Research on Health and Air Pollution in Perth

Morbidity and Mortality: A Case-Crossover Analysis 1992-1997

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Glossary

ABS	Australian Bureau of Statistics
AMA	Australian Medical Association
APHEA	Air Pollution and Health European Approach
AQCC	Air Quality Co-ordinating Committee
AQMP	Air Quality Management Plan
Bsp	a coefficient with units of m^{-1} indicating the back scatter of light at a reference wavelength due to the presence of particles in the air. Local visual distance (in metres) can be determined from Bsp
CI	confidence interval
COPD	chronic obstructive pulmonary disease
CO	carbon monoxide
CVD	cardiovascular disease
DEP	Department of Environmental Protection
Fev1	forced expiratory volume in 1 second
FVC	forced vital capacity
HiVol	high volume air sampler
ICD9	International Classification of Diseases version 9
NEPM	National Environment Protection Measure
NO_2	nitrogen dioxide
O_3	ozone
PM2.5	particles each having an equivalent aerodynamic diameter of less then 2.5 microns
PM10	particles each having an aerodynamic diameter of less than 10 microns
ppm	parts per million by volume
ppb	parts per billion by volume
ROC	reactive organic compound
RR	relative risk
SO_2	sulfur dioxide
TEOM	tapered element oscillating microbalance
$\mu g/m^3$	micrograms per cubic metre, expressed at 0^oC Celsius and 1.0 atmosphere pressure (101.325 kilopascals).
WHO	World Health Organisation

Executive Summary

Studies around the world have reported significant relationships between air pollution levels and health effects including hospitalisation, exacerbation of existing diseases and premature mortality. Groups most susceptible to high levels of pollutants include children, the elderly and those with pre-existing illness. There have been few studies on the effects of air pollution on health in Australia, although recent studies in Sydney, Brisbane and Melbourne all report significant relationships between selected air pollutants and hospitalisation, supporting the findings reported in the international literature.

In 1998, a Western Australian Parliamentary Select Committee on Perth's Air Quality outlined its concerns about the potential health impacts of photochemical smog and particle haze in the Perth metropolitan region. Both photochemical smog (measured as ozone) and particle haze (measured as PM10) concentrations exceed national and international standards at various times through the year, ozone in summer and particulate haze in winter. Concentrations of sulfur dioxide (SO_2), nitrogen dioxide (NO_2) and carbon monoxide (CO) in Perth are all below national and international standards.

As part of the Government's response to the Parliamentary Select Committee report, an Air Quality Management Plan (AQMP) has been developed which, among many other issues, addresses the health impacts of air quality in Perth. Health research is an integral part of the plan to assist in decision making about priority areas for intervention and implementation of control and management strategies. As a first stage in the health effects research section of the Air Quality Management Plan for Perth, the relationship between changes in daily air quality and hospitalisation and mortality was investigated, initially using a preliminary time series analysis protocol as recommended by the Air Pollution and Health European Approach (APHEA) and then followed up with a case-crossover study design and analysis. The case-crossover study design is an alternative to the traditional time series analysis and considers the effects of transient risk factors such as changes in air pollution concentrations on acute events such as mortality and hospitalisation.

The mortality investigation considered daily death rates between 1992 and 1997. Counts of daily deaths were obtained from the Registrar General of WA from January 1992 to December 1997. Data were collected for all causes of death, excluding accidental deaths, and categorised as deaths from respiratory, cardiovascular and other causes for two age groups (all ages and ages 65 and over).

The hospitalisation investigation considered daily hospitalisation rates between 1992 and 1997. Hospitalisation records were collected for respiratory disease, chronic obstructive pulmonary disease (COPD), asthma, and cardiovascular disease (CVD). Data were also collected for gastrointestinal disease as a control disease. Daily air quality data for ozone, nitrogen dioxide, carbon monoxide, and

particulate matter measured as visibility (using a light scattering factor – Bsp) were obtained from air monitoring stations which were operational for the study period and encompassed most of the criteria pollutants under consideration. PM2.5 data were obtained by modelling air quality data for the study period.

The data were analysed using case-crossover conditional logistic regression which involves comparing all the varying exposure matrices for the day in question with daily exposures one week before and one week after.

The case-crossover analysis found associations between changes in daily ozone concentration and cardiovascular mortality. An association between daily carbon monoxide concentrations and 'other' mortality was also observed. The results of the preliminary time series regression analysis showed associations between nitrogen dioxide and all-cause mortality, respiratory mortality and 'other ' (non-specified disease group) mortality. No other significant associations were observed.

Significant associations were observed for daily changes in particle and ozone concentrations and hospitalisations for asthma, chronic obstructive pulmonary disease (COPD), pneumonia and respiratory disease for all ages using the case-crossover analysis. Specific age groups were investigated and significant results were observed for people aged over 65 years of age for CVD, COPD, pneumonia and respiratory diseases. Significant associations were also observed for changes in daily particle and ozone concentrations and asthma hospitalisations for children aged under 15 years, with an estimated 0.3% increase in hospitalisations with every 10 $\mu g/m^3$ of modelled PM2.5 concentrations. These results were supported by those obtained by the preliminary time series analysis, appended to this report.

These results support those reported in Sydney, Melbourne and Brisbane as well as the numerous international studies. The results for Perth indicate that air quality is impacting on the health of Western Australians and provide a useful basis for setting priorities for implementing the Perth Air Quality Management Plan and evaluating the success of the associated implementation strategies.

Contents

1. Introduction

A large number of studies that have investigated the relationship between air quality and health effects. Air pollution is cited as the major environmental issue of concern to the community (Australian Bureau of Statistics 1998).

In the 1950s large increases in illness and death were associated with air pollution, most notably in London (e.g. Mazumdar *et al*. 1982; Schwartz and Marcus 1990; Dockery and Pope 1994). By the 1970s, the relationship between respiratory disease and air pollution (particles and SO_2) was established but there was considerable debate about the concentrations at which impacts might occur (Dockery and Pope 1994). Since that time many studies in Europe and the USA have been conducted which have further established the relationship between air pollution and health effects. Increases in hospitalisation, increases in emergency room attendance and decreased lung function have been associated with a range of common air pollutants, the so-called criteria pollutants: carbon monoxide, nitrogen dioxide, inhalable particles (measured as PM10), photochemical oxidants (measured as ozone) and sulfur dioxide.

In 1998, the Parliamentary Select Committee on Perth's Air Quality outlined its concern about the potential health impacts of air quality in Perth, in particular photochemical smog and particulate haze. Both ozone and particulate concentrations exceed national and international standards on occasions throughout the year. Concentrations of SO_2, NO_2 and CO are all below national and international standards in Perth with elevated levels being observed in specific areas impacted by point source emissions (Department of Environmental Protection 2000).

Given the number of exceedences of ozone and particulate ambient air quality standards, the Select Committee recommended that the Department of Environmental Protection (DEP) and Department of Health of Western Australia facilitate research into air quality and health effects. The Government response to the Select Committee report supported this view and recommended an evaluation of the research requirements into health effects of air pollution and provision of advice on attainment of funding for health research activities.

As part of the development of the Air Quality Management Plan (AQMP) for Perth, an Air Quality Co-ordinating Committee (AQCC) was established with eight working groups, one of which was the Health Research Working Group. The principal purpose of this group was to obtain expert opinion and advice on health related research needs and priorities for Western Australia in the specific area of air quality and to develop a preliminary list of research needs and priorities for further evaluation.

Only a few Australian studies have been conducted which have investigated the relationship between

daily ambient air quality and health indicators. These studies have used one of the most common types of study in air pollution research, the time series study, to investigate the health effects of air pollution. Studies conducted in Sydney and Melbourne have shown that increases in air pollution levels are associated with increased risks for daily mortality (Morgan *et al*. 1998; EPA Victoria 2000). The Melbourne mortality study showed significant associations between ozone and nitrogen dioxide for all-cause mortality and respiratory deaths (EPA Victoria 2000). Studies in Sydney, Melbourne and Brisbane all reported increases in hospitalisations associated with air pollutants (Simpson *et al.* 1997; Petroechevsky *et al.* 2000; Morgan *et al.* 1998; EPA Victoria 2001). Such studies provide some support for the findings reported in the international literature (Dockery and Pope 1994; Schwartz 1994a, 1994b; Department of Health (UK) 1998).

Time series studies involve the collection and analysis of air pollution and health effects data (such as mortality data) over a specified time period. Statistical analysis determines whether there is a relationship between changes in air pollution levels and changes in mortality for specific diseases.

The numerous international time series studies have found significant associations between the criteria pollutants and mortality. Many have shown a consistency across studies and the range of meteorology, topographies and lifestyles of the different study centres (Pope *et al*. 1995; Katsouyanni *et al.* 1995, 1996; Health Effects Institute 2000a, 2000b).

In the past few years, many researchers have been utilising the case-crossover design to investigate the health effects of air pollution. The case-crossover design measures the effects of transient risk factors for acute disease events. Each case is its own matched control and compared at different exposure periods. The study design controls for all measured and unmeasured variables which do not vary with time (Bateson and Schwartz 2001).

No comprehensive outdoor air pollution studies have been undertaken in Perth and it is possible that factors such as lifestyle, meteorology and topography may influence exposure to pollutants even though similar levels of pollutants are observed in Perth compared with other major Australian cities. The Health Research Working Group considered that this may make it difficult to apply the results from overseas studies to air pollution data available for Perth.

One of the highest priorities identified by the Health Research Working Group, therefore, was the need to investigate the relationship between air quality and daily mortality and hospitalisations. Perth provides an excellent opportunity to investigate such associations because it has a well established air monitoring network and an accessible mortality and hospitalisation database. It is comparatively isolated and has a population of about 1.25 million. There are several pockets of high industrial activity and because the population is spread out, there is a high dependency on private motor transport compared with public transport use. The city is generally free from trans-boundary pollution

events, being generally subject to consistent wind from the Indian Ocean where the nearest land is thousands of kilometres away and to offshore winds from regions of very low population density. On certain occasions however in the colder months, temperature inversions occur when levels of some pollutants become quite high, sometimes on several consecutive days. In addition the potential health effects of ozone and particulates can be explored in the absence of other confounding chemicals such as SO_2.

This report outlines the study conducted and the findings for available air quality data and daily mortality and daily hospitalisations.

1.1 Background on Common Air Pollutants in Perth

The following section introduces the sources, health and environmental effects, and ambient air quality standards or guidelines of the main air pollutants monitored in the Perth metropolitan region.

The sources of air pollution are diverse and may be the result of direct emissions to the atmosphere (i.e. primary pollutants) or undergo chemical changes in the air (i.e. secondary pollutants). The key pollutants of interest are considered to be carbon monoxide (CO), nitrogen dioxide (NO_2), particles (measured as PM10 and Bsp) and photochemical smog (measured as ozone). SO_2 was not included in the analysis due to the negligible SO_2 concentrations in the Perth metropolitan area, most being below the limit of detection.

Monitoring in Perth and subsequent trend analysis shows that Perth has levels of air pollution which may present health impacts. The levels of photochemical smog (ozone) in Perth during summer regularly exceed national standards. During the winter months, a smoke haze frequently hangs over Perth. In most cases these episodes of unacceptable air quality are influenced by weather conditions that prevent air pollutants from dispersing rapidly. Concentrations of these pollutants are monitored via a network of 13 ambient air quality stations which monitor a range of air pollutants. Different parameters are monitored at the respective stations.

A detailed analysis of the air quality in the Perth metropolitan region is contained in the DEP's *Air Quality in Perth 1992-1999* report (DEP 2001). In summary the report shows that:

- Ozone levels in summer are tending to remain high, approaching or exceeding the acceptable standard;
- Background levels of ozone are increasing;
- Airborne particle (haze) levels in winter are relatively high, and during spring and autumn are relatively low.

The effects of air pollutants on human health range from a mild irritation of the airway to damage of major organs such as the heart or lungs. Some air pollutants (e.g. ozone) may also exacerbate existing conditions such as asthma or bronchitis. The level and duration of human exposure to a particular air pollutant are linked to the health effects observed. For example, nitrogen dioxide may cause serious health effects after several hours or days of exposure to high ambient concentrations whereas other effects may be induced at higher concentrations over a much shorter time frame.

Ambient air quality standards, such as the National Environment Protection Measure for Ambient Air Quality (Air NEPM), have primarily been based on human health data and existing Australian and international standards (National Environment Protection Council, 1998). Established in June 1998, the Air NEPM specifies air quality standards for the six most common urban air pollutants, the 'criteria pollutants': carbon monoxide (CO), nitrogen dioxide (NO_2), photochemical smog (ozone), sulfur dioxide (SO_2), particles (PM10) and lead.

These air pollutants have impacts on regional and local air quality, but their significance lies in their ubiquity and their effects on human health and the environment.

The goal of the Air NEPM is to achieve compliance by June 2008 with specified standards set down in it. All governments are required to report annually on progress towards meeting the Air NEPM goal. Programs to improve or maintain air quality, such as those developed through policies or management plans, are the responsibility of each State or Territory government. It is proposed to implement the Air NEPM in WA via a Statewide Environmental Protection Policy (EPP), which is currently being developed. Achievement of compliance in Perth will be through Perth's Air Quality Management Plan.

Different averaging periods have been set for pollutants based on health, economic, social, technological and environmental data. For example, there are two Air NEPM standards for NO_2: a 1-hour average and a 1-year average. The health effects of lead are related to long-term exposure, so the averaging periods set in goals or standards tend to be for periods in the order of a few months or one year as in the Air NEPM. Further details can be found in Appendix 1.

1.2 Health Effects of Air Pollutants

The health effects of air pollution, chiefly from industrial and transport emissions, have been studied worldwide for many years (Comstock 1998).

A number of reviews have outlined the associations between air quality parameters and morbidity which have shown consistency across studies and the range of weather patterns, topographies and lifestyles of the different study centres (Pope *et al.* 1995; Thurston *et al.* 1992).

Over the past 30 years, numerous studies from a variety of different locations have reported associations between increases in air pollution and increases in hospital admissions (morbidity) (Dockery *et al*. 1993; Schwartz 1994a, 1994b, 1994c; HEI 2000a; Samet *et al.* 2000). In Europe the collaborative effort of researchers in 15 cities has also found such associations (Katsouyanni *et al.* 1995, 1996). A meta-analysis of 109 time series studies from 94 geographic areas in North America, South America, Mexico, Europe, Asia, Australia and New Zealand found an increased risk of respiratory mortality for all pollutants other than ozone, but not for circulatory mortality (Stieb *et al*. 2002). The research has demonstrated clear associations between mortality from many different causes and levels of air pollution, and also rates of hospital admissions and pollution levels for many different diseases. The precise components of the pollution that have the greater impact on health are still issues of debate.

More recently, long-term effects have been investigated and increased risks of lung cancer and cardiopulmonary mortality observed with increases in fine particulate concentrations (Pope *et al.* 2002).

Particulate and ozone concentrations have been associated with daily mortality and hospital admissions in Australia (Morgan *et al.* 1998; Simpson *et al.* 1997, 2000; EPA Victoria 2000, 2001).

Several other studies have examined the effects of ambient air quality on asthma and other respiratory disorders in Australia (Rennick and Jarman 1992; Christie *et al.* 1992; Abramson and Voigt 1991; Robertson *et al.* 1992). One study of daily hospital admissions in the La Trobe Valley, Victoria, found significant relationships between particulates (visibility) and nitrogen dioxide and hospital admissions for chronic obstructive airways disease (Voigt *et al.* 1998). Photochemical smog has also been associated with increased hospital admissions for asthma and COPD (Abramson *et al.* 1994).

A summary of some of the outcomes of some studies conducted to-date, relating to specific pollutants, is outlined in Appendix 1. The information on the principal sources and health effects of the common air pollutants presented in this section is taken from the *Final Impact Statement for the Ambient Air Quality National Environment Protection Measure* (NEPC, 1998) and selected references.

1.3 The Case-Crossover Study Design

The case-crossover design is used to study transient effects on the risk of acute-onset diseases. This design samples cases, comparing each subject's exposure to disease events to the subject's own exposure before the occurrence of the disease. Exposures for each subject during a designated period before the disease event are compared with the distribution of exposure from a separate time period.

The case-crossover conditional logistic regression analysis (Bateson and Schwartz 1999) is more robust and less prone to bias and confounding than the more standard time series regression analysis. The case-crossover design does not require modelling of all variables. All season and trend effects are removed. Because the event is the unit of analysis, in contrast with the day as in time series, factors such as age and sex can also be estimated much more easily.

Another advantage of the case-crossover design is its ability to control potential confounders that do not vary with time, since each individual (or population) forms its own stratum in which those factors are identical (Bateson and Schwartz 1999).

An important step in the design of a case-crossover study is the choice of time interval between the day of death or hospitalisation and at the control interval. The symmetric bi-directional design, with two control points equally spaced either side of the hazard period or event is regarded as close to optimal (Bateson and Schwartz 1999, 2001).

2. Objectives

The objectives of this study were to investigate the relationship between daily ambient air quality and daily hospitalisation and daily mortality in Perth.

3. Methods

3.1 Study Design, Area and Population

A case-crossover design was used in this study to investigate the relationship between daily ambient air quality and daily hospitalisation and daily mortality in the Perth metropolitan area between 1 January 1992 and 31 December 1997. A time series regression analysis as per the APHEA (Air Pollution and Health European Approach) protocol was originally performed, however was limited in the extent of modelling undertaken and was superseded by the case-crossover study design. The method and results of the time series study are presented in Appendix 2.

The study area encompassed metropolitan Perth (the Australian Bureau of Statistics (ABS) Statistical Division) which is the area bounded by Rockingham in the south, Quinns Rocks in the north and Rolling Green in the east (Figure 1). The area contains 13 monitoring stations also shown on Figure 1. The 1996 ABS census estimate for this study area was 1,244,320 persons.

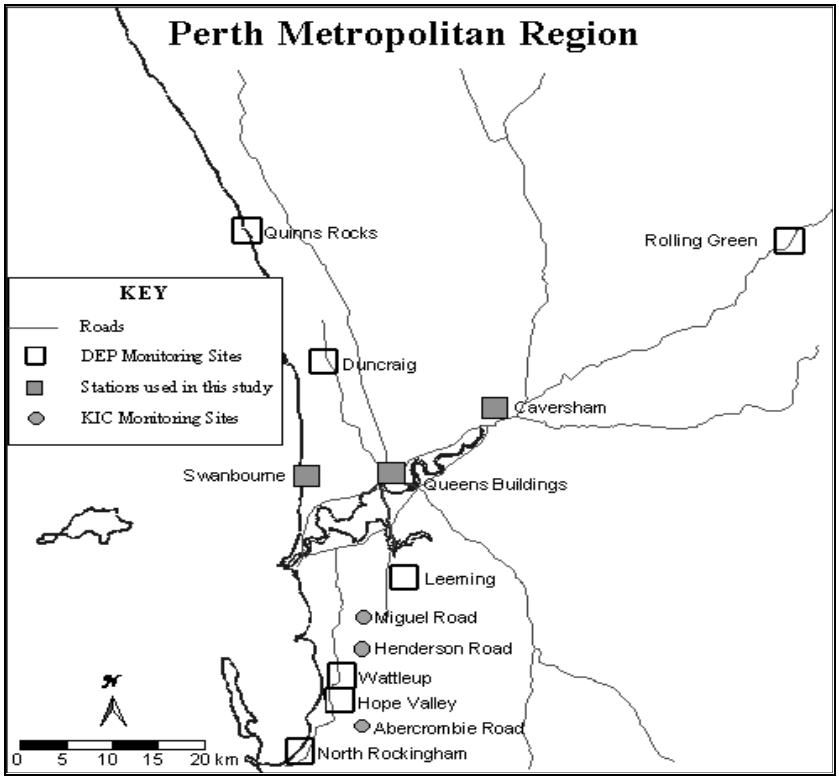


Figure 1 - Map of air quality monitoring sites in Perth as at 1/1/2000. (KIC, Kwinana Industries Council: DEP Department of Environmental Protection)

3.2 Data Collection

3.2.1 Mortality Data

Mortality data were made available by the Registrar General of Western Australia. Information available included underlying cause of death coded to The International Classification of Diseases version 9 (ICD9) by the Australian Bureau of Statistics, date of birth, sex, postcode of residence and date of death. There are approximately 12,000 deaths recorded annually in Perth.

Cause of death codes are made available on an annual basis by date of registration and so deaths were taken from the 1/1/1992 to 30/12/1997. Many deaths are registered in the month following the actual date of death and ICD codes for 1998 deaths were not available. Therefore, deaths registered in 1998

were also checked for the date of death in case any 1997 deaths had been missed.

Four groups of causes of death were analysed separately: all causes excluding accidents, poisoning and violence (ICD9 codes 001 to 799), cardiovascular (ICD9 codes 390 to 459), respiratory (ICD9 codes 460 to 529), and the first group excluding cardiovascular and respiratory causes for two age groups (all ages and age 65 and over only).

3.2.2 Hospital Morbidity Data

Hospital morbidity data for the same period for all hospitals in the Perth metropolitan area were selected. Only non-booked admissions were selected for respiratory, cardiovascular and gastrointestinal admissions (ICD9-CM codes 390.00 to 579.99), noting that a record is completed following discharge, not admission. Diagnoses were based on primary discharge obtained from the patients' charts. Other co-morbidity conditions could not be assessed. The hospitalisation data provides an indication of acute episodes of illness and does not account for a history of health, which may be important in predisposing an individual to an acute episode arising from an external insult such as air pollution. There are approximately 500,000 hospital admissions each year for Western Australia.

Eight groups of diagnoses were analysed separately: all respiratory (all ages and 65 and over only), asthma (all ages and under 15 only), all cardiovascular, chronic obstructive pulmonary disease (COPD: ICD codes 490.00 to 496.99 excluding asthma), pneumonia/influenza (ICD codes 480.00 to 489.99), and all gastrointestinal diseases (ICD codes 520.00 to 579.99) as a 'control' group of admissions, for which there was no reason for anticipating any effects of air pollution.

All transfers from other hospitals were subsequently excluded. Residents living outside the metropolitan area were excluded from the analysis as were death or hospitalisation from industry (including exposure and injury).

3.2.3 Air Quality Data

Daily ozone, PM10, NO_2, CO and Bsp have been measured since 1990. PM2.5 has been monitored since 1994 using a tapered element oscillating microbalance (TEOM). PM10 was collected every 6 days by high volume sampler (HiVol). The number and location of stations monitoring specific pollutants has varied depending on specific projects. Appendix 3 shows the monitoring data available in the Perth metropolitan area for this study.

The most complete data set with the highest number of days with co-located instruments was from the Caversham, Swanbourne and Queens Buildings sites and these were used to generate the daily air

quality data for the pollutants investigated with the exception of PM2.5 which is discussed below. PM10 was not included in this study due to the sampling frequency. It should be noted that ozone is not monitored at the Queens Buildings site and CO is not monitored at the Swanbourne site. While Queens Buildings is located in the centre of Perth City, the concentrations of ozone and particulates are not necessarily elevated at this site compared with other suburban sites. SO_2 was not included in the analysis due to the negligible SO_2 concentrations in the Perth metropolitan area, most being below the limit of detection.

Two approaches to aggregating air quality data were used to provide a daily exposure estimate for each parameter. Data were averaged for the combined metropolitan sites and the three sites used to generate daily data for this study. The averages for each pollutant and each method of generating a daily estimate were comparable with no statistically significant differences, indicating that the use of the three sites was acceptable as a surrogate for data across the entire network. The use of the three locations was in accordance with the APHEA protocol that attempts to ensure the use of data from co-located monitors.

The averaging times selected were based on both international and Australian standards (Table 3.1). The averaging periods listed in Table 3.1 were used for each pollutant in subsequent analyses.

Table 3.1 - Averaging periods and units of measure for each pollutant

Pollutant	Averaging Time	Unit of Measure
Ozone	1h, 4h, 8h	ppb
Nitrogen Dioxide	1h, 24h	ppb
CO	8h	ppm
PM2.5	24h*	$\mu g/m^3$
Bsp	1h, 24h	$2.1 \times 10^{-4}\ m^{-1}$

* Modelled estimates

Daily temperature, wind speed and direction, dew point temperature and relative humidity were obtained by averaging the data from the monitoring stations. These data were compared with the Bureau of Meteorology averages to validate the use of data collected from individual fixed air monitoring stations.

3.2.4 Particulate (PM2.5) Dispersion Modelling

Measurements made at a mix of locations in the Perth region have shown that in winter when high particulate concentrations are most common, there is a strong relationship between the density of population about each site and the particulate concentrations measured. Measurement of particles and numerical modelling studies have confirmed that daily averages vary considerably across the region, being largest near centres of highest population density.

Due to the limited number of particulate monitoring sites and the large variations in population density and subsequent particle concentrations, no single site value, nor a simple average over all sites, was considered likely to provide an accurate estimate of the average exposure of the city's population.

Had the trend of population between the monitoring sites been uniform, a simple interpolation, generating a map of average concentrations, would have formed a useful basis for exposure assessment. However, given the noted variations of population density, this approach was unlikely to provide reasonable estimates.

While a totally accurate model was not available, modelling studies had shown that it was relatively easy to simulate the overall daily average concentration pattern. Although there remained significant errors (probably related to the limited knowledge of meteorological conditions and of the actual particulate emissions pattern), it was considered that the modelled concentration distribution, scaled to match available measurements, would form a better basis for exposure assessment.

The modelling work was conducted using a Guassian plume dispersion model, with the particulate emission rate for each location presumed proportional to population density. Each day of a winter season was modelled separately, giving for each a grid map of estimated daily average concentrations. From this map, modelled concentrations for each particulate measurement site were determined.

For a totally accurate model, these concentrations would have matched measurements, giving a ratio of one at each site. In practice, the ratios differed from one, and varied across the modelled region. However, it was possible to interpolate this set of ratios, to develop a grid map of correction factors for the region.

The best estimate for each model grid point was then taken as the product of the modelled concentration and the interpolated ratio of measured to modelled concentration. These estimates matched all measurements, and also varied between measurement sites in a manner consistent with model calculations.

To further enhance the accuracy of dosage estimates, the relationship between particulate concentrations and optical backscatter coefficient (B_{sp}) was studied. For the winter haze events, it was found that there was a linear relationship, which for B_{sp} over 2 was:

$$[PM2.5] = 17.86\ B_{sp} + 3.69\ \mu g/m^3$$

With this relationship, it was possible to use B_{sp} measurements to enhance the data set used in interpolations.

Appendix 4 shows the interpolated and modelled PM2.5 concentration contours for Perth.

3.3 Statistical Analysis

This study used symmetric bi-directional design with a referent spacing interval of seven days and conditional logistic regression to produce odds ratios. This short interval adjusts for weekly seasonality automatically. This choice was considered to reduce bias (Bateson and Schwartz 2001).

Individual admissions were considered as cases and matched to exposure levels 7 days prior to and post the actual day of admission or mortality, providing two control admissions. Comparisons between cases and controls were then made using conditional logistic regression (Bateson and Schwartz 1999). Before examining the effect of pollutants, all daily weather variables (temperature and humidity) were examined. Variables indicating public holidays and day of the week were included. However, only the most significant effects were included when pollutants were added to the model. In every case, this was with the lag 1 values. Each pollutant was entered into its own predictive model for different lag periods. For each particular pollutant, the residuals from the adjusted model for each separate outcome were regressed on each combination of each type of lag (0,1,2,3,0-1, 0-2, 0-3) days and each averaging period (1h, 4h, 8h, 24h).

Each category of hospitalisation and mortality data was analysed in relation to NO_2, O_3, particulate (PM2.5 modelled and Bsp) and CO concentrations. Gastrointestinal hospitalisations were used as a comparison group. Additional analyses were performed for hospitalisations for those aged over 65 years and for asthma hospitalisations for those aged under 15 years.

Days for which no measures of a particular pollutant had been made were ignored in the analysis involving the specific pollutant. When days of missing data occur randomly—as was the case in this data set—this method is not likely to introduce selection bias.

To explore the potential interaction of season and exposure, a binary season indicator was included and a likelihood ratio test for its interaction with exposure was undertaken.

In the context of case-crossover study, the odds ratios produced by the analysis are equivalent to relative risks.

4. Results

4.1 Mortality

Between 1992 and 1996 the average number of daily deaths was 26.8 with an average of between 2.1 and 2.4 deaths attributable to respiratory disease. By comparison 11.6 deaths were attributed to cardiovascular disease. Most of these deaths occur in the age group over 65 years (Appendix 5).

Daily pollutant concentrations and meteorological parameter measures are presented in Appendix 6. The association between air pollutant concentrations, seasonality and meteorological parameters is also shown in Appendix 7. As expected significant correlations between ozone and particulates and temperature as well as CO concentrations and NO_2 and particulates were found.

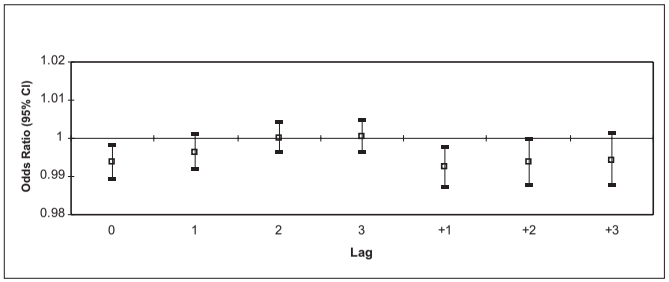
The odds ratio and 95% confidence intervals were plotted for each pollutant and disease category for the case-crossover analysis. These are shown in Figures 2-10.

The results of the case-crossover analysis showed no significant associations between changes in daily pollutant concentrations and respiratory mortality (Appendix 8.2). Significant odds ratios were observed for changes in 4-hr and 8-hr ozone concentrations and daily cardiovascular disease (CVD) mortality with an estimated 0.2% to 0.45% increase per ppb of ozone (Figures 6 and 7). Elevated odds ratios were also observed for the 1-hr ozone concentration and CVD mortality, however the estimates did not reach statistical significance.

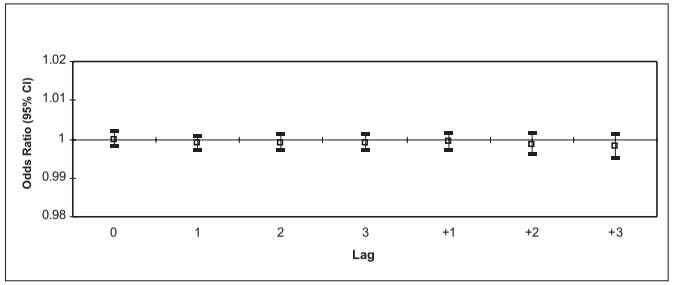
A significant association was also observed for the relationship between changes in the 8-hour carbon monoxide concentrations and 'other' mortality (Appendix 8.3).

No associations were observed for concentrations of particles, CO, O_3 and NO_2 and all mortality.

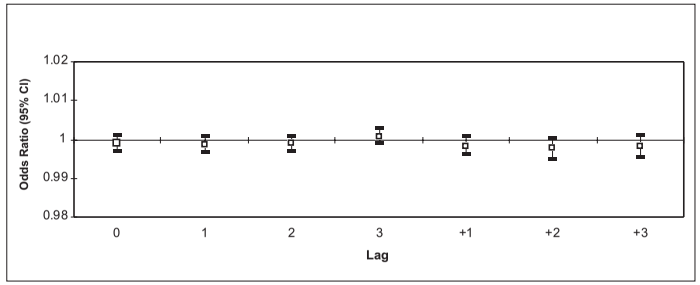
Respiratory Mortality



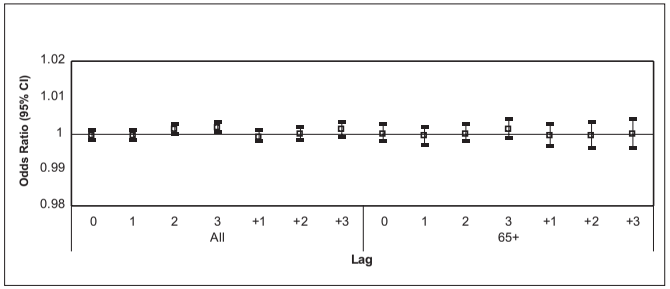
Other Mortality



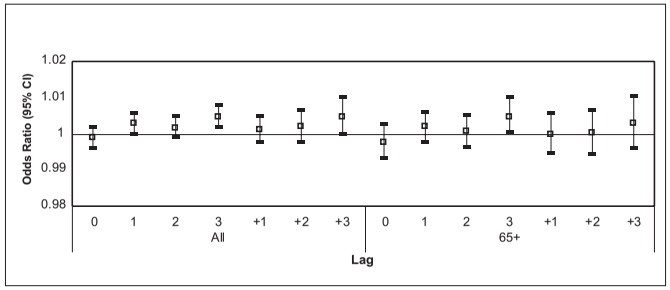
CVD Mortality



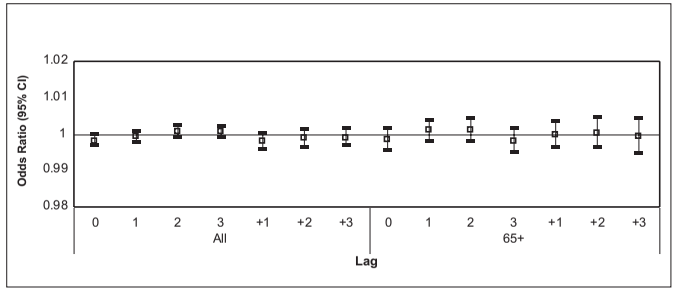
Respiratory Morbidity



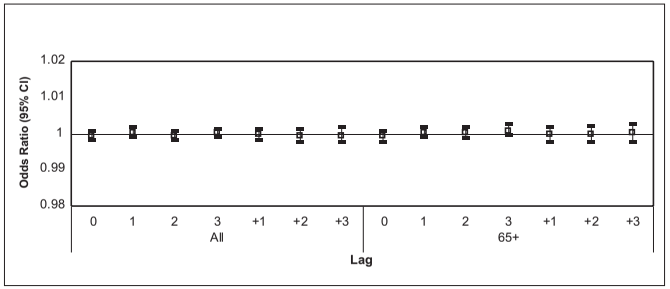
Pneumonia Morbidity



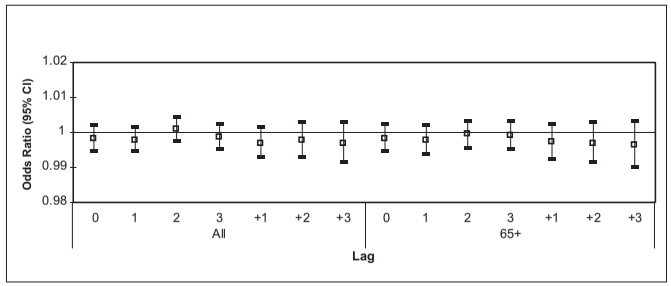
GI Morbidity



CVD Morbidity



COPD Morbidity



Asthma Morbidity

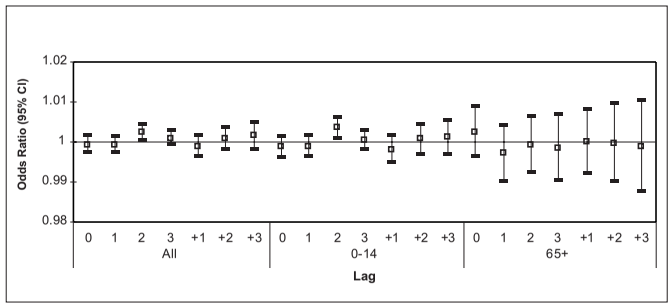


FIGURE 2: Odds Ratio Per Unit Increase in PM2.5 (24-hr) (μg/m^3)

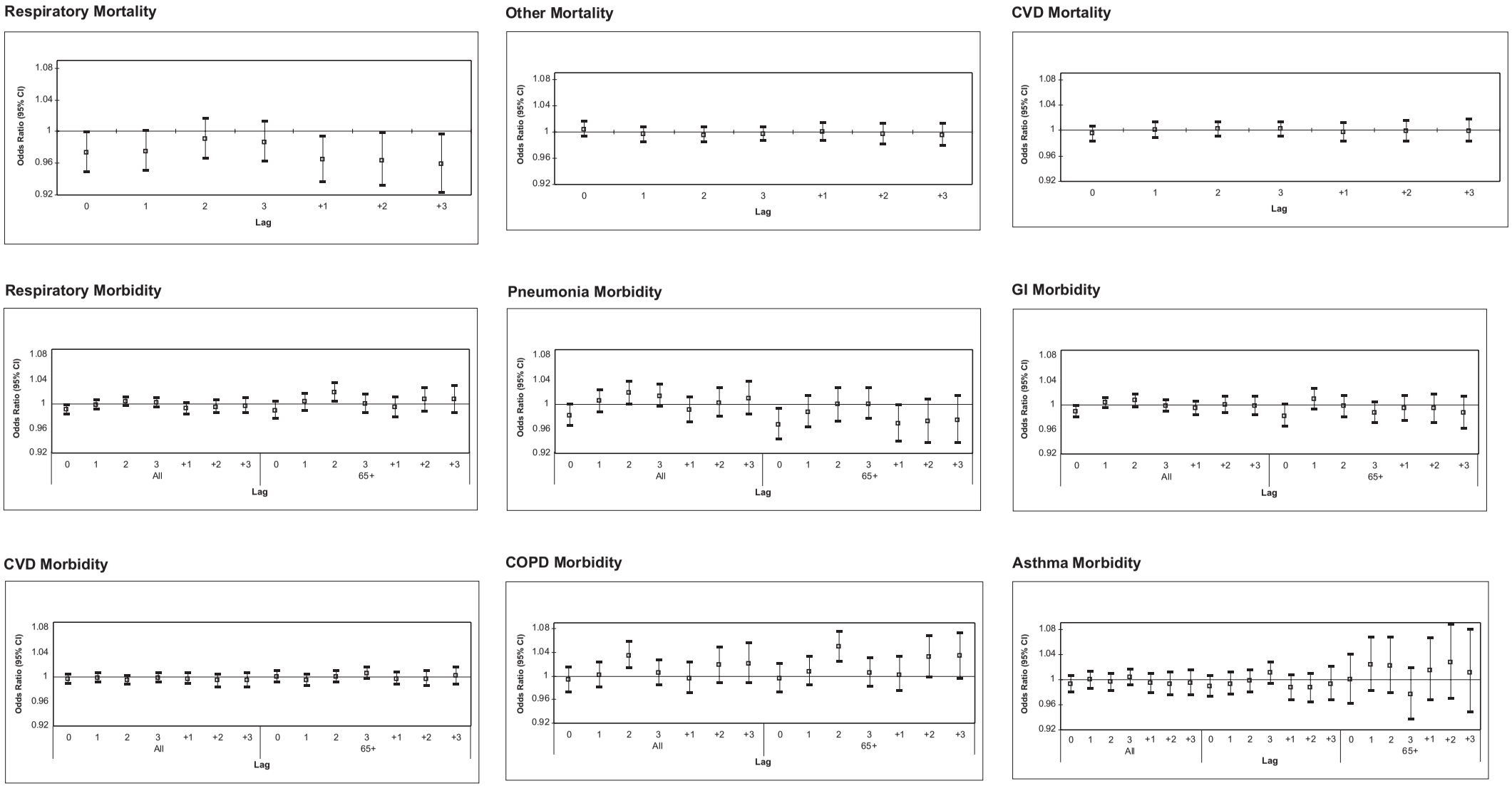


FIGURE 3: Odds Ratio Per Unit Increase in Neph (1-hr)(Bsp)

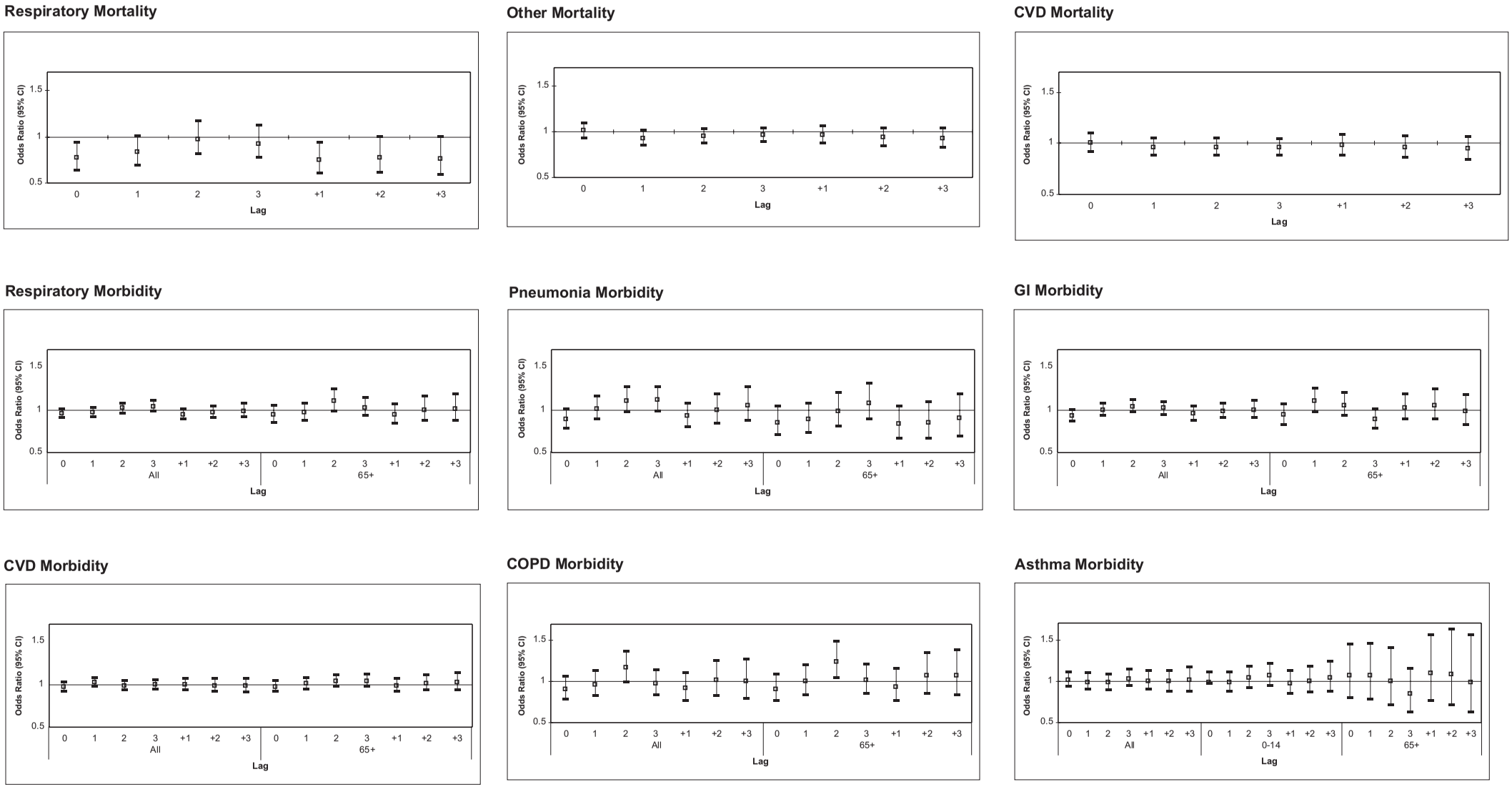


FIGURE 4: Odds Ratio Per Unit Increase in Neph (24-hr)(Bsp)

4.2 Hospitalisations

The number of hospitalisations on an average day for respiratory disease was between 19.3 (summer) and 31.0 (winter) with 6.9 and 10.7 respectively for asthma, with just over half of these being children (Appendix 9).

Daily pollutant concentrations and meteorological parameter measures are presented in Appendix 6. The association between air pollutant concentrations, seasonality and meteorological parameters is also shown in Appendix 7. As expected significant correlations between ozone and particulates and temperature as well as CO concentrations and NO_2 and particulates were found.

The odds ratio and 95% confidence intervals were plotted for each pollutant and disease category. These are shown in Figures 2-10.

4.2.1 Particle Concentrations

Changes in CVD hospitalisations with particle concentrations as derived from modelled PM2.5 and nephelometry (Bsp) were examined (Figures 2-4). There were no significant associations observed, however there were significant interactions with season in the winter direction (Appendices 10.1 and 10.2).

Changes in 24-hour PM2.5 concentrations were significantly associated with respiratory hospitalisation, with an estimated 0.17% increase in respiratory disease hospitalisation for a change in 1 $\mu g/m^3$ (Figure 2; Appendix 10.3). When respiratory hospitalisations for those aged 65 and over were examined, a significant odds ratio was observed for changes in 1-hour maximum particle concentrations as derived from nephelometer measurements (Bsp), with elevated odds ratios observed for the 24-hr Bsp metric (Figures 3 and 4; Appendix 10.4).

The results for asthma hospitalisation showed a significant association with changes in 24-hour PM2.5 concentrations for all age groups and those aged under 15 years (Figure 2; Appendices 10.5 and 10.6). An estimated 0.3% increase in asthma hospitalisation for children (0 to 14 years of age) was observed with changes in 1 $\mu g/m^3$. However, for those aged 65 years and over, no significant associations were found between particles and asthma hospitalisation (Appendix 10.7).

The 1-hour maximum and 24-hr average particle concentrations derived from nephelometer measurements were associated with a significant odds ratio for COPD hospitalisation for all ages and for those over 65 years of age with estimates of between 4 and 24% per unit increase.(Figures 3 and 4; Appendices 10.8 and 10.9). No other statistically significant results were observed, although elevated odds ratios were evident. No significant seasonal interactions were seen.

Pneumonia hospitalisations for all ages were associated with increases in 24-hour PM2.5 and 1-hour maximum particle concentrations as derived from nephelometer measurements (Bsp) and for those aged 65 years and over. Elevated, but not significant odds ratios were also observed for the association between pneumonia hospitalisations and 24-hr particle measured by nephelometer (Figures 2-4; Appendices 10.10 and 10.11).

There were no associations observed between changes in particle concentrations and gastrointestinal diseases.

4.2.2 Ozone Concentrations

No significant associations were seen between CVD hospitalisations and ozone (Figures 5-7). However, there were significant interactions with season in the winter direction (Appendices 10.1 and 10.2).

Changes in daily ozone concentrations were associated with respiratory morbidity, reaching significance at lag 0 for both the 1-hour and 4-hour maxima concentrations (Figures 5 and 6; Appendix 10.3). There was also a significant seasonal interaction for winter. When hospitalisations for respiratory disease for those aged over 65 were examined, no significant odds ratios were observed, however there was a significant interaction for season in the winter direction (Appendix 10.4).

Elevated odds ratios were observed for the relationship between asthma hospitalisation and daily ozone concentrations for all ages, reaching significance for the 1-hour ozone concentration at lag 0 with an estimated 0.3% increase in asthma hospitalisation for children aged between 0 and 14 years (Figure 5; Appendices 10.5 and 10.6). While elevated odds ratios were seen for those over 65 years, the results did not reach significance (Appendix 10.7). No significant seasonal interactions were seen.

A significant odds ratio was also observed for COPD morbidity in association with 4-hour ozone concentrations with elevated estimates for all other averaging periods (Figure 6; Appendix 10.8). Similar patterns were observed for those aged over 65 years, however the results did not reach significance (Appendix 10.9).

No significant associations were found between ozone concentrations and pneumonia hospitalisation for all ages and for those aged 65 years and over, however many elevated odds ratios were observed with wide confidence intervals (Figures 5-7; Appendices 10.10 and 10.11).

There were no associations between changes in ozone concentrations and hospitalisations for gastrointestinal diseases.

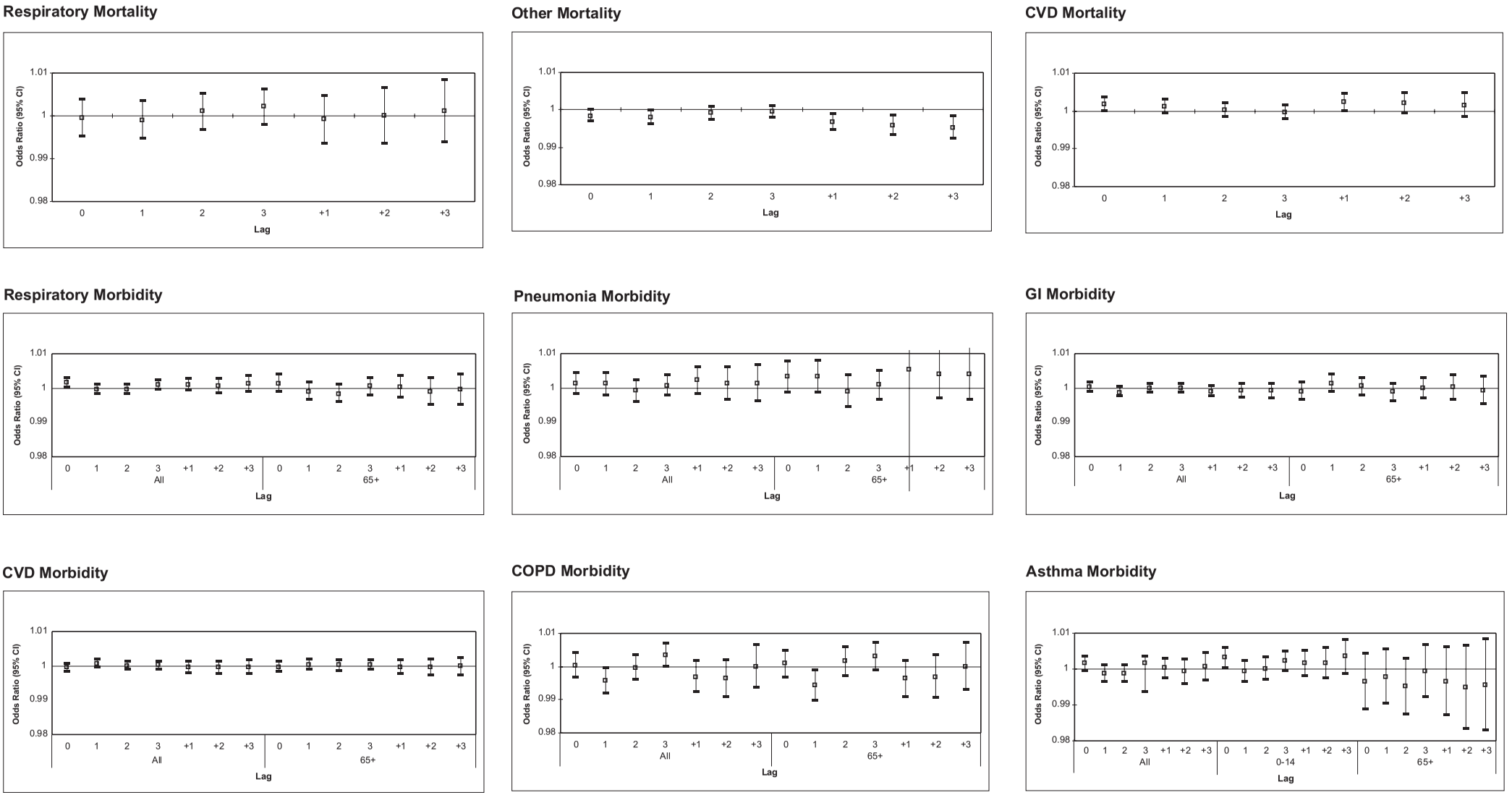
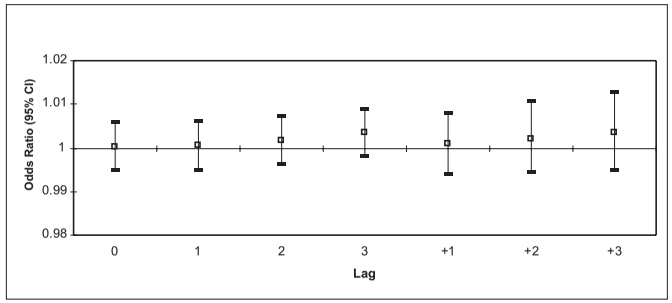
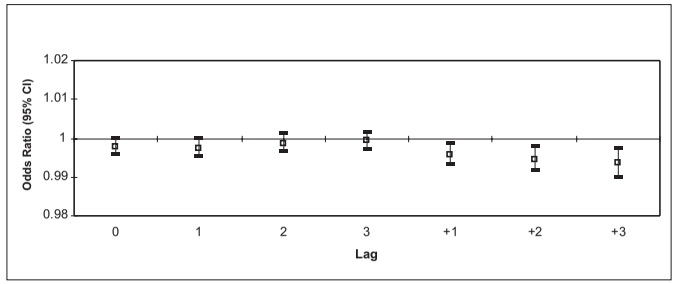


FIGURE 5: Odds Ratio Per Unit Increase in O_3 (1-hr)(ppb)

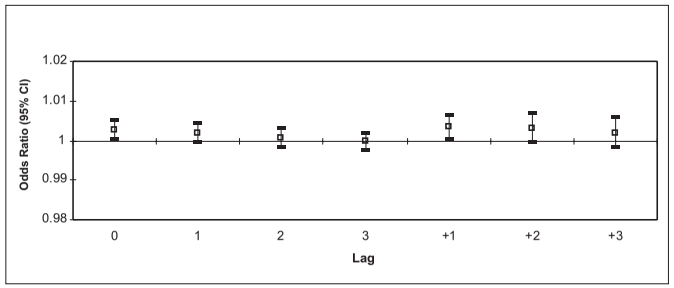
Respiratory Mortality



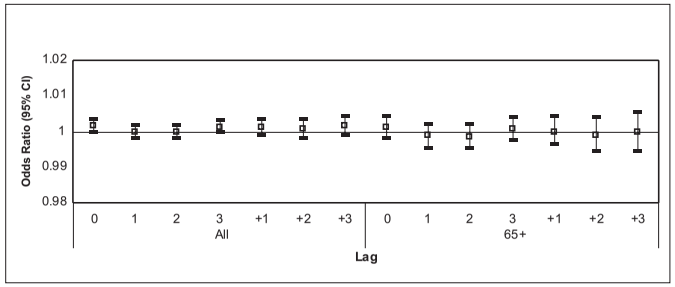
Other Mortality



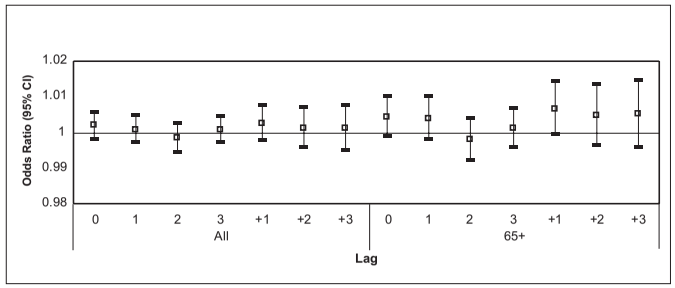
CVD Mortality



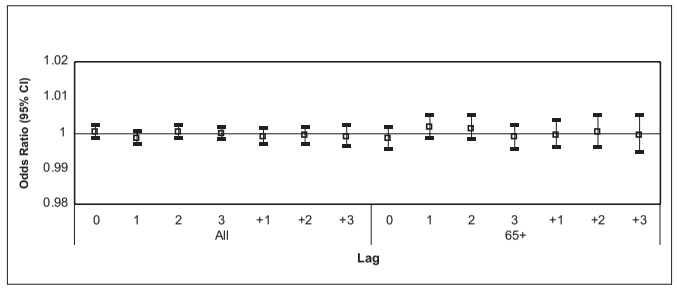
Respiratory Morbidity



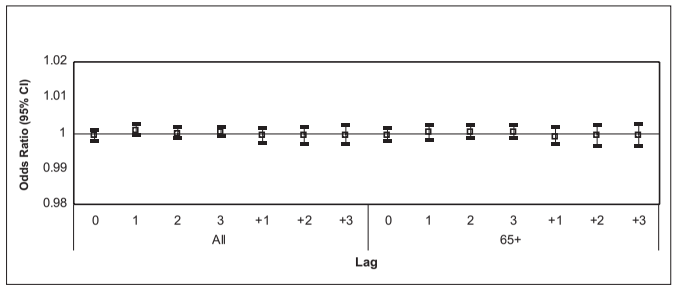
Pneumonia Morbidity



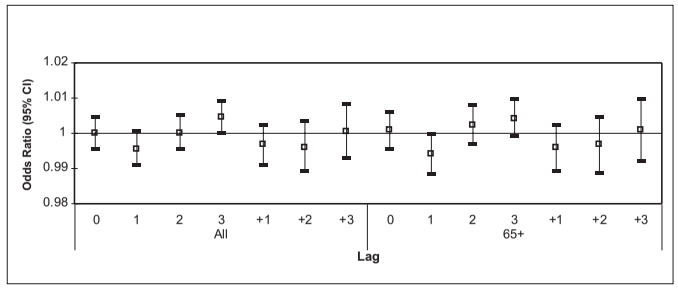
GI Morbidity



CVD Morbidity



COPD Morbidity



Asthma Morbidity

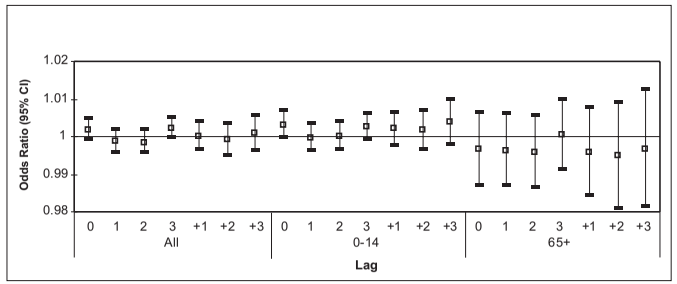


FIGURE 6: Odds Ratio Per Unit Increase in O_3 (4-hr)(ppb)

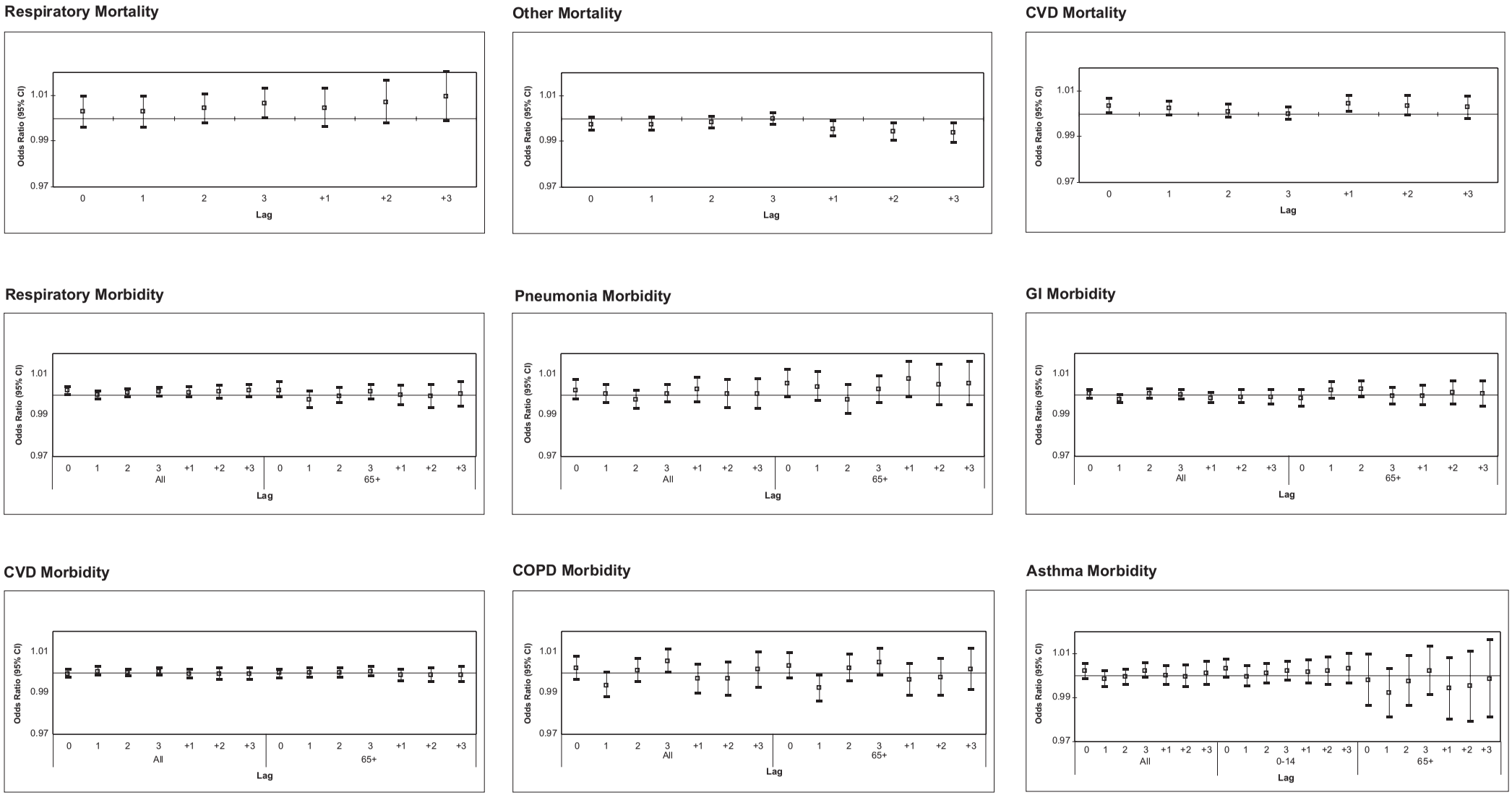


FIGURE 7: Odds Ratio Per Unit Increase in O_3 (8-hr)(ppb)

4.2.3 Nitrogen Dioxide

For the all ages and 65+ years age groups, a significant association was observed between changes in 24-hr NO_2 concentrations and CVD hospitalisations (Figure 9; Appendices 10.1 and 10.2). A significant association between 1-hr NO_2 concentrations and CVD hospitalisations was also observed for those aged 65 years and over (Figure 8).

No associations were observed between changes in daily NO_2 concentrations and asthma hospitalisations (Figures 8 and 9; Appendices 10.3 and 10.5-10.7). A significant association was observed between 24-hr NO_2 concentrations and respiratory disease hospitalisations for the 65+ years age group for lag 1 with elevated odds ratios observed for other lags (Appendix 10.4). This estimate has a wide confidence interval.

No associations were observed between changes in daily NO_2 concentrations and COPD and pneumonia hospitalisations although, as with other disease categories, elevated odds ratios with wide confidence intervals were observed (Figures 8 and 9; Appendices 10.8-10.11).

No associations were seen between nitrogen dioxide concentrations and gastrointestinal diseases.

4.2.4 Carbon Monoxide

No associations were observed between changes in daily CO concentrations and respiratory, asthma, COPD, pneumonia or CVD hospitalisations (Figure 10). Where elevated estimates were observed, these were associated with very wide confidence intervals.

No significant associations were observed between daily CO concentrations and the control disease group of gastrointestinal diseases (Figure 10; Appendices 10.12 and 10.13). Some significant interactions for winter were observed for those aged over 65 years (Appendix 10.13).

5. Discussion

The results of this study showed that changes in air quality in Perth may increase the risks of cardiovascular mortality. The significant finding for ozone concentrations provides estimates of between 2 and 4% increase in mortality with every 10 ppb change in ozone concentration. Elevated odds ratios for cardiovascular mortality in relation to NO_2 concentrations were also observed, however none of the results reached statistical significance. Similar findings have been reported for Brisbane and Sydney as well as for other cities around the world (Appendix 11)(Morgan *et al*. 1998; Simpson *et al.* 1997; Dockery *et al*. 1993).

The significant finding for carbon monoxide and 'other mortality' is difficult to interpret as 'other' mortality consists of many diseases which may not be associated with air pollution and CO concentrations are considered relatively low in Perth. The finding may be a spurious one, particularly in view of the generally low point estimates for the different lags tested.

No other significant relationships between daily mortality and daily changes in pollutant concentrations were detected.

Changes in daily 1-hour and 4-hour ozone concentrations were also associated with increased hospitalisation for respiratory diseases (all ages), asthma (0-14 years of age) and COPD (all ages). Studies in Brisbane and Melbourne have also found similar associations (Appendix 12) (Petroeschevsky *et al.* 2001; EPA Victoria 2001). They were not associated with CVD hospitalisations despite changes in ozone concentration being associated with CVD mortality.

Daily NO_2 concentrations were found to be significantly associated with respiratory disease hospitalisation in the 65 years and above age group, with elevated non-significant estimates also observed. NO_2 concentrations were also associated with significant CVD hospitalisations for the all age category and those aged over 65, supporting the findings of studies in Sydney and Melbourne. This result is surprising given the low, NO_2 levels in Perth, however the result pertains to changes in pollutant levels and not concentration ranges per se. Other Australian studies have shown mixed results for associations between daily nitrogen dioxide concentrations and hospitalisations (Appendix 12). No significant relationships between nitrogen dioxide and respiratory or cardiovascular hospital admissions were found in Brisbane, while strong significant associations between nitrogen dioxide and hospital admissions for asthma in the 0-14 years age group as well as for heart disease have been reported for Sydney (Petroeschevsky *et al.* 2001; Morgan *et al.* 1998).

In this study, no associations were observed between changes in daily CO concentrations and respiratory, asthma, COPD, pneumonia or CVD hospitalisations, unlike other Australian and

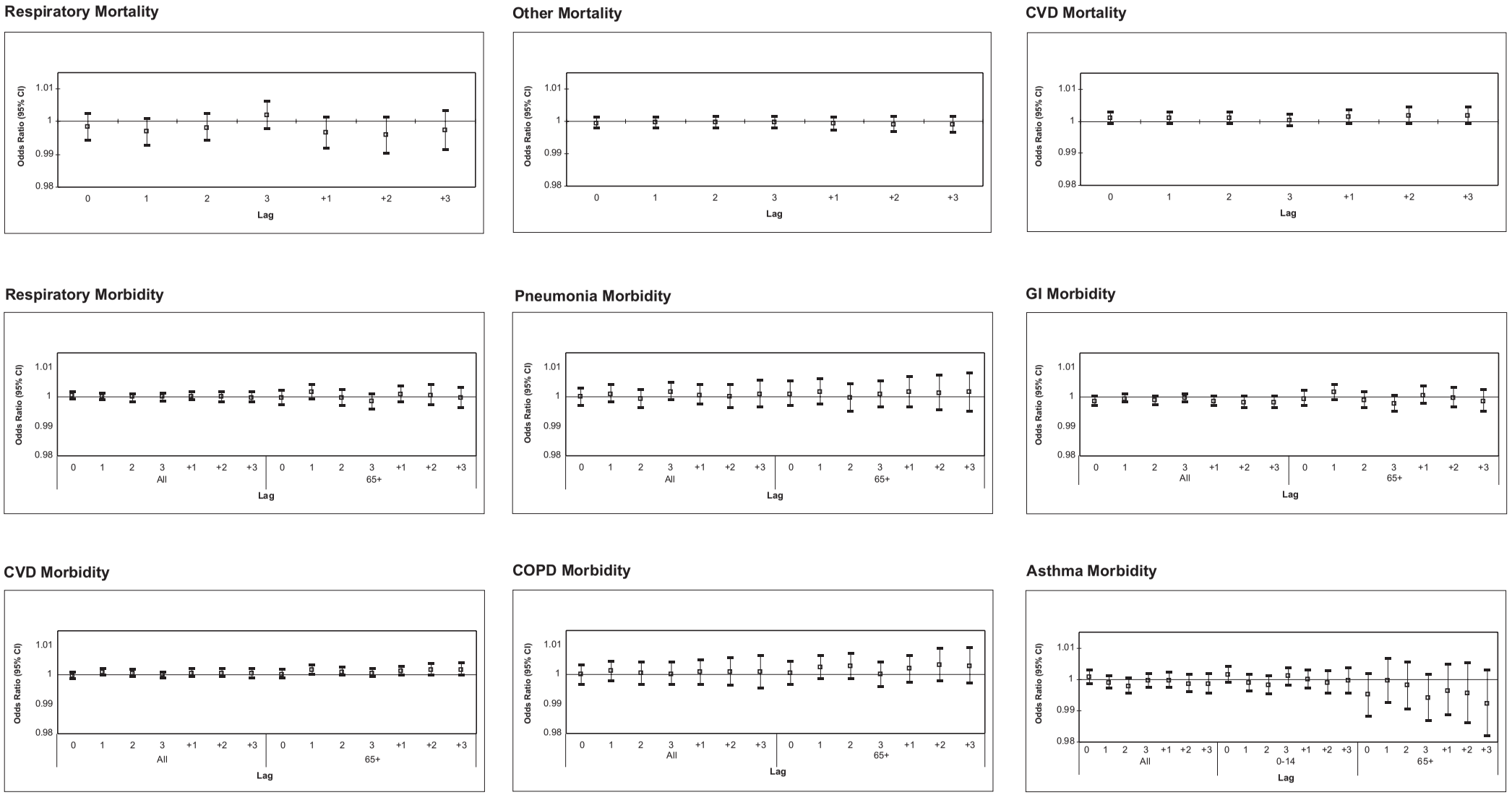


FIGURE 8: Odds Ratio Per Unit Increase in NO_2 (1-hr)(ppb)

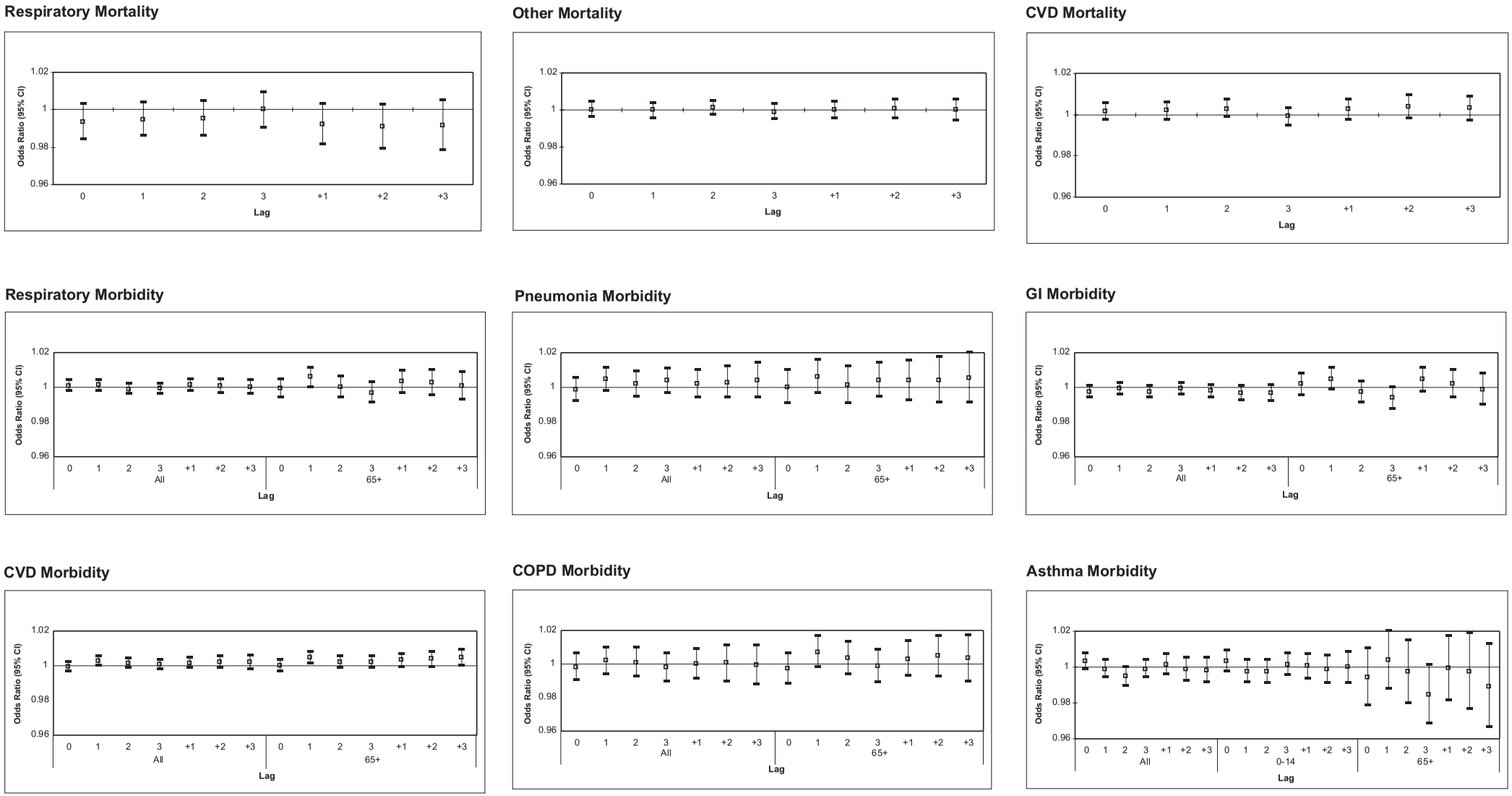
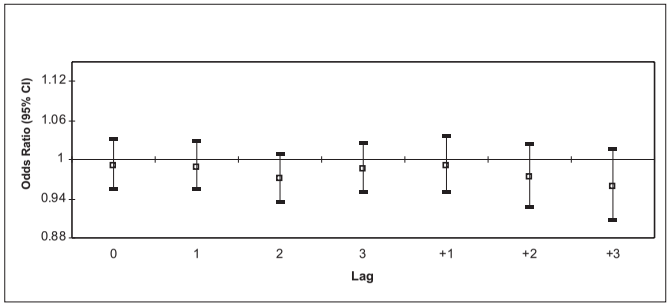
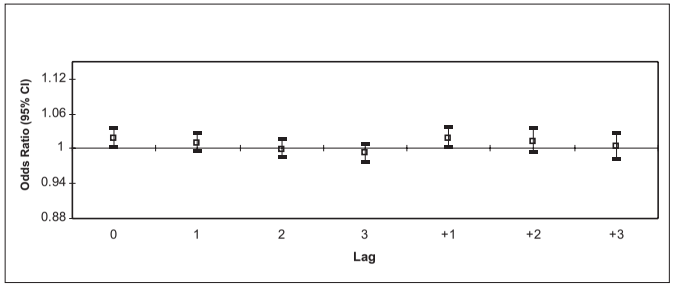


FIGURE 9: Odds Ratio Per Unit Increase in NO_2 (24-hr)(ppb)

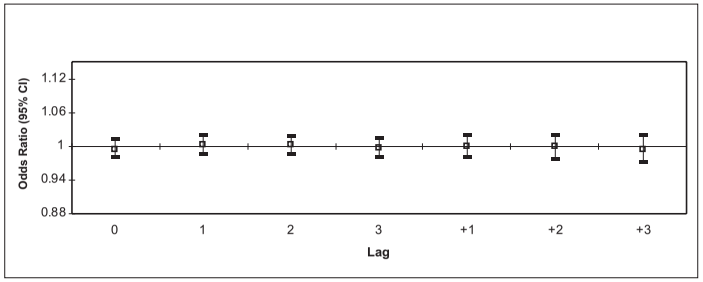
Respiratory Mortality



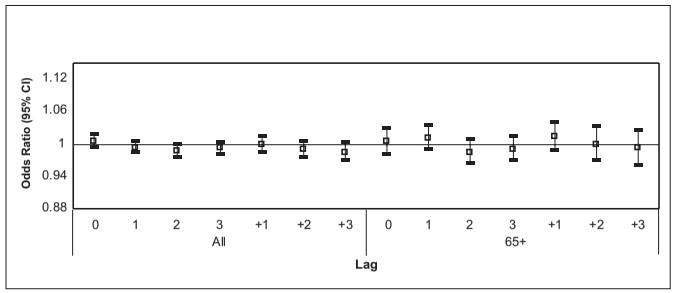
Other Mortality



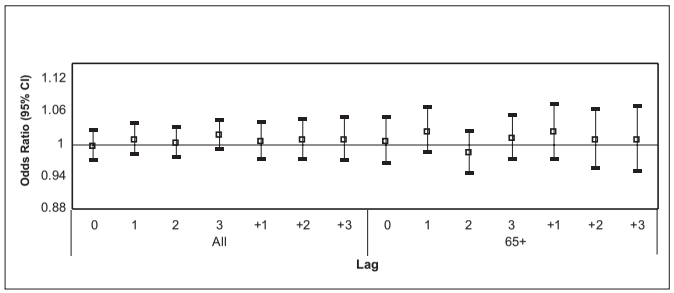
CVD Mortality



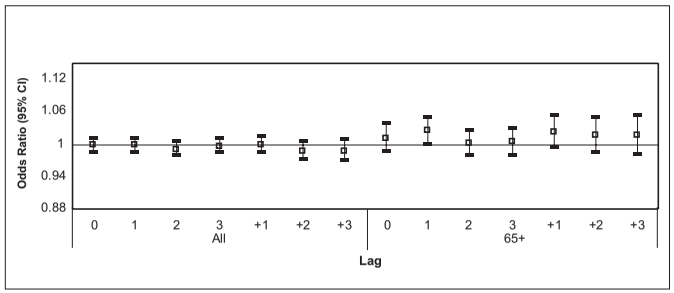
Respiratory Morbidity



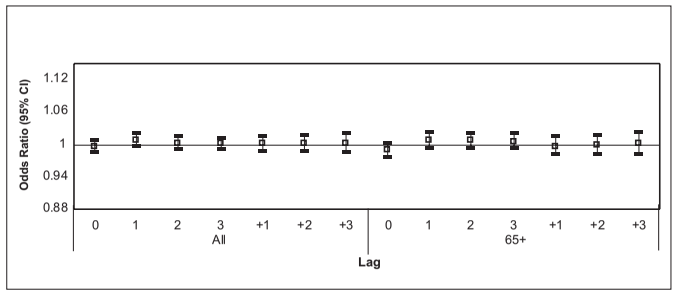
Pneumonia Morbidity



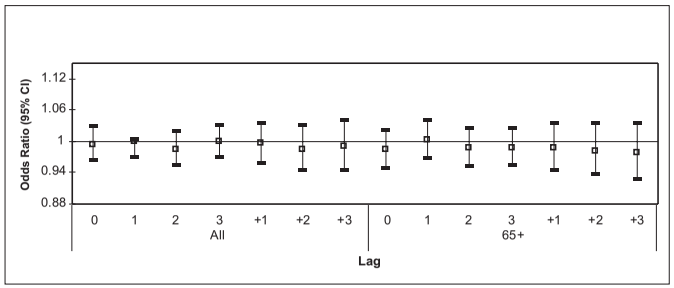
GI Morbidity



CVD Morbidity



COPD Morbidity



Asthma Morbidity

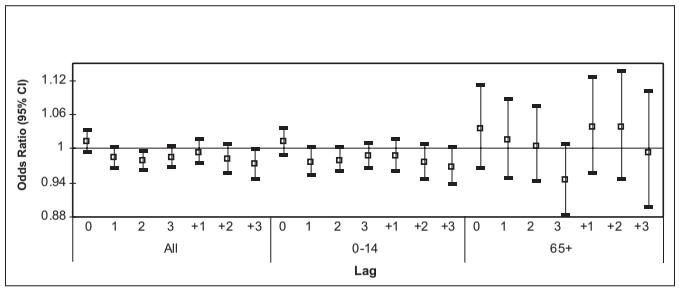


FIGURE 10: Odds Ratio Per Unit Increase in CO (8-hr)(ppm)

international studies (EPA Victoria 2001; Burnett *et al.* 1997). This may be explained by the relatively low CO concentrations experienced in Perth and a decreasing trend in carbon monoxide concentrations.

Changes in daily PM2.5 concentrations were found to be significantly associated with respiratory disease and asthma hospitalisation. Similar findings have been reported for Brisbane, Melbourne and Sydney (Petroeschevsky *et al.* 2001; EPA Victoria 2001; Morgan *et al.* 1998). Strong associations between particles and hospital admissions for respiratory diseases as well as cardiovascular disease have been found in many international studies (e.g. Schwartz, 1994a, 1994b, 1994c, 1996; Samet *et al*. 2000).

While there are differences in the statistical analysis methodology, the significant associations found in the case-crossover study were comparable to those found in other Australian studies and the time series analysis reported in Appendix 2.The time series analysis of the same data as that used in the case-crossover study showed similar associations with the exception of the mortality results (Appendix 2). The time series study design used and analysis method may not have been the optimal model for Perth. As some pollutants may be sensitive to the type of temporal filter used, different temporal filters need to be tested on the Perth data to determine the robustness of the results. Nevertheless, the use of two analysis methods has yielded similar results confirming subsequent conclusions. The significant effects noted for gastrointestinal (GI) disease in the time series study (Appendix 2) can be explained by chance as effects were only observed for one lag period and GI admissions did follow a markedly seasonal pattern with many more admissions in summer. Curiously, GI admissions showed an almost 100% increase from 1996 to 1997 which indicates coding changes or some other error that requires follow up with the Health Department. The APHEA approach may have resulted in mis-specification of the time series model inducing a spurious association.

6. Limitations

The limitations of this study are similar to those of time series studies: its inability to distinguish between mixtures of pollutants; its difficulty in determining independent effects from a range of interrelated, correlated factors; and the confounding of co-pollutant effects. One of the most significant problems is the limited exposure data available. The exposure data were taken from only

three monitoring stations in the network and therefore may not be representative of the study population.

The case-crossover study design can allow selection bias and confounding by factors that vary with time (Bateson and Schwartz 2001). Selection bias occurs when the reference periods are selected on days that are not in the study base that produced the cases. All days in our study served as hazard and reference days, hence selection bias does not occur.

7. Concluding Remarks

Air quality in Perth has been associated with increased risks of hospitalisation for cardiovascular and respiratory diseases including asthma and increased risks of cardiovascular mortality. Particulate concentrations were significantly associated with increased respiratory disease hospitalisations for the elderly and those aged under 14 years. These findings are robust with the application of different analysis techniques and are consistent with the findings of other researchers reported in the national and international literature.

For the first time we have population level data for Perth which suggests emissions to the airshed are having impacts on the health of Western Australians. These data will aid the further development and implementation of the Air Quality Management Plan for Perth, and development of a targetted research program to confirm the impact of Perth's air quality on the health of Western Australians and to measure the success of air quality programs into the future.

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Appendix 1. Summary of Health Effects of the Criteria Pollutants

1.1 Photochemical Oxidants (measured as ozone, O_3)

The concentration of ozone in a polluted atmosphere is usually taken as an indicator of the amount of photochemical smog, because ozone usually comprises about 85 per cent of the total photochemical smog concentration. The rate of production of photochemical smog is limited by the amount of sunlight and reactive organic compounds (ROCs) available, and the quantity produced is generally limited by the amount of NO_x available. Ambient concentrations of ozone vary from season to season and year to year. Ozone and its precursors can be transported long distances, and thus weather conditions principally influence their dispersal.

Ozone in the troposphere as a pollutant should not be confused with its presence in the stratosphere, where it serves the essential function of screening out a large proportion of the sun's harmful ultra-violet rays.

Sources

Ozone (O_3) is a colourless, highly reactive gas with a distinct odour. The reactions between nitrogen oxides (NO_x) and (ROCs) form ozone, in the presence of heat and light. The principal sources of these precursors are motor vehicles (for both), combustion processes (for NO_x), and refining, petrochemical and solvent-based industries (for ROCs).

Air NEPM Standard

The Air NEPM standards for ozone are 0.10 ppm for a 1-hour average and 0.08 ppm for a 4-hour average. These levels are not to be exceeded more than one day per calendar year. Prior to the implementation of the Air NEPM standards, the World Health Organisation (WHO) goals were used for air quality management of the criteria air pollutants in WA. The WHO goal for ozone was 0.08 ppm for a 1-hour average.

Symptoms of exposure to ozone include irritation of the airways and minor lung function changes in both healthy and susceptible individuals. Ozone concentrations have been associated with increased hospital admissions, specifically with respiratory disease, lung function impairment and cardiovascular disease in those 65 years of age or older (Schwartz *et al*. 1996; Ostro and Rothschild 1989; Castillejos *et al.* 1992; Sunyer *et al.* 1996). The available Australian hospitalisation studies conducted in

Brisbane and Sydney show ozone concentrations are also associated with asthma and respiratory disease (Petroeschevsky *et al.* 2001; Morgan *et al.* 1998).

Laden and co-workers (2000) identified several distinct source-related fractions of fine particles and examined the association of these fractions with daily mortality for each city of the Harvard Six Cities Studies using Poisson regression in a generalised additive model. In their meta-analysis of the six cities, a 10 $\mu g/m^3$ increase in PM2.5 from mobile sources accounted for a 3.4% increase in daily mortality (CI: 1.7-5.2%) and from coal combustion sources, a 1.1% increase (CI: 0.3-2.0%). No association was found for PM2.5 from crustal particles and daily mortality.

Based on analyses using 1980 U.S. Vital Statistics data and ambient air pollution data, Ozkaynak and Thurston (1987) concluded the particles from iron and steel emissions and coal combustion were more significant contributors to mortality than soil-derived particles. Recent studies of days dominated by windblown dust and of dust storms support these conclusions (e.g. Schwartz *et al.* 1999; Pope *et al*. 1999).

1.2 Carbon Monoxide (CO)

Sources

Carbon monoxide (CO) is a colourless and odourless gas produced by the incomplete combustion of any carbon-based fuel (e.g. petrol, diesel, oil, gas, wood or coal). In urban areas, motor vehicles are the principal source of CO. In Perth, up to 80% of all CO emissions are a result of motor vehicle exhaust (Australian Academy of Technological Sciences and Engineering 1997).

Power generation, domestic solid fuel heaters and burning vegetation are other significant sources. CO may also be formed in the atmosphere by the oxidation of methane.

Air NEPM Standard

The Air NEPM standard for ambient CO is 9.0 ppm for an 8-hour averaging period. This concentration may not be exceeded on more than one day per calendar year.

Carbon monoxide is absorbed via the lungs, enters the bloodstream and reduces the blood's ability to deliver oxygen to organs and tissues. CO is poisonous to humans at high exposure levels.

Exposure to high levels of CO may result in increased incidence and duration of angina pectoris (chest pain sometimes leading to heart attack), visual impairment, reduced motor skills, poor learning ability, difficulty in performing complex tasks, and low birth weight.

In people with ischaemic heart disease, whose cardiovascular system may already be under considerable stress, exposure to carbon monoxide may increase the risk of adverse health effects. Results from numerous scientific studies indicate that even moderately raised levels of CO can trigger or worsen cardiovascular problems (Morris *et al.* 1995; Burnett *et al.* 1997; Yang *et al.* 1998).

A recent study in London (Poloniecki *et al.* 1997) found significant relationships between average daily CO concentrations and hospital admissions. A rise of 10 ppm in average daily CO corresponded to 23% more admissions for acute myocardial infarction, 6.9% more admissions for heart failure and 23% more admissions for circulatory diseases. Studies elsewhere have found similar results (Morris *et al.* 1995; Burnett *et al.* 1997; Schwartz 1997). Studies in Los Angeles have found a rise in deaths overall as well as a rise in deaths from cardiovascular disease in relation to increases in CO levels after taking into account other pollutants and temperature.

Total mortality was significantly associated with CO and NO_2 ($p<0.05$) in Phoenix (an arid city), while cardiovascular mortality was significantly associated with CO, NO_2, SO_2, PM2.5 and PM10 (Mar *et al.* 2000).

1.3 Nitrogen Dioxide (NO_2)

Sources

Nitrogen dioxide (NO_2), a light brown gas, is a precursor of photochemical smog. The majority of air emissions are in the form of nitric oxide (NO), which can be transformed to NO_2.

At high temperatures, atmospheric nitrogen combines with oxygen to form a mixture of nitric oxide (NO) and nitrogen dioxide (NO_2), referred to as oxides of nitrogen (NO_x). Both gases are emitted by motor vehicle engines, industrial and commercial boilers, in power generation and from other industrial combustion processes. In urban areas motor vehicle emissions are a major source of NO_2.

Indoor sources of NO_2 include unflued gas appliances and other combustion devices. Biogenic sources of NO_2 are lightning and the oxidation of ammonia—a very small component of the total NO_2 emissions in urban areas.

Air NEPM Standard

The Air NEPM standards for NO_2 are 0.12 ppm for a 1-hour average and 0.03 ppm for a 1-year average. The 1-hour average is not to be exceeded more than one day per calendar year. There are no allowable exceedences for the 1-year average.

Nitrogen dioxide irritates the lungs and may lower immunity to respiratory infections. Exposure to high levels of NO_2 causes severe lung injury. NO_2 has been demonstrated to increase the effects of exposure to other pollutants such as ozone, sulfur dioxide and inhalable particles.

Although there have been many studies which have investigated the health effects of nitrogen dioxide, no consistent impacts have been demonstrated. Increases in nitrogen dioxide concentration have been associated with increases in respiratory hospital admissions for all age groups in the UK (Walters *et al.* 1995). A Swiss study (SAPALDIA) group has also reported increases in rhinitis, dyspnoea, wheeze, increase in sputum and chronic cough associated with increases in nitrogen dioxide concentrations in air (Leuenberger 1995).

High numbers of medical consultations for hay fever have been observed in areas with high nitrogen dioxide concentrations and low pollen count in Aichi, Japan (Ozawa *et al*. 1994). It is hypothesised that NO_2 may intensify the allergic response, often in combination with SO_2 (Moseholm *et al.* 1993; Tunnicliffe *et al.* 1994; Wang *et al*. 1995). The Swiss SAPALDIA study has also reported decreases in lung function with increases in nitrogen dioxide concentrations (Schindler *et al*. 1998). In Hong Kong (a sub-tropical city), Wong *et al*. (2001) found significant effects on all the mortality outcomes in the study for NO_2, SO_2 and O_3 during the cool season. Concentrations of NO_2 were associated with daily hospital emergency transports for angina, cardiac insufficiency, myocardial infarction, asthma, acute and chronic bronchitis, and pneumonia.

1.4 Sulfur Dioxide (SO_2)

Sources

Sulfur dioxide (SO_2) is a colourless gas with a sharp irritating odour. It is produced in the combustion of coal and oil, and in the smelting of metallic sulfide ores. Ambient SO_2 concentrations vary seasonally, with higher levels occurring in the winter.

Air NEPM and EPP Standards and Limits

The Air NEPM standards for SO_2 are 0.20 ppm for a 1-hour average, 0.08 ppm for a 24-hour average, and 0.02 ppm for a 1-year average. The 1-hour and 1-day averages are not to be exceeded more than one day per calendar year. There are no allowable exceedences for the 1-year average.

Sulfur dioxide acts directly on the respiratory system, triggering rapid responses within minutes. The maximum effect occurs within 10 to 15 minutes, particularly in those individuals with sensitive airways, such as asthmatics. The most common symptoms are coughing, wheezing, and shortness of breath. Asthmatics are most sensitive to SO_2, usually suffering an acute response associated with a sharp short-term decrease in lung function. However, repeated exposure does not appear to induce a worsening of symptoms. Exercise has been shown to exacerbate the effect of sulfur dioxide on lung function (Bascom *et al.* 1996).

A number of studies report a range of health effects associated with small increases in SO_2 (Petroeschevsky *et al*. 2001; Chen *et al.* 1998; Bascom e*t al.* 1996). However, it is often difficult to separate SO_2 from particulate matter when investigating such issues. The secondary formation of sulfate particles from sulfur dioxide may also have an additional impact.

Several studies have investigated the relationship between SO_2 concentrations in air and lung function, hospital admissions and mortality (Katsouyanni *et al*. 1997; Roemer *et al.* 1993; Lebowitz 1996). One study in Spain reported an increase in emergency room admissions of 6% in summer for an increase of 25 $\mu g/m^3$ of SO_2 (Sunyer *et al*. 1996). A similar association with respiratory hospital admissions for asthma have also been found in Birmingham, UK, and Oulu, Finland (Walters *et al*. 1994; Rossi *et al.* 1993).

1.5 Particles

Airborne particles are a broad class of diverse substances that may be solid or liquid (liquid particles are often called aerosols) and are produced by a wide range of natural and human activities.

Airborne particles are commonly classified by their size as total suspended particles (TSP), visibility-reducing particles (PM2), and inhalable particles (coarse fraction PM10 and fine fraction PM2.5). Total suspended particles are taken to be particles of an equivalent aerodynamic diameter of less than 50 µm. Inhalable particles are grouped into two size categories: those with a diameter of up to 10 µm (PM10) and those with a diameter of up to 2.5 µm (PM2.5). Visibility-reducing particles are small airborne particles ranging from 0.1 to 2.0 µm in size, reducing our ability to see objects at a distance

by scattering light.

Sources

Human activity sources include combustion processes in motor vehicles (particularly diesel-fuelled vehicles), industrial and commercial boilers and incinerators, power generating plants, solid fuel domestic heating, domestic incineration and the burning of vegetation (e.g. for bushfire prevention or clearing of agricultural stubble). Natural sources of airborne particles include fine soil particles and smoke particles from bushfires.

PM10

Inhalable particles are associated with increases in respiratory illnesses such as asthma, bronchitis and emphysema, with an increase in risk related to their size, chemical composition and concentration. Particles in the PM10 size fraction have been strongly associated with increases in the daily prevalence of respiratory symptoms, hospital admissions and mortality.

There is some evidence to suggest that particles within the PM10 fraction, such as PM2.5 and PM1.0, might be more deleterious to health than other size fractions.

Air NEPM Standard

The Air NEPM standard for PM10 is 50 μg/m^3 for a 24-hour averaging period, which may not be exceeded more than 5 days per year.

Research to date suggests that particles less than 10 μm in aerodynamic diameter (PM10) are of a health concern. These small irritant particles easily enter into the lungs and aggravate the lung tissue, the degree to which is determined by their chemical composition (Dockery *et al*. 1992; Ransom and Pope 1992).

Many studies have reported positive relationships between airborne particles and hospitalisation and/or emergency room visits for respiratory illness (Pope *et al.* 1991; Schwartz 1994a; Wordley *et al.* 1997; Thurston 1996; Pope 1996).

For changes of 10 μg/m^3 in daily PM10, an increase in mortality of 1% has been reported (Ostro *et al.* 1996). Several of these studies have reported these associations with concentrations below current air quality standards for particulate matter. Specifically, increases in daily PM10 concentrations have been found to increase the risk of hospital admissions for pneumonia and COPD for individuals aged 65 years or older (Schwartz 1994a, 1994b, 1994c). A similar study found an association between daily PM10 concentrations and ischaemic heart disease admissions (Schwartz and Morris 1995).

Results from the Adventist Health and Smog study suggest that long-term concentrations of airborne

particulate matter have a greater effect on respiratory disease than other air pollutants (Abbey *et al*. 1993a, 1993b, 1995). Associations between particulate air pollution and poor respiratory health in asthmatic children have been reported (Dockery and Pope 1994; Bates *et al.* 1995; Timonen and Pekkanen 1997). These associations have been found at relatively low concentrations of particulate matter (13 to 18 $\mu g/m^3$).

No lower limit for the onset of adverse health effects has yet been observed (Schwartz 1994b; Wordley *et al.* 1997).

PM2.5

Increasing evidence suggests that PM2.5 may be strongly implicated as the major influence on the health effects associated with PM10. Particles in the PM2.5 size fraction can be inhaled more deeply into the lungs than PM10, and have been associated with health effects similar to those of PM10. No lower limit for the onset of adverse health effects has yet been observed.

Currently, an Australian air quality standard or guideline for PM2.5 has not been developed. The US EPA standard is 65 $\mu g/m^3$ (24-hour average).

PM2.5 (estimated from visibility data) has been positively associated with the development of chronic bronchitis (Abbey *et al.* 1995).

Visibility Reducing Particles (Bsp)

Fine particles, mainly in the PM2 size range, cause a reduction in visibility and hence adversely impact visual amenity through the presence of haze.

Visibility reduction may persist for short or quite long periods but, because haze is readily noticeable, a short averaging period of one hour is usually adopted. The Victorian EPA standard for visibility is a local visual distance of 20 kilometres which is equivalent a Bsp value of 2.10×10^{-4} m^{-1} (Bsp is the light scattering coefficient for particles as measured by an integrating nephelometer). This same standard is used as a guideline for visibility in Western Australia.

Appendix 2. Preliminary Time Series Regression Analysis

Previous studies of air pollution and health have used Poisson regression time series analyses. In this study, we followed the APHEA (Air Pollution and Health European Approach) protocol (Katsouyanni *et al.* 1996; Schwartz *et al.* 1996) so that our results could be compared with similar studies elsewhere.

2.1 Study Design

The study design was an ecological time series analysis as utilised by the Air Pollution and Health European Approach (APHEA) group (Katsouyanni *et al.* 1995). It was used to investigate the relationship between daily mortality and daily hospitalisation in the Perth metropolitan area between 1 January 1992 and 31 December 1997.

Time series studies of morbidity are ecological epidemiological studies that look at daily emergency attendance or hospital admissions data over a specified period of time. In the context of air pollution, these studies involve the analysis of daily air pollution data in relation to daily emergency attendance or hospital admissions data. Due to the low number of daily hospital admissions as well as the minor contribution of air pollution to the day-to-day variation of such hospital admissions, these studies require data from large populations over a number of years. The data used in these time series studies consist of routinely collected records of hospital admissions and air pollution from defined geographical areas, thereby eliminating the need for accessing information on individuals per se.

The advantages of time series studies are that they are non-invasive, use routinely collected data, and reduce confounding, since factors such as smoking, blood pressure, diet, socioeconomic factors and genetic predisposition do not vary from day to day and are not correlated with air pollution. As a result, time series studies are relatively inexpensive.

However, time series studies do suffer from a number of limitations. Confounding from variables such as meteorology and temperature may occur, but these factors can be taken into account by the analytical method used, unlike other ecological study designs. Other limitations include:

- The inability to distinguish between mixtures of pollutants in ambient air;
- The use of selected fixed monitoring sites for measuring pollutant levels of ambient air which may not be representative of the study area;
- The inability to differentiate between acute and chronic effects;

- The difficulty in determining independent effects from a range of interrelated, correlated factors;
- The confounding co-pollutant effects;
- The use of ambient data as opposed to the use of personal measures of exposure (Hall 1996).

Time series studies are used to explore correlations between environmental exposures and daily morbidity over time. However, they cannot ascertain direct causal relationships between health status and a pollutant or combined pollutants. Different chemical species within pollutants can interact, causing additive or synergistic effects and it is not possible to include such effects in this type of study design.

Despite these limitations, there have been numerous international studies, using different analytical methods, which have shown remarkably consistent results in regards to the relationship of air pollution and increased morbidity. These studies are consistent with other studies of air pollution using case-control and cohort designs, indicating that the observed associations are relatively robust and are not simply an artefact of confounding.

2.2 Study Area, Study Population and Data Collection

The study area, study population and data collection were as per the case-crossover study and are described in detail in Sections 3.1 and 3.2.

2.3 Statistical Analysis

The APHEA protocol includes a parametric method of analysing epidemiological time series data to detect the short-term health effects of air pollutants. As parts of the protocol may be open to different interpretations, the procedures undertaken in this study are described below.

The basic model fitted was a Gaussian linear regression model for the log of the number of deaths. For all groups, except ‘all age-all causes’, 1 was added to each day’s deaths before log transformation. At each stage of the modelling process, residuals from the fitted model were plotted against time and examined for trends, cyclic variation, outliers, and partial auto-correlation.

Seasonality was controlled by inclusion of the functions:

$$\alpha \sin(2\pi kt/365.25) + \beta \cos(2\pi kt/365.25)$$

for k = 0.5,1,2,3,4,5,6 and t = 1 to 2161 (the number of days of study)

The calculations cover all possible variations in seasonality.

Long-term trends were controlled by the inclusion of the day number in the time series as a variable (t=1 to 2161) and its square. Further dummy variables for year were also tested.

This process of de-trending and de-seasonalising was carried out first for the event data, second for temperature and humidity, and finally for the pollutants themselves. Residuals from each of the processes were plotted against each other to examine the form of the dose-response curve. For each particular pollutant, the residuals from the adjusted model for each separate outcome were regressed on each combination of each type of lag (0, 1, 2, 3, 0-1, 0-2, 0-3) days and each averaging time (1-hr, 4-hr, 8-hr, 24-hr) individually. Only the most statistically significant positive effect was included in the final model as specified in the APHEA protocol. Any significant or large single pollutant effects were subsequently modelled together in pairs.

Exploratory analyses were carried out as described in the APHEA protocol. The computer routine Arpois (Tobias and Campbell 1998) in the statistical computer package STATA (Stata Corporation 1999) was used for the sets of time series analyses. Arpois estimates a Poisson model allowing for auto-correlation and overdispersion (Schwartz *et al.* 1996). This model was developed for the APHEA project (Katsouyanni *et al*. 1996), and has been extensively used to analyse time series regression for counts. The models used were second order auto-regressive models.

Variables indicating public holidays and day of week were included. Influenza epidemics were accounted for by numbers of virus isolates reported by the State Health Laboratories. These were only available for the middle 4 years of the study period and were provided as aggregated monthly figures.

There were very few days for which there were no data available to generate a distribution of air quality concentrations. Days for which no measures of any particular pollutants had been made were ignored in the analyses involving the particular pollutant. When days of missing data occur randomly—as appeared to be the case in this study—this method is not likely to introduce selection bias.

Analyses were also carried out separately for ‘summer’ (November to April) and ‘winter’ (May to October). In addition to the day of the week variables, a separate dummy variable that indicated

public holidays (whose dates vary from year to year) was included. Daily meteorological variables were included with lags applied in the same manner as for the air pollutants. Influenza epidemics were accounted for by numbers of virus isolates reported by the State Health Laboratories. These were only available for the middle 4 years of the study period and were provided as aggregated monthly figures.

2.4 Results

Day-of-the-week effects were not significant for the mortality models, but there were significant non-linear effects of both temperature and humidity. Plots of pollutant effects against residuals from the de-trended de-seasonalised temperature and humidity model did not show any obvious non-linearity for any of the pollutants. The results of the mortality study showed a significant association between nitrogen dioxide and all-cause, respiratory and 'other' (non-specified disease group) mortality in summer. No other significant associations were observed.

The morbidity models used a sixth order model. Day-of-the-week effects were highly significant in the morbidity models. There were also significant non-linear effects of both temperature and humidity in these models. Plots of pollutant effects against residuals from the de-trended de-seasonalised temperature and humidity model did not show any obvious non-linearity for any of the pollutants. Effect sizes and 95% confidence intervals were generated and are expressed in terms of the percent change in rate of a particular response for an increase of either 1 unit (CO and Bsp) or 10 units (NO_2, O_3 and PM2.5). These are of the same order as the amount of variability of each pollutant. They therefore represent the percentage increase in numbers of deaths or numbers of hospitalisations that are predicted to occur when the level of CO increases by 1 part per million, the visibility level increases by 1 bscat/10^4, the NO_2 level increases by 10 parts per billion, the O_3 levels increases by 10 parts per billion, and the modelled PM2.5 levels increase by 10 $\mu g/m^3$. They all therefore predict linear increases (or decreases) on the percentage or logarithmic scale.

The associations were generated for two seasons: summer (November to April) and winter (May to October) as well as all year.

The results of the hospitalisation study showed positive significant relationships between daily hospitalisation for respiratory disease, cardiovascular disease and asthma with daily ambient ozone concentrations in summer with risk estimates per 10 ppb of ozone between 3 and 13% (Table 2.1). Ozone concentration was associated with a 13% increase in risk of hospitalisations of children with asthma. Increases in the risk of daily hospitalisation for all respiratory, asthma, COPD and pneumonia/flu were associated with daily particulate concentrations (Bsp and PM2.5 modelled), with a significant increase of 3.9% in respiratory hospitalisation for the elderly for every 10 $\mu g/m^3$ increase

in concentration.

Increases in risk were also observed for nitrogen dioxide and cardiovascular disease hospitalisation and ozone and gastrointestinal disease hospitalisation.

No significant associations were observed for carbon monoxide and hospitalisation, although positive non-significant relationships were observed for cardiovascular, pneumonia and respiratory disease (> 65 years of age).

Table 2.1 - Hospitalisation and Pollutants (% Change in Risk and 95%CI)

	Averaging Time	Lag	Summer	p	Winter	p	AllYear	p
All respiratory								
[#]NO_2	1h	3	0.8 -0.9,2.5	.36	1.1 -0.6,2.7	.20	1.0 -0.1,2.2	.08
[#]O_3	4h	0-3	**5.0** **1.3,8.8**	.007	-1.6 -5.9,3.0	.49	2.7 -0.1,5.5	.05
[#]$PM_{2.5}$	24h	1	2.9 -1.5,6.9	.20	1.1 -0.2,1.9	.09	1.2 -0.1,1.8	.06
[†]BSP	1h	0-3	1.5 -1.9,4.9	.39	1.2 -0.0,2.4	.06	**1.4** **0.4,2.5**	.008
[‡]CO	8h	0	0.4 -1.2,2.5	.61	-0.5 -1.9,2.1	.45	0.0 -1.1,1.0	.99
Asthma								
NO_2	24h	0	5.3 -1.9,11.3	.15	-2.1 -6.6,7.3	.37	2.0 -1.8,6.0	.30
O_3	4h	0-3	**8.6** **2.4,15.2**	.006	-1.9 -8.9,5.6	.60	**4.8** **0.2,9.6**	.04
$PM_{2.5}$	24h	0-3	5.4 -6.4,19.6	.39	**3.7** **0.3,5.2**	.03	**3.6** **0.4,4.9**	.03
BSP	24h	3	-6.1 -21.9,32.1	.51	**11.2** **1.2,22.2**	.03	7.3 -1.4,16.8	.10
CO	8h	0	0.3 -2.4,3.0	.85	-0.6 -2.8,1.6	.57	-0.3 -1.9,1.4	.72
Cardiovascular								
NO_2	24h	0-2	**9.4** **4.4,14.6**	<.001	0.5 -2.8,3.9	.78	3.7 1.0,6.3	.006
O_3	4h	0-3	**3.1** **0.0,6.4**	.05	2.9 -1.5,7.5	.20	2.8 0.4,5.3	.02
$PM_{2.5}$	24h	0-3	3.9 -2.3,10.4	.22	0.9 -1.2,3.0	.43	1.3 -0.6,3.2	.19
BSP	1h	3	0.2 -1.3,1.7	.80	0.4 -0.4,1.2	.28	0.6 -0.1,1.2	.11
CO	8h	2	-0.2 -1.7,1.3	.79	0.9 -0.5,2.4	.21	0.7 -0.3,1.8	.17
COPD								
NO_2	1h	2	0.9 -4.1,6.1	.74	-3.4 -7.6,1.0	.13	-1.1 -4.2,2.1	.49
O_3	1h	2	1.3 -3.4,6.2	.61	-3.9 -10.7,3.3	.28	0.5 -3.3,4.4	.80
$PM_{2.5}$	24h	1	**15.6** **3.4,29.4**	.01	-0.7 -4.3,3.0	.62	0.7 -2.5,4.0	.67
BSP	24h	3	17.2 -13.6,58.9	.31	**17.2** **0.5,36.5**	.04	**17.7** **3.1,34.3**	.02
CO	8h	0	0.1 -4.2,4.5	.98	-0.9 -4.4,2.8	.62	-0.1 -2.8,2.7	.94

	Averaging Time	Lag	Summer	p	Winter	p	All Year	p
Pneumonia/flu								
[#]NO_2	1h	3	2.9 -0.9,6.9	.14	-1.6 -5.2,2.1	.39	1.3 -1.2,3.9	.32
[#]O_3	8h	3	4.5 -1.1,10.3	.11	1.4 -5.5,8.7	.70	4.2 0.0,8.7	.05
[#]$PM_{2.5}$	24h	2	-0.9 -10.5,9.7	.86	**3.6** **0.8,6.5**	.01	**3.3** **0.7,6.0**	.01
[†]BSP	1h	0-3	5.1 -2.5,13.1	.19	1.6 -1.1,4.4	.24	**3.0** **0.6,5.4**	.02
[‡]CO	8h	0-1	1.7 -2.6,6.3	.44	3.2 -0.7,7.2	.11	2.7 -0.1,5.7	.06
Asthma < 15y								
NO_2	24h	0	5.3 -3.5,14.9	.25	-3.6 -9.3,2.4	.24	1.2 -3.6,6.2	.65
O_3	4h	0-3	**13.0** **5.1,21.6**	.001	-5.7 -14.3,3.8	.23	5.3 -0.5,11.4	.08
$PM_{2.5}$	24h	1	6.3 -2.7,16.2	.17	2.3 -0.1,4.8	.06	**2.6** **0.2,5.0**	.03
BSP	24h	3	.5 -19.5,25.5	.97	**13.1** **0.0,27.9**	.05	10.9 -0.5,23.5	.06
CO	8h	0	0.6 -2.6,3.9	.71	-1.6 -4.4,1.2	.26	-0.7 -2.8,1.4	.51
Respiratory > 64y								
NO_2	1h	3	2.3 -0.8,5.4	.15	-0.1 -2.9,2.8	.95	1.5 -0.5,3.5	.15
O_3	4h	0-3	0.6 -5.7,7.3	.86	-4.7 -11.9,3.1	.23	-0.7 -5.4,4.1	.77
$PM_{2.5}$	24h	1	7.1 -0.9,15.7	.08	0.3 -2.1,2.7	.83	1.0 -1.2,3.2	.39
BSP	1h	0-3	5.3 -0.7,11.7	.09	**3.2** **1.1,5.4**	.003	**3.9** **2.0,5.8**	<.001
CO	8h	0	2.1 -0.8,5.2	.16	0.2 -2.2,2.7	.88	1.4 -0.5,3.3	.16
Gastrointestinal								
NO_2	24h	2	2.2 -2.4,6.9	.35	**5.3** **1.2,9.6**	.01	**3.1** **0.2,6.1**	.04
O_3	4h	0-3	**4.1** **0.3,8.0**	.03	**11.3** **5.0,18.0**	<.001	**4.9** **1.8,8.1**	.002
$PM_{2.5}$	24h	1	3.8 -0.8,8.6	.11	1.1 -0.6,2.8	.21	1.5 -0.1,3.0	.06
BSP	1h	1	1.3 -0.6,3.2	.18	0.5 -0.6,1.6	.35	0.8 -0.1,1.7	.08
CO	8h	3	-1.9 -3.6,-0.2	.03	**2.2** **0.3,4.1**	.03	0.1 -1.1,1.4	.84

[#]ppb; [†]bscat/10^4; [‡]**ppm**

2.5 Discussion

Time series studies conducted around the world indicate ambient air quality is associated with increases in morbidity, the most common associations being the effect of particulate and ozone concentrations and respiratory and cardiovascular disease outcomes (Loomis 2000).

The results of this study support these findings. The preliminary time series analysis demonstrated a significant association between NO_2, O_3, and particulate (PM2.5 and Bsp) concentrations and respiratory, asthma, COPD, pneumonia, and cardiovascular hospitalisations. These results are consistent with a number of international studies (Appendix 12)(Schwartz 1994a, 1994b, 1994c, 1996; Department of Health 1998). They are also consistent with those obtained in both the Brisbane and Sydney studies (Petroeschevsky *et al.* 2001).

In particular, ozone was associated with the risk of hospitalisation for all respiratory diseases, cardiovascular diseases and asthma. The effect estimate was higher for asthma patients under 15 years of age. Particulate concentrations were significantly associated with respiratory conditions (all ages and >64 years of age), asthma (all ages and <15 years of age), COPD and pneumonia/flu. The effects estimate was higher for those over 64 years. Again, these results are consistent with the Brisbane and Sydney studies among others (Schwartz 1994a, 1994b; Petroeschevsky *et al.* 2001; Morgan *et al.* 1998).

The Perth results show that when modelled PM2.5 concentrations were used in the analysis as compared to Bsp, similar associations were observed. However, the 95% confidence intervals for PM2.5 were much smaller, indicating a higher level of confidence in the results. The results from this modelled PM2.5 analysis are consistent with the international literature (Loomis 2000).

The significant effects noted for gastrointestinal (GI) disease admissions are difficult to explain, however GI admissions did follow a markedly seasonal pattern (many more admissions in summer) and curiously showed an almost 100% increase from 1996 to 1997 which indicates coding changes or some other error that requires follow up with the Department of Health. The APHEA approach could have resulted in mis-specification of the time series model inducing a spurious association.

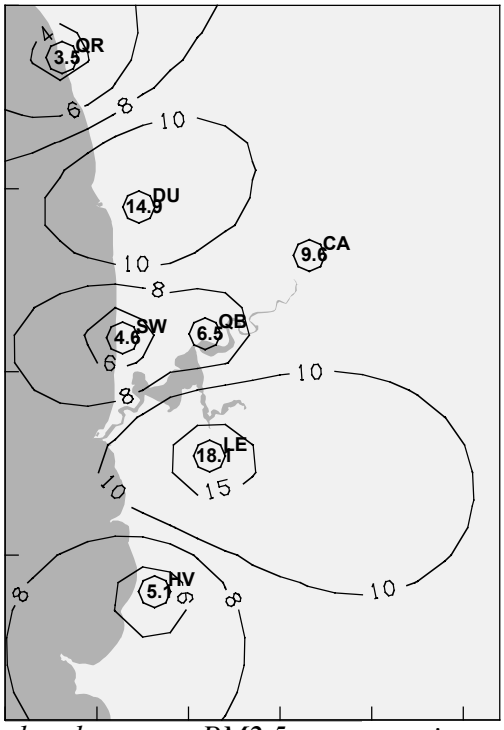
Appendix 3. Commencement Month and Year of Monitoring for Air Pollutants in the Perth Metropolitan Area

Station	SO2	O3	NO2	CO	PM10*	PM2.5	Bsp
Caversham		1/90	9/90	1/92	1/94	1/94	1/90
Duncraig			8/95	8/95	9/94	1/94	1/94
Hope Valley	1/89		1/90				1/90
Leeming			1/97	10/96		1/97	10/96
Queens Buildings			1/90	1/89	1/82		1/90
Quinns Rock		1/93	1/93				1/96
Rolling Green		1/93	1/93				
Rockingham	1/90	1/96	1/96	1/90			
Swanbourne		10/92	10/92		3/94		1/93
Cullucabardee**		93-95	93-95				
Jandakot**		94-95	94-95				
Kenwick**		92-95	92-95				
Rottnest**		93-95	93-95				
Two Rocks**		92-94	92-94				

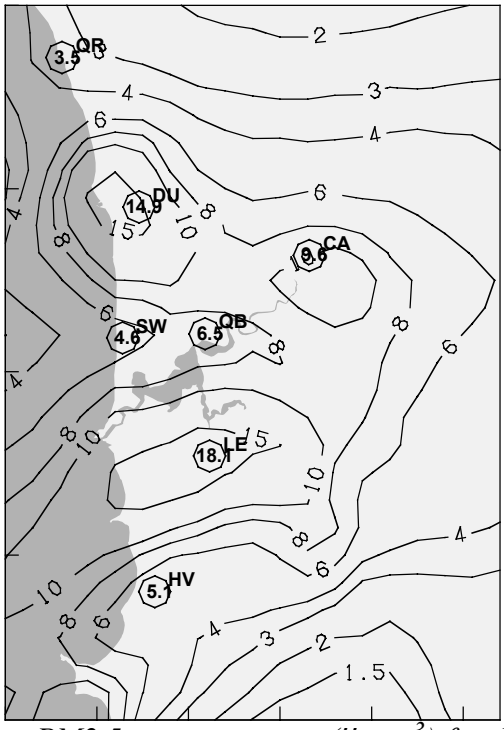
*Sample taken by HIVOL every 6 days (not included in the analysis and not marked on network map in Fig 1)

** Samples taken for the photochemical smog study conducted between 1992 and 1995

Appendix 4. Selected Interpolated and Modelled PM2.5 Concentration Contours for Perth



Interpolated average PM2.5 concentrations ($\mu g\ m^{-3}$), for the period June-July 1997



Average PM2.5 concentrations ($\mu g\ m^{-3}$) for the Perth region, modelled for the period June-July 1997, and adjusted to match measured and estimated PM2.5 values at all monitoring sites.

Appendix 5. Daily Number of Deaths between 1992 and 1996

	November-April				May-October				All year			
	Mean	**SD**	**10th centile**	**90th centile**	**Mean**	**SD**	**10th centile**	**90th centile**	**Mean**	**SD**	**10th centile**	**90th centile**
All deaths	24.2	5.4	18	31	29.2	6.2	21	37	26.8	6.3	19	35
All deaths 65 years and over	18.8	4.7	13	25	23.4	5.6	17	31	21.1	5.7	14	29
CVD deaths	10.2	3.4	6	15	12.9	3.8	8	18	11.6	3.9	7	17
CVD deaths 65 and over		3.1	5	13	11.4	3.6	7	16	10.1	3.6	6	15
Respiratory deaths	1.8	1.4	0	4	2.9	2.0	1	5	2.4	1.8	0	5
Respiratory deaths > 65	1.5	1.3	0	3	2.6	1.9	0	5	2.1	1.7	0	4

Appendix 6. Air Pollution Concentrations and Meteorological Parameters

	November-April				May-October				All year			
	Mean	SD	10^{th} centile	90^{th} centile	Mean	SD	10^{th} centile	90^{th} centile	Mean	SD	10^{th} centile	90^{th} centile
NO_2 - 24h ave (ppb)	9.6	4.8	4.3	15.7	11.1	5.1	4.8	18.0	10.3	5.0	4.4	17.1
NO_2 - 1h max (ppb)	24.7	11.1	12.4	39.2	24.9	8.9	14.4	35.7	24.8	10.1	13.3	37.5
O_3 - 8h max (ppb)	27.7	7.4	19.5	38.3	24.2	4.9	18.4	30.5	25.9	6.5	18.9	34.1
O_3 - 4h max (ppb)	31.1	9.4	20.8	45.0	26.6	5.0	21.1	32.3	28.8	7.8	21	39.5
O_3 - 1h max (ppb)	35.0	12.4	22.1	53.7	28.3	5.6	22.4	34.0	31.6	10.2	22.2	46.1
Bsp - 24h ave (bscat/10^4)	0.20	0.13	0.09	0.33	0.30	0.23	0.12	0.57	0.25	0.20	0.10	0.47
Bsp - 1h max (bscat/10^4)	0.74	0.87	0.25	1.39	1.61	1.70	0.39	3.73	1.2	1.4	0.3	2.6
CO - 8h max (ppm)	2.2	1.3	0.8	4.2	2.4	1.2	1.1	4.2	2.3	1.3	0.9	4.2
*PM_{10} - 24h ave (ppb)	20.6	7.7	12.6	29.7	18.8	7.8	10.7	29.0	19.6	7.8	11.3	29.5
**$PM_{2.5}$ – 24h ave (ppb)	8.6	3.8	4.9	13.1	9.7	4.7	5.1	16.2	9.2	4.3	5.0	14.5
Temperature - 24h ave	21.8	3.4	17.8	26.7	15.1	2.5	12.0	18.2	18.4	4.5	13.0	24.7
Humidity – 24h ave	56.2	15.3	35	75.5	71.4	12.2	53.5	85.0	64.0	15.8	40.5	82.5

* Available only weekly
** Modelled $PM_{2.5}$ data

Appendix 7. Association between Meteorological and Pollution Measures

CORRELATION COEFFICIENTS BETWEEN POLLUTION MEASURES

		NO_2 24-hr	O_3 8-hr	Bsp	CO 24-hr
O_3	**All year**	-0.06			
	Warm	0.35			
	Cool	-0.11			
Bsp	**All year**	0.39	0.01		
	Warm	0.26	0.28		
	Cool	0.42	0.02		
CO	**All year**	0.57	0.00	0.35	
	Warm	0.57	0.16	0.24	
	Cool	0.55	0.00	0.37	
Temp	**All year**	-0.12	0.20	-0.27	
	Warm	0.22	0.51	0.04	0.04
	Cool	-0.19	0.04	-0.31	-0.11
Humidity	**All year**	0.18	-0.12	0.25	
	Warm	0.01	-0.31	0.18	0.26
	Cool	0.15	0.00	0.11	0.33

CORRELATION COEFFICIENTS BETWEEN POLLUTION MEASURES AFTER ADJUSTMENT FOR TREND, SEASONALITY AND METEOROLOGICAL VARIABLES

		NO_2	O_3	B_{SP}
O_3	All year	0.08		
	Summer	0.42		
	Winter	0.04		
B_{SP}	All year	0.41	0.07	
	Summer	0.31	0.32	
	Winter	0.47	0.04	
CO	All year	0.42	0.15	0.34
	Summer	0.25	0.31	0.25
	Winter	0.43	0.15	0.43

Note: Summer is defined as months between November and April, and winter, May to October.

Appendix 8. Case-Crossover Analysis Results – Mortality

8.1 Cardiovascular Disease Mortality: Odds ratio of death per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9990 (0.9971,1.0009)	0.07	n.s.
PM2.5 (μg/m^3)	lag 1	0.9986 (0.9967,1.0005)	0.13	n.s.
PM2.5 (μg/m^3)	lag 2	0.9988 (0.9968,1.0007)	0.18	n.s.
PM2.5 (μg/m^3)	lag 3	1.0007 (0.9988,1.0026)	0.93	n.s.
PM2.5 (μg/m^3)	current day + lag 1	0.9982 (0.9959,1.0005)	0.05	Summer (+)
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	0.9976 (0.9949,1.0003)	0.05	Summer (+)
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	0.9982 (0.9952,1.0012)	0.12	n.s.
Neph, 1-hr max (Bsp)	lag 0	0.9949 (0.9834,1.0065)	0.60	n.s.
Neph, 1-hr max (Bsp)	lag 1	1.0004 (0.9888,1.0122)	0.44	n.s.
Neph, 1-hr max (Bsp)	lag 2	1.0017 (0.9902,1.0133)	0.07	n.s.
Neph, 1-hr max (Bsp)	lag 3	1.0020 (0.9906,1.0136)	0.36	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1	0.9966 (0.9829,1.0106)	0.44	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9982 (0.9827,1.0140)	0.13	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9996 (0.9825,1.0170)	0.37	n.s.
Neph, 24-hr average (Bsp)	lag 0	1.0009 (0.9197,1.0894)	0.20	n.s.
Neph, 24-hr average (Bsp)	lag 1	0.9622 (0.8837,1.0478)	0.67	n.s.
Neph, 24-hr average (Bsp)	lag 2	0.9597 (0.8820,1.0443)	0.23	n.s.
Neph, 24-hr average (Bsp)	lag 3	0.9586 (0.8805,1.0435)	0.76	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1	0.9778 (0.8868,1.0781)	0.35	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.9615 (0.8622,1.0723)	0.26	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9442 (0.8379,1.0641)	0.46	n.s.
CO, 8-hr max (ppm)	lag 0	0.9946 (0.9781,1.0115)	0.32	n.s.
CO, 8-hr max (ppm)	lag 1	1.0025 (0.9861,1.0191)	0.68	n.s.
CO, 8-hr max (ppm)	lag 2	1.0008 (0.9844,1.0175)	0.85	n.s.
CO, 8-hr max (ppm)	lag 3	0.9967 (0.9804,1.0133)	0.64	n.s.
CO, 8-hr max (ppm)	current day + lag 1	0.9993 (0.9802,1.0188)	0.65	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	0.9980 (0.9767,1.0199)	0.93	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	0.9939 (0.9705,1.0180)	0.58	n.s.

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	1.0018 (0.9999,1.0036)	0.93	n.s.
O_3, 1-hr max (ppb)	lag 1	1.0012 (0.9992,1.0031)	0.59	n.s.
O_3, 1-hr max (ppb)	lag 2	1.0003 (0.9985,1.0021)	0.40	n.s.
O_3, 1-hr max (ppb)	lag 3	0.9997 (0.9979,1.0015)	0.31	n.s.
O_3, 1-hr max (ppb)	current day + lag 1	1.0023 (0.9999,1.0046)	0.69	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0020 (0.9992,1.0049)	0.96	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0015 (0.9984,1.0047)	0.89	n.s.
O_3, 4-hr max (ppb)	lag 0	**1.0026 (1.0003,1.0049)**	0.88	n.s.
O_3, 4-hr max (ppb)	lag 1	1.0018 (0.9993,1.0042)	0.66	n.s.
O_3, 4-hr max (ppb)	lag 2	1.0007 (0.9983,1.0030)	0.51	n.s.
O_3, 4-hr max (ppb)	lag 3	0.9996 (0.9973,1.0018)	0.49	n.s.
O_3, 4-hr max (ppb)	current day + lag 1	**1.0033 (1.0003,1.0063)**	0.67	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	1.0029 (0.9994,1.0065)	0.85	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0020 (0.9981,1.0060)	0.94	n.s.
O_3, 8-hr max (ppb)	lag 0	**1.0034 (1.0005,1.0063)**	0.81	n.s.
O_3, 8-hr max (ppb)	lag 1	1.0025 (0.9995,1.0055)	0.92	n.s.
O_3, 8-hr max (ppb)	lag 2	1.0010 (0.9981,1.0038)	0.55	n.s.
O_3, 8-hr max (ppb)	lag 3	1.0000 (0.9973,1.0028)	0.70	n.s.
O_3, 8-hr max (ppb)	current day + lag 1	**1.0042 (1.0006,1.0079)**	0.77	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	1.0035 (0.9993,1.0078)	0.85	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0027 (0.9980,1.0073)	0.87	n.s.
NO_2, 24-hr average (ppb)	lag 0	1.0015 (0.9973,1.0057)	0.16	n.s.
NO_2, 24-hr average (ppb)	lag 1	1.0020 (0.9978,1.0062)	0.19	n.s.
NO_2, 24-hr average (ppb)	lag 2	1.0027 (0.9985,1.0069)	0.14	n.s.
NO_2, 24-hr average (ppb)	lag 3	0.9992 (0.9950,1.0034)	0.71	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1	1.0025 (0.9977,1.0073)	0.15	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0037 (0.9983,1.0091)	0.11	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	1.0030 (0.9972,1.0090)	0.17	n.s.
NO_2, 1-hr max (ppb)	lag 0	1.0008 (0.9990,1.0026)	0.12	n.s.
NO_2, 1-hr max (ppb)	lag 1	1.0011 (0.9992,1.0029)	0.09	n.s.
NO_2, 1-hr max (ppb)	lag 2	1.0009 (0.9991,1.0027)	0.06	n.s.
NO_2, 1-hr max (ppb)	lag 3	1.0001 (0.9983,1.0019)	0.45	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1	1.0013 (0.9992,1.0034)	0.09	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0017 (0.9993,1.0041)	0.05	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0017 (0.9991,1.0043)	0.08	n.s.

8.2 Respiratory Mortality: Odds ratio of death per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9936 (0.9892,0.9981)	0.59	n.s.
PM2.5 (μg/m^3)	lag 1	0.9962 (0.9917,1.0007)	0.39	n.s.
PM2.5 (μg/m^3)	lag 2	1.0002 (0.9962,1.0042)	0.28	n.s.
PM2.5 (μg/m^3)	lag 3	1.0004 (0.9963,1.0046)	0.61	n.s.
PM2.5 (μg/m^3)	current day + lag 1	0.9923 (0.9869,0.9977)	0.85	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	0.9937 (0.9877,0.9997)	0.56	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	0.9943 (0.9876,1.0011)	0.53	n.s.
Neph, 1-hr max (Bsp)	lag 0	0.9733 (0.9487,0.9986)	0.38	n.s.
Neph, 1-hr max (Bsp)	lag 1	0.9747 (0.9496,1.0005)	0.72	n.s.
Neph, 1-hr max (Bsp)	lag 2	0.9909 (0.9663,1.0162)	0.96	n.s.
Neph, 1-hr max (Bsp)	lag 3	0.9868 (0.9625,1.0117)	0.30	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1	0.9643 (0.9355,0.9941)	0.34	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9638 (0.9312,0.9976)	0.45	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9591 (0.9232,0.9965)	0.82	n.s.
Neph, 24-hr average (Bsp)	lag 0	0.7678 (0.6340,0.9299)	0.40	n.s.
Neph, 24-hr average (Bsp)	lag 1	0.8334 (0.6870,1.0111)	0.49	n.s.
Neph, 24-hr average (Bsp)	lag 2	0.9697 (0.8052,1.1679)	0.22	n.s.
Neph, 24-hr average (Bsp)	lag 3	0.9267 (0.7681,1.1181)	0.37	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1	0.7466 (0.5987,0.9311)	0.83	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.7744 (0.6064,0.9890)	0.74	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.7614 (0.5818,0.9963)	0.58	n.s.
CO, 8-hr max (ppm)	lag 0	0.9903 (0.9526,1.0294)	0.46	n.s.
CO, 8-hr max (ppm)	lag 1	0.9892 (0.9527,1.0270)	0.61	n.s.
CO, 8-hr max (ppm)	lag 2	0.9709 (0.9348,1.0083)	0.86	n.s.
CO, 8-hr max (ppm)	lag 3	0.9862 (0.9495,1.0244)	0.93	n.s.
CO, 8-hr max (ppm)	current day + lag 1	0.9906 (0.9475,1.0355)	0.89	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	0.9735 (0.9264,1.0231)	0.94	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	0.9595 (0.9081,1.0138)	0.81	n.s.

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	0.9995 (0.9951,1.0039)	0.92	n.s.
O_3, 1-hr max (ppb)	lag 1	0.9990 (0.9945,1.0034)	0.77	n.s.
O_3, 1-hr max (ppb)	lag 2	1.0010 (0.9968,1.0052)	0.27	n.s.
O_3, 1-hr max (ppb)	lag 3	1.0021 (0.9978,1.0063)	0.78	n.s.
O_3, 1-hr max (ppb)	current day + lag 1	0.9991 (0.9935,1.0046)	0.79	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0001 (0.9936,1.0066)	0.57	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0010 (0.9938,1.0084)	0.67	n.s.
O_3, 4-hr max (ppb)	lag 0	1.0001 (0.9946,1.0057)	0.92	n.s.
O_3, 4-hr max (ppb)	lag 1	1.0004 (0.9948,1.0060)	0.82	n.s.
O_3, 4-hr max (ppb)	lag 2	1.0016 (0.9963,1.0070)	0.37	n.s.
O_3, 4-hr max (ppb)	lag 3	1.0035 (0.9981,1.0088)	0.86	n.s.
O_3, 4-hr max (ppb)	current day + lag 1	1.0009 (0.9939,1.0078)	0.82	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	1.0022 (0.9942,1.0103)	0.65	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0036 (0.9945,1.0127)	0.75	n.s.
O_3, 8-hr max (ppb)	lag 0	1.0026 (0.9958,1.0093)	0.91	n.s.
O_3, 8-hr max (ppb)	lag 1	1.0028 (0.9960,1.0095)	0.92	n.s.
O_3, 8-hr max (ppb)	lag 2	1.0042 (0.9978,1.0106)	0.42	n.s.
O_3, 8-hr max (ppb)	lag 3	1.0062 (0.9998,1.0127)	0.88	n.s.
O_3, 8-hr max (ppb)	current day + lag 1	1.0045 (0.9963,1.0129)	0.87	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	1.0071 (0.9977,1.0167)	0.69	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0093 (0.9988,1.0199)	0.79	n.s.
NO_2, 24-hr average (ppb)	lag 0	0.9937 (0.9844,1.0031)	0.43	n.s.
NO_2, 24-hr average (ppb)	lag 1	0.9949 (0.9857,1.0042)	1.00	n.s.
NO_2, 24-hr average (ppb)	lag 2	0.9952 (0.9860,1.0046)	0.65	n.s.
NO_2, 24-hr average (ppb)	lag 3	0.9999 (0.9905,1.0096)	0.22	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1	0.9923 (0.9817,1.0031)	0.64	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	0.9909 (0.9791,1.0028)	0.59	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	0.9918 (0.9787,1.0051)	0.41	n.s.
NO_2, 1-hr max (ppb)	lag 0	0.9983 (0.9942,1.0024)	0.48	n.s.
NO_2, 1-hr max (ppb)	lag 1	0.9968 (0.9927,1.0008)	0.49	n.s.
NO_2, 1-hr max (ppb)	lag 2	0.9980 (0.9940,1.0021)	0.45	n.s.
NO_2, 1-hr max (ppb)	lag 3	1.0018 (0.9976,1.0060)	0.19	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1	0.9965 (0.9917,1.0013)	0.94	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9958 (0.9904,1.0012)	0.71	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9973 (0.9913,1.0032)	0.46	n.s.

8.3 'Other' Mortality: Odds ratio of death per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 ($\mu g/m^3$)	lag 0	0.9999 (0.9981,1.0019)	0.87	n.s.
PM2.5 ($\mu g/m^3$)	lag 1	0.9989 (0.9970,1.0008)	0.94	n.s.
PM2.5 ($\mu g/m^3$)	lag 2	0.9991 (0.9971,1.0010)	0.34	n.s.
PM2.5 ($\mu g/m^3$)	lag 3	0.9989 (0.9969,1.0009)	0.21	n.s.
PM2.5 ($\mu g/m^3$)	current day + lag 1	0.9992 (0.9969,1.0015)	0.97	n.s.
PM2.5 ($\mu g/m^3$)	current day + lag 1 + lag 2	0.9987 (0.9960,1.0013)	0.66	n.s.
PM2.5 ($\mu g/m^3$)	current day + lag 1 + lag 2 + lag 3	0.9980 (0.9950,1.0011)	0.45	n.s.
Neph, 1-hr max (Bsp)	lag 0	1.0033 (0.9921,1.0147)	0.89	n.s.
Neph, 1-hr max (Bsp)	lag 1	0.9959 (0.9846,1.0073)	0.64	n.s.
Neph, 1-hr max (Bsp)	lag 2	0.9952 (0.9839,1.0066)	0.20	n.s.
Neph, 1-hr max (Bsp)	lag 3	0.9964 (0.9853,1.0075)	0.72	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1	0.9995 (0.9861,1.0131)	0.71	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9966 (0.9815,1.0121)	0.39	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9949 (0.9783,1.0119)	0.42	n.s.
Neph, 24-hr average (Bsp)	lag 0	1.0063 (0.9265,1.0931)	0.87	n.s.
Neph, 24-hr average (Bsp)	lag 1	0.9299 (0.8550,1.0113)	0.62	n.s.
Neph, 24-hr average (Bsp)	lag 2	0.9452 (0.8696,1.0274)	0.18	n.s.
Neph, 24-hr average (Bsp)	lag 3	0.9604 (0.8843,1.0432)	0.39	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1	0.9594 (0.8714,1.0563)	0.91	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.9358 (0.8402,1.0422)	0.55	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9255 (0.8229,1.0410)	0.49	n.s.
CO, 8-hr max (ppm)	lag 0	**1.0176 (1.0013,1.0342)**	0.69	n.s.
CO, 8-hr max (ppm)	lag 1	1.0106 (0.9948,1.0267)	0.35	n.s.
CO, 8-hr max (ppm)	lag 2	0.9998 (0.9840,1.0157)	0.22	n.s.
CO, 8-hr max (ppm)	lag 3	0.9916 (0.9760,1.0076)	0.30	n.s.
CO, 8-hr max (ppm)	current day + lag 1	**1.0187 (1.0000,1.0378)**	0.51	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	1.0135 (0.9927,1.0347)	0.53	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	1.0033 (0.9807,1.0264)	0.55	n.s.

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	0.9984 (0.9969,1.0000)	0.13	n.s.
O_3, 1-hr max (ppb)	lag 1	0.9980 (0.9962,0.9997)	0.07	n.s.
O_3, 1-hr max (ppb)	lag 2	0.9991 (0.9974,1.0008)	0.10	n.s.
O_3, 1-hr max (ppb)	lag 3	0.9994 (0.9978,1.0010)	0.04	Summer (+)
O_3, 1-hr max (ppb)	current day + lag 1	0.9966 (0.9944,0.9988)	0.13	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9959 (0.9933,0.9985)	0.17	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9952 (0.9924,0.9981)	0.13	n.s.
O_3, 4-hr max (ppb)	lag 0	0.9978 (0.9956,0.9999)	0.14	n.s.
O_3, 4-hr max (ppb)	lag 1	0.9973 (0.9951,0.9996)	0.12	n.s.
O_3, 4-hr max (ppb)	lag 2	0.9987 (0.9965,1.0009)	0.12	n.s.
O_3, 4-hr max (ppb)	lag 3	0.9992 (0.9971,1.0013)	0.08	n.s.
O_3, 4-hr max (ppb)	current day + lag 1	0.9957 (0.9930,0.9985)	0.16	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	0.9946 (0.9914,0.9979)	0.20	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9937 (0.9901,0.9973)	0.19	n.s.
O_3, 8-hr max (ppb)	lag 0	0.9974 (0.9948,1.0001)	0.19	n.s.
O_3, 8-hr max (ppb)	lag 1	0.9973 (0.9945,1.0001)	0.12	n.s.
O_3, 8-hr max (ppb)	lag 2	0.9984 (0.9957,1.0010)	0.07	n.s.
O_3, 8-hr max (ppb)	lag 3	0.9998 (0.9973,1.0024)	0.16	n.s.
O_3, 8-hr max (ppb)	current day + lag 1	0.9954 (0.9920,0.9988)	0.19	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	0.9940 (0.9901,0.9980)	0.20	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9936 (0.9893,0.9979)	0.23	n.s.
NO_2, 24-hr average (ppb)	lag 0	1.0001 (0.9961,1.0041)	0.10	n.s.
NO_2, 24-hr average (ppb)	lag 1	0.9997 (0.9958,1.0037)	0.12	n.s.
NO_2, 24-hr average (ppb)	lag 2	1.0012 (0.9973,1.0052)	0.15	n.s.
NO_2, 24-hr average (ppb)	lag 3	0.9989 (0.9950,1.0029)	0.01	Summer (+)
NO_2, 24-hr average (ppb)	current day + lag 1	0.9999 (0.9954,1.0046)	0.08	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0007 (0.9955,1.0058)	0.07	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	1.0000 (0.9945,1.0057)	0.03	Summer (+)
NO_2, 1-hr max (ppb)	lag 0	0.9994 (0.9977,1.0011)	0.15	n.s.
NO_2, 1-hr max (ppb)	lag 1	0.9995 (0.9977,1.0012)	0.07	n.s.
NO_2, 1-hr max (ppb)	lag 2	0.9997 (0.9980,1.0014)	0.04	Summer (+)
NO_2, 1-hr max (ppb)	lag 3	0.9997 (0.9980,1.0014)	0.01	Summer (+)
NO_2, 1-hr max (ppb)	current day + lag 1	0.9992 (0.9972,1.0012)	0.08	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9991 (0.9968,1.0014)	0.04	Summer (+)
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9990 (0.9965,1.0016)	0.02	Summer (+)

Appendix 9. Daily Number of Hospitalisations between 1992 and 1996

	November-April				May-October				All year			
	Mean	SD	10th centile	90th centile	Mean	SD	10th centile	90th centile	Mean	SD	10th centile	90th centile
All respiratory	19.3	6.0	12	27	31.0	8.7	20	42	25.3	9.5	14	38
Asthma	6.9	3.7	3	12	10.7	4.1	6	16	8.8	4.3	4	15
Other COPD	2.4	1.7	0	5	4.1	2.3	1	7	3.3	2.2	1	6
All cardiovascular	24.9	6.4	17	33	28.1	6.4	20	36	26.5	6.6	19	35
Pneumonia & 'flu	3.6	2.1	1	6	5.7	3.1	2	10	4.6	2.9	1	8
Asthma under 15years of age	4.7	3.1	1	9	6.6	3.4	3	11	5.6	3.4	2	10
Gastrointestinal	17.2	5.0	11	24	16.3	4.8	10	22	16.8	4.9	11	23

Appendix 10. Case-Crossover Analysis Results - Hospitalisations

10.1 Cardiovascular Disease Hospitalisations: Odds ratio per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9994 (0.9982,1.0007)	0.049	Winter (+)
PM2.5 (μg/m^3)	lag 1	1.0002 (0.9989,1.0014)	0.26	ns
PM2.5 (μg/m^3)	lag 2	0.9995 (0.9982,1.0007)	0.0098	Winter (+)
PM2.5 (μg/m^3)	lag 3	1.0001 (0.9988,1.0013)	0.00001	Winter (+)
PM2.5 (μg/m^3)	current day + lag 1	0.9997 (0.9982,1.0012)	0.073	ns
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	0.9994 (0.9976,1.0012)	0.017	Winter (+)
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	0.9995 (0.9975,1.0015)	0.001	Winter (+)
Neph, 1-hr max (Bsp)	lag 0	0.9969 (0.9895,1.0043)	0.0292	Winter (+)
Neph, 1-hr max (Bsp)	lag 1	0.9988 (0.9915,1.0061)	0.2449	ns
Neph, 1-hr max (Bsp)	lag 2	0.9943 (0.9869,1.0016)	0.2467	ns
Neph, 1-hr max (Bsp)	lag 3	0.9993 (0.9919,1.0066)	0.0001	Winter (+)
Neph, 1-hr max (Bsp)	current day + lag 1	0.9970 (0.9883,1.0058)	0.0294	Winter (+)
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9941 (0.9843,1.0040)	0.0188	Winter (+)
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9943 (0.9834,1.0052)	0.0005	Winter (+)
Neph, 24-hr average (Bsp)	lag 0	0.9662 (0.9153,1.0199)	0.1216	ns
Neph, 24-hr average (Bsp)	lag 1	1.0225 (0.9685,1.0796)	0.44	ns
Neph, 24-hr average (Bsp)	lag 2	0.9764 (0.9242,1.0315)	0.017	Winter (+)
Neph, 24-hr average (Bsp)	lag 3	0.9878 (0.9383,1.0428)	0.0001	Winter (+)
Neph, 24-hr average (Bsp)	current day + lag 1	0.9933 (0.9331,1.0575)	0.168	ns
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.9827 (0.9158,1.0544)	0.0402	Winter (+)
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9793 (0.9066,1.0578)	0.0028	Winter (+)
CO, 8-hr max (ppm)	lag 0	0.9951 (0.9843,1.0061)	0.0058	Winter (+)
CO, 8-hr max (ppm)	lag 1	1.0087 (0.9960,1.0195)	0.0579	ns
CO, 8-hr max (ppm)	lag 2	1.0022 (0.9909,1.0136)	0.0180	Winter (+)
CO, 8-hr max (ppm)	lag 3	1.0007 (0.9901,1.0115)	0.0002	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1	1.0010 (0.9878,1.0144)	0.0041	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	1.0011 (0.9859,1.0165)	0.0012	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	1.0012 (0.9847,1.0180)	0.0001	Winter (+)

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	0.9995 (0.9983,1.0006)	0.0020	Winter (+)
O_3, 1-hr max (ppb)	lag 1	1.0007 (0.9995,1.0019)	0.0006	Winter (+)
O_3, 1-hr max (ppb)	lag 2	0.9999 (0.9989,1.0011)	0.0013	Winter (+)
O_3, 1-hr max (ppb)	lag 3	1.0001 (0.9990,1.0012)	0.0002	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1	0.9996 (0.9980,1.0012)	0.0002	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9994 (0.9976,1.0013)	0.0001	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9995 (0.9975,1.0016)	0.0000	Winter (+)
O_3, 4-hr max (ppb)	lag 0	0.9992 (0.9977,1.0007)	0.0015	Winter (+)
O_3, 4-hr max (ppb)	lag 1	1.0008 (0.9992,1.0023)	0.0006	Winter (+)
O_3, 4-hr max (ppb)	lag 2	0.9999 (0.9985,1.0014)	0.0010	Winter (+)
O_3, 4-hr max (ppb)	lag 3	1.0002 (0.9988,1.0016)	0.0002	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1	0.9993 (0.9973,1.0013)	0.0002	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	0.9992 (0.9968,1.0015)	0.0001	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9992 (0.9966,1.0018)	0.0000	Winter (+)
O_3, 8-hr max (ppb)	lag 0	0.9993 (0.9975,1.0012)	0.0004	Winter (+)
O_3, 8-hr max (ppb)	lag 1	1.0005 (0.9987,1.0024)	0.0006	Winter (+)
O_3, 8-hr max (ppb)	lag 2	0.9999 (0.9982,1.0017)	0.0006	Winter (+)
O_3, 8-hr max (ppb)	lag 3	1.0003 (0.9986,1.0020)	0.0002	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1	0.9992 (0.9968,1.0016)	0.0001	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	0.9991 (0.9964,1.0018)	0.0000	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9992 (0.9963,1.0022)	0.0000	Winter (+)
NO_2, 24-hr average (ppb)	lag 0	0.9993 (0.9966,1.0019)	0.0044	Winter (+)
NO_2, 24-hr average (ppb)	lag 1	**1.0029 (1.0002,1.0056)**	0.0500	ns
NO_2, 24-hr average (ppb)	lag 2	1.0012 (0.9984,1.0039)	0.0106	Winter (+)
NO_2, 24-hr average (ppb)	lag 3	1.0005 (0.9978,1.0031)	0.0009	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1	1.0015 (0.9984,1.0046)	0.0087	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0019 (0.9984,1.0054)	0.0068	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	1.0020 (0.9982,1.0058)	0.0023	Winter (+)
NO_2, 1-hr max (ppb)	lag 0	0.9998 (0.9986,1.0009)	0.0117	Winter (+)
NO_2, 1-hr max (ppb)	lag 1	1.0010 (0.9998,1.0021)	0.0135	Winter (+)
NO_2, 1-hr max (ppb)	lag 2	1.0003 (0.9991,1.0015)	0.0025	Winter (+)
NO_2, 1-hr max (ppb)	lag 3	0.9999 (0.9988,1.0010)	0.0035	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1	1.0005 (0.9992,1.0019)	0.0058	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0006 (0.9991,1.0022)	0.0031	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0005 (0.9988,1.0022)	0.0016	Winter (+)

10.2 Cardiovascular Disease Hospitalisations (65+ Years): Odds ratio per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9993 (0.9977,1.0008)	0.14	n.s.
PM2.5 (μg/m^3)	lag 1	1.0002 (0.9987,1.0017)	0.15	n.s.
PM2.5 (μg/m^3)	lag 2	1.0002 (0.9986,1.0017)	0.16	n.s.
PM2.5 (μg/m^3)	lag 3	1.0007 (0.9992,1.0022)	0.03	Winter (+)
PM2.5 (μg/m^3)	current day + lag 1	0.9996 (0.9977,1.0015)	0.09	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	0.9997 (0.9976,1.0019)	0.07	n.s.
PM2.5 (μg/m^3)	current day + lag 1 +lag 2 +lag 3	1.0002 (0.9978,1.0026)	0.03	Winter (+)
Neph, 1-hr max (Bsp)	lag 0	1.0008 (0.9918,1.0099)	0.21	n.s.
Neph, 1-hr max (Bsp)	lag 1	0.9951 (0.9862,1.0040)	0.15	n.s.
Neph, 1-hr max (Bsp)	lag 2	0.9997 (0.9908,1.0088)	0.52	n.s.
Neph, 1-hr max (Bsp)	lag 3	1.0063 (0.9974,1.0153)	0.003	Winter (+)
Neph, 1-hr max (Bsp)	current day + lag 1	0.9971 (0.9865,1.0078)	0.08	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9975 (0.9855,1.0096)	0.10	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 +lag 2 +lag 3	1.0013 (0.9880,1.0147)	0.01	Winter (+)
Neph, 24-hr average (Bsp)	lag 0	0.9691 (0.9070,1.0354)	0.25	n.s.
Neph, 24-hr average (Bsp)	lag 1	1.0077 (0.9424,1.0774)	0.38	n.s.
Neph, 24-hr average (Bsp)	lag 2	1.0288 (0.9618,1.1005)	0.23	n.s.
Neph, 24-hr average (Bsp)	lag 3	1.0391 (0.9719,1.1109)	0.03	Winter (+)
Neph, 24-hr average (Bsp)	current day + lag 1	0.9857 (0.9129,1.0642)	0.23	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	1.0043 (0.9213,1.0948)	0.18	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	1.0242 (0.9319,1.1256)	0.07	n.s.
CO, 8-hr max (ppm)	lag 0	0.9891 (0.9758,1.0026)	0.007	Winter (+)
CO, 8-hr max (ppm)	lag 1	1.0080 (0.9936,1.0227)	0.13	n.s.
CO, 8-hr max (ppm)	lag 2	1.0062 (0.9922,1.0205)	0.008	Winter (+)
CO, 8-hr max (ppm)	lag 3	1.0053 (0.9921,1.0187)	0.0009	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1	0.9969 (0.9806,1.0133)	0.01	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	0.9989 (0.9802,1.0179)	0.002	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	1.0011 (0.9808,1.0219)	0.003	Winter (+)

Pollutant	Lag	All Year OR (95 % CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	0.9997 (0.9983,1.0011)	0.11	n.s.
O_3, 1-hr max (ppb)	lag 1	1.0003 (0.9988,1.0018)	0.008	Winter (+)
O_3, 1-hr max (ppb)	lag 2	1.0001 (0.9987,1.0015)	0.04	Winter (+)
O_3, 1-hr max (ppb)	lag 3	1.0002 (0.9989,1.0016)	0.004	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1	0.9995 (0.9975,1.0015)	0.01	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9996 (0.9972,1.0019)	0.01	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9998 (0.9972,1.0023)	0.004	Winter (+)
O_3, 4-hr max (ppb)	lag 0	0.9994 (0.9976,1.0013)	0.09	n.s.
O_3, 4-hr max (ppb)	lag 1	1.0001 (0.9982,1.0020)	0.008	Winter (+)
O_3, 4-hr max (ppb)	lag 2	1.0001 (0.9983,1.0019)	0.03	Winter (+)
O_3, 4-hr max (ppb)	lag 3	1.0002 (0.9985,1.0020)	0.006	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1	0.9990 (0.9965,1.0015)	0.01	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	0.9992 (0.9963,1.0021)	0.008	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9993 (0.9962,1.0025)	0.004	Winter (+)
O_3, 8-hr max (ppb)	lag 0	0.9995 (0.9972,1.0017)	0.05	n.s.
O_3, 8-hr max (ppb)	lag 1	0.9995 (0.9973,1.0018)	0.01	Winter (+)
O_3, 8-hr max (ppb)	lag 2	0.9998 (0.9976,1.0019)	0.02	Winter (+)
O_3, 8-hr max (ppb)	lag 3	1.0003 (0.9981,1.0024)	0.007	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1	0.9986 (0.9957,1.0016)	0.01	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	0.9986 (0.9952,1.0019)	0.005	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9989 (0.9953,1.0026)	0.003	Winter (+)
NO_2, 24-hr average (ppb)	lag 0	0.99995 (0.9967,1.0033)	0.04	Winter (+)
NO_2, 24-hr average (ppb)	lag 1	**1.0047 (1.0013,1.0081)**	0.05	Winter (+)
NO_2, 24-hr average (ppb)	lag 2	1.0023 (0.9989,1.0056)	0.01	Winter (+)
NO_2, 24-hr average (ppb)	lag 3	1.0020 (0.9987,1.0053)	0.003	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1	1.0031 (0.9993,1.0070)	0.03	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0039 (0.9996,1.0083)	0.02	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	1.0046 (0.9999,1.0093)	0.007	Winter (+)
NO_2, 1-hr max (ppb)	lag 0	1.0001 (0.9987,1.0016)	0.09	n.s.
NO_2, 1-hr max (ppb)	lag 1	**1.0016 (1.0001, 1.0031)**	0.02	Winter (+)
NO_2, 1-hr max (ppb)	lag 2	1.0009 (0.9995,1.0024)	0.003	Winter (+)
NO_2, 1-hr max (ppb)	lag 3	1.0005 (0.9991,1.0019)	0.01	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1	1.0012 (0.9995,1.0029)	0.03	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0016 (0.9996,1.0035)	0.01	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0018 (0.9996,1.0039)	0.007	Winter (+)

10.3 Respiratory Hospitalisations: Odds ratio per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9992 (0.9980,1.0005)	0.2402	ns
PM2.5 (μg/m^3)	lag 1	0.9995 (0.9982,1.0007)	0.2066	ns
PM2.5 (μg/m^3)	lag 2	1.0011 (0.9998,1.0024)	0.7151	ns
PM2.5 (μg/m^3)	lag 3	**1.0017 (1.0004,1.0029)**	0.2866	ns
PM2.5 (μg/m^3)	current day + lag 1	0.9990 (0.9975,1.0005)	0.1521	ns
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	0.9998 (0.9980,1.0016)	0.2362	ns
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	1.0009 (0.9989,1.0030)	0.2066	ns
Neph, 1-hr max (Bsp)	lag 0	0.9912 (0.9838,0.9986)	0.9450	ns
Neph, 1-hr max (Bsp)	lag 1	0.9984 (0.9908,1.0060)	0.2230	ns
Neph, 1-hr max (Bsp)	lag 2	1.0035 (0.9957,1.0114)	0.5076	ns
Neph, 1-hr max (Bsp)	lag 3	1.0021 (0.9946,1.0098)	0.2146	ns
Neph, 1-hr max (Bsp)	current day + lag 1	0.9924 (0.9836,1.0013)	0.3342	ns
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9954 (0.9851,1.0058)	0.6982	ns
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9971 (0.9856,1.0088)	0.4333	ns
Neph, 24-hr average (Bsp)	lag 0	0.9501 (0.8990,1.0042)	0.4462	ns
Neph, 24-hr average (Bsp)	lag 1	0.9651 (0.9119,1.0214)	0.6781	ns
Neph, 24-hr average (Bsp)	lag 2	1.0140 (0.9565,1.0749)	0.4784	ns
Neph, 24-hr average (Bsp)	lag 3	1.0373 (0.9806,1.0972)	0.3753	ns
Neph, 24-hr average (Bsp)	current day + lag 1	0.9446 (0.8857,1.0076)	0.4205	ns
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.9618 (0.8937,1.0353)	0.7679	ns
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9840 (0.9075,1.0670)	0.6257	ns
CO, 8-hr max (ppm)	lag 0	1.0055 (0.9937,1.0174)	0.4654	ns
CO, 8-hr max (ppm)	lag 1	0.9940 (0.9826,1.0054)	0.6695	ns
CO, 8-hr max (ppm)	lag 2	0.9863 (0.9752,0.9975)	0.6412	ns
CO, 8-hr max (ppm)	lag 3	0.9918 (0.9808,1.0028)	0.0692	ns
CO, 8-hr max (ppm)	current day + lag 1	0.9987 (0.9850,1.0125)	0.2570	ns
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	0.9901 (0.9750,1.0054)	0.1175	ns
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	0.9845 (0.9682,1.0011)	0.0392	Winter (+)

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	**1.0015 (1.0002,1.0028)**	0.0118	Winter (+)
O_3, 1-hr max (ppb)	lag 1	0.9996 (0.9983,1.0010)	0.1477	ns
O_3, 1-hr max (ppb)	lag 2	0.9995 (0.9981,1.0008)	0.1366	ns
O_3, 1-hr max (ppb)	lag 3	1.0009 (0.9997,1.0022)	0.3939	ns
O_3, 1-hr max (ppb)	current day + lag 1	1.0010 (0.9993,1.0027)	0.0241	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0004 (0.9984,1.0025)	0.0271	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0013 (0.9990,1.0035)	0.0461	Winter (+)
O_3, 4-hr max (ppb)	lag 0	**1.0016 (1.0000,1.0033)**	0.0124	Winter (+)
O_3, 4-hr max (ppb)	lag 1	0.9997 (0.9980,1.0014)	0.1631	ns
O_3, 4-hr max (ppb)	lag 2	0.9997 (0.9980,1.0014)	0.1306	ns
O_3, 4-hr max (ppb)	lag 3	1.0012 (0.9996,1.0028)	0.3128	ns
O_3, 4-hr max (ppb)	current day + lag 1	1.0011 (0.9990,1.0032)	0.0285	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	1.0007 (0.9982,1.0032)	0.0301	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0016 (0.9988,1.0044)	0.0430	Winter (+)
O_3, 8-hr max (ppb)	lag 0	1.0018 (0.9998,1.0038)	0.0140	Winter (+)
O_3, 8-hr max (ppb)	lag 1	0.9995 (0.9974,1.0015)	0.1727	ns
O_3, 8-hr max (ppb)	lag 2	1.0007 (0.9987,1.0027)	0.1437	ns
O_3, 8-hr max (ppb)	lag 3	1.0013 (0.9993,1.0032)	0.4554	ns
O_3, 8-hr max (ppb)	current day + lag 1	1.0010 (0.9985,1.0035)	0.0308	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	1.0012 (0.9983,1.0041)	0.0322	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0019 (0.9987,1.0051)	0.0496	Winter (+)
NO_2, 24-hr average (ppb)	lag 0	1.0009 (0.9981,1.0038)	0.8043	ns
NO_2, 24-hr average (ppb)	lag 1	1.0011 (0.9983,1.0040)	0.2323	ns
NO_2, 24-hr average (ppb)	lag 2	0.9990 (0.9959,1.0021)	0.3074	ns
NO_2, 24-hr average (ppb)	lag 3	0.9991 (0.9962,1.0020)	0.3132	ns
NO_2, 24-hr average (ppb)	current day + lag 1	1.0014 (0.9981,1.0047)	0.4436	ns
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0006 (0.9968,1.0044)	0.3660	ns
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	1.0000 (0.9958,1.0042)	0.3266	ns
NO_2, 1-hr max (ppb)	lag 0	1.0003 (0.9991,1.0015)	0.4612	ns
NO_2, 1-hr max (ppb)	lag 1	1.0000 (0.9987,1.0012)	0.1750	ns
NO_2, 1-hr max (ppb)	lag 2	1.0000 (0.9982,1.0009)	0.0662	ns
NO_2, 1-hr max (ppb)	lag 3	0.9999 (0.9986,1.0012)	0.3643	ns
NO_2, 1-hr max (ppb)	current day + lag 1	1.0002 (0.9987,1.0016)	0.2711	ns
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9999 (0.9982,1.0016)	0.1479	ns
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9998 (0.9979,1.0018)	0.1740	ns

10.4 Respiratory Hospitalisations (65+ Years): Odds ratio per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9999 (0.9976,1.0022)	0.4142	n.s.
PM2.5 (μg/m^3)	lag 1	0.9992 (0.9967,1.0016)	0.2476	n.s.
PM2.5 (μg/m^3)	lag 2	0.9998 (0.9974,1.0022)	0.9228	n.s.
PM2.5 (μg/m^3)	lag 3	1.0011 (0.9985,1.0036)	0.2785	n.s.
PM2.5 (μg/m^3)	current day + lag 1	0.9993 (0.9964,1.0023)	0.2515	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	0.9992 (0.9958,1.0027)	0.4285	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	0.9999 (0.9959,1.0038)	0.3295	n.s.
Neph, 1-hr max (Bsp)	lag 0	0.9894 (0.9756,1.0035)	0.5466	n.s.
Neph, 1-hr max (Bsp)	lag 1	1.0036 (0.9894,1.0179)	0.1550	n.s.
Neph, 1-hr max (Bsp)	lag 2	**1.0196 (1.0048,1.0347)**	0.7535	n.s.
Neph, 1-hr max (Bsp)	lag 3	1.0003 (0.9863,1.0145)	0.0789	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1	0.9948 (0.9782,1.0117)	0.1821	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	1.0070 (0.9874,1.0269)	0.5026	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	1.0069 (0.9850,1.0293)	0.2080	n.s.
Neph, 24-hr average (Bsp)	lag 0	0.9363 (0.8426,1.0404)	0.5468	n.s.
Neph, 24-hr average (Bsp)	lag 1	0.9684 (0.8694,1.0787)	0.6357	n.s.
Neph, 24-hr average (Bsp)	lag 2	1.1008 (0.9866,1.2284)	0.5202	n.s.
Neph, 24-hr average (Bsp)	lag 3	1.0254 (0.9225,1.1399)	0.1463	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1	0.9378 (0.8298,1.0600)	0.4898	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.9985 (0.8691,1.1472)	0.8833	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	1.0116 (0.8686,1.1783)	0.5426	n.s.
CO, 8-hr max (ppm)	lag 0	1.0046 (0.9817,1.0281)	0.0704	n.s.
CO, 8-hr max (ppm)	lag 1	1.0113 (0.9888,1.0342)	0.7269	n.s.
CO, 8-hr max (ppm)	lag 2	0.9841 (0.9626,1.0062)	0.4363	n.s.
CO, 8-hr max (ppm)	lag 3	0.9903 (0.9690,1.0122)	0.1071	n.s.
CO, 8-hr max (ppm)	current day + lag 1	1.0125 (0.9856,1.0401)	0.2197	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	1.0000 (0.9703,1.0308)	0.0992	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	0.9926 (0.9602,1.0260)	0.0388	Winter (+)

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	1.0012 (0.9983,1.0043)	0.2765	n.s.
O_3, 1-hr max (ppb)	lag 1	1.0011 (0.9980,1.0043)	0.1912	n.s.
O_3, 1-hr max (ppb)	lag 2	0.9991 (0.9960,1.0023)	0.3802	n.s.
O_3, 1-hr max (ppb)	lag 3	1.0006 (0.9977,1.0035)	0.4501	n.s.
O_3, 1-hr max (ppb)	current day + lag 1	1.0021 (0.9982,1.0060)	0.1602	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0013 (0.9966,1.0060)	0.2094	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0014 (0.9963,1.0066)	0.2305	n.s.
O_3, 4-hr max (ppb)	lag 0	1.0020 (0.9982,1.0058)	0.1650	n.s.
O_3, 4-hr max (ppb)	lag 1	1.0008 (0.9969,1.0048)	0.3461	n.s.
O_3, 4-hr max (ppb)	lag 2	0.9985 (0.9946,1.0025)	0.3899	n.s.
O_3, 4-hr max (ppb)	lag 3	1.0005 (0.9969,1.0042)	0.4846	n.s.
O_3, 4-hr max (ppb)	current day + lag 1	1.0025 (0.9976,1.0074)	0.1879	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	1.0013 (0.9956,1.0071)	0.2156	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0013 (0.9950,1.0076)	0.2387	n.s.
O_3, 8-hr max (ppb)	lag 0	1.0023 (0.9977,1.0069)	0.2132	n.s.
O_3, 8-hr max (ppb)	lag 1	1.0003 (0.9956,1.0051)	0.5279	n.s.
O_3, 8-hr max (ppb)	lag 2	0.9977 (0.9932,1.0023)	0.4444	n.s.
O_3, 8-hr max (ppb)	lag 3	1.0006 (0.9962,1.0050)	0.5800	n.s.
O_3, 8-hr max (ppb)	current day + lag 1	1.0024 (0.9966,1.0082)	0.3039	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	1.0004 (0.9938,1.0071)	0.2910	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0004 (0.9933,1.0077)	0.3142	n.s.
NO_2, 24-hr average (ppb)	lag 0	0.9986 (0.9920,1.0053)	0.9083	n.s.
NO_2, 24-hr average (ppb)	lag 1	1.0045 (0.9977,1.0113)	0.1415	n.s.
NO_2, 24-hr average (ppb)	lag 2	1.0020 (0.9948,1.0093)	0.2166	n.s.
NO_2, 24-hr average (ppb)	lag 3	1.0039 (0.9970,1.0108)	0.3334	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1	1.0020 (0.9943,1.0097)	0.3950	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0027 (0.9939,1.0117)	0.2876	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	1.0041 (0.9942,1.0140)	0.2840	n.s.
NO_2, 1-hr max (ppb)	lag 0	0.9999 (0.9970,1.0028)	0.6553	n.s.
NO_2, 1-hr max (ppb)	lag 1	1.0010 (0.9981,1.0039)	0.1642	n.s.
NO_2, 1-hr max (ppb)	lag 2	0.9993 (0.9961,1.0025)	0.1680	n.s.
NO_2, 1-hr max (ppb)	lag 3	1.0018 (0.9988,1.0048)	0.2547	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1	1.0006 (0.9972,1.0040)	0.3226	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0001 (0.9962,1.0041)	0.2186	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0009 (0.9965,1.0054)	0.2202	n.s.

10.11 Pneumonia Hospitalisations (65+ Years): Odds ratio per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9978 (0.9934,1.0023)	0.8553	n.s.
PM2.5 (μg/m^3)	lag 1	1.0019 (0.9977,1.0061)	0.8061	n.s.
PM2.5 (μg/m^3)	lag 2	1.0005 (0.9961,1.0049)	0.6714	n.s.
PM2.5 (μg/m^3)	lag 3	**1.0048 (1.0001,1.0097)**	0.8475	n.s.
PM2.5 (μg/m^3)	current day + lag 1	0.9999 (0.9946,1.0053)	0.9541	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	1.0002 (0.9940,1.0065)	0.8255	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	1.0030 (0.9958,1.0102)	0.8455	n.s.
Neph, 1-hr max (Bsp)	lag 0	0.9676 (0.9427,0.9932)	0.2991	n.s.
Neph, 1-hr max (Bsp)	lag 1	0.9879 (0.9627,1.0137)	0.5216	n.s.
Neph, 1-hr max (Bsp)	lag 2	0.9996 (0.9730,1.0269)	0.4840	n.s.
Neph, 1-hr max (Bsp)	lag 3	1.0007 (0.9753,1.0267)	0.8466	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1	0.9688 (0.9395,0.9991)	0.7502	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9718 (0.9377,1.0071)	0.9347	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9744 (0.9365,1.0138)	0.9790	n.s.
Neph, 24-hr average (Bsp)	lag 0	0.8531 (0.7029,1.0355)	0.4738	n.s.
Neph, 24-hr average (Bsp)	lag 1	0.8804 (0.7228,1.0724)	0.1925	n.s.
Neph, 24-hr average (Bsp)	lag 2	0.9830 (0.8052,1.2000)	0.8037	n.s.
Neph, 24-hr average (Bsp)	lag 3	1.0730 (0.8879,1.2967)	0.8844	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1	0.8296 (0.6630,1.0381)	0.2827	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.8481 (0.6593,1.0908)	0.3919	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.8948 (0.6803,1.1770)	0.4558	n.s.
CO, 8-hr max (ppm)	lag 0	1.0057 (0.9645,1.0487)	0.6414	n.s.
CO, 8-hr max (ppm)	lag 1	1.0234 (0.9829,1.0657)	0.4923	n.s.
CO, 8-hr max (ppm)	lag 2	0.9831 (0.9445,1.0233)	0.5593	n.s.
CO, 8-hr max (ppm)	lag 3	1.0114 (0.9720,1.0523)	0.9580	n.s.
CO, 8-hr max (ppm)	current day + lag 1	1.0215 (0.9731,1.0724)	0.4730	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	1.0076 (0.9543,1.0638)	0.2176	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	1.0066 (0.9481,1.0688)	0.1463	n.s.

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	1.0031 (0.9987,1.0075)	0.1508	n.s.
O_3, 1-hr max (ppb)	lag 1	1.0032 (0.9986,1.0079)	0.3397	n.s.
O_3, 1-hr max (ppb)	lag 2	0.9990 (0.9944,1.0036)	0.4384	n.s.
O_3, 1-hr max (ppb)	lag 3	1.0009 (0.9966,1.0051)	0.3033	n.s.
O_3, 1-hr max (ppb)	current day + lag 1	1.0052 (0.9699,1.0110)	0.1906	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0040 (0.9970,1.0110)	0.2341	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0041 (0.9965,1.0118)	0.2191	n.s.
O_3, 4-hr max (ppb)	lag 0	1.0043 (0.9987,1.0099)	0.1272	n.s.
O_3, 4-hr max (ppb)	lag 1	1.0040 (0.9981,1.0099)	0.5245	n.s.
O_3, 4-hr max (ppb)	lag 2	0.9981 (0.9923,1.0039)	0.4173	n.s.
O_3, 4-hr max (ppb)	lag 3	1.0013 (0.9959,1.0067)	0.3314	n.s.
O_3, 4-hr max (ppb)	current day + lag 1	1.0067 (0.9995,1.0140)	0.2520	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	1.0048 (0.9961,1.0134)	0.2769	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0050 (0.9956,1.0145)	0.2506	n.s.
O_3, 8-hr max (ppb)	lag 0	1.0054 (0.9986,1.0123)	0.1401	n.s.
O_3, 8-hr max (ppb)	lag 1	1.0038 (0.9968,1.0109)	0.6712	n.s.
O_3, 8-hr max (ppb)	lag 2	0.9978 (0.9910,1.0046)	0.3509	n.s.
O_3, 8-hr max (ppb)	lag 3	1.0024 (0.9959,1.0089)	0.3273	n.s.
O_3, 8-hr max (ppb)	current day + lag 1	1.0074 (0.9987,1.0161)	0.3277	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	1.0047 (0.9948,1.0146)	0.3002	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0053 (0.9946,1.0161)	0.2662	n.s.
NO_2, 24-hr average (ppb)	lag 0	1.0003 (0.9906,1.0101)	0.8398	n.s.
NO_2, 24-hr average (ppb)	lag 1	1.0063 (0.9964,1.0163)	0.9221	n.s.
NO_2, 24-hr average (ppb)	lag 2	1.0013 (0.9907,1.0119)	0.7505	n.s.
NO_2, 24-hr average (ppb)	lag 3	1.0043 (0.9944,1.0143)	0.9362	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1	1.0041 (0.9929,1.0155)	0.8474	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0040 (0.9911,1.0172)	0.9696	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	1.0055 (0.9912,1.0200)	0.9334	n.s.
NO_2, 1-hr max (ppb)	lag 0	1.0010 (0.9968,1.0053)	0.3846	n.s.
NO_2, 1-hr max (ppb)	lag 1	1.0015 (0.9972,1.0058)	0.8072	n.s.
NO_2, 1-hr max (ppb)	lag 2	0.9997 (0.9950,1.0044)	0.5304	n.s.
NO_2, 1-hr max (ppb)	lag 3	1.0010 (0.9967,1.0053)	0.7824	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1	1.0016 (0.9966,1.0066)	0.5689	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0012 (0.9953,1.0070)	0.5398	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0015 (0.9950,1.0080)	0.6063	n.s.

10.12 Gastrointestinal Disease Hospitalisations: Odds ratio per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9981 (0.9965,0.9998)	0.3616	ns
PM2.5 (μg/m^3)	lag 1	0.9992 (0.9976,1.0008)	0.2169	ns
PM2.5 (μg/m^3)	lag 2	1.0007 (0.9991,1.0023)	0.2134	ns
PM2.5 (μg/m^3)	lag 3	1.0005 (0.9989,1.0021)	0.0116	Winter (+)
PM2.5 (μg/m^3)	current day + lag 1	0.9980 (0.9960,1.0000)	0.2102	ns
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	0.9987 (0.9964,1.0010)	0.1561	ns
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	0.9991 (0.9965,1.0017)	0.0473	Winter (+)
Neph, 1-hr max (Bsp)	lag 0	0.9885 (0.9789,0.9982)	0.3196	ns
Neph, 1-hr max (Bsp)	lag 1	1.0037 (0.9940,1.0124)	0.2299	ns
Neph, 1-hr max (Bsp)	lag 2	1.0069 (0.9971,1.0168)	0.1656	ns
Neph, 1-hr max (Bsp)	lag 3	0.9981 (0.9882,1.0080)	0.0056	Winter (+)
Neph, 1-hr max (Bsp)	current day + lag 1	0.9944 (0.9829,1.0060)	0.1381	ns
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9995 (0.9865,1.0127)	0.0949	ns
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9985 (0.9841,1.0131)	0.0128	Winter (+)
Neph, 24-hr average (Bsp)	lag 0	0.9290 (0.8658,0.9967)	0.2874	ns
Neph, 24-hr average (Bsp)	lag 1	0.9984 (0.9308,1.0709)	0.1254	ns
Neph, 24-hr average (Bsp)	lag 2	1.0380 (0.9666,1.1141)	0.2448	ns
Neph, 24-hr average (Bsp)	lag 3	1.0149 (0.9445,1.0905)	0.0023	Winter (+)
Neph, 24-hr average (Bsp)	current day + lag 1	0.9520 (0.8778,1.0325)	0.1164	ns
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	0.9826 (0.8973,1.0759)	0.1086	ns
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9904 (0.8960,1.0946)	0.0217	Winter (+)
CO, 8-hr max (ppm)	lag 0	0.9975 (0.9840,1.0111)	0.2325	ns
CO, 8-hr max (ppm)	lag 1	0.9981 (0.9848,1.0116)	0.0902	ns
CO, 8-hr max (ppm)	lag 2	0.9898 (0.9765,1.0034)	0.0063	Winter (+)
CO, 8-hr max (ppm)	lag 3	0.9966 (0.9833,1.0101)	0.0054	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1	0.9980 (0.9824,1.0137)	0.0751	ns
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	0.9880 (0.9707,1.0056)	0.0130	Winter (+)
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	0.9868 (0.9678,1.0062)	0.0022	Winter (+)

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	1.0002 (0.9989,1.0015)	0.0211	Winter (+)
O_3, 1-hr max (ppb)	lag 1	0.9987 (0.9974,1.0001)	0.0312	Winter (+)
O_3, 1-hr max (ppb)	lag 2	0.9999 (0.9986,1.0013)	0.0006	Winter (+)
O_3, 1-hr max (ppb)	lag 3	0.9999 (0.9986,1.0013)	0.0093	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1	0.9990 (0.9974,1.0007)	0.0145	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9993 (0.9973,1.0012)	0.0021	Winter (+)
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9992 (0.9970,1.0014)	0.0015	Winter (+)
O_3, 4-hr max (ppb)	lag 0	1.0002 (0.9984,1.0019)	0.0202	Winter (+)
O_3, 4-hr max (ppb)	lag 1	0.9984 (0.9967,1.0001)	0.0253	Winter (+)
O_3, 4-hr max (ppb)	lag 2	1.0002 (0.9984,1.0020)	0.0009	Winter (+)
O_3, 4-hr max (ppb)	lag 3	0.9999 (0.9982,1.0017)	0.0128	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1	0.9988 (0.9966,1.0009)	0.0136	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	0.9992 (0.9967,1.0017)	0.0024	Winter (+)
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9991 (0.9963,1.0019)	0.0019	Winter (+)
O_3, 8-hr max (ppb)	lag 0	1.0004 (0.9979,1.0022)	0.0182	Winter (+)
O_3, 8-hr max (ppb)	lag 1	0.9978 (0.9957,1.0000)	0.0282	Winter (+)
O_3, 8-hr max (ppb)	lag 2	1.0005 (0.9983,1.0026)	0.0007	Winter (+)
O_3, 8-hr max (ppb)	lag 3	0.9997 (0.9976,1.0019)	0.0213	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1	0.9982 (0.9956,1.0008)	0.0128	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	0.9988 (0.9958,1.0018)	0.0022	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9987 (0.9954,1.0021)	0.0021	Winter (+)
NO_2, 24-hr average (ppb)	lag 0	0.9972 (0.9939,1.0006)	0.2230	ns
NO_2, 24-hr average (ppb)	lag 1	0.9994 (0.9960,1.0027)	0.0396	Winter (+)
NO_2, 24-hr average (ppb)	lag 2	0.9971 (0.9937,1.0004)	0.0161	Winter (+)
NO_2, 24-hr average (ppb)	lag 3	0.9993 (0.9959,1.0027)	0.0263	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1	0.9977 (0.9939,1.0016)	0.0775	ns
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	0.9967 (0.9924,1.0010)	0.0378	Winter (+)
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	0.9968 (0.9921,1.0015)	0.0220	Winter (+)
NO_2, 1-hr max (ppb)	lag 0	0.9985 (0.9971,0.9999)	0.1477	ns
NO_2, 1-hr max (ppb)	lag 1	0.9993 (0.9979,1.0007)	0.0590	ns
NO_2, 1-hr max (ppb)	lag 2	0.9987 (0.9973,1.0002)	0.0251	Winter (+)
NO_2, 1-hr max (ppb)	lag 3	0.9996 (0.9981,1.0010)	0.0196	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1	0.9984 (0.9968,1.0000)	0.0689	ns
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9980 (0.9962,0.9999)	0.0393	Winter (+)
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9980 (0.9960,1.0001)	0.0201	Winter (+)

10.13 Gastrointestinal Disease Hospitalisations (65+ Years): Odds ratio per unit increase in pollutant, with 95% confidence intervals

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
PM2.5 (μg/m^3)	lag 0	0.9983 (0.9953,1.0014)	0.6605	n.s.
PM2.5 (μg/m^3)	lag 1	1.0011 (0.9982,1.0039)	0.9731	n.s.
PM2.5 (μg/m^3)	lag 2	1.0010 (0.9980,1.0040)	0.6332	n.s.
PM2.5 (μg/m^3)	lag 3	0.9982 (0.9950,1.0014)	0.8095	n.s.
PM2.5 (μg/m^3)	current day + lag 1	0.9997 (0.9961,1.0032)	0.8095	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2	1.0003 (0.9962,1.0045)	0.7192	n.s.
PM2.5 (μg/m^3)	current day + lag 1 + lag 2 + lag 3	0.9993 (0.9946,1.0040)	0.3474	n.s.
Neph, 1-hr max (Bsp)	lag 0	0.9822 (0.9650,0.9997)	0.0919	n.s.
Neph, 1-hr max (Bsp)	lag 1	1.0098 (0.9925,1.0273)	0.6759	n.s.
Neph, 1-hr max (Bsp)	lag 2	0.9982 (0.9807,1.0160)	0.1183	n.s.
Neph, 1-hr max (Bsp)	lag 3	0.9871 (0.9697,1.0048)	0.0564	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1	0.9944 (0.9739,1.0152)	0.1782	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2	0.9940 (0.9708,1.0178)	0.0732	n.s.
Neph, 1-hr max (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9873 (0.9617,1.0136)	0.0193	Winter (+)
Neph, 24-hr average (Bsp)	lag 0	0.9359 (0.8254,1.0610)	0.3929	n.s.
Neph, 24-hr average (Bsp)	lag 1	1.1013 (0.9706,1.2497)	0.5088	n.s.
Neph, 24-hr average (Bsp)	lag 2	1.0485 (0.9210,1.1937)	0.2298	n.s.
Neph, 24-hr average (Bsp)	lag 3	0.8816 (0.7735,1.0049)	0.1245	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1	1.0193 (0.8813,1.1790)	0.4024	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2	1.0441 (0.8862,1.2300)	0.2844	n.s.
Neph, 24-hr average (Bsp)	current day + lag 1 + lag 2 + lag 3	0.9770 (0.8149,1.1713)	0.1413	n.s.
CO, 8-hr max (ppm)	lag 0	1.0110 (0.9863,1.0363)	0.3828	n.s.
CO, 8-hr max (ppm)	lag 1	1.0247 (0.9998,1.0502)	0.6957	n.s.
CO, 8-hr max (ppm)	lag 2	1.0010 (0.9765,1.0261)	0.7092	n.s.
CO, 8-hr max (ppm)	lag 3	1.0038 (0.9793,1.0289)	0.3977	n.s.
CO, 8-hr max (ppm)	current day + lag 1	1.0222 (0.9933,1.0519)	0.5799	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2	1.0154 (0.9831,1.0488)	0.7974	n.s.
CO, 8-hr max (ppm)	current day + lag 1 + lag 2 + lag 3	1.0152 (0.9797,1.0520)	0.9892	n.s.

Pollutant	Lag	All Year OR (95% CI)	Seasonal Difference (*p*-value)	Seasonal Direction
O_3, 1-hr max (ppb)	lag 0	0.9990 (0.9965,1.0015)	0.1983	n.s.
O_3, 1-hr max (ppb)	lag 1	1.0014 (0.9989,1.0039)	0.1839	n.s.
O_3, 1-hr max (ppb)	lag 2	1.0005 (0.9980,1.0030)	0.0459	Winter (+)
O_3, 1-hr max (ppb)	lag 3	0.9988 (0.9963,1.0013)	0.3386	n.s.
O_3, 1-hr max (ppb)	current day + lag 1	0.9998 (0.9968,1.0029)	0.1393	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2	1.0001 (0.9966,1.0037)	0.0712	n.s.
O_3, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9991 (0.9951,1.0032)	0.0809	n.s.
O_3, 4-hr max (ppb)	lag 0	0.9984 (0.9952,1.0016)	0.2358	n.s.
O_3, 4-hr max (ppb)	lag 1	1.0017 (0.9986,1.0049)	0.1206	n.s.
O_3, 4-hr max (ppb)	lag 2	1.0013 (0.9982,1.0045)	0.0265	Winter (+)
O_3, 4-hr max (ppb)	lag 3	0.9988 (0.9955,1.0020)	0.3182	n.s.
O_3, 4-hr max (ppb)	current day + lag 1	0.9995 (0.9956,1.0034)	0.1284	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2	1.0004 (0.9959,1.0049)	0.0557	n.s.
O_3, 4-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9994 (0.9943,1.0046)	0.0704	n.s.
O_3, 8-hr max (ppb)	lag 0	0.9980 (0.9942,1.0019)	0.1893	n.s.
O_3, 8-hr max (ppb)	lag 1	1.0020 (0.9981,1.0059)	0.1504	n.s.
O_3, 8-hr max (ppb)	lag 2	1.0027 (0.9988,1.0067)	0.0104	Winter (+)
O_3, 8-hr max (ppb)	lag 3	0.9992 (0.9952,1.0032)	0.2181	n.s.
O_3, 8-hr max (ppb)	current day + lag 1	0.9992 (0.9945,1.0040)	0.1222	n.s.
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2	1.0010 (0.9955,1.0064)	0.0405	Winter (+)
O_3, 8-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	1.0002 (0.9941,1.0064)	0.0490	Winter (+)
NO_2, 24-hr average (ppb)	lag 0	1.0017 (0.9956,1.0079)	0.8876	n.s.
NO_2, 24-hr average (ppb)	lag 1	1.0050 (0.9988,1.0111)	0.6706	n.s.
NO_2, 24-hr average (ppb)	lag 2	0.9973 (0.9911,1.0035)	0.5276	n.s.
NO_2, 24-hr average (ppb)	lag 3	0.9938 (0.9876,1.0001)	0.8269	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1	1.0044 (0.9974,1.0115)	0.7584	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2	1.0022 (0.9943,1.0102)	0.6948	n.s.
NO_2, 24-hr average (ppb)	current day + lag 1 + lag 2 + lag 3	0.9990 (0.9903,1.0077)	0.6972	n.s.
NO_2, 1-hr max (ppb)	lag 0	0.9994 (0.9968,1.0020)	0.5464	n.s.
NO_2, 1-hr max (ppb)	lag 1	1.0015 (0.9989,1.0040)	0.5410	n.s.
NO_2, 1-hr max (ppb)	lag 2	0.9989 (0.9963,1.0015)	0.6796	n.s.
NO_2, 1-hr max (ppb)	lag 3	0.9977 (0.9950,1.0003)	0.8373	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1	1.0006 (0.9976,1.0036)	0.5071	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2	0.9998 (0.9964,1.0032)	0.5705	n.s.
NO_2, 1-hr max (ppb)	current day + lag 1 + lag 2 + lag 3	0.9986 (0.9949,1.0024)	0.5990	n.s.

Appendix 11. Comparison to Other Studies - Mortality

Appendix 11.1 Respiratory and Cardiovascular Mortality: Risk of death per unit increase in O_3 (with 95% confidence intervals) from various studies

Pollutant	Outcome	Perth[1] OR (95% CI)	Melbourne[2] RR (95% CI)	Sydney[3] RR (95% CI)	Brisbane[4] RR (95% CI)
O_3 8-hour maximum	Respiratory Deaths	1.0026 (0.9958, 1.0093)	1.0029 (0.9999, 1.0059)		
O_3 4-hour maximum	Respiratory Deaths	1.0001 (0.9946, 1.0057)	**1.0027*** (1.0003, 1.0051)		
O_3 1-hour maximum	Respiratory Deaths	0.9995 (0.9951, 1.0039)	**1.0023*** (1.0001, 1.0045)	0.9916 (0.9284,1.0591)	1.013 (0.977, 1.050)
O_3 8-hour maximum	Cardiovascular Deaths	**1.0034*** (1.0005, 1.0063)			
O_3 8-hour maximum (lag 2)	Cardiovascular Deaths	1.0010 (0.9981, 1.0038)	1.0005 (0.9991, 1.0019)		
O_3 8-hour maximum (current day + lag 1)	Cardiovascular Deaths	**1.0042*** (1.0006, 1.0079)			
O_3 4-hour maximum	Cardiovascular Deaths	**1.0026*** (1.0003, 1.0049)			
O_3 4-hour maximum (lag 1)	Cardiovascular Deaths	1.0018 (0.9993, 1.0042)	1.0004 (0.9994, 1.0014)		
O_3 4-hour maximum (current day + lag 1)	Cardiovascular Deaths	**1.0033*** (1.0003, 1.0063)			
O_3 1-hour maximum	Cardiovascular Deaths	1.0018 (0.9999, 1.0036)		1.0252 (0.9975, 1.0538)	1.012 (0.994, 1.031)
O_3 1-hour maximum (lag 1)	Cardiovascular Deaths	1.0012 (0.9992, 1.0031)	1.0005 (0.9995, 1.0015)		

***** statistically significant at the $P<0.05$ level

[1] Conditional logistic regression; per increase of 1 ppb

[2] GAM (Melbourne Mortality Study 1991-1996); per increase of 1 ppb

[3] Poisson regression with Generalised Estimating Equations model (Sydney 1989-1993)

[4] Poisson regression with Generalised Estimating Equations model; per increase of 10 ppb

Appendix 11.2 Respiratory and Cardiovascular Mortality: Risk of death per unit increase in NO_2 (with 95% confidence intervals) from various studies

Pollutant	Outcome	Perth[1] OR (95% CI)	Melbourne[2] RR (95% CI)	Melbourne[3] RR (95% CI)	Sydney[4] RR (95% CI)
NO_2 24-hour average	Respiratory Deaths	0.9937 (0.9844, 1.0031)	1.0035 (0.9985, 1.0085)	1.0027 (0.9983, 1.0071)	
NO_2 24-hour average (lag 1)	Respiratory Deaths	0.9949 (0.9857, 1.0042)			1.0681 (0.9926, 1.1493)
NO_2 24-hour average (lag 1)	Cardiovascular Deaths	1.0020 (0.9978, 1.0062)	**1.0025*** (1.0003, 1.0047)	**1.0021*** (1.0003, 1.0039)	1.0234 (0.9918, 1.0561)
NO_2 1-hour maximum (lag 1)	Cardiovascular Deaths	1.0011 (0.9992, 1.0029)	1.0010 (0.9998, 1.0022)		1.0096 (0.9847, 1.0352)

***** statistically significant at the $P<0.05$ level

[1] Conditional logistic regression; per increase of 1 ppb

[2] Trigonometric modelling (Melbourne Mortality Study 1991-1996); per increase of 1 ppb

[3] GAM (Melbourne Mortality Study 1991-1996); per increase of 1 ppb

[4] Poisson regression using Generalised Estimating Equations model (Sydney 1989-1993)

Appendix 11.3 Cardiovascular Mortality: Risk of death per unit increase in fine particles (with 95% confidence intervals) from various studies

Pollutant	Outcome	Perth[1] OR (95% CI)	Melbourne[2] RR (95% CI)	Sydney[3] RR (95% CI)	Brisbane[4] RR (95% CI)
Bscat/10^4 24-hour average	Cardiovascular Deaths			1.0221 (0.9994, 1.0454)	
Bscat/10^4 1-hour maximum	Cardiovascular Deaths			**1.0296*** (1.0082, 1.0514)	
Bsp 24-hour average	Cardiovascular Deaths	1.0009 (0.9197, 1.0894)			1.010 (0.998, 1.002)
Bsp 24-hour average (lag 1)	Cardiovascular Deaths	0.9622 (0.8837, 1.0478)	1.0361 (0.9939, 1.0802)		
PM10 24-hour average (lag 1)	Cardiovascular Deaths		1.0011 (0.9997, 1.0025)		
PM10 24-hour average (lag 1)**	Cardiovascular Deaths		1.0019 (0.9989, 1.0049)		
PM2.5 24-hour average (lag 1)	Cardiovascular Deaths	0.9986 (0.9967, 1.0005)	1.0025 (0.9997, 1.0053)		

*statistically significant at P<0.01 level
**every 6th day data
[1] Conditional logistic regression
[2] Trigonometric modelling (Melbourne Mortality Study 1991-1996)
[3] Poisson regression using Generalised Estimating Equations (Sydney 1989-1993)
[4] Poisson regression with Generalised Estimating Equations model

Appendix 12 Comparison to Other Studies - Hospitalisations

Appendix 12.1 Respiratory and Cardiovascular Hospitalisations: Risk of hospital admission per unit increase in O_3 (with 95% confidence intervals) from various studies

Pollutant	Outcome	Perth[1] OR (95% CI)	Perth[2] RR (95% CI)	Melbourne[3] RR (95% CI)
O_3 8-hour maximum	Respiratory Disease (All ages)	1.0018 (0.9998,1.0038) lag 0		**1.0014 (1.0006,1.0022) lag2**
O_3 4-hour maximum	Respiratory Disease (65+ years)	1.0013 (0.9981,1.0044) lag 0	0.9930 (0.9460,1.0410) lag0-3	**1.0016 (1.0002,1.0030) lag2**
O_3 4-hour maximum	Respiratory Disease (All ages)	**1.0016 (1.0000,1.0033) lag 0**	1.0270 (0.9990,1.0550) lag0-3	**1.0014 (1.0006,1.0022) lag2**
O_3 1-hour maximum	Respiratory Disease (65+ years)	1.0014 (0.9989,1.0039) lag 0		**1.0015 (1.0003,1.0027) lag2**
O_3 1-hour maximum	Respiratory Disease (All ages)	**1.0015 (1.0002,1.0028) lag 0**		**1.0013 (1.0005,1.0021) lag2**
O_3 4-hour maximum	COPD (All ages)	**1.0047 (1.0002,1.0092) lag 3**		
O_3 1-hour maximum	COPD (All ages)	1.0003 (0.9967,1.0039) lag 0	1.0050 (0.9670,1.0440) lag 2	
O_3 4-hour maximum	Asthma (0-14 years)	1.0031 (0.9997,1.0066) lag 0	1.0530 (0.9950,1.1140) lag0-3	
O_3 4-hour maximum	Asthma (All ages)	1.0017 (0.9990,1.0045) lag 0	**1.0480 (1.0020,1.0960) lag0-3**	
O_3 1-hour maximum	Asthma (0-14 years)	**1.0031 (1.0003,1.0058) lag 0**		0.9992 (0.9980,1.0004) lag 0
O_3 1-hour maximum	Asthma (All ages)	1.0015 (0.9993,1.0037) lag 0		0.9993 (0.9979,1.0007) lag 1
O_3 8-hour maximum	Cardiovascular Disease (65+ years)	0.9995 (0.9972,1.0017) lag 0		0.9981 (0.9965,0.9997) 5-day avg
O_3 8-hour maximum	Cardiovascular Disease (All ages)	0.9993 (0.9975,1.0012) lag 0		0.9976 (0.9964,0.9988) 5-day avg
O_3 4-hour maximum	Cardiovascular Disease (65+ years)	0.9994 (0.9976,1.0013) lag 0		0.9985 (0.9972,0.9999) 5-day avg
O_3 4-hour maximum	Cardiovascular Disease (All ages)	0.9992 (0.9977,1.0007) lag 0	1.0280 (1.0040,1.0530) lag0-3	0.9981 (0.9969,0.9993) 5-day avg
O_3 1-hour maximum	Cardiovascular Disease (65+ years)	0.9997 (0.9983,1.0011) lag 0		0.9987 (0.9973,1.0001) 5-day avg
O_3 1-hour maximum	Cardiovascular Disease (All ages)	0.9995 (0.9983,1.0006) lag 0		0.9982 (0.9972,0.9992) 5-day avg

[1] Case-crossover conditional logistic regression; per increase of 1 ppb; Perth 1992-1997
[2] Time series analysis; per increase of 10 ppb; Perth 1992-1997
[3] GAM; per increase of 1 ppb; Melbourne 1994-1997 (EPA Victoria, 2001)

Appendix 12.2 Respiratory and Cardiovascular Hospitalisations: Risk of hospital admission per unit increase in particles (with 95% confidence intervals) from various studies

Pollutant	Outcome	Perth[1] OR (95% CI)	Perth[2] RR (95% CI)	Melbourne[3] RR (95% CI)
Bsp 24-hour average	Respiratory Disease (0-14 years)			0.9811 (0.9360,1.0283) lag 2
Bsp 24-hour average	Respiratory Disease (15-64 years)			**1.0784 (1.0121,1.1491) 3-day avg**
Bsp 24-hour average	Respiratory Disease (65+ years)	1.1008 (0.9866,1.2284) lag 2		**1.0745 (1.0041,1.1499) 5-day avg**
Bsp 24-hour average	Respiratory Disease (All ages)	1.0373 (0.9806,1.0972) lag 3		1.0239 (0.9927,1.0561) lag 0
Bsp 1-hour maximum	Respiratory Disease (0-14 years)			0.9940 (0.9700,1.0187) lag 0
Bsp 1-hour maximum	Respiratory Disease (15-64 years)			**1.0383 (1.0063,1.0714) 3-day avg**
Bsp 1-hour maximum	Respiratory Disease (65+ years)	**1.0196 (1.0048,1.0347) lag 2**	**1.0390 (1.0200,1.0580) lag0-3**	**1.0430 (1.0088,1.0783) 5-day avg**
Bsp 1-hour maximum	Respiratory Disease (All ages)	1.0035 (0.9957,1.0114) lag 2	**1.0140 (1.0040,1.0250) lag0-3**	1.0119 (0.9967,1.0273) lag 0
Bsp 24-hour average	COPD (All ages)	1.1656 (0.9931,1.3681) lag 2	**1.1770 (1.0310,1.3430) lag 3**	
Bsp 1-hour maximum	COPD (All ages)	**1.0347 (1.0125,1.0573) lag 2**		
Bsp 24-hour average	Asthma (0-14 years)	1.0675 (0.9460,1.2045) lag 3	1.1090 (0.9950,1.2350) lag 3	**1.1481 (1.0628,1.2403) lag 0**
Bsp 24-hour average	Asthma (All ages)	1.0274 (0.9345,1.1295) lag 3	1.0730 (0.9860,1.1680) lag 3	**1.1394 (1.0582,1.2268) 5-day avg**
Bsp 1-hour maximum	Asthma (0-14 years)	1.0101 (0.9931,1.0274) lag 3		**1.0592 (1.0197,1.1002) lag 0**
Bsp 1-hour maximum	Asthma (All ages)	1.0031 (0.9900,1.0164) lag 3		**1.0766 (1.0374,1.1172) 5-day avg**
Bsp 24-hour average	Cardiovascular Disease (65+ years)	1.0391 (0.9719,1.1109) lag 3		**1.0560 (1.0208,1.0924) lag 1**
Bsp 24-hour average	Cardiovascular Disease (All ages)	1.0225 (0.9685,1.0796) lag 1		**1.0461 (1.0174,1.0757) lag 1**
Bsp 1-hour maximum	Cardiovascular Disease (65+ years)	1.0063 (0.9974,1.0153) lag 3		**1.0352 (1.0143,1.0565) 3-day avg**
Bsp 1-hour maximum	Cardiovascular Disease (All ages)	0.9993 (0.9919,1.0066) lag 3	1.0060 (0.9990,1.0120) lag 3	**1.0274 (1.0104,1.0446) 3-day avg**

[1] Case-crossover conditional logistic regression; Perth 1992-1997
[2] Time series analysis; Perth 1992-1997
[3] GAM; Melbourne 1994-1997 (EPA Victoria, 2001)

Appendix 12.3 Respiratory and Cardiovascular Hospitalisations: Risk of hospital admission per unit increase in NO_2 (with 95% confidence intervals) from various studies

Pollutant	Outcome	Perth[1] OR (95% CI)	Perth[2] RR (95% CI)	Melbourne[3] RR (95% CI)
NO_2 24-hour average	Respiratory Disease (0-14 years)			**1.0079 (1.0038,1.0121) 5-day avg**
NO_2 24-hour average	Respiratory Disease (15-64 years)			**1.0084 (1.0043,1.0126) 5-day avg**
NO_2 24-hour average	Respiratory Disease (65+ years)	**1.0058 (1.0003,1.0113) lag 1**		**1.0110 (1.0070,1.0149) 5-day avg**
NO_2 24-hour average	Respiratory Disease (All ages)	1.0014 ((0.9981,1.0047) current day + lag 1		**1.0078 (1.0055,1.0102) 5-day avg**
NO_2 1-hour maximum	Respiratory Disease (15-64 years)			**1.0045 (1.0020,1.0071) 5-day avg**
NO_2 1-hour maximum	Respiratory Disease (65+ years)	1.0017 (0.9993,1.0041) lag 1	1.0150 (0.9950,1.0350) lag 3	**1.0048 (1.0025,1.0072) 5-day avg**
NO_2 1-hour maximum	Respiratory Disease (All ages)	1.0003 (0.9991,1.0015) lag 0	1.0000 (0.9990,1.0220) lag 3	**1.0043 (1.0029,1.0057) 5-day avg**
NO_2 24-hour average	COPD (All ages)	1.0020 (0.9941,1.0100) lag 1		
NO_2 1-hour maximum	COPD (All ages)	1.0011 (0.9976,1.0045) lag 1	0.9890 (0.9580,1.0210) lag 2	
NO_2 24-hour average	Asthma (0-14 years)	1.0030 (0.9971,1.0090) lag 0	1.0120 (0.9640,1.0620) lag 0	**1.0118 (1.0058,1.0177) 5-day avg**
NO_2 24-hour average	Asthma (All ages)	1.0032 (0.9985,1.0080) lag 0	2.0000 (0.9820,1.0600) lag 0	**1.0145 (1.0099,1.0191) 5-day avg**
NO_2 1-hour maximum	Asthma (0-14 years)	1.0012 (0.9987,1.0038) lag 0		**1.0048 (1.0013,1.0084) 5-day avg**
NO_2 1-hour maximum	Asthma (All ages)	1.0006 (0.9986,1.0027) lag 0		**1.0059 (1.0032,1.0087) 5-day avg**
NO_2 24-hour average	Cardiovascular Disease (0-64 years)			**1.0036 (1.0011,1.0062) lag 0**
NO_2 24-hour average	Cardiovascular Disease (65+ years)	1.0391 (0.9719,1.1109) lag 3		**1.0045 (1.0023,1.0067) 3-day avg**
NO_2 24-hour average	Cardiovascular Disease (All ages)	**1.0029 (1.0002,1.0056) lag 1**	**1.0370 (1.0100,1.0630) lag 0-2**	**1.0040 (1.0022,1.0058) 3-day avg**
NO_2 1-hour maximum	Cardiovascular Disease (0-64 years)			1.0013 (0.9999,1.0027) lag 0
NO_2 1-hour maximum	Cardiovascular Disease (65+ years)	1.0063 (0.9974,1.0153) lag 3		**1.0020 (1.0008,1.0032) 3-day avg**
NO_2 1-hour maximum	Cardiovascular Disease (All ages)	1.0010 (0.9998,1.0021) lag 1		**1.0017 (1.0007,1.0027) 3-day avg**

[1] Case-crossover conditional logistic regression; per increase of 1 ppb; Perth 1992-1997
[2] Time series analysis; per increase of 10 ppb; Perth 1992-1997
[3] GAM; per increase of 1 ppb; Melbourne 1994-1997 (EPA Victoria, 2001)

Appendix 12.4 Respiratory and Cardiovascular Hospitalisations: Risk of hospital admission per unit increase in CO (with 95% confidence intervals) from various studies

Pollutant	**Outcome**	**Perth[1] OR (95% CI)**	**Perth[2] RR (95% CI)**	**Melbourne[3] RR (95% CI)**
CO 8-hour maximum	Respiratory Disease (15-64 years)			**1.0328 (1.0098,1.0564) 3-day avg**
CO 8-hour maximum	Respiratory Disease (65+ years)	1.0113 (0.9888,1.0342) lag 1	1.0140 (0.9950,1.0330) lag 0	**1.0305 (1.0069,1.0546) 5-day avg**
CO 8-hour maximum	Respiratory Disease (All ages)	1.0055 (0.9937,1.0174) lag 0	0.0000 (0.9890,1.0100) lag 0	1.0090 ((0.9992,1.0190) lag 1
CO 1-hour maximum	Respiratory Disease (15-64 years)			**1.0195 (1.0050,1.0342) 3-day avg**
CO 1-hour maximum	Respiratory Disease (65+ years)			**1.0210 (1.0059,1.0363) 5-day avg**
CO 1-hour maximum	Respiratory Disease (All ages)			**1.0101 (1.0010,1.0192) 5-day avg**
CO 8-hour maximum	Asthma (0-14 years)	1.0109 (0.9869,1.0355) lag 0	0.9930 (0.9720,1.0140) lag 0	**1.0606 (1.0274,1.0948) 3-day avg**
CO 8-hour maximum	Asthma (All ages)	1.0130 (0.9936,1.0327) lag 0	0.9970 (0.9810,1.0140) lag 0	**1.0310 (1.0100,1.0524) 5-day avg**
CO 1-hour maximum	Asthma (0-14 years)			**1.0639 (1.0363,1.0922) 3-day avg**
CO 1-hour maximum	Asthma (All ages)			**1.0398 (1.0222,1.0577) 5-day avg**
CO 8-hour maximum	Cardiovascular Disease (0-64 years)			**1.0248 (1.0043,1.0457) 3-day avg**
CO 8-hour maximum	Cardiovascular Disease (65+ years)	1.0080 (0.9936,1.0227) lag 1		**1.0329 (1.0185,1.0476) 3-day avg**
CO 8-hour maximum	Cardiovascular Disease (All ages)	1.0087 (0.9960,1.0195) lag 1	1.0070 (0.9970,1.0180) lag 2	**1.0272 (1.0154,1.0391) 3-day avg**
CO 1-hour maximum	Cardiovascular Disease (0-64 years)			**1.0118 (1.0021,1.0215) lag 0**
CO 1-hour maximum	Cardiovascular Disease (65+ years)			**1.0205 (1.0113,1.0297) 3-day avg**
CO 1-hour maximum	Cardiovascular Disease (All ages)			**1.0173 (1.0098,1.0250) 3-day avg**

[1] Case-crossover conditional logistic regression; per increase of 1 ppm; Perth 1992-1997
[2] Time series analysis; per increase of 1 ppm; Perth 1992-1997
[3] GAM; per increase of 1 ppb; Melbourne 1994-1997 (EPA Victoria, 2001)