AMBIENT AIR POLLUTION
AND DAILY HOSPITAL ADMISSIONS
IN MELBOURNE

1994-1997

RESEARCH TEAM

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FOREWORD – HOSPITAL ADMISSIONS STUDY

EPA has been monitoring Melbourne's air quality since the 1970's. Recently, this information has been the subject of studies by air quality scientists and experts in the field of human health to generate information about the impacts of our air quality on the health and well being of Melburnians.

While we can be reassured that Melbourne's air quality has generally improved over the last twenty years, we also know that the levels of air pollution we experience still have an impact on people. The Melbourne Mortality Study released in June 2000 showed that current levels of air pollution in Melbourne are associated with increases in daily mortality.

This report - Ambient Air Pollution and Daily Hospital Admissions in Melbourne 1994-1997 - provides new information which shows that air pollution in Melbourne is associated with increases in hospital admissions for respiratory and cardiovascular disease. Admissions for asthma in children are also associated with air pollution levels in Melbourne.

The pollutants studied - ozone, nitrogen dioxide, fine particles and carbon monoxide - arise as a result of emissions from many sources, but in particular motor vehicles, industry and domestic wood heaters.

These results confirm that despite the improvements in air quality we have seen in Melbourne, we cannot afford to be complacent as air pollution is still affecting the lives of people in our community.

EPA has recently updated the State environment protection policy (Air Quality Management) and is currently finalising the Air Quality Improvement Plan for the Port Phillip Regions to ensure that Melbourne's air quality continues to improve and that the impacts of air pollution on people's health and well being are reduced. The SEPP and AQIP will provide the framework for taking action that ensures the community's aspirations for clean air are achieved, having regard to the community's social and economic considerations too.

Improvements will only come though if all sectors of society - government, industry and the community - take the necessary steps within this framework to reduce emissions into the air environment. Ultimately it is up to us all as individuals to make decisions and take the actions that improve air quality.

I would like to thank the authors of this report: Dr Lyn Denison from EPA Victoria; Associate Professor Rod Simpson and Anna Petroeschevsky from the University of the Sunshine Coast; and Associate Professor Gail Williams from the University of Queensland for their work in generating this valuable information.

BRIAN ROBINSON

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EXECUTIVE SUMMARY

In recent years studies into the effects of air pollution on human health have shown that increases in daily mortality, hospital admissions and emergency room attendances for respiratory and cardiovascular disease, exacerbation of asthma and respiratory symptoms, are all associated with increases in air pollution. Most of these studies have been conducted in the US and Europe, however recent studies in Australia have shown that the effects observed overseas are also observed here. The results of the Melbourne Mortality study showed that air pollution levels in Melbourne are associated with increases in daily mortality from respiratory and cardiovascular causes.

This current study has been conducted to explore the relationship between air pollution and daily emergency admissions to hospitals in Melbourne. The study period covers 1994 to 1997 and the pollutants considered are ozone, nitrogen dioxide, fine particles and carbon monoxide. Emergency admissions for respiratory disease, cardiovascular disease and asthma were examined in the all ages, 0-14 years, 15-64 years and greater than 65 years age groups.

The results of the study show that, after controlling for the effects of weather, air pollution in Melbourne is associated with increases in hospital admissions for respiratory and cardiovascular disease. Ozone, nitrogen dioxide, fine particles and carbon monoxide were all found to associated with admissions for respiratory disease and for asthma. Fine particles, nitrogen dioxide and carbon monoxide were also associated with admissions for cardiovascular disease in the elderly. For ozone the strongest effects were observed in the warm season. For the other pollutants the strongest effects were observed in the cool season.

The main sources of ozone, nitrogen dioxide, fine particles and carbon monoxide in Melbourne are motor vehicles, some industrial processes and domestic wood burning. The results of this study suggest that strategies to reduce these pollutants are important to reduce the risk of adverse health effects in the community. The development of the Air Quality Improvement Plan for Melbourne addresses these issues.

T A B L E   O F   C O N T E N T S

INTRODUCTION

In recent years epidemiological studies, such as time series studies, have been used extensively in the study of air pollution and health effects. Associations between air pollution and increases in daily mortality and morbidity outcomes, such as hospital admissions and emergency room attendances for respiratory and cardiovascular disease, have been observed in many studies in various parts of the world.

Although the focus of many of these studies has been on the effect of air pollution on daily mortality, relationships have also been observed between daily hospital emergency admissions and ozone, particles, NO_2 and CO. Although many studies have observed associations between hospital admissions and ozone and particles, until recently NO_2 and CO at current ambient levels were not thought to be of concern with respect to public health. With increasing sensitivity in the statistical methods used in epidemiological studies, associations are now being observed between these pollutants and adverse health effects which has led to increased interest in these pollutants.

In Australia studies investigating the effects of air pollution on health have been conducted in Melbourne, Brisbane and Sydney. The results of these studies have shown that although air pollution levels in Australia are generally lower than those observed in the US and Europe, the associations between ambient air pollution and daily mortality observed in overseas studies also occur in Australia.

1.1   Aim

The aim of this study was to examine the relationship between ambient air pollution and daily emergency admissions to hospitals in Melbourne. The study period covered the years 1994 through 1997. The pollutants considered were fine particles (measured by nephelometry), ozone, nitrogen dioxide and carbon monoxide. Sulfur dioxide was not included in the analysis as levels in Melbourne are very low and frequently fall below detectable levels.

The analysis involved an epidemiological time-series study investigating the relationship between ambient levels of air pollution in Melbourne and daily hospital admissions for respiratory and cardiovascular disease. The statistical methodology used followed that developed by researchers in Europe and the United States.

1.2   Background

Air quality is often rated as the most important environmental issue within the community. The potential adverse health effects associated with exposure to air pollution is of growing concern and an understanding of the impacts of air pollution is critical in the development of air quality standards, policies and programs aimed at improving air quality.

The National Environment Protection Measure (NEPM) for ambient air quality sets national air quality standards in Australia for the six major pollutants nitrogen dioxide (NO_2), particles (as PM_{10}), carbon monoxide (CO), ozone (O_3), sulfur dioxide (SO_2), and lead (Pb). The NEPM (Ambient Air Quality) standards have been set to be protective of human health. These standards are based primarily on the results of studies that have been conducted overseas. Whether these findings can be extrapolated to the Australian situation has been the subject of much debate. In general, air quality in Australian cities is much better than that observed in many American and European cities. Recent work in Melbourne (EPA, Victoria, 2000; Simspon et al., 2000), Sydney (Morgan et al. 1998a and b) and Brisbane (Simpson et al. 1997; Petroeschevsky et al., 2001) has indicated that current levels of ambient air pollution in these cities are making significant contributions to variations in daily mortality and hospital admissions for cardiovascular and respiratory disease, and that the effects observed overseas do occur here as well. There is a commitment in the NEPM to undertake a review of

the particle standard by the year 2001 and a full review by 2005. An important part of this review will be the expansion of the local database relating to air quality and health issues to which this study will contribute.

Local studies into the health effects of air pollution are also important to target actions to reduce emissions of priority pollutants. The results of the Melbourne studies have been an important input into the development of the Air Quality Improvement Plan (AQIP) for the Port Phillip Region, which includes both the Melbourne Metropolitan area and Geelong. Ongoing health studies by EPA will provide information for further development of actions contained in AQIP and reductions in emissions of pollutants of concern.

Despite being the second largest city in Australia, few studies have investigated the association between air pollution and health in Melbourne. Particle pollution is a concern during the autumn and winter given the widespread use of wood fires for heating during this time. Generally, the highest particle levels are observed during this period although high levels can also be observed during the summer. However, levels of SO2, which in North American and European cities is often highly correlated with particles, are very low in Melbourne due the absence of significant point sources in the area and the low sulfur content of Australian fossil fuels. Melbourne thus provides a good opportunity to examine the health effects of particles and other pollutants without the potential confounding effects of SO_2.

2. REVIEW OF THE HEALTH EFFECTS OF AIR POLLUTION

The health effects of the criteria air pollutants have been known for many years. This information has been derived mainly from controlled human exposure and population based epidemiological studies, as well as animal toxicological studies. Studies of the health effects of air pollution have shown associations between ambient air pollution levels and adverse health effects, including increases in daily mortality, increases in hospital admissions and emergency room attendances, as well as exacerbation of asthma. Most the observed effects have been associated with respiratory (including asthma) and cardiovascular disease. These studies have been conducted in various parts of the world with differing climates, socioeconomic status and pollutant levels and mixes. Most of the recent information has arisen from epidemiological studies.

Epidemiological studies evaluate the incidence of diseases or effects and risk factors and associate these with air pollution data; they do not demonstrate causality or provide clear evidence of the mechanisms of such diseases or effects. Specifically, epidemiological studies concentrate on showing whether associations exist, rather than how they might be explained at a pathogenic or mechanistic level. Animal studies and experimental exposure, or chamber, studies with humans help to generate data from which hypotheses concerning the mechanisms for the effects of air pollution can be formulated. This in turn can aid in the design of epidemiological studies.

In recent years much of the research into the health effects of air pollution has focussed on particles, PM_{10} and $PM_{2.5}$. Illustrations of the adverse health effects of particles date back to the dramatic pollution episodes of Belgium's industrial Meuse Valley (1930), Donora Pennsylvania (1948), and London England (1952). During the high pollution episode in London, 4,000 excess deaths occurred, with 2,000 occurring in London County (Bascom et al., 1996). The health effects of particles include increases in daily mortality, hospital admissions and emergency room (ER) attendances, and exacerbation of respiratory symptoms and asthma. Populations that have been shown to be susceptible to the effects of particles include the elderly; people with existing respiratory disease such as asthma, chronic obstructive pulmonary disease (COPD) and bronchitis; people with cardiovascular disease;

people with infections such as pneumonia; and children. Results of epidemiological studies have provided little evidence for the existence of a threshold value below which no adverse health effects are observed.

Recent work conducted in the US as part of the National Morbidity, Mortality and Air Pollution Study (NMMAPS) (Samet et al., 2000) has investigated the association between particles and daily mortality in twenty US cities using a standardised statistical approach. The results of this study have shown that there is substantial variability in the observed associations and effect estimates between study locations. Combining the results of the individual analyses yielded an overall effect estimate for the cities studied of 0.34% per 10 $\mu g/m^3$ increase in daily PM_{10}. The results of this study also found that the observed associations were more similar for locations close to each other than for cities further away. There is evidence that the association between PM_{10} and mortality in the western US was larger than in the east or south. A similar analysis undertaken for ninety US cities (NMMAPS II) found stronger effects in the north-east US than observed in NMMAPS and the meta-analysis indicated that a 0.5% increase in daily mortality was associated with a 10 $\mu g/m^3$ increase in daily PM_{10} across these cities.

Studies conducted in Europe as part of the Air Pollution and Health: A European Approach (APHEA) program have shown that although associations are observed between particles and adverse health effects, the results are not as consistent as those observed in the US studies. In general the effect estimates observed in the UK are similar to those observed in NMMAPS.

The results of studies to date have not shown a clear role for particle size or composition in the health effects attributed to particles in epidemiological studies. Adverse health effects, such as increases in daily mortality and increases in hospital admissions, have been associated with PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$ and ultrafine particles. Studies conducted using $PM_{2.5}$,

and to a greater extent $PM_{10-2.5}$, are limited due to the lack of monitoring data in these size fractions, making it difficult to accurately assess the relative importance of each size fraction. The role of particle composition in the observed health effects is also unclear, with adverse effects attributed to both crustal and combustion particles.

Ozone is another pollutant that is the focus of a large amount of research. Subgroups within the population that are susceptible to the effects of ozone include the elderly and asthmatics, however, healthy individuals, including elite athletes, have shown adverse effects on exposure to ozone.

Much of information available on the health effects of ozone has been derived from studies investigating short-term, or acute, effects. Early clinical studies evolved from reports of irritant symptoms and respiratory effects in workers. Workers who were exposed to elevated levels of ozone showed decreases in lung function and chest pain on inspiration. Some of these effects could be reproduced in controlled human exposure studies. These effects have subsequently been shown to occur in the general population. The acute effects of ozone appear to be reversible and there is some evidence that an adaptation process may occur in both animals and humans. The effects of repeated exposures are still unclear. Ambient levels of ozone have been found to be associated with increases in daily mortality and hospital admissions for both cardiovascular and respiratory disease (EPA Victoria, 2000; Touloumi et al, 1997; Sartor et al, 1997; Borja-Arburto et al, 1997; Simpson et al, 1997; Loomis et al, 1996). In a meta-analysis of the APHEA studies investigating the effects of ozone on daily mortality (Touloumi et al., 1997) the association was greater for mortality from cardiovascular causes than from respiratory disease. The O_3 association remained significant with the inclusion of other pollutants, although the effect was slightly reduced with the inclusion of black smoke. The authors concluded that the results of the meta-analysis support the

hypothesis of a causal relationship between increases in O_3 concentrations and daily mortality.

The UK Department of Health has recently reviewed the health effects attributed to ozone exposure (UK Department of Health, 1998). Using the data from the APHEA studies it was estimated that 12,500 premature deaths per year could be attributed to O3 levels in the UK. This estimate was based on the assumption that no threshold exists for the effects of O3 on daily mortality.

There have also been a number of studies that have found association between daily ozone levels and hospital admissions for respiratory and cardiovascular disease (Anderson et al., 1997; Spix et al., 1998; Burnett et al., 1997a; USEPA 1996; Bascom et al., 1996). The results of meta-analyses conducted as part of the APHEA studies (Anderson et al., 1997; Spix et al., 1998) have shown that the strongest effects are observed during the summer months and in the elderly. The UK Department of Health estimates that 9900 hospital admissions per year could be attributed to ambient O_3 levels in the UK (UK Department of Health, 1998).

Increases in respiratory symptoms and decreases in lung function have also been attributed to exposure to ozone. Many of these studies have focussed on the effects of ozone on children's respiratory health. Ozone has also been shown to impact on the lung function in healthy adults (Korrick et al, 1998). The results of the Korrick study found that low level exposure to ozone was associated with decreases in lung function in healthy adult hikers. The effects were stronger in hikers that had a history of asthma or wheeze with a four-fold greater responsiveness to ozone observed.

Recent epidemiological studies have also investigated the long term, or chronic, effects associated with exposure to ozone. The results of these studies have shown that long-term exposure to elevated levels of ozone is associated with decreases in lung function and may also be associated with an

increase in lung cancer. A study by Beeson et al., (1998) showed an association between long term exposure to ozone and an increase in the incidence of lung cancer in non-smoking males. This study followed a cohort of non-smoking adults over a period of 15 years. No associations were observed between ozone and the incidence of lung cancer in females. In a similar study, Abbey et al., (1999), a strong association was found between exposure to ozone and lung cancer mortality. Long-term exposure to ozone has also been found to be associated with decreases in lung function in children (Frischer et al, 1999; Gong et al., 1998; Kunzli et al., 1997).

Exposure to nitrogen dioxide has also been associated with increases in daily mortality, increases in hospital admissions and emergency room attendances (respiratory and cardiovascular disease). Exacerbation of asthma, increases in respiratory symptoms and decreases in lung function have also been found to be associated with exposure to NO_2. Asthmatics have been shown to be particularly susceptible to the effects of NO_2. Some studies suggest that there is a concentration of NO_2 below which adverse effects are not observed. This concentration, termed the No Observed Adverse Effects Level (NOAEL), is approximately 0.2 ppm (Bascom et al., 1996; WHO, 1999).

Nitrogen dioxide may also sensitise individuals to the effects of other pollutants. In some controlled human exposure studies, prior exposure to NO_2 increases the response to ozone and sulfur dioxide compared to subjects not exposed to NO_2. Nitrogen dioxide has also been shown to increase the response to allergens. This may be significant for asthmatics. Exposure to NO_2 may impair the host defence mechanisms of the body that leads to an increase in susceptibility to infection. This is thought to be significant in children, as a higher incidence in respiratory infections as children has been associated with the development of chronic respiratory disease in adult life (Bascom et al., 1996; Streeton, 1997). Exposure to NO_2 has also been

shown to cause reversible effects on lung function and airway responsiveness. Repetitive exposure in animals can produce changes in lung structure, lung metabolism, and lung defences against bacterial infection.

Epidemiological studies on the effect of NO_2 on daily mortality have indicated that exposure at ambient levels increases daily mortality from respiratory and cardiovascular causes (EPA Victoria, 2000; Morgan et al., 1998; Burnett et al., 1998; Touloumi et al., 1997; Poloniecki et al., 1997). Effects are seen in all age groups. A recent study by Burnett et al (1998) has found an association between 24-hour average NO_2 levels and increases in daily mortality in 11 Canadian cities. Effects were observed for CO, SO_2 and O_3 but these weren't as strong as the NO_2 effect.

A meta-analysis from the APHEA studies has shown an association between daily mortality and daily 1-hour maximum NO_2 levels (Touloumi et al, 1997). This analysis was conducted on the results of studies conducted in six cities across Europe. The effect of NO_2 was greatest in cities that had high levels of black smoke. The associations observed for NO_2 were consistent across all cities studied.

Anderson et al (1997) have conducted a meta-analysis of the APHEA studies associating NO_2 exposure to hospital admissions for COPD. Analysis was also conducted for O_3, SO_2, black smoke and TSP. An association was observed for both 24-hour average and daily 1-hour maximum NO_2 levels. The effects were only significant during the warm season. A similar meta-analysis on the APHEA results has been conducted by Sunyer et al (1997) for emergency room attendances for asthma. A significant positive association was found between 24-hour average NO_2 levels and emergency room attendances for adult asthma. No seasonal variation was observed. The observed effect was independent of the effect of black smoke.

Hospital admissions and emergency room visits for cardiovascular causes have also been associated with NO_2 levels in various studies. Poloniecki et al

(1997) found an association between hospital admissionsfor cardiovascular disease in London and ambient NO_2 levels. Daily average NO_2 levels were associated with hospital admissions for acute myocardial infarction (winter only), arrhythmia and combined circulatory disease.

Studies in children have found associations between the incidence and duration of respiratory illness and ambient NO_2 levels. An earlier study by Pershagen et al (1995) found an association between ambient NO_2 levels and wheezing bronchitis in children. The effects were stronger in girls than in boys. Wheezing bronchitis is a common cause of hospitalisation in infants and these children run an increased risk of developing asthma. The association was strongest in girls less than 18 months of age.

Epidemiological studies on lung function response to NO_2 have shown that exposure to ambient levels of NO_2 are associated with decreases in lung function in asthmatic children. Effects in healthy children appear to be minimal. A considerable number of studies have investigated the lung function response to NO_2 in healthy subjects, asthmatics and to a lesser extent, patients with chronic obstructive pulmonary disease (COPD). These results have been quite variable over a wide range of concentrations (Streeton 1997; Bascom et al., 1996).

The health effects of carbon monoxide (CO) are associated with the level of carboxyhaemoglobin (COHb) levels in blood. It is generally accepted that maintaining a COHb level below 2.5% will be protective of adverse effects related to exposure to CO. The binding of CO with haemoglobin to form COHb reduces the oxygen carrying capacity of the blood and impairs the release of oxygen from haemoglobin. The toxic effects of CO first become evident in organs and tissues with high oxygen consumption, such as the brain, heart, exercising skeletal muscle and the developing foetus.

The health effects of CO include increases in daily mortality and hospital admissions (mainly

cardiovascular disease), increases in angina attacks (patients with ischaemic heart disease), decreases in athletic performance and low birth weights. Many of the early studies showing these effects were conducted on populations exposed to relatively high levels of CO. More recent studies have shown that increases in daily mortality and hospital admissions for cardiovascular disease are observed at levels currently experienced in most cities in Australia and overseas (Linn et al., 2000; Sheppard et al., 1999; Yang et al., 1998; Schwartz 1997; Schwartz 1999; Morris and Naumova 1998; Burnett et al., 1997b; Morris et al., 1995; Burnett et al., 1998; Polniecki et al., 1997). Until recently it was thought that current exposure levels were unlikely to produce serious health outcomes, however the results of these studies brings this into question.

There have been several studies that have shown associations between increases in daily mortality and ambient CO levels. Early studies showed associations between daily mortality and ambient CO levels but at levels much higher that those currently observed in urban areas (UK Department of Health 1998; Bascom et al., 1996). More recent studies have also shown such an association even at the lower levels currently experienced. The recent studies have found that the observed associations are stronger for mortality from respiratory and cardiovascular causes than for all cause mortality (Fairley et al., 1999; Burnett et al., 1998; Touloumi et al., 1997).

The association between ambient CO levels and increases in hospital admissions is another area that has received considerable attention in recent years (Linn et al., 2000; Sheppard et al., 1999; Yang et al., 1998; Schwartz,1999, 1997; Morris and Naumova, 1998; Burnett et al., 1997; Morris et al., 1995). Most of these studies have focussed on the association between ambient CO levels and admissions for cardiovascular diseases.

A recent study by Linn et al., (2000) found that hospital admissions for cardiovascular disease in Los Angeles are associated with increases in ambient CO levels. Similar results have been found in earlier studies (Yang et al., 1998; Schwartz, 1997, 1999). The strongest effects appear to be in the elderly. The effects of CO appear to be independent of the effects of other pollutants in these studies. Several studies have focussed on the association between CO and admissions for congestive heart failure (Morris and Naumova, 1998; Burnett et al., 1997b; Morris et al., 1995). The results of these studies have found that the observed associations are independent of other pollutants and weather although in the study by Morris and Naumova (1998) there was some evidence that temperatures below $4^{\circ}C$ may increase the effects of CO on individuals with congestive heart failure.

A study by Sheppard et al (1999) has also shown an association between hospital admissions for asthma and ambient levels of CO in a non-elderly population in Seattle. PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$ and CO were found to be associated with an increase in hospital admissions for asthma, with the highest risk observed during autumn and spring. An association was also found for O_3 during the summer but no effect was observed for SO_2 for any season. Linn et al., (2000) also found associations between CO and admissions for respiratory disease in Los Angeles.

The biological mechanisms by which air pollution may cause increased morbidity and mortality have been the focus of a large amount of research. For ozone and nitrogen dioxide it is clear the free radicals play a role in leading to an inflammatory response in the airways. For carbon monoxide the mechanism involves the binding of CO to haemoglobin in the blood which interferes with oxygen transport in the body. For particles the biological mechanism is still unclear but there is increasing evidence that inflammation of the airways may be important in the biological response. The role of the macrophage in the observed responses is emerging as a key issue. The alveolar macrophage is the central lung cell in the regulation of the immune response to inhaled pathogens and the development

of inflammation. Recent studies have shown that increases in daily particle levels, PM_{10} and TSP, are associated with increases in heart rate, plasma fibrinogen, blood pressure and pulse rate (Ibald-Mulli et al., 2001; Pekkanen et al., 2000; Peters et al., 1999; Pope et al., 1999; Peters et al., 1997). All these factors increase the risk of cardiovascular conditions and may relate to the changes in cardiovascular autonomic control. This may explain the associations observed between increases in daily mortality and hospital admissions for cardiovascular disease and PM_{10} that have been reported in epidemiological time-series studies. It is also apparent from the epidemiological studies that there are groups within the population that are particularly susceptible to the effects of air pollution. These include the elderly, people with existing respiratory and cardiovascular disease, asthmatics, and children.

Attempts to attribute effects to only one pollutant have prompted questions as to whether it is possible to do so. However, the US studies include those performed in settings where there is only minimal concern about the effects of these copollutants. For example, the Utah Valley experiences high levels of particles during the winter months that arise mainly from a local steel mill, and very low SO_2 levels. Ozone is only of concern during the summer and therefore does not confound the effects of particles during winter. As the population is predominantly a Mormon community they have a very small proportion, approximately 6%, of the population who smoke. The studies conducted in this area have looked at increases in daily mortality, hospital admissions, decreases in lung function, increases in respiratory symptoms and increased school absenteeism using a variety of statistical methods and have found consistent associations between exposure to particles and adverse health effects.

It has also been claimed that the observed effects may be due to confounding by meteorological variables such as temperature or humidity. It is well documented that extremes of temperature do cause adverse health effects such as increases in daily mortality. In the majority of the time series studies conducted to investigate the health effects of air pollution, the impact of meteorological variables on the health outcomes is controlled before the analysis of the pollutant effect is undertaken. These studies have shown that even after controlling for these parameters there is an independent effect of air pollutants. In some cases the statistical approach used for the control of the meteorological variables can affect the size of the effect estimate, but in general the results are robust within the statistical method used for analysis.

Similarly, epidemics of diseases such as influenza can also confound the effect attributable to air pollution. Where the information is available, these episodes are also controlled for in the statistical analysis.

Studies conducted in Australia into the health effects of air pollution have shown that the effects observed overseas are also observed here even though air pollution levels are generally lower in Australia compared with the US or Europe. Associations between daily mortality and particles, O_3 and NO_2 have been found in Melbourne, Sydney and Brisbane (EPA Victoria, 2000; Simpson et al., 2000; Morgan et al., 1998a; Simpson et al., 1997). In addition CO was also found to be associated with increases in daily mortality in Melbourne (EPA Victoria, 2000). Hospital admissions have also been found to be associated with air pollution in Sydney and Brisbane (Morgan et al., 1998b; Petroeschevsky et al., 2001). The results of this current study will contribute to the Australian database and provide local information to guide policy development to improve the quality of our air.

3. STUDY AREA

The city of Melbourne is located on the southeast coast of Australia, on the northern edge of Port Phillip Bay. The study area is contained within a 30 km radius of the CBD and is the major part of the Melbourne Statistical Division identified by the Australian Bureau of Statistics. This area contains several air monitoring stations which are thought to be representative of air quality within the region. The Melbourne Statistical Division recorded a population of 2,994,280 persons in the 1996 Census. The city of Geelong, located to the south west of Melbourne, is not included in the study area.

Melbourne's climate, best described as Mediterranean, is dominated by the proximity of Port Phillip Bay. Melbourne is located on the northern edge of the bay. The weather is variable but generally cool in spring, warm in summer, mild in autumn and cold in winter. The topography and climate combine to make the region an area of high pollution potential during still weather. The study area to the east of Melbourne is bounded by the Dandenong Ranges that rise to about 700 metres. The Great Dividing Range which runs east-west is located approximately 100 kilometres to the north and rises to about 2000m. In summer elevated ozone concentrations are associated with warm temperatures and stable atmospheric conditions. In autumn and winter the major concern is fine particles.

4. METHODS

4.1 Data

4.1.1 Hospital Admissions Data

Data on daily emergency hospital admissions for Melbourne residents occurring between January 1991 to December 1997 were obtained from the Department of Human Services (Victoria). However up until July 1994 private hospitals were not required to provide data to the Victorian Government resulting in an incomplete data set for the period prior to this date. The analysis was therefore restricted to emergency admissions occurring between July 1, 1994 and December 31, 1997. Emergency admissions of Melbourne residents to hospitals outside the Melbourne region were excluded, as were admissions of non-residents of the Melbourne region to Melbourne hospitals.

The diagnostic categories examined were grouped according to the International Classification of Diseases version 9 (ICD-9), and included asthma (ICD-9 493), ischaemic heart disease (ICD-9 410-413), all respiratory (ICD-9 460-519) and all cardiovascular (ICD-9 390-459). Admissions due to influenza (ICD-9 487) were also included for modelling purposes. Four age groups were examined; 0-14 years, 15-64 years, 65+ years and all ages. For asthma admissions the analysis was restricted to 0-14 years and all ages, and in the case of ischaemic heart disease, only the all ages group was considered.

4.1.2 Air Pollution Data

Air pollution data were obtained from the Environment Protection Authority (EPA Victoria). The pollutants considered in the analysis included particles (measured by nephelometry, bsp, 24-hour average, 1-hour maximum), ozone (8-hour maximum, 4-hour maximum, 1-hour maximum), nitrogen dioxide (24-hour average, 1-hour maximum) and carbon monoxide (8-hour maximum, 1-hour maximum). Measurements for ozone were collected from 7 sites within the study region, for nitrogen dioxide and bsp from 6 stations, and for carbon monoxide from 4 sites in the region. Missing data from individual sites were estimated by adjusting the mean of the non-missing sites for each day that was missing with the 3-month seasonal mean of the missing site over the 3-month seasonal mean of the network. Daily concentrations for each pollutant were then calculated by averaging across the network. Measurements taken on the same day of admission, the previous day (lag 1), and the day 2 days prior to admission (lag 2) were considered in the analysis, as were cumulative averages over the same day and

previous 2 days (3-day average), and over the same day and previous 4-days (5-day average).

4.1.3   Meteorological Data

Data on temperature (minimum, maximum and average) and dew point temperature (minimum and maximum) were obtained from EPA Victoria at each of the sites in the study region for which pollutant data was obtained. Same day, lag 1 and lag 2 as well as 3- and 4-day cumulative averages for each of the meteorological variables were considered. Rainfall data (daily total) was obtained from the Australian Bureau of Meteorology.

4.2   Statistical Analysis

The statistical approach used to detect the short-term effects of air pollution on daily hospital admissions in Melbourne utilised Generalised Additive Models (GAM) (Hastie and Tibshirani, 1990) using LOESS smoothing. Separate models were built for each of the outcomes considered. The approach followed that used by the APHEA group in Europe (see for example, Katsouyanni et al., 1996), and the analysis was performed in consultation with the APHEA statistical group. The optimal trend for time was selected using a combination of tools including partial autocorrelation functions, fitted and residual plots, t-statistics and to some degree, Akaike's Information Criterion (AIC). This combination of tools minimised the chance of overspecifying the model. To simplify the selection process, the choice of smoothing parameter was restricted to periods of 365, 275, 182.5, 90, 60 and 40 days. In the case of admissions in school-age children, a smoothed school term variable was found to be of more use than the trend for time. Day of week was included in all models. A dummy variable representing local public holidays and admissions for influenza were included in the models where significant.

Relationships between meteorological variables and each of the outcomes were examined using residual plots. Their inclusion was a step-wise process, with the meteorological variable illustrating the strongest

relationship in the residual plots included first. The steps in the model-building process are outlined below.

Step 1 Smoothed function of time: The optimal trend for time was selected using a combination of tools including partial autocorrelation functions, fitted and residual plots, t-statistics and to some extent, the AIC. The AIC optimises the smoothing function by indicating whether the decrease in deviance resulting from the addition of a new variable is worth the use of additional degrees of freedom (Hoek et al. 1997). However, sole use of the AIC in selecting the best trend for time can result in overspecification of the model, so less weight was given to the AIC in this step of the model selection process. To simplify the selection process, the choice of smoothing parameter was restricted to periods of 365, 275, 182.5, 90, 60 and 40 days.

Step 2 Other temporal variables: Hospital admissions vary considerably over the course of a week, therefore day of week was included in all models. In the case of admissions in school-age children, a smoothed school term variable was found to be of more use than the trend for time described in Step 1. A dummy variable representing local public holidays was included in the model where significant.

Step 3 Influenza: Influenza can have a major influence on hospital admissions, and as its occurrence is unrelated to daily variations in air pollution it is important to control for it. In this analysis influenza admissions were included in the model where they made a significant contribution to the model fit.

Step 4 Meteorological variables: Relationships between meteorological variables and each of the outcomes were examined using residual plots. Their inclusion was a step-wise process, with the meteorological variable illustrating the strongest relationship in the residual plots included first. The optimal lag and smoothing parameter for each variable was selected using the AIC. Not all of the meteorological variables examined were included – although each model included at least one temperature and a dew point temperature variable.

Rain was included only where it was found to be significantly related to the outcome. In a number of models it was necessary to control for one or two days following January 1, 1996, on which the study area received 80 mm of rain, and was followed by a day of relative cool (15°C in mid summer), moist conditions. This resulted in lower admissions the day after, but much higher admissions two days after the heavy rain. It is most likely that the variation of admissions was due to the inclement conditions (with patients delaying seeking treatment or affected by the sudden onset of cooler conditions), although some other factor such as an increase in mould and fungi attributable to the heavy rain should not be discounted.

Step 5 Control for outliers: In cases where the model created in the first 4 steps did not adequately control for days with extremely high admission counts, dummy variables representing these days were added to the model. This ensures that any relationship found between air pollution and the outcome is not influenced by admissions occurring on just one or two days over the study period.

Step 6 Addition of pollutants: Each of the pollutants was then added as a linear term to a Poisson regression model (all results are reported as linear terms).

Step 7 Multi-pollutant models: In an attempt to determine whether any of the relationships observed in Step 6 were independent investigations were performed using two-pollutant models. Only the most significant of either the same day or lagged concentrations were considered in this step - cumulative averages were not included.
The approach used in this analysis included the two pollutants in the same model (the strongest lag for each pollutant), regardless of the level of correlation between the two variables. While there are problems with this approach (including highly correlated variables in the same model can result in unstable estimates), it is the approach used most often in studies overseas. Results from this step should therefore be treated with caution.

Step 8: Seasonal investigations: Seasonal analyses were performed to determine whether the pollutant effects varied by season. In this analysis seasonal dummy variables and season-pollutant interaction terms were added to two separate models – one for the warm season and the other for the cool season. The cool season (April-October) corresponds to the winter smog season in Melbourne, which is characterised by high particle levels. The warm season on the other hand (November-March) is characterised by high O_3 levels, corresponding to the summer smog season.

5.   RESULTS

5.1   Descriptive Statistics

5.1.1   Health Data

Descriptive statistics for the admission categories considered in the analysis are presented in Table 1. Over the study period an average of 66 Melbourne residents per day were admitted to Melbourne hospitals with a respiratory condition, just over a quarter of which were attributable to asthma. In 0-14 year old children, who made up one third of all respiratory admissions, almost half of the respiratory admissions were due to asthma. An average of 84 persons per day were admitted for cardiovascular conditions, the majority of whom were over the age of 65 years.

Admissions for all diagnostic categories were slightly higher on average in the cool season, although for asthma and ischaemic heart disease (IHD) the seasonal differences were only slight.

Figures 2 to 5 show the time-series for the major admission groups examined (all-ages only) (see Appendix A for other variables). Strong seasonal variation is apparent for respiratory (Figure 2) and cardiovascular admissions (Figure 3), but the seasonal patterns for asthma (Figure 4) and IHD (Figure 5) are more complex. In Figure 4, the troughs occurring over December/January of each year are the result of reduced admissions in school holiday periods.

Table 1   Daily hospital admissions, Melbourne, July 1994 – December 1997

	Whole study period				Cool season[a]				Warm season[b]			
	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max
Asthma 0-14	9.65	4.64	0	29	9.94	4.18	1	26	9.21	5.22	0	29
Total asthma	18.47	6.45	3	52	19.60	5.92	7	38	16.78	6.83	3	52
Respiratory 0-14	21.93	8.92	2	54	25.53	8.03	7	54	16.58	7.38	2	40
Respiratory 15-64	19.86	6.19	4	54	21.59	6.24	5	54	17.29	5.15	4	37
Respiratory 65+	24.08	8.37	8	68	26.96	8.78	10	68	19.80	5.41	8	38
Total Respiratory	65.87	18.32	21	132	74.08	16.99	35	132	53.68	12.53	21	105
Cardiovas. 0-64	27.93	6.14	11	51	28.47	6.09	12	51	27.13	6.13	11	46
Cardiovas. 65+	56.11	9.34	29	88	58.45	9.17	33	88	52.65	8.48	29	79
Total cardiovas.	84.04	12.53	47	129	86.91	12.09	48	129	79.78	11.94	47	112
Total IHD	31.25	6.23	12	56	32.06	6.07	17	56	30.04	6.27	12	52

Data source: Department of Human Services, Victoria
[a]April-October
[b]November-March

Figure 2   Hospital Admissions for Respiratory Disease, all ages, Melbourne,
July 1994 – December 1997
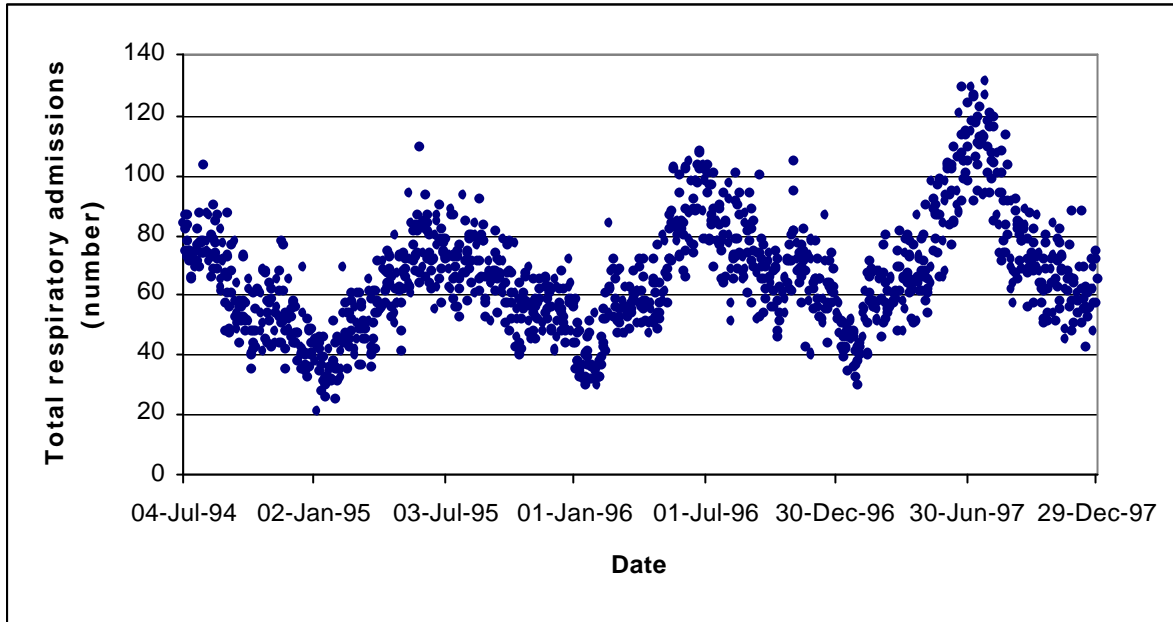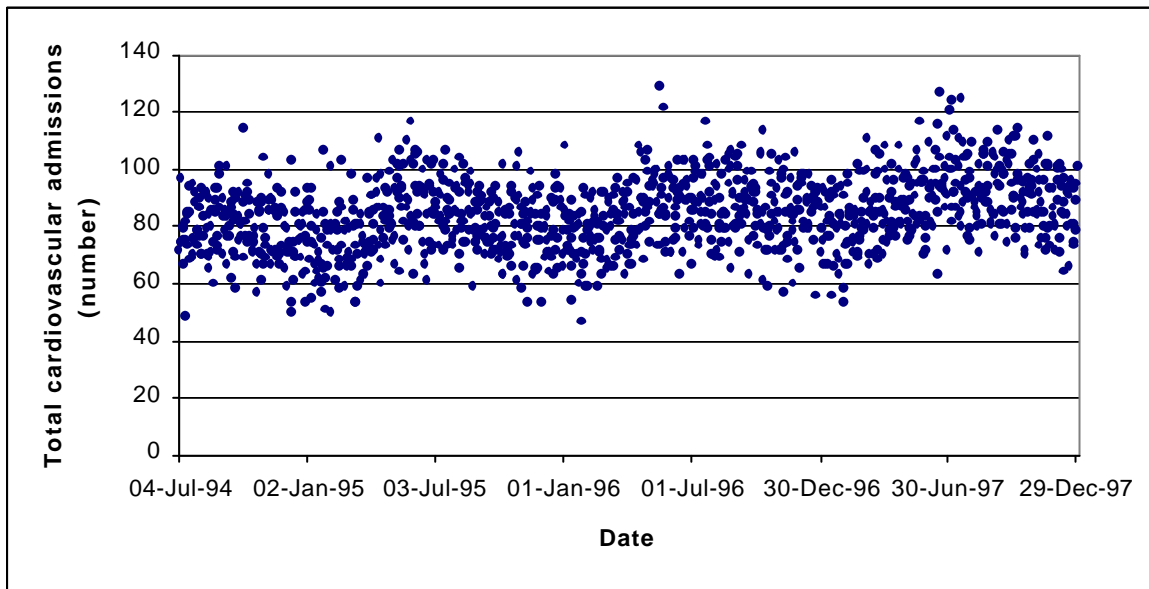

Figure 3   Hospital Admissions for Cardiovascular Disease, all ages, Melbourne,
July 1994 – December 1997

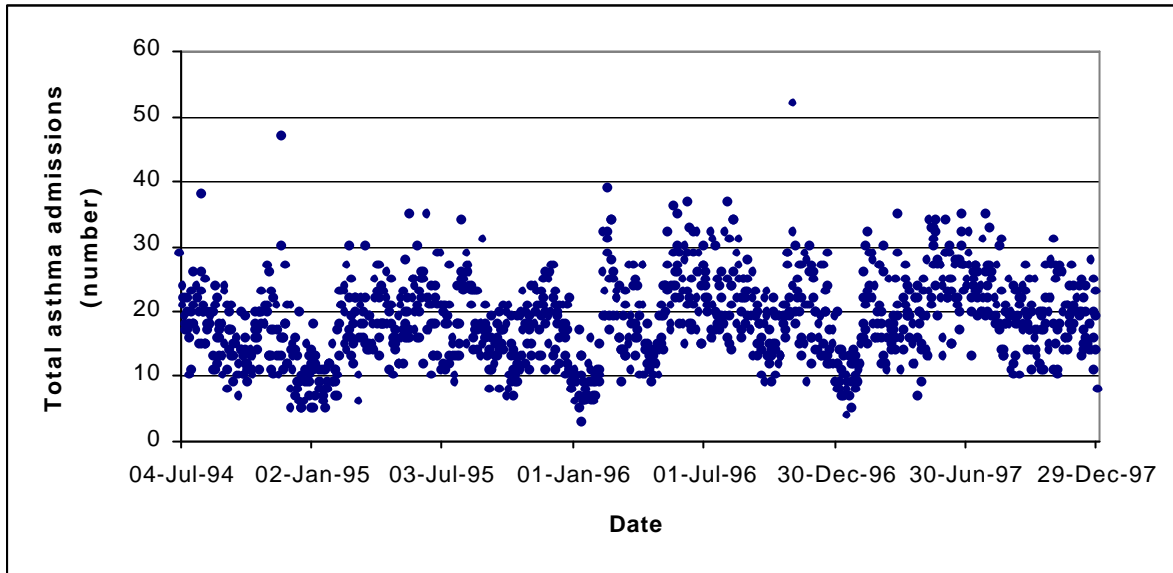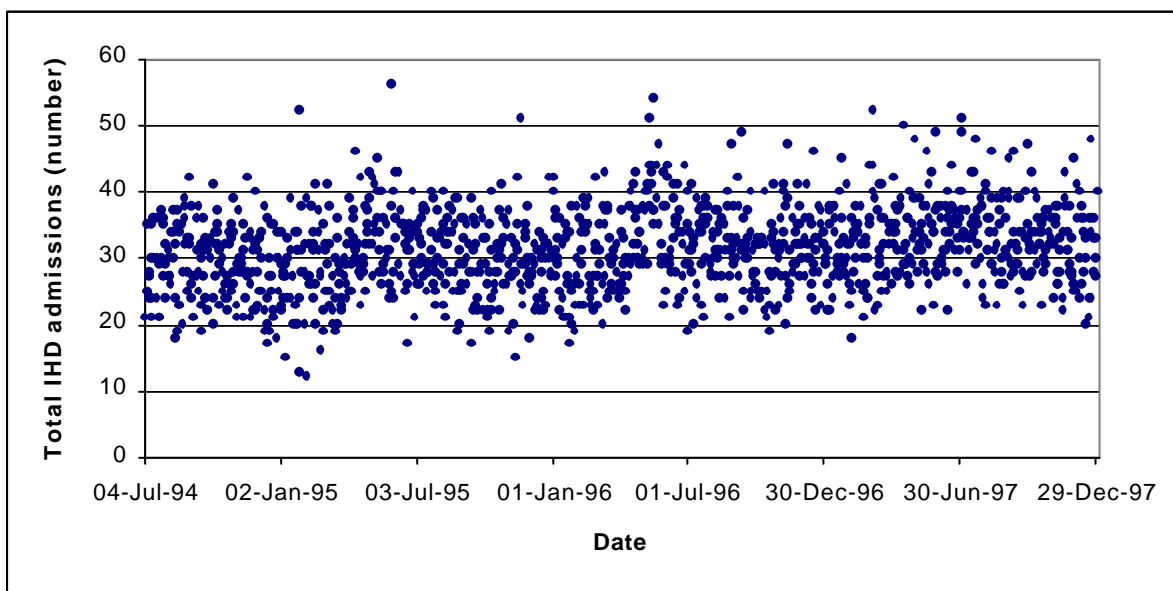Figure 4   Hospital Admissions for Asthma, all ages, Melbourne,
July 1994 – December 1997

Figure 5   Hospital Admissions for Ischaemic Heart Disease, all ages, Melbourne,
July 1994 – December 1997

5.1.2   Pollutant Data

Ozone was monitored at 7 sites within the study area: Alphington, Dandenong, Footscray, Point Cook, Paisley, Brighton and Box Hill (see Figure 1). Correlations between levels collected at these sites were very high, ranging from 0.76 to 0.94. The highest O_3 concentrations (all averaging times) during the study period were measured at Point Cook. For site specific results see Appendix A.

Nitrogen dioxide and particles were measured at 6 sites: Alphington, Dandenong, Footscray, Point Cook, Paisley and Box Hill. Correlations between sites ranged from 0.37 to 0.83 for NO_2 and 0.63 to 0.92 for bsp. Peak concentations for bsp were measured at Alphington, while for NO_2, highest levels for the 1-hour concentration were recorded at Footscray. Although the 24-hour measure for NO_2 was on average highest at Alphington, peak levels were recorded at Pt Cook and Paisley.

Carbon monoxide was recorded at 3-sites in the study area: Alphington, Footscray and Box Hill. Correlations between pollutant levels recorded at these sites ranged between 0.72 and 0.90. Highest levels were recorded at Alphington.

The descriptive statistics for the network averages are presented in Table 2. Levels of O_3 were higher in the warm season while NO_2, CO and particles, bsp, were on average higher in the cool season.

High correlations were observed between the network averages for bsp, NO_2 and CO, with coefficients ranging 0.5-0.8. Ozone on the other hand was not highly correlated with other pollutants, with the strongest relationship (a moderate 0.32) found between the 1-hour concentration and NO_2 (Table 3a).

Table 2   Mean daily pollutant concentrations (network average), Melbourne, July 1994 – December 1997

	Whole study period				Cool season[a]				Warm season[b]			
	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max
O_3 (ppb)												
8 hour	21.79	8.89	0.99	77.57	19.57	6.07	0.99	56.14	25.07	11.13	11.14	77.57
4 hour	24.65	9.99	2.01	87.86	22.28	6.10	2.01	63.00	28.18	13.13	11.57	87.86
1 hour	26.35	11.11	2.00	97.57	23.66	6.07	2.00	67.57	30.35	15.02	10.73	97.57
Particles,bsp $(10^{-4}m^{-1})$												
24 hour	0.24	0.23	0.03	2.00	0.27	0.27	0.03	2.00	0.19	0.13	0.03	1.25
1 hour	0.55	0.48	0.07	3.26	0.66	0.54	0.07	3.26	0.40	0.31	0.08	2.73
NO_2 (ppb)												
24 hour	11.35	4.62	2.47	27.29	13.03	4.34	2.87	27.29	8.85	3.83	2.47	24.67
1 hour	22.90	8.39	5.17	64.29	25.12	7.28	6.50	64.29	19.6	8.83	5.17	52.15
CO (ppm)												
8 hour	0.92	0.75	0.10	5.68	1.15	0.86	0.10	5.68	0.58	0.32	0.10	2.35
1 hour	1.51	1.19	0.17	9.33	1.88	1.33	0.20	9.33	0.95	0.60	0.17	3.57

[a]April-October
[b]November-March

However, network correlation coefficients were found to vary significantly by season. Tables 3b and 3c show the correlation coefficient matrices for the cool and warm season respectively. Ozone in particular shows a dramatic variation by season, with moderate to high positive correlations with the other pollutants observed in the warm season, compared with mostly negative correlations found in the cool season.

It should be noted that NO_2, particles and CO all arise from combustion processes and are therefore highly correlated. As these pollutants have common sources it is important to note that separating the effects of these individual pollutants can be difficult and it may be that one of these pollutants is acting as a surrogate for another. The results presented in this report should be interpreted with this in mind. Figures 6 to 14 illustrate the temporal variations present in the pollutant data over the study period.

Table 3a   Network average correlation coefficients, Melbourne, July 1994-December 1997*

	4 hour O_3	1 hour O_3	24 hour NO_2	1 hour NO_2	24 hour bsp	1 hour bsp	8 hour CO	1 hour CO
8 hour O_3	0.98	0.96	-0.03[ns]	0.21	-0.13	-0.15	-0.25	-0.23
4 hour O_3		0.99	0.06[a]	0.29	0.06[a]	-0.07[a]	-0.17	-0.15
1 hour O_3			0.11	0.32	-0.01[ns]	-0.03[ns]	-0.13	-0.11
24 hour NO_2				0.90	0.64	0.69	0.75	0.77
1 hour NO_2					0.56	0.61	0.64	0.68
24 hour bsp						0.93	0.70	0.66
1 hour bsp							0.78	0.75
8 hour CO								0.96

* all coefficients significant at p=0.0001 unless indicated
[a] significant at p<0.05
[b] significant at p<0.01
[c] Significant at p<0.001
[ns] not significant

Table 3b   Network average correlation coefficients, Melbourne,
July 1994-December 1997: Cool season*

	4 hour O_3	1 hour O_3	24 hour NO_2	1 hour NO_2	24 hour bsp	1 hour bsp	8 hour CO	1 hour CO
8 hour O_3	0.97	0.92	-0.41	-0.15	-0.45	-0.44	-0.47	-0.44
4 hour O_3		0.98	-0.26	-0.004[ns]	-0.37	-0.36	-0.36	-0.32
1 hour O_3			-0.17	0.08[a]	-0.30	-0.29	-0.29	-0.26
24 hour NO_2				0.89	0.68	0.71	0.73	0.75
1 hour NO_2					0.59	0.64	0.67	0.69
24 hour bsp						0.93	0.72	0.69
1 hour bsp							0.81	0.79
8 hour CO								0.96

* April-October, all coefficients significant at p=0.0001 unless indicated
[a] significant at p<0.05
[b] significant at p<0.01
[c] Significant at p<0.001
[ns] not significant

Table 3c   Network average correlation coefficients, Melbourne, July 1994-December 1997: Warm season*

	4 hour O_3	1 hour O_3	24 hour NO_2	1 hour NO_2	24 hour bsp	1 hour bsp	8 hour CO	1 hour CO
8 hour O_3	0.99	0.97	0.61	0.68	0.48	0.41	0.37	0.34
4 hour O_3		0.99	0.63	0.69	0.50	0.44	0.39	0.36
1 hour O_3			0.64	0.69	0.51	0.44	0.40	0.36
24 hour NO_2				0.93	0.60	0.57	0.78	0.76
1 hour NO_2					0.56	0.55	0.72	0.72
24 hour bsp						0.92	0.48	0.41
1 hour bsp							0.47	0.41
8 hour CO								0.91

* November-March, all coefficients significant at p=0.001

Figure 6   Time series: 8-hour Ozone (network average), Melbourne, July 1994-December 1997
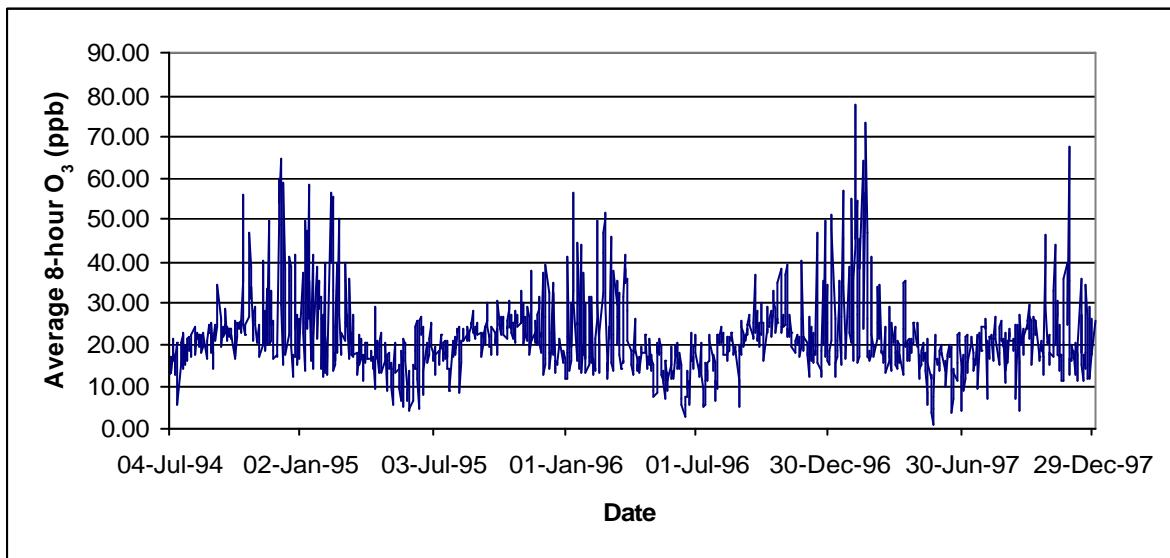
Figure 7   Time series: 4-hour Ozone (network average), Melbourne, July 1994-December 1997
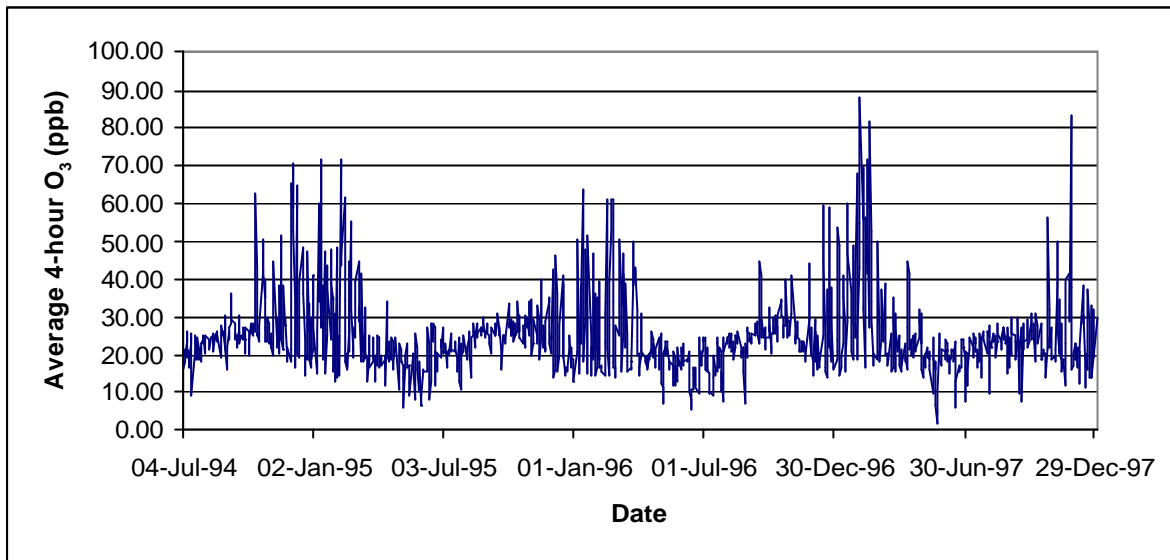
Figure 8   Time series: 1-hour Ozone (network average), Melbourne, July 1994-December 1997
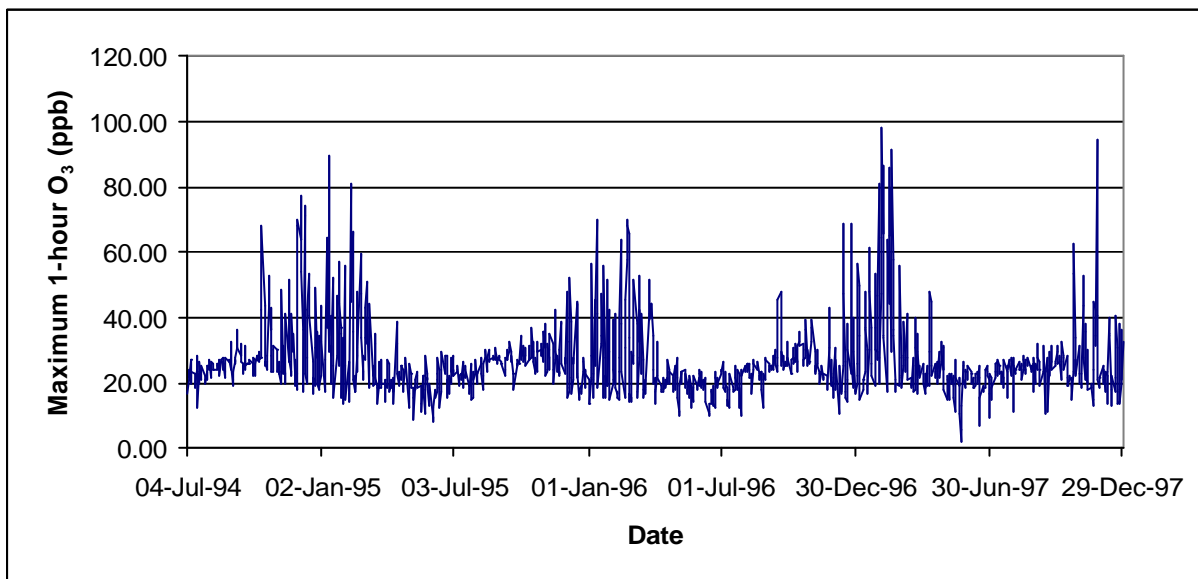
Figure 9   Time series: 24-hour bsp (network average), Melbourne, July 1994-December 1997
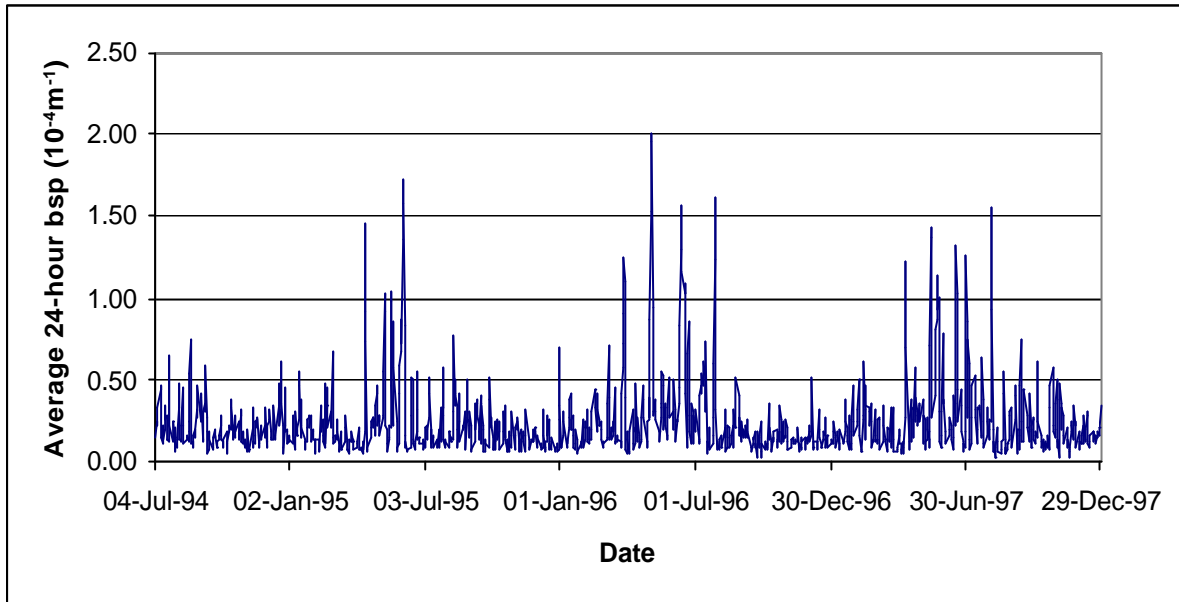
Figure 10   Time series: 1-hour bsp (network average), Melbourne, July 1994-December 1997
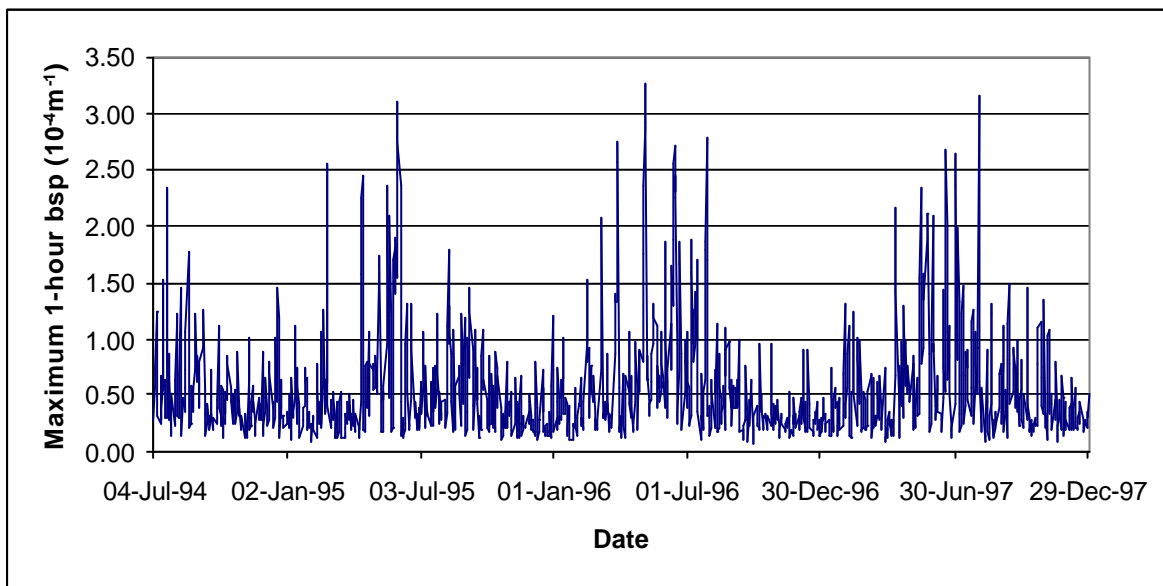
Figure 11   Time series: 24-hour Nitrogen Dioxide (network average), Melbourne, July 1994-December 1997
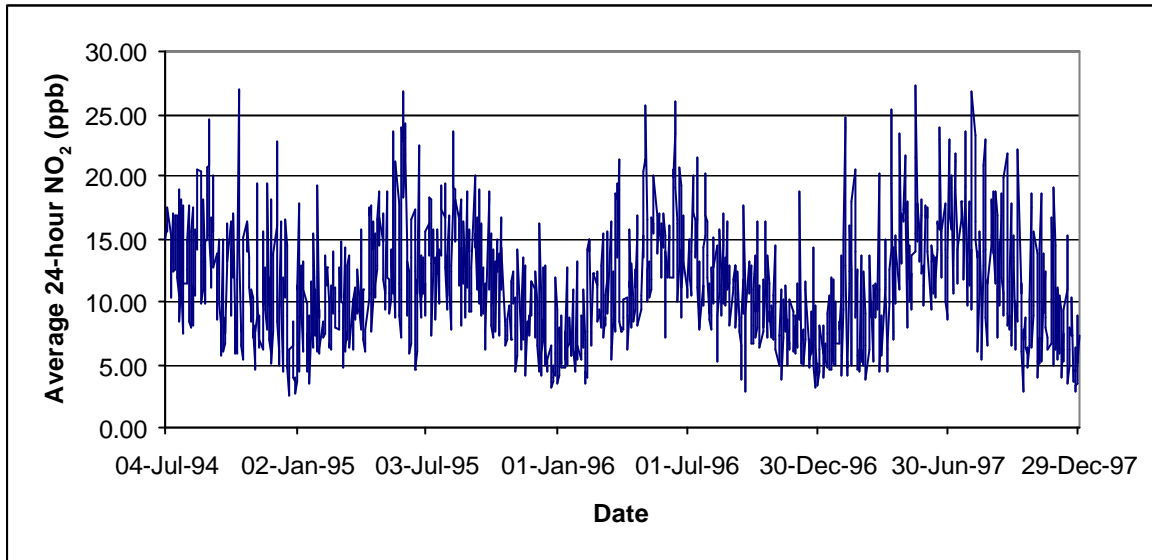
Figure 12   Time series: 1-hour Nitrogen Dioxide (network average), Melbourne, July 1994-December 1997
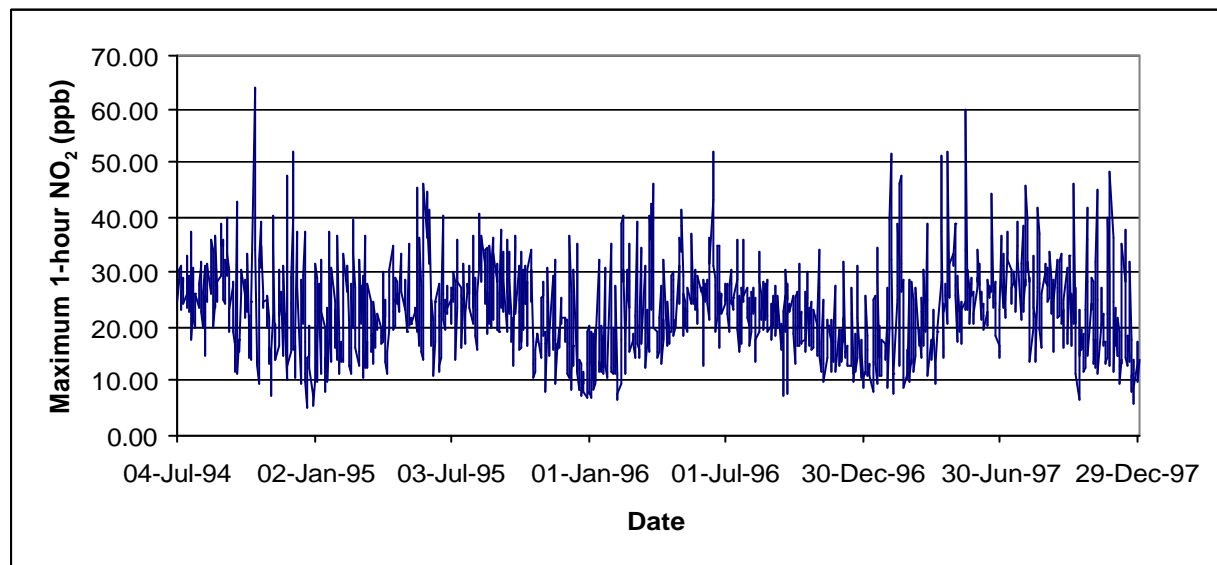
Figure 13   Time series: 8-hour Carbon Monoxide (network average), Melbourne, July 1994-December 1997
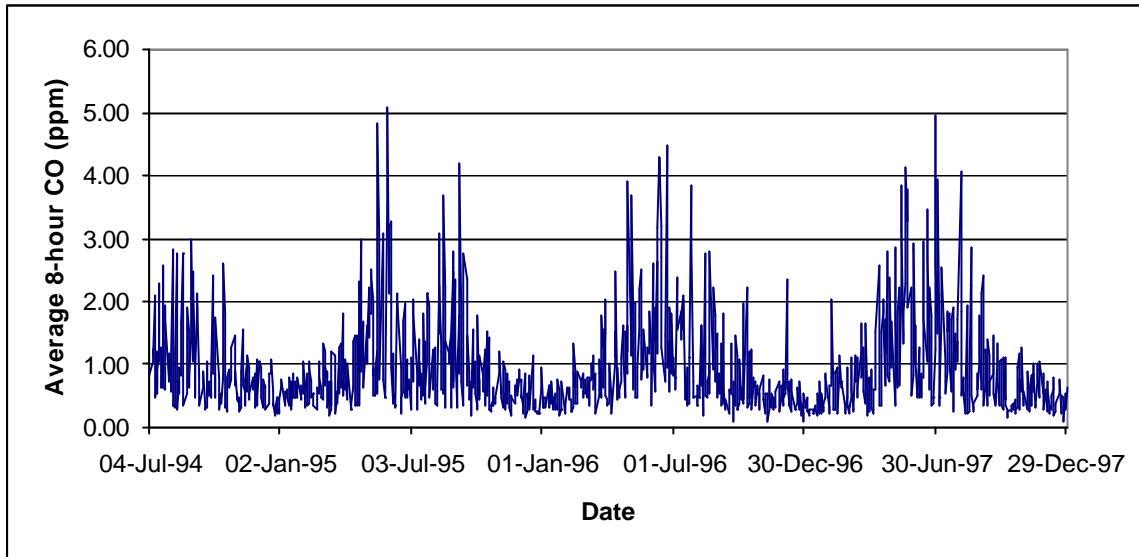
Figure 14   Time series: 1-hour Carbon Monoxide (network average), Melbourne, July 1994-December 1997
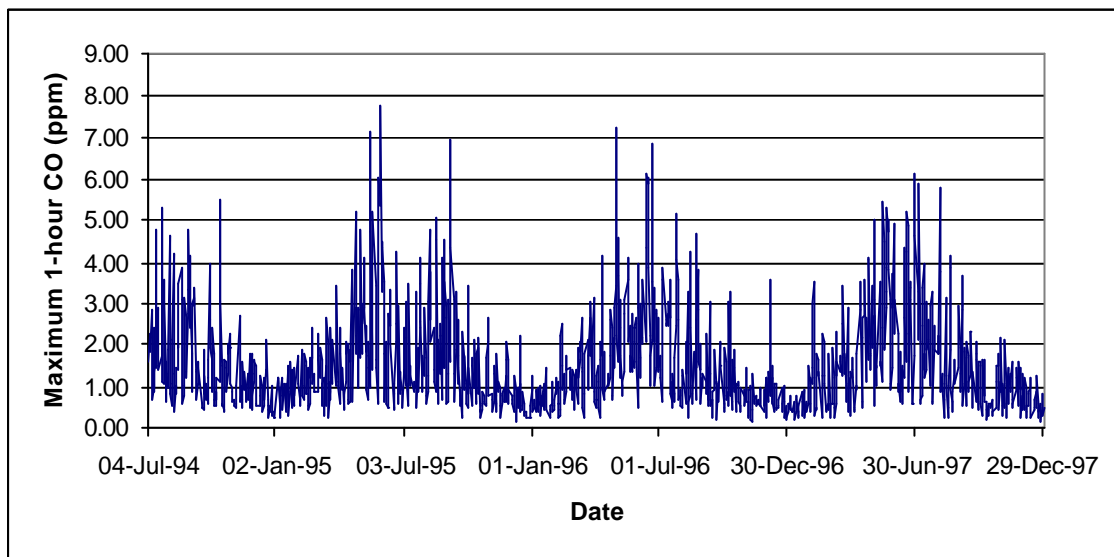
5.1.3 Meteorological Data

Descriptive statistics for the meteorological variables considered in the analysis are presented in Table 4. While the average temperatures indicate relatively mild conditions in Melbourne, the extremes in both the maximum and minimum temperatures illustrate the range of conditions that Melbourne residents experience.

5.2 Pollutant Effects

5.2.1 Ozone

The ozone analysis was conducted using data from the Alphington, Brighton, Dandenong, Box Hill, Footscray, Paisley and Pt. Cook air monitoring stations. These stations provided a good spatial coverage of the Melbourne Statistical Sub-Division. Data from Mt. Cottrell could not be included as O_3 data is only collected during the summer at this site. The data from the CBD site were not included due to discontinuity in the station operation leading to large amounts of missing data during the study period. This situation arose due to relocation of the air monitoring station.

Analysis was conducted using 1-hour, 4-hour and 8-hour maximum O_3 data from each of the monitoring

stations. The data was averaged across the sites to obtain a network average. Table 5 presents the results for O_3 analysis for each of the health outcomes considered. The results are reported as the relative risk (RR) of admission (with 95% confidence limits) associated with a 1 ppb increase in O_3. Only the strongest of the associations found (whether positive or negative) for each concentration are presented due to the large number of pollutant concentrations and lag periods examined in the analysis (for complete tables of results see Appendix B1).

The results of the analysis show significant ($p<0.05$) positive associations between O_3 and admissions for respiratory disease in the 65+ years and all-ages groups. Associations between O_3 levels and admissions for cardiovascular disease, ischaemic heart disease and asthma were not statistically significant (Table 5). In the 65+ years age group only 1-hour and 4-hour maximum concentrations showed significant associations with respiratory admissions. Relationships between each of the O_3 concentrations (1-hour, 4-hour and 8-hour maximum) and respiratory admissions in these age groups were most consistent at lag 2. That is, pollution levels occurring 2 days prior to admission showed the strongest effect.

Table 4   Meteorological variables, Melbourne, July 1994 – December 1997

	Whole study period				Cool season[a]				Warm season[b]			
	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max
Min temp (°C)	9.78	4.06	-1.44	25.60	7.59	3.02	-1.44	16.95	13.02	3.16	5.00	25.60
Max temp (°C)	19.10	5.84	7.10	41.48	16.08	3.75	7.10	32.80	23.57	5.52	11.62	41.48
Average temp (°C)	14.34	4.55	4.22	33.54	11.77	2.90	4.22	23.50	18.15	3.81	9.08	33.54
Dew point min (°C)	6.34	3.24	-2.05	17.15	4.86	2.49	-2.05	13.35	8.53	2.97	2.00	17.15
Dew point max (°C)	10.75	3.38	2.95	20.85	9.07	2.48	2.95	17.40	13.24	2.98	6.20	20.85
Rain (mm)	1.66	4.93	0	80.40	1.69	3.96	0	36.60	1.60	6.10	0	80.40

[a]April-October
[b]November-March

A 1 ppb increase in 4-hour O_3 (lagged 2 days) was associated with a 0.16% increase in the risk of admission in the 65+ age group and a 0.14% increase in the risk of admission in the all ages group.

Multi-pollutant analyses were conducted to determine whether the relationships identified for O_3 in the single pollutant models remained after controlling for other pollutants. In this study only respiratory admissions in the 65+ and all-ages groups had significant associations with O_3 in the whole year models.

The size and significance of effects for O_3 in these outcomes were found to reduce slightly when controlling for NO_2 but were not significantly altered by controlling for CO or fine particles (measured as bsp) in the model. Results of the multi-pollutant analyses are shown in Appendix B2.

Table 5 Relative risk of admission per 1 ppb increase in O_3, with 95% confidence intervals

Admissions category	Averaging Time	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 years	8-hour	3-day average	0.9976	0.9956-0.9996
	4-hour	3-day average	0.9986	0.9966-1.0006
	1-hour	same day	0.9992	0.9980-1.0004
Respiratory 15-64 years	8-hour	5-day average	0.9993	0.9970-1.0017
	4-hour	lag 2	1.0005	0.9991-1.0019
	1-hour	lag 2	1.0005	0.9991-1.0019
Respiratory 65+ years	8-hour	lag 2	1.0014	0.9998-1.0030
	4-hour	lag 2	1.0016	1.0002-1.0030
	1-hour	lag 2	1.0015	1.0003-1.0027
Respiratory all ages	8-hour	lag 2	1.0014	1.0006-1.0022
	4-hour	lag 2	1.0014	1.0006-1.0022
	1-hour	lag 2	1.0013	1.0005-1.0021
Asthma 0-14 years	8-hour	lag 1	0.9978	0.9955-1.0002
	4-hour	lag 1	0.9985	0.9965-1.0005
	1-hour	5-day average	1.0021	0.9992-1.0051
Asthma all ages	8-hour	lag 1	0.9983	0.9966-1.0000
	4-hour	lag 1	0.9989	0.9973-1.0005
	1-hour	lag 1	0.9993	0.9979-1.0007
Cardiovascular 0-64 years	8-hour	5-day average	0.9963	0.9944-0.9983
	4-hour	5-day average	0.9968	0.9950-0.9986
	1-hour	5-day average	0.9971	0.9955-0.9987
Cardiovascular 65+ years	8-hour	5-day average	0.9981	0.9965-0.9997
	4-hour	5-day average	0.9985	0.9972-0.9999
	1-hour	5-day average	0.9987	0.9973-1.0001
Cardiovascular all ages	8-hour	5-day average	0.9976	0.9964-0.9988
	4-hour	5-day average	0.9981	0.9969-0.9993
	1-hour	5-day average	0.9982	0.9972-0.9992
Ischaemic heart disease All ages	8-hour	5-day average	0.9966	0.9948-0.9984
	4-hour	5-day average	0.9972	0.9956-0.9988
	1-hour	5-day average	0.9974	0.9958-0.9990

A seasonal analysis was conducted for all of the health outcomes considered. The results of this analysis revealed significant positive associations with O_3 in the warm season across most of the admission categories investigated (Table 6). Significant associations were found for respiratory admissions in the 0-14 years, 65+ years and all-ages groups. Admissions for asthma in the 0-14 years and all ages group, and admissions for cardiovascular disease and ischaemic heart disease in the 65+ age group were also significantly associated with O_3 in the warm season. While the 5-day cumulative average concentrations showed the strongest associations across the range of outcomes examined, significant relationships were also found for other averaging times and at other lag periods. No significant positive associations were observed for any of the admission categories in the cool season.

Table 6   Seasonal analysis: relative risk of admission per 1 ppb increase in O_3, with 95% confidence intervals

Admissions category	Averaging Time	Cool season			Warm season		
		Pollutant lag	Relative Risk	95% CI	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 years	8-hour	5-day av	0.9896	0.9865-0.9927	5-day av	1.0050	1.0013-1.0088
	4-hour	5-day av	0.9907	0.9876-0.9939	5-day av	1.0044	1.0013-1.0076
	1-hour	5-day av	0.9907	0.9874-0.9940	5-day av	1.0040	1.0013-1.0068
Respiratory 15-64 years	8-hour	5-day av	0.9924	0.9891-0.9957	3-day av	1.0021	0.9996-1.0047
	4-hour	5-day av	0.9939	0.9904-0.9974	3-day av	1.0018	0.9996-1.0040
	1-hour	5-day av	0.9937	0.9902-0.9972	3-day av	1.0013	0.9993-1.0033
Respiratory 65+ years	8-hour	5-day av	0.9944	0.9915-0.9973	lag 2	1.0025	1.0007-1.0043
	4-hour	5-day av	0.9961	0.9930-0.9992	lag 2	1.0021	1.0005-1.0037
	1-hour	same day	0.9980	0.9957-1.0004	lag 2	1.0018	1.0004-1.0032
Respiratory all ages	8-hour	5-day av	0.9943	0.9926-0.9961	5-day av	1.0039	1.0021-1.0057
	4-hour	5-day av	0.9958	0.9939-0.9978	5-day av	1.0032	1.0016-1.0048
	1-hour	5-day av	0.9961	0.9942-0.9981	5-day av	1.0026	1.0012-1.0040
Asthma 0-14 years	8-hour	3-day av	0.9871	0.9826-0.9915	5-day av	1.0072	1.0027-1.0118
	4-hour	3-day av	0.9896	0.9851-0.9940	5-day av	1.0062	1.0025-1.0100
	1-hour	3-day av	0.9906	0.9862-0.9951	5-day av	1.0056	1.0023-1.0090
Asthma all ages	8-hour	5-day av	0.9868	0.9833-0.9903	5-day av	1.0054	1.0021-1.0088
	4-hour	5-day av	0.9879	0.9842-0.9916	5-day av	1.0045	1.0016-1.0075
	1-hour	5-day av	0.9883	0.9844-0.9922	5-day av	1.0038	1.0013-1.0064
Cardiovascular 0-64 years	8-hour	same day	0.9960	0.9937-0.9984	5-day av	0.9965	0.9940-0.9990
	4-hour	same day	0.9968	0.9947-0.9990	5-day av	0.9970	0.9949-0.9992
	1-hour	same day	0.9975	0.9952-0.9999	5-day av	0.9972	0.9953-0.9992
Cardiovascular 65+ years	8-hour	5-day av	0.9962	0.9941-0.9984	lag 2	1.0015	1.0003-1.0027
	4-hour	5-day av	0.9966	0.9945-0.9988	lag 2	1.0012	1.0002-1.0022
	1-hour	5-day av	0.9967	0.9946-0.9989	lag 2	1.0010	1.0000-1.0020
Cardiovascular All ages	8-hour	5-day av	0.9965	0.9947-0.9983	5-day av	0.9982	0.9966-0.9998
	4-hour	5-day av	0.9970	0.9952-0.9988	5-day av	0.9984	0.9970-0.9998
	1-hour	5-day av	0.9972	0.9954-0.9990	5-day av	0.9985	0.9973-0.9997
Ischaemic heart disease all ages	8-hour	5-day av	0.9939	0.9912-0.9966	same day	0.9988	0.9974-1.0002
	4-hour	5-day av	0.9950	0.9923-0.9977	5-day av	0.9982	0.9962-1.0002
	1-hour	5-day av	0.9951	0.9924-0.9978	5-day av	0.9982	0.9964-1.0000

AMBIENT AIR POLLUTION AND DAILY HOSPITAL ADMISSIONS IN MELBOURNE

SUMMARY

The results of this study have shown that ambient levels of ozone in Melbourne are associated with increases in hospital admissions for respiratory disease. The strongest associations were observed in the all ages and 65+ years age groups. No significant associations were observed for cardiovascular disease or asthma in whole year models. Seasonal analysis revealed significant positive associations between ozone and admissions for respiratory disease (0-14 years, 65+ years, all ages), asthma (0-14 years, all ages) and cardiovascular disease (65+ years) in the warm season. No significant associations were found in the cool season. Results of the multi-pollutants analysis indicated that these effects were independent of particles and carbon monoxide but were sensitive to the inclusion of NO_2 in the model.

KEY FINDINGS

1. Ambient O_3 levels are associated with daily hospital admissions in Melbourne.

2. The strongest associations are observed for admissions for respiratory disease in whole year models.

3. The observed effects are greatest in the warm season.

4. Associations were also observed for admissions for asthma and cardiovascular disease in the warm season.

5. The results of this study are consistent with studies overseas and in Australia.

5.2.2   Fine Particles (measured as bsp)

The particle analysis was conducted using nephelometry data from the Alphington, Dandenong, Box Hill, Footscray, Paisley and Pt. Cook air monitoring stations. Data from the Brighton and CBD sites could not be included in the analysis due to a large amount of missing data during the study period. Both 1-hour maximum and 24-hour average bsp concentrations were used in the analysis.

The results of this study show that fine particles were significantly (p<0.05) associated with admissions for respiratory disease (15-64 years, 65+ years), asthma (0-14 years, all ages), cardiovascular disease (65+ years, all ages) and ischaemic heart disease (all ages) (Table 7). For respiratory admissions in the 15-64 age group, significant associations were observed across most of the lag periods and averaging times considered, with the strongest associations occurring at the 3-day cumulative average. A unit ($1 \times 10^{-4} m^{-1}$) increase in 1-hour bsp (equivalent to 15 µg/m^3 PM$_{2.5}$) was associated with a 3.8% increase in risk of admission for respiratory disease in this age group. In the 65+ age group, bsp was associated with a 7.5% and 4.3% increase in risk of admission for the 24-hour and maximum 1-hour concentrations respectively. No significant associations were found for respiratory admissions in the 0-14 years or all ages groups.

In the case of asthma, strong associations were observed across most of the lag periods and averaging times examined in both the 0-14 years and all ages groups. In the 0-14 years age group, a unit increase in same day 24-hour bsp was associated with a 14.8% increase in risk of admission (Table 7). In the all ages category the strongest associations were found with the 5-day cumulative average, where a unit increase in the 24-hour concentration was associated with a 13.9% increase in risk of admission for asthma.

Admissions for cardiovascular disease and ischaemic heart disease also showed strong, consistent associations with particles in this study, in particular the 24-hour concentration (Table 7). While only marginally significant associations were found in the 0-64 years age group, admissions for cardiovascular disease in the 65+ years and all ages groups were consistently associated with bsp levels (1-hour maximum and 24-hour average) across most of the pollutant lag periods and averaging times examined (same day, lag 1 and 3-day average). The 24-hour bsp concentration (lagged 1 day) was associated with a 5.6% and 4.6% increase in risk of admission for cardiovascular disease in the 65+ years and all ages groups, respectively, per unit increase in bsp. Strongest associations for the maximum 1-hour particle concentration were found with the 3-day cumulative average, with a 3.5% and 2.7% increase in risk of admission per unit increase in bsp for cardiovascular disease in the 65+ years age group and all ages group respectively. Similar results were obtained for ischaemic heart disease, with strongest associations found for same day bsp levels (Table 7).

The results of the multi-pollutant analysis showed that many of the significant associations observed between hospital admissions and fine particles in the single pollutant analysis were reduced in size and significance after controlling for the effects of other pollutants, in particular NO$_2$ and CO (see Appendix B). Controlling for NO$_2$ in the model resulted in a reduction in the size and significance of the association between bsp and admissions for each of the outcomes with which it was significantly associated in the single-pollutant analysis (Table 7). The one exception was cardiovascular admissions in the all ages group where the significance of the particle effect was retained after controlling for NO$_2$.

A slight reduction in the size of the effect estimate was observed. Including CO in the model also resulted in a loss of the particle effect associated with admissions for respiratory and cardiovascular disease (including IHD). However, for asthma admissions the particle effect was retained after controlling for CO in the model. Positive associations between particles and respiratory admissions in the 65+ years age group were retained after controlling for O_3 in the model, suggesting that the impacts of these pollutants are independent for this outcome.

Table 7 Relative risk of admission per unit increase (1×10^{-4} m^{-1}) in bsp with 95% confidence intervals

Admissions category	Averaging Time	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 years	24-hour 1-hour	lag 2 same day	0.9811 0.9940	0.9360-1.0283 0.9700-1.0187
Respiratory 15-64 years	24-hour 1-hour	3-day average 3-day average	1.0784 1.0383	1.0121-1.1491 1.0063-1.0714
Respiratory 65+ years	24-hour 1-hour	5-day average 5-day average	1.0745 1.0430	1.0041-1.1499 1.0088-1.0783
Respiratory all ages	24-hour 1-hour	same day same day	1.0239 1.0119	0.9927-1.0561 0.9967-1.0273
Asthma 0-14 years	24-hour 1-hour	same day same day	1.1481 1.0592	1.0628-1.2403 1.0197-1.1002
Asthma all ages	24-hour 1-hour	5-day average 5-day average	1.1394 1.0766	1.0582-1.2268 1.0374-1.1172
Cardiovascular 0-64 years	24-hour 1-hour	same day same day	1.0424 1.0190	0.9949-1.0921 0.9963-1.0422
Cardiovascular 65+ years	24-hour 1-hour	lag 1 3-day average	1.0560 1.0352	1.0208-1.0924 1.0143-1.0565
Cardiovascular all ages	24-hour 1-hour	lag 1 3-day average	1.0461 1.0274	1.0174-1.0757 1.0104-1.0446
Ischaemic heart disease	24-hour 1-hour	same day same day	1.0631 1.0297	1.0188-1.1093 1.0090-1.0509

Table 8 shows the results of the seasonal investigation of the associations between hospital admissions and particle pollution in Melbourne. Strong cool season effects were found for respiratory admissions in the 15-64 years age group, admissions for cardiovascular disease (0-64 years, 65+ years, all ages), ischaemic heart disease (all ages) and admissions for asthma (0-14 years, all ages). Significant associations were found across most of the lags and averaging times considered for these outcomes. Effects were also found in the cool season for respiratory admissions in the 65+ and all ages groups, however effects in the warm season for these outcomes were stronger and more consistent. Same day 24-hour bsp was also significantly associated with asthma (all ages) in the warm season. No significant seasonal effects were found for respiratory admissions in the 0-14 years age group. A complete table of the results can be found in Appendix B.

Table 8   Seasonal analysis: relative risk of admission per unit ($1 \times 10^{-4}\,\mathrm{m}^{-1}$) increase in bsp, with 95% confidence intervals

Admissions category	Averaging Time	Cool season			Warm season		
		Pollutant lag	Relative Risk	95% CI	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 years	24-hour 1-hour	lag 2 lag 1	0.9875 1.0104	0.9397-1.0377 0.9844-1.0370	5-day av lag 1	1.1640 0.9673	0.9163-1.4788 0.9034-1.0358
Respiratory 15-64 years	24-hour 1-hour	3-day av same day	1.0837 1.0428	1.0125-1.1600 1.0120-1.0745	lag 2 5-day av	1.0563 0.9682	0.8944-1.2476 0.8643-1.0846
Respiratory 65+ years	24-hour 1-hour	5-day av 5-day av	1.0678 1.0412	0.9931-1.1481 1.0040-1.0799	lag 1 lag 1	1.2247 1.0827	1.0513-1.4267 1.0165-1.1533
Respiratory all ages	24-hour 1-hour	same day same day	1.0269 1.0177	0.9938-1.0610 1.0010-1.0345	5-day av 5-day av	1.2389 1.0675	1.0817-1.4188 1.0030-1.1361
Asthma 0-14 years	24-hour 1-hour	same day 3-day av	1.1401 1.0715	1.0481-1.2401 1.0193-1.1265	same day same day	1.2131 1.0573	0.9857-1.4930 0.9713-1.1509
Asthma all ages	24-hour 1-hour	5-day av 5-day av	1.1361 1.0872	1.0496-1.2297 1.0438-1.1324	same day same day	1.2031 1.0293	1.0277-1.4084 0.9639-1.0992
Cardiovascular 0-64 years	24-hour 1-hour	lag 1 lag 1	1.0543 1.0305	1.0015-1.1099 1.0039-1.0577	lag 2 lag 1	0.9203 0.9663	0.8029-1.0548 0.9150-1.204
Cardiovascular 65+ years	24-hour 1-hour	lag 1 lag 1	1.0588 1.0306	1.0215-1.0974 1.0119-1.0495	5-day av same day	0.9578 1.0127	0.8300-1.1054 0.9741-1.0528
Cardiovascular all ages	24-hour 1-hour	lag 1 lag 1	1.0489 1.0254	1.0183-1.0804 1.0103-1.0408	same day 5-day av	1.0295 0.9790	0.9547-1.1102 0.9291-1.0316
Ischaemic heart disease	24-hour 1-hour	same day same day	1.0680 1.0315	1.0203-1.1179 1.0081-1.0554	5-day av 5-day av	0.7940 0.8975	0.6563-0.9606 0.8239-0.9778

AMBIENT AIR POLLUTION AND DAILY HOSPITAL ADMISSIONS IN MELBOURNE

SUMMARY

The results of this study show that fine particles in Melbourne are associated with hospital admissions for respiratory disease, with the strongest effects observed in the elderly, 0-64 years and all ages groups. Strong associations were also found for admissions for asthma in children (0-14 years) and all ages groups.

Admissions for cardiovascular disease in the elderly and all ages groups, and ischaemic heart disease were also strongly associated with exposure to particles. The strongest effects were observed in the cool season, however associations were observed for admissions for asthma (all ages) and respiratory disease (all ages and 65+ age group) in the warm season.

KEY FINDINGS

1. Ambient particle levels are associated with daily hospital admissions in Melbourne.

2. The strongest associations are observed for admissions for respiratory disease and asthma.

3. Associations are also observed for admissions for cardiovascular disease in the elderly.

4. The observed effects are greatest in the cool season, although effects were also observed for admissions for asthma and respiratory disease in the warm season.

5. The results of this study are consistent with studies overseas and in Australia.

5.2.3 Nitrogen Dioxide

The NO_2 analysis was conducted using data from the Alphington, Dandenong, Box Hill, Footscray, Paisley and Pt Cook air monitoring stations. The data from other stations were not included due to discontinuity in station operation and large amounts of missing data during the study period. Both 1-hour maximum and 24-hour average NO_2 concentrations were included in the analysis.

Of all the pollutants considered in this study, NO_2 showed the most consistent relationship with hospital admissions for the outcomes considered (Table 9). In whole-year models NO_2 was significantly associated with all of the outcomes examined across the majority of lags and averaging times. The associations observed for the 24-hour concentration showed the most consistent relationship (see Appendix B for a complete table of the results). Effects for 24-hour NO_2 (averaged over 5 days) ranged from a 0.78% increase in risk of admission for respiratory disease in the all ages group to 1.1% increase in risk of admission for respiratory disease in the 65+ year age group. For asthma admissions a 1 ppb increase in NO_2 was associated with a 1.18% increase in risk of admission in the 0-14 years age group and a 1.45% increase in risk of admission in the all ages group. Same day 24-hour NO_2 was

associated with a 0.37% increase in risk of admission for IHD and a 0.36% increase for cardiovascular admissions in the 0-64 age group per 1 ppb increase in NO_2. The 3-day average concentration was associated with a 0.45% and 0.4% increase in risk of admission for cardiovascular disease in the 65+ and all ages groups respectively (Table 9).

Controlling for CO in the multi-pollutant analysis removed the observed associations between NO_2 and cardiovascular admissions (65+ years and all ages groups). Controlling for O_3 reduced the size of the NO_2 effects although the significance ($p < 0.05$) was retained in relationships with respiratory admissions in the 65+ and all ages groups. Controlling for particles in the model resulted in a reduction in the size and loss of the significance of the NO_2 effect in most outcomes. The exceptions were respiratory admissions in the 65+ year age group and cardiovascular admissions in the all ages groups. For both of these outcomes only a slight reduction in effect size occurred and the significance of the effect was retained. The results of the multi-pollutant analysis should be interpreted with caution due to the high correlation between pollutants making it difficult to separate effects of individual pollutants.

Table 9   Relative risk of admission per 1 ppb increase in NO_2, with 95% confidence intervals

Admissions category	Averaging Time	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 years	24-hour 1-hour	5-day average 5-day average	1.0079 1.0025	1.0038-1.0121 0.9999-1.0051
Respiratory 15-64 years	24-hour 1-hour	5-day average 5-day average	1.0084 1.0045	1.0043-1.0126 1.0020-1.0071
Respiratory 65+ years	24-hour 1-hour	5-day average 5-day average	1.0110 1.0048	1.0070-1.0149 1.0025-1.0072
Respiratory all ages	24-hour 1-hour	5-day average 5-day average	1.0078 1.0043	1.0055-1.0102 1.0029-1.0057
Asthma 0-14 years	24-hour 1-hour	5-day average 5-day average	1.0118 1.0048	1.0058-1.0177 1.0013-1.0084
Asthma all ages	24-hour 1-hour	5-day average 5-day average	1.0145 1.0059	1.0099-1.0191 1.0032-1.0087
Cardiovascular 0-64 years	24-hour 1-hour	same day same day	1.0036 1.0013	1.0011-1.0062 0.9999-1.0027
Cardiovascular 65+ years	24-hour 1-hour	3-day average 3-day average	1.0045 1.0020	1.0023-1.0067 1.0008-1.0032
Cardiovascular all ages	24-hour 1-hour	3-day average 3-day average	1.0040 1.0017	1.0022-1.0058 1.0007-1.0027
Ischaemic heart disease	24-hour 1-hour	same day same day	1.0037 1.0012	1.0013-1.0061 1.0000-1.0025

Results of the seasonal analysis for NO_2 are shown in Table 10. Strong positive associations were observed between both 1-hour maximum and 24-hour average NO_2 with all outcomes in the cool season.

The strongest associations were observed for the 24-hour concentration. Significant effects were also found in the warm season for respiratory admissions in the 0-14 years, 65+ years and all ages groups, and admissions for asthma (0-14 years, all ages). No significant associations in the warm season were found for admissions for cardiovascular disease or ischaemic heart disease.

Table 10   Seasonal analysis: relative risk of admission per 1 ppb increase in NO_2, with 95% confidence intervals

Admissions category	Averaging Time	Cool season			Warm season		
		Pollutant lag	Relative Risk	95% CI	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 years	24-hour 1-hour	3-day av 3-day av	1.0063 1.0017	1.0018-1.0109 0.9990-1.0045	5-day av 5-day av	1.0229 1.0087	1.0113-1.0346 1.0036-1.0139
Respiratory 15-64 years	24-hour 1-hour	5-day av 5-day av	1.0110 1.0061	1.0054-1.0165 1.0026-1.0097	3-day av 3-day av	1.0035 1.0027	0.9961-1.0110 0.9994-1.0061
Respiratory 65+ years	24-hour 1-hour	5-day av 5-day av	1.0143 1.0075	1.0091-1.0195 1.0042-1.0109	lag 1 lag 1	1.0046 1.0023	0.9993-1.0099 1.0000-1.0046
Respiratory all ages	24-hour 1-hour	5-day av 5-day av	1.0082 1.0045	1.0053-1.0112 1.0025-1.0065	5-day av 5-day av	1.0129 1.0064	1.0069-1.0189 1.0039-1.0090
Asthma 0-14 years	24-hour 1-hour	Same day Same day	1.0094 1.0034	1.0037-1.0152 1.0000-1.0067	5-day av 5-day av	1.0285 1.0115	1.0139-1.0433 1.0051-1.0178
Asthma all ages	24-hour 1-hour	5-day av 5-day av	1.0128 1.0042	1.0070-1.0186 1.0005-1.0080	5-day av 5-day av	1.0215 1.0085	1.0106-1.0326 1.0038-1.0133
Cardiovascular 0-64 years	24-hour 1-hour	3-day av 3-day av	1.0050 1.0026	1.0009-1.0092 0.9999-1.0052	lag 2 lag 1	0.9966 0.9986	0.9921-1.0011 0.9966-1.0006
Cardiovascular 65+ years	24-hour 1-hour	3-day av lag 1	1.0049 1.0018	1.0020-1.0079 1.0004-1.0032	lag 2 lag 1	1.0017 1.0010	0.9984-1.0050 0.9996-1.0024
Cardiovascular all ages	24-hour 1-hour	3-day av 3-day av	1.0047 1.0024	1.0024-1.0071 1.0008-1.0040	3-day av lag 2	1.0010 1.0006	0.9975-1.0045 0.9994-1.0018
Ischaemic heart disease	24-hour 1-hour	3-day av 3-day av	1.0064 1.0028	1.0025-1.0104 1.0004-1.0052	5-day av 5-day av	0.9942 0.9977	0.9874-1.0011 0.9948-1.0006

AMBIENT AIR POLLUTION AND DAILY HOSPITAL ADMISSIONS IN MELBOURNE

SUMMARY

Strong significant positive associations were found between NO_2 and daily hospital admissions for respiratory and cardiovascular disease in this study. Effects were particularly strong for the 24-hour concentration, which was significant at most lags for most of the outcomes considered in both whole-year and cool season models. Positive associations were observed for all outcomes considered, with the strongest effects observed for admissions for asthma in the 0-14 years and all ages groups. The strongest effects were observed in the cool season but significant effects were also observed for admissions for asthma and respiratory disease in the warm season. The results of the multi-pollutant analysis indicate that the associations observed for NO_2 and the outcomes under consideration were not independent of the effects of other pollutants.

KEY FINDINGS

1. Ambient NO2 levels are associated with daily hospital admissions in Melbourne.

2. The strongest associations are observed for admissions for respiratory disease and asthma.

3. Associations are also observed for admissions for cardiovascular disease in the elderly.

4. The observed effects are greatest in the cool season, although effects were also observed for admissions for asthma and respiratory disease in the warm season.

5. The results of this study are consistent with studies overseas and in Australia.

5.2.4   Carbon Monoxide

The analysis for CO was conducted using data from three air monitoring stations within the study area; Alphington, Box Hill and Footscray. CO levels were on average highest in Alphington and lowest at Footscray. (See Appendix A for site-specific information). Both 1-hour and 8-hour maximum CO concentrations were used in the analysis.

Carbon monoxide was found to be associated with all outcomes considered in this study, with the exception of admissions for respiratory disease in the 0-14 age group, where positive but non-significant relationships were found (Table 11). The most consistent relationships were found for cardiovascular admissions in the 65+ years and all ages groups, with significant associations found at all of the lag periods and averaging times considered (see Appendix B). A 1 ppm increase in 3-day average 8-hour CO was associated with a 3.29% and 2.72% increase in risk of admission for cardiovascular disease in the 65+ and all ages groups respectively (Table 11). A 3.68% and 2.3% increase in admissions for ischaemic heart disease was associated with a 1 ppm increase in 1-hour maximum and 8-hour maximum CO respectively.

Table 11   Relative risk of admission per 1 ppm increase in CO, with 95% confidence intervals

Admissions category	Averaging Time	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 year	8-hour	lag 1	1.0082	0.9916-1.0252
	1-hour	lag 1	1.0056	0.9950-1.0163
Respiratory 15-64 year	8-hour	3-day av	1.0328	1.0098-1.0564
	1-hour	3-day av	1.0195	1.0050-1.0342
Respiratory 65+ year	8-hour	5-day av	1.0305	1.0069-1.0546
	1-hour	5-day av	1.0210	1.0059-1.0363
Respiratory all ages	8-hour	lag 1	1.0090	0.9992-1.0190
	1-hour	5-day av	1.0101	1.0010-1.0192
Asthma 0-14 years	8-hour	3-day av	1.0606	1.0274-1.0948
	1-hour	3-day av	1.0310	1.0100-1.0524
Asthma all ages	8-hour	5-day av	1.0639	1.0363-1.0922
	1-hour	5-day av	1.0398	1.0222-1.0577
Cardiovascular 0-64 years	8-hour	3-day av	1.0248	1.0043-1.0457
	1-hour	same day	1.0118	1.0021-1.0215
Cardiovascular 65+ years	8-hour	3-day av	1.0329	1.0185-1.0476
	1-hour	3-day av	1.0205	1.0113-1.0297
Cardiovascular all ages	8-hour	3-day av	1.0272	1.0154-1.0391
	1-hour	3-day av	1.0173	1.0098-1.0250
Ischaemic heart disease	8-hour	3-day av	1.0368	1.0180-1.0558
	1-hour	3-day av	1.0227	1.0107-1.0348

For asthma in the 0-14 year age group, a 1 ppm increase in 3-day average 8-hour maximum CO was associated with a 6.1% increase in risk in admission. The increased risk of admission associated with a 1ppm increase in the 3-day average 1-hour maximum concentration was 3.1%. Significant associations between CO and respiratory admissions in the 15-64 years age group were found across most of the lag periods and averaging times considered. The strongest association was observed with the 3-day average concentration, with a 3.3% and 2% increase in risk of admission found in this age group for a 1 ppm increase in the 8-hour and 1-hour CO concentration respectively.

The results of the multi-pollutant analysis showed that controlling for the other pollutants in the CO model resulted in a reduction in the size and significance of the CO effect for admissions for respiratory disease and asthma. For asthma admissions in the 0-14 year age group and respiratory admissions the CO effect was completely removed (ie. effect size reduced dramatically and no longer significant) after controlling for NO_2 and particles. For cardiovascular admissions however the observed association between CO and admissions for cardiovascular disease and ischaemic heart disease generally retained significance although the effect size was in some cases reduced by the inclusion of NO_2 or bsp. In the outcomes where O_3

was also examined (respiratory 65+ years and all ages), the CO effect was unchanged after controlling for the effects of O_3. Table 12 shows the results of the seasonal analysis for CO. Significant cool season effects were found for all of the outcomes considered, including respiratory admissions in the 0-14 years age group which was not found to be significantly associated with CO in whole-year models. A 1 ppm increase in 1-hour maximum CO (lag 1) in the cool season was associated with a 1.13% increase in risk of admission for respiratory disease in the 0-14 years age group. Significant positive associations were also found for respiratory admissions in the 65+ years and all ages groups, asthma admissions in the 0-14 years age group and cardiovascular admissions in the 0-64 years age group in the warm season. Marginally significant associations were also found for respiratory admissions in the 0-14 years age group, asthma admissions (all ages), cardiovascular admissions (all ages) and admissions for ischaemic heart disease in the warm season.

As for the other pollutants, the risk associated with a 1 ppm increase in CO was larger in the warm season compared with the cool season, although the cool season effects were more consistent across the range of lag periods and averaging times considered, particularly for cardiovascular admissions (see Appendix B).

Table 12 Seasonal analysis: relative risk of admission per 1 ppm increase in CO, with 95% confidence intervals

Admissions category	Averaging Time	Cool season			Warm season		
		Pollutant lag	Relative Risk	95% CI	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 years	8-hour 1-hour	lag 1 lag 1	1.0147 1.0113	0.9974-1.0324 1.0000-1.0226	5-day av 5-day av	1.1119 1.0616	0.9859-1.2541 0.9953-1.1323
Respiratory 15-64 years	8-hour 1-hour	3-day av 5-day av	1.0380 1.0263	1.0133-1.0633 1.0080-1.0450	5-day av lag 1	0.9615 1.0099	0.8629-1.0713 0.9746-1.0466
Respiratory 65+ years	8-hour 1-hour	5-day av 5-day av	1.0353 1.0237	1.0097-1.0616 1.0070-1.0407	3-day av 3-day av	1.0802 1.0537	0.9928-1.1751 1.0074-1.1021
Respiratory all ages	8-hour 1-hour	5-day av 5-day av	1.0202 1.0142	1.0047-1.0359 1.0041-1.0244	5-day av 5-day av	1.0907 1.0737	1.0180-1.1686 1.0350-1.1138
Asthma 0-14 years	8-hour 1-hour	3-day av 3-day av	1.0619 1.0373	1.0261-1.0990 1.0140-1.0611	5-day av 5-day av	1.2170 1.0890	1.0400-1.4242 1.0008-1.1850
Asthma all ages	8-hour 1-hour	5-day av 5-day av	1.0635 1.0391	1.0341-1.0938 1.0202-1.0585	5-day av lag 2	1.0832 1.0340	0.9589-1.2236 0.9960-1.0734
Cardiovascular 0-64 years	8-hour 1-hour	lag 1 3-day av	1.0204 1.0152	1.0035-1.0375 1.0010-1.0296	same day same day	1.0689 1.0375	1.0142-1.1265 1.0092-1.0666
Cardiovascular 65+ years	8-hour 1-hour	3-day av 3-day av	1.0312 1.0192	1.0157-1.0469 1.0090-1.0294	5-day av 5-day av	1.0373 1.0227	0.9752-1.1033 0.9901-1.0563
Cardiovascular all ages	8-hour 1-hour	lag 1 3-day av	1.0199 1.0160	1.0101-1.0297 1.0077-1.0244	5-day av same day	1.0410 1.0164	0.9899-1.0948 0.9998-1.0333
Ischaemic heart disease	8-hour 1-hour	3-day av 3-day av	1.0360 1.0224	1.0161-1.0563 1.0095-1.0356	5-day av same day	0.9658 1.0173	0.8902-1.0478 0.9908-1.0446

AMBIENT AIR POLLUTION AND DAILY HOSPITAL ADMISSIONS IN MELBOURNE

SUMMARY

Strong significant positive associations were found between CO and hospital admissions for all outcomes considered in this study except respiratory admissions in the 0-14 year age group. The strongest associations were found for admissions for cardiovascular disease in the elderly (65+ years) and all ages groups, admissions for ischaemic heart disease and admissions for asthma in the 0-14 year age group. The results of the seasonal analysis revealed that the associations were strongest in the cool season, although significant positive associations were also observed for respiratory admissions (65+ years and all ages), asthma admissions (0-14 years) and cardiovascular admissions in the warm season.

KEY FINDINGS

1. Ambient CO levels are associated with daily hospital admissions in Melbourne.

2. The strongest associations are observed for admissions for cardiovascular disease and ischaemic heart disaese.

3. Associations are also observed for admissions for asthma in children and respiratory disease.

4. The observed effects are greatest in the cool season, although effects were also observed for admissions for asthma, respiratory and cardiovascular disease in the warm season.

5. The results of this study are consistent with studies overseas and in Australia.

6. DISCUSSION

The findings of this study indicate a strong association between ambient air pollution and daily hospital admissions for cardiovascular and respiratory disease in Melbourne. The strongest effects are observed in the elderly and children. The results show that the pollutants considered are making significant contributions to the variation in daily admissions in both the cool and warm seasons.

6.1 Ozone

In this study O_3 was significantly associated with admissions mostly in the warm season, although associations were also found in whole-year models for respiratory admissions in the 65+ and all ages groups. Effects were most consistent with the 5-day cumulative lag. Significant negative effects were observed for all outcomes in the cool season. Where multi-pollutant analyses were performed (respiratory admissions in the 65+ and all ages groups) the observed O_3 effects were, in general, robust to the inclusion of other pollutants in the model indicating independent effects. These findings are consistent with other studies conducted in Australia (Petroeschevsky et al., 2001; Morgan et al., 1998b) and overseas (Moolgavkar et al., 2000a, b; Gwynn et al., 2000; Wong et al., 1999; Burnett et al., 1999, 1997; Anderson et al., 1998; Sunyer et al., 1997; Schwartz, 1994a,b, 1996).

There are many studies world-wide that have shown associations between O_3 and increases in hospital admissions for respiratory and cardiovascular disease (Moolgavkar et al., 2000a, b; Gwynn et al., 2000; Wong et al., 1999; Burnett et al., 1999, 1997; Anderson et al., 1998; Sunyer et al., 1997; Schwartz, 1994a,b, 1996). In Europe, significant associations between O_3 and admissions for asthma have been found in London (Anderson et al, 1998, Sunyer et al 1997), Barcelona (Sunyer et al 1997), Helsinki (Ponka and Virtanen 1996) and Rotterdam (Schouten et al 1996). Numerous studies throughout North America have also found associations between O_3 and

hospital admissions for asthma and other respiratory conditions (Thurston et al 1994, Schwartz 1994a,b, 1996, Burnett et al. 1997a, Moolgavkar et al 1997, Moolgavkar 2000b, Gwynn et al 2000).

Fewer studies have investigated the association between O_3 and admissions for cardiovascular disease. The results of these studies have been inconsistent. No significant positive association between O_3 and admissions for circulatory diseases was found in a London study (Poloniecki et al 1997) or in studies conducted in a number a number of US cities (Gwynn et al 2000, Linn et al 2000, Moolgavkar 2000a). However, a study in Hong Kong has found significant associations between O_3 and admissions for a range of cardiovascular and respiratory complaints (Wong et al. 1999). Cardiovascular admissions in the 65+ years and all ages groups were the two of the three outcomes to show a significant positive association with O_3 in a more recent London study (Atkinson et al., 1999). In the Melbourne study cardiovascular admissions in the elderly were the only outcome to show a significant positive association with O_3 and this was only observed in the warm season.

In Brisbane, significant associations were found between O_3 and admissions for asthma and respiratory conditions. No associations were found for cardiovascular conditions in the Brisbane study (Petroeschevsky et al, 2001). In Sydney, Morgan et al (1998) found positive non-significant associations between O_3 and admissions for COPD and heart disease in the elderly and for asthma admissions in the 15-64 year age group. In the Melbourne study associations were found for admissions for respiratory disease (0-14 years, 65+ years and all ages groups), asthma (0-14 years and all ages groups) and cardiovascular and ischaemic heart disease (65+ years) in the warm season, consistent with the findings of the Brisbane and Sydney studies.

The results of the current study show strong negative associations between O_3 and all of the outcomes considered in the cool season. O_3 is negatively

correlated with the other pollutants in the cool season. The strong negative associations produced by the regression models could be a reflection of these negative relationships. Negative associations between O_3 and hospital admissions have been reported elsewhere including London (Anderson et al 1998, Atkinson et al 1999) and Los Angeles (Linn et al 2000).

6.2 Particles

Strong, consistent associations between bsp and daily hospital admissions were found in this study. In particular, admissions for asthma (0-14 years, all ages), respiratory admissions in the 15-64 years age group and cardiovascular admissions in the 65+ years and all ages groups showed significant associations with bsp across most of the lag periods examined in whole year models. Strong associations were also observed in the cool season for each of these outcomes. For admissions for respiratory disease in the 65+ and all ages groups, associations with particles were strongest in the warm season, although cool season effects were also observed. Few positive associations were observed for cardiovascular admissions in the warm season.

In previous Australian studies, significant associations between particles and hospital admissions have been found in Brisbane and Sydney. In Brisbane, bsp was associated with admissions for respiratory disease in the 15-64 years and all ages groups. These effects were stronger in the spring/summer periods. No effects were observed for cardiovascular admissions (Petroeschevsky et al, 2001). In Sydney significant associations were found between bsp and admissions for heart disease. Relationships with asthma and COPD admissions were generally positive but not significant (Morgan et al., 1998b).

There have been many studies overseas that link exposure to particles with hospital admissions for respiratory and cardiovascular disease (Hruba et al., 2001; Braga et al., 2001; Petroeschevsky et al., 2001; Samet 2000a,b; Norris et al., 2000; Linn et al., 2000; Moolgavkar 2000; Schwartz, 2000; Hagen et al., 2000; Chen et al., 2000; Gwynn et al., 2000; Wong et al., 1999; Atkinson et al., 1999; Sheppard et al., 1999; Wordley et al., 1997). The results of the Melbourne study are consistent with these studies.

In the NMMAPS 14-city analysis of the association between PM_{10} and daily hospital admissions for cardiovascular disease, an 8.5% increase in admissions per 50 $\mu g/m^3$ increase in PM_{10} was observed for the 65+ age group. A study by Schwartz (1999), found consistent associations between PM_{10} and cardiovascular admissions for cardiovascualr disease in 8 US metropolitan areas, with effect estimates ranging from 1.8 to 4.2% per 25 $\mu g/m^3$ increase in PM_{10}. A recent study conducted in Los Angeles (Linn et al., 2000) found that PM_{10}, NO_2, and CO were all associated with admissions for cardiovascular disease in people 30 years and older.

Strong associations between particles and admissions for a range of respiratory outcomes have been found in London (Atkinson et al., 1999), Birmingham (UK) (Wordley et al., 1997), Birmingham (US) (Schwartz, 1994a), Detroit (Schwartz, 1994b), Los Angeles (Linn et al., 2000; Moolgavkar, 2000b) and a number of other US regions (Chen et al., 2000; Moolgavkar et al, 2000; Gwynn et al, 2000; Samet et al., 2000a,b). The effect estimates observed in the studies conducted in the US generally range between 5 to 25% per 50 $\mu g/m^3$ increase in daily PM_{10}, with larger effects observed for asthma than for admissions for COPD or pneumonia.

Sheppard et al., (1999) found strong positive associations between admissions for asthma in the non-elderly with PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ in Seattle.

The results of the Sheppard study are consistent with those observed in Melbourne where approximately a 10% increase in admissions for asthma in children (0-14 years) was associated with a 10 µg/m³ increase in 24-hour $PM_{2.5}$ (estimated from the relationship between $PM_{2.5}$ and bsp).

6.3   Nitrogen dioxide

Nitrogen dioxide was also found to be significantly associated with each of the outcomes examined in this study. Strong positive associations were found in whole-year models and in the cool season for each outcome. Effects were particularly strong for the 24-hour concentration, which was significant at most lags for most of the outcomes considered in both whole-year and cool season models. Significant warm season associations were also found for respiratory admissions in the 0-14 years, 65+ years and all ages groups and admissions for asthma (0-14 years, all ages groups).

Morgan et al (1998b) found strong support for an NO_2 association in their study of the health effects of air pollution in Sydney. NO_2 was found to be significantly associated with admissions for heart disease in each of the age groups considered, and for admissions for asthma in the 1-14 years age group. Relationships between NO_2 and the remainder of the respiratory groups considered were positive but not significant. In contrast, no significant relationships were found in Brisbane between NO_2 and admissions for respiratory or cardiovascular conditions (Petroeschevsky et al., 2001).

Several overseas studies have found associations between daily hospital admissions for respiratory and cardiovascular disease and ambient levels of NO_2. The results of the Melbourne study are consistent with the findings of the overseas studies. Significant positive associations for a range of respiratory and cardiovascular conditions have been found in the UK (Atkinson et al., 1999; Anderson et al., 1998; Poloniecki et al., 1997) Europe (Anderson et al., 1997; Sunyer et al., 1997) and the United States (Linn et al., 2000; Moolgavkar 2000a; Moolgavkar, 2000b). Significant associations for respiratory and cardiovascular admissions including COPD, asthma, pneumonia, and heart failure have also been found in Hong Kong (Wong et al 1999). The results of a study by Gwynn et al (2000) in Buffalo, New York, found no significant relationships between NO_2 and admissions for cardiovascular and respiratory disease, although a significant positive association was observed for total admissions. Similarly, Schwartz (1997) found no significant relationship between NO_2 and admissions for cardiovascular disease in Tuscon, Arizona. In a meta-analysis of four European cities, Spix et al (1998) found no association between NO_2 and admissions for respiratory disease. In a recent study conducted in Valencia, Spain, strong significant associations were found between admissions for cerebrovascular diseases and daily levels of NO_2 (Ballester et al., 2001). Associations were also observed in this study for Black Smoke, SO_2 and CO and admissions for cardiovascular disease, heart admissions and admissions for cerebrovascular disease.

Studies conducted in Australia have also shown mixed results for associations between daily NO2 levels and admissions for respiratory and cardiovascular disease (Petroeschevsky et al., 2001; Morgan et al., 1998b). Morgan et al., (1998b) found strong significant associations between NO_2 admissions for heart disease in each of the age groups considered, and for admissions for asthma in the 1-14 years age group. Relationships between NO_2 and the remainder of the respiratory groups considered were positive but not significant. In contrast, no significant relationships were found in Brisbane between NO_2 and admissions for respiratory or cardiovascular conditions, with little consistency found in the direction of association (Petroeschevsky et al., 2001).

6.4   Carbon Monoxide

In this study CO was significantly associated with most of the admissions categories examined in single pollutant models. The most consistent associations were found for admissions for cardiovascular disease and ischaemic heart disease. CO generally lost significance in two-pollutant models containing bsp or NO2, possibly indicating that the relationship observed in the single-pollutant models was simply a reflection of the high correlation that exists between CO and the actual pollutant responsible. However, for cardiovascular admissions the CO effect was generally retained.

The health effects of ambient levels of CO have not been studied extensively in Australia. However there is strong support for a link between CO and hospital admissions from studies conducted overseas, in particular cardiovascular admissions. Several overseas studies have found associations between admissions for cardiovascular disease and ambient CO levels. Significant associations have been found between CO and admissions for cardiovascular disease in numerous studies conducted throughout North America. In a study of 10 Canadian cities, Burnett et al (1997b) found CO was strongly associated with admissions for congestive heart failure in the elderly. The observed relationships were relatively stable after controlling for other pollutants and weather. Schwartz (1999) found significant effects for CO on cardiovascular admissions in 6 of the 8 US cities examined in this study. Again, the observed effects were independent of other pollutants and weather. Associations have also been found in Los Angeles (Linn et al 2000, Moolgavkar 2000a), Tuscon (Schwartz 1997), Reno-Sparks (Yang et al 1998) and Cook County (Moolgavkar 2000a). Studies conducted in London have also found CO to be associated with admissions for cardiovascular conditions (Poloniecki et al 1997, Atkinson et al 1999).

Only a few studies have examined the relationship between CO and admissions for respiratory conditions. In those that have, the results have been somewhat less consistent than those for cardiovascular disease. In a study conducted in London (Atkinson et al 1999), few significant associations were found between CO and admissions for respiratory diseases (although results were generally positive). Moolgavkar et al (2000b) studied the effects of CO along with other pollutants on admissions for COPD in King County, USA. While CO exhibited the strongest effects of the pollutants considered and remained stable in multi-pollutant models, the associations were only observed in the 0-19 years age group. No significant associations were found in the 20-64 years or 65+ years age groups. In a separate study Moolgavkar (2000a) found strong associations between CO and admissions for COPD in three metropolitan areas in the US – Cook County, Illinois; Los Angeles County, California; and Maricopa County, Arizona. Significant positive associations were found for CO in all ages group in Los Angeles County and in the elderly in both Maricopa and Cook Counties. Associations were also observed for CO, PM_{10} and NO_2 and admissions for cardiovascular disease. Linn et al., (2000) also found strong associations between CO and admissions for asthma and COPD in another Los Angeles study. However the authors concluded it was not possible to distinguish the effects of CO from PM_{10} and NO_2 on admissions for the range of cardiovascular and respiratory outcomes examined in that study.

In the current Melbourne study an association was also found between ambient CO levels and admissions for asthma in children. The study by Sheppard et al., (1999) in Seattle also shows an association between ambient CO and admissions for asthma in the non-elderly. An increase of 6% in admission rate for asthma was observed for a 1ppm increase in CO lagged 3 days.

In the current study a 1 ppm increase in the 3-day average 8-hour maximum CO concentration was associated with a 6% increase in admissions for asthma in children (0-14 years).

The mechanism by which CO impacts upon health may explain the stability of relationships found with cardiovascular conditions in this study and the relative instability of respiratory associations in multi-pollutant models. CO has no direct toxic effect on the lungs. Rather, CO binds with haemoglobin in the blood, thereby interfering with O_2 transport around the body, which has a direct impact upon the tissues that have high oxygen consumption such as the heart and brain (Bascom et al., 1996).

6.5   Coherence

Bates (1992) has highlighted the importance of coherence in epidemiological studies of this type used in this study. Coherence refers to the finding consistent associations for a particular health outcome in different types of studies within the same setting. For example, finding associations between particles and hospital admissions for respiratory disease as well as increases in daily mortality within the same city provides evidence of coherence.

The Melbourne Mortality Study examined the effects of O_3, bsp, NO_2 and CO on daily mortality due to respiratory, cardiovascular and all causes (< ICD-9 800) in the 65+ years and all ages groups for the period 1991-1996 (EPA Victoria, 2000). The results for O_3 in this admissions study closely reflect those of the mortality study, which found significant relationships in whole-year models for respiratory admissions in the 65+ years and all ages groups, along with all cause mortality in the 65+ years age group. Strong warm season associations were also observed for these outcomes and for all cause mortality in the all ages group (EPA Victoria, 2000).

NO_2 also shows a reasonable level of coherence, particularly for respiratory conditions and cardiovascular conditions in whole year models.

In the mortality study, NO_2 was found to be significantly associated with most of the outcomes considered in whole-year models, with significant associations also found in the warm season for each outcome in the 65+ years and all ages groups. However, no significant relationships were found in the cool season for the mortality outcomes (EPA Victoria, 2000), which is in contrast to the strong cool season associations found with admissions in this study.

In this study, significant associations were observed for admissions for respiratory and cardiovascular disease and asthma with particles in whole-year models and the cool season. Effects were also observed in the warm season for admissions for asthma and respiratory disease. In the Melbourne Mortality study significant positive associations between particles and respiratory mortality were found in the warm season while no significant relationships were observed in the cool season or in whole year models. Only a marginally significant association was observed for cardiovascular mortality in the 65+ years age group in whole year models, with no significant seasonal results (EPA Victoria, 2000).

For CO, relatively weak associations were found with daily mortality in Melbourne compared with the consistency of findings for admissions in this study. CO was significantly related to cardiovascular mortality (all ages) in whole year models, but did not show a strong seasonal effect with that outcome. Significant warm season results were however found for respiratory and all cause mortality (EPA Victoria, 2000). In this study significant positive associations were found for cardiovascular, respiratory and asthma admissions and CO in whole year models and in the cool season. Associations were also observed in the warm season for these outcomes and CO. A similar pattern, with the effects for morbidity being stronger than those observed for mortality, for CO has been found in London by Poloniecki and colleagues (1997), who suggest that the relationship

between CO and cardiovascular conditions is perhaps more consistent with morbidity than mortality.

Some of the differences observed in the mortality and admissions studies could be explained to some extent by what is termed 'mortality displacement'. Schwartz et al., (2000) have investigated whether the shortening of life is a matter of a few days or if it were a more significant length period. The results have shown that for COPD it appears that the shortening of life is displaced by a few weeks; for pneumonia, heart attacks and all cause mortality, the results suggest that longer time periods are involved. As admissions to hospital are a more sensitive health endpoint it is more likely to observe effects for these outcomes.

6.6   Identifying the pollutants responsible

In multi-pollutant analyses conducted in this study, many of the significant relationships identified in single-pollutant models were lost after controlling for other pollutants. In general, the effects of O_3 were independent of the other pollutants with the associations between O_3 and respiratory admissions were retained after controlling for bsp or CO. However the size of the effect estimates were reduced slightly after controlling for NO_2. The effects of CO were generally removed from respiratory models after controlling for other pollutants, but were retained for cardiovascular admissions. The particle effect was not affected by the inclusion of O_3 in the model, but was completely removed by the control of CO in many cases, with the exception of asthma admissions, where the effect was retained. NO_2 generally reduced the size and significance of the particle effect. Similarly, effects for NO_2 were reduced slightly after controlling for bsp and O_3, while the inclusion of CO in the NO_2 model removed the significant associations found for cardiovascular admissions.

These results suggest the relationships between CO and cardiovascular admissions, O_3 and respiratory admissions, particles and admissions for asthma, appear to be independent of the other pollutants considered. However, attributing the observed effects to one pollutant, especially where the pollutants are highly correlated as is the case for particles, CO and NO_2, is very difficult. In this situation it may be that the observed effects are attributable to the pollution mix of which a given pollutant is an indicator. Overall it appears that the effects attributed to ozone may be separated out from the effects of the other pollutants due to the low correlation between O_3 and these pollutants.

7.   CONCLUSION

The results of this study indicate ambient levels of air pollution are associated with increases in daily hospital admissions for respiratory and cardiovascular conditions in Melbourne. Associations were also observed for most of the pollutants considered and admissions for asthma. The elderly and children appear to be the most affected by these pollutants. While it is difficult to establish exactly which pollutants are responsible for these associations, there appears to be strong associations for all pollutants considered in this study and hospital admissions. The main sources of these pollutants in Melbourne are motor vehicles, industrial emissions, wood heaters and other combustion process. The results of this study are consistent with other studies conducted in Australia and overseas.

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