A case-crossover analysis of traffic-related air pollution and emergency department presentations for asthma in Perth, Western Australia

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Recent studies have reported significant associations between air pollution and the exacerbation of asthma. Traffic-related air pollution, the largest anthropogenic contributor to ambient air pollution in many cities, has been particularly implicated. However, the strength of the effect of air pollution on asthma exacerbation resulting in use of hospital services has not yet been adequately assessed.

Various studies have measured hospital admissions rather than emergency department (ED) presentations. Patients experiencing milder asthma symptoms are under-represented in such studies because 60% of patients who present at an ED with asthma show “mild” symptoms and only 13% of these patients are subsequently admitted. ED presentations are also of interest in relation to health service use given the typically high workloads and time-critical responsibilities of ED staff.

A common method for estimating background pollution is to average the concentrations obtained from a set of monitoring sites. However, this estimate is more accurate for patients living close to monitoring sites, and may be biased by the higher records from monitors positioned in “problem spots”. An alternative approach based on modelling exposure using monitored pollutant concentrations has potential to overcome these problems, and was adopted for our study.

In Australia, research on the relation between asthma ED presentation and ambient air pollution is limited, although a 2005 study conducted in Sydney reported significant associations with all criteria pollutants examined, despite the generally low levels of ambient air pollution. We were motivated by these results to determine whether such associations are to be found in the city of Perth, Western Australia, whose airshed is characterised by different pollutant mixtures and meteorological conditions.

**METHODS**

**Study design and rationale**

A record-based, time-stratified case-crossover method was used.

Studies of the impacts on health of air pollution often involve time-series designs, many of which are prone to seasonal or other temporal confounding effects, including those that pertain to the patient. These can be minimised by the use of a case-crossover design, which inherently controls for both measured and unmeasured time-invariant confounders (eg, socioeconomic position) including those that slowly vary with respect to the reference time-period window. The case-crossover design achieves this by comparing the patient’s exposure directly before an ED presentation to their exposure at other times when they did not present at an ED. For example, the design can control for socioeconomic position because this is unlikely to change within a relatively short reference time period (eg, a calendar month, as in our study). By comparing exposure preceding the ED presentation with exposure on days shortly before and after the ED presentation, one can also remove effects associated with seasonal trends. Other examples of patients’ risk factors and study confounders that can be controlled for under this design are body mass index, diet, seasonal comorbidities and exposure to pollen, environmental tobacco smoke and indoor allergens.

**Study area**

The study area included 613 census collection districts (CDs), within south-west metropolitan Perth. CDs are geographical areas for which demographic statistics are disseminated by the Australian Bureau of Statistics, and each on average included 225 dwellings. Perth is protected from air pollution entering from other urban airsheds by its location on a coastal plain between the Indian Ocean to the west and the 300–400 m-high Darling escarpment to the east. It is also more than 2000 km from the nearest major city. Perth’s south-western area was selected because it is traversed by both major metropolitan vehicle corridors and quieter local roads; it is geographically isolated from major industrial sources of air pollution; and its population has a wide socioeconomic range. The total population in the area was 269,734.

**ABSTRACT**

**Objective:** To determine whether changes in 24-hour average background ozone ($O_3$), nitrogen dioxide ($NO_2$), carbon monoxide (CO) and particulates <10 μm (PM$_{10}$) increase the risk of hospital emergency department (ED) presentations for asthma among children.

**Design, setting and subjects:** A time-stratified case-crossover method was used to analyse data of 603 children and young adults aged 0–19 years who were resident in a south-west metropolitan area of Perth, Western Australia, and who had presented with asthma at any public ED within Perth between 1 January 2002 and 31 December 2006. Effect sizes were assessed in relation to age group, sex and season of exposure. City-wide background air pollution was estimated from air monitoring network data.

**Main outcome measures:** ED presentation with asthma.

**Results:** Patients 0–4 years with 1-day lagged exposure to $NO_2$ and CO showed the most significant risk of ED presentation for asthma. An interquartile range (IQR) increase in $NO_2$ resulted in an odds ratio (OR) of 1.70 (95% CI, 1.08–2.69). An IQR increase in CO resulted in an OR of 1.40 (95% CI, 1.06–1.84).

**Conclusions:** The effect sizes observed in this study were higher than those of past studies, and indicated that children aged 0–4 years were the most vulnerable to the effects of air pollution. The period of exposure most clinically relevant is the day before ED presentation.

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Data collection

Data were collected from WA Department of Health records on patients aged 0–19 years, living in the study area, who had presented at any of the nine public hospital EDs within Perth between 1 January 2002 and 31 December 2006 and had been assigned a principal diagnosis of asthma (International classification of diseases, 10th revision [ICD-10]: J45) or status asthmaticus (ICD-10: J46). Seven different patient exposure periods were investigated for all patients: 0, 1, 2, 3, 0–1, 0–2, and 0–3 days prior to the ED visit. Each patient’s exposure day was matched to a set of referent control exposure days for the same patient falling on the same day of the week, in the same calendar month, in the same year as the day of the ED presentation.

Exposure assessment

To obtain a city-wide pollutant concentration for each day, a model for the back-ground air pollution. Validation was performed by using Pearson correlations to compare the modelled daily concentrations with those derived from averaging across all monitoring stations. Correlations were subsequently stratified by sex and age group for lag periods where an elevated risk was observed. Due to the difference in lung function across the ages of the study population, age was initially grouped into four equal bands for analyses but later grouped into three bands (ie, 0–4, 5–9 and 10–19 years) to better accommodate the sample size.

Ethics

Ethics approval was obtained from the University of Western Australia Human Research Ethics Committee and the Department of Health Western Australia Human Research Ethics Committee.

RESULTS

Demographics

Between 2002 and 2006, there were 603 children and young adults aged up to 19 years who were resident in the study area and had presented at a Perth ED with a primary diagnosis of asthma (Box 1). Most of the children were boys (57% [341]); under the age of 10 (66% [400]); and presented at the ED on Sundays (19% [112]) and in autumn or winter (60% [364]). Forty children (7%) were classified as Indigenous. The socioeconomic index for areas (SEIFA) has a mean of 1000 (SD 100) across Australia. Relative to this distribution, the study area was of slightly higher socioeconomic status, with the 40th percentile (1028) being higher than the Australian mean.

Air pollutant concentrations

The 25th percentile (P25) and 75th percentile (P75) of the observed 24-hour pollutant concentrations at the nearest monitoring station (South Lake) over the study period were: 5 ppb (P25) and 10 ppb (P75) for NO2; 0.1 ppm (P25) and 0.3 ppm (P75) for CO; 13 ìg/m3 (P25) and 22 ìg/m3 (P75) for PM10; and 12 ppm (P25) and 20 ppm (P75) for O3. The modelled pollutant concentration exposures for the study population over the study period are displayed in Box 2. The pollutants showed a clear seasonal cycle with peak NO2 and CO occurring in the winter months and peak O3 and PM10 occurring in the summer months. Significant correlations were observed between daily concentrations of O3 and PM10 (correlation coefficient [r] = 0.42, P < 0.01), and between CO and NO2 (r = 0.85, P < 0.01). O3 and PM10 were negatively correlated with CO and NO2. The highest
negative correlation was observed between O\textsubscript{3} and CO ($r = -0.81$).

### Air pollution and ED presentations for asthma

A total of 18 patients re-presented during their referent period, and were subsequently removed from the analyses. Statistically significant increases in odds of ED presentation were only observed for NO\textsubscript{2} and CO\textsubscript{i}, most notably for a 1-day lag, which empirically suggested a 1-day induction period. Analyses for NO\textsubscript{2} and CO were then restricted to this lag period and stratified by age and sex. Significant odds ratios (ORs) were observed for the lowest age group (0–4 years) (Box 3). These patients exhibited a 21% increase in odds per 1 ppb increase in NO\textsubscript{2} (OR, 1.21 [95% CI, 1.03–1.43]), and almost a fivefold increase in odds of ED presentation per 1 ppm increase in CO (OR, 4.92 [95% CI, 1.33–18.15]). When the study sample was stratified by sex, only boys exhibited significant ORs. Among this group, odds of ED presentation were 16% (OR, 1.16 [95% CI, 1.01–1.32]) and four times (OR, 4.13 [95% CI, 3.71–18.15]) higher per 1 unit increase in NO\textsubscript{2} and CO, respectively.

### DISCUSSION

The results indicate that increases in daily background air pollution increase the risk of ED presentation of children for asthma. The most recent comparable Australian study was of presentations between 1997 and 2001 in Sydney, and reported a 0.1%–0.4% increase in risk per 1 ppb increase in 1-hour maximum NO\textsubscript{2}, and a 3.2% increase in risk per 1 ppm increase in 8-hour average CO.\textsuperscript{16} An interquartile range increase in 1-hour NO\textsubscript{2} and 8-hour CO was associated with 1.4% (95% CI, 0.5%–2.4%) and 1.5% (95% CI, 0.6%–2.3%) respective increases in ED presentations among the 1–4-years age group in the Sydney study\textsuperscript{16}. The researchers noted that the associations were most consistent for all assessed air pollutants in the lowest age group. Our findings also suggest greater vulnerability among this group. This is yet to be confirmed by international studies — past international studies have tended not to include the very young as a study subgroup\textsuperscript{20,21} despite their potentially greater vulnerability. Our study assessed more recent presentations (2002–2006) and showed a higher risk of ED presentation compared with the Sydney study. We observed a 70% increase in risk per interquartile range increase in 24-hour NO\textsubscript{2} and a 40% increase in risk per interquartile range change in 24-hour CO among the 0–4-years age group. We used 24-hour average concentrations based on the hypothesis that this was the most aetiologically relevant period, considering the potential for cumulative exposure. This period also matched the frequency at which the ED presentations were obtained. Although this may explain the stronger effect sizes observed for NO\textsubscript{2} and CO, it may also have contributed to the non-detection of an effect for other pollutants.

Despite the importance of our findings, this study has some methodological limitations. The power of the study prevented a comprehensive analysis of co-pollutant effects, such as by matching (eg, to investigate the effect of NO\textsubscript{2}, we would have had to ensure case and control periods had the same level of CO).\textsuperscript{22} The possible effects of CO and NO\textsubscript{2} concentrations on the risk of ED presentation with asthma could not be separated from each other. In addition, the high negative correlation between these two pollutants and O\textsubscript{3} and PM\textsubscript{10}, possibly because of contrasting meteorological conditions, meant that the strong effects of CO and NO\textsubscript{2} may have masked those of O\textsubscript{3} and PM\textsubscript{10}. However, these results may not be significantly confounded by meteorological effects (such as temperature), given that a
recent multi-city study on hospitalisations for respiratory illness and criteria air pollution found little change in effect estimates after pollutant concentrations were matched to within 1°C of temperature.7

Although we restricted our study to an area with minimal non-traffic sources of air pollution, it was impractical to find an urban area completely unaffected by such sources. Non-traffic sources contribute to the modelled background concentrations used as exposures in this study. However, 80% of ambient CO in Perth is derived from traffic-related sources.36 Non-traffic sources contribute to urban areas completely unaffected by such sources. Therefore, CO, nitrogen oxides, depending on the exact pollutionary event, as it is formed by reactions with nitrogen oxides, depending on the exact atmospheric composition. Therefore, CO, NO2 and O3 may be reasonably considered as traffic-related ambient air pollutants. Traffic emissions also contain PM10, although the modelled background concentrations are likely to have significant contributions from other sources.

Although we obtained all public ED presentations for our study period, we could not obtain information on those at private hospitals. Such a sample loss may imply that our results relate more to the low-middle socioeconomic class, but this would have little effect on internal validity. External validity is supported by the existence of only one major private hospital with an ED in the study area.

We know of no changes to the diagnostic process or any documented change to the recording of ED information during the study period. However, there is a risk of misdiagnosis because of asthma’s similarity with other respiratory diseases such as bronchiolitis, a risk possibly more pronounced for the youngest age group. The observed association with air pollution may relate to symptoms common to asthma and bronchiolitis. A post hoc sensitivity analysis for 1-day lagged NO2 and CO exposure showed negligible influence on the effect estimates from that presented in Box 3. The OR for a 1 ppm increase in NO2 among a 1–4-years age group was the same as that for a 0–4-years age group (1.21 [95% CI, 1.03-1.43]). Similarly, there was little change in the OR for a 1 ppm increase in CO exposure (4.87 [95% CI 1.32-17.99) for a 1–4-years age group.

The results of this study indicated that children aged under 5 years were the most vulnerable to increases in traffic-related air pollution and that the most clinically relevant period is exposure on the day before ED presentation.

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COMPETING INTERESTS

None identified.

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