile code

A deceased person's usual residential address is represented by the Statistics New Zealand health domicile code in the mortality database. The health domicile code is created from the Statistics New Zealand census area unit code and is used for collecting health data. There is a one to one match between the health domicile code and the census area unit code. For deaths registered from 1988 to 1992, the 1986 health domicile code was used, which have been mapped to 1991 codes by NZHIS. The 1991 health domicile code (HDOM91) was used for deaths registered from 1993 to 1997 and the 1996 health domicile code (HDOM96) was used for deaths registered from 1998 to 2002 (New Zealand Health Information Service, 2004). The HDOM91 and HDOM96 codes are linked with the Statistics New Zealand 1991 census area unit code and 1996 census area unit code respectively.

Mortality data with health domicile codes that matched Christchurch census area units, were extracted from the mortality database for the period from 1988 to 1998. Mortality data in this period have two groups of health domicile codes; HDOM91 (linked with the 1991 census area unit code) for pre-1998 deaths and HDOM96 (linked with the 1996 census area unit code) for 1998-1999 deaths.

Except for the domicile codes shown in Table 3-5, the area boundaries of all other HDOM91 matched the area boundaries of HDOM96 in Christchurch. Although there were a few changes in the area boundaries of health domicile codes from 1991 to 1996, changes were within the city of Christchurch. This ensured that mortality data

were extracted from the same study area (Christchurch) for all years in the study period. Census population data were based on 2001 CAUs. Although mortality data and population data were based on the CAUs of different censuses, exact matching of the 1996 CAU boundaries with the 2001 CAU boundaries ensured consistency between population and mortality data.

Table 3-5. Non-matching 1991 and 1996 health domicile codes in Christchurch

HDOM91	1991 CAU		HDOM96	1996 CAU	
	Area unit code	Area unit description		Area unit code	Area unit description
2688	595900	South Brighton	2700	595900	South Brighton
2688	595900	South Brighton	2701	596101	Avon-Heathcote Estuary
2690	596100	Moncks Bay	2701	596101	Avon-Heathcote Estuary
2690	596100	Moncks Bay	2702	596102	Moncks Bay

3.7.2 Causes of death

Causes of deaths were coded according to the International Classification of Disease. Deaths registered up to the year 1999 were coded according to the International Classification of Disease, Version 9 (ICD-9 codes) and the deaths registered in 2000 and after were coded according to ICD code, Version 10 (ICD-10 codes) (New Zealand Health Information Service, 2004). Thus, some of the pre-2000 deaths, which were registered in 2000 (mostly deaths, occurring in the second half of December 1999), were coded in ICD-10 codes. Equivalent ICD-9 codes were identified for these deaths using the mapping documentations developed by the New Zealand Health Information Service to map ICD-9 codes to ICD-10 codes and vice versa (New Zealand Health Information Service, 2000).

Based on the first three digits of the codes, the causes of deaths were grouped into the following broad categories:

- All non-external causes (ICD-9 code: 1-799)
- Circulatory causes (ICD-9 code: 390-459)
- Respiratory causes (ICD-9 code: 460-519)

Non-external cause deaths included all deaths except those due to external causes such as vehicle accidents, poisoning, drowning, fire, etc (ICD-9 code: 800-999). Deaths due to external causes were considered to be independent of air quality and excluding them would reduce potential dilution of the air pollution effect. Because of very small numbers of daily deaths (averages of 3 deaths per day for circulatory mortality and 0.8

death per day for respiratory mortality), circulatory and respiratory mortality were not subdivided further to specific causes of death.

Causes of death were identified based only on the primary underlying cause of death. Other relevant diagnoses and other contributing causes were not analysed to classify the deaths into the above groups. This could exclude some deaths in which air pollution might have played a contributory role.

3.7.3 Age group classification

Due to the small number of daily deaths, only four age groups were used. The age of a deceased person was classified into one of the following groups:

- <1 year
- 1-14 years
- 15-64 years
- 65+ years

Preliminary analysis of daily deaths in the three younger age groups showed no clear association with air pollution, which may be due to a lack of power to detect statistically significant associations in those age groups. Most analyses therefore were focussed on deaths in the population aged 65+ years. While the associations between air pollution and mortality were analysed both for the 65+ years age group population and the whole population (all ages) for non-external cause mortality, the analyses were carried out only for the 65+ years age group population for circulatory and respiratory mortality.

3.7.4 Computation of daily deaths

Daily numbers of deaths for the study period of June 1988 to December 1999 were computed for the following subsets of death:

- Non-external cause mortality of all ages
- Non-external cause mortality of those aged 65+ years
- Circulatory mortality of those aged 65+ years
- Respiratory mortality of those aged 65+ years

All analyses were separately carried out for each of the above subsets of death in this thesis.

3.8 Statistical analysis

Statistical modelling techniques used in each chapter are briefly mentioned here, with details provided in the “Methods” section of the respective chapter.

The first part of this study (analysis of research objectives 1 to 4 in Chapters 6 to 8) is a time series study with “day” as the unit of analysis. Time series study design associates daily variation in mortality with daily variation in air pollutant concentrations after controlling for the variables that vary over time such as a long-term trend and seasonal variations in daily mortality, weather variables and other pollutants. The second part (analysis of research objective 5 in Chapter 9) is an ecological cross-sectional study.

Poisson regression models were used to associate daily air pollutant concentrations with daily mortality controlling for a long-term trend and seasonal variations in daily mortality, and the confounding effects of weather variables. In Chapter 6, the models were used to quantify the acute effects of single day exposure to air pollutants on daily mortality.

Using the LOESS smooths with different smoothing window sizes, time series data were decomposed into a number of independent time series representing daily variations in those series, which vary with the period of different time scales such as long-term variation, mid-term variation and short-term variation. The associations between daily PM₁₀ and daily mortality were analysed using the mid-term variations of daily data in Chapter 7 to examine if any association between short-term exposure to air pollution and daily mortality was due to mortality displacement by a few days. Polynomial distributed lag models were used in Chapter 8 to estimate the extended effects of PM₁₀ on daily mortality, distributed over several days and to analyse the shape of the distribution of effects of PM₁₀ on daily mortality over lag days. Logistic regression models across spatial units were used to analyse the association between annual average PM₁₀ concentrations and annual mortality in Chapter 9. These statistical methods are discussed in detail in the respective chapters.

Effect size estimates were presented with their 95% confidence intervals. Statistical significance of the results were generally assessed at 5% level of significance (p-value < 0.05), unless otherwise stated.

Chapter 4: Descriptive analysis of population and mortality data

4.1 Introduction

This chapter is divided into two sections. The first section describes the population structure of Christchurch and compares it between the three censuses. The second section provides a descriptive analysis of mortality, including an analysis of mortality trends over the study period and seasonal variations in mortality.

4.2 Descriptive analysis of population data

Christchurch has the second largest population of all the territorial local authorities in New Zealand after Auckland, making it the second largest urban area in New Zealand. According to the 2001 Census of Population and Dwellings, Christchurch had the usually resident population of approximately 316 thousand, which was about 8.5% of the country's 3.7 million residents (Statistics New Zealand, 2002).

The population growth rate in Christchurch fluctuated over the study period of 1988 to 1999. Between the census years 1986 and 1991, the average annual growth was 0.49%, which increased to 1.38% per year between 1991 and 1996. The rate of increase slowed to 0.46% per year between the 1996 and 2001 censuses (Statistics New Zealand, 1998; 2002). Figure 4-1 shows the numbers of males and females in the population in the three census years. The percentages of males and females were approximately the same in all three census years. The sex ratio (males per 100 females) remained relatively constant at 93 in the whole study period.

4.2.1 Age distribution

In Christchurch, a little over one per cent of the total population were under one year old, approximately 18% were between 1 and 14 years old, around 67% were between 15 and 64 years old and a little over 13% were 65+ years old (Figure 4-2). The proportion of less than one year old population decreased slightly between 1991 and 2001 whereas the proportion of people aged 65+ years increased slightly in the same period.

Figure 4-1. Comparison of the male and female population in the three census years (percentages indicate proportion of each sex), Christchurch

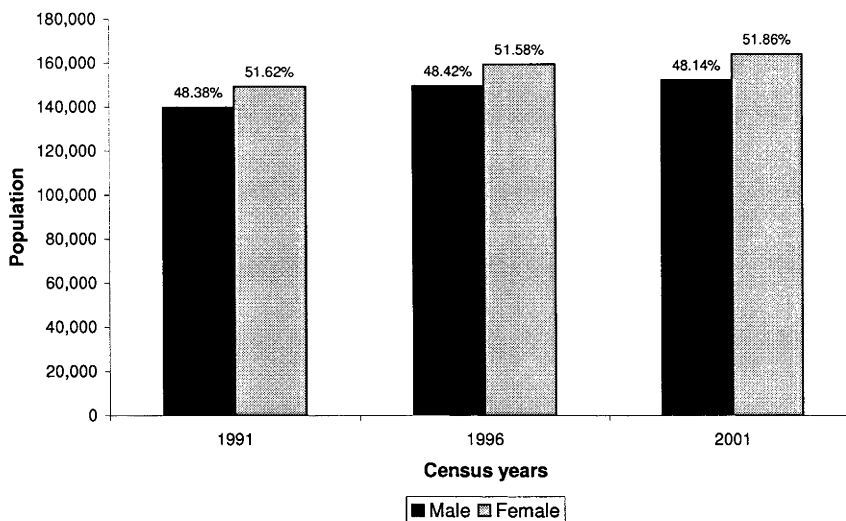
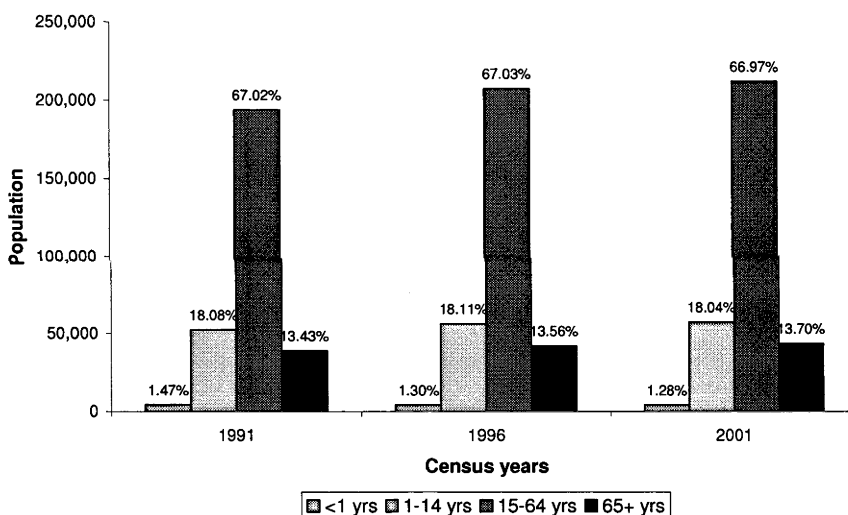


Figure 4-2. Age distribution of Christchurch population in the three census years



4.3 Descriptive analysis of mortality data

4.3.1 Mortality by cause of death

The total number of deaths in Christchurch during the study period of 1988 to 1999 was 31,921, with an average of 2,660 deaths each year, or 7.3 deaths each day. About 94.3% of these deaths were due to non-external causes and the remainder (about 5.7%) were due to external causes, which included accidents, poisoning, drowning, fire etc. (Table 4-1). Among non-external deaths, circulatory disease was the major cause of deaths, constituting about 48% of all non-external deaths and about 45% of total deaths.

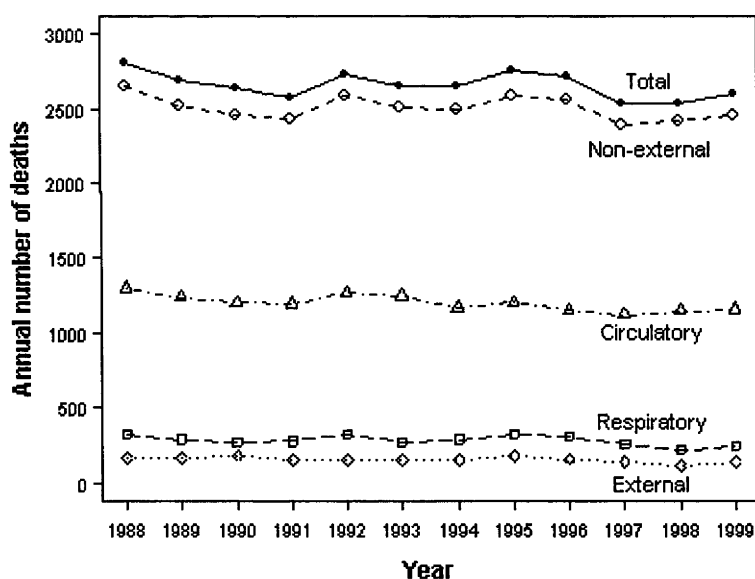
Respiratory mortality constituted about 11% of non-external mortality and 10.5% of total mortality.

Table 4-1. Mortality by cause of death, Christchurch 1988 – 1999

	All causes	Non-external causes	External causes	Circulatory causes	Respiratory causes
Mortality count	31,921	30,111	1,810	14,327	3,352
Percentage of all causes mortality	100%	94.3%	5.7%	44.9%	10.5%
Daily average mortality	7.3	6.9	0.4	3.3	0.8

Figure 4-3 shows the number of deaths by year for different causes of deaths for the twelve years from 1988 to 1999. The trends for the annual number of deaths showed slight declines in all cause mortality, non-external mortality, circulatory mortality and respiratory mortality over the twelve year period.

Figure 4-3. Mortality trends by cause of death, Christchurch 1988 – 1999



4.3.2 Age distribution of cause-specific mortality

Except for external cause deaths, the age distributions of mortality for the other three categories (non-external, circulatory and respiratory) were similar (Figure 4-4). Table 4-2 shows the percentage of deaths by age group within each mortality category. For non-external, circulatory and respiratory cause mortality, those aged 65+ years made up the highest percentage of deaths (over 80% within each category) whereas for external cause mortality, the highest percentage occurred in the 15 – 64 years age group,

followed by the 65+ years group. Those under 15 years of age constituted a very small proportion of deaths, less than 2% within each category except external cause mortality.

Figure 4-4. Age distribution of mortality within each category, Christchurch 1988 – 1999

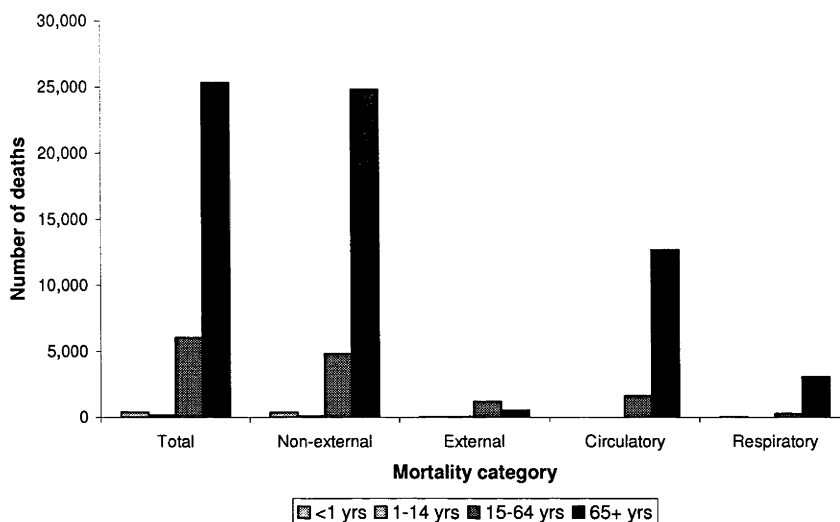
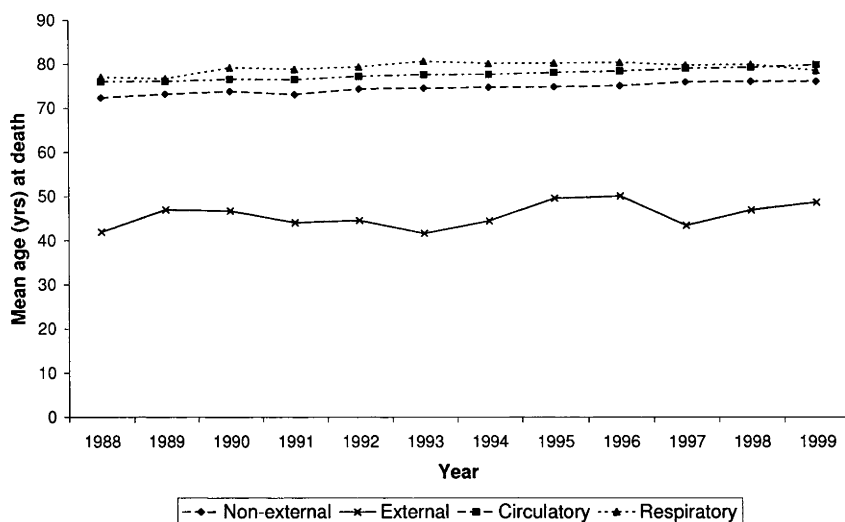


Table 4-2. Percentage of deaths by age group within each mortality category, Christchurch 1988 – 1999

Age group	All causes	Non-external causes	External causes	Circulatory causes	Respiratory causes
<1 year	1.2	1.2	0.8	0.03	0.5
1 – 14 years	0.5	0.3	3.4	0.03	0.2
15 – 64 years	18.9	16.03	66.9	11.4	7.9
65+ years	79.4	82.4	28.8	88.5	91.5

The mean age of Christchurch residents who died due to non-external causes was 74.5 years (sd 15.7 years). For circulatory and respiratory mortality, the mean age at death was 77.6 years (sd 11.5 years) and 79.2 years (sd 12.5 years) respectively. The trend of mean age at death for each mortality category over the twelve year period is shown in Figure 4-5. There were slight upward trends in the mean age of death over the twelve year period for all the causes presented here.

Figure 4-5. Trend of mean age at death by mortality category, Christchurch 1988 – 1999



4.3.3 Seasonal variation in mortality

The age groups under 15 years (<1 year and 1-14 years) constituted a very small percentage of total number of deaths, less than 2% (Section 4.3.2). Due to the very low number of daily deaths in these age groups, the age groups under 15 years were merged with the 15-64 years age group to analyse the seasonal variation in daily mortality. Since the population aged 65+ years is likely to have the highest mortality risk associated with air pollution, seasonal variation, and the association between air pollution and mortality were analysed separately for total population and for the population aged 65+ years for non-external mortality. Since most of the circulatory and respiratory deaths occurred in the population aged 65+ years (more than 88% of circulatory and respiratory deaths), analysis was carried out only for the population aged 65+ years for those mortality categories.

Figures 4-6 and 4-7 show time series plots of daily mortality. A centred 31-day moving average smoothing filter was applied to reveal any pattern present in each time series plot. Figure 4-8 shows the monthly variations in average number of daily deaths. There was a strong seasonal variation in non-external cause mortality, circulatory mortality and respiratory mortality in the population aged 65+ years, with peaks occurring during the winter months (June-August) and troughs during the summer months (December-February). Non-external mortality in the population under 65 years old did not have a noticeable seasonal variation. A marked seasonal variation in non-external cause mortality of all ages was related to the seasonal variation in non-external

cause mortality of the elderly population (65+ years old). External cause mortality did not show any seasonal pattern.

Figure 4-6. Time series plots of daily deaths with a 31-day moving average filter, Christchurch 1988 – 1999

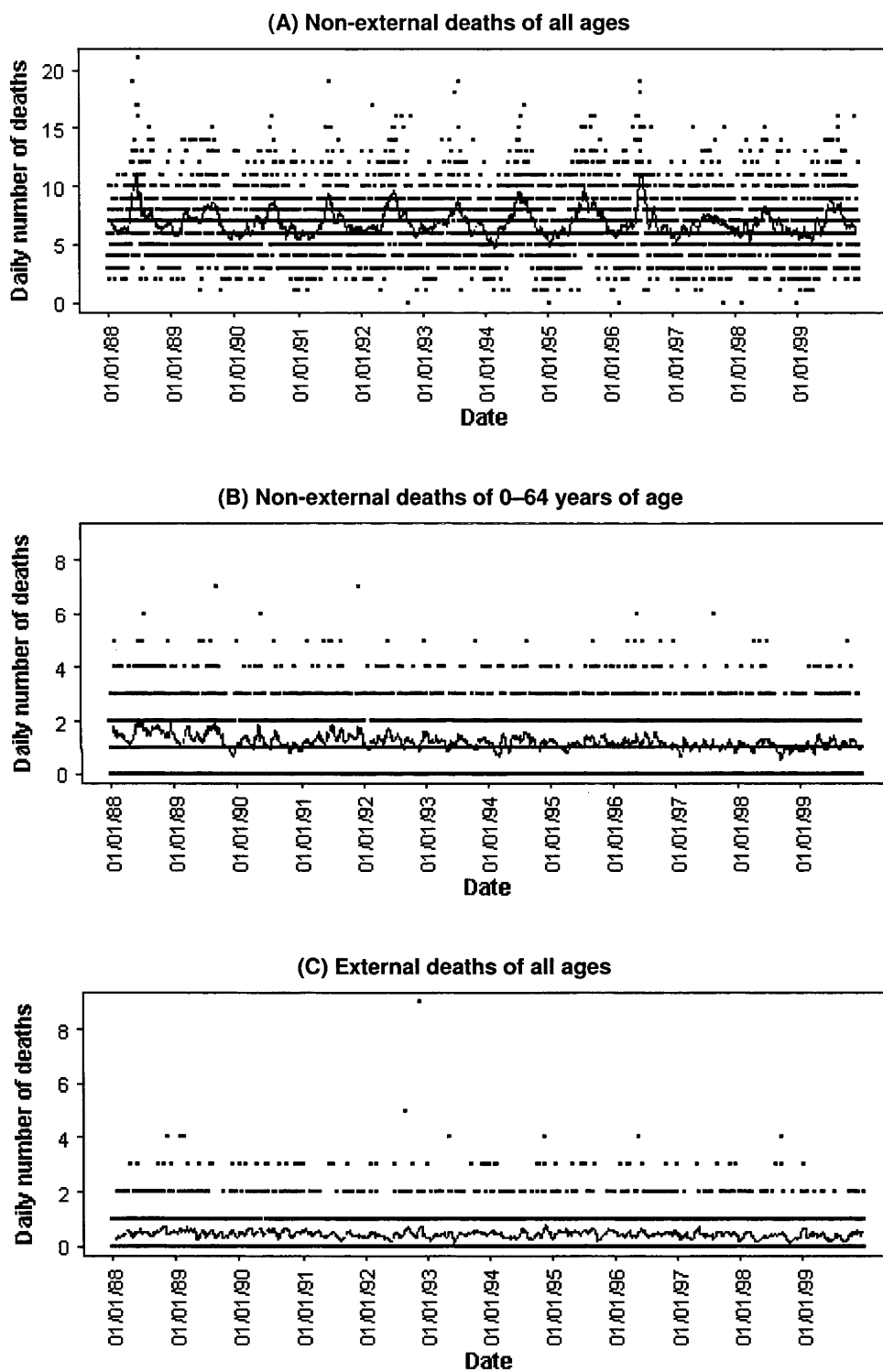


Figure 4-7. Time series plots of daily deaths with a 31-day moving average filter in the population aged 65+ years, Christchurch 1988 – 1999

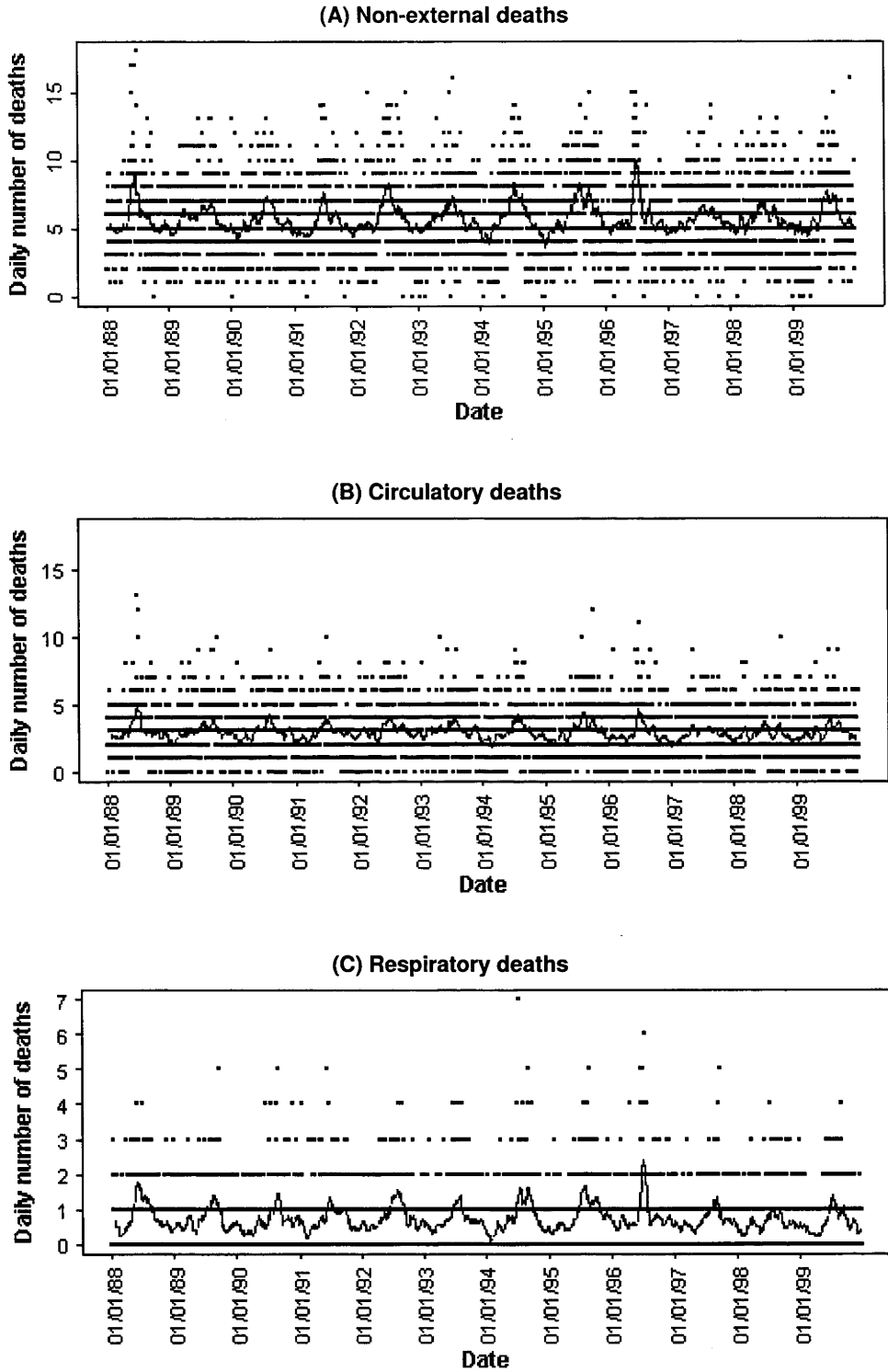
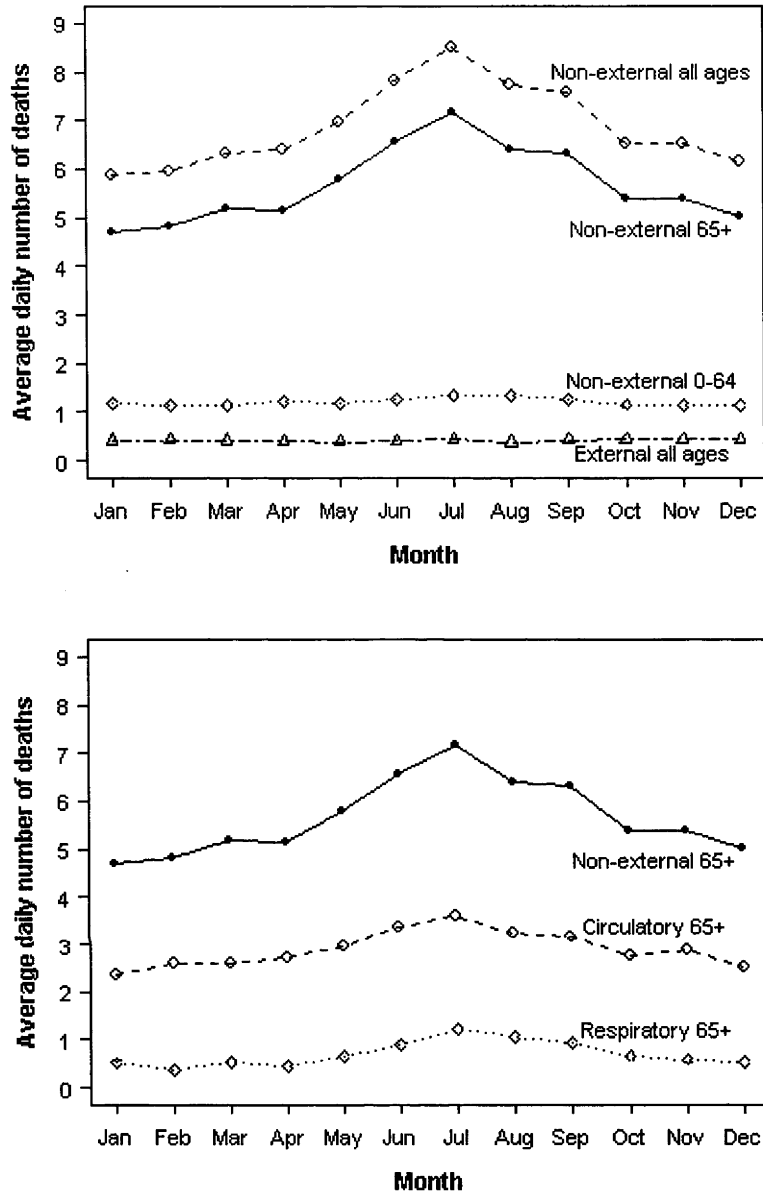


Figure 4-8. Monthly variations of average daily deaths by mortality category, Christchurch 1988 – 1999



In order to estimate the seasonal differences in daily mortality, the months were grouped into three different periods based on the dominant source of air pollution in those months. The first four months from January to April and the last four months from September to December, when motor vehicle emissions were the primary source of air pollution with very small contributions from the emissions from home heating to the ambient air pollution, were grouped as the warmer period 1 and the warmer period 2 respectively. The four months from May to August, when emissions from home heating were the dominant source of air pollution, were grouped as the cooler period. Relative risks of mortality for each period were calculated for each mortality category using Poisson regression models with the warmer period 1 (January – April) as a reference

period (Table 4-3). Except for external cause mortality, the risks of dying were significantly higher in the cooler period than in the warmer periods. Relative risks were similar for non-external mortality and circulatory mortality of the 65+ years age group. In the population aged 65+ years, the risks of dying due to non-external causes, circulatory causes and respiratory causes were significantly higher in the warmer period 2 (September – December) than in the warmer period 1 (January – April). Respiratory mortality in 65+ years age group had the strongest seasonal variation, with the cooler period's average daily mortality 2.07 times higher (95% CI: 1.89, 2.27) than the average daily mortality in the warmer period 1.

Table 4-3. Relative risk of mortality (95% CI) in each period for different mortality categories, Christchurch 1988 – 1999

Season	65+ years population			Non-external mortality of less than 65 years old	External cause mortality of all ages
	Non-external mortality	Circulatory mortality	Respiratory mortality		
Warmer period 1 (Jan-Apr)	Reference	Reference	Reference	Reference	Reference
Cooler period (May-Aug)	1.30*** (1.27, 1.35)	1.28*** (1.23, 1.34)	2.07*** (1.89, 2.27)	1.09** (1.03, 1.17)	0.96 (0.86, 1.08)
Warmer period 2 (Sep-Dec)	1.11*** (1.08, 1.15)	1.09*** (1.05, 1.15)	1.46*** (1.32, 1.61)	1.004 (0.94, 1.07)	1.05 (0.94, 1.18)

* p-value < 0.05; ** p-value < 0.01; *** p-value < 0.001

The associations between daily air pollution and mortality were analysed by season in time series analysis in Chapter 6. For the seasonal analysis, the two warmer periods (warmer period 1 and warmer period 2), covering eight months from September to the following April, were combined as the emissions from home heating generally contribute very low to the ambient air pollution in those periods. Motor vehicle emissions were the primary source of air pollution in those months.

4.3.4 Daily variation in mortality across a week

Average daily numbers of deaths by day of week with 95% confidence interval bars are shown in Figure 4-9. The average daily number of deaths did not vary across a week in any discernible manner for any mortality category except for external cause mortality. The average daily number of deaths due to external causes was slightly lower on Tuesday, Wednesday and Thursday than on other days. Relative risks of mortality for each day of the week were calculated using Poisson regression models with Sunday as a

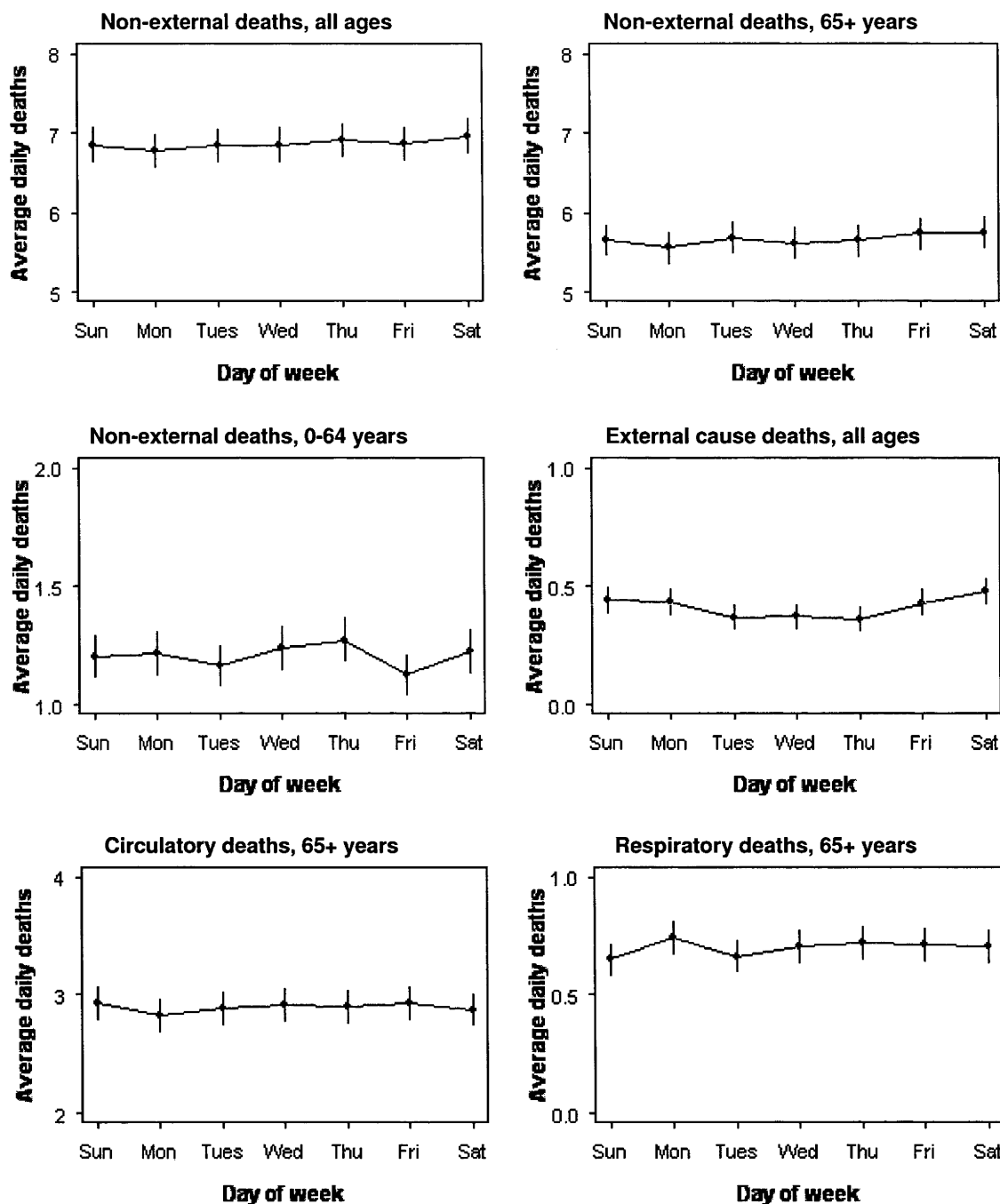
reference day (Table 4-4). The risk of dying due to respiratory causes in the population aged 65+ years was high on Monday compared with the risk on Sunday. It is not clear why the number of respiratory deaths of those aged 65+ years was higher on Monday than on Sunday. It may be due to registration errors (Barnett and Dobson, 2004). However, there was no evidence of higher deaths occurring on Monday for other causes of deaths. The results did not provide any evidence that the risks of mortality were associated with days of the week for non-external mortality of all ages or for non-external deaths and circulatory deaths of those aged 65+ years.

Table 4-4. Relative risk (95% CI) of mortality for each day of the week for different mortality categories, Christchurch 1988 – 1999

Day of week	65+ years population			Non-external mortality of less than 65 years old	External cause mortality of all ages
	Non-external mortality	Circulatory mortality	Respiratory mortality		
Sunday	Reference	Reference	Reference	Reference	Reference
Monday	0.98 (0.94, 1.03)	0.96 (0.90, 1.03)	1.14* (1.00, 1.31)	1.01 (0.91, 1.11)	0.98 (0.83, 1.16)
Tuesday	1.01 (0.96, 1.05)	0.99 (0.93, 1.05)	1.01 (0.88, 1.16)	0.97 (0.87, 1.07)	0.83* (0.69, 0.99)
Wednesday	0.99 (0.95, 1.04)	0.99 (0.93, 1.06)	1.08 (0.95, 1.24)	1.03 (0.93, 1.14)	0.84[#] (0.71, 1.01)
Thursday	0.99 (0.95, 1.05)	0.99 (0.93, 1.06)	1.11 (0.97, 1.27)	1.06 (0.96, 1.17)	0.81* (0.68, 0.97)
Friday	1.02 (0.97, 1.06)	1.00 (0.94, 1.07)	1.09 (0.96, 1.26)	0.93 (0.84, 1.03)	0.97 (0.82, 1.15)
Saturday	1.02 (0.97, 1.07)	0.98 (0.92, 1.05)	1.08 (0.94, 1.24)	1.01 (0.92, 1.12)	1.08 (0.92, 1.28)

[#] p-value < 0.1; * p-value < 0.05; ** p-value < 0.01; *** p-value < 0.001

Figure 4-9. Variations of average daily deaths across a week by mortality category, Christchurch 1988 – 1999



Note: Y-axis scales vary between plots.

4.3.5 Distribution of daily mortality

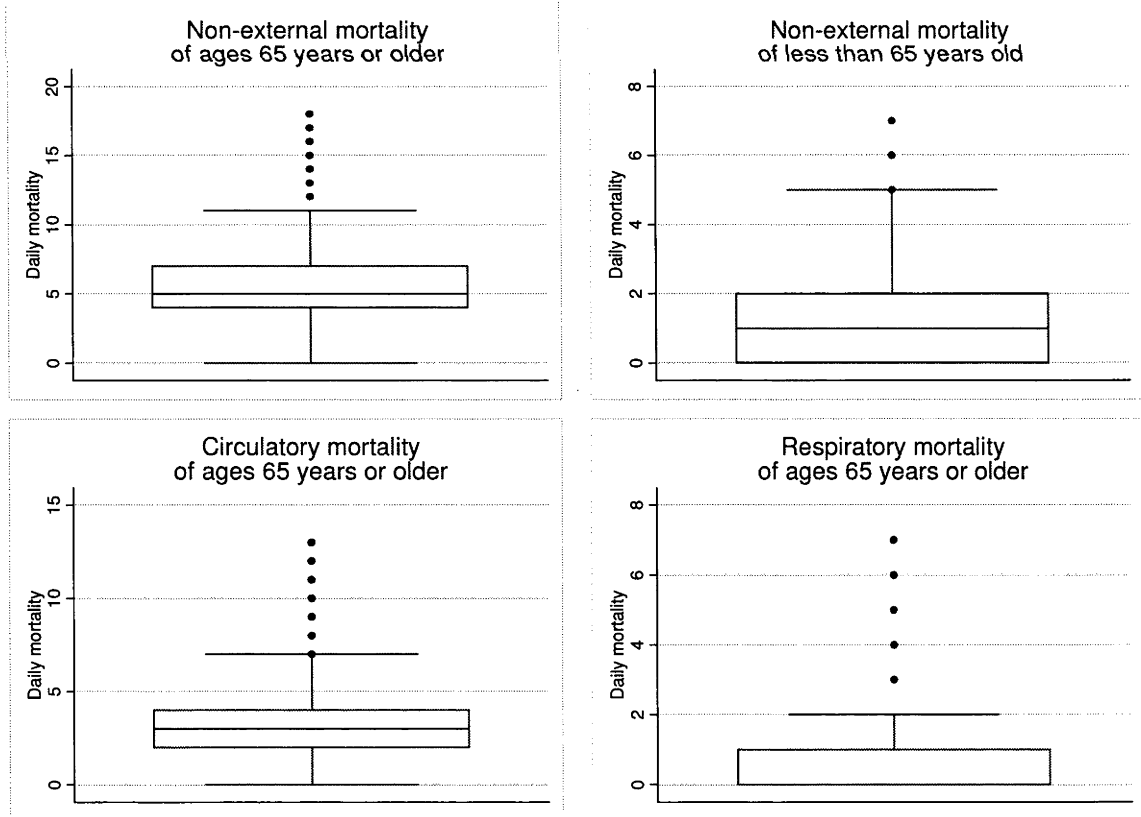
Table 4-5 summarises daily mortality data for each mortality category and Figure 4-10 shows the distribution of daily mortality by mortality category. The mean number of daily respiratory deaths in the population aged 65+ years was about 12% of the mean number of daily non-external cause deaths of the same age group. About 50% of daily non-external deaths in the population aged 65+ years were due to circulatory causes. The distributions of daily deaths were skewed towards higher values (right skewed) for

all mortality categories, with daily respiratory mortality having the most skewed distribution.

Table 4-5. Summary statistics for daily mortality for each mortality category, Christchurch 1988 – 1999

Mortality category	Mean (sd)	Minimum	Quartile			Maximum
			1 st	Median	3 rd	
Non-external mortality:						
less than 65 years old	1.2 (1.1)	0	0	1	2	7
65+ years old	5.7 (2.6)	0	4	5	7	18
Circulatory mortality (65+ years old)	2.9 (1.8)	0	2	3	4	13
Respiratory mortality (65+ years old)	0.7 (0.9)	0	0	0	1	7
External mortality of all ages	0.4 (0.7)	0	0	0	1	9

Figure 4-10. Distribution of daily mortality by mortality category, Christchurch 1988 – 1999



4.4 Summary

This chapter provided a general description of the population structure of Christchurch. Mortality data were summarised by age group for non-external mortality, circulatory mortality and respiratory mortality. More than 80% of all non-external cause deaths occurred in the population aged 65+ years. For both circulatory and respiratory mortality categories, the 65+ years population constituted more than 88% of deaths. On average, there were 5.7 non-external cause deaths per day in the population aged 65+ years. For circulatory and respiratory cause deaths, the average daily numbers of deaths were 2.9 and 0.7 respectively.

Annual numbers of deaths declined slightly over the study period for all mortality categories analysed. Except for external cause deaths, there were strong seasonal variations in other mortality categories, with peaks occurring during the winter months (June – August) and troughs during the summer months (December – February).

Chapter 5: Descriptive analysis of weather and air pollution data

5.1 Introduction

This chapter provides a descriptive analysis of weather (temperature and relative humidity) and air pollutant (PM_{10} , CO and NO_2) data. Air pollution levels depend highly upon local weather conditions. Emissions from the burning of solid fuels for domestic heating and the local meteorological conditions cause very high levels of air pollution during winter in Christchurch. Due to local topography, temperature inversions occur frequently during calm evenings in the winter. Temperature inversions and calm weather restrict the dispersion of air pollutants both horizontally and vertically, resulting in higher levels of air pollution. Seasonal variations in weather data and air pollutants and the correlations between them are examined in this chapter.

5.2 Seasonal variation in weather variables

Figures 5-1 and 5-2 show time series plots of daily temperature and daily relative humidity from June 1988 to December 1999, with a centred 31-day moving average smoothing filter. Time series plots of three temperature variables (average hourly temperature, maximum hourly temperature and minimum hourly temperature) are shown in Figure 5-1. As expected, temperature had a strong seasonal variation, with lower levels occurring in the winter months (June – August) and higher levels occurring in the summer months (December – February). The seasonal patterns are illustrated in the plots showing monthly variations in daily temperature and daily relative humidity (Figure 5-3). Seasonal variations were consistent for all three temperature variables. Relative humidity had a seasonal variation opposite to the seasonal variation of temperature. Monthly average relative humidity was higher between May and July and lower between October and December.

Figure 5-1. Time series plots of daily temperature with a 31-day moving average filter, Christchurch June 1988 – December 1999

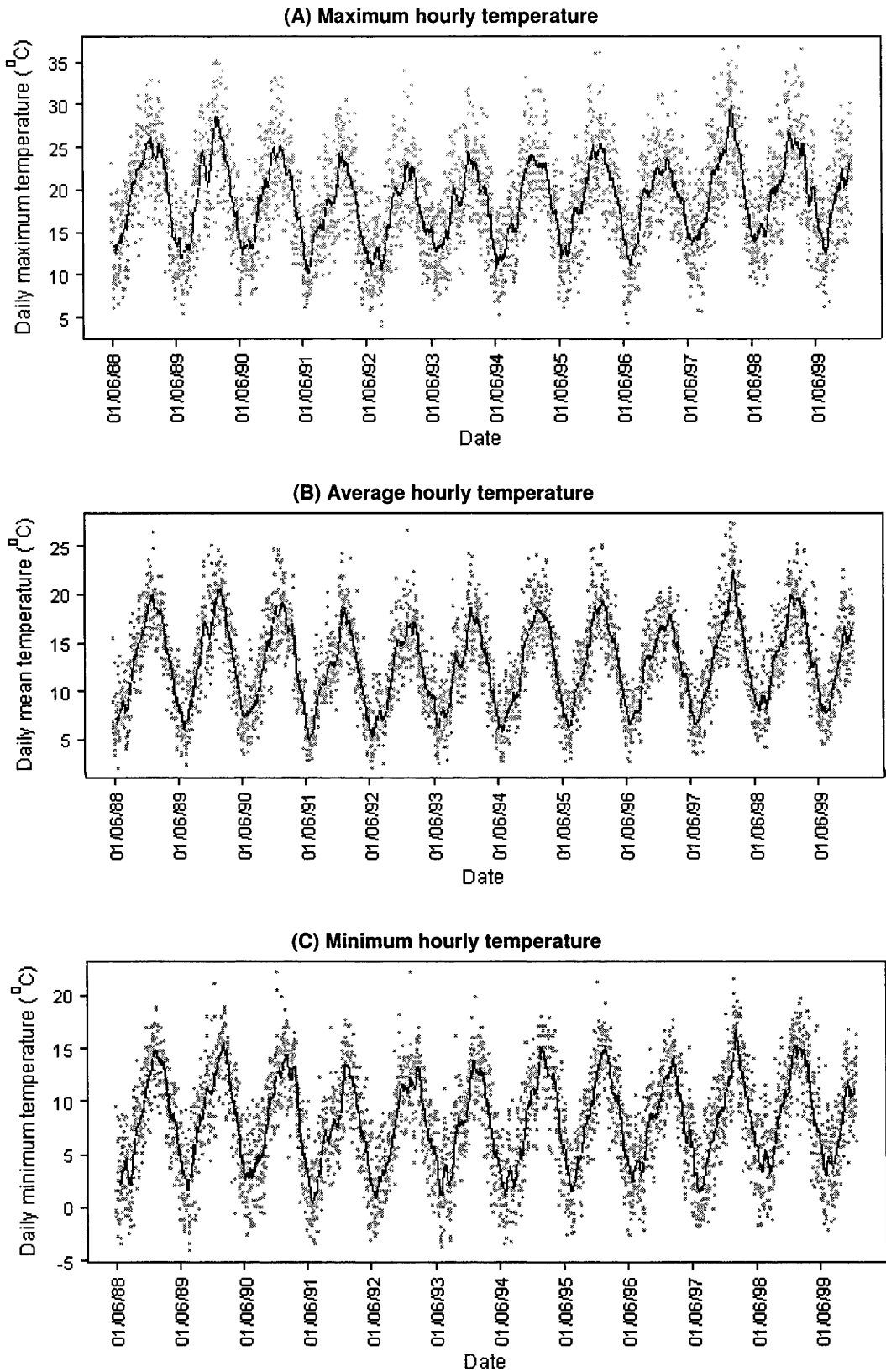


Figure 5-2. Time series plot of daily average of hourly relative humidity with a 31-day moving average filter, Christchurch June 1988 – December 1999

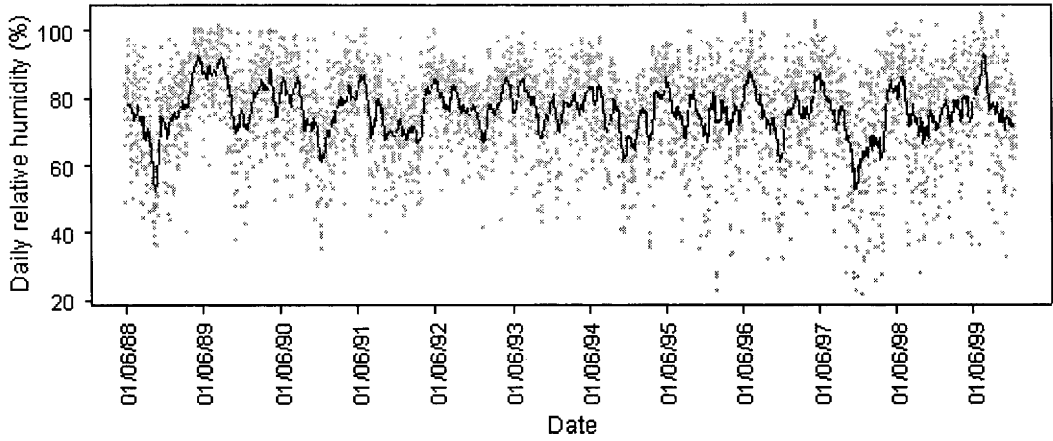
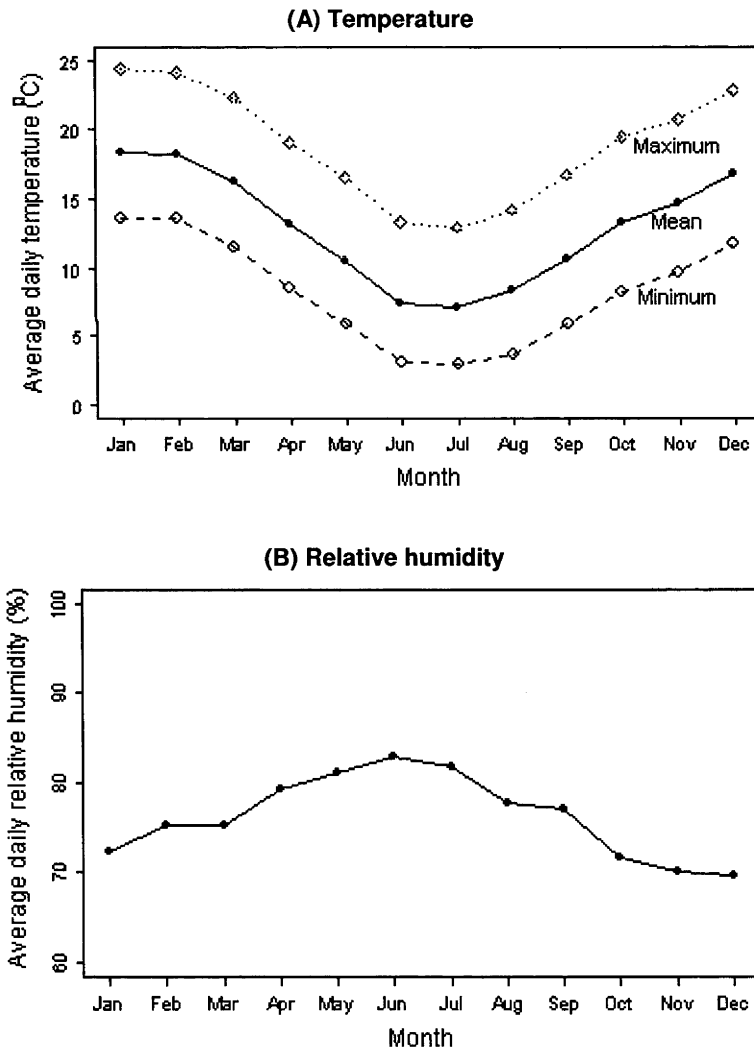


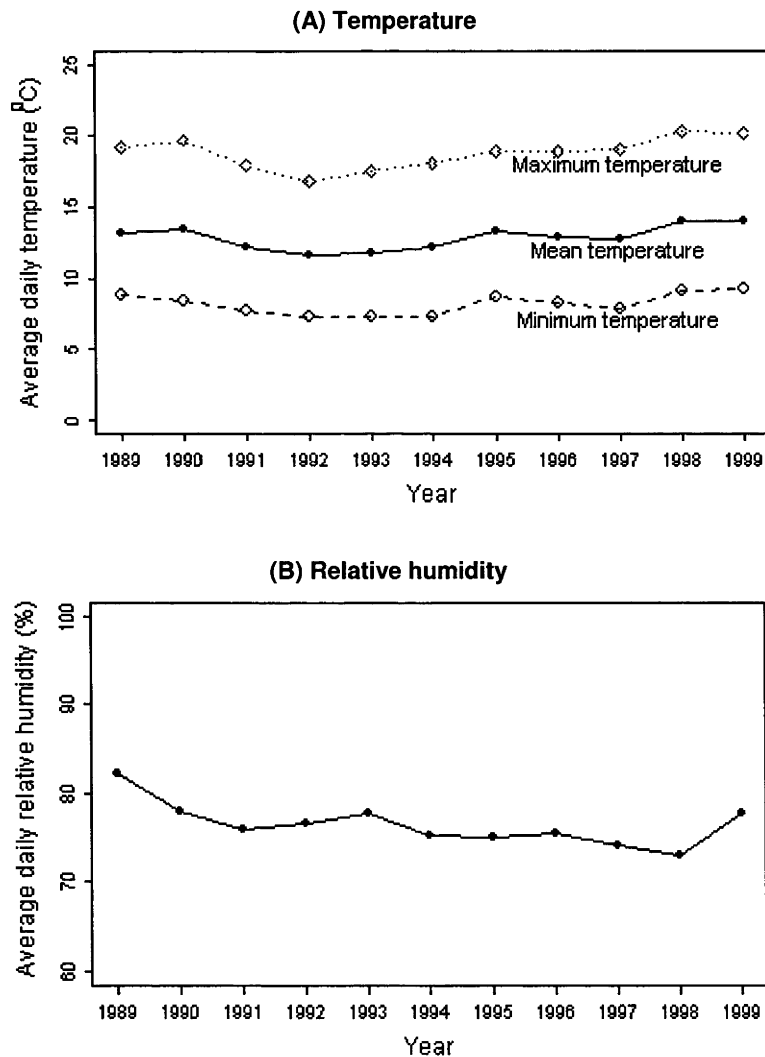
Figure 5-3. Monthly variations in average daily temperature and average daily relative humidity, Christchurch June 1988 – December 1999



5.3 Annual trends in weather variables

No long-term trend in daily temperature or daily relative humidity was noticeable in the time series plots in Figures 5-1 and 5-2. Figure 5-4 shows annual averages of daily temperature and daily relative humidity from 1989 to 1999. Since data were available only from June 1988 for this study (almost half of the warmer period data were not available for 1988), the 1988 annual averages were not comparable with annual averages for the other years and thus were not plotted in Figure 5-4.

Figure 5-4. Annual trends in average daily temperature and average daily relative humidity, Christchurch 1989 – 1999



There were slight upward trends in the annual averages of daily temperature for all three temperature variables (hourly maximum temperature, hourly average temperature and hourly minimum temperature). The upward trends were clearly visible after 1992. There was a slight downward trend in the annual average of daily relative humidity.

5.4 Seasonal variation in air pollution variables

Figure 5-5 shows time series plots of daily PM₁₀, CO and NO₂ concentrations from June 1988 to December 1999 with a centred 31-day moving average smoothing filter. Time series plot of daily NO₂ concentrations shows that daily NO₂ concentrations in the winter season in the years after 1992 were lower than the concentrations in the pre 1992 winter season with no apparent change in the warmer seasons. The standard deviation of daily NO₂ concentrations after 1992 was little more than half of the standard deviation of daily NO₂ concentrations before 1992. The other two pollutants (PM₁₀ and CO) did not have any specific long-term trend. The differences in NO₂ concentrations before and after 1992 may be due to the increasing number of old vehicles being gradually replaced by the vehicles fitted with catalytic converters and other emission control equipment.

All three pollutants (PM₁₀, CO and NO₂) had strong seasonal patterns, with higher levels occurring between May and August and lower levels occurring between September and April. The seasonal patterns are illustrated in the plots showing monthly variations in daily PM₁₀, CO and NO₂ concentrations (Figure 5-6). Though all three pollutants had similar seasonal patterns, the monthly variation of NO₂ was slightly different from those of PM₁₀ and CO. While the monthly averages of PM₁₀ and CO were almost constant from October to March, when the concentration levels were low, the monthly averages of NO₂ gradually increased in the first three months of the year (January – March) and decreased in the last three months of the year (October – December), after having attained higher concentration levels in the winter.

Figure 5-5. Time series plots of daily air pollutant concentrations with a 31-day moving average filter, Christchurch June 1988 – December 1999

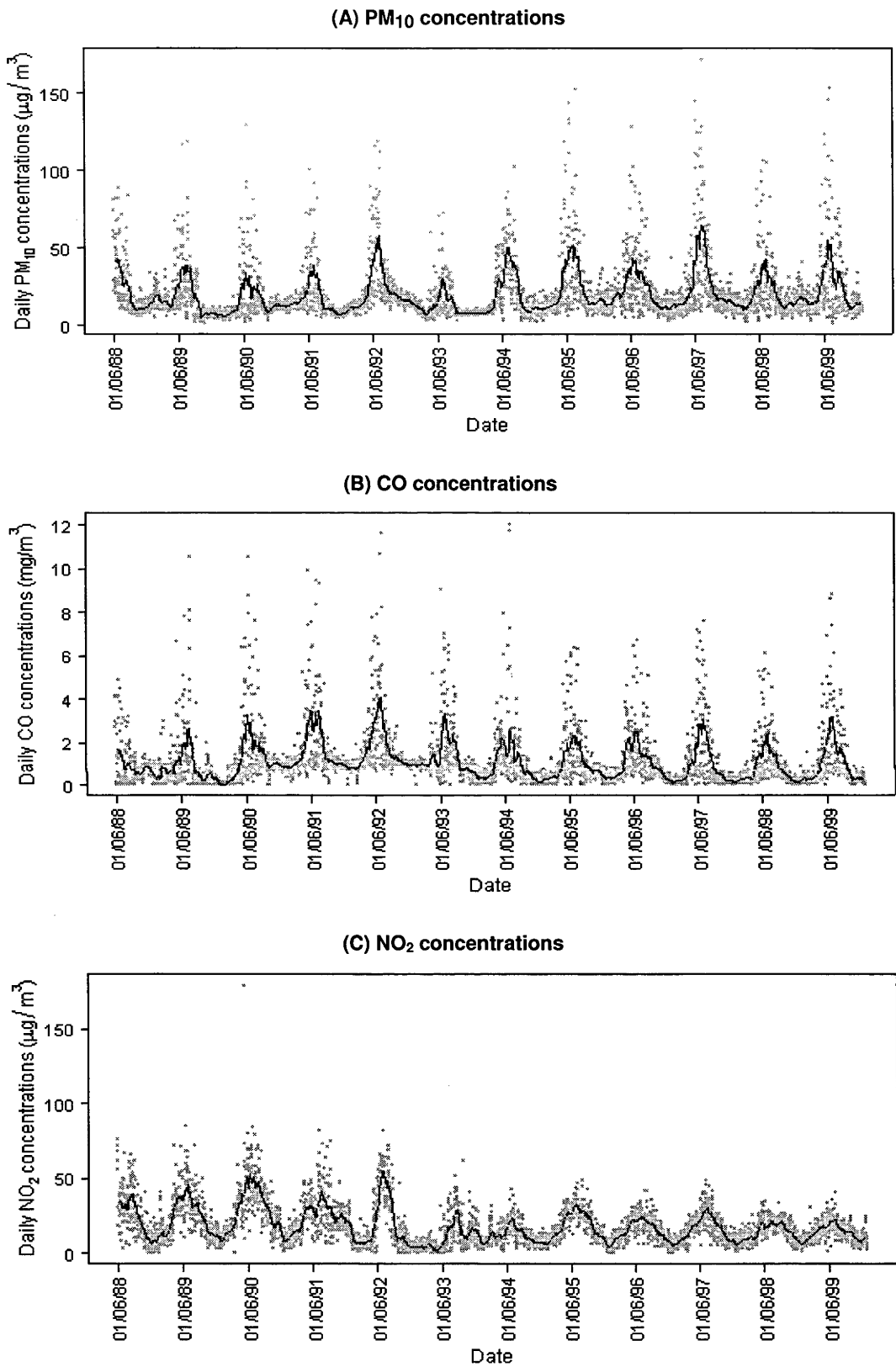
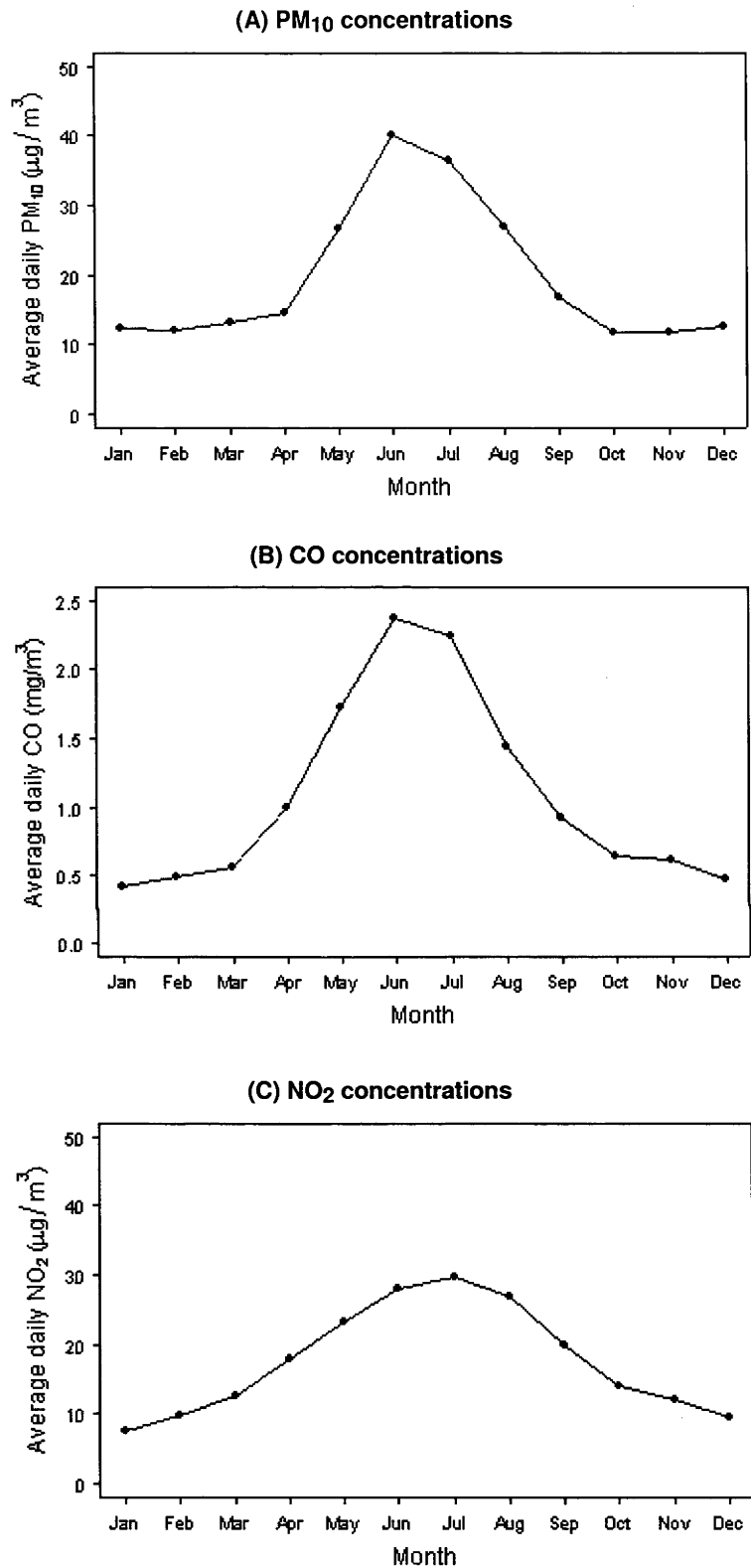


Figure 5-6. Monthly variations in average daily air pollutant concentrations, Christchurch June 1988 – December 1999



All three pollutants had peaks in June and July, which are the coldest and the calmest months (Figure 5-3). The low wind speed and the presence of temperature inversions during these months cause highly stable air. Besides meteorological

conditions, the higher concentrations of pollutants during June and July were also due to higher levels of emissions from domestic heating, such as the burning of coal and firewood to keep houses warm.

The primary sources of air pollutants are different for summer and winter in Christchurch. Vehicle emissions are the main source of air pollution in the summer. Apart from vehicle emissions, sea spray, dusts and pollens may also contribute some pollutants especially PM₁₀. The contributions of these sources are likely to be high during the summer when wind speeds are elevated. In the winter, air pollution from domestic heating, which dominates all other sources, is added to the summer level air pollution. This results in very high pollution levels in the winter. Another source of air pollution is industry emissions, which is approximately the same throughout the year.

In the winter months, people burn firewood during cold mornings and evenings to keep their houses warm. The sudden increase in PM₁₀ concentrations in the coldest months is most likely due to the burning of firewood in these months. The concentrations of NO₂ started gradually increasing during the summer months as well when people had not started burning firewood. Emissions of NO₂ in the environment is mainly caused by vehicle emissions, which is more likely to be the same throughout the year, with slightly lower level of emissions in December and January. The gradual increase and decrease in NO₂ concentrations is likely to be mainly due to changes in wind speed and in temperature inversion conditions.

5.5 Annual trends in air pollution variables

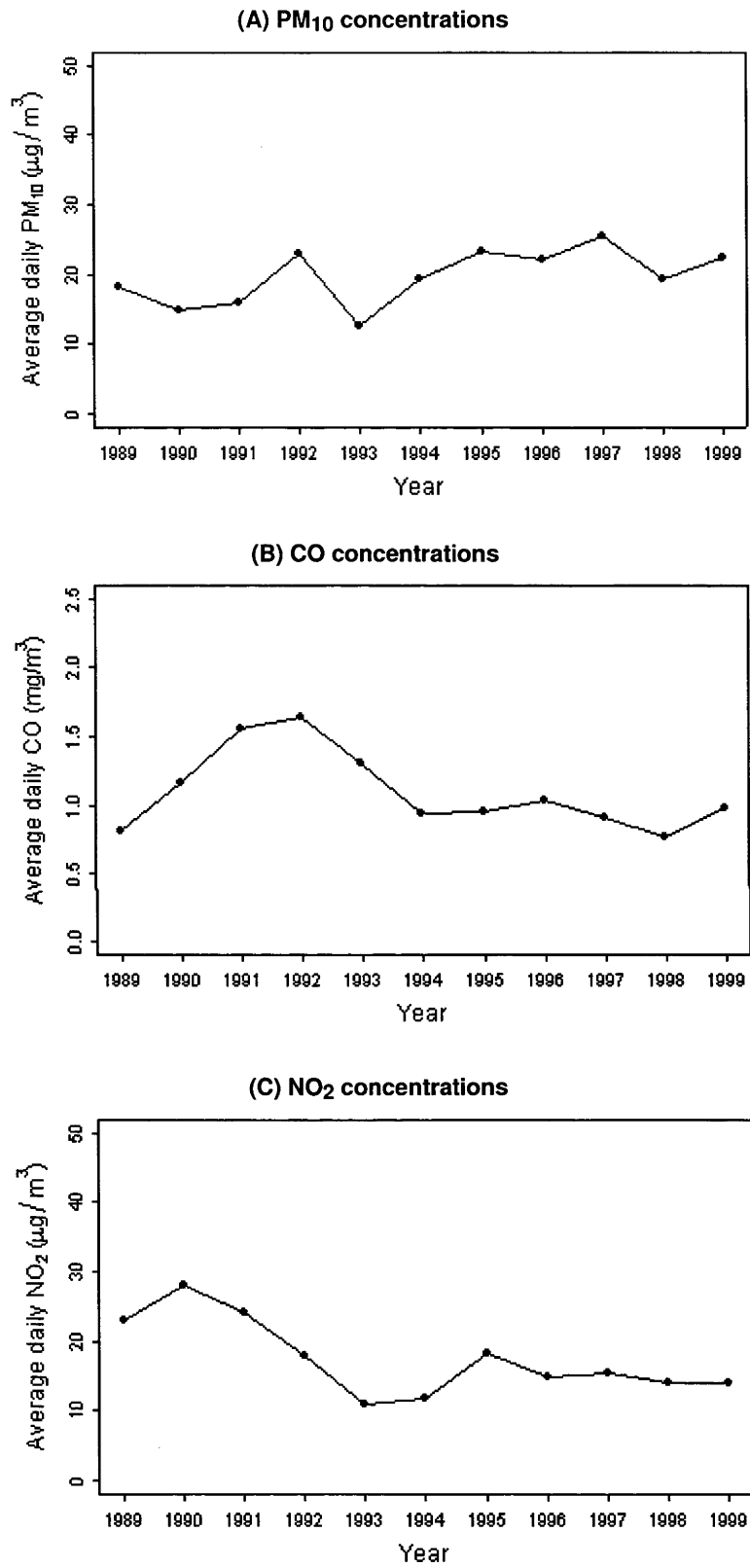
Time series plots of air pollutant concentrations in Figure 5-5 show no specific long-term trend in daily PM₁₀ and CO concentrations. The NO₂ plot shows that daily NO₂ concentrations in the winter season in the years after 1992 were lower than daily NO₂ concentrations of the pre 1992 winter season, with no apparent changes in the warmer season. The higher concentrations in the pre 1992 winters caused higher annual averages of daily NO₂ concentrations in the years earlier than 1992 (Figure 5-7). The plot of the annual averages of daily CO concentrations shows that annual averages were higher in the years between 1990 and 1993. There was no specific long-term trend in the annual averages of daily PM₁₀ concentrations (Figure 5-7).

The primary sources of three main pollutants were different in Christchurch, with domestic home heating being the dominant source of PM₁₀ and emissions from motor vehicles being the main source of NO₂. Both domestic home heating and emissions from motor vehicles contributed almost equally to CO, with emissions from

motor vehicles contributing about 4% more than from domestic home heating (Scott and Gunatilaka, 2004). Differences between the long-term trend of PM₁₀ and those of CO and NO₂ may be due to the increasing number of old vehicles being gradually replaced by the vehicles fitted with catalytic converters and other emission control equipment, which might have reduced NO₂ and CO concentrations dramatically without making any noticeable change in PM₁₀ concentrations.

Annual averages for 1988 were not plotted in Figure 5-7 as data were not available for the full year. Since the period of this study commenced June 1988, only the seven months of data from June to December were available for 1988. Almost half of the warmer period data were not available for 1988.

Figure 5-7. Annual trends in average daily air pollutant concentrations, Christchurch 1989 – 1999



5.6 Distribution of weather and air pollution variables

Summary statistics for daily weather variables and air pollutants are presented in Table 5-1 and their distributions are shown in Figure 5-8. Except for daily temperature, all other variables had skewed distributions, with all three pollutants having highly skewed distributions. While the distribution of daily relative humidity is left skewed, the distributions of all three daily pollutants were highly right skewed.

Figure 5-8. Distributions of daily weather variables and air pollutants, Christchurch June 1988 – December 1999

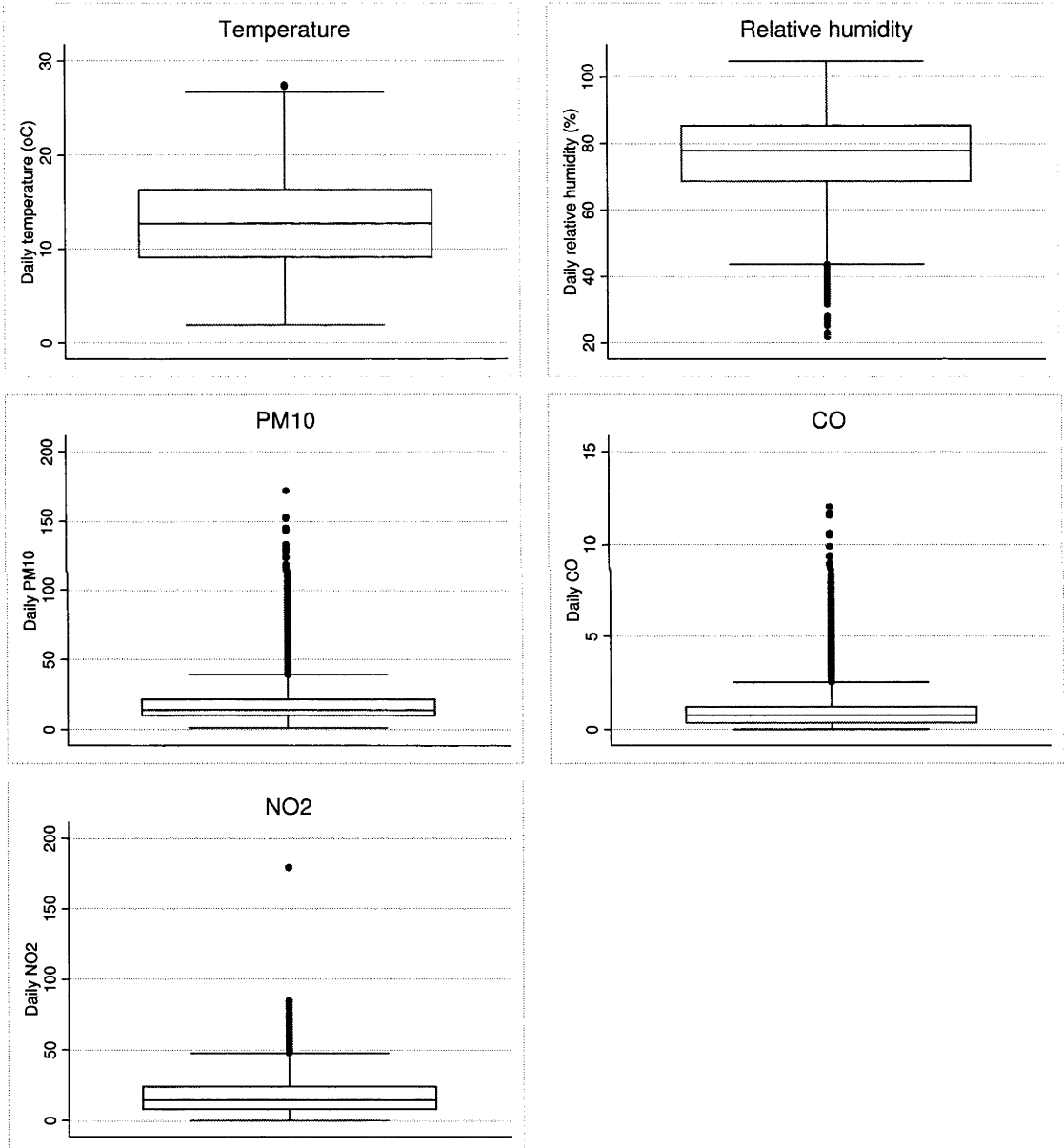


Table 5-1. Summary statistics for daily weather and air pollutants, Christchurch June 1988 – December 1999

Variables	Mean (sd)	Min	Quartile			Max
			1 st	Median	3 rd	
Weather variables						
<i>Temperature</i>						
24-hour average (°C)	12.8 (4.8)	1.9	9.1	12.7	16.4	27.4
Maximum hourly (°C)	18.8 (5.7)	3.9	14.6	18.7	22.9	36.7
Minimum hourly (°C)	8.1 (4.9)	-4.0	4.5	8.3	11.7	22.1
<i>Relative humidity</i>						
24-hour average (%)	76.1 (13.3)	21.8	68.7	77.8	85.4	104.8
Pollutants						
<i>Particulate matter (PM₁₀)</i>						
24-hour average (µg/m ³)	19.8 (18.1)	1.1	9.8	13.9	21.5	171.9
Maximum hourly (µg/m ³)	59.7 (74.8)	4.2	21.5	31.3	60.8	806.1
<i>Carbon monoxide (CO)</i>						
24-hour average (mg/m ³)	1.1 (1.3)	0	0.33	0.75	1.2	12.04
Maximum hourly (mg/m ³)	3.6 (4.9)	0	1	1.7	4.0	40.6
<i>Nitrogen dioxide (NO₂)</i>						
24-hour average (µg/m ³)	17.8 (13.4)	0	8.2	14.4	23.9	179.5
Maximum hourly (µg/m ³)	41.1 (28.9)	0	21.8	34.4	53.0	412

5.7 Correlations between exposure variables

The Pearson's correlation coefficients between daily pollutants and daily weather variables by season are shown in Table 5-2. Seasons were defined similar to those

defined for the seasonal analysis of the association between daily air pollution and mortality in Chapter 6. Four months from May to August were classified as the cooler season and the remaining eight months from September to the following April were classified as the warmer season. The correlation coefficients of temperature with CO and NO₂ were similar in both seasons but the correlation with PM₁₀ was stronger in the cooler season. Relative humidity was weakly correlated with all three pollutants in both seasons.

All three pollutants were negatively correlated with daily mean temperature and daily minimum temperature. The strongest correlation was with daily minimum temperature (Table 5-3). The negative correlation of pollutants with temperature is consistent with the environmental process. People start burning firewood in the winter months when the temperature is very low, which results in higher concentrations of air pollutants. Temperature inversion, the other meteorological phenomenon, which restricts the dispersion of air pollutants by limiting the vertical movement of air and thus increases the local air pollution levels, is created when the ground level temperature is low.

Pollutants were strongly correlated with each other, with the strongest correlation observed between PM₁₀ and CO (correlation coefficient = 0.77 for the cooler season). The correlations between pollutants were stronger in the cooler season than in the warmer season. The strong correlations between air pollutants were most likely due to the dominating influence of weather on daily pollutant variations. Temperature inversions and calm weather, which occur more frequently in the cooler season in Christchurch, restrict the dispersion of air pollutants both horizontally and vertically and cause the higher levels of all air pollutants in the cooler season.

Table 5-2. Correlations between daily pollutants and weather variables by season, Christchurch June 1988 – December 1999

	PM ₁₀	CO	NO ₂	Mean Temperature
Cooler season (May – Aug)				
CO	0.77			
NO ₂	0.32	0.41		
Mean temperature	-0.39	-0.39	-0.29	
Relative humidity	-0.08	-0.09	-0.08	-0.28
Warmer season (Jan – Apr; Sept – Dec)				
CO	0.35			
NO ₂	0.10	0.32		
Mean temperature	-0.07	-0.36	-0.30	
Relative humidity	-0.05	0.03	0.04	-0.35

Table 5-3. Correlations between daily pollutants and temperature variables by season, Christchurch June 1988 – December 1999

	Daily Temperature		
	Mean	Maximum	Minimum
Cooler season (May – Aug)			
PM ₁₀	-0.39	0.13	-0.55
CO	-0.39	0.11	-0.55
NO ₂	-0.29	-0.006	-0.35
Warmer season (Jan – Apr; Sept – Dec)			
PM ₁₀	-0.07	0.02	-0.15
CO	-0.36	-0.23	-0.43
NO ₂	-0.30	-0.15	-0.38

5.8 Summary

This chapter provided a general description of daily weather and air pollution data in Christchurch. Long-term trends and seasonal variations in weather and air pollution data were explored. All weather variables and air pollutants had strong seasonal variations. Relative humidity had a seasonal variation opposite to the seasonal variation in

temperature variables. Monthly average relative humidity was higher in May – July and lower in October – December. All three pollutant concentrations had similar seasonal patterns, with higher concentrations in the cooler months and lower concentrations in the warmer months. There was a strong correlation between PM₁₀ and CO, especially in the cooler season. The correlations between air pollutants were stronger in the cooler season than in the warmer season. This is most likely because of the dominating influence of winter weather conditions, such as calm weather and temperature inversions, on the dispersion of air pollutant concentrations.

Chapter 6: Short-term effects of air pollution on mortality

6.1 Introduction

The main aims of this chapter are to examine the association between daily mortality and air pollution adjusting for confounding variables, and to assess the sensitivity of the association to different methods by which the confounders are adjusted for. The main hypothesis explored in this chapter is that an increase in daily air pollutant concentration is associated with an increase in daily mortality.

Time series regression models are used to model daily counts of deaths with “day” as the unit of analysis. The probability of dying on any given day is very small and the distribution of daily mortality can take only non-negative integers. This suggests that daily counts of deaths are counts of rare events and thus follow a Poisson distribution. Poisson regression analysis was used to quantify the association between daily mortality and air pollutant concentrations adjusting for other co-variates such as a long-term trend and seasonal variation in mortality data, days of the week and daily weather data. Various approaches were used to adjust for confounders and the sensitivity of the association to different approaches were analysed.

6.2 Confounders

Confounding in the association between mortality and air pollution occurs when an extraneous variable is associated with an increase in mortality and it is also correlated with air pollutant concentrations. The key analysis in a time series study design is to associate daily variation in air pollutant concentrations with daily variation in mortality. The most likely confounders in time series study design are the variables that have systematic variation over time, such as a long-term trend, seasonal variation, weather variables, and epidemics of seasonal infectious diseases such as flu epidemics. As individual characteristics like age, sex, smoking habit, and socioeconomic class do not vary from day-to-day, these variables do not confound the association between air pollution and mortality.

Daily mortality may have a long-term trend due to change in population structure over time. If daily air pollutant concentration also has a long-term trend, it will induce correlations between air pollution and mortality even if they are not causally related. Analysis to look for any causal association must adjust for these long-term trends.

Chapters 4 and 5 show that mortality, meteorological and air pollution variables had pronounced seasonal variations. Both daily number of deaths and air pollutant concentrations peaked during the winter. Because of similar seasonal variations, mortality and air pollution would be positively correlated and the correlations could be very high. Although high PM₁₀ concentrations in the winter may contribute to higher deaths in the winter, most of the seasonal variation in mortality may be due to other factors. So in order to analyse the acute effects of air pollution on mortality, these seasonal patterns need to be adequately adjusted for in the analysis. Failure to control for seasonal patterns in the analysis could provide false evidence of the association between mortality and air pollution.

Weather variables and co-pollutants that vary on short time scales are the other likely confounders in the short-term association between daily mortality and air pollution. Weather variables, especially temperature, are highly correlated with air pollutants and at the same time they are also risk factors for increased mortality. Extreme temperatures, both hot and cold, are associated with increased daily mortality. So if the confounding effects of weather variables are not adjusted for, the increased mortality may be wrongly linked only to air pollutants even though they are not the only risk factors. This may result in an overestimation of the effect of air pollutants on mortality. Co-pollutants may also confound the true association between the pollutant of interest and mortality. For example, PM₁₀ and CO concentrations are highly correlated. Thus, the likely effect of CO on mortality may contribute to the association between PM₁₀ and mortality resulting in an exaggerated effect of PM₁₀ if the confounding effect of CO is not adequately adjusted for.

A long-term trend and seasonal variation in daily mortality and the confounding effects of weather variables have been taken into account in this analysis. Before adding air pollutant variables, the models were checked to confirm that a long-term trend and seasonal variation were adequately controlled for. The confounding effects of co-pollutants were analysed by fitting multi-pollutant models.

6.3 Methods

Poisson regression models were fitted for daily mortality as shown in equation (6-1).

$$E(Y_t) = Pop \times \exp\left(\beta_0 + \beta_1 PM_{t-q} + \sum_j \beta_j f(X_{j,t})\right) \quad ; j > 1, \quad 0 \leq q \leq 3 \quad (6-1)$$

Where Y_t is the number of deaths on day “ t ”, Pop is the population at risk which is used as an “offset” in the models, PM_{t-q} is PM₁₀ concentrations on day “ $t-q$ ”, “ q ” is the lag

number and $X_{j,t}$ are other daily confounding variables. By varying the values of q from 0 to 3, the lagged effects of air pollutant can be estimated for lags 0 (same day) to 3 days. The effect of each lag was estimated one at a time by fitting separate models with only one lagged air pollutant variable each time. The annual population was used as the population at risk for each day in a year. For census years, the “usually resident” census population for Christchurch was used as an annual population and for non-census years it was estimated by linear interpolation from the “usually resident population” of two census years.

Various functions of time were used to adjust for a long-term trend and seasonal variation in daily mortality. Models were also adjusted for weather variables such as daily temperature and relative humidity.

The associations between mortality and air pollutants were examined for non-external cause mortality, respiratory mortality and circulatory mortality by separately fitting the Poisson regression models (equation (6-1)) for each of the mortality categories. Models were fitted for daily mortality of different age groups to analyse age-specific associations between air pollutants and mortality. Analysis was performed separately for all ages and for the 65+ years age group. The analyses of respiratory mortality and circulatory mortality were restricted to the population aged 65+ years only.

Two different approaches were used to adjust for seasonality and weather variables. They were:

1. Using trigonometric filtering to adjust for seasonality in daily mortality and a linear or a piecewise linear term for weather variables to control for the confounding effects of weather variables based on the analysis of their associations with daily mortality.
2. Using regression spline functions of confounders including time, temperature and relative humidity to adjust for them.

These methods are discussed in detail in the following sections.

6.3.1 Controlling for season and trend

Method 1

In the first method, the seasonal variation in mortality was controlled by using harmonic waves (sinusoidal terms). Although the annual cycle of daily mortality is strongly periodic with higher deaths in the winter and lower deaths in the summer, it is highly unlikely that mortality patterns over time can be explained by just one sinusoidal

function with a period of one year. Some risk factors that vary seasonally may cause the seasonal variation of a six month period in daily mortality while others may cause the seasonal variations of even shorter period for example four months, three months etc. Thus, in addition to the annual cycle, the pattern of daily deaths is more likely to have a number of other cycles of less than a one year period. This would require the sum of harmonic waves with increasing frequencies to adjust for the complex seasonal pattern of daily deaths.

Seasonal patterns in mortality were modelled using sinusoidal functions

$$\sum_k (\alpha_k \sin kT + \beta_k \cos kT) \quad (6-2)$$

Where $T = 2\pi t/365.25$, t is the day of the year, α_k and β_k are the regression coefficients to be estimated from the model fitting. The values of k determine the period of seasonal cycle. For annual cycle, the value of k is set to 1. The integer values of $k = 2,3,4,5$ approximately correspond to the cycles of 6 months, 4 months, 3 months and 73 days respectively. Modelling seasonal pattern using sinusoidal functions like this is commonly known as a cosinor analysis.

Sinusoidal functions were included in the models to control for significant cycles of up to 2 months ($k = 6$). The short-term variation of less than 2 months in mortality was used to estimate the association between mortality, air pollution and weather. The maximum number of sinusoidal functions to control for seasonal patterns was selected based on the likelihood ratio test for the model. Sinusoidal functions of one-year period, 6 months cycle and the other smaller cycles were entered into the model in a forward stepwise fashion until the likelihood ratio test between the higher order model and the nested model within it showed that further additions of sinusoidal functions improved the model fit significantly. Statistical significance was set at the 10% level (p -value < 0.1). Both sine and cosine terms of the same period were included simultaneously in the models to create the sinusoidal functions.

Long-term trends in mortality over the 13 year period were modelled using a linear time trend. Influenza or other epidemics may cause higher mortality in the epidemic years than in non-epidemic years. This results in non-monotonic year to year fluctuations in mortality. Analysis was carried out to investigate whether there were year to year fluctuations in mortality by including dummy variables for each year in the models. A statistically significant improvement (p -value < 0.1) in the model fit would indicate year to year fluctuations in mortality. If there was evidence of year to year fluctuations in mortality, dummy variables were included in the models. This would

control for any between-year variations in daily number of deaths that may be caused by influenza epidemics or epidemics of other infectious diseases. Flu or other epidemics may also cause the variations in daily number of deaths within a year. Adding dummy variables for each year would not control for this within-year variations. However, since most epidemics including flu are seasonal, controlling for seasonal variations in daily number of deaths would control for within-year variations to some extent. Because of the lack of data on flu or other epidemics, the variations in daily number of deaths caused by the epidemics could not be completely controlled for.

In order to control for daily variation in mortality across the week, dummy variables for day of the week were included in the models irrespective of whether they were statistically significant or not.

Method 2

While seasonal variation in mortality was modelled with a parametric approach using sinusoidal functions in Method 1, it was modelled semi parametrically in Method 2 using regression spline functions of calendar time. Natural cubic splines of calendar time were used to fit the seasonal variation and a long-term trend in daily mortality. Natural cubic spline function of calendar time divides the whole time period of analysis into a number of intervals as defined by the degrees of freedom used in the natural cubic spline function and a cubic polynomial is fitted to each interval. Cubic polynomials are fitted such that they join smoothly at the boundaries of the intervals. Beyond the boundary points, a linear function is fitted.

Seven degrees of freedom (df) per year of data was chosen (a total of 81 df) to create natural cubic splines of calendar time. Using 7 df per year of data would adjust for the long-term variation in daily mortality of approximately more than two months leaving only the short-term variation in mortality of less than two months to estimate the association between mortality, air pollution and weather. This would adjust for the confounding effect from a long-term trend in mortality, year to year variations in mortality arising from influenza or other epidemics and seasonal variations in mortality. Dummy variables for days of the week were included in the models irrespective of whether they were statistically significant or not in order to control for daily variation in mortality across the week.

Diagnostic plots

Several diagnostic plots were examined to assess whether the models reasonably adjusted for seasonal variations and long-term trends in daily mortality data. Time series

plots of residuals were examined for any long wavelength pattern remained in the data. Time series plots of model prediction against the real data are helpful in assessing if the models are reasonably fit to the seasonal variation and a long-term trend in data. The models adjusted for the variation in daily mortality of approximately more than two months. The spectral analysis on the residuals was carried out to see if residual periodogram had larger values at the periods above two months. Large values at these periods would indicate an insufficient adjustment of seasonal variations. The partial autocorrelation functions (PACF) of the residuals were plotted to check for any large values at the first lags, which would indicate an insufficient adjustment of seasonal variations.

6.3.2 Controlling for weather variables

Method 1

Relationships between weather variables and mortality were first explored graphically using the residual-residual plots of daily mortality data against daily weather data. The residuals from the basic model (discussed in Section 6.3.1) for daily mortality that adjusted for a long-term trend and seasonal variations were plotted against the residuals from the model for daily weather variables that adjusted for a long-term trend and seasonal variations in daily weather data. The same sets of variables were used to control for a long-term trend and seasonal variations in both mortality and weather variables. A loess smoothing technique was used to smooth the scatter plots and to reveal any pattern present in them (Cleveland, 1979). The decisions on which weather terms (i.e. linear, piecewise linear, second order polynomial) to include in the models to control for their confounding effects were made based on the shape of the plots.

Because of the increased risk of mortality associated with hot and cold weather, daily mortality is likely to increase with change in temperature at both high and low temperature. Variables, based on daily maximum temperature and daily minimum temperature, were included in the models to control for the confounding effects of heat and cold respectively. Similarly, variables, based on daily average relative humidity, were included in the model to control for the confounding effect of relative humidity on mortality.

If the residual-residual plots showed that there was an increase in mortality with increase in daily maximum temperature above a certain temperature (heat effect) and increase in daily mortality with decrease in daily minimum temperature below a certain

temperature (cold effect), two new temperature variables would be created as follows to include in the models:

$$\begin{aligned}
 HOT &= \begin{cases} tempgx - A & ; \text{ if } tempgx \geq A \\ 0 & ; \text{ else} \end{cases} \\
 COLD &= \begin{cases} B - tempgm & ; \text{ if } tempgm \leq B \\ 0 & ; \text{ else} \end{cases}
 \end{aligned}$$

Where *tempgx* and *tempgm* are daily maximum hourly temperature and daily minimum hourly temperature; *A* and *B* (*A* > *B*) are the temperatures where the associations of mortality with maximum hourly temperature and minimum hourly temperature changed.

Temperature *A* is the daily maximum hourly temperature above which there is an increased risk of daily mortality and temperature *B* is the daily minimum hourly temperature below which there is an increased risk of mortality. Days with maximum hourly temperature below the temperature *A* and minimum hourly temperature above the temperature *B* can be described as the comfort days when the risk of increased mortality associated with daily temperature is almost zero. The optimal change-points *A* and *B* were estimated by fitting models with a range of possible temperatures as change-points and choosing the ones that best fitted the data. Model fit was evaluated based on the AIC.

Similarly, new relative humidity variables were created from daily average relative humidity based on the shape of the associations shown in the residual-residual plot for relative humidity. These new variables were included in the models to control for the confounding effects of relative humidity.

The new weather variables were created, as discussed above, based on the shape of the associations of weather variables with non-external mortality in the 65+ years age group and the same weather variables were included in the models for other mortality categories.

The lagged effects of weather variables were adjusted for by including the moving averages of the last three days' (lag 1 to lag 3) maximum temperature, minimum temperature, and relative humidity. It was assumed that the relationships of these lagged weather variables with daily mortality would be of the same shape as the relationships of the corresponding same day weather variables with daily mortality. Both temperature and relative humidity variables were kept in the model irrespective of

their statistical significances in order to control for the confounding effects of weather variables.

Method 2

Like modelling long-term trend and seasonal variations in daily mortality, the effects of weather variables on daily mortality were modelled using natural cubic splines of temperature and relative humidity. A weather model similar to the one used in the 90 US cities analysis was used in this analysis (Samet *et al.*, 2000b; Samet *et al.*, 2000c). The model included

- a natural cubic spline of the same day's average hourly temperature with 6 df
- a natural cubic spline of the moving average of the preceding three days' (lag 1 to lag 3) average hourly temperature with 6 df
- a natural cubic spline of the same day's average hourly relative humidity with 3 df
- a natural cubic spline of moving average of the preceding three days' (lag 1 to lag 3) average hourly relative humidity with 3 df.

In addition, several sensitivity analyses were performed to examine the sensitivity of the effect of PM₁₀ on acute mortality to adjustment for a long-term trend and seasonal variation in mortality and to control for the confounding effects of weather variables. The choice of degrees of freedom for a natural cubic spline smooth function of time to control for a long-term trend and seasonal variation in mortality can change the estimates of effect coefficients considerably and as such it is an important issue in time series analysis of air pollution and mortality (Health Effects Institute, 2003). The degrees of freedom for the smooth function of time were varied to investigate the sensitivity of effect estimates of daily PM₁₀ on acute mortality to the adjustment for a long-term trend and seasonal variation in daily mortality. Similarly, the sensitivity of the estimates of PM₁₀ effect to the degrees of freedom for natural cubic spline smooth functions of weather variables was examined. Sensitivity analysis is discussed in detail in Section 6.4.3.

6.3.3 Adding air pollution variables

Daily measurement of air pollutant concentrations (24-hour average of hourly concentrations) was added as a linear term to the models that controlled for a long-term trend, seasonal variation and weather variables to estimate the relative risk of daily mortality associated with an increase in daily air pollutant concentrations. The lagged effects of air pollutant were estimated for lags 0 (same day) to 3 days. The effect of each

lag was separately estimated by fitting separate models with different lags of air pollutants one at a time in this chapter. Each model had only one air pollution exposure variable. Estimation of the combined effect of all lags on daily mortality using distributed lag model is discussed in detail in Chapter 8.

Multi-pollutant models were fitted to investigate whether the association between daily mortality and PM_{10} changed after controlling for CO or NO_2 . All air pollutant variables were added as linear terms to the model.

6.3.4 Effect modification by season

The possible differences in the association between mortality and PM_{10} in different seasons were examined by adding a dummy variable for season and an interaction term between the season variable and PM_{10} to the model. For this purpose, the whole year was divided into two seasons; cool season (May-August) and warm season (September-April). The cool season corresponds to the winter months with very high PM_{10} concentrations, when particulate matter from wood smoke dominates all other sources. The warm season corresponds to the non-winter months with relatively low PM_{10} concentrations with almost no particulate matter from wood smoke. Vehicle emissions, industrial emissions and other insignificant sources such as farm dusts, sea spray and pollens are the sources of particulate matter in the warm season.

6.3.5 Overdispersion and autocorrelation

The mean and variance of a Poisson distribution are equal and as such, the dispersion parameter for a Poisson distribution is one. Many count processes such as daily counts of deaths generally have a variance greater than the mean of the distribution. Such distribution has a dispersion parameter greater than one. This is called overdispersion. The dispersion parameter for the final models for daily mortality was checked to examine whether or not the distribution was overdispersed. If there was evidence of overdispersion, Negative Binomial regression models would be used instead of Poisson regression models to allow for overdispersion.

It is very common to get serially correlated residuals from the models fitted for time series data. Autocorrelation, if remaining in the residuals of the final models, would be adjusted for by re-fitting autoregressive models.

6.4 Results

6.4.1 Method 1: Using sinusoidal functions

Controlling for season and trend

Table 6-1 shows a systematic development of the basic model to describe a long-term trend and seasonal variations in daily non-external deaths in the 65+ years age group. A long-term temporal trend in mortality over the 13 year period was modelled using a linear time trend. Seasonal variations were modelled by first fitting the simplest model with sine and cosine terms for a one-year period to model the annual cycle and then gradually adding sinusoidal terms for other shorter period cycles. In each step, the likelihood ratio test was performed to test if adding new terms significantly improved the model fit. The p-values for the likelihood ratio tests are shown in the table. The p-values less than 0.1 were considered statistically significant. All models shown in Table 6-1 had a linear time trend as a co-variate to capture a long-term trend in daily mortality.

Table 6-1. Sequential development of a basic model for daily non-external deaths in the 65+ years age group

Model	Model Description	df [#]	p-value [^]
M ₁	Annual cycle ($k^* = 1$)	4	
M ₂	Annual and six months cycle ($k^* = 1, 2$)	6	< 0.001
M ₃	Annual, six months and four months cycle ($k^* = 1, 2, 3$)	8	0.007
M ₄	With $k^* = 1, 2, 3, 4$	10	0.007
M ₅	With $k^* = 1, 2, 3, 4, 5$	12	0.005
M ₆	With $k^* = 1, 2, 3, 4, 5, 6$	14	0.063

All models had a linear time trend as a co-variate.

[#] df: degrees of freedom of model (the number of parameters in the model)

[^] p-value: p-value for the likelihood ratio test to compare the model with the one immediately above it.

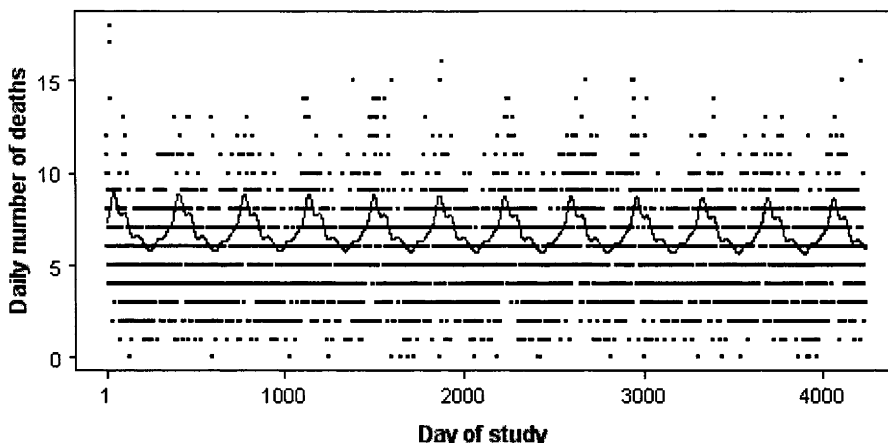
* Values of k determine the period of seasonal cycle. $k = 1, 2, 3, 4, 5, 6$ approximately correspond to annual, 6-month, 4-month, 3-month, 73-day and 2-month cycle respectively.

For non-external deaths in the 65+ years age group, the sinusoidal terms of up to 6th order corresponding to the seasonal cycles of 1 year, 6 months, 4 months, 3 months, 73 days and 2 months were statistically significant. Dummy variables for each year were included in the model to test if there was any remaining year to year variation in daily non-external deaths in the 65+ years age group. There was no evidence of remaining year to year variations after controlling for a long-term trend and seasonal

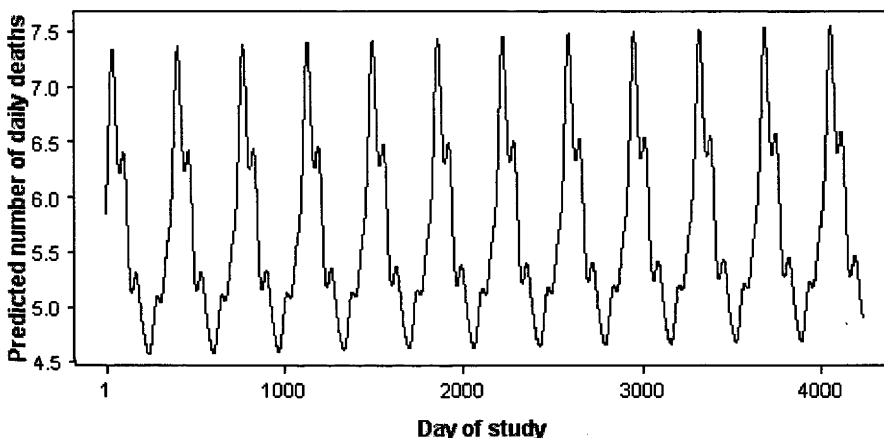
variations, and thus dummy variables were dropped from the model. The basic model for daily non-external deaths in the 65+ years age group had sinusoidal terms for the periods of 1 year, 6 months, 4 months, 3 months, 73 days and 2 months, and a linear term for time trend. A time series plot of daily number of non-external deaths in the 65+ years age group with the predicted daily number of deaths from the basic model are shown in Figure 6-1. The plot confirms that the basic model approximately captures the seasonal variation in daily mortality. In order to control for any daily variation in mortality across the week, dummy variables for days of the week were added into the basic model.

The basic models that adjust for a long-term trend and seasonal variations in daily mortality were developed for circulatory deaths in the 65+ years age group, respiratory deaths in the 65+ years age group, and total non-external mortality of all ages. The sequential development of these models (similar to Table 6-1) is shown in Appendix A. Plots of the predicted number of daily deaths based on the final basic models for daily circulatory and respiratory deaths in the 65+ years age group and daily non-external deaths in the whole population are shown in Appendix B. There was a long-term time trend present in all analysed daily mortality data. Among seasonal cycles of different periods, the annual cycle was the most dominant seasonal cycle for all analysed daily mortality categories.

Figure 6-1. Time series plot of daily number of non-external deaths in the population aged 65+ years with the predicted number of daily non-external deaths based on the cosinor model for daily non-external deaths in the 65+ years age group



Predicted number of daily non-external deaths of the population aged 65+ years based on the cosinor model for daily non-external deaths in the 65+ years age group



Note: Y-axis scale is different

The final basic model for daily circulatory deaths in the 65+ years age group had sinusoidal terms for the periods of 1 year, 6 months, 4 months and 73 days, and a linear term for time trend. There was no significant remaining non-monotonic year to year variation in daily circulatory deaths in this age group.

The final basic model for respiratory deaths in the 65+ years age group included seasonal cycles of 1 year, 6 months, 4 months, 3 months and 2 months, and a linear term for time trend. The likelihood ratio test for the comparison between two models; one with dummy variables for each year and another without dummy variables showed that addition of dummy variables into the model improved the model fit significantly, suggesting a remaining year to year variation in daily respiratory deaths in the 65+ years age group. Dummy variables for each year were included in the final model to adjust for the remaining year to year variation in daily mortality.

The final basic model for non-external mortality of all ages included sinusoidal terms for the periods of 1 year, 6 months, 4 months, 3 months and 73 days, and a linear term for time trend. The test showed that there was a remaining year to year variation in daily non-external mortality of all ages. Thus, dummy variables for each year were included in the model to adjust for the remaining year to year variation in daily non-external mortality of all ages. In order to control for any daily variation in mortality across the week, dummy variables for days of the week were added into the basic models for all mortality categories.

Residual diagnosis

In order to check whether the basic models adequately adjusted for a long-term temporal trend and seasonal variation in daily deaths, various diagnostic analyses of residuals were performed. Figures 6-2, 6-3(B) and 6-4(B) show diagnostic plots for residuals of the basic model for non-external deaths in the 65+ years age group. A plot of residuals of the basic model for non-external deaths in the 65+ years age group against day of study (Figure 6-2) shows that the seasonal pattern in daily deaths have been reasonably removed.

Figure 6-2. A temporal plot of the residuals after adjusting for a long-term trend and seasonal variation in daily non-external deaths in 65+years age group

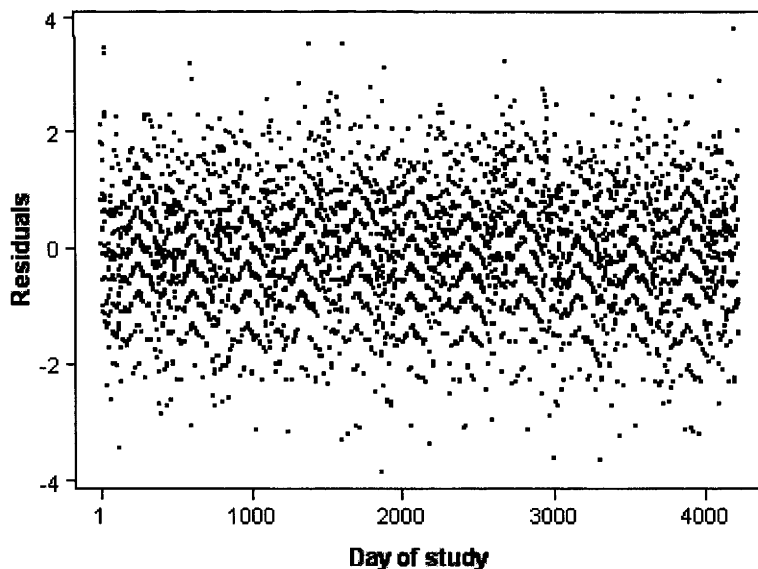


Figure 6-3 compares the partial autocorrelation functions (Partial ACF) for daily non-external deaths in the 65+ years age group and for the residuals from the basic model for daily non-external deaths in the same age group. Unlike in daily non-external deaths, there was no large partial autocorrelations in the residual series. The absence of

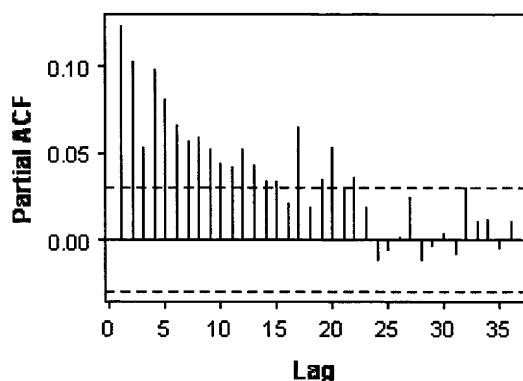
large positive values at the first lags suggests that the basic model adequately adjusted for seasonal variations.

Figure 6-4 compares the spectral density analysis of daily non-external deaths in the 65+ years age group and the residuals from the basic model for daily non-external deaths in the same age group. The periodogram shows the estimates of spectral density (spectrum) at various frequencies, which are the number of periods per day. Large spectrum indicates a cyclical pattern of the frequency at which the large spectrum was estimated. The periodogram for daily non-external deaths had some large values at different frequencies (Figure 6-4 (A)) suggesting that the data series had a number of cyclical patterns of different period lengths. The periodogram for residual series did not have any large value left. This shows that the seasonal variation and a long-term trend in daily non-external deaths in the 65+ years age group had been adequately controlled for by the basic model.

The diagnoses of the residuals were performed for other mortality categories (i.e. daily non-external deaths of all ages, daily circulatory deaths in the 65+ years age group and daily respiratory deaths in the 65+ years age group). The diagnosis confirmed that the basic models for respective mortality categories reasonably controlled for seasonal variation and a long-trend in daily mortality.

Figure 6-3. Partial autocorrelation plots

(A) For daily non-external deaths in 65+ years age group



(B) For the residuals after adjusting for a long-term trend and seasonal variation in daily non-external deaths in 65+ year age group

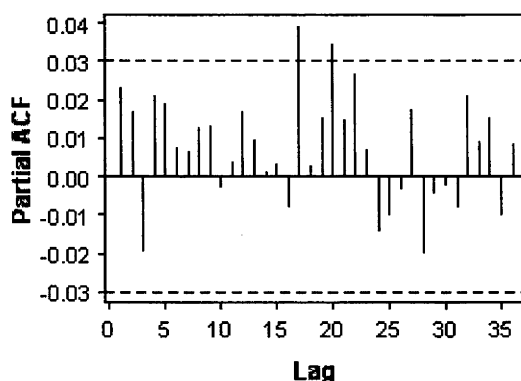
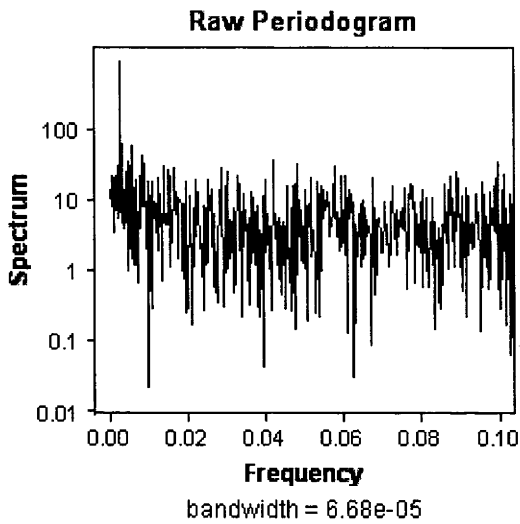
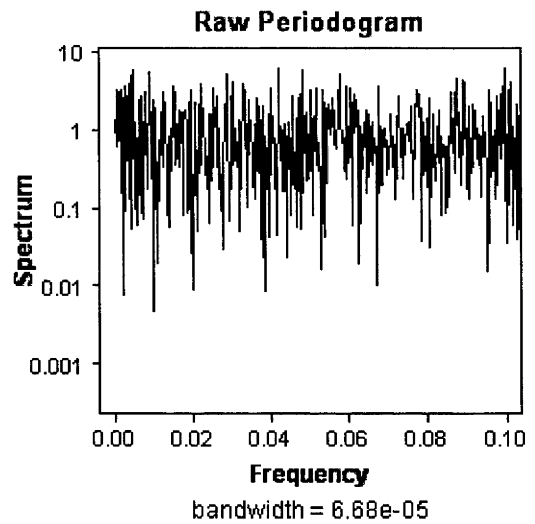


Figure 6-4. Spectral density analysis

(A) Periodogram plot for daily non-external deaths in 65+ years age group



(B) Periodogram plot for residual series after adjusting for a long-term trend and seasonal variation in daily non-external deaths in 65+ year age group



Controlling for weather variables

The short-term relationships between daily mortality and weather variables after controlling for a long-term trend and seasonal variation were explored graphically using the residual-residual plots. Models with the same set of variables as in the basic model were fitted for weather variables and their residuals were plotted against the residuals of the basic model for non-external mortality in the 65+ years age group. Figures 6-5, 6-6 and 6-7 show the residual-residual plots of daily mortality against the same day's maximum temperature, minimum temperature and relative humidity respectively. The non-parametric smooth (a lowess smooth) shows the pattern present in the scatter plots. In order to reveal the pattern present in more detail, only a part of the residual-residual plot from -1 to 1 of the y-axis is shown in the right hand side plot of all three figures.

The shape of the association in the residual-residual plots of daily non-external deaths in the 65+ years age group against the same day's maximum temperature (Figure 6-5) suggested that there was an increase in daily number of deaths for an increase in the same day's maximum temperature above a certain threshold maximum temperature but no association below the threshold maximum temperature. This suggests an increased risk of mortality associated with high temperature (effect of heat) above a certain threshold temperature after controlling for seasonal variation and a long-term trend in daily mortality.

In order to identify the threshold temperature for heat effect, a new variable “*hot*” was created as explained in Section 6.3.2. The value of “*hot*” was set to 0 if the daily maximum temperature was below the threshold maximum temperature and set to *maximum temperature minus threshold temperature* if the daily maximum temperature was above or equal to the threshold temperature. Models were fitted using a sequence of threshold maximum temperature ranging over the minimum and maximum values of daily maximum temperature with an increment of 0.1°C. The threshold maximum temperature for the heat effect on mortality, i.e. the temperature above which the risk of increased daily mortality was associated with an increase in maximum temperature, was chosen based on the best model fit as decided by minimising the AIC. The best model fit was with a threshold temperature of 26.2°C for daily maximum temperature. The new variable “*hot*” with the threshold temperature of 26.2°C for daily maximum temperature was included in the final models to adjust for an increase in daily mortality associated with hot temperatures (the effect of heat on daily mortality).

The shape of the association in the residual-residual plots of daily non-external deaths in the 65+ years age group against the same day’s minimum temperature (Figure 6-6) suggested that there was an increase in daily number of deaths for a decrease in the same day’s minimum temperature. This suggests an increased risk of mortality associated with low temperatures (effect of cold). The plots neither showed a piecewise linear relationship between daily mortality and minimum temperature nor supported any existence of the threshold temperature for the effect of cold temperature on mortality. Thus daily minimum temperature was included in the final models as a linear term to adjust for an increase in daily mortality associated with cold temperatures (the effect of cold on daily mortality).

The shape of the association in the residual-residual plots of daily non-external deaths in the 65+ years age group against the same day’s relative humidity (Figure 6-7) suggested that there were increases in daily number of deaths for an increase in the same day’s relative humidity above a certain threshold relative humidity and also for a decrease in the same day’s relative humidity below a certain threshold relative humidity but no association between the two threshold levels.

In order to identify the two threshold levels for relative humidity, two new variables “*high.humid*” and “*low.humid*” were created as follows:

$$\begin{aligned}
 \text{high.humid} &= \begin{cases} rh - rh_A & ; \text{ if } rh \geq rh_A \\ 0 & ; \text{ else} \end{cases} \\
 \text{low.humid} &= \begin{cases} rh_B - rh & ; \text{ if } rh \leq rh_B \\ 0 & ; \text{ else} \end{cases}
 \end{aligned}$$

Where rh is daily relative humidity; rh_A and rh_B ($rh_A > rh_B$) are the two threshold levels for relative humidity where the association of mortality with relative humidity changed.

Models were fitted using a sequence of threshold relative humidity ranging over the minimum and maximum values of daily relative humidity with an increment of one. The two levels of relative humidity rh_A and rh_B were chosen based on the best model fit as decided by minimising the AIC. The best model fit occurred when the values of rh_A and rh_B were 61% and 52% respectively. This shows that there was an increased risk of mortality associated with a decrease in relative humidity below 52% and also associated with an increase in relative humidity above 61%. The effect of new relative humidity variable *low.humid* (i.e. the negative association between daily mortality and the same day’s relative humidity below 52%) was not statistically significant after controlling for a long-term trend, seasonal variation in daily mortality and the effect of temperature on daily mortality. Also, there was not much improvement in the model fit when the new relative humidity variable *low.humid* was used in the model.

Although exploratory analysis shows a negative association between daily number of deaths and the same day’s relative humidity below 52%, the association disappeared when a long-term trend, seasonal variation in daily mortality and the effect of temperature on daily mortality were controlled for. The positive association between daily number of deaths and the same day’s relative humidity above 61% remained statistically significant. This suggested that there was no association between daily number of deaths and relative humidity below 61%. Thus, only one relative humidity variable *high.humid* with the threshold level of 61% was chosen to use in the final models.

The model with the relative humidity variable with a threshold (*high.humid*) was compared with the model with a linear term for relative humidity and quadratic terms for relative humidity. The threshold model fitted better to the data than others and thus the relative humidity variable with a threshold (*high.humid*) was used in the final

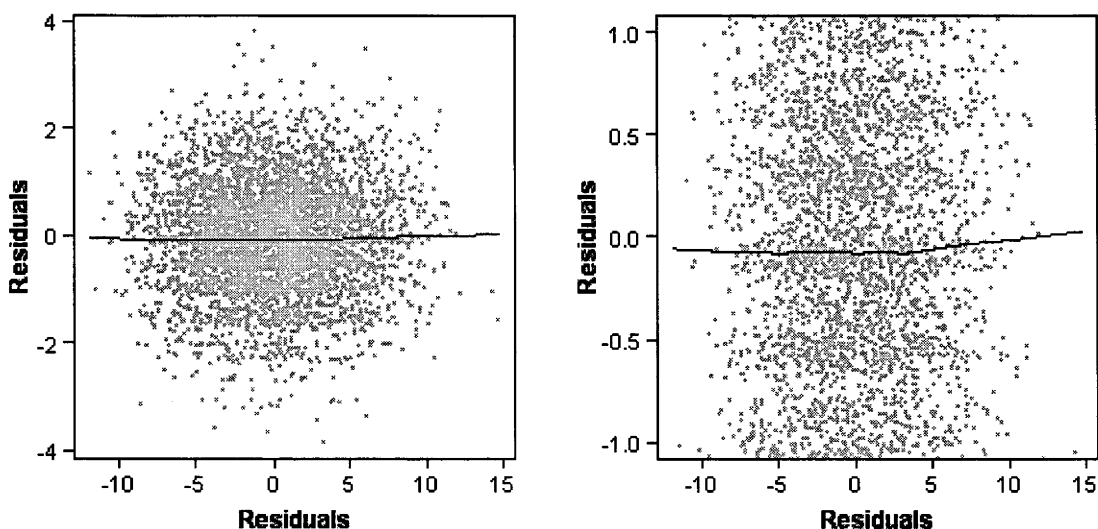
models to adjust for the confounding effect of the same day relative humidity on daily mortality.

The lagged effects of weather variables were adjusted for by including the moving averages of the last three days' (lag 1 to lag 3) maximum temperature, minimum temperature and relative humidity. A new lagged “*hot*” variable similar to the same day “*hot*” variable with the threshold temperature of 26.2°C was created from the moving average of the preceding three days' maximum temperature. Similarly, a new lagged “*high.humid*” variable similar to the same day “*high.humid*” variable with the threshold relative humidity level of 61% was created from the moving average of the preceding three days' relative humidity. In order to control for the lagged effects of cold weather, the moving average of the preceding three days' minimum temperature was included in the model. These variables were kept in all models irrespective of their statistical significances.

Interactions between temperature and relative humidity variables were added into the model to examine if including them in the model changed the associations between daily air pollutant concentrations and daily mortality. The interaction between temperature and relative humidity was statistically significant. However, no noticeable difference was found between the effect size estimates of air pollutants on daily mortality from the models with and without interaction terms. Thus, the interactions between temperature and relative humidity variables were not included in the final models.

Figure 6-5. Residual - residual plot: Plots of residuals of the basic model for non-external deaths in 65+ years age group against residuals of the same model for daily maximum temperature

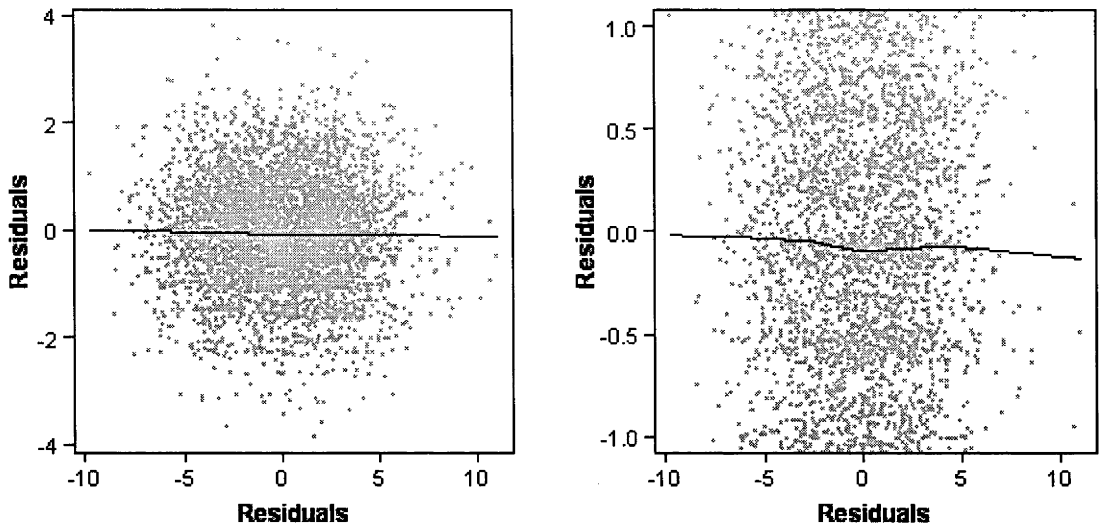
(Right hand side plot shows part of the left hand side plot from -1 to 1 of y-axis)



Note: Y-axis scale is different

Figure 6-6. Residual - residual plot: Plots of residuals of the basic model for non-external deaths in 65+ years age group against residuals of the same model for daily minimum temperature

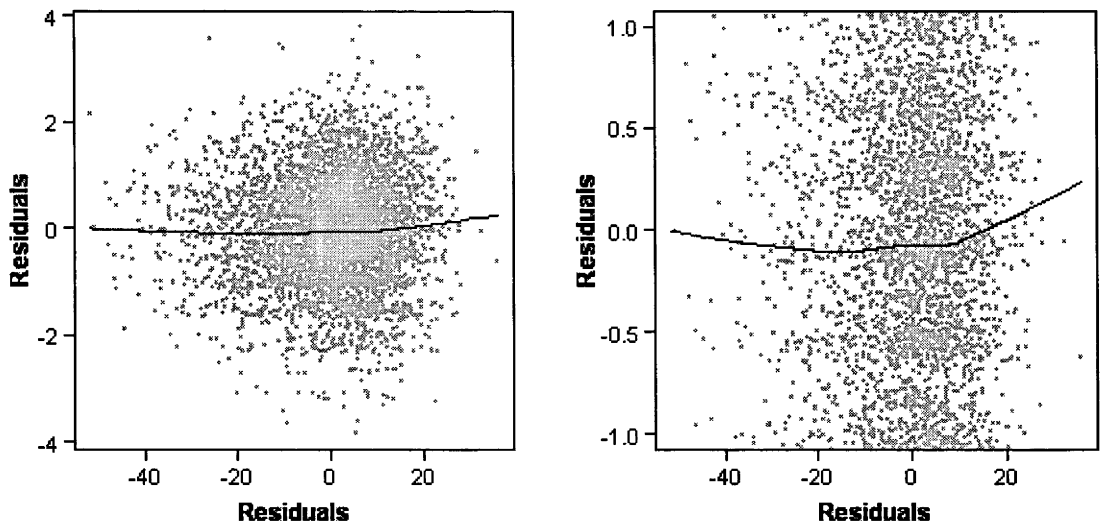
(Right hand side plot shows part of the left hand side plot from -1 to 1 of y-axis)



Note: Y-axis scale is different

Figure 6-7. Residual - residual plot: Plots of residuals of the basic model for non-external deaths in 65+ years age group against residuals of the same model for daily relative humidity

(Right hand side plot shows part of the left hand side plot from -1 to 1 of y-axis)



Note: Y-axis scale is different

Single pollutant analysis

Table 6-2 shows the results of single pollutant analysis for each mortality category. The effects of air pollutants were estimated by fitting separate models with only one lag of air pollutant at a time. The effect of air pollutant on daily mortality is expressed as a

percentage increase in daily mortality associated with one interquartile range increase in respective air pollutant concentrations. Figures 6-8, 6-9 and 6-10 show the comparisons of the effects of PM₁₀, CO and NO₂ on daily mortality for zero (the same day) to three day lags.

A significant positive association between daily non-external mortality and PM₁₀ was found for up to two days after exposure, with the largest risk for mortality two days after exposure. The associations of PM₁₀ with daily non-external mortality were stronger in the population aged 65+ years than in the whole population. An interquartile range (11.7 µg/m³) increase in two-day lag PM₁₀ concentrations was associated with an increase of 1.69% (95% CI: 0.70, 2.69) in non-external mortality in the population aged 65+ years. For the whole population, the estimated increase in non-external mortality for one interquartile range increase in two-day lag PM₁₀ concentrations was 1.31% (95% CI: 0.37, 2.26).

Carbon monoxide was associated with an increase in daily non-external mortality only in the elderly population (65+ years age group). In this age group, the statistically significant associations between CO and non-external mortality were observed for up to two days after exposure. The association was at the borderline significance for non-external deaths of the same day. An interquartile range (0.88 mg/m³) increase in two-day lag CO concentrations was associated with a 1.10% (95% CI: 0.07, 2.14) increase in non-external mortality in the elderly population. There was no statistically significant association between CO and daily non-external mortality for the whole population.

There was no evidence of a significant association of PM₁₀ and CO with same day circulatory mortality in the 65+ years age group but both pollutants were associated with an increase in next day mortality. For respiratory mortality in the 65+ years age group, no statistically significant association between PM₁₀ and mortality was found for same day mortality or next day mortality. PM₁₀ associated significant increased risk was found only for respiratory mortality two days after exposure. There was no significant association between CO and respiratory mortality in the 65+ years age group. Analysis did not provide any evidence of significant association between NO₂ and daily mortality for any mortality categories analysed.

The overdispersion parameters for the above models were approximately equal to one suggesting no overdispersion. Residual autocorrelation coefficients for various lags were estimated. No residual autocorrelation coefficient was greater than 0.05, which suggested that there was no serial correlation left in the residuals.

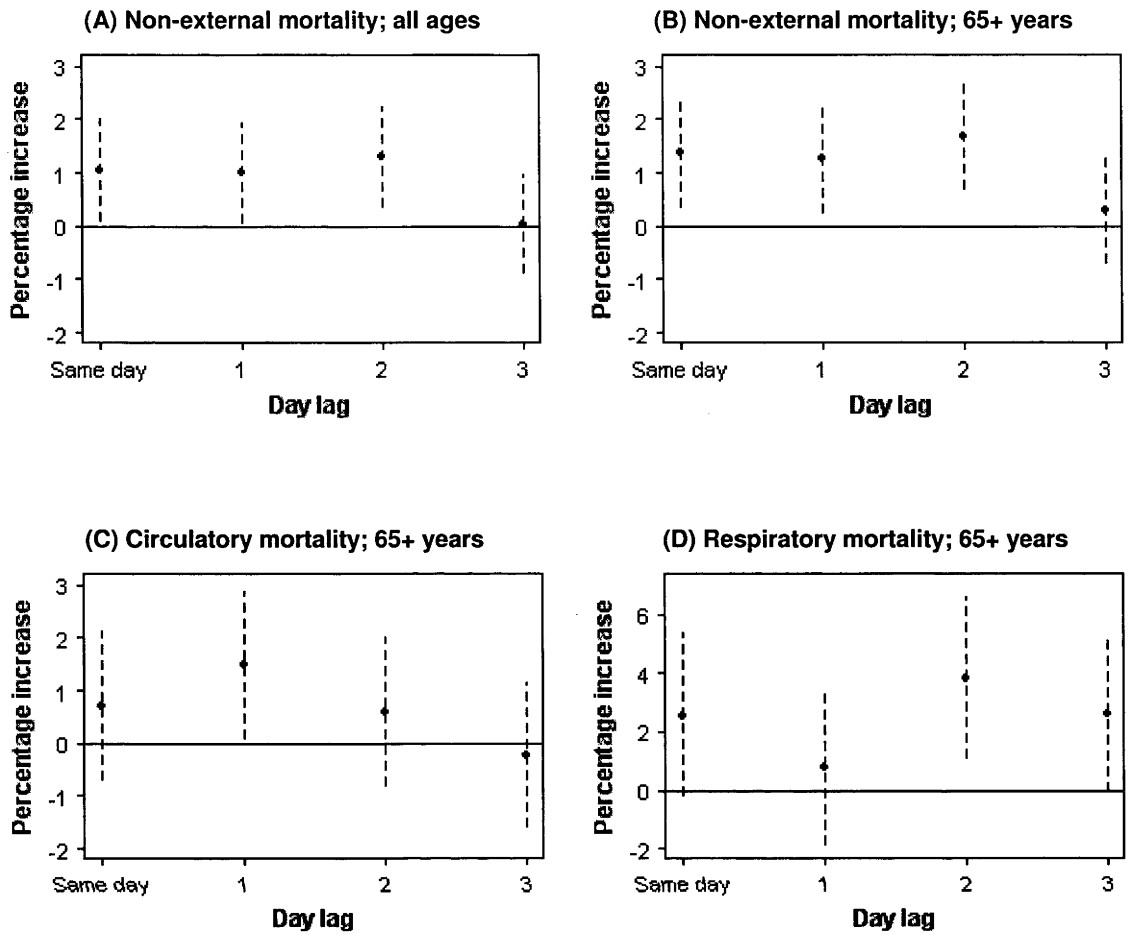
Table 6-2. Single pollutant model: Percentage increase (95% CI) in daily mortality associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³), daily CO concentrations (0.88 mg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories

Pollutant	Lag	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
PM ₁₀	Same day	1.05* (0.10, 2.01)	1.37** (0.37, 2.38)	0.71 (-0.70, 2.13)	2.54 (-0.19, 5.35)
	1	1.02* (0.09, 1.95)	1.25* (0.27, 2.25)	1.49* (0.11, 2.89)	0.77 (-1.89, 3.49)
	2	1.31** (0.37, 2.26)	1.69*** (0.70, 2.69)	0.60 (-0.80, 2.02)	3.81** (1.13, 6.56)
	3	0.05 (-0.87, 0.97)	0.28 (-0.69, 1.27)	-0.23 (-1.60, 1.16)	2.57 (-0.02, 5.24)
CO	Same day	0.75 (-0.22, 1.74)	1.01 (-0.04, 2.06)	1.01 (-0.44, 2.48)	0.37 (-2.43, 3.25)
	1	0.88 (-0.07, 1.84)	1.05* (0.04, 2.08)	1.93** (0.52, 3.36)	0.52 (-2.19, 3.30)
	2	0.76 (-0.21, 1.73)	1.10* (0.07, 2.14)	0.82 (-0.62, 2.28)	1.85 (-0.88, 4.67)
	3	-0.60 (-1.54, 0.36)	-0.47 (-1.48, 0.55)	-0.87 (-2.28, 0.56)	0.37 (-2.29, 3.11)
NO ₂	Same day	0.03 (-1.90, 2.00)	-0.41 (-2.35, 1.58)	-1.01 (-3.69, 1.75)	-2.65 (-8.29, 3.33)
	1	0.41 (-1.49, 2.34)	0.03 (-1.89, 1.98)	-0.88 (-3.51, 1.83)	-3.04 (-8.54, 2.79)
	2	0.48 (-1.44, 2.43)	0.29 (-1.64, 2.26)	-0.26 (-2.93, 2.48)	-1.76 (-7.34, 4.17)
	3	-0.79 (-2.67, 1.12)	-1.22 (-3.12, 0.72)	-0.77 (-3.41, 1.94)	-5.39 (-10.78, 0.31)

* p < 0.05; ** p < 0.01; *** p < 0.001

Lag 1, lag 2 and lag 3 represent the pollutant levels on the previous day, the 2 days before and the 3 days before respectively.

Figure 6-8. Percentage increase in daily deaths and 95% CI associated with one interquartile range ($11.7 \mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories



Note: Y-axis scale is different

Figure 6-9. Percentage increase in daily deaths and 95% CI associated with one interquartile range (0.88 mg/m³) increase in daily CO concentrations for various mortality categories

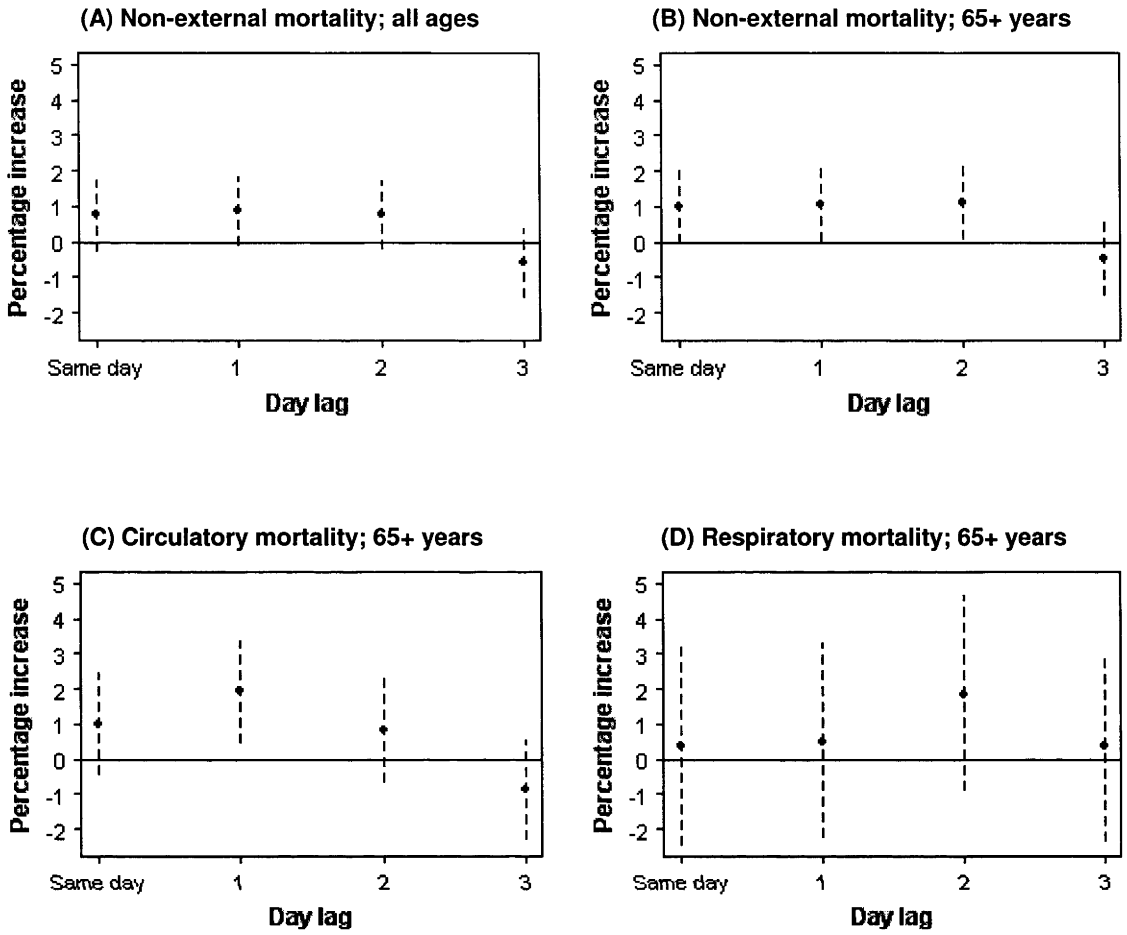
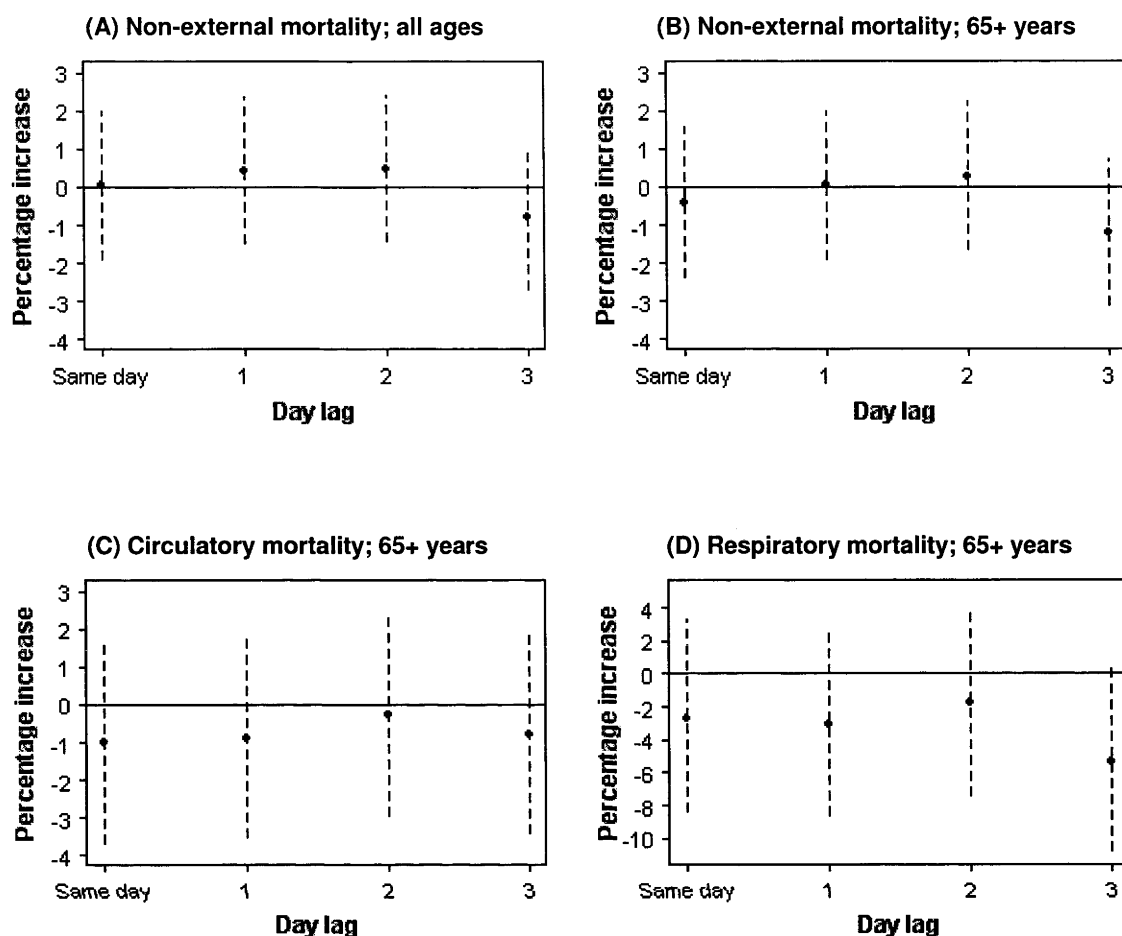


Figure 6-10. Percentage increase in daily deaths and 95% CI associated with one interquartile range (15.8 $\mu\text{g}/\text{m}^3$) increase in daily NO_2 concentrations for various mortality categories



Note: Y-axis scale is different

Multi-pollutant analysis

Table 6-3 summarises the results of multi-pollutant models for daily mortality with PM_{10} and CO as exposure variables. The effects of air pollutants were estimated by fitting separate models with only one lag of air pollutants at a time, with the same lag for both PM_{10} and CO. The same day and the lagged effects of pollutants on daily mortality from multi-pollutant analysis are compared in Figure 6-11. The statistically significant association between CO and non-external mortality in the 65+ years age group in single pollutant analysis (Table 6-2) became statistically non-significant when PM_{10} was included in the model in multi-pollutant analysis. Similarly, the associations of non-external mortality with PM_{10} were weakened except for the association with two-day lag PM_{10} when CO was included. The estimated PM_{10} effect sizes at this lag for both non-external mortality in the 65+ years age group and the whole population were larger in the multi-pollutant analysis. The increased risk of non-external mortality in the

65+ years age group associated with the same day's PM₁₀ was statistically significant only at the 10% level (p-value < 0.1).

The statistically significant positive associations of PM₁₀ and CO with daily circulatory mortality of 65+ years age group at lag 1 observed in single pollutant analysis disappeared in multi-pollutant analysis (Figure 6-11(C)). For respiratory mortality in the 65+ years age group, the estimated effect size of PM₁₀ was larger in multi-pollutant analysis (Figure 6-11(D)).

Table 6-3. Multi-pollutant model: Percentage increase (95% CI) in daily mortality associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily CO concentrations (0.88 mg/m³) for various mortality categories

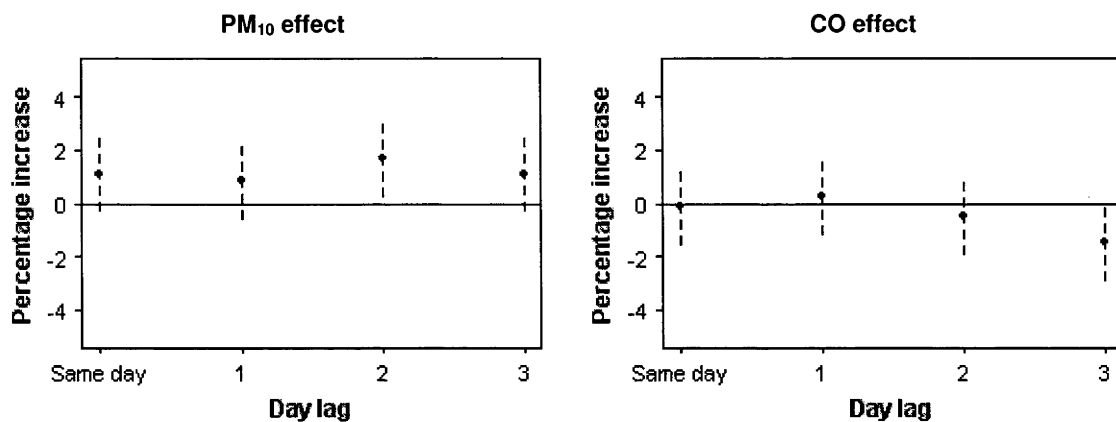
Pollutant	Lag	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
Model 1					
PM ₁₀	Same day	1.11 (-0.28, 2.52)	1.33 (-0.04, 2.72)	0.07 (-1.83, 2.01)	5.05* (0.92, 9.36)
CO	Same day	-0.08 (-1.50, 1.36)	0.06 (-1.35, 1.49)	0.96 (-1.01, 2.98)	-3.36 (-7.38, 0.84)
Model 2					
PM ₁₀	1	0.84 (-0.55, 2.24)	1.06 (-0.31, 2.44)	0.35 (-1.54, 2.28)	0.88 (-3.12, 5.04)
CO	1	0.25 (-1.16, 1.67)	0.29 (-1.11, 1.72)	1.68 (-0.28, 3.67)	-0.15 (-4.19, 4.06)
Model 3					
PM ₁₀	2	1.67* (0.28, 3.09)	1.83** (0.45, 3.22)	0.11 (-1.80, 2.06)	5.56** (1.48, 9.80)
CO	2	-0.50 (-1.91, 0.93)	-0.20 (-1.61, 1.22)	0.74 (-1.23, 2.75)	-2.30 (-6.27, 1.83)

* p < 0.05; ** p < 0.01; *** p < 0.001

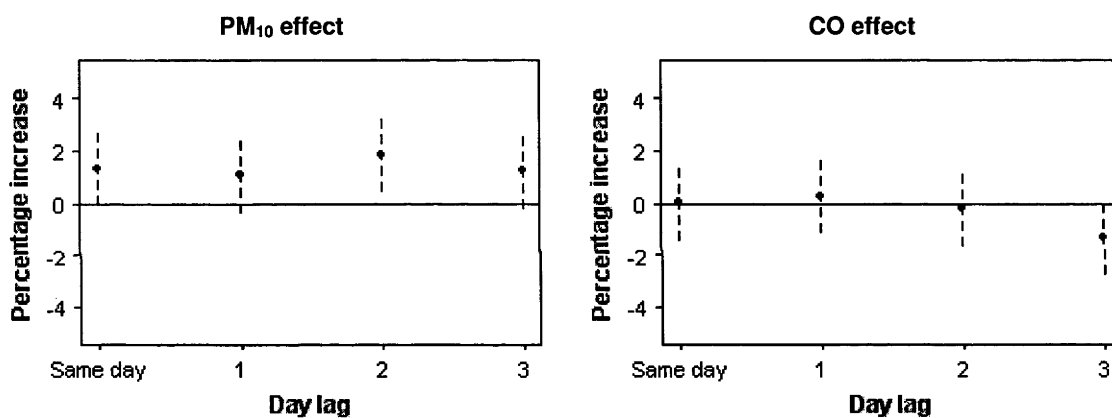
Lag 1, lag 2 and lag 3 represent the pollutant levels on the previous day, the 2 days before and the 3 days before respectively.

Figure 6-11. Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 μg/m³) and daily CO concentrations (0.88 mg/m³) for various mortality categories from multi-pollutant models

(A) Non-external mortality; all ages



(B) Non-external mortality; 65+ years



(C) Circulatory mortality; 65+ years

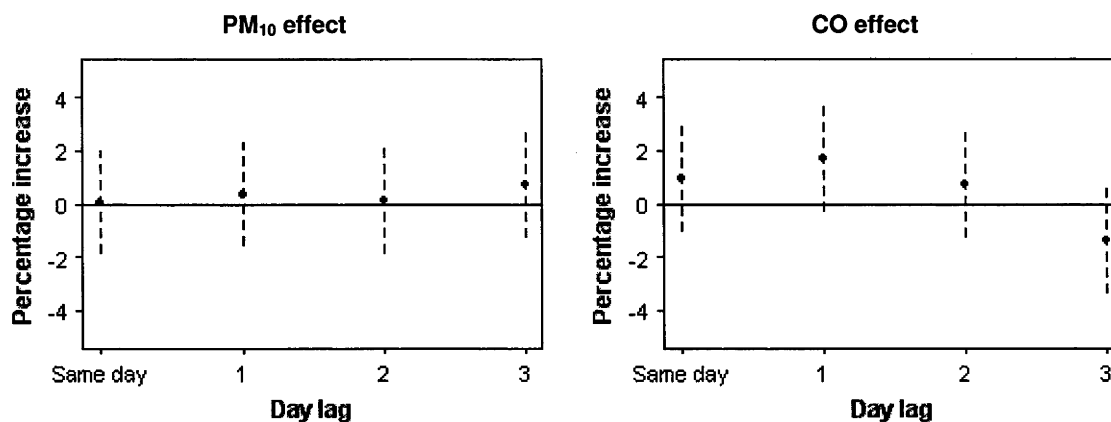
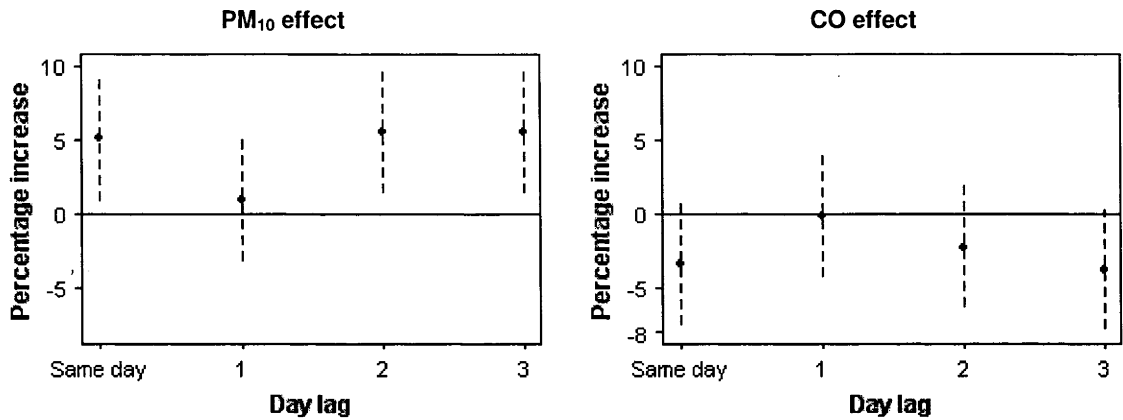


Figure 6-11 (cont.). Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily CO concentrations (0.88 mg/m³) for various mortality categories from multi-pollutant models

(D) Respiratory mortality; 65+ years



The results of multi-pollutant models for daily mortality with PM₁₀ and NO₂ as exposure variables are reported in Table 6-4. The effects of air pollutants were estimated by fitting separate models with only one lag of air pollutants at a time, with the same lag for both PM₁₀ and NO₂. The same day and the lagged effects of PM₁₀ and NO₂ on daily mortality from multi-pollutant models are compared in Figure 6-12. Adjusting for NO₂ did not change the associations between daily mortality and PM₁₀ for non-external mortality and circulatory mortality. For respiratory mortality in the 65+ years age group, the estimated PM₁₀ effect sizes on mortality were slightly larger in multi-pollutant models compared to single pollutant analysis (Figure 6-12(D)). The statistically non-significant associations between daily mortality and NO₂ for the mortality categories analysed in the single pollutant analysis remained unchanged in the multi-pollutant analysis, except for the association between three-day lag NO₂ and respiratory mortality in the 65+ years age group. A statistically significant negative association was observed between three-day lag NO₂ and respiratory mortality when three-day lag PM₁₀ was included in the model.

No significant association between daily mortality and NO₂ was found in either single or multi-pollutant analysis, except for the statistically significant negative association between three-day lag NO₂ and respiratory mortality in the 65+ years age group in multi-pollutant analysis. There was an association between CO and daily non-external mortality in the population aged 65+ years in single pollutant analysis but the association disappeared in multi-pollutant analysis. Although adding CO slightly weakened the association of PM₁₀ with daily non-external mortality in the population

aged 65+ years, the association was still statistically significant at the 10% level. Daily mortality appeared to be independently associated with only PM₁₀. Thus, only the association between daily mortality and PM₁₀ was analysed in the analysis of the association by season.

Table 6-4. Multi-pollutant model: Percentage increase (95% CI) in daily mortality associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories

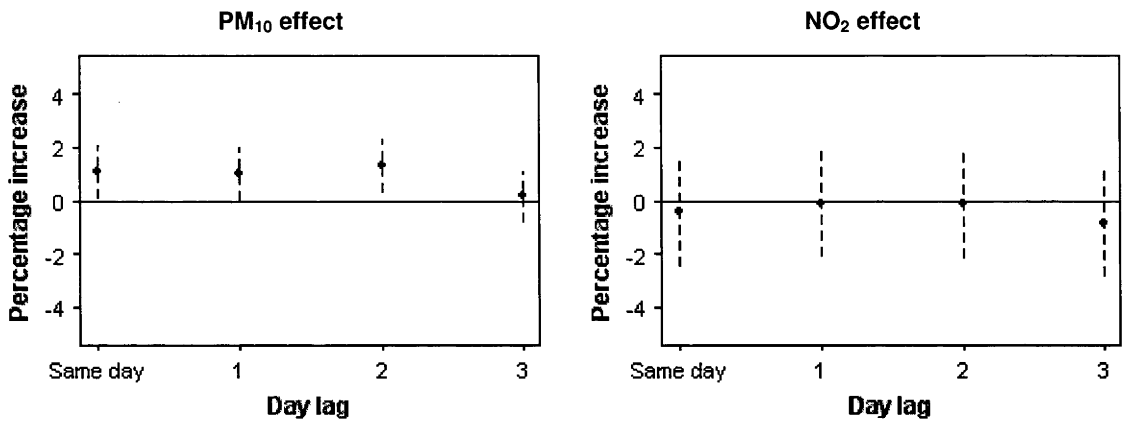
Pollutant	Lag	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
Model 1					
PM ₁₀	Same day	1.09* (0.13, 2.08)	1.50** (0.47, 2.54)	0.88 (-0.57, 2.34)	2.96* (0.16, 5.84)
NO ₂	Same day	-0.45 (-2.42, 1.56)	-1.08 (-3.07, 0.95)	-1.39 (-4.14, 1.43)	-4.00 (-9.69, 2.05)
Model 2					
PM ₁₀	1	1.03* (0.07, 1.99)	1.34** (0.32, 2.37)	1.73* (0.30, 3.18)	1.21 (-1.54, 4.04)
NO ₂	1	-0.11 (-2.05, 1.88)	-0.66 (-2.62, 1.35)	-1.74 (-4.45, 1.04)	-3.69 (-9.34, 2.31)
Model 3					
PM ₁₀	2	1.32** (0.36, 2.29)	1.76*** (0.74, 2.80)	0.68 (-0.77, 2.14)	4.25** (1.49, 7.08)
NO ₂	2	-0.15 (-2.10, 1.85)	-0.56 (-2.54, 1.46)	-0.58 (-3.33, 2.24)	-3.86 (-9.48, 2.12)

* p < 0.05; ** p < 0.01; *** p < 0.001

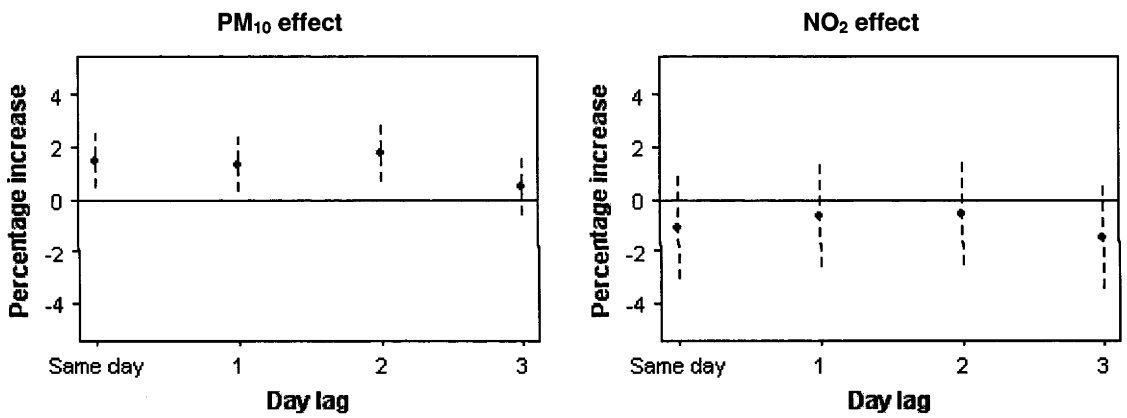
Lag 1 and lag 2 represent the pollutant levels on the previous day and the 2 days before respectively.

Figure 6-12. Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories from multi-pollutant models

(A) Non-external mortality; all ages



(B) Non-external mortality; 65+ years



(C) Circulatory mortality; 65+ years

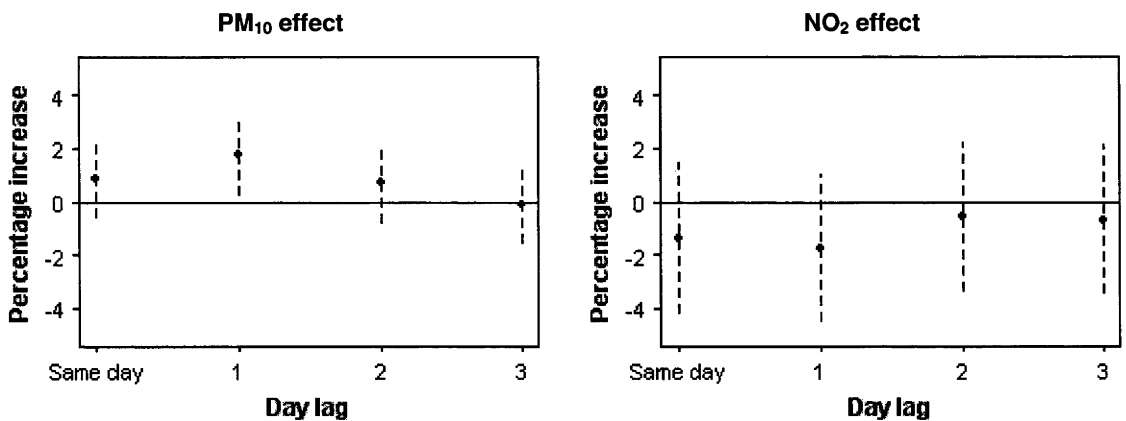
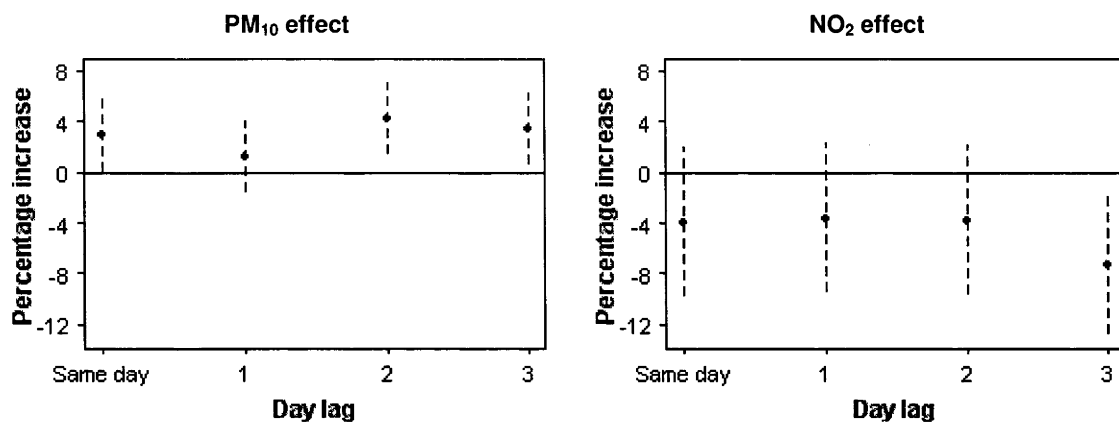


Figure 6-12 (cont.). Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories from multi-pollutant models

(D) Respiratory mortality; 65+ years



Effect modification by season

Table 6-5 summarises the results of seasonal analysis of the association between PM₁₀ and daily mortality for up to two day lags. Figure 6-13 gives graphical representations of the associations by season. The percentage increases in daily mortality and their 95% confidence interval for one interquartile range (11.7 µg/m³) increase in daily PM₁₀ concentrations are reported by season for various mortality categories together with the p-values for the significance test of the interaction between the season variable and daily PM₁₀ concentrations. Significant p-values indicate seasonal differences in the association between PM₁₀ and daily mortality.

There was a significant seasonal difference in the association between PM₁₀ and non-external mortality both in whole population and in the elderly population (65+ years). The PM₁₀ associated risks of non-external mortality were higher in the warm season (September - April) than in the cool season (May - August). In the warm season, significantly increased risks were found for up to two days after exposure for both the whole population and the elderly population. In the cool season, the increased risk of non-external mortality for the whole population was significant only for mortality two days after exposure.

The analysis for circulatory mortality in the elderly population did not show statistically significant seasonal differences in the association between PM₁₀ and daily circulatory mortality. The results were consistent across different lags. There was no evidence of association between PM₁₀ and daily circulatory mortality in both seasons. The significant positive association of PM₁₀ with the next day circulatory mortality in

the 65+ years age group (Table 6-2) disappeared when the associations were analysed by season.

For respiratory mortality in the 65+ years age group, there was a strong seasonal difference in the association between daily mortality and PM₁₀. It was consistent across different lags. The PM₁₀ associated risks of respiratory mortality were significantly higher in the warm season (September - April) than in the cool season (May - August). One interquartile range increase in PM₁₀ concentrations was associated with a 15.61% (95% CI: 6.07, 26.02) increase in same day respiratory mortality in the warm season compared to a 1.66% (95% CI: -1.14, 4.55) increase in the cool season (Table 6-5).

The 95% confidence intervals of the effect estimates were much wider in the warm season than in the cool season. It was most likely due to the greater variability in daily PM₁₀ concentrations in the cool season than in the warm season. The standard deviation of daily PM₁₀ was 25.03 µg/m³ in the cool season compared to 6.41 µg/m³ in the warm season.

Table 6-5. Percentage increase (95% CI) in daily mortality, by season, associated with one interquartile range (11.7 µg/m³) increase in daily PM₁₀ concentrations for various mortality categories

Lag	Season [‡]	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
Same day	Warm	3.93^{**} (0.99, 6.96)	4.26^{**} (1.12, 7.49)	0.78 (-3.48, 5.22)	15.61^{***} (6.07, 26.02)
		0.82 (-0.16, 1.81)	1.09[*] (0.05, 2.15)	0.70 (-0.76, 2.18)	1.66 (-1.14, 4.55)
	p-value [§]	0.043	0.057	0.974	0.004
1	Warm	4.17^{**} (1.25, 7.18)	4.75^{**} (1.63, 7.97)	3.74 (-0.56, 8.22)	11.21[*] (1.93, 21.33)
		0.75 (-0.21, 1.73)	0.92 (-0.11, 1.95)	1.27 (-0.17, 2.73)	0.04 (-2.69, 2.85)
	p-value [§]	0.025	0.020	0.284	0.020
2	Warm	4.43^{**} (1.49, 7.44)	5.31^{***} (2.17, 8.55)	2.06 (-2.21, 6.51)	14.19^{**} (4.71, 24.54)
		1.05[*] (0.08, 2.03)	1.35[*] (0.31, 2.39)	0.46 (-1.003, 1.94)	3.15[*] (0.40, 5.97)
	p-value [§]	0.028	0.017	0.486	0.024

[‡] Warm season (Non-winter months): September to the following April;

Cool season (Winter months): May - August

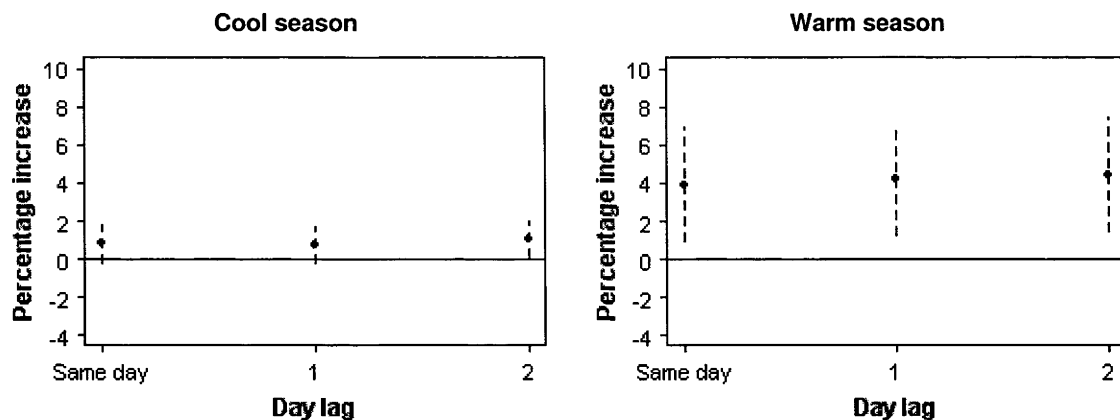
* p < 0.05; ** p < 0.01; *** p < 0.001

Lag 1 and lag 2 represent the pollutant levels on the previous day and the 2 days before respectively.

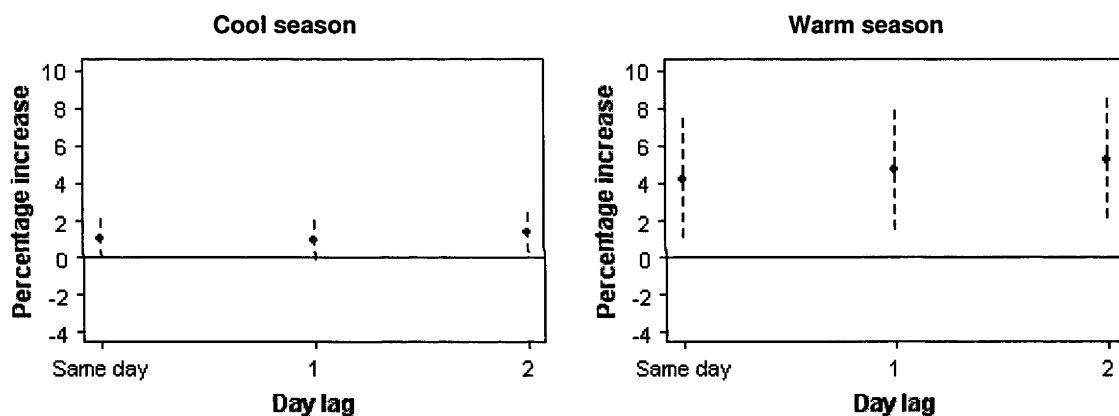
§ P-value for the significance test of the interaction between season and daily PM₁₀ concentration

Figure 6-13. Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories by season

(A) Non-external mortality; all ages



(B) Non-external mortality; 65+ years



(C) Circulatory mortality; 65+ years

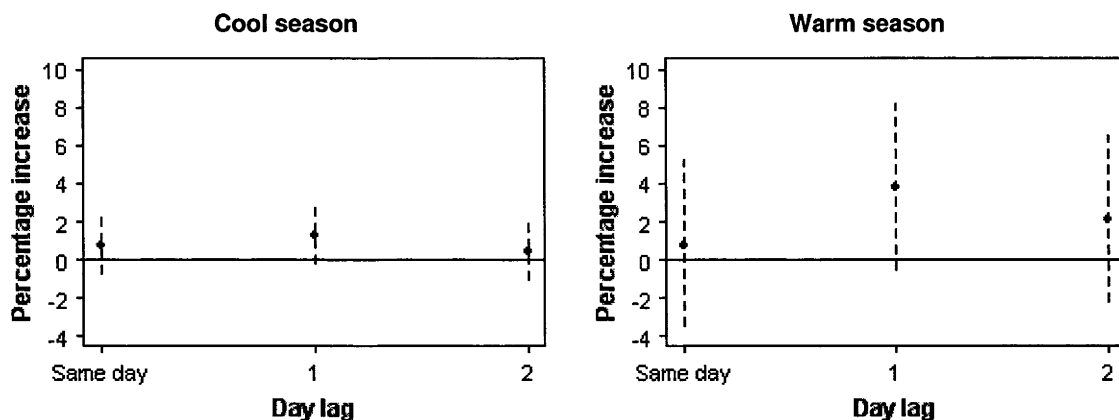
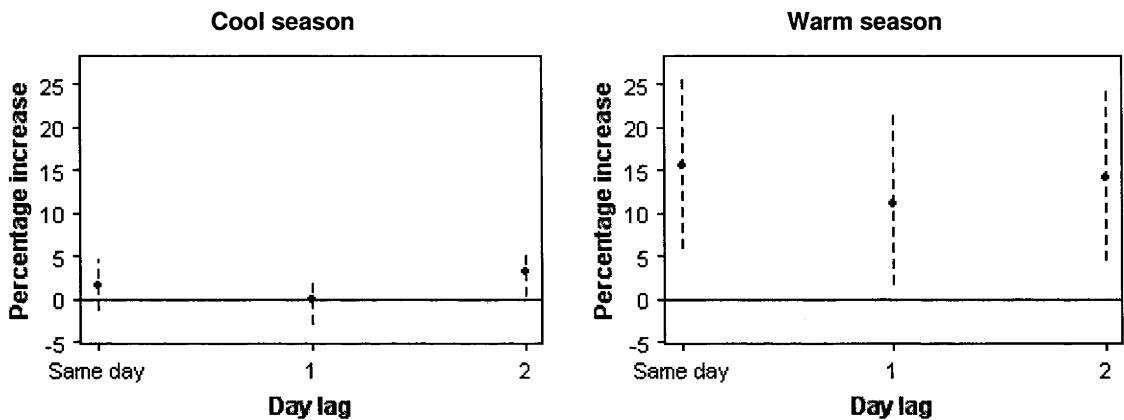


Figure 6-13 (cont.). Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories by season

(D) Respiratory mortality; 65+ years



6.4.2 Method 2: Using natural splines

In the second method, the confounding effects of long-term trend, seasonal variation and weather variables were adjusted for by using natural cubic spline smooth functions of all confounders. The associations between daily mortality and daily pollutant concentrations were analysed separately for each mortality category using the Poisson regression model

$$E(Y_t) = Pop \times \exp(\beta_0 + ns(time, 81) + \gamma DOW_t + ns(tempg_t, 6) + ns(tempg_{1-3}, 6) + ns(rh_t, 3) + ns(rh_{1-3}, 3) + \beta_1 PM_{t-q}) \quad (6-3)$$

Where:

t refers to the day of the study.

q refers to the lag number.

$E(Y_t)$ is the expected number of deaths on day “ t ”.

Pop is the population at risk, which is the annual usual resident population.

DOW_t is the set of dummy variables for days of week.

γ is the set of coefficients that adjusted the daily variation in mortality across the week.

$ns(X, k)$ refers to a natural cubic spline smooth function of variable “ X ” with “ k ” degrees of freedom.

$ns(time, 81)$ is a natural cubic spline smooth function of time variable with 81 degrees of freedom i.e. approximately 7 df / year of data.

$tempg_t$ is the same day’s average hourly temperature.

$temp_{1-3}$ is the average of three previous days' average hourly temperature (average of lags 1 to 3).

rh_t is the same day's average hourly relative humidity.

rh_{1-3} is the average of three previous days' average relative humidity (average of lags 1 to 3).

PM_{t-q} is PM_{10} concentrations on day " $t-q$ ".

β_I is the log relative risk of mortality associated with an increase of $1 \mu\text{g}/\text{m}^3$ in PM_{10} on " $t-q$ "th day. The percentage increase in daily mortality associated with one interquartile range increase in daily PM_{10} concentrations is calculated as $100[(e^{IQR \beta_I}) - 1]\%$, where IQR is one interquartile range of daily PM_{10} concentrations.

The above Poisson regression model (equation (6-3)) was first fitted to estimate the percentage increase in mortality associated with an increase in the same day's PM_{10} (PM_t). Replacing same day PM_{10} by previous days PM_{10} (PM_{t-1} , PM_{t-2} , PM_{t-3}), the lagged effects of PM_{10} were estimated separately for up to 3 day lags. Similarly, using CO and NO_2 instead of PM_{10} , the corresponding percentage increases in daily mortality associated with increases in CO and NO_2 concentrations were estimated. For multi-pollutant models, I fitted the models with two pollutant variables at the same time.

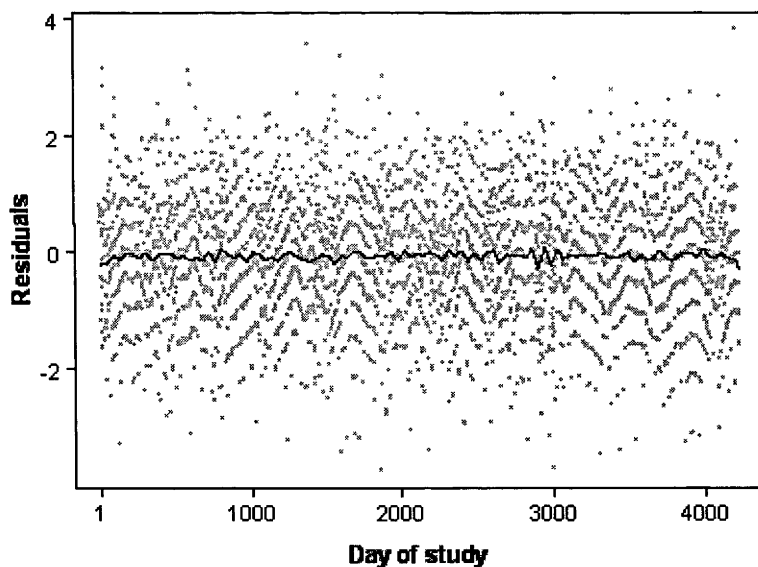
Residual diagnosis

Before including weather variables and air pollutant variables, various diagnoses of the residuals of the basic models, which only had a natural cubic spline smooth function of time and dummy variables for days of the week, were performed. This was to check whether the basic models adequately adjusted for seasonal variation and a long-term temporal trend in daily deaths. Diagnoses were separately performed for the residuals of the basic models for each of the mortality categories (i.e. daily non-external deaths of all ages, daily non-external deaths in the 65+ years age group, daily circulatory deaths in the 65+ years age group, and daily respiratory deaths in the 65+ years age group). Figures 6-14 and 6-15 show the diagnostic plots for residuals of the basic model for non-external deaths in the 65+ years age group that adjusted for seasonal variation and a long-term temporal trend.

Figure 6-14 shows residuals of the basic model for non-external deaths in the 65+ years age group plotted against day of study. The grey points in the plot are the residuals for each day. The dark line is a non-parametric smoothing (lowess smoothing with a bandwidth of 0.02) of the scatter plot to reveal any pattern present in the plot.

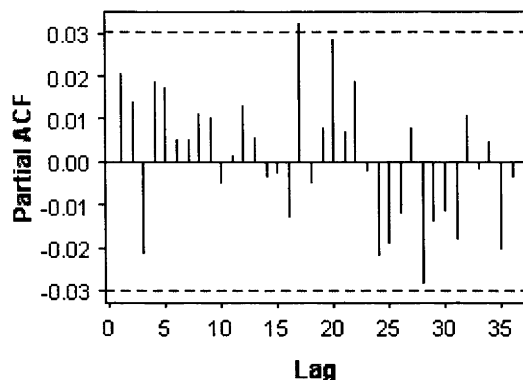
There was no seasonal pattern left in the residuals suggesting that the basic model adequately adjusted a long-term trend and seasonal variation in daily non-external deaths in the 65+ years age group. Figure 6-15 shows the partial autocorrelation functions (Partial ACF) for the residuals from the basic model for daily non-external deaths in the 65+ years age group, which only had a natural cubic spline smooth function of time and dummy variables for days of the week. There was no large partial autocorrelations in the residual series. The original daily mortality series had large positive partial autocorrelations (Figure 6-3 (A)). The absence of large positive values at the first lags in the partial autocorrelations of the residual series suggests that the basic model adequately adjusted for seasonal variation. The diagnosis of the residuals of the basic models for other mortality categories also confirmed that the basic models for respective mortality categories reasonably controlled for seasonal variation and a long-term trend in daily mortality.

Figure 6-14. Residuals after adjusting for a long-term trend and seasonal variation in daily non-external deaths in 65+years age group against day of study



Bandwidth for lowess smoothing = 0.02

Figure 6-15. Partial autocorrelation plot for residuals after adjusting for seasonal variation and a long-term trend in daily non-external deaths in 65+ years age group



Single pollutant analysis

Results of single pollutant analysis for each mortality category are shown in Table 6-6. The effects of air pollutants were estimated by fitting separate models with only one lag of air pollutant at a time. The comparisons of the same day and the lagged effects of PM₁₀, CO and NO₂ on daily mortality are shown in Figures 6-16, 6-17 and 6-18 respectively for various mortality categories. The estimated percentage increases in daily mortality and their 95% confidence intervals shown in Table 6-6 and Figures 6-16, 6-17 and 6-18 are for one interquartile range increase in respective daily pollutant concentrations. Results are shown for up to three day lags.

Both PM₁₀ and CO were positively associated with an increase in daily non-external mortality. The statistically significant positive associations between daily non-external mortality and PM₁₀ and CO were observed for up to 2 days after exposure, with the largest effects occurring on the same day mortality. The associations of PM₁₀ and CO with daily non-external mortality were stronger in the population aged 65+ years than in the whole population. An interquartile range (11.7 µg/m³) increase in PM₁₀ concentrations was associated with an increase of 1.82% (95% CI: 0.71, 2.95) in same day non-external mortality of the elderly population. For all ages, there was an estimated increase of 1.54% (95% CI: 0.52, 2.57) in same day non-external mortality for one interquartile range increase in PM₁₀. An interquartile range (0.88 mg/m³) increase in CO was associated with 1.57% (95% CI: 0.46, 2.70) increase in same day non-external mortality in the elderly population and 1.35% (95% CI: 0.33, 2.37) increase in same day non-external mortality in the whole population.

Only previous day's PM₁₀ and CO concentrations were significantly associated with an increase in daily circulatory mortality of people aged 65+ years. For respiratory mortality of people aged 65+ years, estimated PM₁₀ effect was statistically significant

only on the mortality occurring two days after exposure. Although the associations of PM_{10} with respiratory mortality in the population aged 65+ years were not statistically significant, PM_{10} had a higher effect on respiratory mortality than on other mortality categories.

No significant association between CO and respiratory mortality of those aged 65+ years was observed. Analysis did not provide any evidence of a significant association between NO_2 and daily mortality for any mortality categories analysed.

The check of dispersion parameter and residual autocorrelation coefficients for the above models provided no evidence of overdispersion and high residual autocorrelations. The dispersion parameters were approximately equal to one. The residual autocorrelation coefficients for lag 1 up to lag 365 were estimated without conducting any statistical test. None of the residual autocorrelation coefficients was greater than 0.1, suggesting there was no serial autocorrelation left in the residuals.

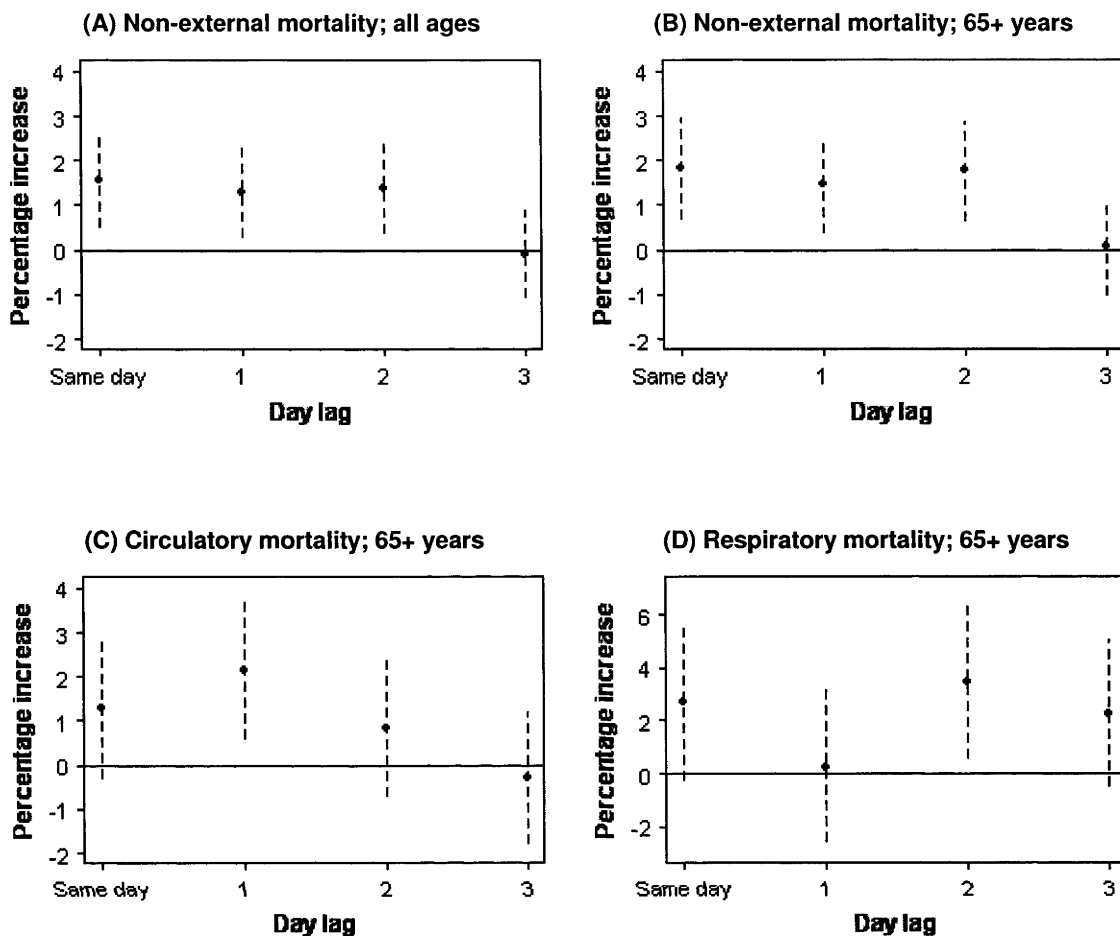
Table 6-6. Single pollutant model: Percentage increase (95% CI) in daily mortality associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³), daily CO concentrations (0.88 mg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories

Pollutant	Lag	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
PM ₁₀	Same day	1.54** (0.52, 2.57)	1.82** (0.71, 2.95)	1.30 (-0.26, 2.88)	2.69 (-0.21, 5.68)
	1	1.28* (0.29, 2.28)	1.46** (0.38, 2.55)	2.16** (0.64, 3.71)	0.26 (-2.53, 3.14)
	2	1.39** (0.39, 2.40)	1.77** (0.68, 2.88)	0.84 (-0.70, 2.41)	3.48* (0.65, 6.39)
	3	-0.09 (-1.05, 0.88)	0.06 (-0.99, 1.11)	-0.29 (-1.77, 1.20)	2.30 (-0.40, 5.07)
CO	Same day	1.35** (0.33, 2.37)	1.57** (0.46, 2.70)	1.49 (-0.06, 3.06)	0.69 (-2.20, 3.66)
	1	1.24* (0.25, 2.24)	1.36* (0.28, 2.46)	2.36** (0.85, 3.90)	0.47 (-2.33, 3.36)
	2	0.88 (-0.12, 1.89)	1.21* (0.11, 2.31)	0.86 (-0.68, 2.42)	1.63 (-1.20, 4.53)
	3	-0.62 (-1.58, 0.36)	-0.57 (-1.62, 0.49)	-1.14 (-2.62, 0.36)	0.29 (-2.42, 3.08)
NO ₂	Same day	0.98 (-1.31, 3.32)	0.99 (-1.54, 3.58)	0.86 (-2.63, 4.48)	1.33 (-5.45, 8.60)
	1	0.90 (-1.35, 3.20)	1.03 (-1.46, 3.57)	0.19 (-3.22, 3.73)	0.05 (-6.54, 7.11)
	2	0.66 (-1.60, 2.97)	1.13 (-1.37, 3.69)	0.77 (-2.68, 4.33)	1.10 (-5.59, 8.26)
	3	-1.19 (-3.39, 1.05)	-1.38 (-3.80, 1.10)	-0.23 (-3.60, 3.25)	-3.87 (-10.2, 2.96)

* p < 0.05; ** p < 0.01; *** p < 0.001

Lag 1, lag 2 and lag 3 represent the pollutant levels on the previous day, the 2 days before and the 3 days before respectively.

Figure 6-16. Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories



Note: Y-axis scale is different

Figure 6-17. Percentage increase in daily deaths and 95% CI associated with one interquartile range (0.88 mg/m³) increase in daily CO concentrations for various mortality categories

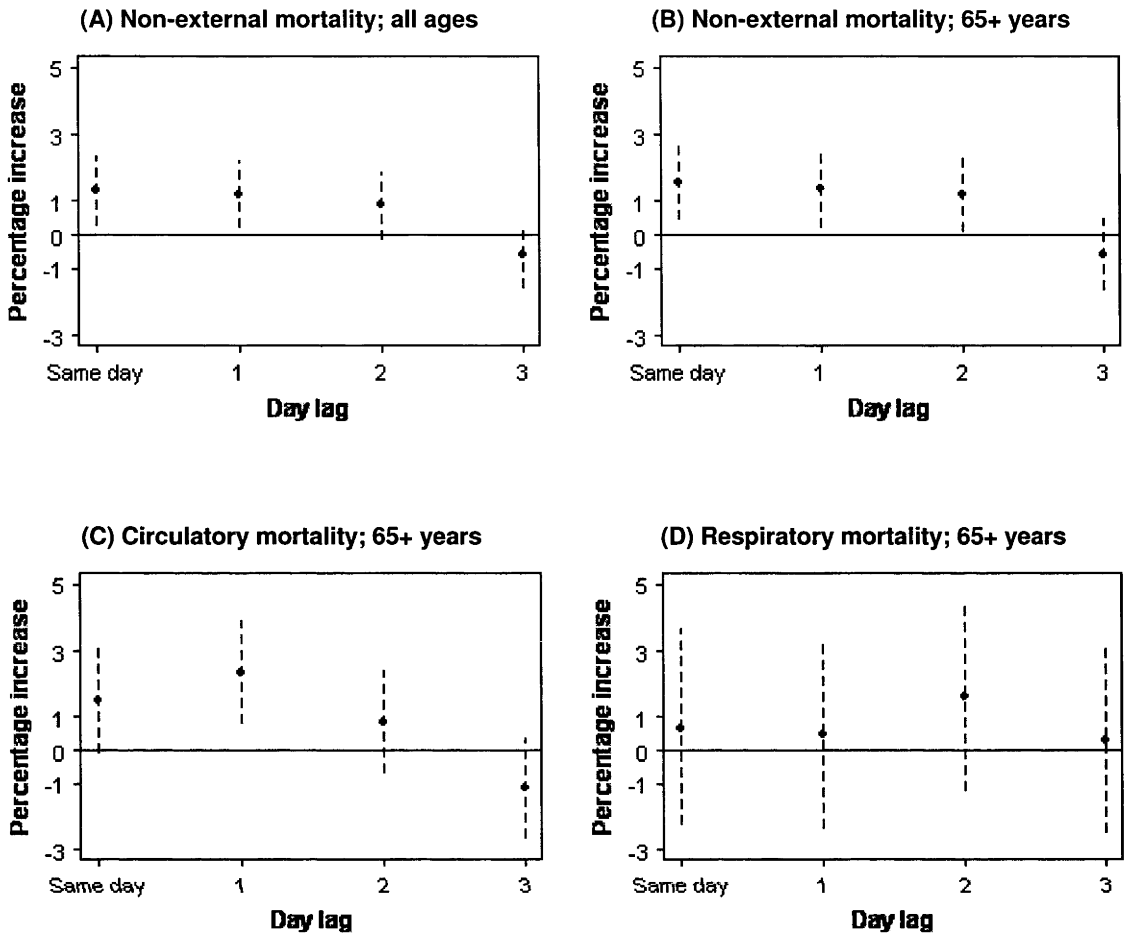
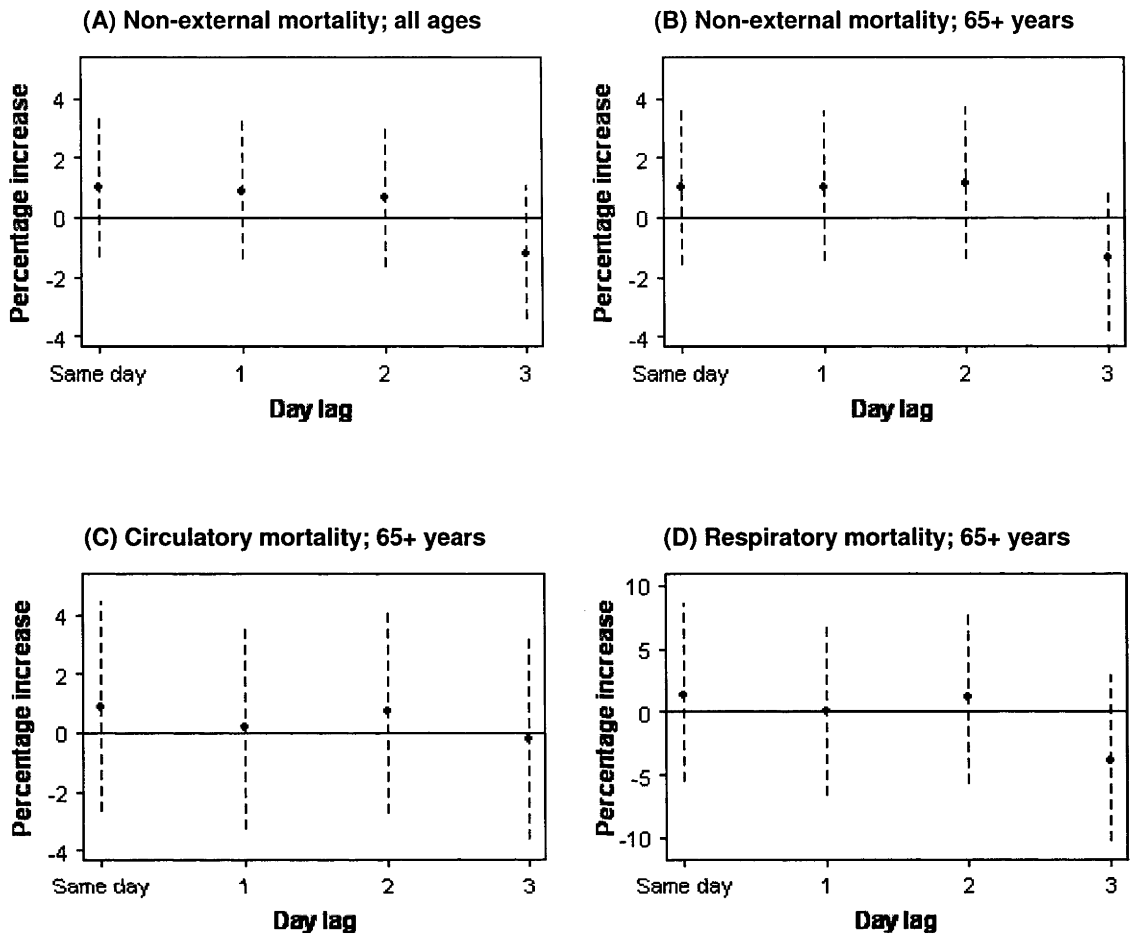


Figure 6-18. Percentage increase in daily deaths and 95% CI associated with one interquartile range ($15.8 \mu\text{g}/\text{m}^3$) increase in daily NO_2 concentrations for various mortality categories



Note: Y-axis scale is different

Multi-pollutant analysis

Table 6-7 summarises the results of multi-pollutant models for daily mortality with PM_{10} and CO as exposure variables. The effects of air pollutants were estimated by fitting separate models with only one lag of air pollutants at a time, with the same lag for both PM_{10} and CO. The same day and the lagged effects of pollutants on daily mortality from multi-pollutant models are compared in Figure 6-19. Single pollutant analysis showed that both CO and PM_{10} were associated with an increase in daily non-external mortality (Table 6-6). The associations were weakened when a second pollutant was included in multi-pollutant analysis. The association of CO with daily non-external mortality was no longer statistically significant at any lag in multi-pollutant analysis. While the associations of PM_{10} with daily non-external mortality at lags 0 and 1 became weaker and were statistically significant only at the 10% level ($p\text{-value} < 0.1$) (Table 6-7), the 2-day lag association remained statistically significant at the 5% level.

The estimated effect sizes of PM₁₀ on daily non-external mortality were smaller for the same day mortality and the next day mortality in multi-pollutant models compared to single pollutant model. However, controlling for CO slightly increased the size of the effect of PM₁₀ two days prior to mortality on non-external mortality.

The statistically significant positive associations of PM₁₀ and CO with daily circulatory mortality of people aged 65+ years at lag 1 observed in single pollutant analysis disappeared in multi-pollutant analysis (Figure 6-19(C)). For respiratory mortality of people aged 65+ years, the estimated effect size of PM₁₀ was larger in multi-pollutant models (Figure 6-19(D)).

Table 6-7. Multi-pollutant model: Percentage increase (95% CI) in daily mortality associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily CO concentrations (0.88 mg/m³) for various mortality categories

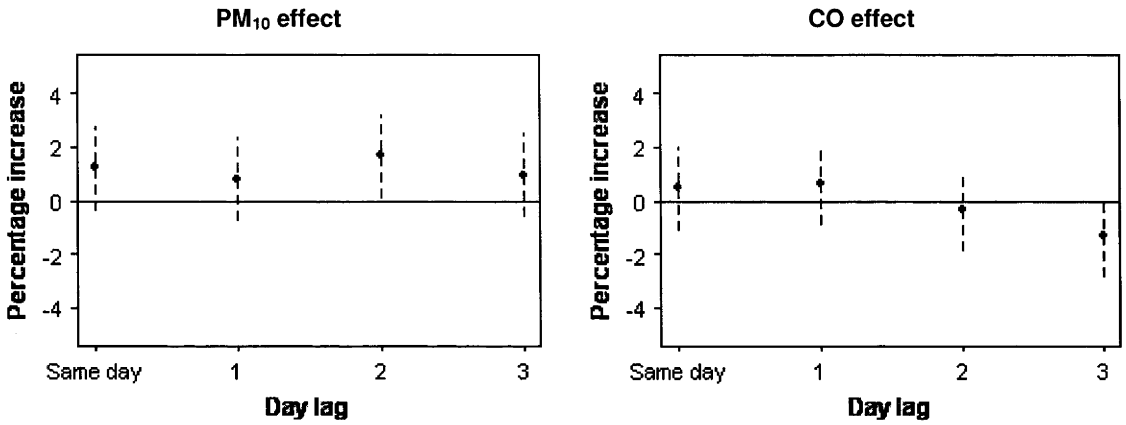
Pollutant	Lag	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
Model 1					
PM ₁₀	Same day	1.20 (-0.32, 2.75)	1.46 (-0.21, 3.16)	0.39 (-1.94, 2.77)	5.08* (0.57, 9.79)
CO	Same day	0.45 (-1.06, 1.99)	0.48 (-1.18, 2.17)	1.20 (-1.12, 3.57)	-3.03 (-7.29, 1.41)
Model 2					
PM ₁₀	1	0.81 (-0.70, 2.33)	1.003 (-0.65, 2.68)	0.85 (-1.46, 3.21)	-0.23 (-4.48, 4.22)
CO	1	0.63 (-0.88, 2.16)	0.60 (-1.05, 2.28)	1.71 (-0.60, 4.08)	0.65 (-3.65, 5.14)
Model 3					
PM ₁₀	2	1.67* (0.14, 3.21)	1.99* (0.33, 3.69)	0.45 (-1.88, 2.83)	5.30* (0.89, 9.91)
CO	2	-0.37 (-1.87, 1.16)	-0.29 (-1.94, 1.38)	0.52 (-1.80, 2.90)	-2.31 (-6.51, 2.07)

* p < 0.05; ** p < 0.01; *** p < 0.001

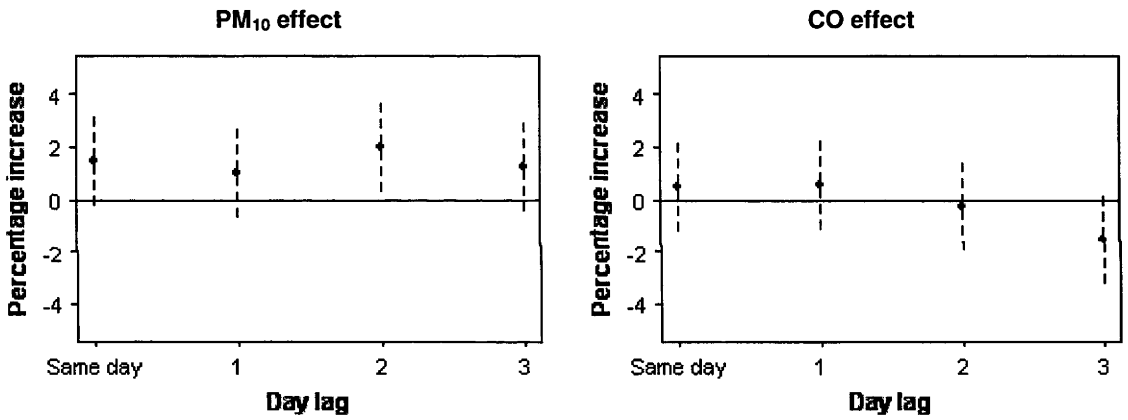
Lag 1, lag 2 and lag 3 represent the pollutant levels on the previous day, the 2 days before and the 3 days before respectively.

Figure 6-19. Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 μg/m³) and daily CO concentrations (0.88 mg/m³) for various mortality categories from multi-pollutant models

(A) Non-external mortality; all ages



(B) Non-external mortality; 65+ years



(C) Circulatory mortality; 65+ years

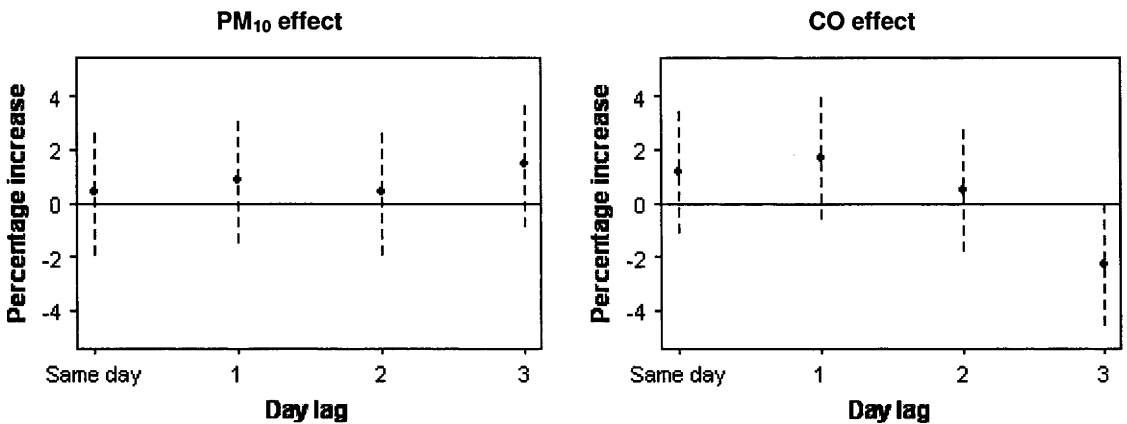
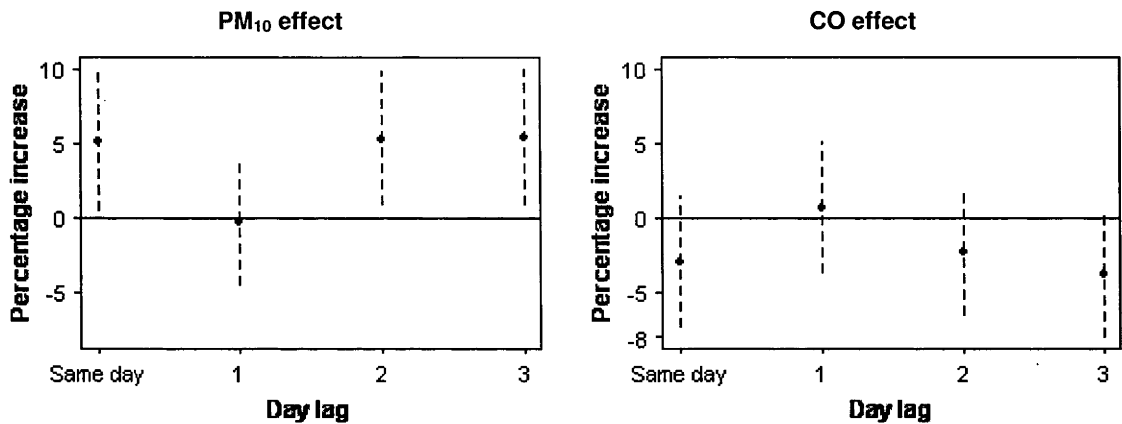


Figure 6-19 (cont.). Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily CO concentrations (0.88 mg/m³) for various mortality categories from multi-pollutant models

(D) Respiratory mortality; 65+ years



The results of multi-pollutant models for daily mortality with PM₁₀ and NO₂ as exposure variables are reported in Table 6-8. The effects of air pollutants were estimated by fitting separate models with only one lag of air pollutants at a time, with the same lag for both PM₁₀ and NO₂. The same day and the lagged effects of PM₁₀ and NO₂ on daily mortality from multi-pollutant models are compared in Figure 6-20. Adjusting for NO₂ did not change the associations between daily mortality and PM₁₀ for non-external mortality and circulatory mortality of people aged 65+ years. For respiratory mortality in the 65+ years age group, the estimated effect sizes of PM₁₀ on mortality two days (lag 2) and three days (lag 3) after exposure were larger in multi-pollutant models (Figure 6-20(D)). The statistically non-significant associations between daily mortality and NO₂ for the mortality categories analysed in single pollutant analysis remained unchanged in multi-pollutant analysis.

Analysis of single pollutant models showed that there was an association between CO and daily non-external mortality but the association disappeared in multi-pollutant analysis when PM₁₀ was included. Although adding CO weakened the associations of PM₁₀ with daily non-external mortality in multi-pollutant models, they were statistically significant at the 10% level. No significant association between daily mortality and NO₂ was observed. Daily mortality appeared to be primarily associated with PM₁₀. Thus, only the associations between daily mortality and PM₁₀ were analysed in the subsequent analyses.

Table 6-8. Multi-pollutant model: Percentage increase (95% CI) in daily mortality associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories

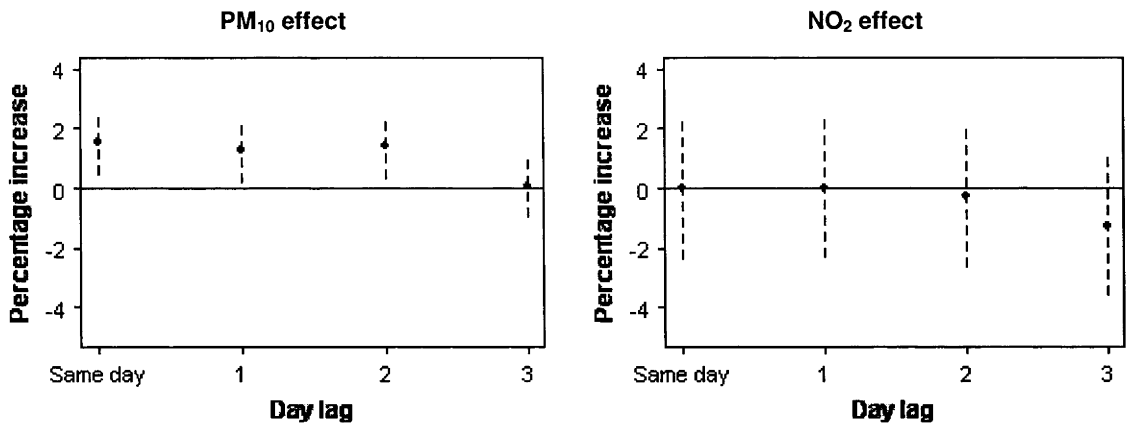
Pollutant	Lag	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
Model 1					
PM ₁₀	Same day	1.54** (0.48, 2.61)	1.85** (0.69, 3.02)	1.29 (-0.33, 2.95)	2.76 (-0.27, 5.87)
NO ₂	Same day	-0.001 (-2.37, 2.42)	-0.20 (-2.80, 2.48)	0.03 (-3.59, 3.78)	-0.53 (-7.48, 6.94)
Model 2					
PM ₁₀	1	1.28* (0.24, 2.33)	1.46* (0.32, 2.60)	2.36** (0.75, 3.98)	0.29 (-2.65, 3.31)
NO ₂	1	0.03 (-2.31, 2.42)	0.02 (-2.56, 2.67)	-1.41 (-4.95, 2.25)	-0.16 (-7.08, 7.27)
Model 3					
PM ₁₀	2	1.42** (0.37, 2.48)	1.78** (0.63, 2.93)	0.81 (-0.80, 2.46)	3.66* (0.70, 6.70)
NO ₂	2	-0.26 (-2.60, 2.13)	-0.05 (-2.63, 2.61)	0.23 (-3.35, 3.94)	-1.46 (-8.29, 5.88)

* p < 0.05; ** p < 0.01; *** p < 0.001

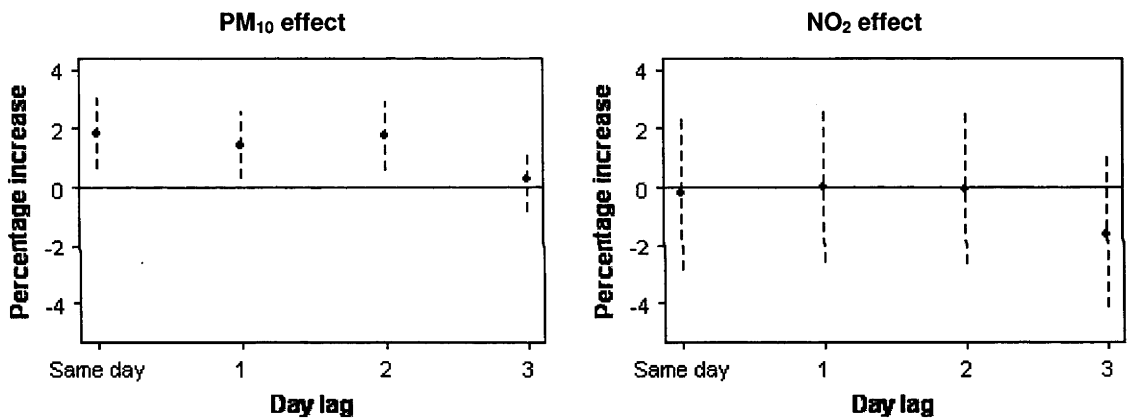
Lag 1 and lag 2 represent the pollutant levels on the previous day and the 2 days before respectively.

Figure 6-20. Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories from multi-pollutant models

(A) Non-external mortality; all ages



(B) Non-external mortality; 65+ years



(C) Circulatory mortality; 65+ years

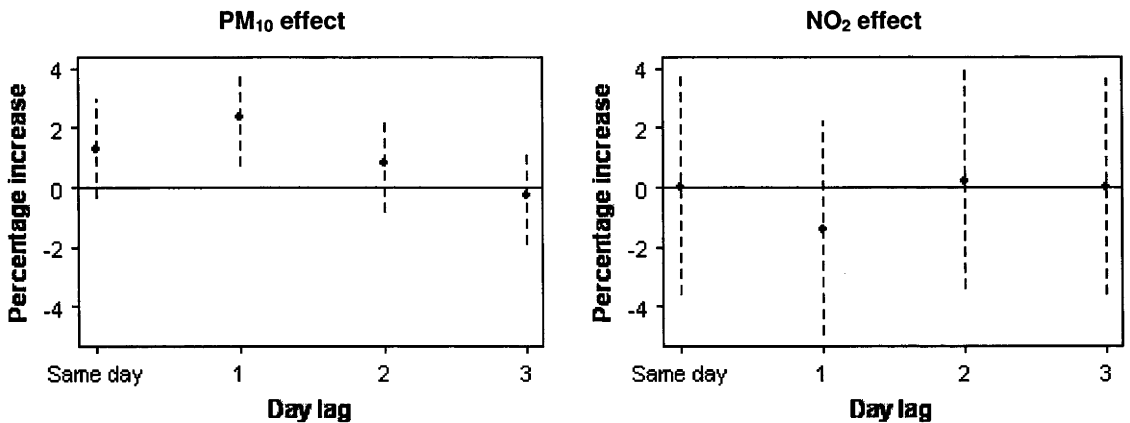
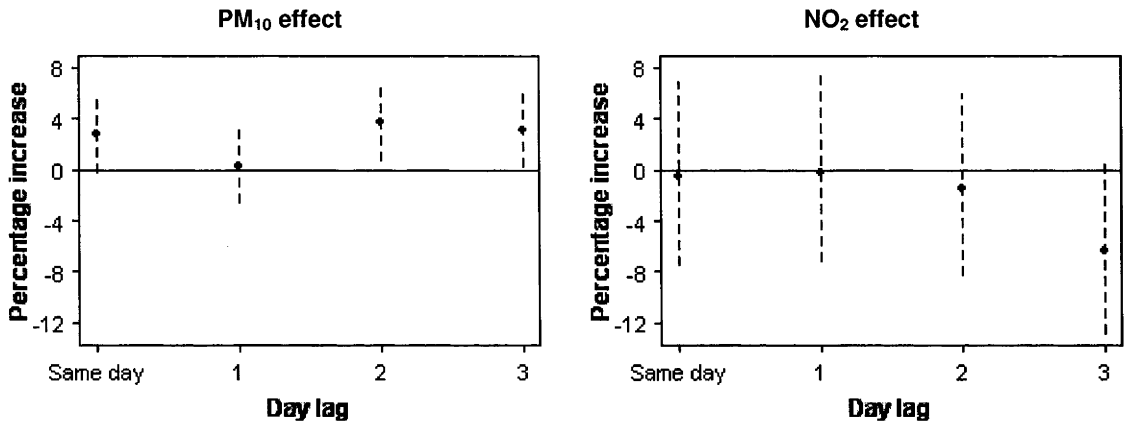


Figure 6-20 (cont.). Percentage increase in daily deaths and 95% CI associated with one interquartile range increase in daily PM₁₀ concentrations (11.7 µg/m³) and daily NO₂ concentrations (15.8 µg/m³) for various mortality categories from multi-pollutant models

(D) Respiratory mortality; 65+ years



Effect modification by season

Table 6-9 summarises the results of seasonal analysis of the association between PM₁₀ and daily mortality for up to two day lags and Figure 6-21 gives graphical representations of the associations by season. The percentage increases in daily mortality and their 95% confidence interval associated with one interquartile range (11.7 µg/m³) increase in daily PM₁₀ concentrations are reported by season for various mortality categories together with the p-values for the significance test of the interaction between the season variable and daily PM₁₀ concentrations. Significant p-values indicate seasonal differences in the association between PM₁₀ and daily mortality.

No statistically significant seasonal difference was observed in the association between PM₁₀ and same day non-external mortality both in whole population and in the elderly population. In the elderly population, the effects of PM₁₀ on non-external mortality at both lag 1 and lag 2 in the cool season (May - August) were significantly lower than the effects in the warm season (September - April). For non-external mortality of all ages, there was weak evidence (p-value < 0.1) of seasonal differences in the associations at lag 1 and lag 2. The statistically significant positive associations between PM₁₀ and daily non-external mortality were consistent across both seasons and all analysed lags. The relative risks of non-external mortality associated with PM₁₀ were higher in the warm season than in the cool season. Although the relative risks were higher in the warm season, the total number of non-external deaths attributed to PM₁₀ would be much higher in the cool season due to the higher daily PM₁₀ concentrations in the cool season.

The analysis for circulatory mortality of those aged 65+ years did not show statistically significant seasonal differences in the associations between PM₁₀ and daily circulatory mortality. The results were consistent across different lags.

For respiratory mortality in the 65+ years age group, there was a strong seasonal difference in the acute effects of PM₁₀ on the same day mortality. One interquartile range increase in PM₁₀ concentration was associated with a 14.5% (95% CI: 4.16, 25.9) increase in same day respiratory mortality of people aged 65+ years in the warm season compared to a 1.81% (95% CI: -1.17, 4.89) increase in the cool season (Table 6-9). For other lag effects of PM₁₀, there were weak evidences (p-value < 0.1) of seasonal differences. No statistically significant association between PM₁₀ and daily respiratory mortality of those aged 65+ years was observed in the cool season (Figure 6-21(D)).

Table 6-9. Percentage increase (95% CI) in daily mortality, by season, associated with one interquartile range (11.7 µg/m³) increase in daily PM₁₀ concentrations for various mortality categories

Lag	Season [‡]	Non-external mortality of all ages	65+ years population		
			Non-external mortality	Circulatory mortality	Respiratory mortality
Same day	Warm	3.95* (0.73, 7.27)	4.47* (0.92, 8.14)	1.25 (-3.63, 6.37)	14.5** (4.16, 25.9)
	Cool	1.33* (0.27, 2.40)	1.59** (0.43, 2.76)	1.31 (-0.31, 2.96)	1.81 (-1.17, 4.89)
	p-value [§]	0.126	0.127	0.981	0.019
1	Warm	4.12* (0.94, 7.40)	4.89** (1.38, 8.51)	4.84 (-0.07, 9.98)	9.05 (-0.87, 19.9)
	Cool	1.03* (0.001, 2.07)	1.16* (0.03, 2.29)	1.93* (0.34, 3.54)	-0.38 (-3.26, 2.58)
	p-value [§]	0.068	0.045	0.269	0.073
2	Warm	4.37** (1.18, 7.66)	5.54** (2.02, 9.19)	2.39 (-2.46, 7.47)	12.6* (2.44, 23.9)
	Cool	1.10* (0.06, 2.16)	1.42* (0.28, 2.57)	0.70 (-0.91, 2.33)	2.81 (-0.09, 5.80)
	p-value [§]	0.053	0.027	0.517	0.067

‡ Warm season (Non-winter months): September to the following April;

Cool season (Winter months): May - August

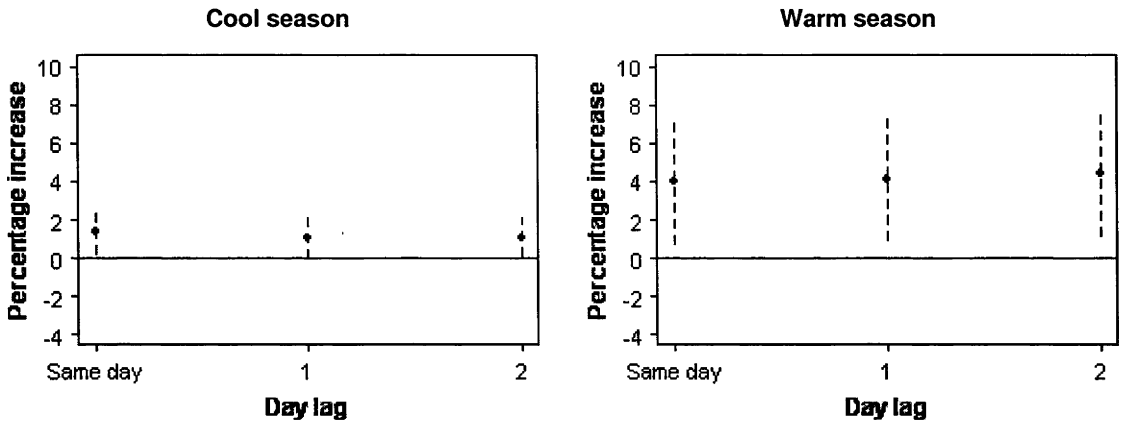
* p < 0.05; ** p < 0.01; *** p < 0.001

Lag 1 and lag 2 represent the pollutant levels on the previous day and the 2 days before respectively.

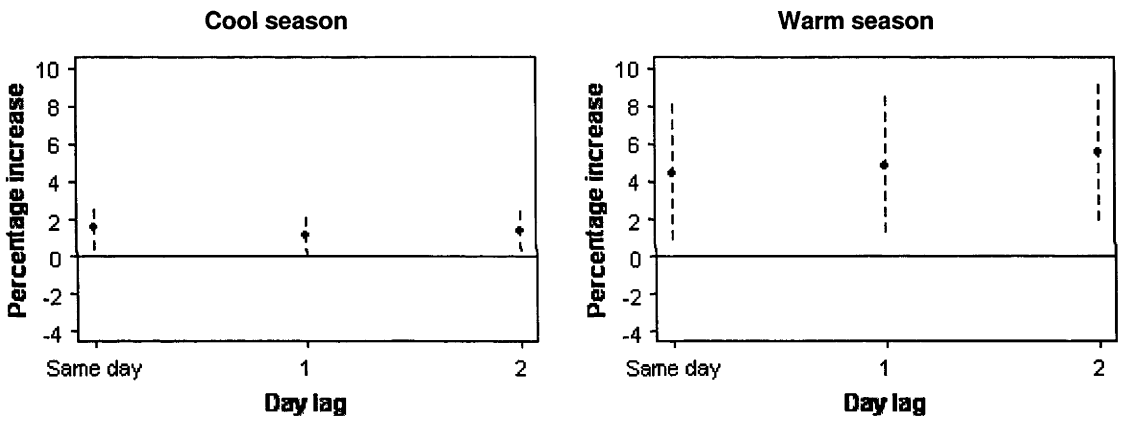
§ P-value for the significance test of the interaction between season and daily PM₁₀ concentration

Figure 6-21. Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories by season

(A) Non-external mortality; all ages



(B) Non-external mortality; 65+ years



(C) Circulatory mortality; 65+ years

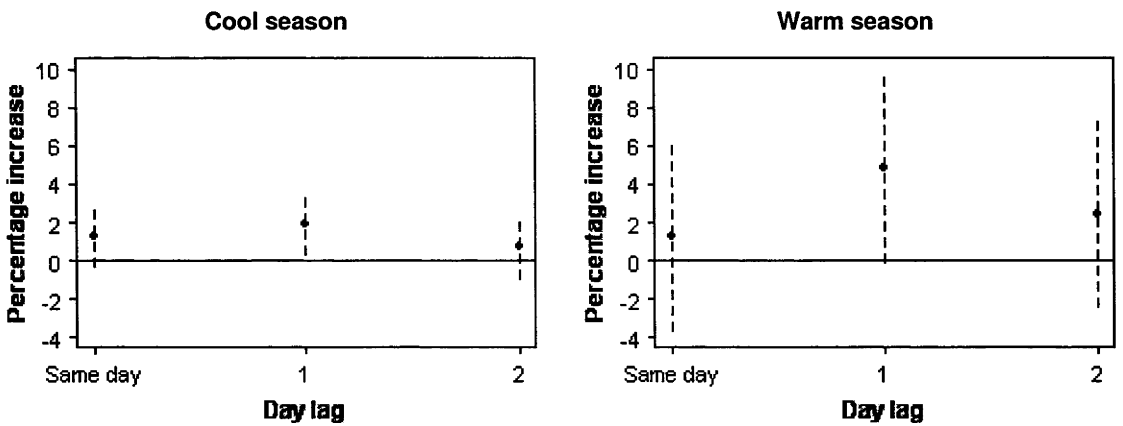
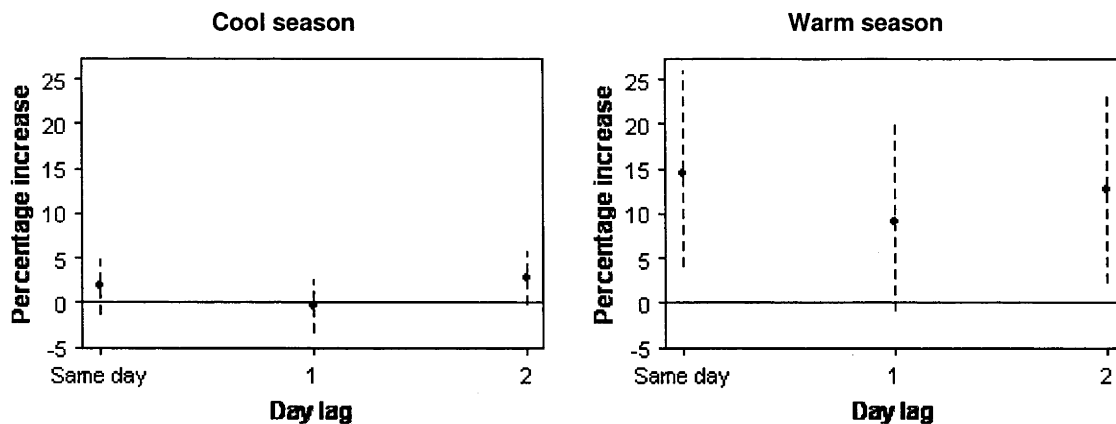


Figure 6-21 (cont.). Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories by season

(D) Respiratory mortality; 65+ years



6.4.3 Sensitivity analysis

The original model (presented in Section 6.4.2) used a natural cubic spline smooth function of time with 7 degrees of freedom per year of data to adjust for a long-term trend and seasonal variation in daily mortality. The analysis was repeated using 3 to 10 degrees of freedom per year of data for the natural cubic spline smooth function of time. Figure 6-22 shows the sensitivity analysis for non-external mortality in the 65+ years age group. Although the use of 3 and 4 degrees of freedom per year of data gave slightly lower estimates of PM_{10} effect on daily mortality, the effect estimates did not greatly depend upon the number of degrees of freedom for the smooth function of time (Figure 6-22 (A)).

In order to examine the sensitivity of the estimates of PM_{10} effect on daily mortality to the adjustment for the temperature effect, the analysis was repeated using 3 to 8 degrees of freedom for the natural cubic spline smooth functions of temperature variables (the same day's temperature and the average of three previous days' temperature). The original analysis used 6 degrees of freedom. The effect estimate was slightly smaller when 2 degrees of freedom was used. The effect estimates and their 95% confidence intervals were almost equal and did not depend upon the degrees of freedom for the smooth functions of temperature variables (Figure 6-22 (B)).

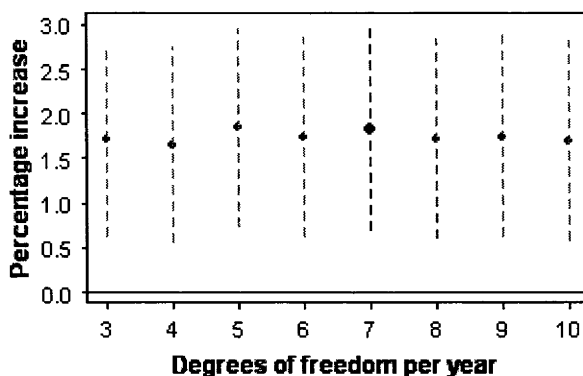
The original model used 3 degrees of freedom for the natural cubic spline smooth functions of relative humidity variables (the same day's relative humidity and the average of three previous days' relative humidity). The analysis was repeated using 1 to 5 degrees of freedom. The estimates of PM_{10} effect on daily mortality did not

depend upon the choice of the number of degrees of freedom for the natural cubic spline smooth functions of relative humidity variables (Figure 6-22 (C))

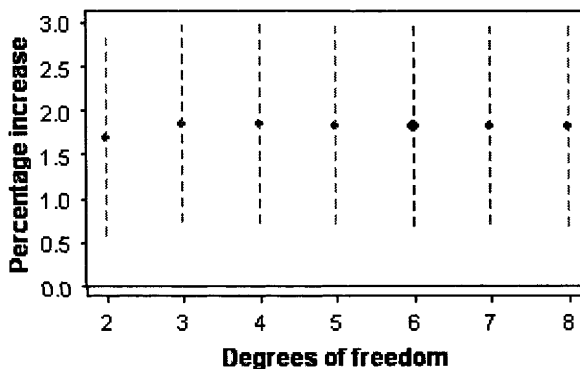
Sensitivity analyses for other mortality groups are presented in Appendix C. The estimates of effect of PM_{10} on acute mortality did not depend upon the degrees of freedom for the natural cubic spline smooth functions of weather variables. Small variations in the estimates of PM_{10} effect were observed when the degrees of freedom for the smooth function of time were varied. However, the variations were not large enough to conclude that the effect estimates largely depended upon the number of degrees of freedom for the smooth function of time.

Figure 6-22. Sensitivity of the effects of PM₁₀ on daily non-external mortality of 65+ years age group to the degrees of freedom set for the smooth functions of confounders. Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 µg/m³) increase in daily PM₁₀ concentrations

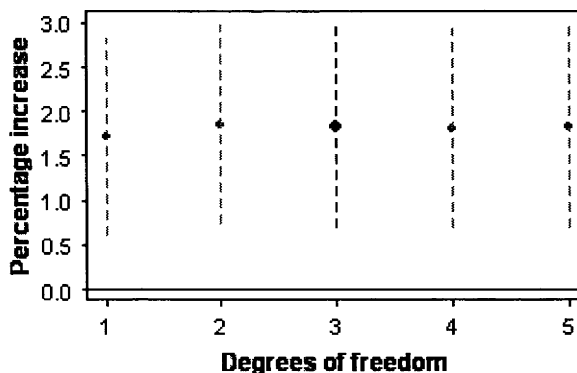
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature



(C) Degrees of freedom for the smooth function of relative humidity



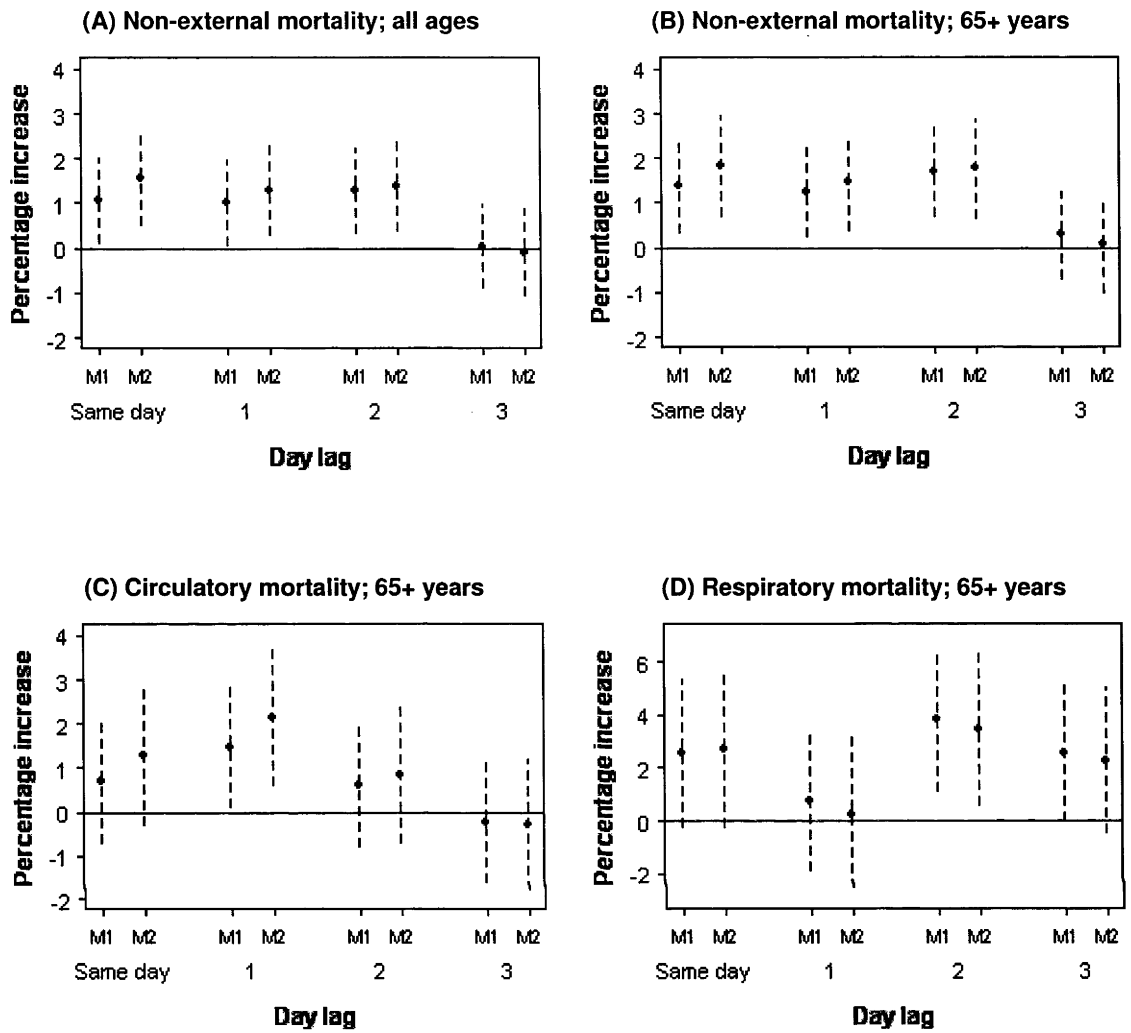
6.5 Comparison of results from the two methods

Figure 6-23 shows the comparison of the estimated effects of PM₁₀ from the two methods for various mortality categories. Both methods reported similar findings about

the statistical associations of daily mortality and PM₁₀. It was consistent for all mortality categories and across different lags analysed (up to lag three). Method 2 estimated slightly higher relative risks of mortality associated with PM₁₀ for non-external mortality of all ages, non-external mortality in the 65+ years age group and circulatory mortality in the 65+ years age group. For respiratory mortality in the 65+ years age group, the estimated relative risk was slightly smaller in Method 2. However, the differences were very small.

The associations of PM₁₀ with daily mortality by season from the two methods are compared in Figure 6-24 for various mortality categories and for different lags. There were very small differences in the estimates of PM₁₀ effects from the two methods. The main findings from both methods were, in general, consistent. It appeared that differences in the methods to control for a long-term trend, seasonal variation, temperature and relative humidity may result in a small difference in the relative risks of mortality associated with PM₁₀. However, this does not change the main findings of the statistically significant associations of PM₁₀ with daily mortality as long as they are adequately adjusted for.

Figure 6-23. Comparison of the results from the two methods: Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories

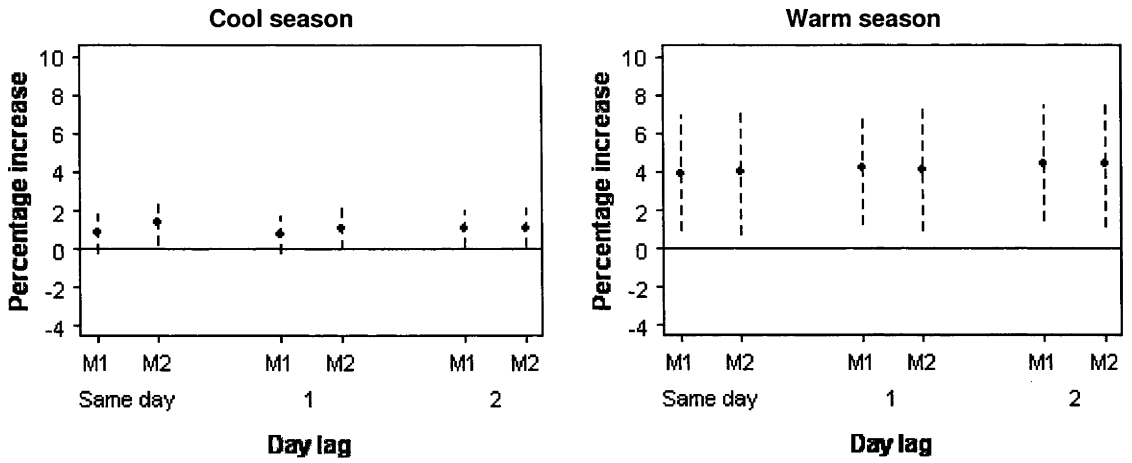


Note: Y-axis scale is different

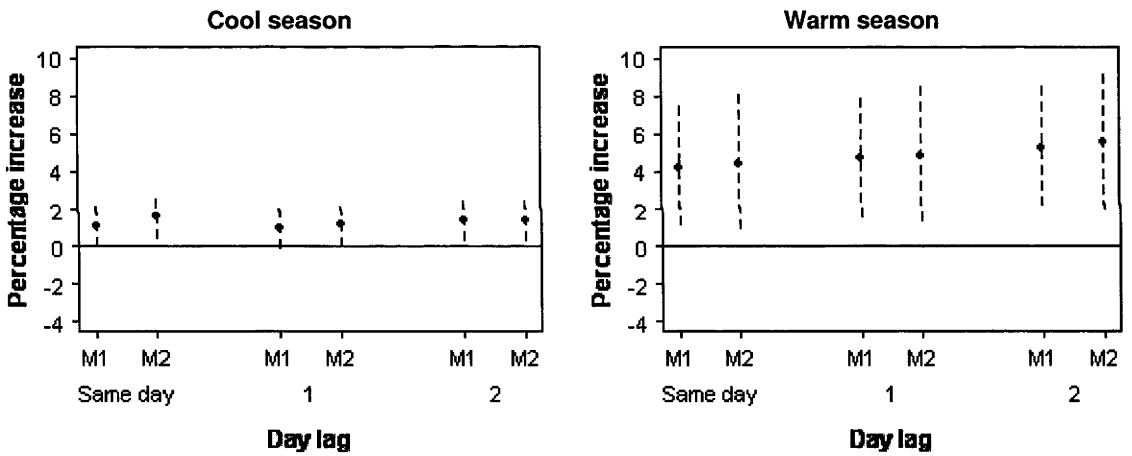
M1 : Method 1
M2 : Method 2

Figure 6-24. Comparison of the results from the two methods: Percentage increase in daily deaths and 95% CI associated with one interquartile range ($11.7 \mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories by season

(A) Non-external mortality; all ages



(B) Non-external mortality; 65+ years



(B) Circulatory mortality; 65+ years

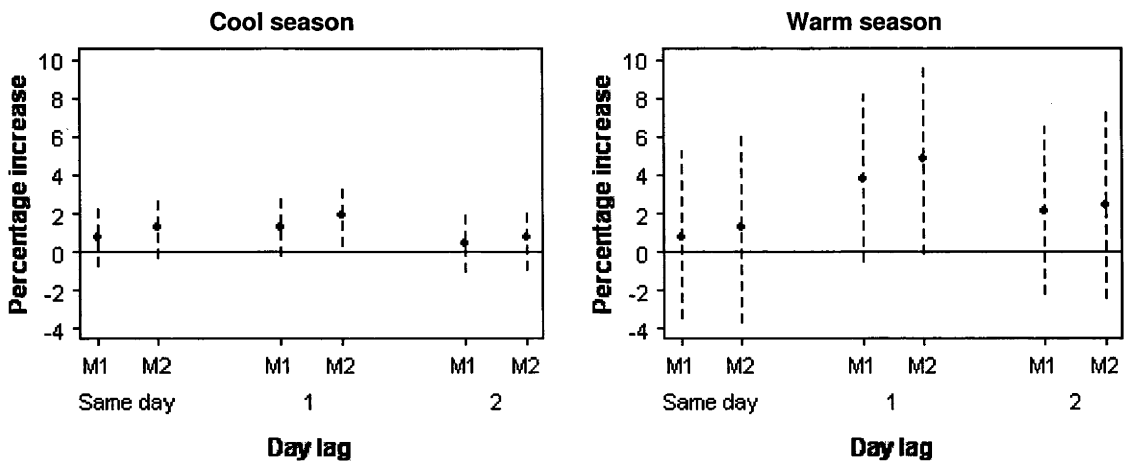
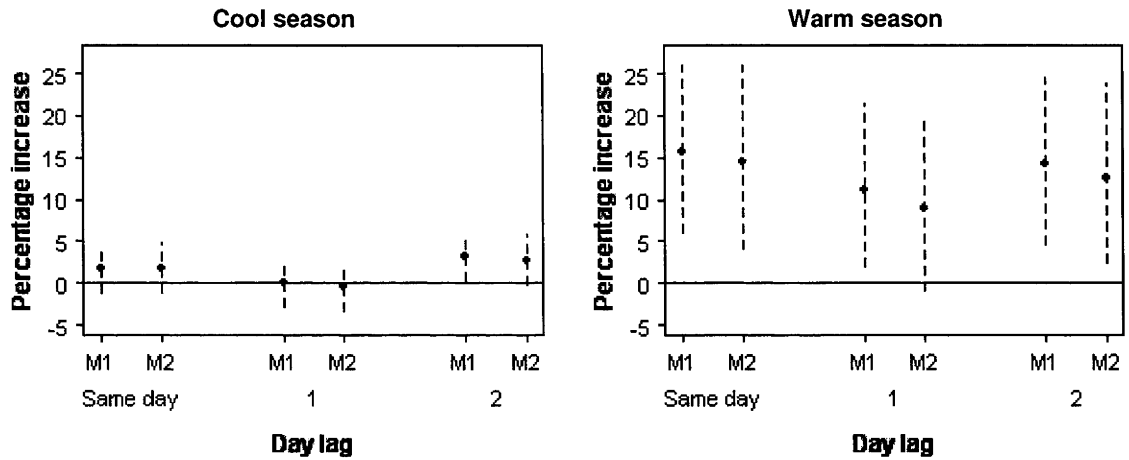


Figure 6-24 (cont.). Comparison of the results from the two methods: Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM_{10} concentrations for various mortality categories by season

(D) Respiratory mortality; 65+ years



6.6 Summary

This chapter quantifies the association between daily mortality and air pollution concentrations using Poisson regression models, adjusting for long-term trend and seasonal variations in mortality, and the confounding effects of weather variables. The possible differences in the association between daily mortality and PM_{10} in different seasons were analysed. Two different methods were used to control for confounders, and the results were compared.

Increase in daily mortality was significantly associated with an increase in daily PM_{10} and CO but not with NO_2 . While PM_{10} was associated with an increase in daily mortality for all mortality categories analysed, CO was associated with non-external mortality of all ages, non-external mortality of people aged 65+ years, and circulatory mortality of people aged 65+ years. No evidence of the association of CO with respiratory mortality of people aged 65+ years was found. The seasonal analysis of the association between PM_{10} and daily mortality shows that the relative risks were larger in the warmer season than in the cooler season. The results from two different methods used in this analysis were consistent suggesting that the associations between daily mortality and air pollution observed in this study is less likely due to the choice of statistical modelling technique and inadequate control of confounders.

Chapter 7: Mortality displacement

7.1 Introduction

Time series analysis of the association between daily mortality and PM_{10} in Chapter 6 provided strong evidence of a significantly increased risk of daily mortality associated with PM_{10} after controlling for potential confounders. The analysis estimated an acute effect of PM_{10} on mortality. However, it was not clear from the analysis whether exposure to high PM_{10} concentrations increased the number of deaths in the general population or only brought forward the time of death of individuals in the frail subpopulation, who would have died in a few days irrespective of PM_{10} concentrations, a phenomenon usually termed “harvesting” of deaths or “mortality displacement”. This is an important question for the public health task of estimating the impact of PM_{10} on mortality. The public health concern of the association of PM_{10} with mortality would be much less if the majority of the deaths associated with PM_{10} were from the pool of frail individuals, who would have died in a few days, compared to PM_{10} increasing the number of deaths in the general population and thus reducing the life span by months or years (McMichael *et al.*, 1998).

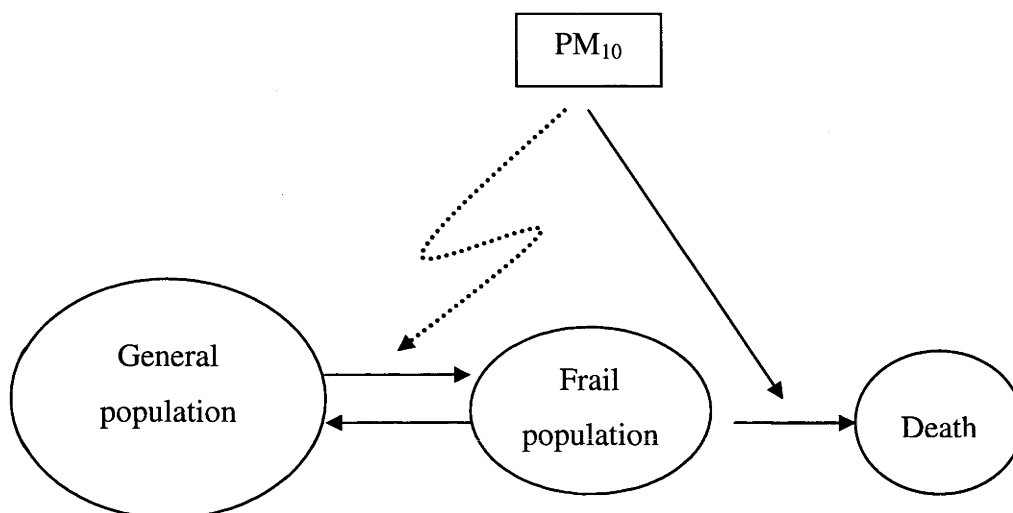
This chapter investigates whether the association between daily mortality and PM_{10} can be entirely attributed to “harvesting”. This was done by analysing the associations at different mid-term time scales. If the association was completely due to “harvesting”, then the association would be observed at short time scales but not at mid to long time scales.

7.2 “Harvesting” hypothesis

Under “harvesting” or mortality displacement hypothesis, PM_{10} only hastens the deaths of individuals in the frail population and is only associated with an increase in deaths of frail individuals. A three state population model, i.e. a model describing the movement of individuals from “healthy” (general population) to “frail” (pool of frail individuals) to death, has been used to analyse the mortality displacement hypothesis (Zeger, Dominici and Samet, 1999; Murray and Nelson, 2000; Zanobetti *et al.*, 2000; Schwartz, 2001; Dominici *et al.*, 2003c; Roberts and Switzer, 2004). Figure 7-1 shows a schematic diagram of this model. Because of various risk factors, individuals in the “general population” become frail and move into the “frail population”. Exposure to PM_{10} may be one of the risk factors that cause the movement of individuals from the “general

population” to the “frail population”. Individuals in the “frail population” either may die (move into “death”) or recover and move back to the “general population”. Individuals in the “frail population” are the ones who may die in near future irrespective of PM_{10} concentrations. The movement of individuals from the “frail population” to “death” may be associated with PM_{10} concentrations.

Figure 7-1. A three state population model for the analysis of harvesting hypothesis



Under the mortality displacement hypothesis, PM_{10} only affects the individuals in the “frail population”, but not the individuals in the “general population”. In other words, PM_{10} is only associated with an increase in deaths of individuals in the “frail population”. It is not associated with the movement of individuals from the “general population” to the “frail population” or the deaths of individuals in the “general population”. This is, of course, only a hypothesis and in reality, people in the “general population” may be affected by PM_{10} and become frail.

The hypothesis assumes that there would be no increase in the number of individuals moving from the “general population” to the “frail population” due to higher PM_{10} concentrations to counter balance the short-term increase in mortality rate in the “frail population” due to higher PM_{10} concentrations. As a result, the size of the “frail population” will deplete to a level below its long-term average. Because of the smaller size of the “frail population”, the number of deaths on subsequent days will be lower than expected (i.e. a decrease in the number of individuals moving from the “frail population” into “death”). As there is no change in the movement of individuals from the “general population” to the “frail population”, the size of the “frail population” would replenish eventually and the number of deaths would return to its long-term average. Until the size of the “frail population” returns to the long-term average, decrease in the number of deaths would continue.

If only individuals in the “frail population” are affected by PM_{10} , then an increase in daily mortality due to PM_{10} will be immediately followed by a drop in daily mortality. The increase in daily mortality will be balanced out by the subsequent drop in daily mortality, if the increase is completely due to short-term displacement of the time of deaths by a few days. There will be no net increase in daily deaths associated with PM_{10} if the daily number of deaths is averaged over a few days. In other words, mortality will no longer be associated with PM_{10} if a moving average of daily data is analysed. If some of the deaths are brought forward by a longer period (“no harvesting”), the increase in daily mortality will be partially cancelled out by the subsequent drop in daily mortality. This will result in a reduced effect of PM_{10} if moving average of daily data is analysed.

Calculating moving average of daily data is similar to smoothing daily time series data. A moving average or a smoothed data series fluctuates less than the original daily data series. The frequency and amplitude of fluctuation reduce with an increase in the number of days over which the moving averages are calculated. The data series that represents daily variations in the original data series due to a short time scale pattern (i.e. shortest-term variations in the original data) fluctuates with high frequency. The data series that represents daily variations in the original data series due to mid-term and longer time scale pattern (i.e. mid-term variations and long-term variations in the original data) fluctuate with mid-scale and low frequency respectively. Because of similar long-term trends and seasonal variations in daily mortality and PM_{10} data, it is more likely that the association between mortality and PM_{10} will be observed at low frequency.

If the association between daily mortality and PM_{10} is completely due to short-term mortality displacement, the association will likely to be observed only at high frequency fluctuations (i.e. in the time series representing the shortest-term variations, those with periods of only a few days), but not at mid-scale frequencies (i.e. in the time series representing the mid-term variations). An increase in daily mortality due to PM_{10} and the drop in daily mortality which follows the increase would have been smoothed over at mid-scale frequencies and thus PM_{10} and mortality will no longer be associated at these frequencies. Conversely, the existence of a positive association between PM_{10} and mortality at mid-scale frequencies would show that the association of daily mortality with PM_{10} is not completely due to mortality displacement and PM_{10} associated deaths are brought forward by a longer period. A significantly positive but reduced effect size of PM_{10} on mortality at mid-scale frequencies indicates that some

deaths, not all, are due to harvesting. This suggests that some deaths are brought forward by a longer period, and the association between daily mortality and PM_{10} cannot be entirely attributed to harvesting.

The “harvesting” hypothesis was tested using the method proposed by Schwartz (2000c) which examines the effects of PM_{10} on daily mortality at different time scales (Schwartz, 2000c).

Schwartz (2000c) suggested decomposing each time series, which includes daily mortality, air pollution and weather data, into three independent parts representing daily variations in the original series that are due to patterns with different time scales. For example, daily PM_{10} data could be decomposed into three independent time series: PM_{10long} , PM_{10mid} , and $PM_{10short}$ which represent the daily variations in the original PM_{10} series that fluctuate with low frequency, mid-scale frequency and high frequency respectively. PM_{10long} , which fluctuates with low frequency, represents temporal trends including a long-term trend and seasonal variations. $PM_{10short}$, which oscillates with high frequency, represents the short-term variations, which is susceptible to short-term mortality displacement. The mid-scale component (PM_{10mid}), which fluctuates with mid-scale frequency, is free from both the long-term trends (including seasonal variation) and the short-term variation. The presence of “harvesting” in the association between PM_{10} and mortality can be examined by analysing the associations in these mid-scale components of the original data series adjusting for the mid-scale components of daily weather data. The existence of a positive association between PM_{10} and mortality in these mid-scale components would show that not all of PM_{10} associated deaths are being advanced by only a few days (Schwartz, 2000c; 2001). Some are brought forward by a longer period.

7.3 Methods

Following the method proposed by Schwartz (2000c), the LOESS smooths (Cleveland, 1979) with different smoothing window sizes were used to decompose a time series data into a number of independent time series representing daily variations in that series, which vary with the period of different time scales such as long-term variation, mid-term variation and short-term variation (Schwartz, 2000c). The LOESS is a non-parametric smoother. The amount of smoothing depends upon the size of the smoothing window, with increased smoothness as the size of the smoothing window increases.

The LOESS smooth with a large smoothing window gives smoothed data that fluctuate with a low frequency. This smoothed series represents the fluctuation in the

original series, which is due to a long-term time trend and seasonal variation. The width of smoothing window was set at 120 days for a long-term trend and seasonal variation in time series data. The residuals from this filtering will have no seasonality and only include the mid-term and short-term time scale components of data series.

Let Y_t be the original time series data and $lo(Y_t, 120)$ be the 120-day LOESS smooth of the series Y_t . The smoothed data series $lo(Y_t, 120)$ is a long-term trend and seasonal variation in daily time series Y_t . The residual series

$$Z_t = Y_t - lo(Y_t, 120)$$

will be free from the long-term trend and seasonal variation of the original series.

The second LOESS filter with a 30-day smoothing window was then applied to the residuals from the first filter, i.e. Z_t .

Let $lo(Z_t, 30)$ be the 30-day LOESS smooth of the series Z_t . This filtered series $lo(Z_t, 30)$ is the mid-term time scale component of the original time series. This second filtered series $lo(Z_t, 30)$ will be free from the longer-term fluctuations and seasonal variations as well as from the short-term fluctuations, which are sensitive to short-term displacement of time of deaths.

The residuals of the second filter, i.e. $Z_t - lo(Z_t, 30)$, represent the fluctuations at short-term time scale of less than 30 days.

The above process was applied to daily mortality, PM_{10} , temperature and relative humidity to generate daily fluctuations in each of the time series data, which are due to mid-scale variations. The second filtered series $lo(Z_t, 30)$ of daily mortality and PM_{10} concentrations were associated to investigate the association at mid-term time scale.

In order to analyse the associations over different mid-term time scales, several mid-scale variations of each of the time series were generated by repeating the above process with different mid-term smoothing windows for the second LOESS filter. In addition to a 30-day smoothing window, mid-term smoothing windows of 15, 45 and 60 days were used for the second LOESS filter.

Analysis was carried out using only the mid-term time scale components of all time series. Regression analysis was undertaken to study the association between mid-term time scale components of daily mortality and PM_{10} concentrations adjusting for mid-term time scale components of weather variables for the mid-scale components on each of the four filter lengths. The mid-term components of the same time scale were used for all variables in the models. The same model as described in daily time series analysis in Section 6.4.2 (p.113) was used in this analysis. Daily time series variables in

the model were replaced by the respective mid-term time scale components. A log-linear regression model was fitted to the mid-scale component of daily mortality to maintain the basis of multiplicative effect of covariates as in the time series analysis of the effect of short-term exposure to PM_{10} on mortality, discussed in Chapter 6. The mean of daily mortality was added back as a constant to the mid-scale component to retain the scaling of original mortality series. The confounding effects of weather variables were controlled for by creating natural cubic spline smooth functions of the same weather variables as in the original model in Section 6.4.2 (p.113). Same number of degrees of freedom was used for the natural cubic spline smooth functions. The models included

- a set of dummy variables for days of week to control for daily variation in mortality across the week;
- a natural cubic spline smooth function of the mid-scale component of the same day's average hourly temperature with 6 df and a natural cubic spline smooth function of the mid-scale component of moving average of the preceding three days' (lag 1 to lag 3) average hourly temperature with 6 df to control for the confounding effect of temperature;
- a natural cubic spline smooth function of the mid-scale component of the same day's average hourly relative humidity with 3 df and a natural cubic spline smooth function of the mid-scale component of moving average of the preceding three days' (lag 1 to lag 3) average hourly relative humidity with 3 df to control for the confounding effect of relative humidity; and
- mid-scale component of daily PM_{10}

7.4 Results

Figures 7-2 and 7-3 illustrate the decomposition of daily time series data of non-external deaths in the population aged 65+ years and daily PM_{10} concentrations into long-term components (top panel) and mid-scale components (central panel). Decompositions for other mortality categories are illustrated in Appendix D. The bottom panels of both figures show daily data of the respective data series. The long-term components (top panels) are the LOESS smooths with a window size of 120 days applied to daily data. They appear to capture a long-term trend and seasonal variations in daily data. The residuals after removing the long-trend and seasonal pattern in daily non-external deaths in the 65+ years age group are shown in Figure 7-4. The residual plot confirms that the

long-term variations were reasonably controlled for by applying the LOESS filter with a 120-day smoothing window.

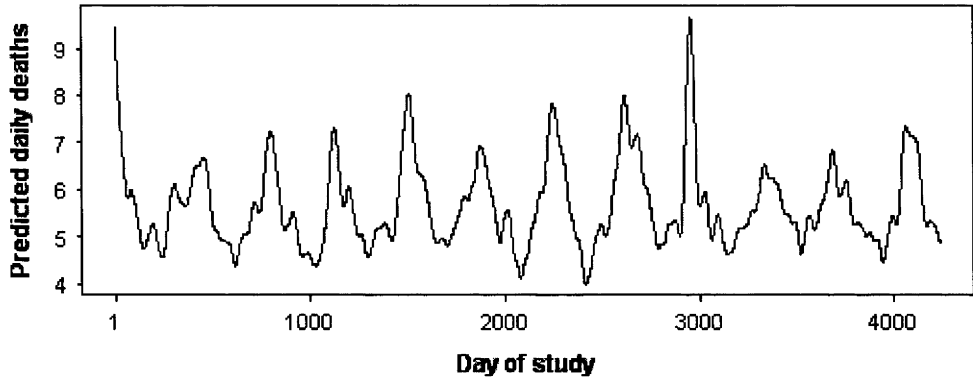
The central panel in Figure 7-2 shows the mid-scale component of daily non-external deaths in the population aged 65+ years, generated by applying the LOESS filter with a 30-day smoothing window to the residuals in Figure 7-4. This series was free from long-term variations including seasonal variations as well as from short-term variations, which were sensitive to short-term mortality displacement. The mean of this series was zero but the original mean of daily non-external deaths of people aged 65+ years was added back to retain the scaling of the original mortality series before fitting the log-linear regression models. Figure 7-5 shows the residuals of the second LOESS filter with a 30-day smoothing window applied to the residual of the first LOESS filter with a 120-day smoothing window (Figure 7-4). This residual series represents the short-term variations in daily time series data.

Figure 7-3(B) shows the mid-scale component of daily PM_{10} data series, generated by applying the LOESS filter with a 30-day smoothing window to the residuals of the LOESS filter with a 120-day smoothing window applied to the original daily PM_{10} data. Daily time series data of other confounders were similarly decomposed and the series representing mid-scale components of each data series were generated using the LOESS filter with smoothing windows of same sizes as in the case of daily mortality and PM_{10} data (plots not shown). The mid-scale component of daily non-external mortality in the 65+ years age group (Figure 7-2(B)) was associated with the mid-scale component of daily PM_{10} concentrations (Figure 7-3(B)) adjusting for the mid-scale components of other confounder data series.

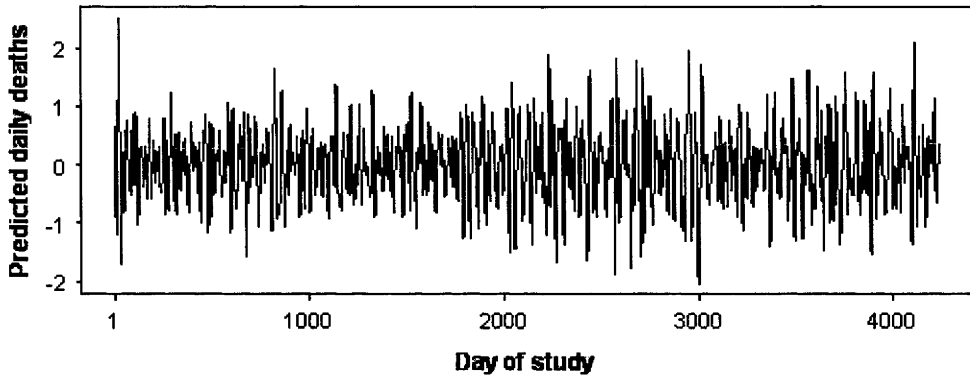
Mid-scale components with mid-term smoothing window sizes of 15, 30, 45 and 60 days were generated in order to analyse the associations over different mid-term time scales. For illustration, the plots of mid-scale components generated with a mid-term smoothing window size of 30 days are shown here in this chapter.

Figure 7-2. Decomposition of daily non-external deaths in 65+ years age group

(A) Long-term trend and seasonal variation with a 120-day smoothing window



(B) Mid-scale component with a 30-day smoothing window



(C) Daily number of deaths

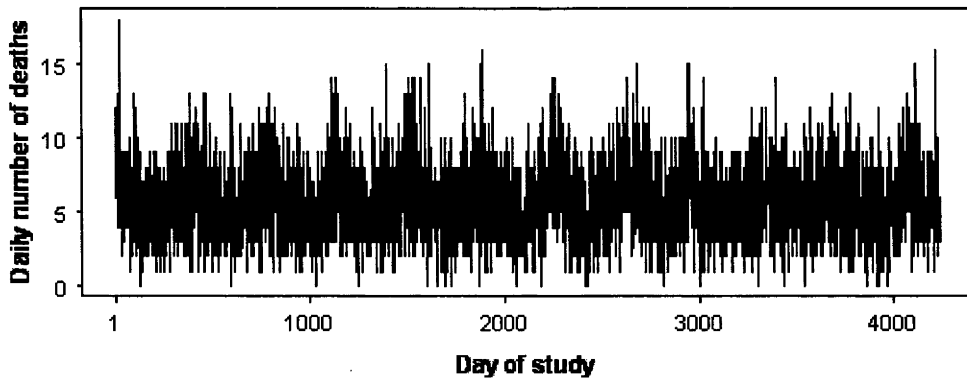


Figure 7-3. Decomposition of daily PM₁₀ concentrations

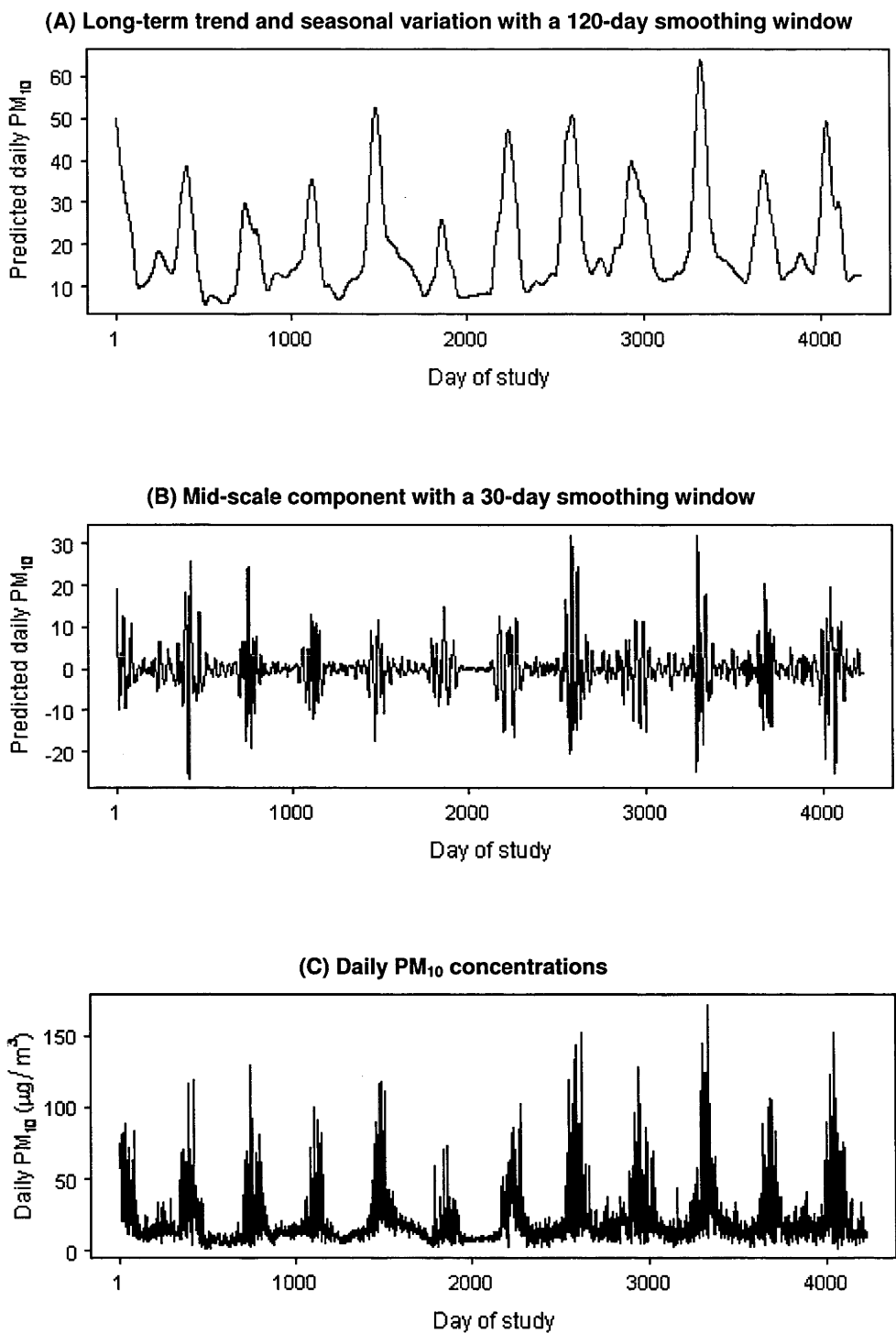


Figure 7-4. Residuals after removing long-term variations in daily non-external deaths in 65+ years age group using the LOESS filter with a 120-day smoothing window

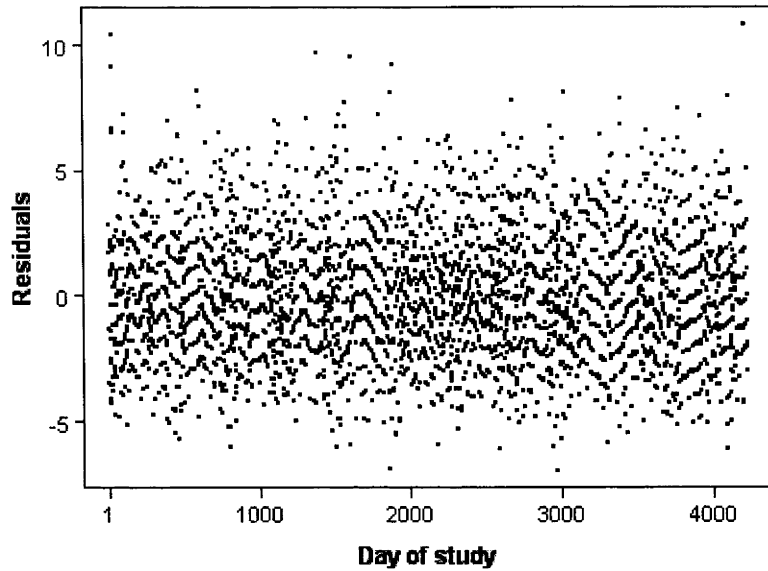
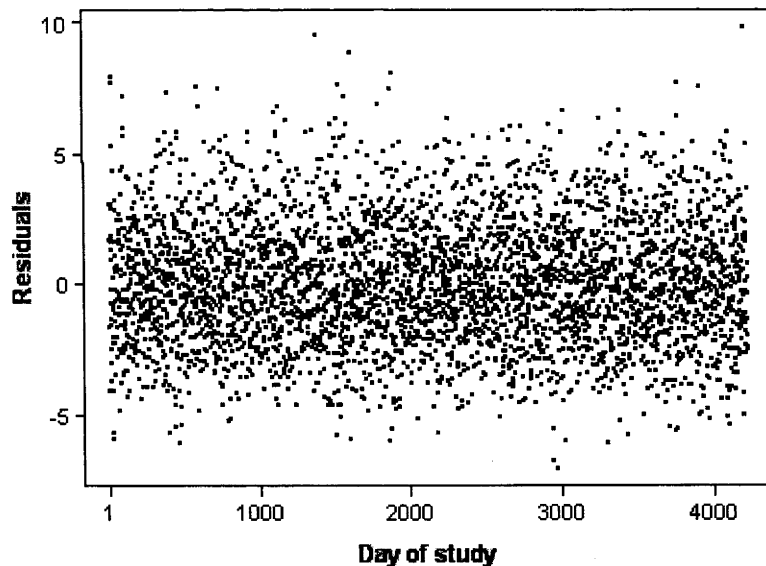


Figure 7-5. Residuals after removing both long-term variations and mid-scale variations in daily non-external deaths in 65+ years age group using the LOESS filter with a 120-day smoothing window and a 30-day smoothing window

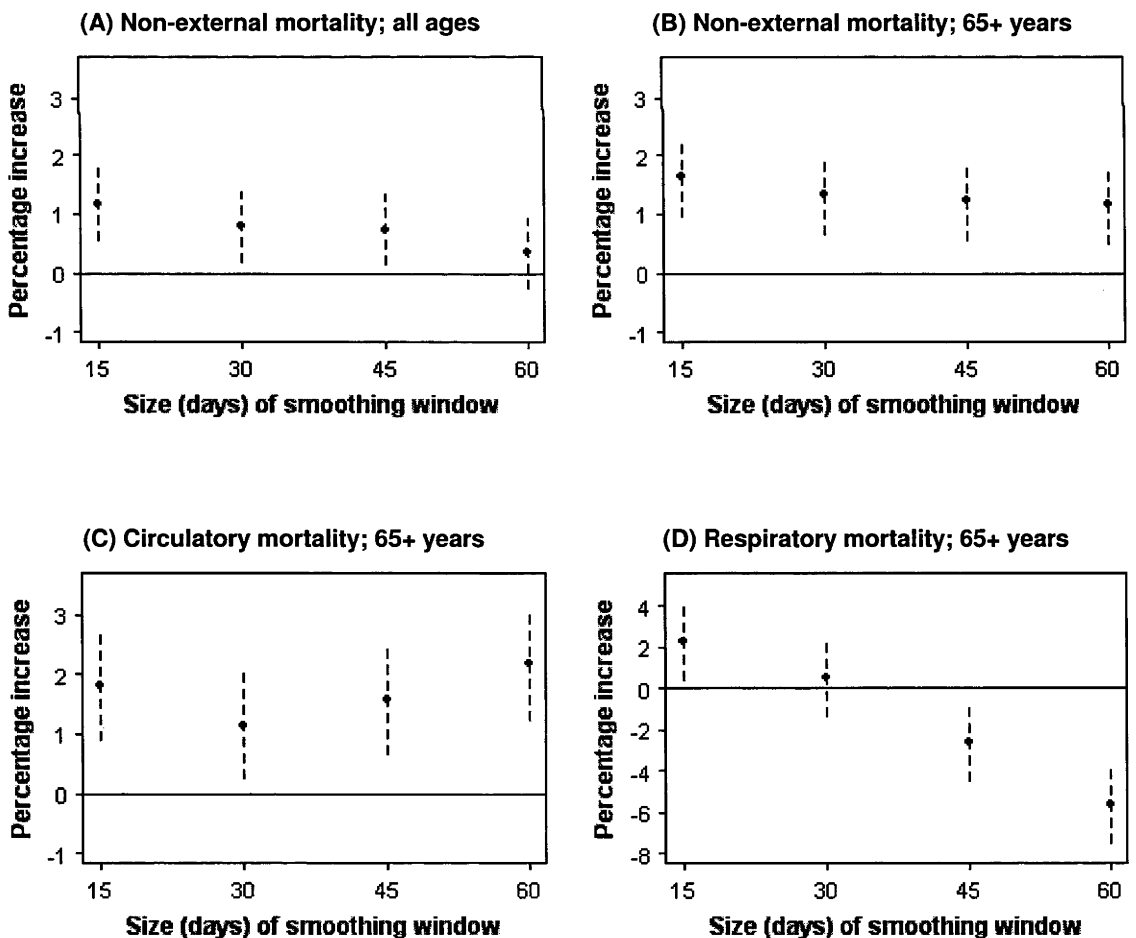


The associations between PM_{10} and mortality at four different mid-term time scales were compared by examining the effect size estimates of PM_{10} on mortality at different mid-term time scales. The effect size estimates were expressed as percentage increases in mid-term components of daily mortality associated with a $10 \mu\text{g}/\text{m}^3$ increase in mid-term components of daily PM_{10} . Figure 7-6 shows the effect size estimates of PM_{10} on daily mortality at four different mid-term time scales for various mortality categories. The corresponding sizes of the mid-scale smoothing windows used

in the analysis of the association are shown in the x-axes. The effect size estimates at four different mid-term time scales are compared with the short-term effect size estimates of PM₁₀ on daily mortality from Chapter 6 in Table 7-1. The estimated percentage increases in daily mortality in Chapter 6 were reported for one interquartile range increase in daily PM₁₀. For the comparison here, the percentage increases in daily mortality were recalculated for a 10 µg/m³ increase in daily PM₁₀ concentrations.

The results presented in Figure 7-6 and Table 7-1 are based on the analysis of the mid-term components of daily mortality and PM₁₀ data series generated using non-parametric smooths. Therefore, the interpretation of the percentage increase in mortality for increase in PM₁₀ is not as clear as the ones reported in Chapter 6, which were based on the analysis of the observed daily number of deaths and daily PM₁₀ concentrations. However, to examine whether or not the association between PM₁₀ and mortality can be entirely attributed to harvesting, we only need to test if there is an association between PM₁₀ and mortality at mid-term time scales.

Figure 7-6. Effect size estimates with 95% CI for various mortality categories associated with a 10 µg/m³ increase in PM₁₀ at various mid-scale components with different smoothing window sizes



Note: Y-axis scale is different

Table 7-1. Percentage increase (95% CI) in mortality associated with a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} at various mid-scale components with different smoothing window sizes for various mortality categories

Smoothing window size for mid-term component	Non-external mortality of all ages	65+ years population		
		Non-external mortality	Circulatory mortality	Respiratory mortality
0 day [#]	1.31 (0.44, 2.18)	1.55 (0.60, 2.50)	1.84 (0.54, 3.15)	2.95 (0.55, 5.40)
15 days	1.16 (0.55, 1.78)	1.64 (0.97, 2.32)	1.79 (0.89, 2.72)	2.25 (0.40, 4.13)
30 days	0.78 (0.18, 1.38)	1.33 (0.67, 1.99)	1.13 (0.24, 2.03)	0.48 (-1.38, 2.36)
45 days	0.73 (0.15, 1.32)	1.22 (0.56, 1.88)	1.55 (0.64, 2.47)	-2.59 (-4.45, -0.69)
60 days	0.34 (-0.24, 0.92)	1.16 (0.49, 1.83)	2.17 (1.24, 3.11)	-5.64 (-7.49, -3.75)

[#]Note: The percentage increases in mortality estimated in the daily time series analysis of mortality and PM_{10} in Chapter 6. The percentage increases were for the PM_{10} lag that had the strongest effect on single day mortality. For non-external mortality, the effects were of the same day's PM_{10} . For circulatory mortality and respiratory mortality, they were the effects of 1-day lagged PM_{10} and 2-day lagged PM_{10} respectively.

For non-external mortality of all ages, a 10 $\mu\text{g}/\text{m}^3$ increase in daily PM_{10} was associated with an increase of 1.31% (95% CI: 0.44, 2.18) in the same day mortality. The estimated PM_{10} effect sizes at mid-term time scales were smaller than the PM_{10} effect at the very short-term time scale of a 1-day period (i.e. the same day effect of PM_{10}). The estimated effect size reduced as the size of the smoothing window for mid-scale components was increased. The associations became statistically non-significant when a 60-day smoothing window was used. This pattern was similar to what we would have expected under the short-term mortality displacement hypothesis. This suggested that the association between PM_{10} and non-external mortality of all ages may be entirely attributed to short-term mortality displacement. However the positive associations for up to 45-day smooth windows suggested that deaths were being advanced by more than a few days, perhaps by a few weeks or months.

For non-external mortality in the 65+ years age group, a 10 $\mu\text{g}/\text{m}^3$ increase in daily PM_{10} was associated with an increase of 1.55% (95% CI: 0.60, 2.50) in the same day mortality. The estimated effect size first slightly increased when a 15-day

smoothing window was used for mid-scale components and then gradually decreased as the size of the smoothing window increased. With smoothing windows larger than 15 days (i.e. 30-day, 45-day and 60-day smoothing window) for mid-scale components, the effect sizes at mid-term time scales were smaller than the effect of same day PM₁₀. Although the estimated effect sizes slightly reduced as the size of the smoothing window for mid-scale components was increased, significant positive associations were observed at all four mid-term time scales. This shows a presence of some harvesting and at the same time shows that some deaths were brought forward by a longer period. This suggests that the association between PM₁₀ and non-external mortality in the population aged 65+ years could not be entirely attributed to short-term mortality displacement.

There was an estimated increase of 1.84% (95% CI: 0.54, 3.15) in circulatory deaths in the population aged 65+ years for each 10 µg/m³ increase in 1-day lagged PM₁₀. Effect sizes at mid-term time scales were slightly lower when a 15-day and a 30-day smooth windows were used, which suggests that there was some harvesting. For the smoothing windows of greater than 30 days, the estimated effect size increased with an increase in the size of the smoothing window. When a 60-day smoothing window was used, the effect size was greater than the effect size at the very short-time scale of a 1-day period (effect of single day PM₁₀ on next day circulatory mortality). There were greater effects at longer time scales, which was not consistent with the harvesting hypothesis.

There was an estimated increase of 2.95% (95% CI: 0.55, 5.40) in respiratory deaths in the population aged 65+ years for each 10 µg/m³ increase in 2-day lagged PM₁₀. The estimated PM₁₀ effect sizes at mid-term time scales were smaller than the effect at the very short-term time scale of a 1-day period and reduced gradually with increase in the size of the smoothing window for mid-term components. There was a positive association when a 15-day smoothing window was used but the associations became negative when 45-day and 60-day smoothing windows were used. Increase in PM₁₀ was associated with decrease in mortality at these time scales. The reason for this statistically significant negative association was unclear. It might be due to a very small number of daily respiratory cause deaths in the population aged 65+ years although we would more likely to get inconclusive results when the numbers are very low.

7.5 Summary

This chapter investigated whether the short-term association between daily mortality and PM₁₀, shown in the time series analysis in Chapter 6, was entirely due to short-

displacement of time of deaths of frail individuals who would have died in a few days irrespective of PM_{10} concentrations, a phenomenon usually termed “harvesting” or “mortality displacement”. A method proposed by Schwartz (2000c) was used to analyse the “harvesting” hypothesis by examining the association of PM_{10} with mortality at different time scales. If the association was completely due to harvesting, the association would be observed only at short time scales but not at mid time scales.

All daily time series data including mortality data, PM_{10} data and weather data were decomposed into three components that represent a long-term variation, a mid-term variation and a short-term variation in the original series. The associations were analysed at mid-term variations only as associations at this time scale would indicate that the association between PM_{10} and daily mortality was not completely due to harvesting. The mid-term component of the mortality series was associated with the mid-term component of PM_{10} series adjusting for the mid-term components of weather variables. Several mid-term components were generated using various mid-term smoothing window sizes and the associations were analysed at different mid-term time scales.

Although the association between PM_{10} and daily non-external mortality of all ages appeared to be due to harvesting, the results suggest that time of death was brought forward by more than a few days, perhaps by a few weeks or months. The analysis for non-external deaths in the 65+ years age group showed that the association was not entirely due to harvesting. Some deaths in this age group were brought forward by a longer period. Similarly, the association between PM_{10} and circulatory mortality in the 65+ years age group was not entirely due to harvesting.

In this analysis, the mid-term components were generated using non-parametric smooth with different smoothing window size. Therefore, the interpretation of the regression coefficients (effect size estimates) from this analysis for an increase in mortality associated with PM_{10} is not as clear as that for daily time series analysis of mortality and PM_{10} . However, to examine whether or not the association between PM_{10} and mortality can be entirely attributed to harvesting, we only need to test if there is an association between PM_{10} and mortality at mid-term time scales and this can be examined using this method. This analysis shows that the association cannot be entirely attributed to harvesting, suggesting that PM_{10} not only affects the deaths of frail individuals but may also increase mortality in the general population over a longer period. This indicates that PM_{10} can have a real impact on long-term mortality.

Chapter 8: Extended effects of PM₁₀ using distributed lag models

8.1 Introduction

Daily mortality is affected not only by the same day's air pollution but also by the air pollution of a number of preceding days. In the same way, air pollution can increase the deaths occurring on the same day as well as on several subsequent days. The effect of air pollution on mortality is more likely to be spread over several days (Zanobetti *et al.*, 2000). The models in Chapter 6 estimated the short-term effects of air pollution on a single day mortality; either on the same day or on the deaths occurring a few days after exposure. It did not combine the effects across several days. In order to measure the real effect of air pollution on mortality, we need a model that combines the effects of air pollution on mortality on the same day and on many subsequent days.

Distributed lag models can estimate the total effect of air pollution on mortality spread over multiple days and indicate how the effects are distributed over multiple days. The main aims of this chapter are to estimate the extended effects of PM₁₀ on daily mortality using polynomial distributed lag models and to explore how the effects are distributed over several days. This chapter also analyses the sensitivity of the extended effects of PM₁₀ on daily mortality to the different methods by which the confounders are adjusted for.

8.2 Distributed lag models

Pope and Schwartz (1996) described the application of distributed lag models in epidemiology (Pope and Schwartz, 1996) and a few studies have used this method to estimate the total effect of daily PM₁₀ on mortality distributed over several days (Schwartz, 2000b; Braga, Zanobetti and Schwartz, 2001; Kim, Kim and Hong, 2003; Goodman, Dockery and Clancy, 2004). The main basis of this model is that the number of deaths on any given day depends on PM₁₀ concentrations of the same day and on several previous days' (lagged) PM₁₀ concentrations. The simplest method is to fit the regression model for daily number of deaths with the same day's PM₁₀, the previous days' PM₁₀ and other covariates as independent variables. The model separately estimates the mortality effects of the same day's PM₁₀ and each previous days' (lagged) PM₁₀. The sum of the estimated mortality effect of a unit increase in the same day's PM₁₀ concentrations and the estimated mortality effects of a unit increase in PM₁₀

concentrations on preceding days gives the estimated total effect of a unit increase in daily PM₁₀ on mortality spread over several days. Such distributed lag models for daily mortality which include the same day's PM₁₀ and the previous days' PM₁₀ as predictor variables are generally known as unconstrained distributed lag models as no constraint is applied for PM₁₀ effects (Schwartz, 2000b).

For Poisson regression, the unconstrained distributed lag model is

$$E(Y_t) = Pop \times \exp \left\{ \mu + \beta_0 PM_t + \beta_1 PM_{t-1} + \beta_2 PM_{t-2} + \dots + \beta_q PM_{t-q} + \sum_j \lambda_j f(X_{j,t}) \right\} \quad (8-1)$$

Where Y_t is the number of deaths on day "t", Pop is the population at risk, $PM_t, PM_{t-1}, PM_{t-2}, \dots, PM_{t-q}$ are PM₁₀ concentrations on day $t, t-1, t-2, \dots, t-q$ respectively, $\beta_0, \beta_1, \beta_2, \dots, \beta_q$ are the log relative risks of mortality associated with an increase of 1 $\mu\text{g}/\text{m}^3$ in $PM_t, PM_{t-1}, PM_{t-2}, \dots, PM_{t-q}$ respectively and $X_{j,t}$ are the other daily confounding variables. The sum $\beta_0 + \beta_1 + \beta_2 + \dots + \beta_q$ gives the estimated total mortality effect of a unit increase in daily PM₁₀ concentrations.

Due to a higher degree of serial correlation in daily PM₁₀ data, the Poisson regression model in equation (8-1) will have a collinearity problem. As a result, it produces unstable estimates of individual regression coefficients. There will be too much noise in the regression coefficients to provide any information on the shape of the distribution of effects of PM₁₀ on daily mortality over several days. Although the model estimates unstable individual PM₁₀ coefficients (β_j 's ; $j = 0, 1, 2, \dots, q$), their sum ($\beta_0 + \beta_1 + \beta_2 + \dots + \beta_q$) gives an unbiased estimate of the total mortality effect of a unit increase in daily PM₁₀ concentration (Schwartz, 2000b).

Constraining the variation of β_j 's to some shape will reduce noise in individual PM₁₀ coefficients and help to explain the shape of the distribution of lagged PM₁₀ effects on daily mortality. This could be done by calculating the weighted sum of lagged PM₁₀ concentrations with weights that reflect the relative mortality effects of the same day and individual lagged PM₁₀ concentrations, and then modelling daily mortality on the weighted sum of the lagged PM₁₀ concentrations instead of the lagged PM₁₀ concentrations themselves. For such models, equation (8-1) can be rewritten as follows:

$$E(Y_t) = Pop \times \exp \left\{ \mu + \beta Z_t + \sum_j \lambda_j f(X_{j,t}) \right\} \quad (8-2)$$

Where Z_t is the weighted sum of lagged PM₁₀ concentrations,

$$Z_t = W_0 PM_t + W_1 PM_{t-1} + W_2 PM_{t-2} + \dots + W_q PM_{t-q} \quad (8-3)$$

and $W_0, W_1, W_2, \dots, W_q$ are the weights reflecting relative mortality effects of the same day's PM_{10} (PM_t) and those on preceding days ($PM_{t-1}, PM_{t-2}, \dots, PM_{t-q}$).

Unlike the unconstrained distributed lag model in equation (8-1), the model in equation (8-2) puts a constraint on the shape of the distribution of daily PM_{10} effects on mortality over several days and is thus referred as a constrained distributed lag model (Schwartz, 2000b). One can change the constraint by changing weights (W_j 's ; $j = 0, 1, 2, \dots, q$) in equation (8-3), which will also change the shape of the variation of PM_{10} effects with lag days.

The models with a single day's PM_{10} exposure (either the same day or the individual lag day) as discussed in Chapter 6, are also a kind of constrained distributed lag model. If we are calculating the same day effect of PM_{10} concentrations on daily mortality, we set the weight W_0 at 1 and all other weights W_j 's at 0. Similarly, to calculate the effect of 1-day lagged PM_{10} concentrations, all the weights W_j 's are set at 0 except for $j = 1$, and W_1 is set at 1. In these models, we assume that the effects of PM_{10} are limited to a single day, either on the same day or on the following days.

A few studies have used a lagged moving average of daily PM_{10} concentrations as an exposure variable (Pope, Schwartz and Ransom, 1992; Morgan *et al.*, 1998; Goldberg *et al.*, 2001; Katsouyanni *et al.*, 2001). These models put a constraint $W_0 = W_1 = W_2 = \dots = W_q$ with the assumption that mortality effects of the same day's PM_{10} and the preceding days' PM_{10} are equal. Both of these models are very restrictive and may not reflect the real risk of mortality associated with daily PM_{10} concentrations (Pope and Schwartz, 1996).

A more flexible and common approach is to constrain the shape of the extended effects of daily PM_{10} over lag days to follow some polynomial function of lag number (Almon, 1965; Schwartz, 2000b). Such models are referred to as polynomial distributed lag models and are discussed in detail in the next section. For these models, weights are calculated as follows

$$W_j = \sum_{k=0}^d v_k j^k \quad ; j = 0, 1, 2, \dots, q \quad (8-4)$$

where d is the degree of polynomial, q is the number of lags and v_k ; $k = 0, 1, 2, \dots, d$ are the parameters of polynomial distributed lag models.

8.3 Polynomial distributed lag models

The polynomial distributed lag model was first introduced by Almon (1965) for Gaussian data and Schwartz (2000b) extended the application of this method for Poisson data in a generalised additive model (Almon, 1965; Schwartz, 2000b). Schwartz (2000b) showed for the first time the use of polynomial distributed lag models in studying the distributed effects of daily PM₁₀ concentrations on mortality over a number of days. Since then, a number of studies have applied this method in time series epidemiological studies to estimate the extended effects of particulate air pollution and temperature on daily mortality (Braga, Zanobetti and Schwartz, 2002; Zanobetti *et al.*, 2002; Kim, Kim and Hong, 2003; Zanobetti *et al.*, 2003; Goodman, Dockery and Clancy, 2004).

The polynomial distributed lag model constrains the coefficients β_j 's in equation (8-1) to follow a polynomial function of lag number j . The polynomial distributed lag model with q number of lags and d degrees of polynomial puts the following constraints on β_j 's in equation (8-1).

$$\beta_j = \sum_{k=0}^d \eta_k j^k \quad ; j = 0, 1, 2, \dots, q \quad q > d \quad (8-5)$$

Substituting β_j 's from equation (8-5) in equation (8-1) and suppressing covariates and other terms except for PM₁₀ terms, equation (8-1) can be rewritten as

$$E(Y_t) = \exp \left(\sum_{j=0}^q \left(\sum_{k=0}^d \eta_k j^k \right) PM_{t-j} \right) \quad (8-6)$$

which becomes, after rearrangement of the terms,

$$E(Y_t) = \exp \left(\sum_{k=0}^d \eta_k \left(\sum_{j=0}^q j^k PM_{t-j} \right) \right) \quad (8-7)$$

Thus, equation (8-1) can be expressed as

$$E(Y_t) = Pop \times \exp \left\{ \mu + \eta_0 Z_0 + \eta_1 Z_1 + \eta_2 Z_2 + \dots + \eta_d Z_d + \sum_j \lambda_j f(X_{j,t}) \right\} \quad (8-8)$$

where Z_i ; $i = 0, 1, 2, \dots, d$ are the weighted sums of lagged PM₁₀ concentrations such that

$$Z_i = \sum_{j=0}^q j^i PM_{t-j} \quad ; i = 0, 1, 2, \dots, d \quad (8-9)$$

and $\eta_0, \eta_1, \eta_2, \dots, \eta_d$ are the parameters of the polynomial distributed lag model. We can create $d+1$ new variables Z_i ; $i = 0, 1, 2, \dots, d$ representing the weighted sums of lagged PM₁₀ concentrations and use them in the Poisson regression model

(equation (8-8)) to fit a polynomial distributed lag model and estimate its parameters η_i ; $i = 0, 1, 2, \dots, d$ (Schwartz, 2000b).

After estimating the parameters of the polynomial distributed lag model (η_i 's), the effect size estimates (β_j 's) of the same day PM₁₀ and the lagged PM₁₀ on daily mortality can be calculated by expanding equation (8-5) as follows:

$$\begin{aligned}
 \beta_0 &= \eta_0 \\
 \beta_1 &= \eta_0 + \eta_1 + \eta_2 + \eta_3 + \dots + \eta_d \\
 \beta_2 &= \eta_0 + \eta_1 2 + \eta_2 2^2 + \eta_3 2^3 + \dots + \eta_d 2^d \\
 \beta_3 &= \eta_0 + \eta_1 3 + \eta_2 3^2 + \eta_3 3^3 + \dots + \eta_d 3^d \\
 &\vdots \\
 \beta_q &= \eta_0 + \eta_1 q + \eta_2 q^2 + \eta_3 q^3 + \dots + \eta_d q^d
 \end{aligned}
 \tag{8-10}$$

where q is the number of lag days and d is the degree of polynomial.

The above set of equations can be expressed in matrix notation as:

$$\boldsymbol{\beta} = \mathbf{J} \boldsymbol{\eta}
 \tag{8-11}$$

where $\boldsymbol{\beta}$ is a vector ($\beta_0, \beta_1, \beta_2, \beta_3, \dots, \beta_q$)^t of length $q+1$, $\boldsymbol{\eta}$ is a vector ($\eta_0, \eta_1, \eta_2, \eta_3, \dots, \eta_d$)^t of length $d+1$ and \mathbf{J} is a $(q+1) \times (d+1)$ matrix

$$\begin{pmatrix}
 1 & 0 & 0 & 0 & \dots & 0 \\
 1 & 1 & 1 & 1 & \dots & 1 \\
 1 & 2 & 2^2 & 2^3 & \dots & 2^d \\
 1 & 3 & 3^2 & 3^3 & \dots & 3^d \\
 1 & 4 & 4^2 & 4^3 & \dots & 4^d \\
 \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\
 1 & q & q^2 & q^3 & \dots & q^d
 \end{pmatrix}$$

The covariance matrix of $\boldsymbol{\beta}$ is

$$\text{cov}(\boldsymbol{\beta}) = \mathbf{J} \text{cov}(\boldsymbol{\eta}) \mathbf{J}^t
 \tag{8-12}$$

and the square root of the $(j+1)$ th diagonal element of $\text{cov}(\boldsymbol{\beta})$, which is the $((j+1), (j+1))$ th element of $\text{cov}(\boldsymbol{\beta})$ gives the standard error of β_j ; $j = 0, 1, 2, \dots, q$.

The sum of β_j 's gives an estimate of the total effect of a unit increase in PM₁₀ on daily mortality. Thus, the total mortality effect β can be calculated as

$$\beta = \mathbf{1}^t \boldsymbol{\beta} \quad (8-13)$$

where $\mathbf{1}$ is a unit vector $(1, 1, 1, 1, \dots, 1)^t$ of length $q+1$. The standard error of β is calculated as

$$\begin{aligned} \text{se}(\beta) &= \sqrt{\mathbf{1}^t \text{cov}(\boldsymbol{\beta}) \mathbf{1}} \\ &= \sqrt{\mathbf{1}^t \mathbf{J} \text{cov}(\boldsymbol{\eta}) \mathbf{J}^t \mathbf{1}} \end{aligned} \quad (8-14)$$

8.4 Methods

The fourth degree polynomial distributed lag model was used to estimate the overall risk of mortality distributed over 0 to 40 days after PM_{10} exposure (over 41 days including the day of exposure). Five new variables representing the weighted sums of PM_{10} concentrations of the same day and up to 40 lag days were created as described in equation (8-9) and the Poisson regression model (equation (8-8)) was fitted for daily mortality to estimate the parameters of the fourth degree polynomial distributed lag model. The parameters define the shape of PM_{10} effects on daily mortality distributed over 41 days. The individual effect coefficients (β_j 's) of the same day PM_{10} and the lagged PM_{10} on daily mortality and their standard errors were estimated as described in equations (8-10) and (8-12). The overall risk of mortality due to PM_{10} concentrations distributed over 0 to 40 days was estimated by totalling the estimated individual effect coefficients (β_j 's), and its standard error was calculated using equation (8-14).

Analyses were separately undertaken for daily non-external deaths of all ages, and for daily non-external deaths, circulatory cause deaths and respiratory cause deaths of the 65+ years age group.

A long-term trend and seasonal variation in daily mortality and the confounding effects of weather variables were controlled for in the Poisson regression model (equation (8-8)) using natural cubic spline smooth functions of calendar time and weather variables, as mentioned in Sections 6.3.1 (Method 2, p.84) and 6.3.2 (Method 2, p.87). In addition to PM_{10} exposure variables, the models included

- a natural cubic spline smooth function of calendar time with 81 degrees of freedom i.e. approximately 7 df per year of data to control for a long-term trend and seasonal variation in daily mortality;
- a set of dummy variables for days of the week to control for daily variation in mortality across the week;
- natural cubic spline smooth functions of the same day's average hourly temperature and a moving average of the preceding three days' (lag 1 to lag 3)

average hourly temperature with 6 df each to control for the confounding effect of temperature; and

- natural cubic spline smooth functions of the same day's average hourly relative humidity and a moving average of the preceding three days' (lag 1 to lag 3) average hourly relative humidity with 3 df each to control for the confounding effect of relative humidity.

The check of overdispersion parameters and residual autocorrelation coefficients for the models with the above variables provided no evidence of over-dispersion or residual autocorrelations for any of the mortality categories analysed (Section 6.4.2, p.117). Thus, controlling for overdispersion and autocorrelation were not required for any of the models.

A quadratic or a cubic distributed lag model is usually flexible enough to approximate a biologically plausible lag structure of PM₁₀ effects on daily mortality (Pope and Schwartz, 1996). However, a fourth degree polynomial was fitted to ensure adequate flexibility to better define the shape of the distribution of PM₁₀ effects on daily mortality over time. A cubic distributed lag model, a quadratic distributed lag model and an unconstrained distributed lag model were also fitted as sensitivity analyses.

Similar to the sensitivity analysis in Section 6.4.3 (p.130), the degrees of freedom for the natural cubic spline smooth function of calendar time were varied to examine the sensitivity of the estimated extended effects of daily PM₁₀ on mortality to adjustment for a long-term trend and seasonal variation in mortality. Additional analyses to examine the sensitivity of PM₁₀ effect estimates to the degrees of freedom for natural cubic spline smooth functions of weather variables were also performed.

8.5 Results

Table 8-1 compares the PM₁₀ associated risk of single day mortality with the total risk of mortality summed over 0 to 40 days after PM₁₀ exposure under various distributed lag models for different mortality categories. Risks are expressed as percentage increases in mortality for each 10 µg/m³ increase in PM₁₀ concentrations. For distributed lag models, the percentage increases in mortality are summed over 41 days, including the day of exposure (0 day after exposure). The results of distributed lag models are compared with the effect of PM₁₀ lag that had the largest effect on single day mortality.

The relative risks of mortality associated with an increase in daily PM₁₀ concentrations were substantially higher when the lagged effects were considered using

distributed lag models for each of the mortality categories analysed. Each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} was associated with a 1.31% (95% CI: 0.44, 2.18) increase in the same day total non-external mortality of all ages. When the lagged effects were considered using the fourth degree polynomial distributed lag model, the overall percentage increase in total non-external mortality of all ages over 41 days, including the day of exposure, was 13.3% (95% CI: 6.1, 20.9) for a $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} . In the population aged 65+ years, the percentage increase in non-external mortality for each $10 \mu\text{g}/\text{m}^3$ in the same day PM_{10} was 1.55% (95% CI: 0.6, 2.5) whereas the overall percentage increase in non-external mortality over 41 days, including the day of exposure, was 14.9% (95% CI: 6.99, 23.4).

For circulatory deaths in the population aged 65+ years, the percentage increase for each $10 \mu\text{g}/\text{m}^3$ increase in 1-day lagged PM_{10} was 1.84% (95% CI: 0.54, 3.15), compared to the overall percentage increase of 18.7% (95% CI: 7.3, 31.3) over 41 days for each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} when the lagged effects were combined using the fourth order polynomial distributed lag model. The estimated percentage increase in mortality due to PM_{10} was considerably greater for respiratory deaths than for other causes of deaths. For respiratory deaths of the 65+ years age group, the overall percentage increase in mortality over 41 days was 26.9% (95% CI: 4.5, 54.1) for each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} .

Although the total effect size estimates were similar for all distributed lag models including the unconstrained distributed lag models, they increased slightly with an increase in the order of models, with unconstrained distributed lag models providing the highest effect size estimates, but with slightly wider confidence intervals. This may indicate that polynomial distributed lag models marginally underestimated the total effect size, given that the estimates from the unconstrained distributed lag models were unbiased.

Table 8-1. Estimated percentage increase in daily mortality (95% CI) associated with each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} concentrations and the estimated overall percentage increase in mortality associated with each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} concentrations under different distributed lag models for 0 to 40 lag days

Causes of deaths	1-day mortality [#]	Distributed lag model			
		4 th order	Cubic	Quadratic	Unconstrained
Total non-external					
All ages	1.31 (0.44, 2.18)	13.3 (6.1, 20.9)	13.0 (5.9, 20.7)	12.9 (5.9, 20.6)	13.9 (6.7, 21.8)
65+ yrs	1.55 (0.60, 2.50)	14.9 (6.99, 23.4)	14.7 (6.9, 23.2)	14.7 (6.8, 23.1)	15.7 (7.6, 24.3)
Circulatory 65+ yrs	1.84 (0.54, 3.15)	18.7 (7.3, 31.3)	18.6 (7.2, 31.1)	18.5 (7.2, 31.1)	18.9 (7.4, 31.7)
Respiratory 65+ yrs	2.95 (0.55, 5.4)	26.9 (4.5, 54.1)	26.3 (4.01, 53.3)	26.1 (3.9, 53.1)	28.7 (5.6, 56.7)

[#]Note: For 1-day mortality, the percentage increases in mortality were for the PM_{10} lag that had the strongest effect on single day mortality. For non-external mortality, the effects were of the same day's PM_{10} . For circulatory mortality and respiratory mortality, they were the effects of 1-day lagged PM_{10} and 2-day lagged PM_{10} respectively.

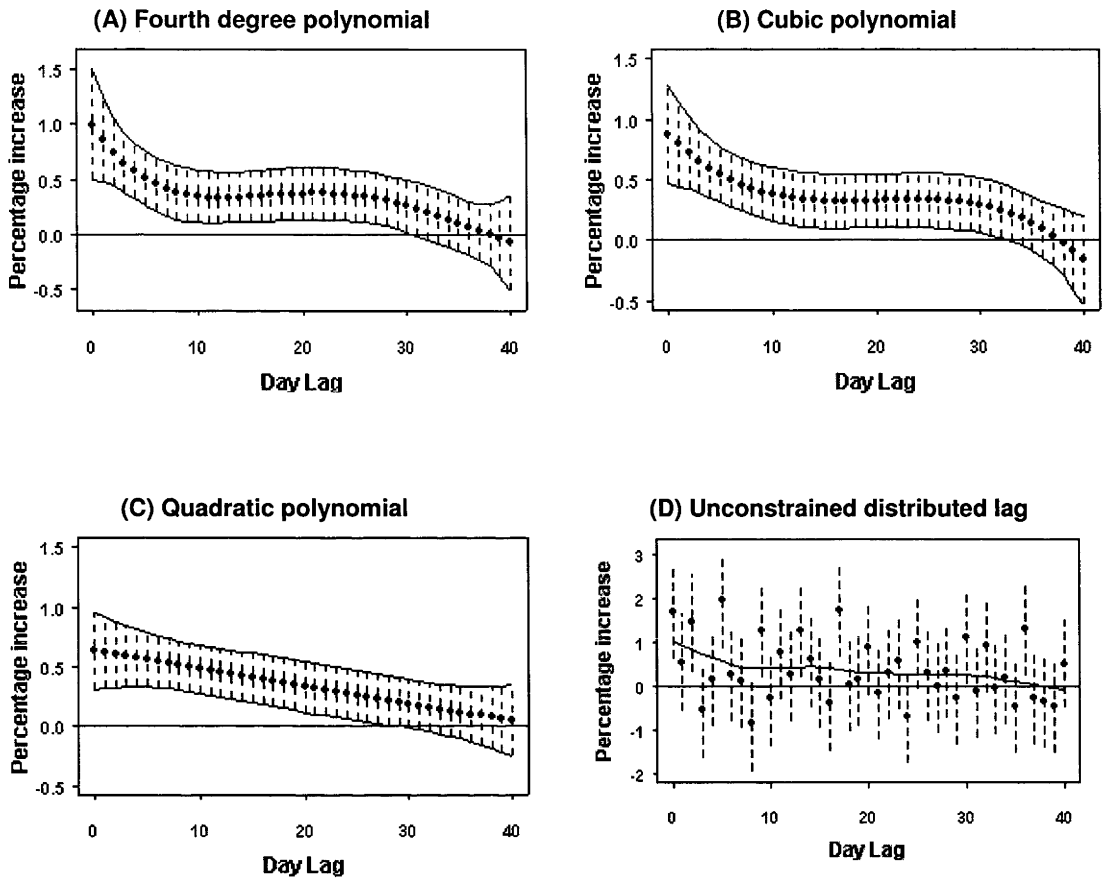
Results of polynomial distributed lag models showed significant increased risks of daily non-external mortality due to PM_{10} up to four weeks after exposure in the population aged 65+ years (Figure 8-1). PM_{10} had the major immediate effect on the first few days after exposure. The delayed effect of PM_{10} decreased but stayed significantly positive for up to four weeks after exposure. Results from the fourth degree polynomial and cubic distributed lag models showed that the effect remained fairly constant between 10 and 28 days after exposure (Figures 8-1(A) and 8-1(B)). Although statistically non-significant, the effect remained positive between 30 and 37 days after exposure. This showed that besides an immediate effect on the same day mortality, PM_{10} had a significant delayed effect on non-external mortality of 65+ years suggesting that the model with single day's PM_{10} underestimates the overall effect of PM_{10} .

Polynomial distributed lag models showed how the PM_{10} effects were distributed over time and thus were useful to explore the shape of the distribution of PM_{10} effects on daily mortality over days. Estimated PM_{10} effects at each lag in unconstrained distributed lag models had too much noise to provide any information about the shape of the distribution of PM_{10} effects on daily mortality over lag days (Figure 8-1(D)). A lowess smooth with a bandwidth of 0.4 was applied to the plot to reveal the shape of the distribution of PM_{10} effects. The smoothed shape approximately matched the shape of the distribution of PM_{10} effects from the polynomial distributed lag models.

Figure 8-2 shows the estimated increased risk of non-external deaths of all ages, circulatory deaths in the 65+ years age group and respiratory deaths in the 65+ years age group, associated with PM_{10} , at each lag with fourth degree polynomial distributed lag models. Although the shapes of the distributions of increased risks of daily deaths due to PM_{10} over lag days differed slightly, they had similar patterns in general. Significantly increased risks of non-external deaths of all ages and circulatory deaths in the 65+ years age group were found up to four weeks after PM_{10} exposure. Similar to the results for non-external deaths in the population aged 65+ years, effects were strongest for the first few days after exposure and then decreased but stayed significantly positive for up to four weeks. For all ages, the risk of total non-external deaths elevated to a second smaller peak about 20 days after exposure (Figure 8-2(A)).

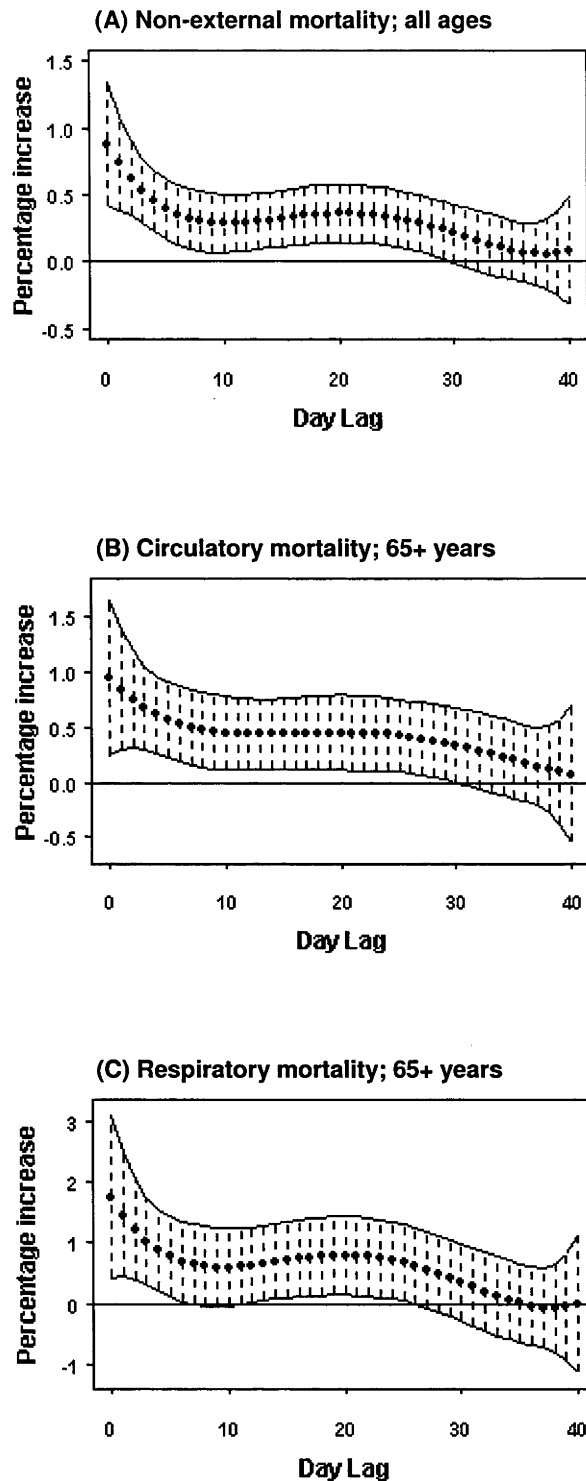
PM_{10} was significantly associated with an increased risk of respiratory deaths in the 65+ years age group in the first week after exposure (Figure 8-2 (C)). Risks associated with PM_{10} were not statistically significant between 7 and 10 days after exposure. The risks then elevated to a second smaller peak around 20 days after exposure and stayed significantly positive for up to 25 lag days before dying out to zero by 40 lag days.

Figure 8-1. Estimated percentage increase in daily non-external deaths of the 65+ years age group associated with a $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} under various distributed lag models



Note: Y-axis scale is different

Figure 8-2. Estimated percentage increase in daily deaths associated with a $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} with fourth degree polynomial distributed lag models for various mortality categories



8.6 Sensitivity analysis

Figure 8-3 shows the sensitivity of the estimated shape of the association between PM_{10} and daily non-external deaths of the 65+ years age group to using various degrees of freedom for the natural cubic spline smooth functions of time for the adjustment of a

long-term trend and seasonal variation in daily mortality, with fourth degree polynomial distributed lag models. In the original analysis (results presented in Section 8.5), 7 degrees of freedom per year of data were used to adjust for a long-term trend and seasonal variations in daily mortality. The analysis was repeated using 3 to 10 degrees of freedom per year of data for the natural cubic spline smooth function of time. Irrespective of the number of degrees of freedom set for the smooth function of time, the distributions of PM_{10} associated increased risks of daily non-external deaths over lag days in the population aged 65+ years had the same general shape (Figure 8-3). Risks of mortality due to daily PM_{10} remained positive for five weeks after exposure, though were statistically non-significant at some lags. When 5, 6 7 and 9 degrees of freedom per year of data were used for the smooth function of time, statistically significant increased risks were found up to four weeks after exposure. Using 3 and 4 degrees of freedom per year of data, the delayed effects were statistically significant up to four weeks after exposure, though were not so during the second and third weeks after exposure.

Using fewer degrees of freedom for the smooth function of time gave lower estimates of the total PM_{10} effects on daily non-external mortality of the 65+ years age group summed over 0 – 40 days (Figure 8-4 (A)). The effect estimates stabilised, however, after seven or more degrees of freedom per year of data were used. Effect estimates had wider confidence intervals when greater degrees of freedom were assigned for the natural cubic spline smooth functions of time.

Figure 8-3. Sensitivity of the estimated shape of PM₁₀ effects to the degrees of freedom set for the smooth function of time. Estimated percentage increase in daily non-external deaths of the 65+ years age group for each 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀, with the fourth degree polynomial distributed lag models.

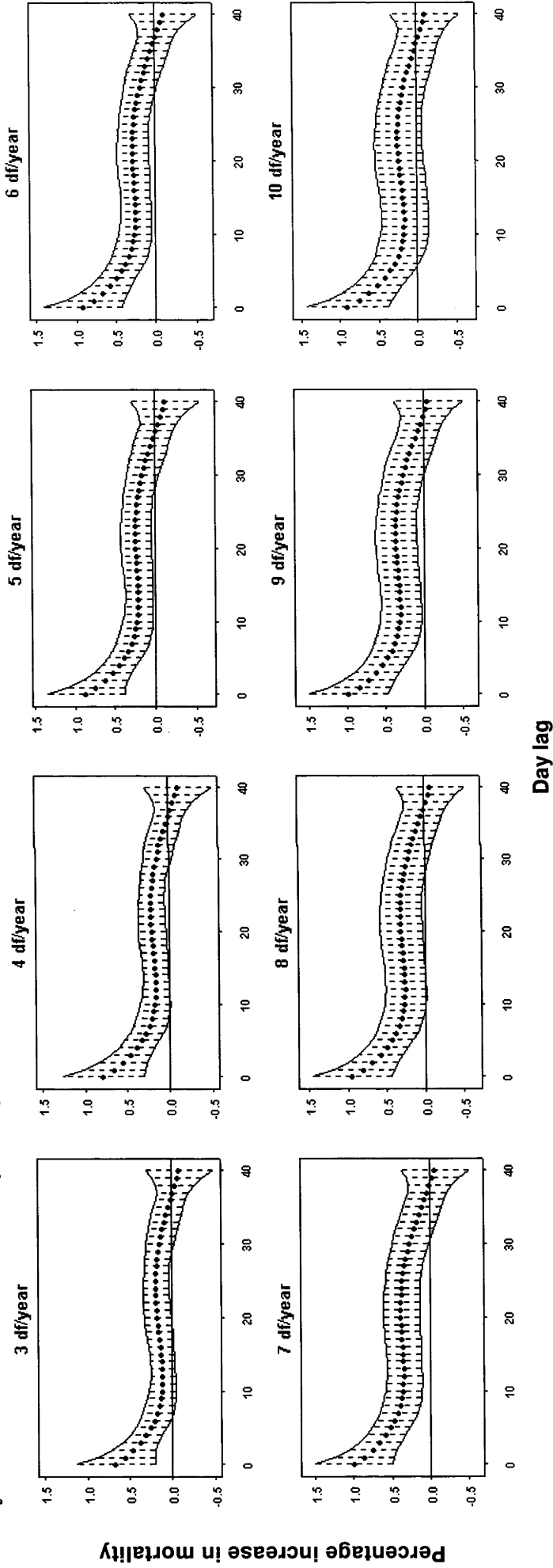
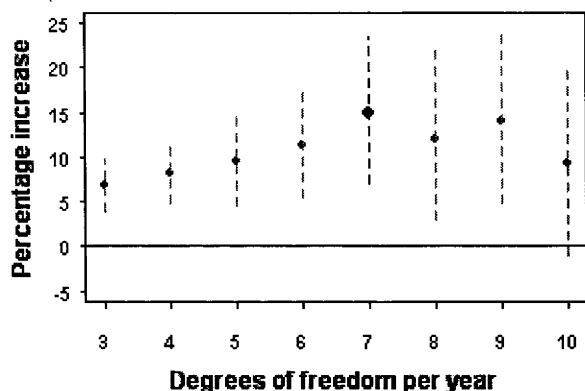
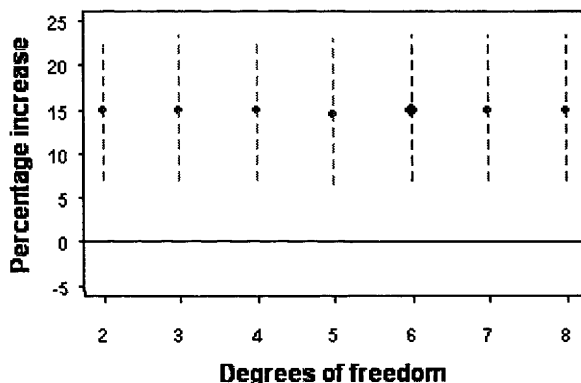


Figure 8-4. Sensitivity of the extended effects of PM₁₀ on daily non-external mortality of the 65+ years age group over 41 days to degrees of freedom set for the smooth functions of confounders. Estimated overall percentage increase in daily deaths and 95% CI for each 10 µg/m³ increase in PM₁₀, with fourth degree polynomial distributed lag models.

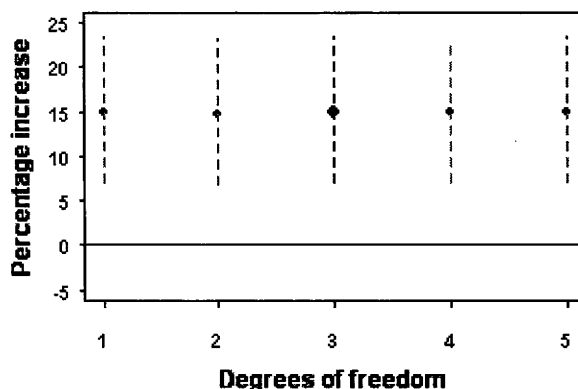
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature



(C) Degrees of freedom for the smooth function of relative humidity



The PM₁₀ associated total risk of non-external deaths in the population aged 65+ years over 0 – 40 days did not depend upon the adjustment for the effect of temperature or relative humidity. Increased risks of mortality due to daily PM₁₀ and their 95% confidence intervals were almost equal, irrespective of the degree of freedom for the

natural cubic spline smooth functions of temperature or relative humidity (Figures 8-4 (B) and 8-4 (C)).

Sensitivity analyses for other mortality groups (non-external deaths of all ages, circulatory deaths of the 65+ years age group and respiratory deaths of the 65+ years age group) are presented in Appendix E. The findings were similar to the results for non-external deaths of the 65+ years age group. They also showed that the PM₁₀ associated increased risk of mortality did not depend upon the degree of freedom for the smooth functions of temperature or relative humidity to control for the effects of temperature or relative humidity whereas it changed considerably with changes in the degrees of freedom for the smooth function of time to control for a long-term trend and seasonality.

8.7 Summary

This chapter describes the use of distributed lag models to estimate the extended effects of PM₁₀ on mortality. The PM₁₀ associated total risks of mortality over 0 – 40 days after exposure under a fourth degree polynomial distributed lag model, a cubic distributed lag model, a quadratic distributed lag model and unconstrained distributed lag model were compared with each other and with the effects of PM₁₀ on single day mortality. Polynomial distributed lag models show that PM₁₀ was associated with significantly increased risks of mortality for several weeks after exposure. The total effects of PM₁₀ spread over several weeks were substantially higher than the effect of PM₁₀ on single day mortality. The results were consistent across various distributed lag models. For non-external deaths in the population aged 65+ years, the total percentage increase in mortality over 0 to 40 days for a 10 µg/m³ increase in PM₁₀ was 14.9% (95% CI: 6.99, 23.4) whereas the percentage increase in the same day mortality for the same increase in PM₁₀ was only 1.55% (95% CI: 0.6, 2.5). This suggests that the studies focussing on single day effects highly underestimate the real effect of PM₁₀ on daily mortality.

The total risks of mortality associated with PM₁₀ did not depend upon the number of degrees of freedom for the natural cubic spline smooth functions of temperature or relative humidity, suggesting that the risk was not sensitive to the way the confounding effects of temperature and relative humidity were adjusted for. The total risk of mortality was, however, sensitive to the number of degrees of freedom set for the natural cubic spline smooth function of time variable used to control for seasonal variation and a long-term trend in daily mortality. The total PM₁₀ effects over 0 to 40

days were slightly lower when fewer degrees of freedom were used for the smooth function of time, but the results were not meaningfully different.

Chapter 9: Association of annual PM₁₀ with mortality

9.1 Introduction

Time series analysis in Chapters 6, 7 and 8 provided evidence of associations between PM₁₀ and mortality over short to mid-term time scales. Time series studies, based on daily data, are designed to pick up short-term increase in number of air pollution related deaths against the background long-term mortality. These studies only estimate the acute effects of air pollution and provide no information on the association of air pollution with the long-term mortality. For the health effect assessment of air pollution including estimating the number of deaths attributed to air pollution, the health effect estimates that combine both the short-term and long-term health effects of air pollution are required. The health effect estimates of long-term exposure to air pollution can provide the combination of both chronic and acute effects of air pollution and thus are the most appropriate effect measurement for the health effect assessment. This chapter quantifies the effects of long-term exposure to PM₁₀ on long-term mortality. It is reasonable to assume annual average air pollution levels as long-term exposure levels for the people living in a spatial area. The association of annual average PM₁₀ concentrations with annual mortality adjusting for confounders, such as age, sex, ethnicity, area-level socioeconomic status and smoking is analysed in this chapter. This chapter also explores the spatial variation in population characteristics and PM₁₀ exposure in Christchurch. Census area unit (CAU) is chosen as a small scale geographic unit to explore spatial variation.

9.2 Study design

This is an ecological cross-sectional study of air pollution and mortality with the spatial unit “CAU” as the unit of analysis. This study associates spatial variation of annual average PM₁₀ concentrations with spatial variation of mortality after controlling for potential confounders. The hypothesis analysed in this study is that the mortality rates at CAUs with higher annual average PM₁₀ concentrations are greater than the mortality rates at CAUs with lower annual average PM₁₀ concentrations. An increase in mortality rates is associated with an increase in annual average PM₁₀ concentrations of CAUs.

The key assumptions in this study are:

- all people within each CAU are exposed to the same level of annual PM₁₀ level;

- personal exposure to annual average PM₁₀ level can be measured at their place of usual residence;
- personal exposure to annual average of weather variables is uniform across Christchurch;
- between-year variations in spatial distribution of annual average PM₁₀ level are negligible; and
- all people within a CAU have the same socioeconomic status as the socioeconomic status calculated for that CAU.

9.3 Confounders

Age, sex and ethnicity are important determinants of mortality. The ethnic differences in mortality in New Zealand have been well documented. Studies have found that Maori and Pacific Islanders have higher mortality rates than Europeans (Ministry of Health, 1999; Blakely *et al.*, 2005; Ministry of Health and University of Otago, 2006). There is no concern for confounding effect of these variables (age, sex and ethnicity) in time series analysis (Chapters 6, 7 and 8) as time series analysis compares short-term temporal variations between air pollution and mortality and these variables do not vary in short-term. Time series studies require controlling for the confounding effects of those variables which vary from day-to-day like weather variables. Unlike time series studies, ecological cross-sectional studies compare geographic areas to analyse the association between air pollution and mortality. The population composition with respect to age, sex and ethnicity can vary significantly between geographic areas. If different geographic areas have different population structure, then these variables may confound the relationship between air pollution and mortality.

Socioeconomic status is another important potential confounder in the relationship between air pollution and mortality (Finkelstein *et al.*, 2003; Jerrett *et al.*, 2005; Naess *et al.*, 2007). Socioeconomic status in different geographic areas can vary significantly (Crampton *et al.*, 2000). People with poor socioeconomic status may be exposed to higher air pollution levels as they have higher probability of living in the areas closer to main roads or near industrial sources where air pollution levels tend to be higher. Socioeconomic status has also been well recognised as a broad determinant of the health status of individuals and populations (Ministry of Health, 1999). A number of studies have reported a strong association of socioeconomic status with all cause mortality and specific causes of mortality (Ministry of Health, 1999; Crampton *et al.*, 2000; Blakely *et al.*, 2002; Ministry of Health, 2002; Ministry of Health and University

of Otago, 2006). The confounding factors such as age, sex, ethnicity, area-level socioeconomic status, smoking variables have been taken into account in this analysis.

9.4 Data

9.4.1 Population data

This analysis used the “usually resident population” of Christchurch CAUs for two census years 1996 and 2001, obtained from the Statistics New Zealand Census of Population and Dwellings. The census data were based on 2001 CAU boundaries. There was no change in CAU boundaries for the 1996 and 2001 census in Christchurch. This ensured that population data of both censuses belonged to the same spatial units.

Statistics New Zealand provided the “usually resident population” data by sex, age group (<1, 1-14, 15-24, 25-44, 45-64, 65-84 and 85+ years) and ethnicity (European, NZ Maori, Pacific people, Asian, Others and “not specified”). Due to very small population and low number of deaths in the age-sex-ethnic groups, data were aggregated into three age groups (<15, 15-64 and 65+ years) and five ethnic groups (European, NZ Maori, Pacific, Asian/Others and “not specified”). Assuming an equal increase in age-sex-ethnicity specific population each year between the two census years, the age-sex-ethnicity specific population for non-census years were estimated by linear interpolation from the population of two census years. As four years of mortality data from 1996 to 1999 were used in this analysis, the estimated populations for the years from 1996 to 1999 were summed in each of age-sex-ethnicity groups to give person-years of residence.

9.4.2 Mortality data

This analysis used mortality data from 1996 to 1999. This dataset was the subset of the mortality data extracted for the earlier analysis (Chapters 6, 7 and 8), which were for the period from 1988 to 1999.

The Statistics New Zealand health domicile code, which represented a deceased person’s usual residential address, was used to map mortality records to census area unit. Mortality data with the health domicile codes that matched Christchurch census area units were extracted for the period from 1996 to 1999. Mortality data in this period (1996 to 1999) had two groups of health domicile codes: the 1991 health domicile code (HDOM91) for 1996/1997 deaths which were linked with the 1991 census area unit code and the 1996 health domicile code (HDOM96) for 1998/1999 deaths which were

linked with the 1996 census area unit code (New Zealand Health Information Service, 2004).

Except for the domicile codes shown in Table 3-5, the area boundaries of all 1991 census area units matched the area boundaries of 1996 census area units in Christchurch. The census area unit boundaries of Christchurch CAUs for the 1996 census and the 2001 census exactly match. The population statistics for all three censuses (1991, 1996 and 2001) were based on the 2001 census area boundaries. For the 1996 census, part of South Brighton and part of Moncks Bay were merged and a new census area unit Avon-Heathcote Estuary was created (Table 3-5). According to the population statistics based on the 2001 census area unit boundaries, there was no one living in Avon-Heathcote Estuary part of both South Brighton and Moncks Bay in 1991. This ensured that the people, who died in 1996/1997 and had HDOM91 of 2688 were living in South Brighton part. Similarly, the people, who died in 1996/1997 and had HDOM91 of 2690 were living in Moncks Bay part. There was no one living in Avon-Heathcote Estuary in 1996 and 2001 and thus this census area unit was excluded from the analysis.

Table 9-1. Non-matching 1991 and 1996 health domicile codes in Christchurch

HDOM91	1991 CAU		HDOM96	1996 CAU	
	Area unit code	Area unit description		Area unit code	Area unit description
2688	595900	South Brighton	2700	595900	South Brighton
2688	595900	South Brighton	2701	596101	Avon-Heathcote Estuary
2690	596100	Moncks Bay	2701	596101	Avon-Heathcote Estuary
2690	596100	Moncks Bay	2702	596102	Moncks Bay

Mortality data were aggregated into three age groups (<15, 15-64 and 65+ years) and five ethnic groups (European, NZ Maori, Pacific, Asian/Others and “not specified”). Deaths were categorised by cause of deaths according to the ICD-9 code. The causes of deaths were grouped into the same three broad causes of deaths which were analysed in Chapters 6, 7 and 8. They were:

- All non-external causes (ICD-9 code: 1-799)
- Circulatory causes (ICD-9 code: 390-459)
- 1. Respiratory causes (ICD-9 code: 460-519)

The total numbers of deaths by age-sex-ethnicity for four years from 1996 to 1999 were calculated for each CAU for the above three causes of deaths. Analyses were separately conducted for each of the above causes of deaths.

9.4.3 Percentage of ever smokers

No smoking data was collected in the 2001 Census of Population and Dwellings. Percentages of ever smokers were estimated based on the 1996 census data. The number of current smokers and ex-smokers by sex, age group and ethnicity were obtained for Christchurch CAUs from the 1996 Census of Population and Dwellings. Data were aggregated into the same age, sex and ethnic groups as in population and mortality data. No data on the number of smokers was available for less than 15 years age group. As such, the numbers of smokers and ex-smokers in this age group were assumed zero. The number of current smokers and ex-smokers were added to calculate the total number of ever smokers by age-sex-ethnic group for each CAU. The age-sex-ethnicity specific percentage of ever smokers was then calculated by dividing the age-sex-ethnicity specific number of ever smokers by the age-sex-ethnicity specific population.

9.4.4 Socioeconomic status

The NZDep96 index of deprivation, created from the 1996 Census of Population and Dwellings data, was used as a measure of socioeconomic status (Crampton *et al.*, 2000). This index is based on nine deprivation variables from the 1996 Census and is therefore a composite indicator of relative social and economic deprivation. These nine variables which reflect eight types of deprivation are shown in Appendix F. The NZDep96 index of deprivation is a small area based measure. It does not describe the socioeconomic position of an individual but reflects the general socioeconomic position of all people living in the small area.

The NZDep96 index of deprivation for Christchurch CAUs was obtained from the publication “Degrees of Deprivation in New Zealand: An atlas of socioeconomic difference” (Crampton *et al.*, 2000). The NZDep96 index of deprivation was created for small areas, which were in general either one standard Statistics New Zealand meshblock or two nearby meshblocks joined together. Each census area unit is made up of many meshblocks or many small areas for which the NZDep96 index was created. The average deprivation score for each census area unit was calculated by calculating the population-weighted average of the deprivation score of the small areas that made up the census area unit. Based on deprivation score, census area units were ranked from

the least deprived (lowest score) to the most deprived (highest score) and were assigned NZDep96 index of deprivation from 1 to 10 with 1 indicating the least deprived 10 per cent of census area units and 10 indicating the most deprived 10 per cent of census area units in New Zealand (Crampton *et al.*, 2000).

9.4.5 Air pollution exposure

Geography Department of Canterbury University, New Zealand estimated annual average PM₁₀ concentrations for Christchurch CAUs as their contribution to the Health and Air Pollution in New Zealand (HAPiNZ) project. The estimates of annual PM₁₀ exposure were produced for the health impact assessment of PM₁₀ in New Zealand (Fisher *et al.*, 2005; Fisher *et al.*, 2007). These CAU based annual PM₁₀ exposures were used to analyse the effect of annual PM₁₀ on mortality in this study.

Canterbury University simulated the meteorology and dispersion of PM₁₀ continuously for 2001 over the Christchurch airshed using the Air Pollution Model (TAPM; version 2). This generated the spatially distributed datasets of hourly PM₁₀ concentrations over the Christchurch airshed. Zawar-Reza *et al.* (2005) validated PM₁₀ concentrations simulated by TAPM version 2 against PM₁₀ concentrations monitored at Christchurch's primary air quality monitoring site at St. Albans. They reported that the simulated PM₁₀ concentrations were in good agreement with observed levels at St. Albans with a very small difference of 4 µg/m³ in annually averaged simulated and monitored PM₁₀ concentrations (Zawar-Reza, Kingham and Pearce, 2005).

The annual average PM₁₀ levels for Christchurch CAUs were derived from the spatially distributed datasets of hourly PM₁₀ concentrations. The Air Pollution Model separately simulated the dispersion of PM₁₀ emissions from different sources such as domestic home heating, motor vehicle and industrial. Using TAPM modelled annual PM₁₀ levels and other variables such as the variables that could affect the pollution concentrations from domestic home heating, motor vehicle emissions such as wood fires per square kilometre and vehicle kilometres travelled per square kilometre, the variables for proximity to other CAUs and the variables describing topography of CAUs, Canterbury University produced more precise estimates of annual average PM₁₀ for Christchurch CAUs for 2001 (Fisher *et al.*, 2007). Canterbury University provided the estimates of annual average PM₁₀ for Christchurch CAUs to use in this study.

9.5 Spatial distribution of population characteristics

9.5.1 Age

Figures 9-1 and 9-2 show the spatial distributions of the proportion of population aged less than 15 years old and 65+ years old respectively. Census area units in the central business area of Christchurch had the lowest proportion (6 to 10%) of their population aged less than 15 years old. Only a few CAUs (i.e. Shirley Easy, Upper Riccarton and Barrington South) had over 20% of their population aged 65+ years old.

Figure 9-1. Percentage of under 15-year population (1996 – 1999)

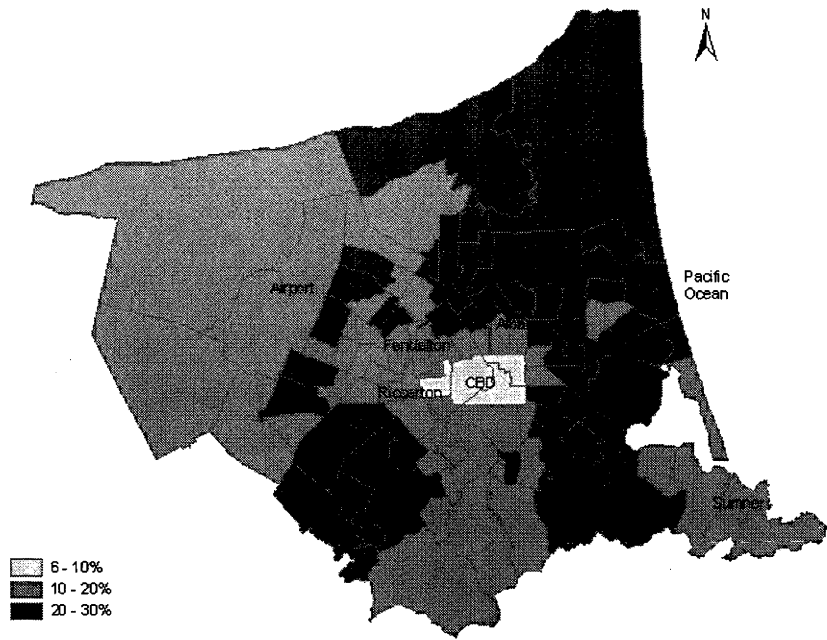
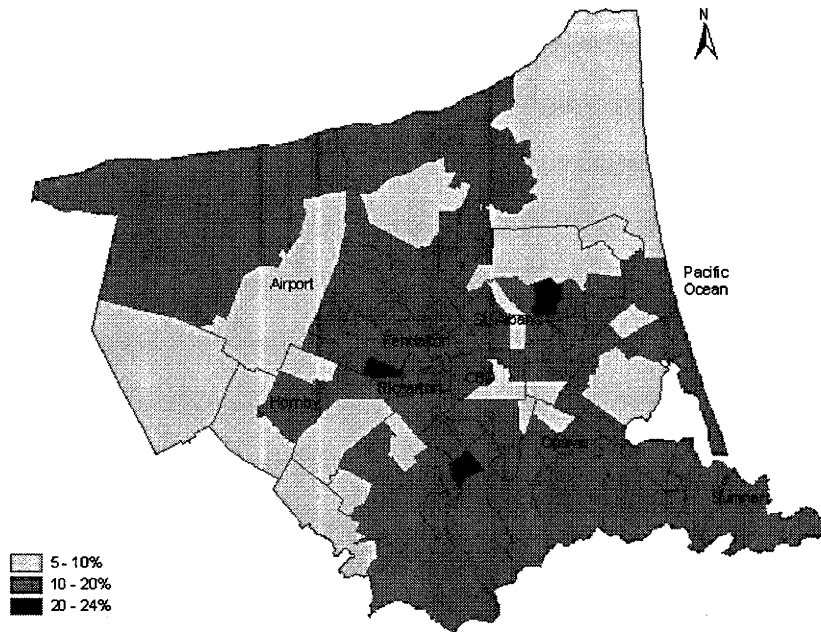


Figure 9-2. Percentage of population aged 65+ years (1996 – 1999)



9.5.2 Ethnicity

The spatial distributions of the proportion of European, Maori, Pacific and Asian/Others populations are shown in Figures 9-3, 9-4, 9-5 and 9-6 respectively. European populations were the most dominant in all CAUs. More than 70% of CAU population were of European in almost all CAUs. More than 90% of the population living in CAUs at Port Hills in the south were Europeans. Except in a few CAUs, there were a very small percentage of Pacific Islanders (less than 5% of CAU population) living in most CAUs. They tend to have higher concentrations at CAUs which had higher proportion of Maori populations. Asian / Other populations tend to have higher concentrations in the central business area of Christchurch and the north-west of central business area towards the airport.

Figure 9-3. Percentage of Europeans (1996 – 1999)

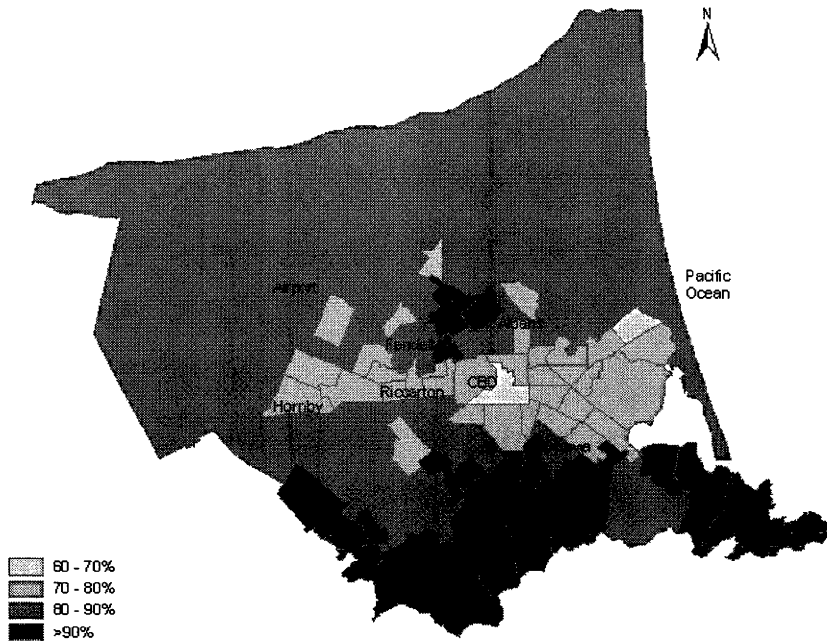


Figure 9-4. Percentage of Maori population (1996 – 1999)

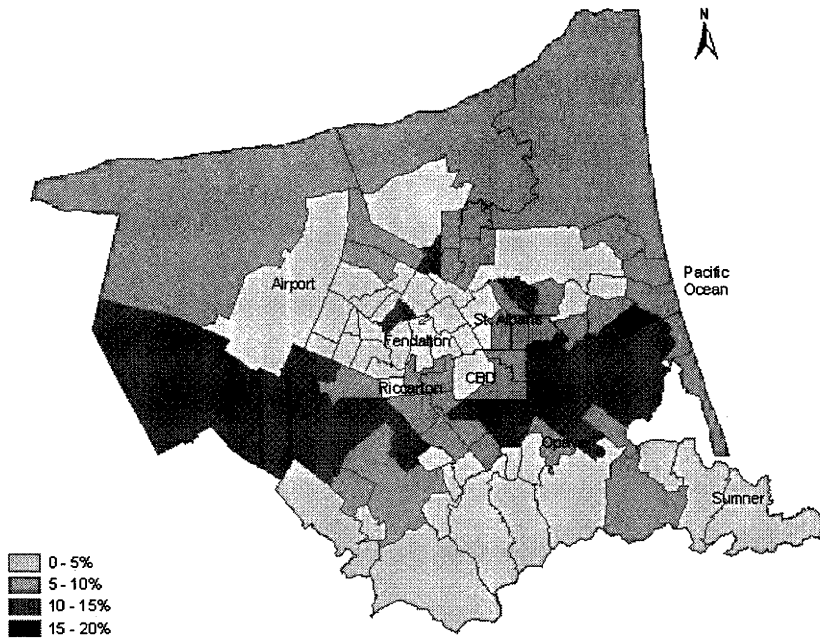


Figure 9-5. Percentage of Pacific Islander population (1996 -1999)

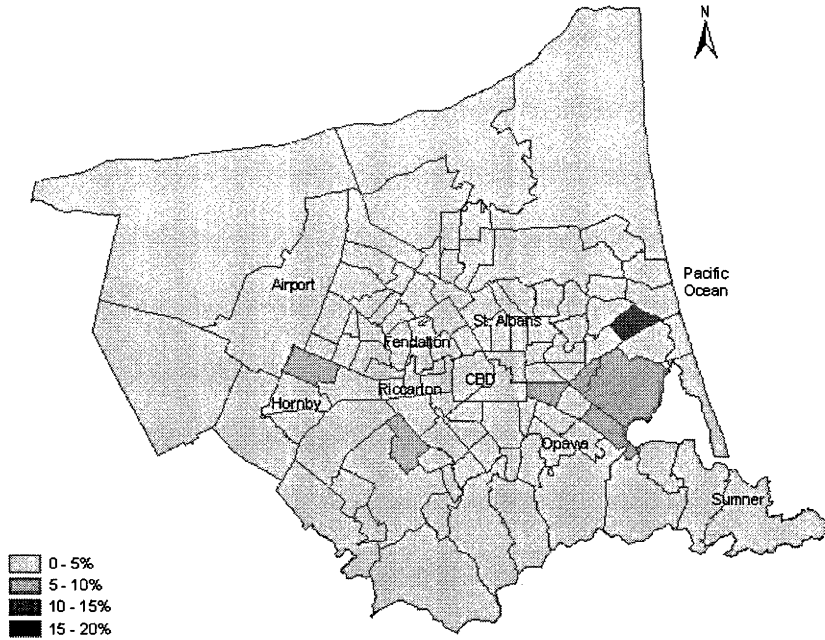
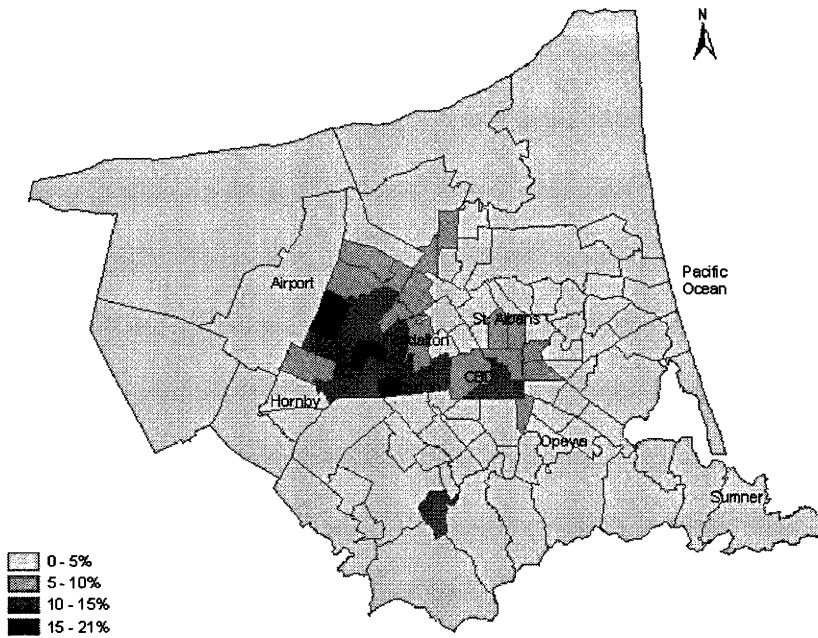


Figure 9-6. Percentage of Asian/Others (1996 - 1999)

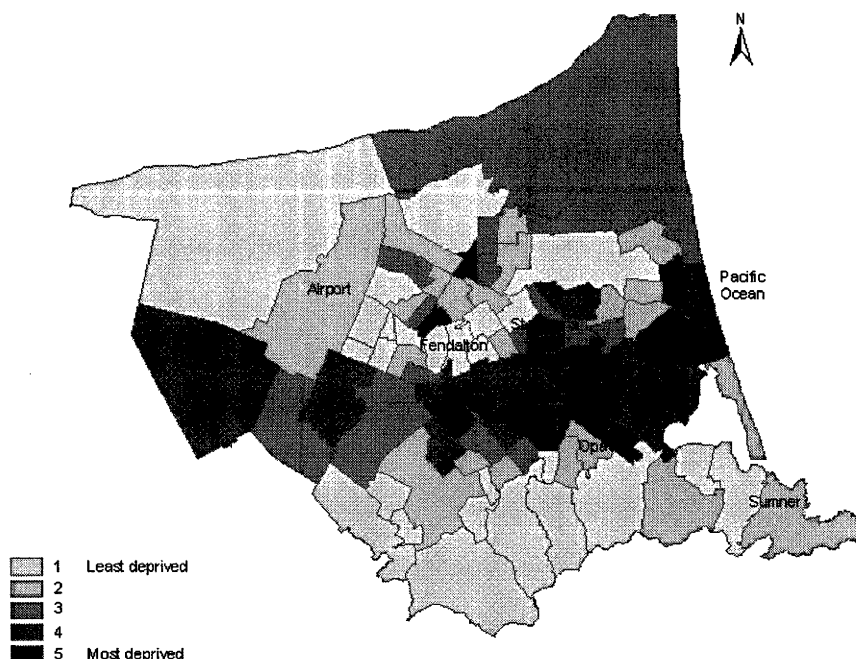


9.5.3 Socioeconomic status

Figure 9-7 shows the spatial distribution of area-level socioeconomic status as measured by the NZDep96 index of deprivation in 1996. NZDep96 quintile 1 represents the least deprived 20% CAUs and quintile 5 represents the most deprived 20% CAUs in New Zealand. There was higher deprivation in the central business area and the surrounding CAUs of central business area except at the CAUs north-west to the central business area.

There was a relationship between socioeconomic status and ethnic groups living in the area. Compared to other CAUs, the most deprived CAUs had higher proportions of Maori and Pacific Islander population (Figures 9-4 and 9-5). The least deprived CAUs at Port Hills in the south had more than 90% of their population from European ethnic groups (Figures 9-3 and 9-7)

Figure 9-7. Socioeconomic status as measured by NZDep96 index of deprivation

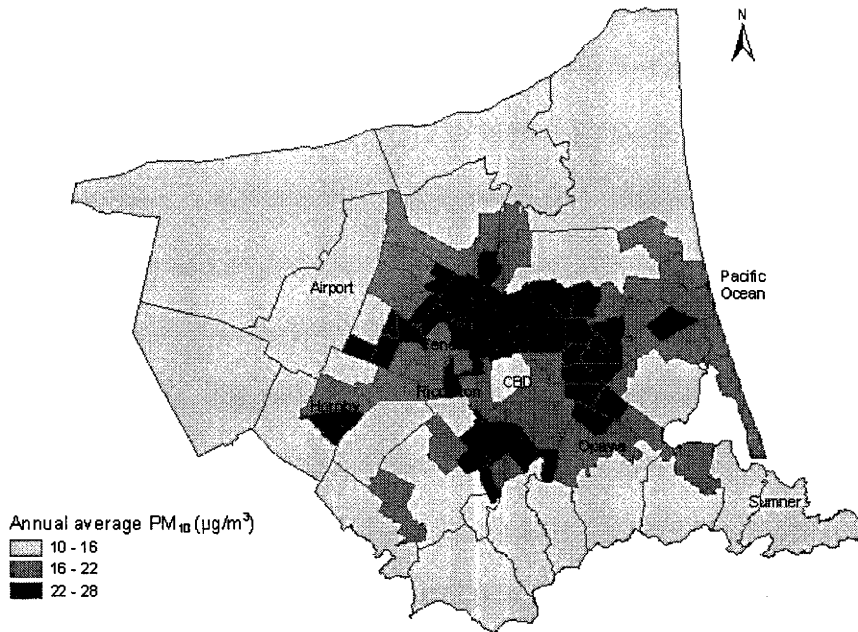


9.6 Air pollution exposure

Spatial distributions of annual average PM₁₀ concentrations for 2001 (Figure 9-8) showed a clear trend of annual PM₁₀ levels, decreasing from the inner parts of Christchurch to the outer parts of Christchurch. Inner city had the highest level of annual PM₁₀ with many CAUs having an annual average of higher than 20 µg/m³, the

Ministry for the Environment guideline level for annual average PM₁₀ (Ministry for the Environment and Ministry of Health, 2002)

Figure 9-8. Spatial distribution of annual average PM₁₀ for 2001



9.7 Methods

Both population and mortality data were aggregated into 3 x 2 x 5 age-sex-ethnicity groups in each CAU as discussed in Section 9.4. Age was categorised into three groups; <15, 15-64 and 65+ years. Sex was categorised into two groups; male and female. Ethnicity was categorised into five groups; European, Maori, Pacific, Asian/Others and “not specified”. Each CAU had one socioeconomic status indicator (NZDep96; 1-10 level) and one annual PM₁₀ exposure level. Data with ethnic group “not specified” were excluded from the analysis. This eliminated 313 (3.2%) non-external deaths, 168 (3.7%) circulatory deaths and 26 (2.5%) respiratory deaths from the analysis. This left 24 (3 x 2 x 4) age-sex-ethnicity groups for each CAU for the analysis.

Logistic regression was used to analyse the association of annual PM₁₀ with mortality controlling for confounders age, sex, ethnicity, socioeconomic status (NZDep96) and the percentage of ever smokers. Poisson regression or Negative Binomial regression is often used to model the count data (i.e. number of deaths in each age-sex-ethnicity group) when the probably of dying in each group (i.e. the probably of dying in age-sex-ethnicity groups) is too small. However, in this case due to very small denominator population in most age-sex-ethnicity groups, the probably of dying in age-sex-ethnicity groups was not small. Therefore, logistic regression was chosen over

Poisson regression or Negative Binomial to analyse how PM₁₀ influences the probability of dying.

The dataset was a grouped data with each row corresponding to an age-sex-ethnic specific group of each CAU. The dataset had the number of deaths and the total number of people for each age-sex-ethnic group of each CAU (i.e. for each row). The probability of dying in this aggregated data was modelled using logistic regression analysis method for grouped data.

Data on the number of current smokers and ever smokers by age, sex and ethnicity were not available for three CAUs; Halswell West, Kennedys Bush and McLeans Island. Data analysis excluded these three CAUs.

Some of the age-sex-ethnicity groups had less number of people than the number of deaths occurred in the group. This anomaly most likely had occurred due to the confidentiality assurance technique used by Statistics New Zealand. All aggregated census data were randomly rounded to base three by Statistics New Zealand in order to protect the confidentiality of the information about individual people and to ensure that no person can be identified from the released data (Statistics New Zealand, 2002). When the data in each cell of a table are randomly rounded to base three, the numbers in each cell become the multiples of three. Estimating the age-sex-ethnicity specific population for non-census years by linear interpolation from the population of two census years could also have contributed to the anomaly. The age-sex-ethnicity groups with the number of deaths greater than population were not included in the analysis. The percentage of deaths in each ethnic group which were excluded from the analysis is shown in Table 9-2.

Table 9-2. Number of deaths (% of deaths) in each ethnic group which were not included in the analysis

Ethnicity	Cause of deaths		
	All non-external	Circulatory	Respiratory
European	0	0	0
Maori	13 (6.2%)	7 (8.1%)	2 (10%)
Pacific Islander	14 (20.0%)	6 (24%)	1 (14.3%)
Asian/Others	13 (14.8%)	7 (19.4%)	2 (28.6%)

The percentage of deaths excluded from the analysis varied across ethnic groups. Asian/Other and Pacific Islanders had higher percentage of deaths excluded from the analysis (Table 9-2). This may have introduced some error in the results. In order to

prevent the necessity of eliminating number of deaths from the analysis, three people were added to all age-sex-ethnicity groups. This made population in each age-sex-ethnicity groups equal or greater than the number of deaths in those groups. The results were compared between the models which had the populations increased by three and the models which had the age-sex-ethnicity groups with the number of deaths greater than population excluded.

Analyses were separately conducted for total non-external deaths, circulatory deaths and respiratory deaths. Models were first fitted only to control for the main effect of confounders. Interaction terms between confounders were then added into the model to test the effect of interaction between confounders and to see how controlling for the interaction effect changes the association between annual PM_{10} and mortality. Interaction terms between confounding variables were retained in the model if adding them improved the model fit and they were statistically significant at the 5% level. Improvements in the model fit were tested using the likelihood ratio test.

The mortality effects of PM_{10} are likely to be stronger in the population aged 65+ years than all age groups combined. In addition to the analysis for the whole population, analyses were also carried out for the population aged 65+ years only. The risks of mortality associated with annual average PM_{10} were compared for the whole population and the population aged 65+ years.

9.8 Results

Out of 106 CAUs in Christchurch, four CAUs (Avon-Heathcote Estuary, Halswell West, Kennedys Bush and McLeans Island) were excluded from the analysis. Avon-Heathcote Estuary did not have any population and Canterbury University did not provide the estimate of annual PM_{10} concentration for this CAU. The other three CAUs did not have data on the number of smokers by age, sex and ethnic groups. This analysis used data from 102 Christchurch CAUs. The following results are based on these 102 Christchurch CAUs and for all age groups.

9.8.1 Descriptive analysis of mortality data

Mortality by cause of death

There were 9828 non-external deaths with an annual average of 2457 non-external deaths in the period from 1996 to 1999 in Christchurch. The total number of non-external deaths reduced to 9515 when the deaths with the ethnic group “not specified” were excluded. Table 9-3 shows annual number of deaths by cause of deaths from 1996

to 1999 after excluding the deaths in the ethnic group “not specified”. About 46% and 11% of non-external deaths were respectively due to circulatory cause and respiratory cause.

Table 9-3. Annual number of deaths by cause of deaths (1996 - 1999)

Year	All non-external cause	Circulatory cause	Respiratory cause
1996	2400	1062	298
1997	2248	1038	244
1998	2415	1136	218
1999	2452	1141	239
Total	9515	4377	999

Mortality by sex

Annual average mortality for the period 1996 to 1999 by sex are presented for various causes of deaths in Table 9-4. This table excluded the deaths with the ethnic group “not specified”. Total Female deaths were slightly higher than total male deaths. About 52% of all non-external deaths were female deaths and the remaining 48% were male deaths. Similarly about 53% of deaths due to circulatory cause were female deaths and 47% were male deaths. For respiratory cause mortality, females made up about 51% and males made up 49% of total deaths. Although the proportion of female deaths was slightly higher than the proportion of male deaths for respiratory mortality, the crude annual average mortality rate was slightly lower for female for respiratory mortality. For all non-external mortality and for circulatory mortality, the crude annual average mortality rates were higher for female.

Table 9-4. Annual average mortality by sex (1996 - 1999)

Sex	All non-external cause		Circulatory cause		Respiratory cause	
	Average Mortality	Crude rate [#]	Average mortality	Crude rate [#]	Average mortality	Crude rate [#]
Male	1137	783	514	354	123	85
Female	1242	796	581	372	127	81

[#]Annual mortality rate per 100,000 population

Mortality by age group

Table 9-5 shows average annual mortality by age group after excluding the deaths in the ethnic group “not specified”. As expected, the 65+ year age group population had both the highest annual average number of deaths and the highest annual mortality rate for all mortality categories analysed. Eighty four percent of non-external deaths occurred in this age group. Similarly, this age group constituted 90% of circulatory cause mortality and 93% of respiratory cause mortality. Those under 15 years of age constituted a very small proportion of deaths, less than 2% for all non-external cause mortality and less than 1% for circulatory mortality and respiratory mortality.

Table 9-5. Annual average mortality by age group (1996 - 1999)

Age group	All non-external cause		Circulatory cause		Respiratory cause	
	Average mortality	Crude rate [#]	Average mortality	Crude rate [#]	Average mortality	Crude rate [#]
< 15 yrs	36	49	0.5	0.9	0.5	0.8
15 – 64 yrs	352	175	109	54	17	8
65+ yrs	1998	4883	985	2406	232	568

[#]Annual mortality rate per 100,000 population

Mortality by ethnicity

Annual average mortality by ethnicity is shown in Table 9-6. The majority of non-external deaths (about 96%) were of European ethnicity. Maori people mortality constituted about 2% of non-external deaths. Each of the ethnic groups Pacific Islanders and Asian/Others constituted less than 1% of non-external deaths. Roughly similar proportions were found for circulatory mortality and respiratory mortality.

Although the majority of deaths occurred in European ethnic group, Pacific Islanders had the highest age-sex standardised mortality rates for non-external deaths and respiratory mortality (Table 9-6). Standardisation was done using direct standardisation method with total Christchurch population (excluding the populations of four CAUs that were not included in the analysis and the population in ethnic group “not specified”) as a standard population. For respiratory mortality, Maori had higher age-sex standardised annual mortality rate than Europeans whereas the age-sex standardised annual mortality rates were higher for Europeans for all non-external mortality and circulatory mortality.

Table 9-6. Annual average mortality by ethnic groups (1996 - 1999)

Ethnicity	All non-external cause		Circulatory cause		Respiratory cause	
	Average mortality	Rate [#]	Average mortality	Rate [#]	Average mortality	Rate [#]
European	2287	797	1058	366	241	83
Maori	53	687	22	298	5	91
Pacific Islander	18	922	6	336	2	107
Asian / Others	22	463	9	221	2	42

[#]Age – sex standardised annual mortality rate per 100,000 population

Mortality by socioeconomic status

Christchurch had a slightly higher proportion of its population living in relatively less deprived areas. More than half of its population were living in the CAUs with NZDep96 deciles from 1 to 5. It had the smallest proportion of its population (about 4%) living in the most deprived areas (NZDep96 decile 10). The fewer number of most deprived CAUs in Christchurch was due to its smaller proportion of Maori and Pacific Islanders population. More than 85% of its population were Europeans and other ethnic groups such as Maori and Pacific Islanders made up a very small proportion. Europeans generally had relatively better socioeconomic status than Maori and Pacific Islanders. The CAUs with higher proportions of Maori and Pacific Islander population were the most deprived (Section 9.5.3).

Although the most deprived area (NZDep96 decile 10) had the lowest annual average number of deaths, the age-sex-ethnicity standardised mortality rate was one of the highest for the CAUs in the most deprived area for non-external mortality, circulatory mortality and respiratory mortality (Table 9-7). The lowest annual average number of deaths in the most deprived area was due to its smallest population size. Table 9-7 shows annual average mortality and age-sex-ethnicity standardised mortality rates by NZDep96 index of deprivation. Standardisation was done using direct standardisation method with total Christchurch population (excluding the populations of four CAUs that were not included in the analysis and the population in ethnic group “not specified”) as a standard population. There was an association between NZDep96 and mortality. The CAUs in relatively less deprived areas (NZDep96 deciles from 1 to 3) had lower age-sex-ethnicity standardised mortality rates whereas the CAUs in more

deprived areas (NZDep96 deciles from 8 to 10) had higher age-sex-ethnicity standardised mortality rates.

Table 9-7. Annual average mortality by socioeconomic status (1996 - 1999)

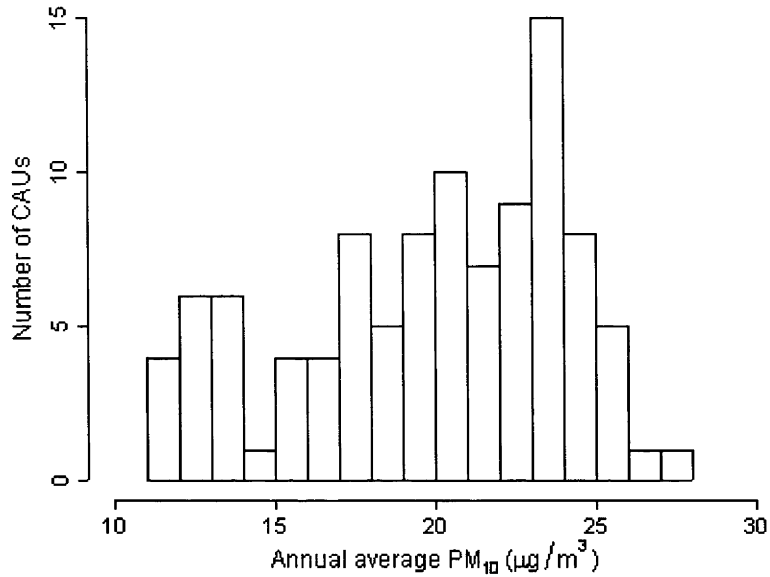
NZDep96	All non-external cause		Circulatory cause		Respiratory cause	
	Average mortality	Rate [#]	Average mortality	Rate [#]	Average mortality	Rate [#]
Decile 1 (Least deprived)	180	588	84	271	16	53
2	285	739	129	339	24	64
3	252	660	120	313	23	60
4	339	926	152	412	39	107
5	194	804	87	360	18	74
6	199	838	93	386	25	104
7	376	790	175	369	39	83
8	183	984	82	438	24	128
9	291	855	135	401	32	97
Decile 10 (Most deprived)	82	888	38	412	9	96

[#]Age-sex-ethnicity standardised annual mortality rate per 100,000 population

9.8.2 Descriptive analysis of PM₁₀ data

The distribution of annual average PM₁₀ was slightly left skewed (Figure 9-9). The mean and median of annual PM₁₀ level were respectively 19.8 µg/m³ (standard deviation of 4.2 µg/m³) and 20.8 µg/m³. Fifty six out of 102 CAUs had annual average PM₁₀ level greater than the Ministry for the Environment ambient air quality guideline level of 20 µg/m³ for annual average PM₁₀ (Ministry for the Environment and Ministry of Health, 2002).

Figure 9-9. Frequency distribution of annual average PM₁₀



9.8.3 Modelling the association between annual average PM₁₀ and mortality

There was a significant positive relationship between mortality and annual average PM₁₀ for all non-external causes of deaths, circulatory deaths and respiratory deaths. All potential confounding variables, which were controlled in the analysis, such as age, sex, ethnicity, socioeconomic status and smoking had statistically significant effect on mortality for all non-external causes of deaths, circulatory deaths and respiratory deaths. Table 9-8 shows the odds ratios for mortality associated with a 10 µg/m³ increase in annual average PM₁₀ for different causes of deaths analysed for all ages.

Table 9-8. Odds ratios (95% CI) for mortality of all ages associated with a 10 µg/m³ increase in annual average PM₁₀

Cause of deaths	Controlling for main effects of confounders only	Controlling for both main effects and interaction effects between confounders
All non-external causes	1.17 (1.10, 1.24)	1.11 (1.04, 1.18)
Circulatory causes	1.18 (1.08, 1.29)	1.13 (1.03, 1.24)
Respiratory causes	1.45 (1.18, 1.76)	1.32 (1.08, 1.61)

In the first stage of analysis, only the main effects of confounders were controlled for. Both main effects and the effects of interaction between confounders were controlled in

the second stage of analysis by adding statistically significant interaction terms between confounders. The odds ratios estimated in the second stage of analysis (controlling for both main and interaction effects) are compared with the odds ratios estimated in the first stage of analysis (controlling for main effects only) in Table 9-8. The odds ratios for mortality were reduced after controlling for interaction effects. The interaction effects of age and sex; age and ethnicity; sex and ethnicity; NZDep96 and age; NZDep96 and sex; age and smoking variable; ethnicity and smoking variable; NZDep96 and smoking variable on non-external cause mortality were statistically significant. For circulatory mortality, the interaction effects of age and sex; age and ethnicity; sex and ethnicity; NZDep96 and age; NZDep96 and sex; NZDep96 and ethnicity; age and smoking variable; NZDep96 and smoking variable were statistically significant. For respiratory mortality, interaction effects of age and sex; age and ethnicity; NZDep96 and age; NZDep96 and sex; NZDep96 and ethnicity; age and smoking variable; ethnicity and smoking variable; NZDep96 and smoking variable were statistically significant.

The effect estimates of annual PM_{10} on non-external cause mortality and circulatory mortality were approximately the same. After controlling for potential confounding variables, a $10 \mu\text{g}/\text{m}^3$ increase in annual PM_{10} was associated with an estimated 11% (95% CI: 4, 18) increase in annual non-external cause mortality and an estimated 13% (95% CI: 3, 24) increase in annual circulatory mortality. Annual average PM_{10} had the strongest effect on annual respiratory mortality compared to others. There was an estimated increase of 32% (95% CI: 8, 61) in annual respiratory mortality per $10 \mu\text{g}/\text{m}^3$ increase in annual PM_{10} .

Table 9-9 compares the odds ratios for mortality associated with a $10 \mu\text{g}/\text{m}^3$ increase annual average PM_{10} for all ages and the population aged 65+ years. Except for circulatory cause of deaths, the odds ratios were higher for the population aged 65+ years showing that the estimated excess risk of mortality due to PM_{10} was higher for the population aged 65+ years compared to the whole population.

Table 9-9. Odds ratios (95% CI) for mortality associated with a 10 $\mu\text{g}/\text{m}^3$ increase in annual average PM_{10}

Cause of deaths	For all ages	65+ years age group
All non-external causes	1.11 (1.04, 1.18)	1.17 (1.09, 1.26)
Circulatory causes	1.13 (1.03, 1.24)	1.13 (1.02, 1.25)
Respiratory causes	1.32 (1.08, 1.61)	1.39 (1.12, 1.73)

The above analysis excluded the age-sex-ethnicity groups with the number of deaths greater than population. In order to eliminate the possibility of errors that might have been introduced due to the exclusion of few deaths in the analysis, populations in each age-sex-ethnicity group was increased by three so that population in each group would be greater or equal to the number of deaths in the same group making it possible to include all age-sex-ethnicity groups in the analysis. Models were refitted with population increased by three in all age-sex-ethnicity groups and the results were compared with the earlier analysis that excluded the age-sex-ethnicity groups which had the number of deaths greater than population (Table 9-10). There was a very small increase in the odds ratios for mortality in the analysis which did not exclude any death. However, the increase was too small to draw any conclusion that excluding deaths in the age-sex-ethnicity groups with the number of deaths greater than population added any error in the results.

Table 9-10. Differences in the results due to excluding few deaths in the analysis

Cause of deaths	Odds ratios (95% CI) for mortality associated with a 10 $\mu\text{g}/\text{m}^3$ increase in annual average PM_{10}	
	Analysis 1	Analysis 2
All non-external causes	1.11 (1.04, 1.18)	1.12 (1.05, 1.19)
Circulatory causes	1.13 (1.03, 1.24)	1.14 (1.04, 1.26)
Respiratory causes	1.32 (1.08, 1.61)	1.35 (1.10, 1.65)

Note: Analysis 1: Excluding deaths in the age-sex-ethnicity groups which had the number of deaths greater than population.

Analysis 2: Increasing the population in each age-sex-ethnicity group by three so that the number of deaths would be greater or equal to population in all age-sex-ethnicity groups. No death was excluded in this analysis.

9.9 Summary

This chapter explored the spatial variation of socio-demographic characteristics and annual average PM₁₀ concentrations and also quantified the association of long-term exposure to PM₁₀ concentrations with annual mortality. Census area unit was chosen as a spatial unit for exploring the spatial variations and for the analysis of the association between PM₁₀ and mortality. The analysis controlled for the confounding effects of age, sex, ethnicity, area-level socioeconomic status and smoking.

There was a distinct spatial pattern of annual average PM₁₀ concentrations within Christchurch with higher levels in the inner parts of the city, which were gradually declining from the inner parts to the outer parts of the city. The mean annual PM₁₀ level was 19.8 µg/m³, with 56 CAUs having annual average PM₁₀ level greater than the Ministry for the Environment ambient air quality guideline level of 20 µg/m³.

An increase in annual average PM₁₀ was significantly associated with an increase in annual mortality. There was an estimated increase of 11% (95% CI: 4, 18) in annual non-external cause mortality, 13% (95% CI: 3, 24) in annual circulatory mortality, and 32% (95% CI: 8, 61) in annual respiratory mortality per 10 µg/m³ increase in annual average PM₁₀. The effect of annual average PM₁₀ on annual mortality was stronger for the population aged 65+ years compared to the whole population.

Chapter 10: Discussion

10.1 Introduction

Air pollution from the burning of solid fuels for domestic heating has been a major environmental concern for several decades in Christchurch. Because of the emissions from domestic heating and local weather conditions, Christchurch experiences very high levels of air pollution especially PM₁₀ in the winter. Twenty-four hour average PM₁₀ concentrations exceeds the Ministry for the Environment ambient air quality guideline of 50 µg/m³, on average, for 30 days in the winter (Canterbury Regional Council, 1997; Ministry for the Environment and Ministry of Health, 2002). There is growing public health concern about air pollution in Christchurch because of its potential health effects. Numerous epidemiological studies have provided evidence of associations of *short-term* air pollution exposure with mortality in various cities including Australian and New Zealand cities (Schwartz, 1993; Ostro *et al.*, 1996; Kelsall *et al.*, 1997; Burnett *et al.*, 1998; Morgan *et al.*, 1998; Ostro *et al.*, 1999; Hales *et al.*, 2000; Hoek *et al.*, 2000; Peters *et al.*, 2000; Simpson *et al.*, 2000). In addition, a few studies have also demonstrated the associations of *long-term* air pollution exposure with mortality (Dockery *et al.*, 1993; Pope *et al.*, 1995; Pope *et al.*, 2002; Nafstad *et al.*, 2004; Scoggins *et al.*, 2004; Filleul *et al.*, 2005; Krewski *et al.*, 2005a; Krewski *et al.*, 2005b).

The main aim of this study is to examine whether or not particulate matter is associated with an increase in mortality in Christchurch. This study analyses the association of particulate matter with mortality at different time scales. The research objectives of this study are:

1. To quantify the effects of short-term exposure to particulate matter on daily mortality.
2. To test whether or not the association between short-term exposure to particulate matter and daily mortality is due to mortality displacement by a few days.
3. To quantify the extended effects of particulate matter on daily mortality using distributed lag models.
4. To explore the shape of the distribution of effects of particulate matter on daily mortality over lag days.
5. To study the association between long-term exposure to particulate matter and annual mortality.

Time series study design was used for research objectives 1 to 4 and ecological cross-sectional study design was used for research objective 5. Chapters 6, 7, 8 and 9 summarised the findings of this study. This chapter discusses the main findings and compares them with the findings of the other studies, with particular reference to any differences in results. Various methodological issues of this research are also discussed in this chapter.

10.2 Short-term effects of air pollution on daily mortality

10.2.1 Analytical methods

One of the major concerns in time series analysis of the association between daily air pollution and mortality is the appropriate method to adequately control for the effects of confounding variables, such as a long-term trend and seasonal variations in daily mortality, and weather variables. Descriptive analyses of daily mortality, weather and air pollution data have shown that daily time series of mortality, weather variables and air pollutants have very strong seasonal patterns with peaks generally occurring during the winter months and troughs occurring during the summer months (Section 4.3.3 in Chapter 4 and Sections 5.2 and 5.4 in Chapter 5). Similar seasonal variations and long-term trends in mortality, weather and air pollution time series will induce correlations among them even if they are not causally related. Although temperature may be responsible for a lot of the seasonal variation in deaths, the long-term trends and seasonal variations in mortality may also be due to unmeasured factors associated with mortality, other than air pollution and weather, which also vary seasonally and have long-term trends over time. Seasonal variations in mortality may occur because of the effect of seasonal infectious disease, such as influenza, and other more general factors such as people spending more time indoors during the winter months (Schwartz *et al.*, 1996). Thus, long-term trends and seasonal variations in mortality need to be adequately controlled for in the analysis of the association of short-term air pollution exposure with daily mortality. In this Christchurch City study, the long-term trends and seasonal variations in daily mortality were adjusted for using the standard statistical methods commonly used in the time series studies of air pollution and health (Schwartz *et al.*, 1996). Various standard diagnostic checks were performed at each step to ensure that the long-term trends and seasonal variations in daily mortality were adequately controlled for.

The long-term trends and seasonal variations in mortality were controlled for using two different methods. In the first method, they were modelled using a linear time

trend and sinusoidal terms. In the second method, they were modelled using a natural cubic spline smooth function of calendar time. In both methods, the long-term variations in daily mortality of more than two months were controlled for leaving only the short-term variations in mortality of less than two months to estimate the association between mortality, air pollution and weather. The European multicity study, the first APHEA project, used harmonic waves (sinusoidal terms) in regression models to control for seasonal variations (Katsouyanni *et al.*, 1996). The US multicity study, the NMMAPS study, used a smooth function of calendar time (Samet *et al.*, 2000c; Dominici *et al.*, 2005) with a smoothing parameter (i.e. number of degrees of freedom) selected based on a priori considerations. The same number of degrees of freedom per year of data as in the NMMAPS study (seven degrees of freedom per year of data) was chosen to create natural cubic splines of calendar time in this Christchurch City study. This would adjust for the long-term variation in daily mortality of approximately more than two months, which would include a long-term trend, seasonal variation and any increase in the number of deaths due to the outbreaks of seasonal infectious diseases such as flu in an epidemic year. However, a very localised spike in the number of deaths, if any, possibly caused by flu outbreaks that lasted only for a few days might not be adequately controlled for.

Another standard method to control for a long-term trend and seasonal variation is to use non-parametric loess smoothing functions of calendar time in the generalised additive models for daily counts of deaths (Anderson *et al.*, 2001; Katsouyanni *et al.*, 2001; Aga *et al.*, 2003).

Besides long-term trends and seasonal variations, the other most likely confounders in the relationship between air pollution and mortality are weather variables. Because of high dependence of local ambient air pollution concentrations on local weather conditions, daily weather data are likely to be highly correlated with daily air pollution data. Temperature plays a major role in emission patterns, thus affecting the ambient air pollutant concentrations. For example, people burn firewood in cold winter temperatures to keep their houses warm. Temperature inversions, which commonly occur in cold weather, create a stable atmospheric condition restricting the dispersion of air pollutants, which results in higher air pollutant concentrations. Furthermore, short-term exposure to extreme temperatures, both hot and cold, and high relative humidity are associated with an increase in daily mortality after controlling for long-term trend and seasonal variation (Huynen *et al.*, 2001; Hajat *et al.*, 2002; O'Neill *et al.*, 2005). Inadequate control for the short-term effect of daily weather variables on

mortality may provide a false evidence of a short-term association between daily air pollution and daily mortality. Such association does not reflect a true causal relationship.

Daily temperature and relative humidity are the two main weather variables that confound the association between daily air pollution and daily mortality, and are usually controlled for in time series analysis of air pollution and mortality. Although it is a standard practice to use temperature and relative humidity as separate variables in the models to control for their confounding effects (Katsouyanni *et al.*, 1996; Samet *et al.*, 2000c), some studies have combined the two variables to construct a single variable such as an apparent temperature that reflects the physiologic effects of temperature and relative humidity, and used it in the model to control for the confounding effects of weather variables (O'Neill, Zanobetti and Schwartz, 2003; O'Neill *et al.*, 2005). In this Christchurch City study, the confounding effects of weather variables were controlled for by including temperature and relative humidity as separate variables in the models.

Two different approaches were used in this study to control for the confounding effects of temperature and relative humidity on daily mortality. In the first method, the relationships of temperature and relative humidity with daily mortality after controlling for a long-term trend and seasonal variations were explored and new weather variables that expressed these relationships were created (Section 6.3.2).

While the first method was based on data, the second method was based on a priori considerations. The second method (Method 2 in Section 6.3.2) adjusted for the confounding effects of weather variables using natural cubic spline smooth functions of weather variables with smoothing parameters same as in the NMMAPS study (Samet *et al.*, 2000c; Dominici *et al.*, 2005).

Despite the uses of two different approaches to adjust for the long-term trends, seasonal variations and the confounding effects of weather variables, the results from both methods were similar. Both methods provided evidence of associations between short-term exposure to air pollutant and daily mortality with similar effect sizes. This strengthens the findings of this study.

The confounding effects of weather variables (i.e. temperature and relative humidity) were adjusted for in the analysis by controlling for the same day effect and the lagged effect of weather variables on daily mortality. The models included the weather variables based on the same day's weather variables and the moving average of the previous three days' weather variables. The same day's weather variable and the moving average of the previous three days' weather variables are likely to be strongly

correlated. When two strongly correlated variables, such as the same day's temperature and the moving average of the previous three days' temperature, are included in the same model, the coefficient estimates will likely be positive for one variable and negative for another making it difficult to interpret the effects of those variables. Thus, the coefficient estimates for the same day's weather variable and the lagged weather variables should not be interpreted independently.

Generalised linear models were fitted for daily counts of deaths. Another common modelling approach for these types of studies is to fit generalised additive models with non-parametric loess smoothing functions of calendar time and weather variables (Burnett *et al.*, 1998; Zanobetti and Schwartz, 2000; Anderson *et al.*, 2001; Braga, Zanobetti and Schwartz, 2001; Aga *et al.*, 2003). A generalised linear model with natural cubic splines is basically a parametric approach for a generalised additive model with smoothing splines such as loess smoothers (Roberts, 2004). It was argued that the generalised linear models with natural cubic splines provide better estimates of air pollution effect and the associated standard errors than the generalised additive models with non-parametric loess smoothing functions (Dominici *et al.*, 2002c). The **gam** function used to fit the generalised additive model in the software SPlus (Version 3.4) was prone to overestimating the risk estimates when the model was fitted with the default convergence criteria and two or more confounding variables were controlled using non-parametric smoothers (Dominici *et al.*, 2002c). It was suggested to fit the model with the **gam** function using stricter convergence criteria instead of using the default convergence criteria (Health Effects Institute, 2003). The risk coefficients from the generalised linear model with parametric nonlinear adjustment (natural spline smoother) were reported to be similar to the risk coefficients from the **gam** with stricter convergence criteria (Dominici *et al.*, 2002c). In addition, the generalised additive models also underestimated the standard errors of the risk coefficients if there were concavity in data (Ramsay, Burnett and Krewski, 2003) up until a new function **gam.exact** was developed to compute the exact standard error of the risk coefficients (McDermott, 2003a; 2003b). The generalised linear models do not have such modelling problems.

The assumption of a no threshold log-linear relationship between daily air pollutant concentrations and daily mortality used in this analysis is comparable with other time series air pollution epidemiological studies (Morgan *et al.*, 1998; Hales *et al.*, 2000; Samet *et al.*, 2000c; Anderson *et al.*, 2001; Aga *et al.*, 2003). A few studies have explored the shape of dose-response relationships of PM₁₀ with mortality and analysed

whether a threshold level exists for mortality effects of PM₁₀. These studies neither found any evidence of a threshold nor any evidence against log-linear dose relationships between PM₁₀ and daily mortality (Daniels *et al.*, 2000; Schwartz and Zanobetti, 2000; Samoli *et al.*, 2005).

10.2.2 Relative risk of daily mortality associated with daily PM₁₀

This study has a major focus on the population aged 65+ years as it is the most vulnerable population group. Studies have reported a higher mortality effect of PM₁₀ for this age group than for younger populations (Gouveia and Fletcher, 2000; Filleul *et al.*, 2004). Preliminary time series analysis of any association between air pollution and mortality in the younger age groups showed no clear association in Christchurch, which may be due to a lack of power to detect statistically significant associations in those age groups. Analysing only the deaths in the older population would reduce the potential “dilution” of any air pollution effect. Because of a very small number of daily deaths (an average of 5.6 deaths per day for non-external deaths) in the population aged 65+ years, data were not analysed for the further sub age groups. For the analysis of non-external mortality, analysis was done for all ages and for the 65+ years age group. For circulatory and respiratory mortality, analysis was conducted for the 65+ years age group only.

Another reason to focus mainly in the population aged 65+ years was the larger proportion of deaths occurring in this age group. More than 80% of non-external cause deaths occurred in this age group. For circulatory and respiratory cause mortality, this age group made up more than 88% of deaths (Section 4.3.2, p.54).

The results of single pollutant models in this study showed positive associations between daily mortality and daily PM₁₀ and CO for non-external mortality of all ages and the 65+ years age group. Statistically significant positive associations between daily non-external mortality and PM₁₀ and CO were observed for up to two days after exposure. There was no evidence of associations between daily mortality and daily NO₂ for any mortality category analysed in Christchurch.

The positive association between daily non-external mortality and PM₁₀ observed in this study is consistent with the results reported by numerous other single city and multicity studies (Morgan *et al.*, 1998; Fairley, 1999; Samet *et al.*, 2000c; Katsouyanni *et al.*, 2001; Fairley, 2003). This study found a 1.3% increase in daily non-external mortality of all ages for each 10 µg/m³ increase in the same day PM₁₀. A review of time series studies of the association between daily mortality and PM₁₀

reported a -0.5% to 2.6% increase in daily non-external mortality for each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} (Levy, Hammitt and Spengler, 2000). The review of air pollution epidemiological studies by the USEPA for its Air Quality Criteria for Particulate Matter reported that the PM_{10} effect size estimates for total non-external mortality were generally in the range of 0.4% to 0.7% for each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} (US Environmental Protection Agency, 2004). A meta-analysis by Dockery and Pope (1994) found a 1% increase in daily non-external mortality for each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} concentrations. Studies reviewed in this meta-analysis reported estimated acute effects of PM_{10} on daily non-external mortality in the range of 0.7% to 1.6% increase for each $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} (Dockery and Pope, 1994). The reviews of acute effects of particulate pollution on daily mortality reported heterogeneity in the results. Although PM_{10} effect size estimates observed in Christchurch were generally within the range of the effect size estimates reported by other studies, they were at the high end.

The slightly larger than average PM_{10} effect size estimate in Christchurch may be due to the air quality monitoring method. An earlier Christchurch study by Hales et al. (2000) using the data from 1988 to 1993 reported a slightly smaller PM_{10} effect estimates (an increase of 1% in daily non-external mortality per $10 \mu\text{g}/\text{m}^3$ increase in PM_{10}) than the findings of the current study (Hales *et al.*, 2000). They used PM_{10} concentrations measured by the beta gauze method. The current study used PM_{10} data monitored by the TEOM method with the temperature of TEOM analyser set at 40°C and analysed for the period from 1988 to 1999. Comparisons of monitored PM_{10} concentrations using two different methods in Chapter 3 showed that the TEOM method underestimates PM_{10} concentrations compared to the beta gauze method. Underestimation of PM_{10} concentrations by the TEOM method has also been reported by a study in Mexico City (O'Neill *et al.*, 2004).

When the analysis was restricted to the population aged 65+ years, the estimates of acute effects of PM_{10} on daily non-external mortality were larger than the effect estimates for the whole population. This suggests that the elderly population had a higher risk of mortality due to PM_{10} exposure than the younger population. Moreover, the preliminary analysis did not show any significant association between PM_{10} and daily deaths in the younger age groups. A larger relative risk of mortality associated with particulate pollution for the population aged 65+ years than for the younger population has been consistently reported by numerous studies (Schwartz and Dockery, 1992a; Schwartz, 1994c; Gouveia and Fletcher, 2000; Filleul *et al.*, 2004). Multicity

studies have reported larger effects of PM₁₀ in the cities with a larger proportion of the population aged 65+ years (Katsouyanni *et al.*, 2001).

For other mortality categories in the population aged 65+ years, this study found that increases in daily circulatory and respiratory mortality were associated with an increase in 1-day lagged PM₁₀ and 2-day lagged PM₁₀ respectively. The significant positive associations between PM₁₀ and daily circulatory mortality and respiratory mortality observed in this study are consistent with the findings of other studies (Zmirou *et al.*, 1998; Ostro *et al.*, 1999). This study found larger relative risks of mortality due to PM₁₀ for respiratory mortality than for circulatory mortality. Studies have generally reported a larger relative risk of respiratory mortality (Zmirou *et al.*, 1998; Fairley, 1999; Ostro *et al.*, 1999; Gouveia and Fletcher, 2000). Although there is growing epidemiologic evidence that the cardiovascular system is affected by PM₁₀, lung is still considered as the primary organ affected by PM₁₀ inhalation (US Environmental Protection Agency, 2004). Experimental studies have shown that exposures to concentrated air particles induce pulmonary inflammation and reduce pulmonary function (Ghio, Kim and Devlin, 2000; Ghio and Devlin, 2001).

The PM₁₀ mortality effect estimates for respiratory mortality had larger standard errors than for other mortality categories analysed, which could be due to a smaller number of average daily deaths from respiratory cause in the population aged 65+ years compared to daily deaths from other causes analysed. The average number of daily deaths from respiratory causes was 0.7 deaths compared to 5.7 from non-external causes and 2.9 from circulatory causes. Because of a fewer number of respiratory and circulatory cause deaths per day, no further analysis was conducted for the specific causes of deaths within the respiratory and circulatory cause categories.

Associations of daily mortality with gaseous air pollutants

This study found a positive association of CO with increase in daily non-external mortality and daily circulatory cause mortality, which is similar to the results reported by other studies (Burnett *et al.*, 1998; Cifuentes *et al.*, 2000). This study did not find any evidence of association between NO₂ and daily mortality for any mortality categories analysed in Christchurch. This contrasts with the results reported by the studies in other cities (Touloumi *et al.*, 1997; Burnett *et al.*, 1998; Cifuentes *et al.*, 2000; Samoli *et al.*, 2006), which have found a positive association between daily mortality and NO₂, but it is consistent with the earlier Christchurch study by Hales *et al.* (2000). They also did not find any association between the oxides of nitrogen (NO_x) and daily mortality in Christchurch (Hales *et al.*, 2000).

Confounding effects of co-pollutants

Most air pollutants are highly correlated with each other, either because of the common source of their emissions or meteorological conditions or both. The primary source of PM₁₀ and CO in Christchurch in the winter months is the emissions from domestic home heating using solid fuels such as wood and coal. The meteorological conditions, such as calm weather and temperature inversions, which frequently occur in the cold winter nights in Christchurch, increase the levels of all air pollutant concentrations in the winter. Depending upon meteorological conditions, all air pollutant concentrations increase or decrease in parallel. Descriptive analysis in Section 5.7 (p.76) shows that PM₁₀, CO and NO₂ concentrations were highly correlated with stronger correlations in the cooler months.

Because of a strong correlation between air pollutants, it can be difficult to interpret the observed association between air pollutant and mortality in single air pollutant models. The air pollutant in single pollutant models may simply be a surrogate for exposure to other air pollutants or the mix of other air pollutants. Even if the air pollutant in single pollutant models is in fact responsible for an increase in mortality, its mortality effect estimates may capture some effects of other air pollutants as well. In other words, co-pollutants confound the relationship. The multi-pollutant models adjust for the confounding effects of co-pollutants.

In multi-pollutant models with PM₁₀ and CO in the model, the statistically significant positive association between CO and mortality disappeared and the association of PM₁₀ with mortality became slightly weaker (statistically significant only at the 10% level; p-value < 0.1). Including CO in multi-pollutant models increased the standard errors of the PM₁₀ effect. This result together with high correlation between PM₁₀ and CO especially in the cooler months (Section 5.7, p.76) suggests that the positive associations of CO with mortality in single pollutant models may be just due to CO acting as a surrogate exposure for particulate matter. The reduction in the strength of the association between PM₁₀ and mortality after adding CO in the model suggests that the effect of PM₁₀ on mortality may not be completely independent of the effect of CO. The results of multi-pollutant models with PM₁₀ and NO₂ suggest that the effect of PM₁₀ on mortality was independent of any NO₂ effect.

Since the effect of CO was not statistically significant in multi-pollutant models with PM₁₀ and CO in the model and including CO in the model increased the standard errors of the PM₁₀ effect, the subsequent analysis of distributed lag modelling and mortality displacement hypothesis did not adjust for CO effect in the modelling.

Another reason for not adjusting the effect of CO in the subsequent analysis was because it was possible that CO was only acting as a surrogate exposure for particulate matter in single pollutant analysis. If this were true, then analysing data with both PM₁₀ and CO in the model would risk putting two variables representing the same risk factor, and thus strongly correlated variables, in a regression model (Zeka, Zanobetti and Schwartz, 2005). As a possible statistical effect, this may result in a negative coefficient estimate for one of the pollutants, suggesting that the pollutant has a protective effect, which in the case of these air pollutants is biologically implausible (Roberts, 2006).

Ground level ozone exposure has been reported to be associated with increased mortality (Simpson *et al.*, 1997; Bell *et al.*, 2004; Penttinen, Tiittanen and Pekkanen, 2004; Levy, Chemerynski and Sarnat, 2005; Parodi *et al.*, 2005; Schwartz, 2005). As ozone levels were not monitored in Christchurch during the study period, this analysis could not examine any effect of ozone on daily mortality or any confounding of the association between PM₁₀ and daily mortality by ozone. In any case, the confounding effects of ozone would be minimal, if any, as ozone concentrations are least likely to be high in the winter when PM₁₀ concentrations are high. Ozone generally peaks in the summer when PM₁₀ concentrations are low (Schwartz, 2000a) and it does not generally have a very strong correlation with PM₁₀ unlike other air pollutants (Morgan, Corbett and Wlodarczyk, 1998). Furthermore, it has been shown that PM₁₀ - mortality effects are not significantly changed after controlling for potential confounding by other co-pollutants including ozone (Samet *et al.*, 2000c; Schwartz, 2000a; Katsouyanni *et al.*, 2001). A case-crossover analysis by Schwartz (2004), that controlled for confounding effects of gaseous pollutants by choosing control days that were matched on each gaseous pollutant in turn, found that PM₁₀ – mortality association was independent of the effects of gaseous pollutants (Schwartz, 2004).

Two different methods were used to adjust for long-term trends, seasonal variations in daily mortality and the confounding effects of weather variables in this study. Comparison of the results from the two methods showed that the results were not dependent upon the method chosen and were consistent with the results reported by the other studies.

10.2.3 Seasonal differences in the effects of PM₁₀ on mortality

In order to analyse the seasonal differences in the effects of PM₁₀ on mortality, the whole year was divided into two seasons; cooler season (May-August) and warmer season (September-April), based on the level of air pollution concentrations and the

primary source of air pollutants. The cooler season corresponds to the winter months with very high PM₁₀ concentrations, when particulate matter from home heating dominates all other sources. The warmer season corresponds to the non-winter months with relatively low PM₁₀ concentrations with home heating contributing very low particles. Although September pollution levels tend to be slightly higher than those in the warmer months, they are still closer to the levels of warmer period than the levels of May to August (Figure 5-6, p.71). Besides, domestic home heating generally contributes lesser in September than in the winter months. Hence, September was grouped into the warmer season.

This study found that the effect estimates of short-term exposure to PM₁₀ on daily mortality varied by season. For non-external deaths of all ages and non-external deaths and respiratory deaths in the population aged 65+ years, the effect estimates were higher in the warmer season (in the non-winter months from September to the following April) than in the cooler season (in the winter months from May to August). For some mortality categories, the estimated percent increases in daily deaths for one interquartile range (11.7 µg/m³) increase in PM₁₀ concentrations were over 10% in the warmer season. For example, for respiratory mortality in the population aged 65+ years, the estimated increase in daily death for one interquartile range increase in the same day PM₁₀ concentrations was 14.5% (95% CI: 4.16, 25.9). This was a very big increase in daily number of deaths and would have larger public health implications than what has been considered until now.

The findings of larger effect estimates in the warmer season are similar to the results of other studies analysing the seasonal difference in the acute effects of PM₁₀ on mortality (Michelozzi *et al.*, 1998). The analysis of 100 US cities in the NMMAPS reported a larger effect size of PM₁₀ in the summer season than in other seasons (Peng *et al.*, 2005). Other multicity studies have reported higher risk of mortality in warmer cities than in cooler cities (Katsouyanni *et al.*, 2001; Aga *et al.*, 2003) suggesting that the effects were stronger in a warmer climate than in a cooler climate. Larger effect estimates in the warmer season than in the cooler season have also been reported for the effects of ozone on daily mortality (Ito, De Leon and Lippmann, 2005).

The stronger effects of PM₁₀ on mortality during the warmer season may be due to the fact that people are likely to have higher personal exposure to ambient air pollution during this season. A study by Sarnat *et al.* (2000) reported a higher correlation between ambient particulate pollution concentrations and personal exposure to particulate pollution in the summer than in the winter (Sarnat, Koutrakis and Suh,

2000). The time spent outdoors varies by season. People tend to spend more time in outdoor activities in the warmer season than in the cooler season and are more likely to keep windows and doors open during the warmer season.

The stronger PM_{10} effect in the warmer season may also be due to the differences in the source of ambient air pollution in the warmer and cooler seasons. Air pollution from one source may be more toxic than another. Sources of ambient air pollution are more likely to be different in the warmer and cooler climate resulting in the differences in the toxicity of air pollution mixtures in the warmer and cooler seasons. In Christchurch, air pollution from domestic heating is the main source of ambient PM_{10} in the winter months (cooler season) with PM_{10} from domestic heating contributing very low to the ambient PM_{10} in the non-winter months (warmer season). PM_{10} from domestic heating contributes about 82% of ambient PM_{10} in the winter months (Scott and Gunatilaka, 2004). Motor vehicle emissions are the main source of PM_{10} in the non-winter months. The stronger PM_{10} effects in the warmer season than in the cooler season may suggest that PM_{10} from motor vehicle emissions is more toxic than PM_{10} from domestic heating. This may be due to the physical and chemical differences in fine particles ($PM_{2.5}$) present in PM_{10} from different sources. Fine particles generated from domestic heating may have different chemical composition than the fine particles generated from motor vehicle emissions have and thus fine particles from one source may be more toxic than another. Laden et al. (2000) reported in their analysis of Harvard Six Cities data that fine particles from motor vehicle emissions had a stronger effect on daily mortality than fine particles from coal combustion (Laden *et al.*, 2000). The proportion of fine particles is also likely to be different in PM_{10} from different sources, which may also explain the differences in mortality risks of PM_{10} from different sources. However, in the absence of source-oriented PM_{10} data in Christchurch, it is not possible to estimate the separate effects of PM_{10} on mortality from motor vehicle emissions and domestic home heating.

Although the relative risks of mortality associated with PM_{10} were lower in the cooler season than in the warmer season, the total number of deaths attributed to PM_{10} could be much larger in the cooler season due to significantly higher PM_{10} concentrations in the cooler season than in the warmer season. PM_{10} concentrations in the cooler season are almost three times the levels of the warmer season (Chapter 5).

10.3 Mortality displacement

The effects of PM₁₀ on mortality net of short-term mortality displacement (Chapter 7) were estimated using the method proposed by Schwartz (2000c) for the assessment of short-term mortality displacement in the association between PM₁₀ and mortality (Schwartz, 2000c). The harvesting hypothesis was tested which assumes that the short-term association between daily PM₁₀ and daily mortality discussed in Section 10.2 is completely due to short-term mortality displacement of the time of deaths of the frail people who are going to die in a few days irrespective of PM₁₀ levels. Under this hypothesis, we would expect an increase in daily mortality to be immediately followed by a drop in daily mortality. There would be no net increase in daily mortality if the data were analysed at time scales of more than a few days. No association between PM₁₀ and mortality would then be observed at time scales of more than a few days. This hypothesis was tested by examining the associations between PM₁₀ and mortality at different mid-term time scales of a few days to a few weeks using the mid-term components of PM₁₀ and mortality data series. Any effect of PM₁₀ observed at these time scales would be an estimate of the effect of PM₁₀ on mortality net of short-term mortality displacement.

The results for non-external mortality of people aged 65+ years suggest that the association between PM₁₀ and mortality cannot be entirely attributed to the short-term mortality displacement. As the mid-term time scales were increased for the analysis by increasing the size of the smoothing window for the mid-term component of data series, the effect size estimates decreased but remained positive confirming that there was an association between PM₁₀ and mortality at all mid-term time scales. The decrease was possibly due to an increase in daily mortality being partially balanced out by a drop in daily mortality that followed soon after the increase. However, the significant positive association between PM₁₀ and mortality of 65+ years at all mid-term time scales confirms that not all the increase in daily mortality was balanced out by the drop in daily mortality and some of the deaths were brought forward by a longer period. This provides evidence that PM₁₀ was associated with an increase in mortality in the population aged 65+ years even after excluding a short-term harvesting effect.

Using different methodologies, many studies have analysed time series data to test if the observed effect of PM₁₀ in time series analysis is entirely due to a short-term harvesting effect (Zeger, Dominici and Samet, 1999; Schwartz, 2000c; 2001; Dominici *et al.*, 2003c; Morgan *et al.*, 2003). These studies have not found results in support of the short-term harvesting hypothesis. Instead, they have reported a larger

effect size at the longer mid-term time scales. However, in Christchurch, a lower effect size was found at the longer mid-term time scales. Although there is not enough statistical evidence to suggest that the effect size is significantly lower at the longer mid-term time scales than at the shorter time scales, the lower effect size at the longer mid-term time scales suggests the presence of some short-term harvesting effect.

No association between PM_{10} and mortality was observed in the analysis using the mid-term components generated with a 60-day smoothing window for non-external mortality of all ages. This indicates that the association between PM_{10} and non-external mortality of all ages may be completely due to short-term mortality displacement. However, the positive associations observed between PM_{10} and mortality for up to 45-day smooth windows suggest that some of the deaths were brought forward by more than just a few days, possibly by a few weeks or months. Comparison between the results for non-external mortality of 65+ years and non-external mortality of all ages suggests that most of the short-term harvesting effect of PM_{10} on non-external mortality of all ages is specific to the population aged less than 65 years. In this age group, it is more likely that PM_{10} only affects the frail individuals without affecting the healthy population resulting in the partial short-term harvesting phenomenon.

For circulatory causes of death, the effect size was slightly reduced when a 30-day smoothing window was used for mid-term components suggesting that some circulatory deaths may be brought forward only by a few weeks. Increasing the averaging period for mid-term components, the effect size increased steadily providing evidence of a larger effect size when the short-term harvesting effect was excluded. Schwartz (2000c) also reported a larger effect size of $PM_{2.5}$ for the deaths from ischemic heart disease in Boston when the longer averaging period was used for the mid-term components (Schwartz, 2000c). Although the time series studies can not estimate the effect of long-term exposure to PM_{10} on mortality like cohort studies (Dockery *et al.*, 1993; Pope *et al.*, 2004), the larger effect size at longer mid-term time scale may be reflecting the effect of long-term exposure to PM_{10} . This may also be due to the reason that PM_{10} increases the number of frail individuals by affecting the healthy people, which results in the higher number of deaths after a few weeks or months and thus increasing the effect size when a longer averaging period for mid-term component was used (Schwartz, 2000c).

The reason for the statistically significant negative associations between the mid-term components of PM_{10} and respiratory mortality of 65+ years (Figure 7-6, p.147), when the longer mid-scale averaging periods was used, is unclear. One possible

explanation for this observation could be the small number of daily respiratory deaths in Christchurch. The average number of daily deaths from respiratory cause was 0.7 deaths per day. This analysis decomposed daily time series data of respiratory mortality of 65+ years into three different time series data that vary at different timescales: longer-term, mid-term and the shortest-term. Because of very low numbers of daily deaths in the original data series, perhaps there was not enough variation in the decomposed data series. We would generally expect non-significant associations in such cases. Although it is unclear, the negative associations observed in this study could also have been occurred because of this. This may suggest that it is not easy to analyse the short-term harvesting hypothesis in the association between daily mortality and air pollution with the method of decomposing data series into different components, when the number of daily deaths is very small.

In order to maintain comparability with daily time series analysis of PM_{10} and mortality in Chapter 6, the same weather model as in Chapter 6 was used to test the harvesting hypothesis. The same weather variables, with their daily time series data replaced by their respective mid-term time scale components, were used in the models. The same numbers of degrees of freedom as in the weather model in Chapter 6 were used for natural cubic spline smooth functions of weather variables.

As Schwartz (2001) has noted, the method used in this analysis to study mortality displacement has certain limitations. Because of the necessity to control for seasonal variation in time series study design, this analysis and other studies that used time series approach to analyse short-term harvesting (Zeger, Dominici and Samet, 1999; Dominici *et al.*, 2003c) can not examine short-term harvesting beyond 3-4 months (Schwartz, 2001). In addition, this approach can not tell exactly by how many days or months, deaths are brought forward by air pollutant exposure. It can only indicate that exposure to air pollutants bring deaths forward by a nontrivial amount of time (Schwartz, 2001).

10.4 Distributed lag models

Polynomial distributed lag models were used to estimate the cumulative effects of PM_{10} of the last 40 days on daily mortality and to examine the pattern of mortality risks over the 40 day period (Chapter 8). Several studies have analysed the cumulative effects of PM_{10} of the last 40 days on daily mortality and examined the shape of the distribution of PM_{10} effects over the same time period (Zanobetti *et al.*, 2002; Zanobetti *et al.*, 2003; Goodman, Dockery and Clancy, 2004). Use of lag 40 days to estimate the extended

effects of PM₁₀ on mortality in this study allowed me to compare the Christchurch results with the results of the other studies conducted in various cities. The analysis of the lag structure of mortality risk for more than a few weeks lag is also useful to analyse if there is any short-term mortality displacement.

Although the models allowed analysing the effects of PM₁₀ of the last 40 days, it was not assumed that there were mortality effects at all 40 lags. If there were no effects at high lags, then the model would show that by giving fitted polynomials with small values at high lags, which means the estimated effects of PM₁₀ on mortality at high lags would be very small to almost zero.

The results show that the risks of daily deaths due to PM₁₀ were the highest on the day of exposure and a few days after exposure. The risks were reduced but stayed statistically significant for several weeks after exposure. For the population aged 65+ years, there was a significant risk of non-external mortality due to daily PM₁₀ for up to four weeks after exposure. This explains why the model with a single day PM₁₀ underestimates the overall effect of PM₁₀ on mortality. Significant risks of mortality for several days after exposure have been reported in several studies. However, most studies found statistically significant risks for a lesser number of days than this study. Zanobetti et al. (2002) reported that the effect of PM₁₀ on daily non-external deaths of all ages decreased to almost zero with a lag of 10 days (Zanobetti *et al.*, 2002). In Dublin, significantly increased risks of non-external deaths of all ages associated with black smoke were found for three days after exposure when the extended effects of both minimum temperature and black smoke were estimated simultaneously through 40 days with polynomial distributed lag models (Goodman, Dockery and Clancy, 2004).

For the population aged 65+ years, the lag structures of mortality risks for respiratory mortality and circulatory mortality were similar to the lag structure of mortality risks of non-external mortality. Although there are some minor differences in the pattern of mortality risks over several days reported by different studies, the results of this Christchurch study are consistent with the basic findings of other studies that the adverse effects of particulate pollution on mortality persist for several weeks (Zanobetti *et al.*, 2002; Goodman, Dockery and Clancy, 2004). The similar adverse effect of air pollution was also observed during the 1952 London smog episode (Anderson, 1999). Toxicological studies also suggest that exposure to particulate matter may have an effect for several subsequent days. Lay et al. (1999) reported that particles instilled in the lung induced an inflammation that took up to 4 days after exposure to resolve (Lay *et al.*, 1999). Respiratory disease such as pneumonia and other chronic diseases progress very

slowly and thus the longer lagged effects of PM₁₀ can be expected for the deaths from these causes (Zanobetti *et al.*, 2003).

Several time series studies have reported an association between increase in daily PM₁₀ concentrations and increase in hospital admissions for different diseases (Morgan, Corbett and Wlodarczyk, 1998) (Ponce de Leon *et al.*, 1996; Schouten, Vonk and de Graaf, 1996). Some of these people, who became sick because of air pollution, may not recover and may die a few weeks after exposure. This may explain some of the delayed effects of PM₁₀ on mortality. People becoming sick and languishing in hospital for a few weeks before dying could be the reason for the small secondary peak in the distribution of mortality risks at around 20 days (Figures 8-2, p.162) after exposure.

The lag structure of mortality risks is also useful to ascertain if there is any short-term mortality displacement. The significant risk of increased mortality for up to four weeks after exposure confirms that the association between PM₁₀ and mortality was not due to short-term harvesting. If it was completely due to short-term harvesting, the increase in the number of daily deaths during the exposure and soon after exposure would be counterbalanced by a decrease in the number of daily deaths on subsequent days. If this were the case, analysis of lag structure in the relationship between PM₁₀ and mortality would show that positive associations in the first few days after exposure would be followed by negative associations at some longer lags. A few studies have explored the mortality displacement hypothesis using distributed lag models and have found no evidence of short-term mortality displacement for non-external mortality, cardiovascular mortality and respiratory mortality (Zanobetti *et al.*, 2000; Zanobetti *et al.*, 2002; Zanobetti *et al.*, 2003). A study in Dublin, however, reported a non-significant negative risk for non-external mortality between one and two weeks after exposure to black smoke suggesting possible short-term harvesting (Goodman, Dockery and Clancy, 2004).

Comparison of the cumulative effect of PM₁₀ of the last 41 days (including the same day PM₁₀) to the effect of a single day PM₁₀ showed that the effect was much lower for a single day PM₁₀. This is consistent with the findings of other studies. Studies focussing on the single day effect or the effect on a couple of days after exposure (Schwartz, 2000a; Katsouyanni *et al.*, 2001) consistently reported a substantially smaller effect size than analysis of the effects over a longer period after exposure (Schwartz, 2000b; Braga, Zanobetti and Schwartz, 2001; Zanobetti *et al.*, 2002; Zanobetti *et al.*, 2003; Goodman, Dockery and Clancy, 2004).

The increase in effect size, when the effects spread over several days were considered, was much larger in this study compared to the other overseas studies. When only a single day PM₁₀ effect was considered, a 10 µg/m³ increase in daily PM₁₀ was associated with a 1.55% (95%CI: 0.6, 2.5) increase in daily non-external mortality of 65+ years. When the effect was summed over 41 days (including the effect on the same day), there was an estimated total increase of 14.9% (95%CI: 6.99, 23.4) for each 10 µg/m³ increase in daily PM₁₀ for non-external mortality of people aged 65+ years. Schwartz (2000b) reported that the estimated total effect of daily PM₁₀ on non-external mortality of people aged 65+ years for five days follow up period was about double the effect of PM₁₀ on same day mortality (Schwartz, 2000b). Goodman et al. (2004) reported that the total estimated effect of black smoke on all non-external cause mortality in the subsequent 41 days (including on the same day) was almost three times the effect of a 3-day moving average of daily black smoke (Goodman, Dockery and Clancy, 2004). Many studies have, however, reported heterogeneity in the results (Schwartz, 2000b; Zanobetti *et al.*, 2002), which could be due to the differences in population structure, and PM₁₀ sources of study cities.

Similar to PM₁₀ associated risks of all cause non-external mortality, the cumulative effects of PM₁₀ of the last 41 days (including the same day PM₁₀) were much larger for circulatory mortality and respiratory mortality compared to the effects of a single day PM₁₀. For circulatory mortality in the population aged 65+ years, the total effect of each 10 µg/m³ increase in PM₁₀ over 0 to 40 days after exposure was a 18.7% (95% CI: 7.31, 31.3) increase which was about 10 times the estimated effect of the same increase in 1-day lagged PM₁₀. For respiratory mortality in the population aged 65+ years, the cumulative effect of each 10 µg/m³ increase in PM₁₀ over 0 to 40 days was 9 times the estimated effect of the same increase in 2-day lagged PM₁₀. The PM₁₀ mortality effect size estimates at different time scales are compared in Table 10-1 in Section 10.6. This analysis, together with the results of other studies, confirms that the estimates of the increased risk of mortality associated with PM₁₀ exposure relying on the same day or a couple of days' exposure underestimate the overall mortality risk associated with daily PM₁₀ concentrations. Thus, distributed lag models should be used to estimate the overall effect of daily PM₁₀ on mortality.

The total mortality effects of PM₁₀ were similar under different distributed lag models such as 4th degree polynomial, cubic polynomial, quadratic polynomial and unconstrained. This confirms that the risks of mortality are not sensitive to the choice of distribution lag model fitted to the data.

Various sensitivity analyses were carried out to check whether the accumulated effects of PM₁₀ were sensitive to the adjustment of confounders like temperature, relative humidity and seasonal variations. The sensitivity of the effect of PM₁₀ on mortality to the adjustment of confounders has been recognised as an important area of research in time series epidemiological studies of air pollution and mortality (Health Effects Institute, 2003). The present study shows that the total effects of PM₁₀ were not sensitive to the number of degrees of freedom used to create the natural cubic splines of temperature and relative humidity. Lack of sensitivity of the estimated total effect of PM₁₀ on mortality to the choice of smoothing parameter for weather variables and the parameters of the distributed lag model strengthens the findings of this study.

However, the PM₁₀ mortality effect estimates varied considerably when the number of degrees of freedom for the smooth function of time was varied to control for long-term trend and seasonal variation. In the original analysis, seven degrees of freedom per year of data was used. Sensitivity analyses were performed using three to ten degrees of freedom per year of data. Using fewer degrees of freedom in the model resulted in lower estimates of PM₁₀ mortality effect. The effect estimates stabilised in the models that used seven or more degrees of freedom per year of data for the natural cubic spline smooth function of time variable. Using more than seven degrees of freedom per year of data did not change the effect estimates but provided more imprecise estimates with wider confidence intervals. A simulation study conducted by Roberts (2005) has shown that the use of too many degrees of freedom for controlling seasonal variation did not create a major problem in the effect estimates. The study reported that the effect estimates were similar to those obtained from using the correct number of degrees of freedom. However, the use of too few degrees of freedom sometimes provided effect estimates substantially larger than those obtained from using the correct number of degrees of freedom (Roberts, 2005). It is unclear why the estimates of PM₁₀ mortality effect in Christchurch were lower when few degrees of freedom were used to control for seasonal variation. Since the effect estimates were stabilised after the use of seven or more degrees of freedom, controlling for seasonal variation with seven degrees of freedom per year of data seemed adequate and appropriate in this analysis. Moreover, the use of natural cubic splines of the time variable with seven degrees of freedom per year of data adjusts for the long-term variation of approximately more than a two-months period so that only the information from time scales less than two months is used to estimate the association between mortality, air pollution and weather.

10.5 Association between annual exposure to PM₁₀ and annual mortality

An ecological cross-sectional study design was used to analyse the association of long-term exposure to PM₁₀ with mortality. The cross-sectional study design associates spatial variation in mortality with spatial variation in air pollutant concentration. This requires controlling for the confounding effects of spatially varying variables such as population structure with respect to age, sex, ethnicity, socioeconomic status etc.

This study found a positive association between annual average PM₁₀ and annual mortality after controlling for age, sex, ethnicity, socioeconomic status and smoking across CAUs in Christchurch. Long-term associations between air pollution and mortality have been reported by both cross-sectional studies (Chappie and Lave, 1982; Ozkaynak and Thurston, 1987) and prospective cohort studies (Dockery *et al.*, 1993; Pope *et al.*, 2002; Finkelstein *et al.*, 2003; Krewski *et al.*, 2005c). Comparison between the relative risks of mortality from the acute effect study (Chapter 6) and the long-term study (Chapter 9) shows that the relative risk of annual mortality associated with annual average PM₁₀ was larger than the relative risk of acute mortality associated with daily PM₁₀. The PM₁₀ mortality effect size estimates at different time scales are compared in Table 10-1 (Section 10.6). This observation is in agreement with the results reported by the other studies. The prospective cohort studies (Dockery *et al.*, 1993; Pope *et al.*, 2002) have reported larger relative risks of mortality than the relative risks of mortality reported by the acute effect studies using time series method (Schwartz, 2000a; Katsouyanni *et al.*, 2001). While time series studies, based on daily data, are designed to estimate the acute effects, the long-term studies estimate a combination of both acute and chronic effects (Dominici, Sheppard and Clyde, 2003) and as such, the larger relative risk in the long-term studies is reflecting any chronic effect of PM₁₀ on mortality.

There is consistency between the results of time series studies discussed in Sections 10.2.2 and 10.4 and this ecological study. Both studies found larger relative risk of mortality for the population aged 65+ years than for whole population. This suggests that the population aged 65+ years is more vulnerable than the younger population. A larger relative risk for respiratory cause mortality than for other causes analysed in the population aged 65+ years was found in both studies.

In principle, cross-sectional studies must adjust for a wide range of variables that may affect mortality rates. However, given the large number of individual and environmental factors that can affect mortality, controlling for all confounders is not

possible in this type of research. This study has controlled for age, sex, ethnicity, socioeconomic status (as measured by the NZDep96 index of deprivation) and smoking but lacked information on other risk factors such as diet and lifestyle factors. Controlling for socioeconomic status probably has mitigated the confounding effects of some variables to some extent. There is a strong socioeconomic gradient in obesity, diet and physical activity in New Zealand. Obesity is significantly more common among people in the most disadvantaged occupation class who are most likely to be in the lowest socioeconomic group. Occupational class is strongly related to socioeconomic status in New Zealand. People in the lower occupational class also have higher intake of saturated fat (Howden-Chapman and Tobias, 2000). Although the use of socioeconomic status in the analysis is likely to control for any confounding effect of some risk factors, there is still a possibility of further residual confounding by the effects of other unmeasured risk factors.

Like all ecological cross-sectional studies, this study used area based measure for some confounding variables. For example, the socioeconomic status variable, the NZDep96 index of deprivation, is a CAU based measure and the analysis assumes that all resident of a deprived CAU are deprived. The smoking variable is based on age-sex-ethnicity specific group within each CAU. The analysis controlled for smoking by creating a smoking variable that represented the percentage of ever smokers in each age-sex-ethnicity stratum in each CAU. The analysis used aggregated data instead of individual level data. Thus, making any inferences about individuals based on this aggregated level data may result in the well known error termed the “ecological fallacy” (US Environmental Protection Agency, 1996). Prospective cohort studies are less subject to confounding by community-level factors as they use individual level data. Because of their ability to control for individual risk factors, they are better than cross-sectional studies. However, since air pollution is population-wide exposure, ecological cross-sectional analyses can also provide important information.

The ecological cross-sectional study used mortality data from 1996 to 1999, which was the subset of the mortality data analysed in the first part of this study. The time series study in the first part of this study analysed daily data from June 1988 to December 1999.

Annual change in population structure with respect to age, sex and ethnicity was adjusted for in the analysis by using the estimated annual population based on the 1996 and 2001 censuses. There were considerable changes in population structure in some CAUs between the two censuses. Since no smoking data was collected in the 2001

Census, percentages of ever smokers were estimated based on the 1996 Census data. This may have resulted in some errors in smoking data. The study used the spatial annual average PM₁₀ concentrations estimated for the year 2001. The main assumption in this study was that between-year variations in spatial distribution of annual PM₁₀ concentrations were negligible in Christchurch. The long-term trend in annual average PM₁₀ concentrations monitored at the St. Albans monitoring site did not show any specific trend (Figure 5-7(A), p.74). Thus, there is no reason to believe that the spatial pattern in annual average PM₁₀ changed between years and hence the assumption is reasonable. However, the possibility of misclassification of exposure can not be excluded.

10.6 Comparison of the effect estimates at different time scale

The effect estimates of PM₁₀ on mortality for different exposure periods are compared in Table 10-1. The effects of short-term exposure to daily PM₁₀ are the acute effects of single day lagged PM₁₀ for the lag that had the largest effect. These are the estimates from the time series study (Chapter 6). The effects of PM₁₀ at medium term are the cumulative effects of PM₁₀ summed over 0 (the day of exposure) to 40 days after exposure. These are the estimates from the polynomial distributed lag models (Chapter 8). The long-term effects are the effects of annual average PM₁₀ on annual average mortality estimated from the ecological cross-sectional study (Chapter 9).

Table 10-1. Comparison of the effect estimates of PM₁₀ for different exposure period. Percentage increase (95% CI) in deaths for each 10 µg/m³ increase in PM₁₀ in the population aged 65+ years

Duration of effect measurement	All non-external Causes	Circulatory causes	Respiratory causes
Short-term [#] (acute effect)	1.5 (0.6, 2.5)	1.8 (0.5, 3.1)	2.9 (0.5, 5.40)
Medium term (distributed lag model)	14.9 (6.9, 23.4)	18.7 (7.3, 31.3)	26.9 (4.5, 54.1)
Long-term (effect of annual exposure)	17 (9, 26)	13 (2, 25)	39 (12, 73)

[#]Note: The acute effects of single day lagged PM₁₀ for the lag that had the strongest effect. For non-external cause mortality, the effects were of the same day PM₁₀. For circulatory cause mortality and respiratory mortality, they were the effects of 1-day lagged PM₁₀ and 2-day lagged PM₁₀ respectively

The finding of an increase in the mortality effect estimates of PM₁₀ with an increase in the duration of the exposure (Table 10-1) confirms the results reported by other studies. The medium term studies using distributed lag models (Schwartz, 2000b; Braga, Zanobetti and Schwartz, 2001; Zanobetti *et al.*, 2002; Zanobetti *et al.*, 2003; Goodman, Dockery and Clancy, 2004) have consistently reported substantially larger effect size than the acute effect studies focussing on the single day effect or the effect on a couple of days after exposure (Schwartz, 2000a; Katsouyanni *et al.*, 2001). The cohort studies analysing the effects of long-term air pollution exposure on mortality (Dockery *et al.*, 1993; Pope *et al.*, 2002; Nafstad *et al.*, 2004) have reported much larger relative risk of mortality than the studies of the short-term and medium term effects.

The acute effect studies only evaluate an increased risk of mortality due to an increase in air pollution over very short intervals of time usually one day to a few days. The medium term effect studies extend the acute effect studies to examine cumulative effects of daily air pollution over several weeks. Both study designs use time series methods. The larger risk of mortality due to PM₁₀ in the medium term effect studies compared to the acute effect studies confirms that the increased risk of mortality due to PM₁₀ exposure is not restricted to a few days. The risk is actually distributed over several weeks.

Because of the study design, time series studies are unable to analyse whether long-term exposure to higher levels of air pollution increase long-term mortality rates (McMichael *et al.*, 1998). They only provide the evidence of the short-term association between daily air pollution and daily mortality, which could be due to short-term mortality displacement. The effects estimated by the long-term studies are not susceptible to short-term mortality displacement. The positive associations of long-term exposure to PM₁₀ and mortality observed in this study show that the association is not just due to short-term mortality displacement. It confirms the findings of other studies that exposure to PM₁₀ shortens life by a significant amount of time. Although the acute effect studies have their own role in demonstrating the association between daily mortality and air pollution, because of their limitation to estimate the chronic effects of air pollution and uncertainty about short-term mortality displacement due to acute exposure, the effect estimates from the acute effects studies are not appropriate to estimate the total impact of air pollution on mortality (McMichael *et al.*, 1998). In order to estimate the number of deaths attributed to air pollution and for other health effect assessment of air pollution, one needs to use more suitable effect estimates from the

long-term exposure studies, which can provide the combined estimates of both acute and chronic effects.

10.7 Air pollution exposure data

10.7.1 Time series studies

One of the major issues in studying the adverse health effects of air pollution is the measurement error in the assessment of exposure. This study, like other time series studies of the association between air pollution and mortality, has used air pollution exposure data from outdoor air pollution monitoring stations rather than personal exposure monitors. Exposure assessment studies have reported differences between outdoor air pollutant concentrations and the readings from personal exposure monitors and have argued that air pollutant concentrations monitored at outdoor monitoring stations do not adequately represent personal exposure (Lioy *et al.*, 1990; Ozkaynak *et al.*, 1996). Schwartz (2000a) concluded that personal exposure monitors measure exposure to particulate matter of all sources (ambient plus indoor generated) including environmental tobacco smoke and cooking aerosols. Thus, outdoor concentrations are not likely to be highly correlated with the readings from personal exposure monitors. However, if the correlation of ambient concentrations with personal exposure to particulate matter of ambient origin only is analysed, they are more likely to be closely related (Schwartz, 2000a). This has been confirmed by others (Janssen *et al.*, 1998; Mage *et al.*, 1999). Janssen *et al.* (1998) has reported that the correlations between personal exposure and outdoor concentrations was much higher when exposure to environmental tobacco smoke was excluded (Janssen *et al.*, 1998). Mage *et al.* (1999) has further reported that if there is any association between indoor generated PM₁₀ and mortality, daily variation in mortality related to daily variation in indoor PM₁₀ is uncorrelated with daily variation in ambient PM₁₀ concentrations. Thus, indoor generated PM₁₀ is less likely to confound the association of daily mortality with ambient PM₁₀ concentrations (Mage *et al.*, 1999). Given that the main aim of this study was to quantify the association between ambient PM₁₀ concentrations and mortality, the use of PM₁₀ concentrations measured at an outdoor monitoring station as surrogates for personal exposure to PM₁₀ of outdoor origin would not create any major bias in the estimates of the mortality effects of ambient PM₁₀.

Ambient air pollutant concentrations at the monitoring station at St. Albans in central Christchurch were used as air pollution exposure in this study. Environment Canterbury monitored air pollutant concentrations at a few other monitoring stations at

different locations within Christchurch but only for a short period of time during the study period (Environet Limited, 2003). Only at St. Albans, monitoring was continued for the whole study period from June 1988 to December 1999. As data were only available for a relatively small period of time at the monitoring stations other than at St. Albans, monitored data from other stations were not used in this study.

The monitoring station at St. Albans is Christchurch's primary air quality monitoring site (Aberkane, 2000). It is centrally located in Christchurch. Because of its location and the flat topography of Christchurch, the measurements at this site are likely to provide a good representation of ambient air quality in much of Christchurch. It has also been suggested that PM_{10} concentrations are homogenous over Christchurch because of relatively even mixing of ambient particulate matter across the Christchurch airshed over a 24-hour period (Foster, 1996). However, the cross-sectional study showed that annual average PM_{10} concentrations varied across Christchurch CAUs with higher levels in the inner parts of the city and the levels gradually decreasing from the inner parts to the outer parts of the city (Section 9.6, p.179).

For time series studies, it is the relative changes in exposure from one day to the next day that matters rather than the absolute exposure. The relative changes from one day to the next day in air pollution at the monitoring station may adequately represent the relative changes in exposure for the whole city (US Environmental Protection Agency, 1996). Thus, it is reasonable to use ambient air pollutant concentrations monitored at a single station at St. Albans as exposure for the whole of Christchurch in this study.

The missing hourly air pollutant concentrations were estimated using regression models based on hourly weather variables and time variables like hour of day for this study (Shrestha, Kjellstrom and Metcalf, 2002). These regression models predicted observed hourly air pollutant levels reasonably well except for some high hourly concentration levels, which were underestimated. This study used 24-hour averages of hourly concentrations instead of individual hourly concentrations as an exposure variable. Although some possibly high levels of hourly concentrations, which were missing, may have been underestimated, the measurement error it may cause in 24-hour average concentrations is likely to be small. However, the possibility of bias in the estimates of mortality effects of PM_{10} resulting from the possible underestimation of missing high levels of hourly concentrations can not be excluded. In addition, the estimates of the missing air pollutant concentrations from the regression models for the years when a high proportion of data is missing may not be as reliable as for the other

years when only a small proportion of data is missing. This may also introduce some bias in the estimates of mortality effects of PM₁₀.

10.7.2 Ecological cross-sectional study

Unlike time series analysis (Chapters 6, 7 and 8), ecological cross-sectional analysis (Chapter 9) compares mortality rates between geographic areas. This study used census area units as small scale geographic areas for comparison. This requires spatial variation in air pollution exposure rather than temporal variation and as such ambient air pollutant concentrations monitored at a single monitoring station at St. Albans will not be sufficient measures of exposure for this analysis. The annual average PM₁₀ exposure levels for each CAU were derived from the simulated spatially distributed PM₁₀ concentrations over Christchurch airshed using an urban airshed model (Fisher *et al.*, 2007). The simulated concentrations from the urban airshed model are likely to represent an average condition rather than an extreme condition (Scoggins, 2003) and thus they may not represent true daily variation in air pollutant concentration but their long-term average would represent true long-term average PM₁₀ exposure. These PM₁₀ exposure estimates may not be suitable for acute effect studies where we correlate daily variation in mortality with daily air pollutant concentrations but are appropriate measures of exposure for long-term studies such as this where our interest of exposure is a long-term average like annual average PM₁₀.

The assumption that people residing in a CAU are exposed to an ambient PM₁₀ concentrations estimated for that CAU is consistent with the assumption made in other studies that ambient air pollutant concentrations can be measured at place of residence (Dockery *et al.*, 1996). While some studies have argued that exposure to an air pollutant not only depends upon a person's usual residential location but also on regular movement across diverse environments with varying levels of air pollutant concentrations such as the person's workplace and the chosen mode of transport (Kingham *et al.*, 1998), others concluded that the residential location is the most useful measure of exposure to ambient air pollution particularly for long-term average exposure (Huang and Batterman, 2000; Kunzli and Tager, 2000). The lack of time-activity patterns and the use of PM₁₀ concentrations at the residential location as exposure measure may have introduced some misclassification of exposure.

This study has also assumed that there was a uniform distribution of annual average PM₁₀ concentrations within a CAU. Given the smaller size of CAUs in

Christchurch city, this assumption is plausible. However, this assumption may still introduce some errors in exposure assessment.

10.8 Population and mortality data

This study obtained population data from the Census of Population and Dwellings for census years and estimated population for non-census years by linear interpolation from the populations of two census years, which is the most commonly used method to estimate the population size for non-census years (Marshall, Scragg and Bourke, 1988).

Mortality data were extracted from a national mortality database. The causes of deaths were coded according to the International Classification of Disease. Although the causes of death in the database are unlikely to be completely accurate, any misclassification of cause of deaths is very unlikely to be related with air pollutant concentrations and thus the association between PM₁₀ and mortality observed in this study cannot be explained by this (Hales *et al.*, 2000). Furthermore, analyses were conducted for broad mortality categories, such as all respiratory cause deaths and all circulatory cause deaths, instead of specific causes of deaths within these categories. This also reduces errors due to misclassification of cause of deaths.

Causes of deaths were identified based only on the primary underlying causes of deaths. Secondary and other contributing causes were not considered to group the deaths. This could have excluded some deaths in which air pollution might have played a contributory role and thus might have resulted in underestimation of air pollution effects.

10.9 Power of the study

This study found statistically significant positive associations between daily mortality and daily PM₁₀ and CO for the whole population and the population aged 65+ years suggesting that the study had adequate power to find the statistically significant associations. However, for certain mortality categories and certain analysis such as multi-pollutant analysis, the study may not have enough power to find the statistically significant results.

Preliminary time series analysis of any association between air pollution and mortality in the population under 65 years old showed no clear association in this age group, which may just be due to a lack of power to find a statistically significant association in that age group. The PM₁₀ associated relative risk that would have been statistically significant at the 5% level for the standard error obtained in the model for

non-external deaths in the population under 65 years old was estimated. For the standard error obtained in the model, the percentage increase in non-external mortality associated with a $10 \mu\text{g}/\text{m}^3$ increase in the same day PM_{10} should be at least 2.2% to become a statistically significant at the 5% level. This effect size was a bit high since the percentage increase in the same day non-external mortality for a $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} was just 1.31% (95% CI: 0.44, 2.18) for the whole population suggesting that the study was underpowered to detect a statistically significant association between daily PM_{10} and daily mortality in the population under 65 years old. Thus, no specific analysis was undertaken for the population of that age group.

The statistically significant association observed between CO and daily mortality in the single pollutant analysis disappeared in the multi-pollutant analysis when PM_{10} was added in the model. While the association between PM_{10} and non-external mortality was statistically significant at the 5% level in the single pollutant analysis, it was statistically significant only at the 10% level in the multi-pollutant analysis. For the standard errors obtained in the multi-pollutant model, the percentage increase in non-external mortality associated with one interquartile range ($11.7 \mu\text{g}/\text{m}^3$) increase in the same day PM_{10} should be at least 1.67% and the percentage increase in non-external mortality associated with one interquartile range ($0.88 \text{mg}/\text{m}^3$) increase in the same day CO should be at least 1.68% to become statistically significant at the 5% level. These effect sizes were similar to the results from the single pollutant analysis and were not unreasonable for daily non-external deaths of the population aged 65+ years suggesting that the study had enough power to detect any effect of the reasonable size. However, the estimated percentage increases in the same day non-external mortality for one interquartile range increases in PM_{10} and CO were respectively 1.46% and 0.48% in the multi-pollutant analysis (Table 6-7, p.122). The study did not have enough power to detect the smaller effects of that magnitude.

10.10 Areas of future research

This study has established associations of both short-term and long-term exposure to PM_{10} with mortality. PM_{10} is a mixture of particles of different physical sizes, all smaller than $10 \mu\text{m}$ aerodynamic diameter. There is a growing evidence that the relative risks of mortality associated with fine particles ($\text{PM}_{2.5}$) (particles less than $2.5 \mu\text{m}$) is greater than the risks associated with coarse particles ($\text{PM}_{10-2.5}$) (Schwartz, Dockery and Neas, 1996; Cifuentes *et al.*, 2000; Fairley, 2003). The fine particles ($\text{PM}_{2.5}$) within the PM_{10} mixture are probably the ones that cause the greatest effects because of their

ability to accumulate and reach the lower region of the respiratory system. It would be useful to conduct a similar study, analysing the association of exposure to fine particles with mortality. Although PM_{2.5} has not been monitored continuously in Christchurch, Environment Canterbury has carried out some monitoring of PM_{2.5} in recent years (Aberkane, Harvey and Webb, 2005). This could be a useful future research topic when continuous monitoring of PM_{2.5} will start.

This study has analysed the association between air pollution and mortality. The number of deaths due to air pollution will be smaller than the number of non-fatal health outcomes such as hospital admissions due to air pollution. Studies have reported an increase in hospital admissions associated with an increase in air pollutant concentrations (Morgan, Corbett and Wlodarczyk, 1998; Schwartz, 1999; Atkinson *et al.*, 2001; Luginaah *et al.*, 2005) . This has also been reported in Christchurch city (McGowan *et al.*, 2002). These studies only analysed an increase in hospital admissions up to a few days after exposure. My study showed that the PM₁₀ effect on mortality is not restricted to a few days. The effect is distributed over several weeks. Focussing on the effects for only a few days after exposure would underestimate the overall effect of PM₁₀. This may be true for hospital admissions as well. A detailed analysis looking at the extended effects of PM₁₀ on daily hospital admissions using distributed lag modelling would be useful for public health decision making, including the allocation of hospital resources according to expected air pollution related demand.

The urban airshed model used in this study is also capable of simulating other pollutants such as NO₂ and CO. Annual average NO₂ simulated by a similar urban airshed model was used to study the association of long-term exposure to NO₂ with annual mortality in the Auckland Region (Scoggins *et al.*, 2004). An increase in annual mortality associated with an increase in annual average NO₂ was reported in that study. The present study did not find any evidence of the acute effects of NO₂ on mortality in Christchurch. The effect of long-term exposure to NO₂ on mortality is, however, unknown for Christchurch. In the absence of data on long-term exposure to other pollutants, it is unknown whether the long-term effect of PM₁₀ on mortality observed in this study is independent of the effect of other pollutants or other pollutants confound the association of long-term exposure to PM₁₀ with mortality. It would be useful to carry out a similar study with annual data on other pollutants.

The variation in the exposure-response relationships among the large number of short-term mortality studies could be due to differences in air pollution sources. Air pollution from one source may be more toxic than another and as such their effects on

mortality will be different. However, in absence of source-oriented PM₁₀ data in Christchurch, it is not possible to estimate the separate effects of PM₁₀ from different sources, such as motor vehicle emissions, domestic home heating and industry. Provided that source-specific detailed PM₁₀ data are available, separate analysis of the association of mortality with PM₁₀ from different sources would be helpful in developing policies aimed at reducing exposure from specific sources.

Chapter 11: Conclusion

There has been growing public health concern about air pollution in New Zealand, Australia and other countries in recent years. The main aim of this study was to establish whether or not ambient particulate air pollution is associated with an increase in mortality in Christchurch. Christchurch has one of the worst air quality problems in New Zealand, especially in the winter, with 24-hour average PM_{10} concentrations exceeding the Ministry for the Environment ambient air quality guideline for about 30 days each winter. The availability of continuously monitored ambient air pollutant concentration going back to 1988 in Christchurch provided an excellent opportunity to carry out a comprehensive time series analysis of the association of daily mortality with air pollution exposure. In addition, the estimated spatial PM_{10} concentrations from an urban airshed model simulated over the Christchurch airshed allowed analysis of the association of long-term exposure to PM_{10} with mortality. This study analysed the association between PM_{10} and mortality over different timescales.

This study shows that PM_{10} is associated with an increase in mortality for all non-external causes, circulatory causes and respiratory causes. The associations were observed for both short-term exposure (acute effect) and long-term exposure to PM_{10} . One important policy question while interpreting the association of short-term exposure to PM_{10} with daily mortality is whether or not this short-term association (acute mortality effect of PM_{10}) is entirely due to short-term mortality displacement. If the majority of the deaths associated with PM_{10} are brought forward just by a few days, there will be much less public health concern of the association of PM_{10} with mortality. This study shows that the short-term association between PM_{10} and daily mortality could not be entirely attributed to short-term mortality displacement or “harvesting”. PM_{10} brought forward the majority of deaths by a non-trivial amount of time.

This study also shows that the mortality effect of PM_{10} was not restricted to a couple of days. The effect was distributed over several weeks after exposure. The increase in risk of mortality due to PM_{10} was much higher when the effects of daily PM_{10} over a longer period of time was considered.

It is the increase in mortality associated with long-term exposure to PM_{10} , such as annual average, that has the most public health importance. This study shows that the effect of annual average PM_{10} on annual mortality was much higher than the acute effect of PM_{10} on daily mortality. This suggests that PM_{10} may be a greater threat to

public health than what has been considered until now based on the findings of time series studies.

Although the PM_{10} associated individual risk of mortality in the general population is low, the implication of the study is important for public health because hundreds of millions of people in the world are exposed to air pollution levels at least as high as those in Christchurch. There need to be policies in place to reduce ambient air pollution from the current level. Christchurch experiences high levels of air pollution mainly in the winter. The major source of air pollution in the winter is the emissions from the burning of solid fuels for domestic heating. Hence, the primary focus of control strategies for reducing air pollution in Christchurch should be to implement policies that will reduce domestic emissions such as banning the use of open fires, replacing solid fuel burners with improved burners, which have lower levels of emissions, and better insulation of houses. Although vehicle emissions make up a relatively small fraction of air pollution in Christchurch, it should not be ignored especially because of the likelihood of a stronger adverse effect of air pollution from this source than other sources. In addition, vehicle emissions are the major contributor in the summer when there is no air pollution from domestic heating. Thus, the long-term control strategies to reduce air pollution should include policies to reduce motor vehicle emissions.

The results of this study are broadly consistent with those of many other studies of the association of particulate pollution with mortality. This analysis, together with the results of other studies, confirms that particulate pollution is associated with an increase in mortality. This study contributes to the growing scientific evidence of mortality effects of PM_{10} both in the short-term and in the long-term and provides evidence that ambient air pollutant concentration in Christchurch is a matter of public health concern, and that there should be policies in place to reduce ambient air pollution.

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Appendix A: Sequential development of basic models for various mortality categories

Sequential development of a basic model for daily *circulatory deaths in the 65+ years age group*

Model	Model Description	df [#]	p-value [^]
M ₁	Annual cycle ($k^* = 1$)	4	
M ₂	Annual and six months cycle ($k^* = 1, 2$)	6	0.052
M ₃	Annual, six months and four month cycle ($k^* = 1, 2, 3$)	8	0.016
M ₄	With $k^* = 1, 2, 3, 4$	10	0.270
M ₅	With $k^* = 1, 2, 3, 4, 5$	12	0.002
M ₅₁	With $k^* = 1, 2, 3, 5$	10	0.001
M ₆	With $k^* = 1, 2, 3, 5, 6$	12	0.807

All models had a linear time trend as a co-variate.

[#] df: degrees of freedom of model (the number of parameters in the model)

[^] p-value: p-value for the likelihood ratio test to compare the model with the one immediately above it except for model M₅₁. Model M₅₁ was compared with model M₃.

* Values of k determine the period of seasonal cycle. $k = 1, 2, 3, 4, 5, 6$ approximately correspond to the seasonal cycles of one year, 6 months, 4 months, 3 months, 73 days and 2 months respectively.

Sequential development of a basic model for daily *respiratory deaths in the 65+ years age group*

Model	Model Description	df [#]	p-value [^]
M ₁	Annual cycle ($k^* = 1$)	4	
M ₂	Annual and six months cycle ($k^* = 1, 2$)	6	< 0.001
M ₃	Annual, six months and four month cycle ($k^* = 1, 2, 3$)	8	0.154
M ₄	With $k^* = 1, 2, 3, 4$	10	0.005
M ₅	With $k^* = 1, 2, 3, 4, 5$	12	0.930
M ₆	With $k^* = 1, 2, 3, 4, 5, 6$	14	<0.001
M ₆₁	With $k^* = 1, 2, 3, 4, 6$	12	0.547
M ₆₂	With $k^* = 1, 2, 4, 5, 6$	12	0.038
M ₇	M ₆₁ + Indicator variables for years	23	< 0.001

All models had a linear time trend as a co-variate.

[#] df: degrees of freedom of model (the number of parameters in the model)

[^] p-value: p-value for the likelihood ratio test to compare the model with the one immediately above it except for model M₆₂ and M₇. Model M₆₂ was compared with model M₆ and M₇ was compared with M₆₁.

* Values of k determine the period of seasonal cycle. $k = 1, 2, 3, 4, 5, 6$ approximately correspond to the seasonal cycles of one year, 6 months, 4 months, 3 months, 73 days and 2 months respectively.

Sequential development of a basic model for daily *non-external* deaths in the whole population

Model	Model Description	df [#]	p-value [^]
M ₁	Annual cycle ($k^* = 1$)	4	
M ₂	Annual and six months cycle ($k^* = 1, 2$)	6	< 0.001
M ₃	Annual, six months and four month cycle ($k^* = 1, 2, 3$)	8	0.012
M ₄	With $k^* = 1, 2, 3, 4$	10	0.012
M ₅	With $k^* = 1, 2, 3, 4, 5$	12	0.014
M ₆	With $k^* = 1, 2, 3, 4, 5, 6$	14	0.115
M ₇	M ₅ + Indicator variables for years	23	0.079

All models had a linear time trend as a co-variate.

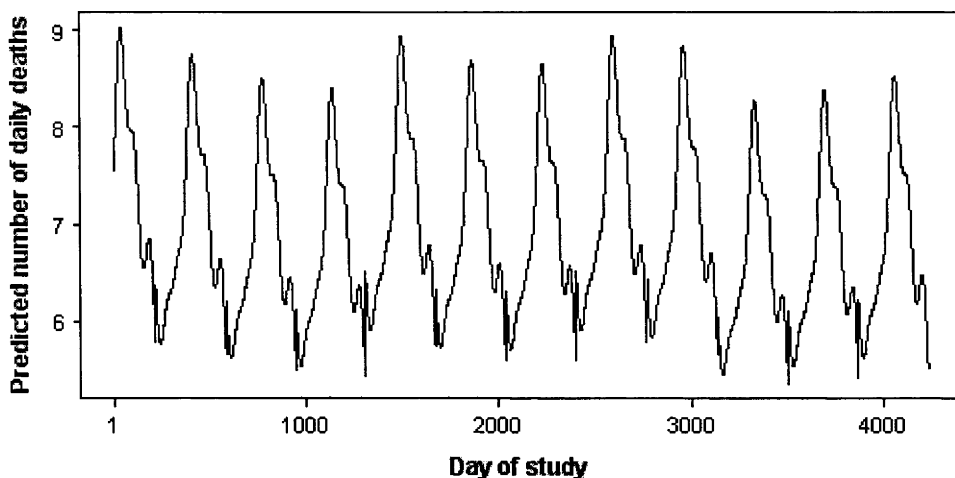
[#] df: degrees of freedom of model (the number of parameters in the model)

[^] p-value: p-value for the likelihood ratio test to compare the model with the one immediately above it except for model M₇. Model M₇ was compared with model M₅.

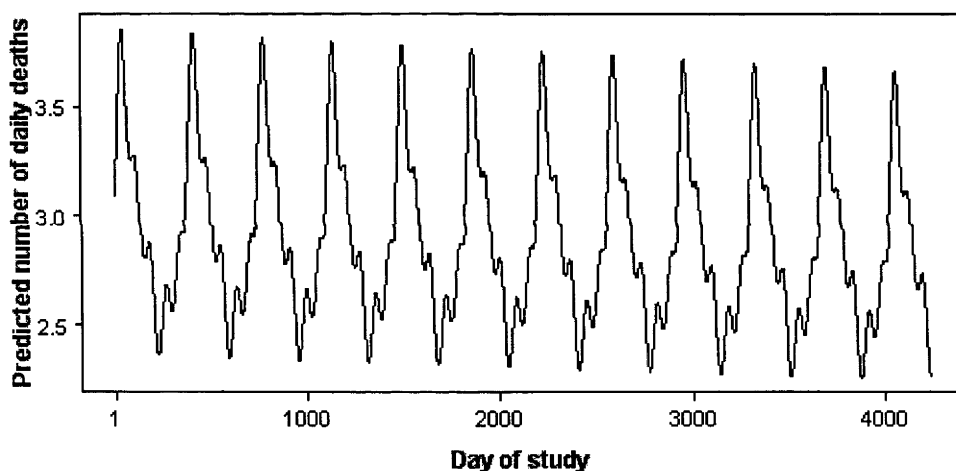
* Values of k determine the period of seasonal cycle. $k = 1, 2, 3, 4, 5, 6$ approximately correspond to the seasonal cycles of one year, 6 months, 4 months, 3 months, 73 days and 2 months respectively.

Appendix B: Plots of the predicted number of daily deaths based on the final cosinor models that adjust for a long-term trend and seasonal variations in daily mortality

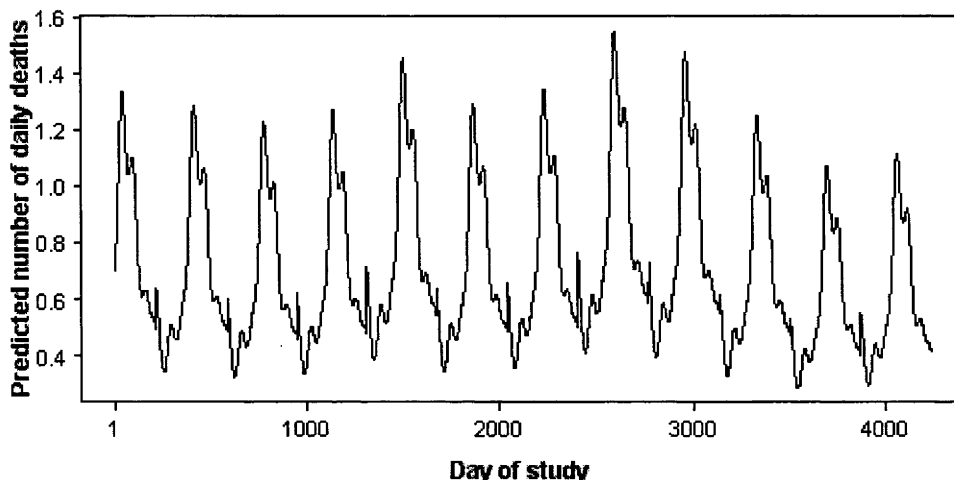
(A) Non-external deaths in the whole population



(B) Circulatory deaths in the 65+ years age group



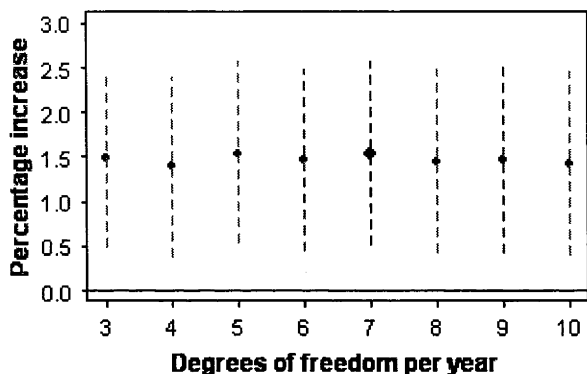
(C) Respiratory deaths in the 65+ years age group



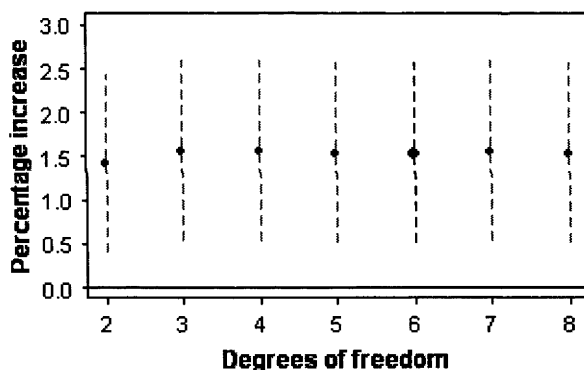
Appendix C: Sensitivity analyses of the effects of PM₁₀ on daily mortality for various mortality categories

Sensitivity of the effects of PM₁₀ on daily *non-external mortality of all ages* to the degrees of freedom set for the smooth functions of confounders. Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 $\mu\text{g}/\text{m}^3$) increase in daily PM₁₀ concentrations

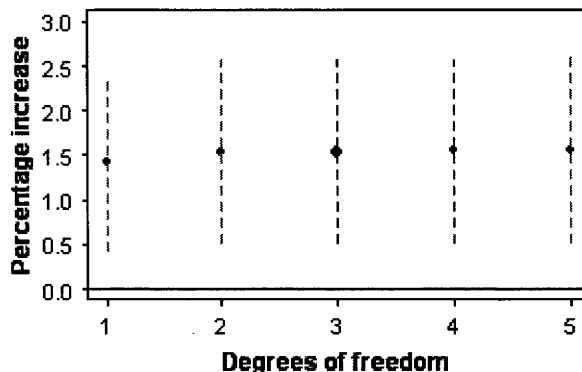
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature

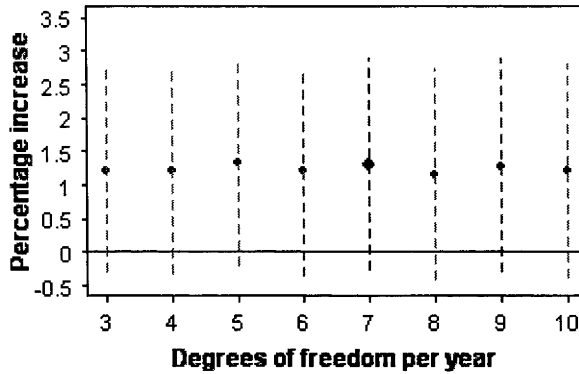


(C) Degrees of freedom for the smooth function of relative humidity

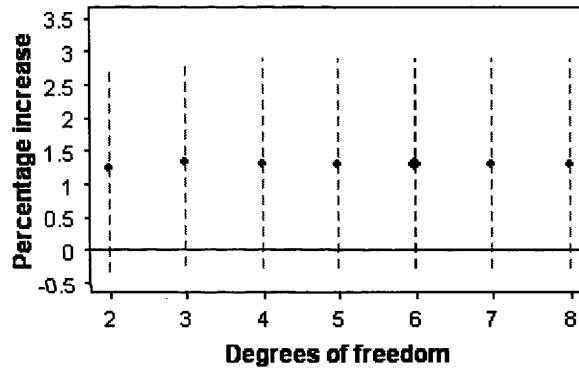


Sensitivity of the effects of PM₁₀ on daily *circulatory mortality of those aged 65+ years old* to the degrees of freedom set for the smooth functions of confounders. Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 μg/m³) increase in daily PM₁₀ concentrations

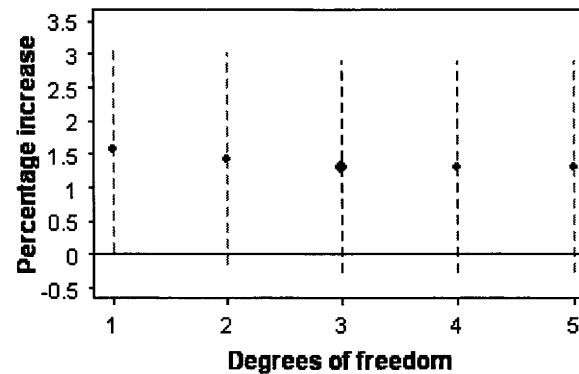
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature

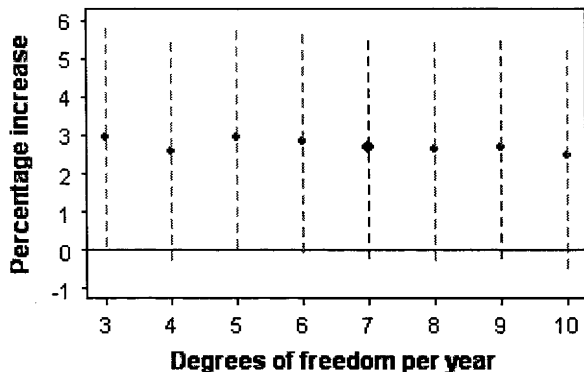


(C) Degrees of freedom for the smooth function of relative humidity

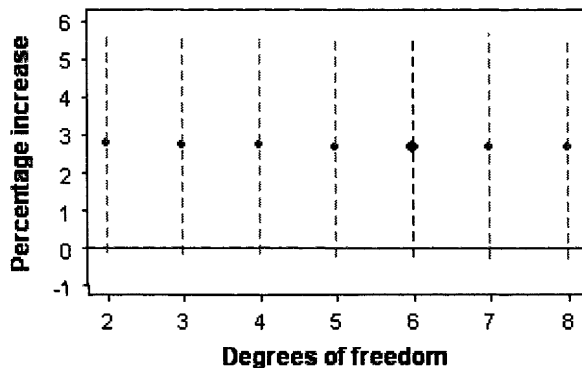


Sensitivity of the effects of PM₁₀ on daily *respiratory mortality of those aged 65+ years old* to the degrees of freedom set for the smooth functions of confounders. Percentage increase in daily deaths and 95% CI associated with one interquartile range (11.7 µg/m³) increase in daily PM₁₀ concentrations

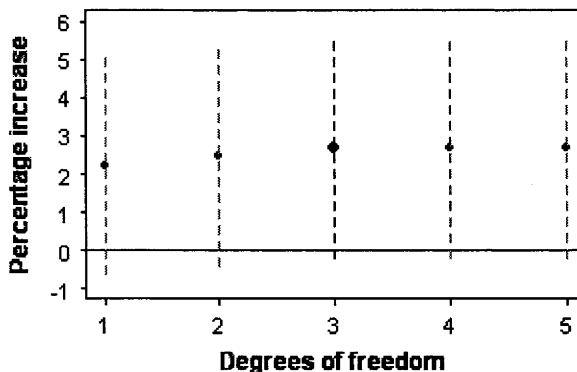
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature



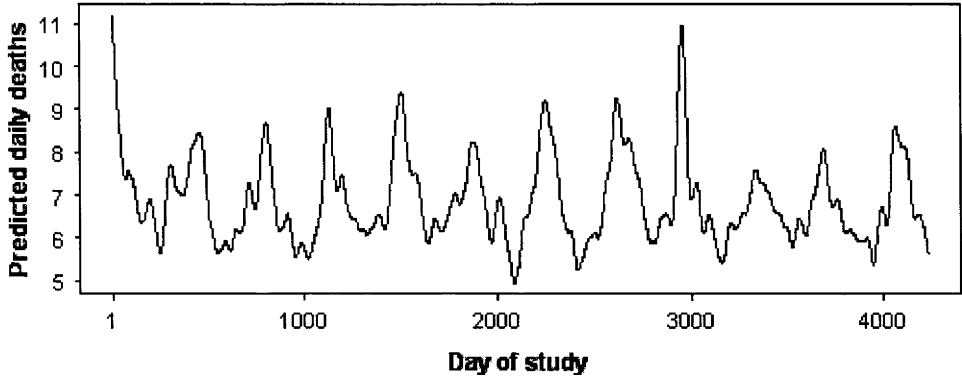
(C) Degrees of freedom for the smooth function of relative humidity



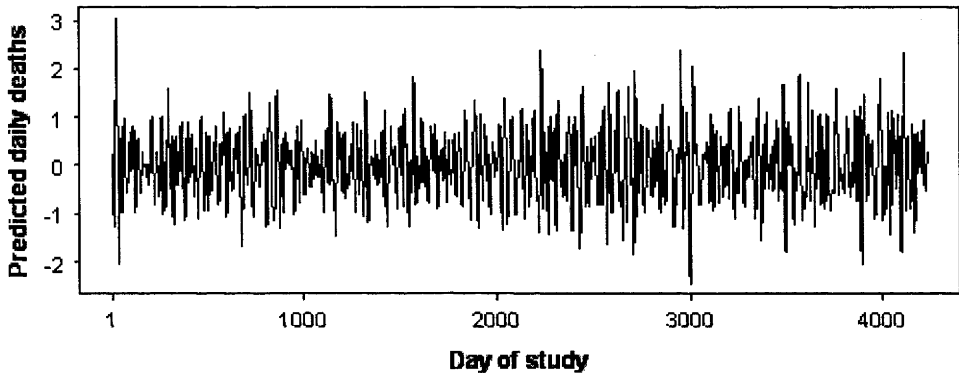
Appendix D: Decomposition of daily mortality data series

Decomposition of daily *non-external deaths in whole population*

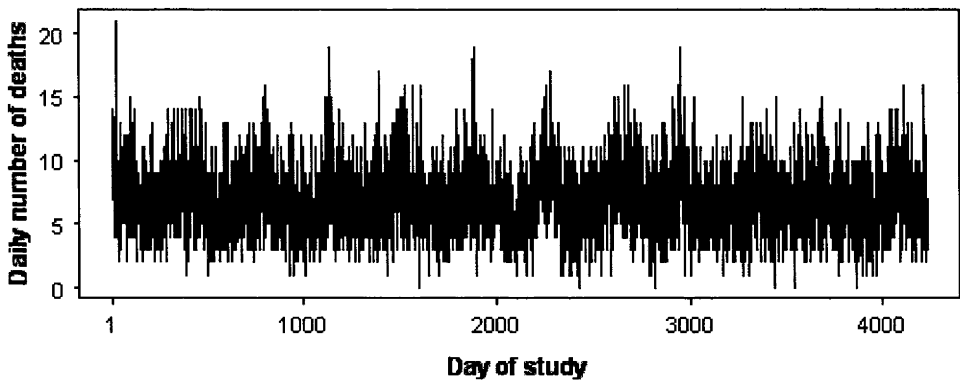
(A) Long-term trend and seasonal variation with a 120 days smoothing window



(B) Mid-scale component with a 30 days smoothing window

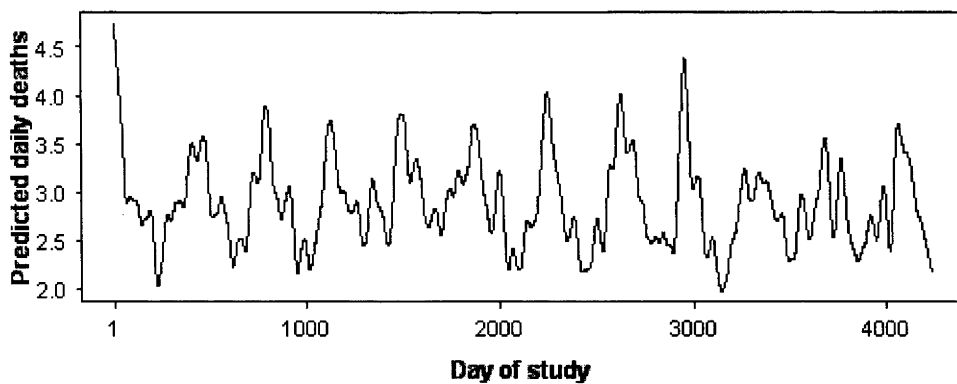


(C) Daily number of deaths

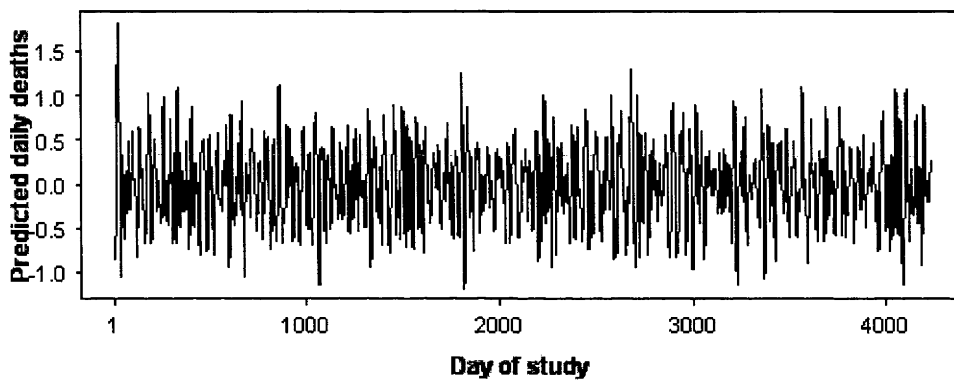


Decomposition of daily *circulatory deaths in the 65+ years age group*

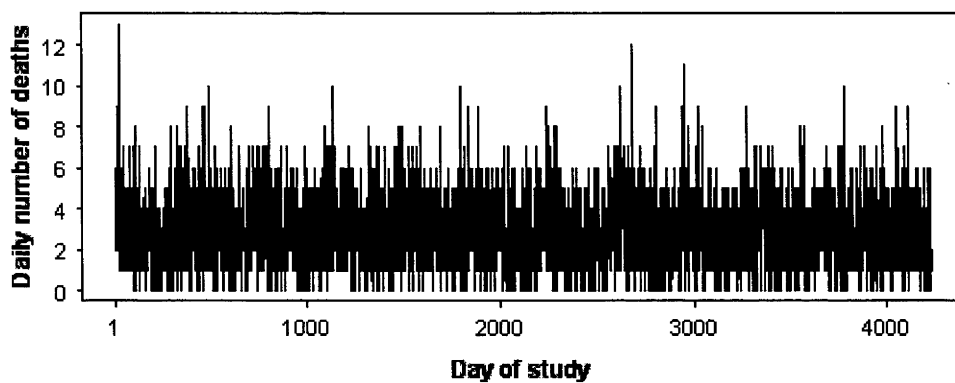
(A) Long-term trend and seasonal variation with a 120 days smoothing window



(B) Mid-scale component with a 30 days smoothing window

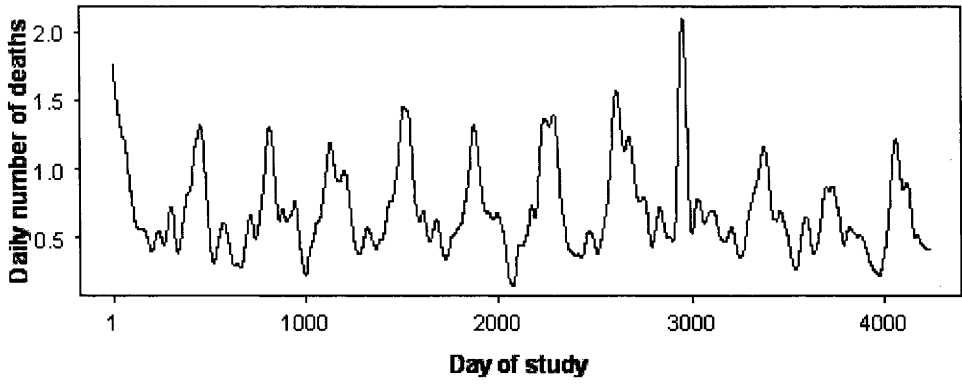


(C) Daily number of deaths

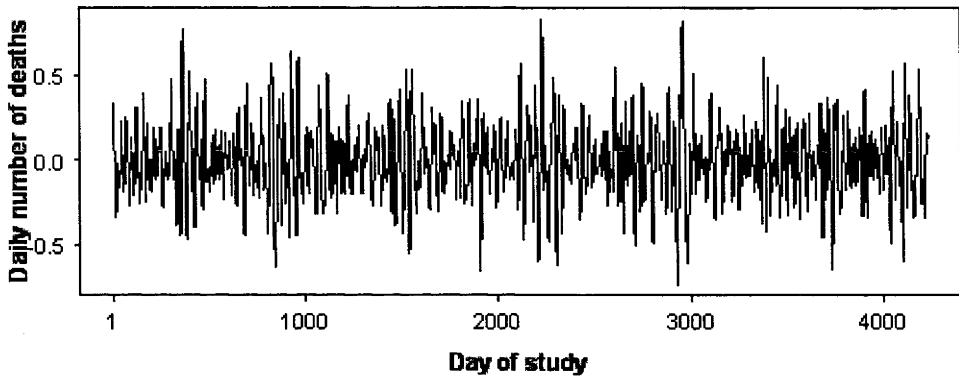


Decomposition of *daily respiratory deaths in the 65+ years age group*

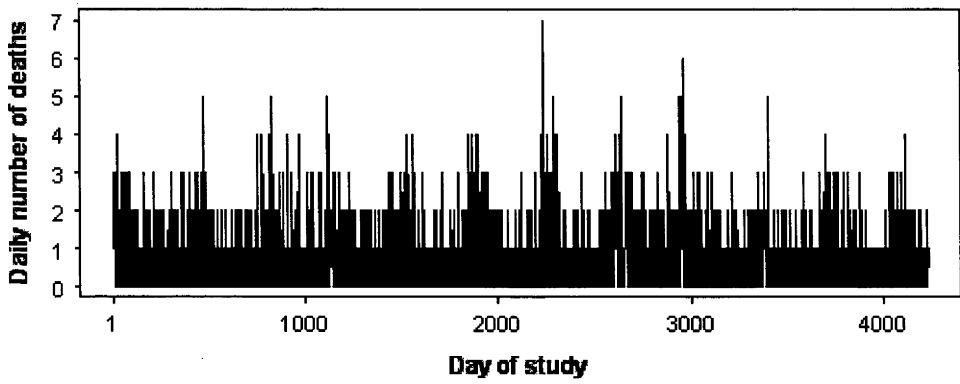
(A) Long-term trend and seasonal variation with a 120 days smoothing window



(B) Mid-scale component with a 30 days smoothing window



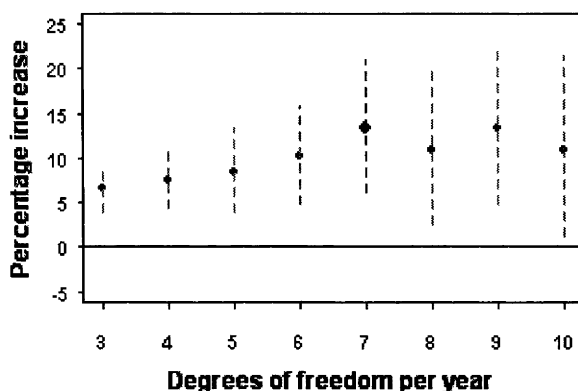
(C) Daily number of deaths



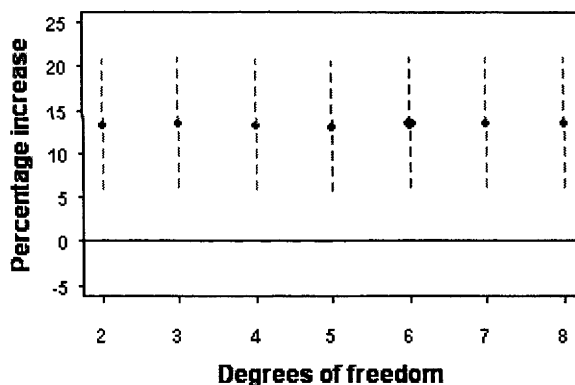
Appendix E: Sensitivity analyses of the total effects of PM₁₀ on daily mortality over 0 – 40 days for various mortality categories

Sensitivity of the total effects of PM₁₀ on daily *non-external mortality of all ages* over 41 days to the degrees of freedom set for the smooth functions of confounders. Estimated overall percentage increase in daily deaths and 95% CI for each 10 µg/m³ increase in PM₁₀, with fourth degree polynomial distributed lag models

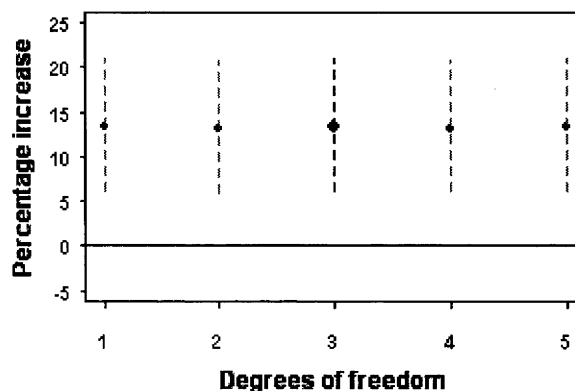
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature

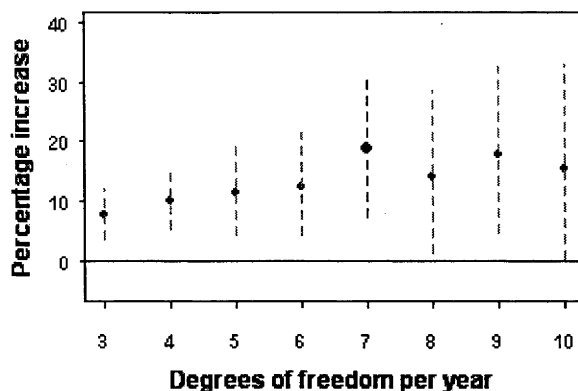


(C) Degrees of freedom for the smooth function of relative humidity

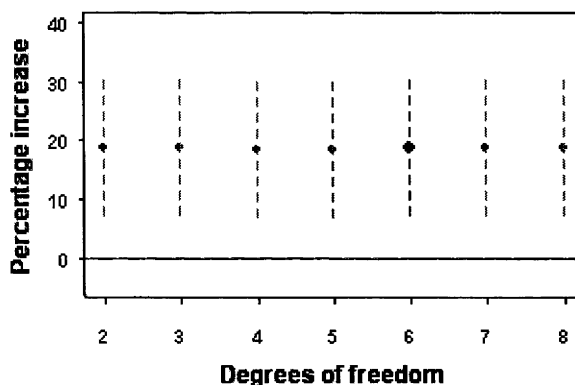


Sensitivity of the total effects of PM₁₀ on daily *circulatory mortality of those aged 65+ years old* over 41 days to the degrees of freedom set for the smooth functions of confounders. Estimated overall percentage increase in daily deaths and 95% CI for each 10 µg/m³ increase in PM₁₀, with fourth degree polynomial distributed lag models

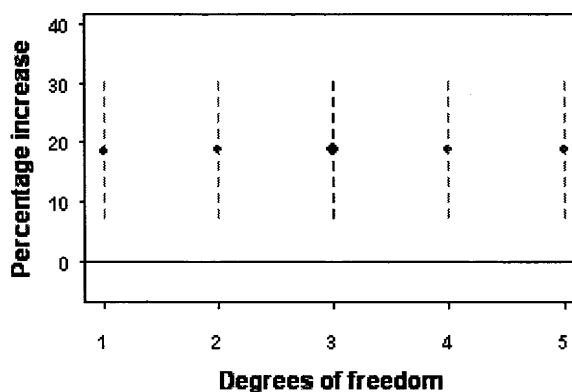
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature

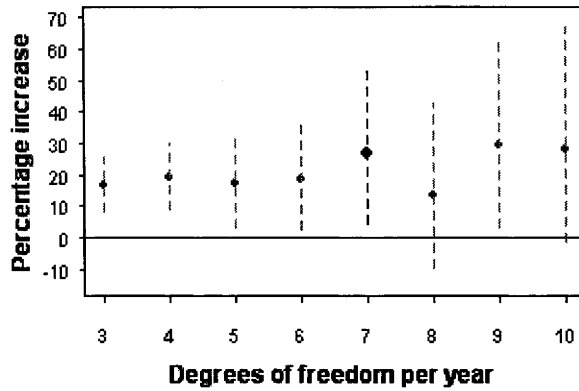


(C) Degrees of freedom for the smooth function of relative humidity

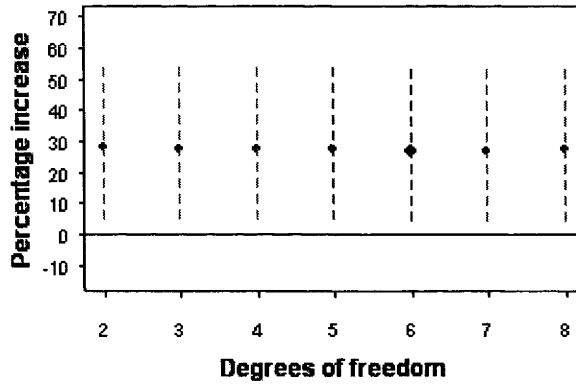


Sensitivity of the total effects of PM₁₀ on daily *respiratory mortality of those aged 65+ years old* over 41 days to the degrees of freedom set for the smooth functions of confounders. Estimated overall percentage increase in daily deaths and 95% CI for each 10 µg/m³ increase in PM₁₀, with fourth degree polynomial distributed lag models

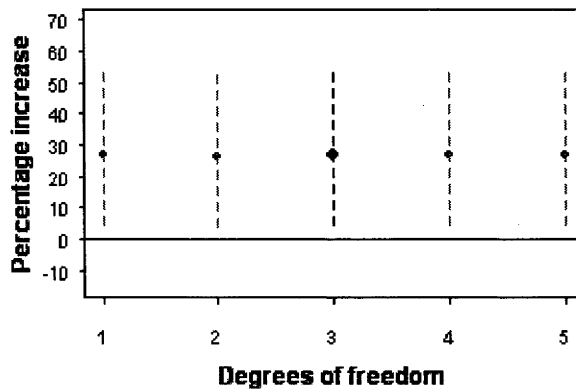
(A) Degrees of freedom for the smooth function of time



(B) Degrees of freedom for the smooth function of temperature



(C) Degrees of freedom for the smooth function of relative humidity



Appendix F: NZDEP96 index of deprivation

Deprivation variables used in the construction of the NZDep96 index of deprivation (in decreasing importance in the index)

Deprivation type	Description (proportions in small areas of people)
Communication	With no access to a telephone
Income	Aged 18-59 years receiving a means-tested benefit
Employment	Aged 18-59 years unemployed
Income	Living in households with equivalised* income below an income threshold
Transport	With no access to a car
Support	Aged less than 60 years living in a single-parent family
Qualifications	Aged 18-59 years without any qualifications
Owned home	Not living in own home
Living space	Living in households above equivalised* bedroom occupancy threshold

* equivalisation refers to methods used to control for household composition. In this way, for example, the standard of living of a single person with an income of \$40,000 could be compared to that of a household consisting of two adults and three children on an income of \$40,000.

Source: (Crampton *et al.*, 2000)