EFFECTS OF PARTICULATE AIR POLLUTION ON CARDIORESPIRATORY ADMISSIONS IN CHRISTCHURCH, NZ.

A thesis submitted in partial fulfilment of the requirements for the Degree of Master of Science in Statistics in the University of Canterbury by James A. McGowan

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Abstract

Objective:

In Christchurch there is concern that winter air pollution, dominated by particulate matter (PM$_{10}$) from domestic heating, causes a local increase in cases of cardiorespiratory disease. Our aim was to investigate whether the particulate levels did influence emergency hospital admissions, and if so to what extent.

Method:

Air pollution and meteorological data was obtained from a Canterbury Regional Council monitoring station. Two local hospitals provided data on emergency admissions for both adults and children with cardiac and respiratory disorders. All data was obtained for the period from June 1988 to December 1998. Missing PM$_{10}$ data was interpolated from other known pollution values when necessary. The PM$_{10}$ data was compared to the admissions data using a time series analysis approach, with weather variables controlled for using a generalised additive model.

Results:

There was a significant association between PM$_{10}$ levels and cardiorespiratory admissions. For children and adults combined there was a 3.4% increase in respiratory admissions for every interquartile (14.8 $\mu g/m^3$) increase in PM$_{10}$. In adults there was a 1.3 % increase in cardiac admissions for each interquartile increase in PM$_{10}$. There was no relationship between PM$_{10}$ levels and appendicitis, the condition that we selected to be our control.
Conclusion:

In Christchurch there is a significant relationship between particulate levels and the admissions for cardiac and respiratory illnesses. The size of the effect is comparable to other international studies, and the greatest impact is seen on the respiratory system.
1. Introduction

Our study looked at the air pollution in Christchurch, and primarily investigated the effects this pollution can have on public health.

There has been much concern in Christchurch in recent years regarding the effects of air pollution. The local council is currently investigating the possibility of new regulations to reduce pollutants, such as banning domestic coal fires.

When this proposal first became public, the Coal Producers’ Federation of New Zealand Ltd, along with other companies in the same industry, was understandably opposed to it. They took the Canterbury Regional Council to court, to prevent the proposal from being passed. Their central argument was that there was no proof that coal smoke had any detrimental effects, in Christchurch at least.

To counter this, the Health Research Council of New Zealand, with Phil Hider and Ian Town from the Christchurch School of Medicine, organised funding for a study into the health effects of particulate pollution in Christchurch. I was hired as the research assistant, and spent one year on the analysis, which is outlined in this thesis. I had no previous knowledge of the statistics, meteorology, or medical theory covered, and so started by spending a lot of time studying textbooks and the literature.

In the winter, the air pollution in Christchurch regularly exceeds the recommended guidelines. The climate of Christchurch is mild in the summers and quite cool in the winters. A lot of domestic heating is used on the colder winter nights, when temperatures often drop to near 0°C Celsius. It is this home heating, principally from coal and wood, which contributes the bulk of the pollutants found in Christchurch’s air (NIWA, 1996). On some evenings the
worst of the pollution has dissipated by the morning, on others it can remain until the next evening.

We concentrated specifically on how this pollution affects cardiac and respiratory systems, as these have been found to be the most susceptible (Pope et al, 1992; Spix et al, 1993; Lipfert & Wiyzga, 1995). The aim was to decide if any relationship existed and, if so, to provide a quantitative description of it. A time series approach was used, looking at data collected over the last twelve years.

This research was initially written up for the Australian and New Zealand Journal of Public Health, and is currently in the process of being submitted there. The article is a lot shorter than this thesis, and is aimed at a medical audience rather than statistical. It has been included in the appendices. At the time of submission for this thesis, the article was still being touched up. Hopefully there are a minimum of mistakes, but it is only included as an alternative approach.

Outline

Chapter 2 is an overview of the statistics used in this thesis. It is aimed to assist a reader without much previous statistical experience.

Chapter 3 gives a summary of the local weather patterns in Christchurch, and how they affect the city’s air pollution levels.

Chapter 4 describes our data sets, where we obtained them from, the problems we initially had with them, and how we converted them from their raw state to a form suitable for use in our analysis.
Chapter 5 goes through the methods employed to reach our final results. It shows how the theory of Chapter 2 was put into practise. This is where the bulk of our time was spent.

Chapter 6 explains the results found, and what they mean in qualitative terms.

Chapter 7 explores one way to interpret the results, by looking at the monetary cost of pollution on the health system.

Chapter 8 is the conclusion to the study, and contains further discussion on positive and negative aspects of the project.

In the appendices are the computer programs that were used, the full results table, and the medical report.

Finally, the references, containing all the sources referred to in this thesis combined with those from the Appendix III report.
2. Statistics Overview

A sequence of observations, recorded at regular intervals over time, is known as a \textit{time series} (Moore & McCabe, 1993). The observations can be made, for example, on an hourly, daily, weekly, monthly, quarterly, or yearly basis. Data of this kind is common enough in our everyday lives, and can be found in such forms as hourly share prices, daily temperatures, monthly unemployment rates, or quarterly reports on the economy.

A time series can be affected by various factors, such as an underlying trend, seasonal factors, or unusual events (e.g. an earthquake). It is the objective of time series analysis to interpret the series while also taking such factors into account (Lancaster, 1974).

As an example, our study concentrated on two groups of time series.

The hospital admissions were observations made on a daily basis. Each observation was a count of the number of emergency admissions for that day. Choosing a particular age group or disease type gave rise to more specific time series.

The meteorological data was collected over intervals of either one hour or ten minutes, depending on the variable. This data was from measurements so the resulting numbers were real, not just integers as before. These two data types needed to be treated differently, as shown later. In order to make useful comparisons this data was recalculated into daily intervals (see chapter 4).

When analysing a time series, it is useful to be able to find any systematic movements in it, and then to extract a sense of order from the often random appearance of the information. The purpose is not only to interpret past behaviour, but also to predict future events. So once we know the relationship (if any) between the two data sets above, we will be able to estimate the number of hospital admissions on a given day, once we know what the
meteorological conditions are. Time series analysis is used extensively on many sorts of economic, social, and business information.

The four basic components of a time series variable are the trend, seasonal variation, cyclical variation, and random variation (Fleming & Nellis, 1994).

The trend \((T)\) is the tendency for a time series to steadily increase or decrease over a long period of time. By measuring the nature of an underlying trend, we are able to make long-term projections into the future. In addition, we may wish to eliminate the trend from the data, to highlight the influence of other components.

Seasonal variation \((S)\) follows a complete cycle through the year, with essentially the same general pattern repeating itself year after year. Obvious examples would be the sales of seasonal items like ice cream and heating fuel. To compare data from different times of the year we would have to convert to a seasonally adjusted form.

Regular variation in a time series with a period longer than one year is known as cyclical variation \((C)\). This is commonly found in economic data affected by periodic upsings and downswings in business confidence. The time-span for our study is not long enough to identify any cyclical variations.

And finally, what remains is called random variation \((R)\). Thus it is the remainder left when the regular effects \((T, S, \text{and } C)\) have been removed from the data. If they are removed correctly then it will be completely random and unpredictable.

A typical time series, including all those in this project, will be a sum of these four components, and can be written

\[ Y_t = T_t + S_t + C_t + R_t, \]

where the \(t\) subscript indicates varying over time. Another possible time series structure would be
\[ Y_t = T_t \cdot S_t \cdot C_t \cdot R_t. \]

In this study we look at multiple time series, and compare how they interact with each other. In any relationship between two time series, the above four components can be treated individually if desired. The trend of one time series will only ever influence the trend in other time series, and never the seasonal, cyclical, or random variations. Similar statements apply for the other three components.

To determine the magnitude of these relationships, a \textit{regression} must be performed. A regression is an algorithm that calculates the best model to connect two or more time series. One variable, the \textit{response}, is determined to be a function of the other variables, the \textit{explanatory variables}. If the time series are sufficiently long then it is unlikely that an exact model will be found, and so the closest approximation is sought instead.

There are many different sorts of regression modelling, and they differ in the possible functions they allow. Simple linear regressions will firstly multiply the explanatory variables \((X_i)\) by constants \((\beta)\), and then add them to get the estimated response \(E(Y)\):

\[ E(Y) = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \cdots + \beta_n X_n. \]

More complicated functions are used in other regressions. In a generalised linear model any smooth function can be used instead of multiplying by constants. A smooth function is simply one whose graph consists of a smooth line (with no breaks or cusps). So such an equation would be

\[ E(Y) = \beta_0 + S_1(X_1) + S_2(X_2) + \cdots + S_n(X_n). \]

And rather than merely adding the explanatory variables together afterwards, they could be multiplied or combined in other more complicated ways.

The time series model that we used is the generalised additive model (Hastie & Tibshirana, 1990; Chambers et al, 1983), another step up from the generalised linear model. Here an extra link is needed, between adding the smoothed variables and getting the estimated
response. This depends on the distribution of the data, and because our admissions are count data, we use a Poisson-log link. This amounts to adding the smoothed explanatory variables together, but then taking the exponent of this sum as our approximation to the response (Lindsay, 1997):

$$\ln[E(Y)] = \beta_0 + S_1(X_1) + S_2(X_2) + \cdots + S_n(X_n)$$

Different regressions are used in different situations. The most appropriate model will always depend on the specific data being analysed. If a model is well fitted then the residual differences between the actual and estimated response values will be random. If not, there is still more structure to be studied.
3. Christchurch Meteorology

It is important to consider the meteorology of Christchurch, because what happens to PM$_{10}$ in the atmosphere is almost entirely determined by the local meteorological conditions. In other words, once the PM$_{10}$ is emitted into the air, it will be transported only where the airflow takes it. Eventually it will be depleted by rainfall, although this process is sufficiently slow that we can ignore it. Most of the other air pollutants are depleted due to such things as chemical reactions, gas-particle interactions, and settling due to gravity.

The geography of Christchurch’s surroundings creates a complex meteorological system. The Port Hills, the Southern Alps, the (gently) sloping Canterbury plains, and the nearby coastline all have their own impact on the airflow, and hence on the air pollutants (McKendry et al, 1986). The important effects fall into three groups.

- Temperature inversions. At night the earth’s surface cools, which can result in a layer of warm air sitting above cool air. A situation like this is very stable, and will prevent much dispersion of the air, leading to high pollution concentrations.

- Local winds. On a large-scale weather chart of New Zealand, the wind direction and speed can be estimated as moving parallel to the air pressure isobars. But on a smaller scale, the local geographic features produce more complicated wind patterns (McKendry et al, 1987). Cooling at night-time can lead to a breeze blowing from the Port Hills and/or the Canterbury Plains toward the city. This is known as drainage flow (or katabatic flow), and is essentially only cool air flowing downhill under the influence of gravity. Christchurch also has a sea breeze, which blows from the east or northeast during the daytime. The local winds are demonstrated in Figure 1.
The mountain barrier. The prevailing winds from the west must either pass over the Southern Alps, or detour through the Cook Straight to approach Christchurch from the northeast. This second option is called the *coastal northeasterly*, and is also associated with a trough of low pressure off the eastern coast of the South Island (McKendry et al, 1987). The flow coming over the mountains may reach ground level (foehn winds), creating the well-known nor'wester wind (Owens and Tapper, 1977). Otherwise it will cross above the coastal northeasterly, as it is warmer and therefore less dense.

*Figure 1: Map of Christchurch, showing the three kinds of local winds.*

Once these effects are understood, a better picture of the general airflow can be constructed. On some nights, different areas of the city can have drastically different wind speeds (Sturman, 1985). The calmer regions are toward the centre of the city, which is also where
most of the pollutants originate, so this is where a stagnant build up of pollution can often occur.

The large-scale winds and the sea breeze are often vigorous enough to ventilate the city, cleansing it of any pollutants. But the drainage flows don’t have the same strength, and tend to only redistribute the pollutants, creating higher concentrations than before in certain areas (Oke, 1978). The Port Hills flow is very variable, and may not reach the city centre or last all night. This will affect the pollution released from the southern suburbs. The cold air drainage from the Canterbury Plains is slower to develop, due to the gentler slopes, but will reach right across the city, bringing the pollutants from the west. The other, brisker winds tend to occur only during the day, so the air pollutants have their highest concentrations during the evening and night-time.

The highest pollution levels occur on nights when the winds are light, and the temperature is low. The low temperatures create strong inversion conditions, trapping emitted pollutants within 25m of the ground. Cold nights also see a great increase in the need for home heating, which in turn increases the quantity of pollution being produced. The light winds also aid in the formation of a steady inversion layer.

By exploring this link with meteorology we were able to build a better understanding as to which factors have important effects on pollution, and which don’t. Using this knowledge it is possible to create a computational ‘Box’ model (van den Assem, 1997) of the system. This uses available weather data to predict the PM$_{10}$ concentrations for nights in the future, or the evening of the current day (enabling the CRC to issue suitable warnings), or for days in the past when data was not available. We decided this approach would unnecessarily complicate our analysis for negligible gain. However it was still very useful to know how the structure operated.
4. Data

Study Area

The study area for this project was the city of Christchurch, NZ. Christchurch is a flat city on the eastern edge of the Canterbury Plains, with an area of 452 square kilometres, and a population of 333,000 (CCC website, 1999).

Hospital Admissions Data

The city is serviced by two hospitals, the Christchurch Hospital in the centre, and the Princess Margaret Hospital to the south. Admission data was obtained from the New Zealand Health Information Service on daily numbers of emergency admissions to these hospitals. We chose only those cases whose medical diagnoses fell into the following categories.

Cardiac:
- Ischaemic heart disease (ICD 410-414)
- Dysrhythmias (ICD 427)
- Heart failure (ICD 428)

Respiratory:
- Acute respiratory infection (ICD 460-466)
- Other diseases of the upper respiratory tract (ICD 470-478)
- Pneumonia and influenza (ICD 480-487)
- Chronic obstructive pulmonary disease (COPD) (ICD 491-492 and 494-496)
- Asthma (ICD 493)
The International Classification of Diseases (ICD) 9th Revision assigns a classification number to each disease type (as shown above). For each case we used the diagnosis given when the patient was discharged (they also get one on admission, which won’t be as accurate), but we included them in the count for the day they were admitted. The original data set we received, unbeknownst to us, wasn’t calculated along these lines, so until we had that corrected our results were worryingly off. For each case we also obtained the age group of the patient, being one of the five-year intervals 0-4, 5-9, 10-14, etc. This information enabled us to focus on certain age ranges, such as the very young or elderly, which may have different relationships.

We chose to get appendicitis admission data in addition to the cardiorespiratory categories, to act as a control variable for our analysis. There is currently no known relationship between appendicitis and air pollution, which was supported by our findings.

The admission data was obtained from June 1988 to December 1998 for both hospitals. Together they service the entire Christchurch area, so the two data sets were added together, creating one that didn’t differentiate between the hospitals. Beginning in January 1992, emergency respiratory admissions were predominantly sent to Christchurch Hospital only. The percentage going to Princess Margaret dropped from 30% to 3%. Similarly, in May 1994 the emergency heart admissions were also redirected. This time Princess Margaret went from 83% of the patients to only 3%. These changes wouldn’t affect the totals though, with the hospitals summed together.

**Air Pollution and Meteorological Data**

All pollution and meteorological data was obtained from the Canterbury Regional Council’s monitoring station in the central suburb of St. Albans. This station has been around for the longest in Christchurch, and the time series for this study begin in June 1988 because that is
when it first became operational. In June 1995 three other monitoring stations were established in the outer suburbs of Hornby, Beckenham and Opawa. The information collected from them correlates highly with the St. Albans data, supporting the assumption (CRC 1996) that the pollution levels across Christchurch are relatively uniform, when taken as a 24 hour average. Based on this, we decided to use the St. Albans information only, due to its appropriate location and abundance of information, expecting it to give a good representation of Christchurch as a whole.

The monitoring stations measure six different air pollutants, and five weather variables:

- Carbon monoxide (CO) in mg/m³
- Nitric oxide (NO) in μg/m³
- Nitrogen dioxide (NO₂) in μg/m³
- Total oxides of nitrogen (NOₓ) in ppb
- Particulate matter (PM₁₀) in μg/m³
- Sulphur dioxide (SO₂) in μg/m³

- Temperature at 1m in °C
- Temperature at 10m in °C
- Relative humidity as a percentage
- Wind speed in m/s
- Wind direction in degrees

All of this data was obtained for the time period of June 1988 to December 1998. The PM₁₀ data was measured at hourly intervals from 1/6/88 to 16/8/94, and then at ten-minute intervals from 17/8/94 to 31/12/98. All other measurements were made at half-hourly intervals from 1/6/88 to 18/2/92, and at ten-minute intervals from 19/2/92 to 31/12/98.

The data was recalculated as 24-hour averages for ease of comparison with the hospital data. In accordance with CRC policy, any daily period missing more than 25% of its
measurements was not calculated. Wind speed and direction were not used, as they would have no effect on hospital admissions (except in extreme circumstances).

<table>
<thead>
<tr>
<th>Cardiac Admissions:</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
<th>IQ range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischaemic heart disease</td>
<td>4.5</td>
<td>2.4</td>
<td>0</td>
<td>18</td>
<td>3</td>
</tr>
<tr>
<td>Heart failure</td>
<td>1.4</td>
<td>1.3</td>
<td>0</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>Disrhythmias</td>
<td>1.0</td>
<td>1.0</td>
<td>0</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>Total Cardiac Admissions</td>
<td>6.8</td>
<td>3.0</td>
<td>0</td>
<td>22</td>
<td>4</td>
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<table>
<thead>
<tr>
<th>Respiratory Admissions:</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
<th>IQ range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute respiratory infections</td>
<td>2.5</td>
<td>2.2</td>
<td>0</td>
<td>17</td>
<td>3</td>
</tr>
<tr>
<td>Other diseases of upper tract</td>
<td>0.3</td>
<td>0.6</td>
<td>0</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Pneumonia and influenza</td>
<td>2.3</td>
<td>2.2</td>
<td>0</td>
<td>17</td>
<td>2</td>
</tr>
<tr>
<td>Chronic obstructive</td>
<td>1.9</td>
<td>1.7</td>
<td>0</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>Asthma</td>
<td>3.1</td>
<td>2.0</td>
<td>0</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>Total Respiratory Admissions</td>
<td>10.2</td>
<td>5.5</td>
<td>0</td>
<td>38</td>
<td>7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Appendicitis</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
<th>IQ range</th>
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<tbody>
<tr>
<td></td>
<td>0.9</td>
<td>1.0</td>
<td>0</td>
<td>6</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pollutant Concentrations:</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
<th>IQ range</th>
</tr>
</thead>
<tbody>
<tr>
<td>SO₂ (μg/m³)</td>
<td>7.8</td>
<td>9.6</td>
<td>0</td>
<td>87</td>
<td>8</td>
</tr>
<tr>
<td>NO (μg/m³)</td>
<td>30.1</td>
<td>59.8</td>
<td>0</td>
<td>709</td>
<td>23</td>
</tr>
<tr>
<td>NO₂ (μg/m³)</td>
<td>19.4</td>
<td>14.7</td>
<td>0</td>
<td>184</td>
<td>17</td>
</tr>
<tr>
<td>NOₓ (ppb)</td>
<td>32.5</td>
<td>49.6</td>
<td>0</td>
<td>566</td>
<td>26</td>
</tr>
<tr>
<td>CO (mg/m³)</td>
<td>1.2</td>
<td>1.5</td>
<td>0</td>
<td>15.7</td>
<td>1</td>
</tr>
<tr>
<td>PM₁₀ (μg/m³)</td>
<td>25.2</td>
<td>25.5</td>
<td>0</td>
<td>283</td>
<td>15</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Meteorological Variables:</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
<th>IQ range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Windspeed (m/s)</td>
<td>2.8</td>
<td>1.1</td>
<td>0</td>
<td>6.9</td>
<td>1.5</td>
</tr>
<tr>
<td>Temperature at 1m (°C)</td>
<td>12.5</td>
<td>4.8</td>
<td>0.6</td>
<td>28.4</td>
<td>7.3</td>
</tr>
<tr>
<td>Temperature at 10m (°C)</td>
<td>11.7</td>
<td>4.4</td>
<td>0.9</td>
<td>26.8</td>
<td>6.5</td>
</tr>
<tr>
<td>Relative Humidity (%)</td>
<td>75.8</td>
<td>14.6</td>
<td>21.1</td>
<td>104.2</td>
<td>18.3</td>
</tr>
</tbody>
</table>

Table 1: Summary statistics for daily measurements of hospital admissions, air pollutants, and meteorological variables, in the city of Christchurch, for 1988 - 1998.
Missing Data

The principal difficulty that arose from the pollution data set was that there were missing values, due to both CRC quality control procedures and the fact that monitoring was not performed on all days of the trial period. The PM$_{10}$ data in particular existed on only 80% of days (Figure 2), which was of concern. The first task was to estimate values for the remaining days, based on all the other available information.

![Graph showing PM$_{10}$ concentration over time](image)

*Figure 2: Average daily concentrations of PM$_{10}$ in Christchurch, 1988-1998, raw data.*
Many of the days for which the PM$_{10}$ was unknown already had measurements for other pollutants. So we looked for a relationship between the different pollutants. The correlation coefficient for CO was particularly high (Table 2), so we began there.

<table>
<thead>
<tr>
<th></th>
<th>Correlation with PM$_{10}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM$_{10}$</td>
<td>1.000</td>
</tr>
<tr>
<td>CO</td>
<td>0.843</td>
</tr>
<tr>
<td>NO$_X$</td>
<td>0.813</td>
</tr>
<tr>
<td>SO$_2$</td>
<td>0.504</td>
</tr>
<tr>
<td>Temp</td>
<td>-0.482</td>
</tr>
</tbody>
</table>

*Table 2: Correlation coefficients between PM$_{10}$ and other pollutants.*

Plotting the PM$_{10}$ values versus CO for those days when both existed, we could then calculate a line of best fit. The points were remarkably linear, and so we chose the straight-line equation of

$$E(P) = 14.4C + 8.8$$

where $C$ is the carbon monoxide concentration for the day, and $E(P)$ is our estimation of the corresponding PM$_{10}$ concentration. The units are still as mentioned above. This connection is in keeping with other international studies (Schwartz, 1994), although the exact proportions differ depending on what the emission sources are.

$E(P)$ must also be multiplied by a variable $W$ that depends on the day of the week. This takes into account the minor variations that different days cause. $W$ was calculated by considering the averages of PM$_{10}$ for each day of the week, and can be seen (Table 3) to be reasonably close to 1.
<table>
<thead>
<tr>
<th>Day</th>
<th>W</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monday</td>
<td>1.037</td>
</tr>
<tr>
<td>Tuesday</td>
<td>1.004</td>
</tr>
<tr>
<td>Wednesday</td>
<td>0.961</td>
</tr>
<tr>
<td>Thursday</td>
<td>0.985</td>
</tr>
<tr>
<td>Friday</td>
<td>0.937</td>
</tr>
<tr>
<td>Saturday</td>
<td>1.013</td>
</tr>
<tr>
<td>Sunday</td>
<td>1.063</td>
</tr>
</tbody>
</table>

*Table 3: Values for W, the modifier for days of the week.*

That helped for those days with CO but not PM$_{10}$, and the same approach was then repeated for NO$_X$ and SO$_2$, eventually satisfying all but the 4% of the days on which no pollution data was available. The bulk of these were during the summer of 1993/94 (see Figure 2), when the equipment was not in operation. Fortunately summer levels are fairly constant (and low), and with nine other summers available to us we calculated the average values. The main effects of this study are seen in winter, when the PM$_{10}$ values are significantly higher, so the summer values are less consequential anyway. The few isolated days remaining were interpolated using a moving average. Thus we arrived at our complete data set, as in Figure 3.
Figure 3: Average daily concentrations of PM$_{10}$ in Christchurch, 1988-1998, including estimations for missing values.
5. Methods

With the data collected, the next step was to analyse it. This chapter will go through stages that were taken to reach the final results.

Considerations

Our purpose was to investigate the effects of airborne particulate (PM$_{10}$) on the hospital admissions data. We didn’t use the other pollutants as they weren’t expected to have as much effect, and weren’t as relevant to the Christchurch scenario. Before starting we knew that if there were a relationship, then we would find admissions on the increase shortly after high pollution episodes. There are other factors that can increase hospital admissions too, though, and they must all be taken into consideration.

The weather conditions in particular are very influential on people’s health. Extremes in temperature and heavy rainfall are both known to make people more susceptible to illness. Some of the diseases we examined were more sensitive than others. The cardiac illnesses were relatively oblivious to the seasonal changes when compared to the respiratory cases, which had a significant increase during the winter. Wintertime is when the temperature drops lowest, and consequently when the air pollution is highest. Both of these factors would be causal, although it is difficult to distinguish between the two as they are closely correlated.

Another consideration on the total number of admissions would be the size of the population. Census data from the last three censuses (1986, 1991, and 1996) was obtained from Statistics New Zealand. This showed that the population of Christchurch increased 10% over our ten-year period, and this was included in our later calculations. Also of interest was the notable
prominence of people born in the early 1930s. As the aged they changed the relative sizes of our age groups, which also needed to be considered when investigating the age structures.

The final issue was viral epidemics, or other infectious maladies. We approached the Christchurch Public Hospital for information on these. Their records were unfortunately still written in exercise books, but after some effort from Phil Hider we had them in electronic form. The data was the results of swabs taken at the hospital. The infections were divided into six types: (Influenza A and B, Respiratory Syncitial Virus, and Parainfluenza 1,2, and 3).

On talking with the local doctors we discovered that the swabs were only taken nearer the beginning of winters, while there was still uncertainty as to what type of infections were around. Once a clear picture had been built, they tended to treat the patients without taking swabs. This meant the data was seriously distorted, and of little use as a daily indicator variable.

Viruses aren't associated with pollution, however. Indeed any effects that PM$_{10}$ has on virus transmission (ie by lowering the immune system) would also apply to the general health. The actual effects that contagions have on public health would be independent to the consequences of PM$_{10}$.

From the information we had, the different winters did appear similar, so because our virus data was inadequate as well as superfluous, we chose to exclude it from our working. Only two of our disease categories would be affected anyway - Pneumonia/Influenza and Acute Respiratory Infections.

**Modelling Weather**

So, to distinguish between temperature effects and pollution effects, we decided to attribute as much of the admissions' variations as possible to the meteorological factors, and then to
compare what was left with the PM$_{10}$. This approach would maximise the temperature’s influence, and minimise the PM$_{10}$’s, so our final results would be on the conservative side. It was important to use this method though, to ensure that if we found a relationship then we could be sure that it actually existed.

This is where we used a generalised additive model, comprehensively used in similar studies (Schwartz, 1994; Schwartz, 1996; Higgins et al, 1995). We wanted to model the weather variables with the admissions, but didn’t know what type of connection there would be. We used three explanatory variables:

- $X_1$ is the temperature at a height of 1m.
- $X_2$ is the temperature at a height of 10m.
- $X_3$ is the dew-point temperature. Relative humidity is dependent on the temperature, so instead of using that data, we first converted it to dew-point, which is independent of temperature. The dew-point is the temperature at which dew will form. Calculating dew-point from relative humidity and temperature is a long and uninformative process. The necessary formulae and tables can be found in Battan (1979) or Lutgens & Tarbuck (1979).

A standard linear regression

$$E(Y) = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3$$

isn’t appropriate in our case because we know the effect of temperature on admissions is not linear. For most of the temperature range there is a neutral connection, and only the coldest (and, in other places, hottest) weather would be influential.

It was important to note here that the hospital data was count data. The values were all integers, rather than the unrestricted decimals from the other measurements. Normally this situation would be a Poisson process, but that also requires a constant mean. With our mean admissions changing over the course of the year, and also between years, we got a distribution that is not strictly Poisson. This calls for a Poisson link in our model, as follows:

$$\ln[E(Y)] = \beta_0 + S_1(X_1) + S_2(X_2) + S_3(X_3) .$$

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The logarithmic component reduces the variances, which in turn reduces the carry-over uncertainties in our results.

This was the model at the heart of our project. The regression process calculated what \( \beta_0, X_1, X_2, \) and \( X_3 \) were. Then the estimated values \( E(Y) \) were considered to be the effect that the weather variables had on admissions, and when subtracted from the actual values left the residuals, which are the portion of admissions unrelated to weather.

**Modelling Residuals**

The residuals are no longer integers, or even necessarily non-negative, so the same approach cannot be used again (one reason being that logarithms only operate on positive numbers). We now wanted to compare these residuals with the PM\(_{10}\) data-set. Initially we were unsure how long it would take for the pollution to affect the hospital data, so different lags (of 0-6 days) were tried for the PM\(_{10}\) data.

Once a lag was chosen, we had two time series of numbers to compare, and had to decide if the peaks in one (PM\(_{10}\)) corresponded to peaks in the other. The best way to check this was a normal linear regression. With only the two variables, this is analogous to plotting the data sets as 3870 (x,y) coordinates (a scatter graph), and then calculating the line of best fit (which is the line that minimises the sum of the squares of the distances from each point to that line).

With this line found we could now write the residuals from the first regression \( R \) as a linear function of the PM\(_{10}\) values \( (P) \): \( R = mP + c \).

We knew that the lower quartile (LQ) and upper quartile (UQ) values for the PM\(_{10}\) were 12.7 \( \mu g/m^3 \) and 27.5 \( \mu g/m^3 \) respectively. The regression equation was then used to calculate the corresponding residual values at these two points. The residuals on their own are meaningless, but on taking the difference between them:
\[ m \times 27.5 + c - m \times 12.7 + c = m \times 14.8, \]

we get the expected increase in admissions as PM\textsubscript{10} rises from its LQ value to its UQ value. Divide this expected increase by the mean admissions value:

\[ \frac{m \times 14.8}{A} \times 100\%, \]

and we get the expected percentage increase in admissions per interquartile increase in PM\textsubscript{10}. This is the statistic that we expressed our results as.

In the end our results were still only estimates though, albeit as accurate as possible. It was important to know just how accurate they were. In doing the linear regression, S-Plus simultaneously computed the standard errors for our model. We wanted to convert these to a 95\% confidence interval for our just calculated answer, which is the interval inside which the actual answer will lie with 95\% certainty. This is done in a similar fashion to before. Firstly, the error in the value of \( m \) (written as \( \Delta m \)) replaces \( m \) in the above calculation to give us the standard error for that answer:

\[ \frac{\Delta m \times 14.8}{A} \times 100\%. \]

Then we multiply by 1.96 to get the 95\% confidence error (Moore & McCabe, 1993):

\[ \frac{\Delta m \times 29}{A} \times 100\%. \]

This 95\% confidence error can then be added to and subtracted from our answer to give the 95\% confidence interval (95\% CI).

**Calculating Lags**

The modelling methods above were computed using S-Plus 4.5. Initially, a program was written (LagsCalc, see Appendix I) to compute the above regressions for a given disease and a given lag. The results from this are shown in Table 4. In each row the larger values signify bigger influences at that particular lag.
<table>
<thead>
<tr>
<th>Disease category</th>
<th>Lag of PM10 (in days)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>0.70</td>
</tr>
<tr>
<td>Heart failure</td>
<td>3.05</td>
</tr>
<tr>
<td>Dysrhythmias</td>
<td>1.08</td>
</tr>
<tr>
<td>Heart Total</td>
<td>1.26</td>
</tr>
<tr>
<td>Acute respiratory infections</td>
<td>3.70</td>
</tr>
<tr>
<td>Other diseases of upper tract</td>
<td>-0.25</td>
</tr>
<tr>
<td>Pneumonia and influenza</td>
<td>2.52</td>
</tr>
<tr>
<td>Chronic obstructive</td>
<td>3.56</td>
</tr>
<tr>
<td>Asthma</td>
<td>1.14</td>
</tr>
<tr>
<td>Respiratory Total</td>
<td>2.52</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>0.38</td>
</tr>
</tbody>
</table>

Table 4: Percentage increase of admissions per interquartile PM10 increase, with varying lags.

It is evident that pollution affects some diseases immediately, and others over a period of a few days. For subsequent calculations we needed to associate each disease with a specific lag, so from these results we decided we would use lags of:

- 0 days for the cardiac diseases (ischaemic heart disease, heart failure, dysrhythmias and heart total) and appendicitis.
- 4 days for the respiratory infections (acute respiratory infections, pneumonia and influenza, and other diseases of the upper tract).
- 2 days for the other respiratory categories (chronic obstructive pulmonary disease and asthma) and respiratory total.

Here a lag of 0 days indicates we are comparing the admissions for one day with the pollution levels from the night before. So 2 days lag means 3 nights earlier, and 4 days lag means 5 night earlier. In choosing the lags we had to take into account the errors associated with the figures, and also the connections between similar illnesses. A single lag was chosen for each category to reduce the number of degrees of freedom. If we chose the lags giving the
biggest influence on a case by case basis, then our results would contain anomalies caused by random patterns in the data set.

With a fixed lag chosen for each disease, the next step was to write a program (ResultsCalc, Appendix I) that varied disease categories and age categories. The smallest age distinctions we could make were the five-year categories our admissions data was stored in. By combining two or more of these smaller categories into one larger one we formed other age ranges. Many different ranges were used in the calculations, and Appendix II has a full table of the results. The next chapter focuses only on some of the more useful ranges.
6. Results

We now had, for each age and disease category, an expected percentage increase due to an interquartile $\text{PM}_{10}$ increase, and the associated 95% confidence errors. In Table 5 we only show these results for the age ranges:

- 0-14 (youth)
- 15-44 (adult)
- 65+ (elderly)
- Total (all ages)

<table>
<thead>
<tr>
<th>Disease Type</th>
<th>0-14</th>
<th>15-44</th>
<th>65+</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischaemic Heart Disease</td>
<td>$1.01 \pm 5.32$</td>
<td>$0.74 \pm 1.37$</td>
<td>$0.70 \pm 1.14$</td>
<td></td>
</tr>
<tr>
<td>Heart Failure</td>
<td>$-11.26 \pm 44.58$</td>
<td>$-6.97 \pm 21.59$</td>
<td>$3.09 \pm 2.00$</td>
<td>$3.05 \pm 1.89$</td>
</tr>
<tr>
<td>Dysrhythmias</td>
<td>$1.45 \pm 15.41$</td>
<td>$6.67 \pm 6.76$</td>
<td>$-0.56 \pm 3.00$</td>
<td>$1.08 \pm 2.32$</td>
</tr>
<tr>
<td>HEART TOTAL</td>
<td>$0.21 \pm 14.70$</td>
<td>$2.85 \pm 4.10$</td>
<td>$1.22 \pm 1.11$</td>
<td>$1.26 \pm 0.95$</td>
</tr>
<tr>
<td>Acute Respiratory Infections</td>
<td>$4.92 \pm 1.82$</td>
<td>$2.19 \pm 5.13$</td>
<td>$1.57 \pm 7.00$</td>
<td>$4.53 \pm 1.71$</td>
</tr>
<tr>
<td>Other Diseases of U. T.</td>
<td>$11.86 \pm 8.18$</td>
<td>$0.77 \pm 5.41$</td>
<td>$9.62 \pm 18.06$</td>
<td>$5.71 \pm 4.35$</td>
</tr>
<tr>
<td>Pneumonia / Influenza</td>
<td>$8.95 \pm 2.80$</td>
<td>$2.73 \pm 4.23$</td>
<td>$2.90 \pm 2.53$</td>
<td>$5.32 \pm 1.86$</td>
</tr>
<tr>
<td>Chronic Obstructive</td>
<td>$7.73 \pm 16.18$</td>
<td>$10.43 \pm 9.03$</td>
<td>$3.70 \pm 2.08$</td>
<td>$3.95 \pm 1.80$</td>
</tr>
<tr>
<td>Asthma</td>
<td>$0.58 \pm 1.77$</td>
<td>$3.26 \pm 2.67$</td>
<td>$1.62 \pm 4.76$</td>
<td>$1.86 \pm 1.38$</td>
</tr>
<tr>
<td>RESPIRATORY TOTAL</td>
<td>$3.62 \pm 1.28$</td>
<td>$2.54 \pm 1.96$</td>
<td>$2.86 \pm 1.63$</td>
<td>$3.37 \pm 1.03$</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>$1.17 \pm 4.86$</td>
<td>$0.52 \pm 2.79$</td>
<td>$-2.74 \pm 11.48$</td>
<td>$0.38 \pm 2.25$</td>
</tr>
</tbody>
</table>

*Table 5: Selected results - Percentage Increases and 95% Confidence Errors*
There is no value in the upper left cell because there were no youth admissions for ischaemic heart disease in our study period.

A result is considered to be significant if its 95% CI (obtained by adding and subtracting the 95% confidence errors to the values) does not contain zero. Most of the categories, particularly respiratory, show a significant positive increase. There are several negative values (indicating a decrease as pollution rises), but they are all very insignificant (the errors being much larger than the value itself).

The larger errors are due primarily to smaller sample sizes. This is especially noticeable in non-elderly heart cases, and in the less common ailments collectively known as other diseases of the upper tract. The totals (age, heart, and respiratory) all encompass more admissions, and thus have reduced errors.

Of most importance were the significant results for heart and respiratory totals over all ages. The increase in respiratory admissions from an interquartile rise in PM$_{10}$ was 3.37%, with a 95% CI of (2.35, 4.40). This increase would occur approximately two days after the PM$_{10}$ rise, once the symptoms had worsened sufficiently for the patient to be taken to the emergency department. The increase in cardiac admissions from an interquartile rise in PM$_{10}$ was 1.26%, 95% CI (0.31, 2.21). These values are both in keeping with the international literature.

Over all ages the greatest expected increases were found to be for other diseases of the respiratory tract 5.71% (1.36, 10.06), pneumonia/influenza 5.32% (3.46, 7.18), and acute respiratory infections 4.53% (2.82, 6.24), which all had the largest lag of 4 days. Insignificant results were found for ischaemic heart disease 0.70% (-0.44, 1.84), dysrhythmias 1.08% (-1.25, 3.40) and appendicitis 0.38% (-1.87, 2.63), although these still had positive increases. A greater sample size would be needed to clarify these.
The appendicitis results were encouraging. In all age ranges the results were highly insignificant (ie zero was near the centre of the 95% CI), which supported our assumption that it has no relationship with pollution. As this was our control data, it helps to confirm the validity of our methodology.

Many diseases showed different effects as different age groups were considered. Heart failure (3.09%) and COPD (3.70%) only showed statistically significant associations in older patients, while acute respiratory infections (4.92%) and other diseases of the respiratory tract (11.86%) were only significant in the young. Pneumonia/influenza was significant for the young (8.95%) and elderly (2.90%) age ranges, but not for adult admissions. Conversely, asthma (3.26%) was found to be only associated with PM$_{10}$ in the adult admissions.

As mentioned previously, the existence of significance is dependent on sufficiently large sample sizes as well as relationships between the admissions and the pollutants.

Due to the conservative nature of our approach, the above results that do show a relationship are likely to be less than their true value.
7. Financial Implications

The above results help to show the effect that pollution has on hospital admissions. We know that there are other indicators of the population’s health that could also be investigated for relationships with air pollution. Rather than emergency admissions, we could have examined numbers of deaths (from suitable causes), visits to the GP, sick days taken off from work, pharmacy sales of certain medicines, or even measurements of lung capacity or bloodstream pollutants in some sample. Each of these would be an entire project in itself, and most have already been studied, if not in Christchurch then elsewhere in the world.

One idea that did seem to be a reasonable extension to our work, however, was to look at the economic effect on the local hospitals. Our results have already found the expected increase in emergency hospital admissions due to air pollution. As air pollution levels rise we can thus estimate how many more admissions to expect and if they fall we can similarly estimate the decrease in admissions. Each patient would cost the hospital money and resources. The question is, how much is attributable to pollution, and so would be saved if pollution could be lowered? By applying this view to our data we hoped to find a different perspective to look at our results from.

Firstly we had to choose how much lower the pollution should be hypothesised to be. The Canterbury Regional Council has a target PM$_{10}$ threshold of 50 μg/m$^3$, which it aims to be the maximum average concentration for any 24-hour period. This is based on World Health Organisation guidelines, and seemed a sensible value to start with.

We next assumed that this target had been achieved. A new data set was created, based on the PM$_{10}$ values for the last ten years. All days which had a concentration higher than 50 μg/m$^3$
were reduced to 50. Any concentrations below 50 \( \mu g/\text{m}^3 \) were left unchanged. This was our imagined pollution situation.

Taking the difference between the actual and imaginary pollutant sets gave the assumed drop in PM\(_{10} \) for each day. On the days when the PM\(_{10} \) levels were assumed to drop there would also be an associated drop in hospital admissions. We calculated these expected reductions in admissions for each age, disease and day.

For each calculation we needed to know: the original pollution level for that day \( (P, \text{ in } \mu g/\text{m}^3) \); the number of admissions in the age/disease category for that day \( (A) \); and the increase in admissions per interquartile PM\(_{10} \) increase for that category \( (I, \text{ as a percentage}) \), which was already calculated.

On any day when \( P \) was below the threshold of 50, there was no expected change in pollution, and so no change in admissions. Otherwise, we expected there to be a drop of \( P - 50 \), which is

\[
\frac{P - 50}{14.8}
\]

when measured in interquartile ranges (the interquartile range for the PM\(_{10} \) data was 14.8 \( \mu g/\text{m}^3 \)).

For each interquartile drop in pollution there would be an \( I \) percent drop in admissions, so this would multiply \( A \) by

\[
1 - \frac{I}{100}
\]

This is for each interquartile drop, so the number of times we have to multiply it to \( A \) is the total number of interquartile drops, which is \( \frac{P - 50}{14.8} \). The expected number of admissions would therefore be

- 32 -
$$A \left(1 - \frac{I}{100}\right)^{\frac{P-50}{14.8}}$$

and so the expected drop in admissions would be

$$A - A \left(1 - \frac{I}{100}\right)^{\frac{P-50}{14.8}}.$$

These savings were summed over all days, age categories and disease categories. The resulting total was 533 prevented admissions, of which 97 were cardiac and 436 were respiratory.

To find the actual amount of money this would save, we obtained information from Crown Public Health Limited, consisting of the average costweight for each patient category. The costweight represents the total amount of money each patient costs the hospital, and one costweight corresponds to NZ$2,500. By multiplying the average costweights by the size of each category, and then adding them, we got the total costweight saved to be NZ$1,025,000.

We repeated the same approach with PM$_{10}$ further reduced to 30 µg/m$^3$ instead of 50µg/m$^3$, and found that 948 admissions would be prevented, saving NZ$1,733,000. With PM$_{10}$ reduced to 25 µg/m$^3$, there would be 1127 admissions and NZ$2,035,00 saved.

These figures give a rough indication of the financial implications of pollution. The 7-digit amounts suggest that it is not trivial. And as well as the expense of hospital care, there would be other costs to the economy such as lost work hours. This money, or a portion of it, could instead be better spent converting homes to efficient heating, or some other initiative to reduce Christchurch’s air pollution. Either way high pollution appears to be costly to the health sector.
8. Conclusion

The information we were provided with to use was not ideal. As already mentioned, the meteorological data had missing values, which had to be recalculated. But aside from that, the information we had available to us was not as useful as we would have liked.

- The temperature values were 24-hour averages. From them we could not estimate how cold the nights were, or how warm the days. This information would have been helpful for several reasons, as pollution levels will only relate to night-time temperatures, and also immune systems become most susceptible during big temperature changes.

- The pollution monitoring program is still in its initial stages. In the future we hope that readings can be collected from more urban centres around Christchurch. This will assist in building a general outlook of the city’s pollution, which in turn would enable us to isolate those areas with higher pollution. We could then obtain our admissions data with location included, and correlate the admissions with the polluted regions more closely.

- Finally, the wind data as a 24-hour average is also unusable. Only the evening wind speed is pertinent, and this may be unrelated to the average if the wind conditions change during the day. The use of 10-minute measurements was looked into, but because wind doesn’t directly influence health, only pollution levels, this data would only be useful if a more complicated air pollution model was desired. Hourly (or more frequent) wind data and temperature data coupled together would also give information on depths of inversion layers, which in turn have a big effect on pollutant concentrations.
One last improvement would be to increase the sample sizes involved (Dawson et al, 1983). In several years time our approach could be repeated to give more accurate values, particularly to those groups with large errors.

None of these issues is particularly major, however, and are more extensions than required improvements. The project had many strengths too.

The disease classification system at the Christchurch hospitals is closely monitored, and is known to have very few misdiagnoses. Even if there were errors in the data, they would not be correlated with pollution levels at all (Delfino et al, 1993).

The meteorological variables, which were possible confounders in our study (Lipfert, 1993), were collected from the same site as the pollution data, as well as being available in ample amounts. This enabled us to extract any of the confounding effects without hassle.

The concentrations of particulate matter in Christchurch are known to be fairly evenly spread over the city (CRC, 1996). This reduces any need for location to be included as a factor. Also the use of 24-hour averages increases the likelihood of getting a true city-wide value.

The main conclusion to be drawn is that the air pollution in Christchurch does have a measurable effect on the health of the general population. As Chapter 6 showed, a wide range of cardiac and respiratory diseases are affected. In some cases, the effects were only noticeable for specific age categories, in others all ages were affected.

These findings compare strongly with the international literature (Ponka, 1996; Schwartz, 1997; Burnett, 1994; Schwartz, 1995; Walters, 1994; Delfino, 1994; Thurston, 1992). Many similar studies have been done in cities around the world (Schwartz, 1997; Gold et al, 1999; Gordian et al, 1996), and in all cases small, but significant, associations were found. This project was funded to discover if such results applied to Christchurch also, and this has been confirmed.
Acknowledgements:

This project was undertaken with funding from the Health Research Council of New Zealand. The Canterbury Regional Council assisted with data acquisition. Many thanks to my supervisors Easaw Chacko and Phil Hider, who were fully involved with the whole project, to the three Ians (McKendry, Town, and Westbrooke) for their advice and assistance, and to my family, friends, and Rebecca, who put up with me during the busier times.
Appendices

Appendix I – S-Plus Program Listings

These are the S-Plus programs written to perform the analysis.

```
# LagsCalc calculated Table 4. It varies the lags and #
# diseases, but keeps the age range as the total.

LagsCalc<-function()
{
  attach(Hosp)
  for (pmlag in 0:6)
    for (k in 19:19)
      for (j in 1:11)
        {
          d<-disnames[j,1]
          a2<-agenames[k,2]
          a3<-agenames[k,3]
          lagincs[j,pmlag]<-p2(d,a2,a3,pmlag)
          lagincses[j,pmlag]<-se
          cat("Increase in ",d," is ",format(lagincs[j,pmlag],nsmall=1,digits=4)," 
          ",format(se,nsmall=1,digits=4),"\n")
        }
}
```
# ResultsCalc calculated the expected increase for given ages
# and diseases, and used the lag previously calculated by
# LagsCalc (0, 2, or 4 days), to give a result.
# Its current settings would create Table 5.

ResultsCalc<-function()
{
attach(Hosp)
for (k in c(19,21,24,34))
for (j in 1:11)
{
  d<-disnames[j,1]
pmlag<-disnames[j,2]
a2<-agenames[k,2]
a3<-agenames[k,3]
newincs[j,k]<-p2(d,a2,a3,pmlag)
newincses[j,k]<-se
cat("Increase in
",d,"is",format(newincs[j,k],nsmall=1,digits=4),"% +-
",format(se,nsmall=1,digits=4),"\n")
}
}
# p2 is used by the other two, and includes the regression calculations which do all the work. As input it requires a disease category, youngest and oldest ages to consider, and the lag for the PM10.

p2<-function(dis="RT",agea=21,agez=21,pmlag=0)
{
  tm <- rowSums(Hosp[Hosp$Type==dis,agea:agez])
  tm <- remove.row(target = tm, start.row = 1, count = 4)
  tm2 <- cbind(weat,tm)
  tm3 <- tm3dummy
  tm3$V7L1 <- V7L7((7-pmlag):(3872-pmlag))
  tm4 <- tm4dummy
  tm4$V7L1 <- quantile(tm3$V7L1,c(.25,.75))
  last.gam <- menuGam(formula = X2 ~ Temp1 + Temp10 + RH,
                      family = poisson, link = log, variance = "constant", data = tm2, na.omit.p = T, trace = F, maxit = 50, epsilon = 0.001,
                      bf.maxit = 10, bf.epsilon = 0.001, print.short.p = F,
                      print.long.p = F, save.name = "tm3", save.fit.p = F,
                      save.resid.working.p = F, save.resid.pearson.p = F,
                      save.resid.deviance.p = F, save.resid.response.p = T,
                      plotResidVsFit.p = F, plotSqrtAbsResid.p = F,
                      plotResponseVsFit.p = F, plotQQ.p = F, smooths.p = F,
                      rugplot.p = F, id.n = 3, plotPartialResid.p = F,
                      plotPartialFit.p = F, rugplotPartialResid.p = F,
                      scalePartialResid.p = T, predict.type = "response", predict.p = F, se.p = F)

  last.lm <- menuLm(formula = resid.response~V7L1, data = tm3,
                 na.omit.p = T, print.short.p = F, print.long.p = F,
                 print.anova.p = F, print.correlation.p = F, save.fit.p = F,

se <<- (tm4[2,1] -
  tm4[1,1])*196/mean(tm2[,5])*summary.lm(last.lm,F)["coefficients"][2,2]

return(100*(tm4[2,2]-tm4[1,2])/mean(tm2[,5]))
}

- 40 -
Appendix II – Full Results Table

Percentage increases in admissions from an interquartile increase in PM$_{10}$:

<table>
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<tr>
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<th>35-39</th>
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95% confidence errors for above increases: (add or subtract to get the 95% confidence limits)

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Percentage increases in admissions from an interquartile increase in PM$_{10}$:

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95% confidence errors for above increases: (add or subtract to get the 95% confidence limits)

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Percentage increases in admissions from an interquartile increase in PM$_{10}$:

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95% confidence errors for above increases: (add or subtract to get the 95% confidence limits)

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Percentage increases in admissions from an interquartile increase in PM$_{10}$:

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95% confidence errors for above increases: (add or subtract to get the 95% confidence limits)

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Appendix III – Journal Article

Particulate Air Pollution and Hospital Admissions In Christchurch

Abstract

J. McGowan*, P. Hider*, E. Chacko*, G. I. Town*
Department of Mathematics & Statistics*, University of Canterbury; New Zealand Health Technology Assessment* and Department of Medicine*, Christchurch School of Medicine, Christchurch, New Zealand

Aims: Winter air pollution in Christchurch is dominated by particulate from solid fuel domestic heating. The aim of the study was to explore the relationship between particulate air pollution and admissions to hospital with cardiorespiratory illnesses.

Methods: Air pollution data (PM$_{10}$) was obtained from the Canterbury Regional Council monitoring station in St Albans. The New Zealand Health Information Service provided data on admissions to the Princess Margaret and Christchurch Hospitals for the period June 1988 through December 1998 for both adults and children with cardiac and respiratory disorders. The relationship between PM$_{10}$ and admissions was explored using a time series analysis approach controlling for weather variables. Missing data was interpolated from carbon monoxide values for the same time period, which showed a close relationship with PM$_{10}$.

Results: There was a significant association between PM$_{10}$ levels and cardiorespiratory admissions. For children and adults combined there was a 3.37% increase in respiratory admissions for each interquartile rise in PM$_{10}$ (interquartile value 14.8 mcg/m$^3$). In adults there was a 1.26% rise in cardiac admissions for each interquartile rise in PM$_{10}$. There was no relationship between PM$_{10}$ and admissions for appendicitis, the control condition selected.

Conclusions: In keeping with international literature, there is evidence in Christchurch of a relationship between particulate levels and admissions with cardiac and respiratory illnesses. The size of the effect is consistent with overseas data with the greatest impact seen on the respiratory system.

Implications: A reduction in ambient particulate pollution levels in Christchurch would significantly reduce hospital admissions for cardiorespiratory conditions.
Aim

To assess the relationship between the daily number of people admitted to Christchurch Hospitals with respiratory or cardiovascular illness and particulate pollution during 1988-1998.

Introduction

High levels of air pollution have been associated with short-term adverse effects on human health in a number of European and North American cities. (Schwartz, 1996; Schwartz and Dockery, 1992; Katsouyanni et al., 1995). The adverse effects have largely been associated with exposure to elevated levels of the particulate component of air pollution (Seaton et al., 1995; Pope et al., 1995) and specifically the small sub ten micron sized particles (PM$_{10}$).

Adverse health effects from PM$_{10}$ exposure have now been documented to be associated with rises in local mortality rates, hospital admissions, emergency department visits, as well as increases in the frequency of symptoms and medication use and with deteriorations in various physiological variables (Committee on the Medical Effects of Air Pollution, 1995). The effects from exposure to high concentrations of inhaled pollutants have largely impacted on respiratory (Schwartz, 1993; Spix et al., 1993) and cardiovascular systems (Pope et al., 1992; Lipfert and Wyngaard, 1995) and have also recently been found from exposures to even relatively moderate air pollution levels.

To date, there has been relatively little New Zealand based research examining the relationship between external air pollution and either the morbidity or mortality related to respiratory or cardiovascular illnesses (Crane, 1996). This lack of research is surprising as parts of New Zealand have clearly been documented to have a significant air pollution problem (most notably the Christchurch urban area) with ambient levels of particulate that sometimes exceed those of major North American or European cities (Public Health Commission, 1995) and regularly are higher than World Health Organisation Environment Guidelines (Canterbury Regional Council, 1993). In addition, the prevalence of respiratory (especially asthma) and cardiovascular illnesses have also been noted to be high in this country (Crane et al., 1994; PHC, 1993).

Unfortunately the few studies that have been conducted in this country have had significant methodological weaknesses; such as a reliance on extrapolated data from overseas work (Foster, 1996) or low power resulting from a small sample size (Dawson et al., 1983).

Christchurch is an ideal site for an investigation of the relationship between outdoor air quality and respiratory/cardiovascular illness as it has a centralised hospital serving the entire city catchment with a computerised patient information system and there is a well-established local system for gathering data on pollution levels.

Uniquely in Christchurch most particulate air pollution (about 90%) comes from the city’s 47,000 wood burners and open fires that are regularly used during the cold winters (NIWA, 1996). Overseas studies have found that the smoke generated by these sources is associated with the most deleterious effects upon the respiratory and cardiovascular health of local inhabitants (Schwartz et al., 1993). Furthermore the local weather patterns that typically occur during Christchurch winters further compound its pollution problem. An inversion layer frequently occurs during colder months which then traps warmer air along with large amounts of airborne particulate matter at low altitude above the city (NIWA, 1996). Local studies suggest that relatively even mixing of the ambient particulate matter occurs across the Christchurch air-shed in a 24 hour period ensuring that there is a generally homogeneous exposure to similar PM$_{10}$ levels for all city residents (Canterbury Regional Council, 1996).

Finally, reliable information on important potential confounders (meteorological variables and epidemics of influenza (Lipfert, 1993; Schwartz, 1996; Vedal, 1997) are readily available in this city.
Method

Study area

Christchurch City (population 333,000) is located on the Canterbury Plains which are bordered by the Pacific Ocean to the East and the Southern Alps to the West.

Hospital admission data

Admission data was obtained from New Zealand Health Information Service on daily counts of all acute admissions with the following primary diagnoses based on the International Classification of Diseases, 9th Revision, (ICD 9) classifications: pneumonia (ICD 480-487), chronic obstructive pulmonary disease (491-492, 494-496), asthma 493, ischaemic heart disease (410-414), dysrhythmia (427), and heart failure (428). Information on appendicitis admissions was also included as a control group. Admission data was obtained for both Christchurch Hospital and Princess Margaret Hospital between June 1988 to December, 1998 and aggregated into a single dataset.

Air pollution and meteorology data

Pollution data was obtained from the Canterbury Regional Council monitoring site in the central Christchurch suburb of St Albans for the period June 1988- December 1998. Data was provided in the form of 24 hour averages on the concentrations of the following pollutants: carbon monoxide (CO), particulate (PM$_{10}$), sulphur dioxide (SO$_2$), and oxides of nitrogen (NO$_x$). Missing values for the 20% of days without particulate pollution data were imputed from information on other pollutants. High correlations existed between PM$_{10}$ and these other pollutants (especially CO). A regression was performed on days when values for both PM$_{10}$ and CO existed and the relationship between the pollutants enabled the determination of the missing PM$_{10}$ data. A similar approach was used with the other pollutants (NO$_x$ and SO$_2$) to fill in some remaining gaps. Data for a small period (<4% of the total) in the summer of 1993/4 was determined by the use of a moving average based on the other relatively constant summer time levels. Measurements of PM$_{10}$ were mainly by TEOM, and were subject to regular quality audit by the CRC.

Meteorological data including wind speed, relative humidity, and temperatures at 1m and 10m above ground level were also obtained from this site as 24-hour averages.

Consent was obtained from the relevant hospitals, the Canterbury Regional Council and the Canterbury Ethics Committee.

Statistical methods

The analysis was undertaken in two stages. Firstly the influence of the meteorological variables on hospital admissions was determined and then the connection between PM$_{10}$ and the residuals was modelled. This approach was considered conservative, and expected to give a minimum value for the pollution's contribution to hospital admissions.

A generalised additive model with a Poisson link was used for the first step because although admission data was count data (the number of admissions on any day is a whole number of people), the mean number of admissions varies over time so that their distribution was not strictly Poisson. The generalised additive model is an analytical methodology well suited to this type of research (Hastie and Tibshirani, 1990; Chambers et al., 1983) and one that has been extensively used by other researchers (Schwartz,1994; Schwartz,1994; Schwartz,1996).
The equation for the model was:

$$\log[E(Y)] = \sum_i S_i(X_i)$$

$X_i$ = predictor meteorological variables (two temperatures and humidity)

$Y$ = either all admissions, or a specific disease.

$S_i$ are smooth functions

This model was used to assess the effect of weather conditions on admissions. The number of admissions predicted by the model were subtracted from the actual known admissions, to leave the residual admissions, which were unrelated to the meteorological variables.

A linear regression model was used for the second stage to compare the admission residuals to the PM$_{10}$ concentration. Because PM$_{10}$ is significantly higher during the night-time, admissions were compared on one day with the pollution from the previous night (calculated from 9am on the previous day to 9am on the same day). This gave a line of best fit, from which the expected increase in admissions were calculated due to a given increase in PM$_{10}$.

For each condition, as well as total respiratory and cardiac conditions an expected percentage increase (or decrease) in admissions for an inter-quartile increase of PM$_{10}$ was calculated according to patients in 5 year age groups. Different lags (from 1-7 days) for the pollution data were investigated to assess the delayed effects of particulate exposure.

The study was calculated to have sufficient sample size to detect a 1% increase in cardiac admissions for people aged over 64 years, and for respiratory admissions for children less than 6 years, in relation to a 10 mcg/m$^3$ rise in particulate with 80% power (alpha = 0.05). These calculations do not include the provision for a threshold, which is consistent with other previous studies (Committee on the Medical Effects of Air pollution,1995; Schwartz,1994; Ostro,1984).

Results

Table 1 presents summary statistics for the daily measurements of pollutants, meteorological variables, and cardiac/respiratory admissions for all age groups in Christchurch between 1988-1999. During the study period the interquartile range for PM$_{10}$ recordings was 14.8 mcg/m$^3$ while the average daily total number of respiratory or cardiac admissions was 10.2 and 6.8 respectively.

Over the study period the average daily hospital admissions for respiratory conditions rose (insert graph of daily resp admissions over 10 years). Weather, pollution, and hospital admission (for most conditions) data all exhibited a cyclical pattern with peak values generally being recorded in winter.

A significant association was found between particulate pollution and admissions for both total cardiac and respiratory conditions. The increase in respiratory admissions in relation to an increase in PM$_{10}$ was higher for respiratory conditions. The increase in respiratory admissions from an interquartile rise in PM$_{10}$ was 3.37% (95% C.I. 2.34% - 4.40%) compared to cardiac conditions (increase in admissions was 1.26%, 95% C.I.0.31% - 2.21%) (Insert table of results for age groups and conditions). For cardiac conditions the associations were strongest when there was no lag in the pollution data. For respiratory admissions there was a lag of two days for most categories, and up to four days for respiratory infections.

The highest increase in admissions for an inter-quartile rise in PM$_{10}$ was for respiratory admissions due to other diseases of the upper respiratory tract (5.71%, 1.36 – 10.06) pneumonia/influenza (5.32%, 3.46 – 6.39), acute respiratory infections (4.53%, 2.82 – 6.24) and COAD (3.95%, 3.15 – 5.75) (Table 2). By contrast, there was no significant increase in admissions for appendicitis, the control condition, (0.38%, 95% C.I. = -2.63 - 1.87%), ischaemic heart disease (0.70%, -0.44 - 1.84), dysrhythmias (1.08%, -1.24 - 3.4), (Table 2).
The relationship between hospital admissions and PM$_{10}$ varied between different age groups for each of the conditions. Admissions for heart failure (3.09%, 1.09 – 5.09) and total cardiac admissions (1.22%, 0.11 – 2.33) only exhibited statistically significant associations with PM$_{10}$ for the group aged over 65 years (Table 2). By contrast, hospitalisations for several respiratory conditions including acute respiratory infections and other diseases of the upper tract were significantly associated with PM$_{10}$ for children but not older patients (Table 2). Admissions for asthma and chronic obstructive airways disease exhibited a different pattern; adults admissions were significantly associated with PM$_{10}$ while the relationship between paediatric and elderly patients was smaller and not statistically significant (Table 2). There was no statistically significant relationship between PM$_{10}$ and appendicitis admissions for any age group.

Discussion

The main finding of this study that small sized particulate air pollution is associated with a significant rise in hospital admissions for total respiratory (especially) and all cardiac conditions is consistent with previous research conducted in several North American and European cities. The size of the effect on admissions in Christchurch is also comparable with previous research that has also examined the relationship between all respiratory or cardiac conditions and PM$_{10}$ (Thurston, 1992; Dab, 1996; Delfino 1994; Walters 1994, Schwartz, 1995; Schwartz 1996; Schwartz 1994; Burnett, 1994; Schwartz, 1997; Ponka, 1996).

The results from age and diagnostic subgroup analyses for respiratory admissions also presented many consistent findings with other previous research. The increase in admissions for pneumonia and COAD among the elderly was similar to the rise found in work by Schwartz (in three previous studies published in 1994, 49, 53, 55) and Moolgavkar, (1997). However, in contrast to (Pope, 1991) there was not a statistically significant relationship between paediatric (unlike elderly) asthma admissions and PM$_{10}$.

The results of age and diagnostic sub-group analyses for admissions in relation to the cardiac conditions were also generally consistent with the findings from previous research. Schwartz (1997, 1999) similarly reported a 1-1.5% increase in admissions for ischaemic heart disease among the elderly in relation to a 15 mcg/m3 rise in particulate, however, unlike these two North American-based studies the association in Christchurch did not achieve statistical significance. This absence of a statistically significant relationship between admissions for ischaemic heart disease and PM$_{10}$ among elderly patients may however be due to an inadequate sample size in this study. While this study had sufficient statistical power to find an association between admissions for all cardiac conditions and PM$_{10}$ among people >65 the number of cardiac admissions over the study period may have been too small to find any significant relationship between individual cardiac conditions and PM$_{10}$ if it existed.

A possible limitation for this study is the absence of any data on ozone exposure. Several studies have found a clear association between ozone levels and admission rates for respiratory illnesses (e.g. Thurston et al, 1994; Schwartz, 1994; Pope, 1991). However, it is notable that this relationship has principally been found in conjunction with summertime, photo-chemical smog and not particulate pollution (Sunyer et al, 1991; Sunyer et al, 1993; Schwartz, 1994). Studies that have concurrently examined the effect of particulates and ozone have typically reported that the effect of each is separate (Schwartz, 1996; Thurston, 1992; Committee on the Medical Effects of Air Pollution, 1995). Christchurch is considered unlikely to experience significant effects from ozone, especially during the winter when particulate levels are highest (NIWA, 1996).

Despite the possible limitations from the lack of data about ozone levels and a small sample size for sub group analyses this study design has a number of important strengths in relation to its ability to minimise bias. The major issues in evaluating the association between particulate pollution and morbidity are the adequacy of covariate control, and the relative impact of measurement error in the assessment of exposure and outcome variables. These factors have been carefully addressed by time series studies. A time series study only needs to control for confounders that might vary on a day to day basis in coincidence with air pollution. Reliable information was available throughout this study to describe the meteorological conditions, which were the most important potential confounders for the study, from the same site used to collect the information about pollutants.
While it is accepted that misdiagnosis or misclassification of diagnoses may occur in this study it is unlikely that coding staff will misclassify patients in a systematic way that correlates with daily air pollution levels. The use of discharge, rather than admission, diagnoses further strengthens this expectation. Furthermore, the use of broad groupings of ICD codes would help to reduce errors due to misdiagnosis. Delfino et al (1993) found that the accuracy of discharge diagnoses were 90% when closely related diagnostic categories were grouped in this type of study.

The relatively homogenous exposure of Christchurch residents to pollution and the relatively minor contribution to pollution from non-particulate matter reduces the potential for the study to misclassify exposure (Vedal, 1997). Although exposure is measured at set locations, the Christchurch air space has been found to have a relatively even mixing of particulate matter. The use of 24 hour averages also increases the likelihood that a true catchment-wide measurement of exposure was obtained. A major issue remains about whether outdoor measurements can function as a proxy for personal exposure given that people spend considerable amount of time indoors and are sometimes exposed to particulate from cigarette or cooking smoke. However, the ability of small diameter particulate matter to freely penetrate walls and thereby rapidly equilibrate between indoor/outdoor environments has been previously described (Wallace et al, 1987; Schwartz and Morris, 1995). Several studies (Suh et al, 1994; Brauer et al, 1989; Thomas et al, 1993; Anuszewski et al, 1996) have found that there is a high correlation between environmental PM_{10} recordings and personal exposure to particulate however this has yet to be adequately examined especially in the New Zealand setting. In the meantime it is notable that almost all the previous studies that have assessed the relationship between particulate pollution and hospital admissions have used daily measurements of ambient concentrations of particulate from fixed point monitors rather than measurement of personal exposures (Committee on the Medical Effects of Air pollutants, 1996). In large population-based studies it is impractical to attempt to measure personal exposures. Furthermore, in practice, policy related issues such as the compliance with standards, or threshold levels for the provision of advice to the public, are based on ambient levels of pollution and not on personal exposure.

Finally, this study addressed the relationship between particulate and hospital admissions and did not consider the role of other pollutants. Although SO_{2} and NO_{2} have been shown to be related to hospital admissions, the relationship is neither as consistent, nor as strong, as that with particulate suggesting that these oxides may be exerting an effect mainly through confounding with airborne particulate (Schwartz, 19995; Pope et al, 19991; Committee on the Medical Effects of Air pollution, 1995).

In conclusion, this study adds to the accumulating evidence that particulate air pollution causes significant morbidity in the community. A reduction in ambient particulate pollution levels can therefore be expected to generate considerable public health benefits. In Christchurch city, for example, if PM_{10} was rigorously maintained below a level of 50ug/m3 then the findings from this study predict that nearly 40 admissions for cardio-respiratory conditions per year would be avoided.

Acknowledgements

The following support for this study is gratefully acknowledged. Major funding was provided by a grant (98/) from the Health Research Council of New Zealand. Assistance with obtaining data was provided by the Canterbury Regional Council. A grant-in-aid was received from Crown Public Health Limited.
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