There are now numerous studies reporting significant relationships between air pollution and mortality (Dockery, Schwartz & Spengler 1992; Dockery et al. 1993; Health Effects Institute 2000; Katsouyanni et al. 1997; Laden et al. 2000; Pope, Hill & Villegas 1999; Samet et al. 2000; Schwartz 1993). The research demonstrates clear associations between mortality for a variety of causes and levels of air pollution (Mar et al. 2000; Ostro et al. 1996; Pope et al. 2002; Ponka, Savela & Virtanen 1998; Sunyer et al. 1996; Wong et al. 2001). In Australia, several studies using time series analysis have been conducted to investigate the relationship between daily ambient air quality and health outcomes including mortality. Studies conducted in Sydney and
Melbourne have shown that increases in air pollution levels are associated with increased risks of daily mortality (Environmental Protection Authority 2000; Morgan et al. 1998). A positive association was observed between ozone and particulate matter (PM) and daily mortality for a study in Brisbane where the results were significant for those older than 65 years (Simpson et al. 1997).

Air Quality in Perth is monitored via a network of 13 ambient air quality stations with different parameters monitored at each station. Perth air quality is considered unsatisfactory for relatively short periods of time each year. The levels of photochemical smog (ozone) in Perth during summer regularly exceed national standards, and, during the winter months, particles are relatively high (Department of Environmental Protection [DEP] 2000). The episodes of unsatisfactory air quality are influenced by weather conditions that prevent air pollutants from dispersing rapidly. By contrast, concentrations of SO2, NO2 and CO are all below national and international standards in Perth with elevated levels being observed in specific areas impacted by point source emissions (DEP 2001).

Figure 1: Map of air quality monitoring sites in Perth as at 1/1/2000

Given the results of national and international studies relating air pollution and health outcomes, it was necessary to establish whether such associations are observed in Perth where there is a different pollutant mix and meteorology and where measures are required to reduce air pollutants to acceptable levels. This study used 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. This design samples only cases with exposures for each subject during a designated period before the disease or hazard event and compares the distribution of exposures during a reference period.

The reference exposures should be representative of the expected distribution of exposures for follow-up times that do not result in a case (Levy et al. 2001). The study design may have advantages in controlling for time trends and seasonality compared to traditional time series analysis and controls for all measured and unmeasured variables which do not vary with time (Bateson & Schwartz 2001). It does not require modelling of all variables and all season and trend effects are removed. Factors such as age and sex can be estimated more easily as the event is the unit of analysis. The case crossover conditional logistic regression analysis is considered to be more robust and less prone to bias and confounding than the more standard time series regression analysis, however, bias may be introduced when the exposure in the reference periods is not equally representative of exposure in the hazard periods (Bateson & Schwartz 2001).

This study investigated the relationship between changes in daily ambient air pollutant concentrations and daily mortality, cardiovascular mortality, and respiratory mortality in the Perth metropolitan area between 1992 and 1998 using a time stratified design, with three or four control cases in the same month as the hazard.
Method

Study area and population
The study area encompassed the metropolitan Perth region according to the Australian Bureau of Statistics (ABS) Statistical Division.

There are several pockets of high industrial activity, the population is spread out and 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 subject to consistent wind from the Indian Ocean where the nearest land is thousands of kilometres away and offshore winds are from regions of very low population density.

Mortality data
Mortality data were made available by the Registrar General of Western Australia from the period 1 January 1992 to 31 December 1997. There are approximately 12,000 deaths recorded annually in Perth. Causes of death codes are made available on an annual basis by date of registration. Deaths registered in 1998 were checked for the date of death in case any 1997 deaths had been missed. Three groups of causes of death using the International Classification of Diseases version 9 (ICD-9) were analysed separately: cardiovascular (ICD9 codes 390 to 459), respiratory (ICD9 codes 460 to 529), and 'other causes' excluding accidents, poisoning, violence, cardiovascular and respiratory causes.

Air quality data
Daily ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), sulphur dioxide (SO_2) and particle concentrations measured by nephelometry (Bsp) have been measured since 1990. PM_{10} has been monitored since 1994 using a tapered element oscillating microbalance (TEOM). PM_{10} was collected every 6 days by high volume sampler (HiVol) and was excluded for analysis.

The most complete data set with the highest number of days with co-located instruments were from the Caversham, Swanbourne and Queens Building sites and these were used to generate the daily air quality data for the pollutants investigated, with the exception of PM_{2.5} which is discussed below. 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.

Data aggregation
Data from Caversham, Queens Building and Swanbourne monitoring stations were aggregated in this study. The aggregate data were then compared with all available data across the network.

The averages for each pollutant and each method of generating a daily estimate were comparable with no statistically significant differences, indicating the use of the three sites was acceptable as a surrogate for data across the entire network.

The averaging times selected were based on the Australian National Environmental Protection Measure Ambient Air standards and were used for each pollutant in subsequent analyses. 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.

Dispersion modelling was utilised to develop daily average concentration of PM_{2.5} due to the limited number of particulate monitoring sites and the large variations in population density and subsequent particle concentrations. 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 (Clench-Aas et al. 1999; Micallef & Colls 1999; Vinitketkumnuen et al. 2002).

Despite the inherent limitations in this approach (related to the limited knowledge of meteorological conditions and of the actual particulate emissions), it was considered that the modelled concentration distribution, scaled to match available measurements, would form a better basis for exposure estimation. The modelling work was conducted using a Gaussian 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, providing for each day 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 enhance further the accuracy of exposure 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:

$$[\text{PM}_{2.5}] = 17.86 \ B_{sp} + 3.69 \ \mu g/m^3$$

With this relationship, it was possible to use $B_{sp}$ measurements to enhance the data set used in interpolations. Figure 2 shows the interpolated and modelled PM$_{2.5}$ concentration contours for Perth.

**Figure 2: Selected interpolated and modelled PM$_{2.5}$ concentration contours for Perth**

Interpolated average PM$_{2.5}$ concentrations ($\mu g/m^3$), for the period June-July 1997

Average PM$_{2.5}$ concentrations ($\mu g/m^3$) for the Perth region, modelled for the period June-July 1997, and adjusted to match measured and estimated PM$_{2.5}$ values at all monitoring sites.
Statistical analysis

This study used a time stratified case crossover design. Individual deaths were considered and the cases were matched to exposure levels during referent days falling on the same day of the week in the same month as the day of mortality. This provided three or four control days depending on the number of days and cases in each month. Comparisons between cases and controls were then made using conditional logistic regression to produce odds ratios (Bateson & Schwartz 1999). The analysis was conducted using Proc PHREG in SAS V8 (SAS Institute Inc., Cary, NC). Before examining the effect of pollutants, temperature and humidity were examined. Variables indicating public holidays and day of the week were included. However, only the most significant effects were included in the pollutant models. Each pollutant was entered into its own predictive model for seven different lag periods, (0,1,2,3,0-1, 0-2, 0-3) days, and four different averaging periods (1hr, 4hr, 8hr, 24hr) where data were available. Each category of mortality data was analysed in relation to NO₂, O₃, particulate (PM₂.₅ modelled and Bsp) and CO concentrations. 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 occurred randomly, as was the case in this data set, ignoring the data was not likely to introduce selection bias.

Results

From 1992 to 1998 the average number of daily deaths was 26.8 with an average of between 2.1 and 2.4 deaths attributable to respiratory disease (Table 1). By comparison 11.6 deaths were attributed to cardiovascular disease. Most of these deaths occurred in the age group greater than 65 years. Daily pollutant concentrations and meteorological parameter measures for the period are presented in Table 2. The association

| Table 1: Daily number of deaths between 1992 to 1998 |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                                 | 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           | 18.8  | 4.7 | 13           | 25           | 22.4  | 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 years           | 8.0   | 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 years   | 1.5   | 1.3 | 0            | 3            | 2.6   | 1.9 | 0            | 5            | 2.1   | 1.7 | 0            | 4            |

| Table 2: Air pollution concentrations and meteorological parameters |
|-------------------------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                                 | November-April  | May-October     | All year        |
|                                 | Mean  | SD  | 10th centile | 90th centile | Mean  | SD  | 10th centile | 90th centile | Mean  | SD  | 10th centile | 90th centile |
| NO₂ - 24h ave (ppb)              | 9.6   | 4.0 | 4.3           | 15.7          | 11.1  | 5.1 | 4.8           | 18.0          | 10.3  | 5.0 | 4.4           | 17.1          |
| NO₃ - 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          |
| O₃ - 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          |
| O₃ - 4h max (ppb)                | 31.1  | 9.4 | 20.8          | 45.0          | 26.6  | 5.0 | 21.1          | 32.3          | 28.8  | 7.8 | 21.0          | 39.5          |
| O₃ - 1h max (ppb)                | 35.0  | 12.4| 22.1          | 53.7          | 28.3  | 5.6 | 22.4          | 34.0          | 31.6  | 10.2| 23.2          | 46.1          |
| Bsp - 24h ave bscat/10⁴          | 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/10⁴)         | 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           |
| **PM₁₀ - 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          |
| **PM₂.₅ - 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               | 21.8  | 3.4 | 17.8          | 26.7          | 15.3  | 2.5 | 12.0          | 18.2          | 18.4  | 4.5 | 13.0          | 24.7          |
| Humidity - 24h                  | 54.2  | 15.3| 35.5          | 75.5          | 71.4  | 12.2| 53.5          | 85.0          | 64.0  | 15.0| 40.5          | 82.5          |

* Available only weekly
** Modeled PM2.5 data
between air pollutant concentrations, seasonality and meteorological parameters is shown in Table 3. As expected, significant correlations between $O_3$ and particulates and temperature, as well as CO concentrations and NO$_2$ and particulates were found.

The resultant odds ratio and 95% confidence intervals (CI) were plotted for particulate ($B_{sp}$), NO$_2$, $O_3$ and CO concentrations and each mortality category (Figures 3 to 6). The odds ratios and 95% CI for each mortality category and lag for 24hr averaging periods for particulates ($B_{sp}$), NO$_2$ and modeled PM$_{2.5}$ is shown in Table 4. The results of analysis showed no significant associations between changes in daily particle concentrations as measured by nephelometry ($B_{sp}$) (1hr and 24hr) and respiratory mortality, cardiovascular diseases (CVD) mortality or ‘other’ mortality (Figure 3, Table 4). Small but significantly elevated

### Table 3: Correlation coefficients between pollution measures

<table>
<thead>
<tr>
<th></th>
<th>NO$_2$ 24-hr</th>
<th>$O_3$ 8-hr</th>
<th>$B_{sp}$ CO 24-hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>$O_3$</td>
<td>-0.04</td>
<td>0.35</td>
<td>-0.11</td>
</tr>
<tr>
<td>Warm</td>
<td>0.35</td>
<td>-0.11</td>
<td>0.35</td>
</tr>
<tr>
<td>Cool</td>
<td>-0.11</td>
<td>0.35</td>
<td>0.35</td>
</tr>
<tr>
<td>$B_{sp}$</td>
<td>0.19</td>
<td>0.01</td>
<td>0.42</td>
</tr>
<tr>
<td>Warm</td>
<td>0.26</td>
<td>0.36</td>
<td>0.42</td>
</tr>
<tr>
<td>Cool</td>
<td>0.42</td>
<td>-0.02</td>
<td>0.36</td>
</tr>
<tr>
<td>CO</td>
<td>0.57</td>
<td>0.00</td>
<td>0.35</td>
</tr>
<tr>
<td>Warm</td>
<td>0.57</td>
<td>0.16</td>
<td>0.24</td>
</tr>
<tr>
<td>Cool</td>
<td>0.55</td>
<td>0.00</td>
<td>0.37</td>
</tr>
<tr>
<td>Temp</td>
<td>-0.12</td>
<td>0.20</td>
<td>-0.27</td>
</tr>
<tr>
<td>Warm</td>
<td>0.22</td>
<td>0.51</td>
<td>0.04</td>
</tr>
<tr>
<td>Cool</td>
<td>-0.19</td>
<td>0.04</td>
<td>-0.31</td>
</tr>
<tr>
<td>Humidity</td>
<td>0.18</td>
<td>-0.12</td>
<td>0.25</td>
</tr>
<tr>
<td>Warm</td>
<td>0.01</td>
<td>-0.31</td>
<td>0.18</td>
</tr>
<tr>
<td>Cool</td>
<td>0.15</td>
<td>0.00</td>
<td>0.11</td>
</tr>
</tbody>
</table>

### Table 4: Odds ratios and 95% confidence intervals (CI) by mortality category, per lag, per unit concentration of 24 hr average of $B_{sp}$, NO$_2$ and PM$_{2.5}$ (modeled)

<table>
<thead>
<tr>
<th>Mortality Category</th>
<th>Lag</th>
<th>NO$_2$ OR (95% CI)</th>
<th>CO OR (95% CI)</th>
<th>$B_{sp}$ OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>0</td>
<td>0.960 (0.7285 - 1.0367)</td>
<td>0.9922 (0.9836 - 1.0099)</td>
<td>0.961 (0.9915 - 1.0023)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>0.9384 (0.7529 - 1.0719)</td>
<td>0.9953 (0.9867 - 1.0040)</td>
<td>0.961 (0.9916 - 1.0015)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.9785 (0.6237 - 1.6162)</td>
<td>0.9971 (0.9807 - 1.0050)</td>
<td>1.0166 (0.9971 - 1.0322)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.9816 (0.8240 - 1.1694)</td>
<td>1.0019 (0.9932 - 1.0106)</td>
<td>1.0005 (0.9950 - 1.0060)</td>
</tr>
<tr>
<td>Cardiovascular Diseases (CVD)</td>
<td>0</td>
<td>1.0536 (0.9740 - 1.0069)</td>
<td>1.0031 (0.9992 - 1.0069)</td>
<td>0.961 (0.9982 - 1.0023)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1.0089 (0.9235 - 1.0073)</td>
<td>1.0004 (0.9995 - 1.0097)</td>
<td>0.9955 (0.9975 - 1.0015)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1.0017 (0.9264 - 1.0075)</td>
<td>1.0027 (0.9998 - 1.0095)</td>
<td>1.0002 (0.9982 - 1.0022)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.9918 (0.9165 - 1.0053)</td>
<td>1.0014 (0.9976 - 1.0053)</td>
<td>1.0000 (0.9978 - 1.0023)</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>1.0075 (0.9740 - 1.0069)</td>
<td>1.0323 (1.007 - 1.016)</td>
<td>0.9999 (0.9972 - 1.0207)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1.0075 (0.9740 - 1.0069)</td>
<td>1.0323 (1.007 - 1.016)</td>
<td>0.9999 (0.9972 - 1.0207)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1.0075 (0.9740 - 1.0069)</td>
<td>1.0323 (1.007 - 1.016)</td>
<td>0.9999 (0.9972 - 1.0207)</td>
</tr>
</tbody>
</table>

Note: Each pollutant was entered into its own predictive model for seven different lag periods, (0,1,2,0-1, 0-2, 0-3) days.
Air Pollution and Mortality in Perth

**Figure 3:** Odds ratios plotted for each mortality category lag per unit increase of particle concentrations as measured by nephelometry (1 hr)

- **Respiratory Mortality Odds Ratio per Unit Increase in Neph:**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 1.05, 1.02, 0.98, 0.99, 0.99, 0.99

- **CVD Mortality Odds Ratio per Unit Increase in Neph:**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 1.05, 1.02, 0.98, 0.99, 0.99, 0.99

- **Other Mortality Odds Ratio per Unit Increase in Neph:**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 1.05, 1.02, 0.98, 0.99, 0.99, 0.99

**Figure 4:** Odds ratios plotted for each mortality category, lag and per unit increase in nitrogen dioxide concentrations ppb (1 hr)

- **Respiratory Mortality Odds Ratio per Unit Increase in NO2:**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 1.05, 1.02, 0.98, 0.99, 0.99, 0.99

- **CVD Mortality Odds Ratio per Unit Increase in NO2:**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 1.05, 1.02, 0.98, 0.99, 0.99, 0.99

- **Other Mortality Odds Ratio per Unit Increase in NO2:**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 1.05, 1.02, 0.98, 0.99, 0.99, 0.99
Figure 5: Odds ratios plotted for each mortality category per unit increase in ozone concentrations ppb (8 hr)

- **Respiratory Mortality Odds Ratio per Unit Increase in Ozone (8 hr)**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 0.98, 0.99, 1, 1.02, 1.04

- **CVD Mortality Odds Ratio per Unit Increase in Ozone (8 hr)**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 0.98, 0.99, 1, 1.02, 1.04

- **Other Mortality Odds Ratio per Unit Increase in Ozone (8 hr)**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 0.98, 0.99, 1, 1.02, 1.04

Figure 6: Odds ratios plotted for each mortality category per unit increase in CO concentration (8hr)

- **Respiratory Mortality Odds Ratio per Unit Increase in CO (8 hr)**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 0.8, 0.9, 1, 1.1, 1.2

- **CVD Mortality Odds Ratio per Unit Increase in CO (8 hr)**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 0.8, 0.9, 1, 1.1, 1.2

- **Other Mortality Odds Ratio per Unit Increase in CO (8 hr)**
  - Lag: 0, 1, 2, 3, +1, +2, +3
  - Odds Ratio (95% CI): 0.8, 0.9, 1, 1.1, 1.2
changes in PM$_{2.5}$ concentrations and mortality (Table 4).

The odds ratios for 1, 4 and 8 hr O$_3$ concentration and respiratory mortality were elevated with wide confidence intervals, with statistical significance observed for 8-hr O$_3$ concentration (Figure 5). Significant odds ratios were observed for changes in O$_3$ concentration (1hr, 4hr, 8hr) and CVD mortality with an estimated 0.2 to 0.45% increase in mortality for every 10 ppb of O$_3$.

Significant associations were also observed for changes in O$_3$ concentration (8hr) and CVD and respiratory mortality (Figure 5). A significant association was also observed for the relationship between changes in the 8-hr CO concentrations and ‘other’ mortality but not for other mortality categories (Figure 6).

**Discussion**

The results of this study suggest that changes in daily air quality in Perth may increase the risks of cardiovascular and respiratory mortality. The significant finding for O$_3$ concentrations provides an estimated 3% increase in mortality with every 10 ppb change in O$_3$ concentration. These results are supported by the recent publication by Gryparis et al. (2004) where a significant increase in the number of cardiovascular deaths was associated with both 1hr and 8hr O$_3$ concentration in Europe. Vedal et al. (2003) report similar findings.

Significantly elevated odds ratios for cardiovascular mortality in relation to NO$_2$ concentrations were also observed. Similar findings have been reported for other studies using different statistical methods (Dockery et al. 1993; Morgan et al. 1998; Simpson et al. 2000). The significant finding for CO 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.

Other case crossover study design studies have been used to investigate daily mortality and air pollution. The Shanghai study reports a significant increased mortality associated with increases of PM$_{10}$, SO$_2$ and NO$_2$ concentrations (Kan, Chen & Jia 2003). The Seoul study found a significant increase in mortality associated with SO$_2$ concentrations and a non significant but elevated odds ratio for mortality associated with 1 hr O$_3$ and particle concentrations (Lee & Schwartz 1999). Although we also found significant increased mortality associated with NO$_2$ and O$_3$ concentrations, it is difficult to compare previous study results with this study since we analysed mortality data into three different categories. In addition, the studies by Lee and Schwartz (1999) and Kan, Chen and Jia (2003) reported increased mortality associated with changes in SO$_2$ concentrations. One of the benefits of this study is the absence of SO$_2$, and the strength of the significant findings for the pollutants in the absence of SO$_2$.

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.

**Conclusion**

Our findings are consistent with the findings of other studies reported in the national and international literature. Since air quality in Perth has been associated with increased risks of cardiovascular mortality further development and implementation of the Air Quality Management Plan for Perth will contribute to reducing air pollutant concentrations and improving the health of Western Australians.

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