

Long-term exposure to gaseous air pollutants and cardio-respiratory mortality in Brisbane, Australia

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Abstract. This study examines the association of long-term exposure to gaseous air pollution with cardio-respiratory mortality in Brisbane, Australia, in the period 1996-2004. The pollutant concentrations were estimated using geographical information system (GIS) techniques at the statistical local area (SLA) level. The generalized estimating equations model was used to investigate the impact of nitrogen dioxide (NO₂), ozone (O₃) and sulphur dioxide (SO₂) on mortality due to cardio-respiratory disease after adjusting for a range of potential confounders. An increase of 4.7% (95% confidence interval = 0.7-8.9%) in cardio-respiratory mortality for 1 part per billion (ppb) increment in annual average concentration of SO₂ was estimated. However, there was no significant association between long-term exposures to NO₂ or O₃ and death due to cardio-respiratory disease. The results indicate that the annual average concentration of SO₂ is associated with cardio-respiratory mortality at the SLA level and this association appears to vary with the geographical area.

Keywords: cardio-respiratory mortality, air pollution, nitrogen dioxide, ozone, sulphur dioxide, spatial analysis, Australia.

Introduction

Air pollution is ranked by the World Health Organization (WHO) as one of the top ten contributors to the global burden of disease and injury. Exposure to gaseous air pollutants, even at a low level, has been associated with cardio-respiratory diseases (Vedal et al., 2003). Most recent epidemiological studies of air pollution have used time-series analyses to explore the relationship between daily mortality or morbidity and daily ambient air pollution concentrations based on the same day or previous days (Hajat et al., 2007).

However, most of the previous studies have examined the association between air pollution and health outcomes using air pollution data from a single monitoring site, or average values from a few monitoring sites, to represent the whole population of the study area. In fact, for a metropolitan city, ambient air pollution levels may differ significantly among different areas and there is increasing concern that the relationships between air pollution and mortality may vary with geographical area (Chen et al., 2007). Additionally, some studies have indicated that socio-economic status can act as a confounding factor when investigating the relation between geographical location and health (Scoggins et al., 2004).

This study examines the spatial variation in the relationship between long-term exposure to gaseous air pollutants – including nitrogen dioxide (NO₂), ozone (O₃) and sulphur dioxide (SO₂) – and cardio-respiratory mortality in Brisbane, Australia, during the period 1996-2004.

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Materials and methods

Study area

Brisbane is the capital of Queensland and the third largest city after Sydney and Melbourne in Australia. Its urban areas cover 1326.8 km² and the population has increased by 16.3% from 824,489 in mid-1996 to 958,504 in mid-2004. In this study, the data analysis was undertaken at the statistical local area (SLA) level, i.e. the basic spatial unit used to collect and analyse data. There were 162 SLAs in Brisbane during the study period.

Gaseous air pollutants

Daily data on maximum 1-hour O₃, NO₂ and SO₂ concentrations in parts per billion (ppb), recorded in 13 monitoring stations for the period of 1 January 1996 to 31 December 2004, were obtained from the Queensland Environmental Protection Agency (QEPA) (Fig. 1). The daily O₃, NO₂ and SO₂ concentrations were aggregated to annual means and

used as long-term gaseous air pollution exposure indicators. Geographical information system (GIS) techniques were used for mapping the spatial patterns of annual average O₃, NO₂ and SO₂ concentrations at the SLA level.

Health data

In order to assess the association between exposure to gaseous air pollutants and cardio-pulmonary mortality at the SLA level, we estimated the annual average potential concentrations of gaseous pollutants by SLA using an inverse distance weighted (IDW) method (O'Sullivan and Unwin, 2003), as most SLAs did not have a monitoring station.

Mortality data were provided by the Office of Economic and Statistical Research of the Queensland Treasury. Apart from date and cause of death, the data used included sex, age and SLA of residence. The cause-specific mortality were categorised according to the International Classification of Diseases version 9 (ICD-9 code) or version 10 (ICD-10 code) and were defined as cardio-respirato-

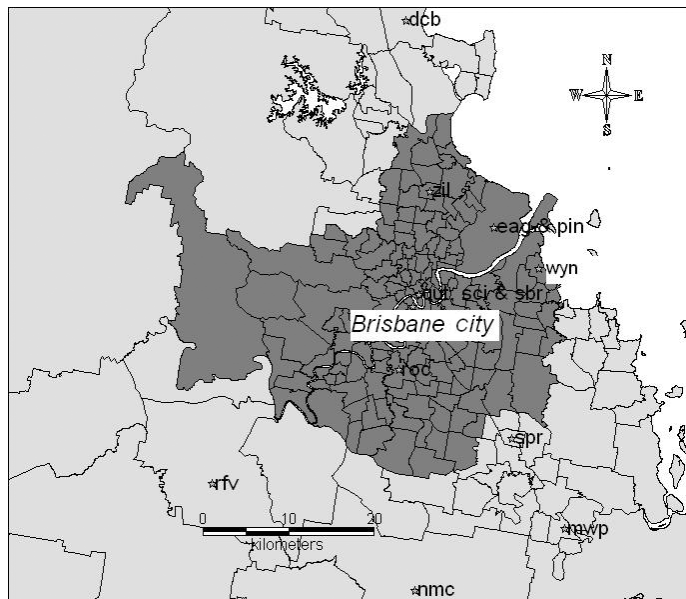


Fig. 1. Locations of air pollution monitoring stations around urban Brisbane city (13 monitoring stations in total: Brisbane CBD (qut), Brisbane CBD (sci), Deception Bay (dcb), Eagle Farm (eag), Flinders View (rfv), Mount Warren Park (mwp), North Maclean (nmc), Pinkenba (pin), Rocklea (roc), South Brisbane (sbr), Springwood (spr), Wynnum (wyn) and Zillmere (zil)).

ry diseases (CRD) (ICD 9: 390-519; ICD 10: I00-199 and J00-J99), including both respiratory and cardio-vascular diseases (Vedal et al., 2003; Scoggins et al., 2004). During the study period, 51,233 deaths were recorded, including 27,480 cardio-respiratory deaths. To compare the spatial patterns of cardio-respiratory mortality across SLAs, an indirect method (i.e. using the Brisbane population as a reference) was used to calculate the age-standardized mortality rate (ASM) for each SLA, adjusted for differences in the age and sex distributions among SLAs (Selvin, 2001).

The 2001 census provided information on resident population, sex, and age groups by SLA for Brisbane city. We also obtained the socio-economic indexes for areas (SEIFA) and data for each SLA in 2001 from the Australian Bureau of Statistics.

Statistical analysis

The generalised estimating equations (GEE) model was used to investigate how gaseous air pollutants influence the probability of deaths as this model is capable of removing spatial autocorrelation of residuals and can implement unbiased regression in spatial analysis (Hedeker and Ebooks, 2006; Carl and Kühn, 2007). We controlled for confounding effects of age

(<14, 15-64, 65-74 and 75+ years), sex, calendar year, the SEIFA disadvantage index, and the population size using an offset term in the model. The dependent variable was the annual counts of cardio-respiratory deaths for each SLA, while the annual means of gaseous air pollutants constituted independent variables. Both single pollutant and multiple pollutants models were performed in relation to cardio-respiratory mortality using the SAS statistical software package. Finally, Moran's *I* statistic, with a contiguity-based spatial weights matrix, was calculated to assess spatial autocorrelation of residuals using ArcGIS 9.2.

Results

The daily maximum 1-h NO₂, O₃ and SO₂ concentrations recorded in Brisbane are presented in Table 1. The overall averages of these gases were 19.8 ppb, 30.1 ppb and 5.4 ppb, respectively.

Pearson correlations between gaseous pollutants were calculated in three different types of monitoring stations: Eagle Farm (light industrial area), Brisbane CBD (qut) (commercial area) and Springwood (residential area) as they all have long-term monitoring data (Table 1). The correlations between NO₂, O₃ and SO₂ concentrations in each station ranged from 0.10 to 0.48. For the same

Table 1. Summary statistics of gaseous air pollutants in all monitoring stations, Brisbane, Australia (1996-2004).

Monitoring site	Area classification	Max 1-h NO ₂		Max 1-h O ₃		Max 1-h SO ₂	
		Mean	SD ^a	Mean	SD ^a	Mean	SD ^a
Brisbane CBD (qut)	Commercial	21.7	8.8	28.2	13.5	4.9	3.9
Brisbane CBD (sci)	Commercial	25.4	10.4	23.7	7.9	3.2	2.4
Deception Bay (dcb)	Residential	16.8	8.6	32.1	9.9	-	-
Eagle Farm (eag)	Light industrial	21.6	9.7	29.9	10.8	6.4	5.4
Flinders View (rfv)	Residential	19.8	8.1	35.6	13.8	7.7	8.7
Mount Warren Park (mwp)	Residential	15.1	8.1	32.1	12.4	-	-
North Maclean (nmc)	Rural	11.0	5.8	35.0	13.9	-	-
Pinkenba (pin)	Residential (adjacent to industry and airport)	18.9	6.6	29.9	8.7	7.8	7.8
Rocklea (roc)	Residential/light industrial	20.1	9.1	34.3	13.5	-	-
South Brisbane (sbr)	Commercial	28.0	9.0	-	-	-	-
Springwood (spr)	Residential	19.4	7.7	27.0	8.7	2.5	2.8
Wynnum (wyn)	Residential (adjacent to industrial zone)	20.8	10.1	25.8	7.6	5.1	5.4
Zillmere (zil)	Light industrial area	18.8	8.3	27.1	11.3	-	-

^aSD = standard deviation; - = not recorded.

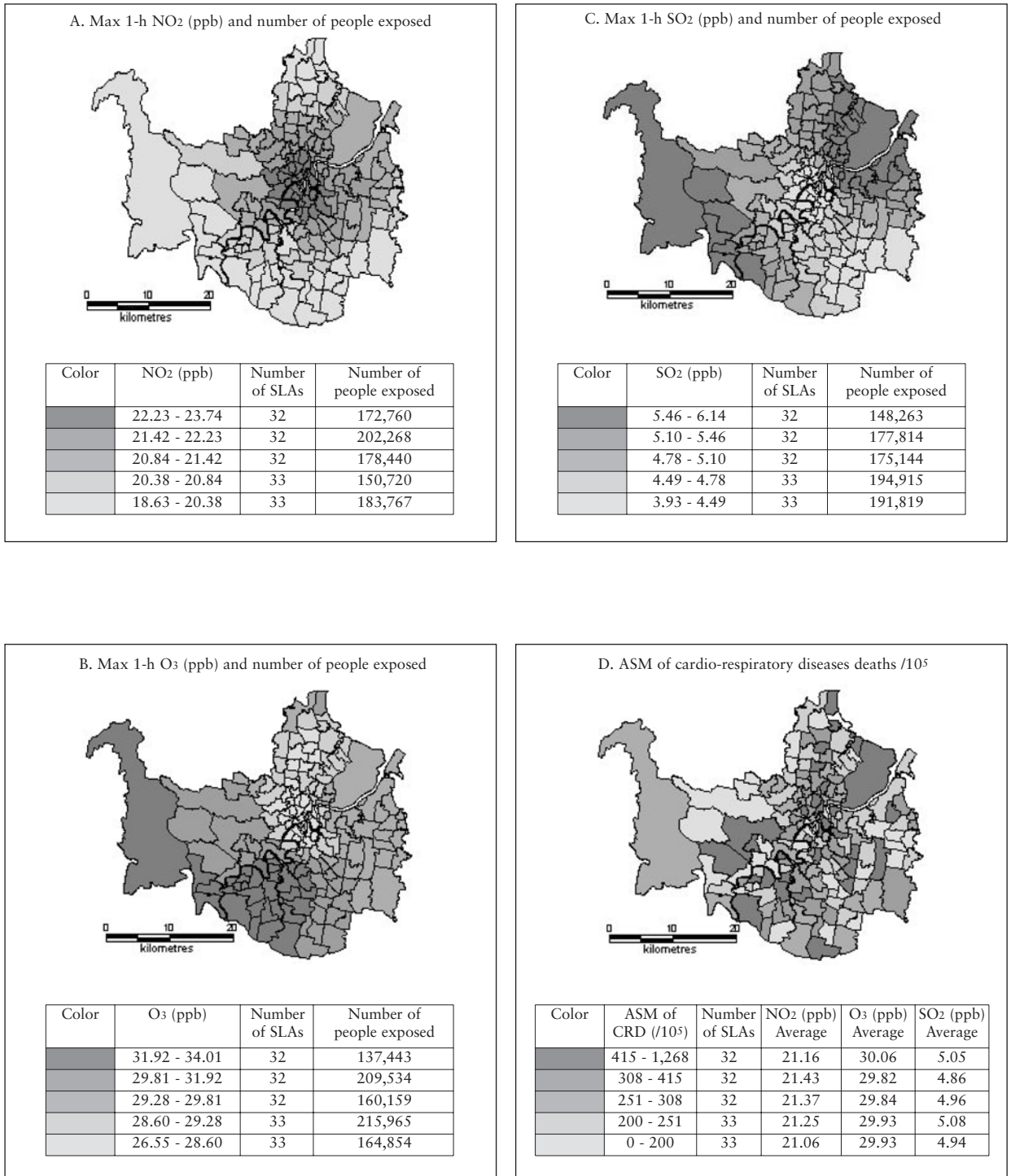


Fig. 2. Spatial patterns of annual average concentrations of NO₂, O₃ and SO₂; annual average ASM of cardio-respiratory mortality in Brisbane, Australia (1996-2004).

gaseous air pollutant across different monitoring stations, there were moderate to high correlations for NO₂ concentrations (ranging from 0.35 to 0.89), and O₃ concentrations (ranging from 0.33 to 0.89), but only moderate correlations for SO₂ concentrations (ranging from -0.22 to 0.53).

Figure 2 shows the spatial patterns of annual average concentrations of gaseous air pollutants (NO₂, O₃ and SO₂) and the annual ASM of cardio-respiratory mortality. The number of people exposed is shown for each of the gaseous pollutants, and the average of the gaseous pollutants is also shown in each category of cardio-respiratory mortality at the SLA level.

The highest values of NO₂ were recorded around the centre of Brisbane (Fig. 2A). The spatial pattern of O₃ appears opposite to the NO₂ plot, although with a different dynamical range (Fig. 2B). A negative correlation was found ($r = -0.69$; $P < 0.01$) between O₃ and NO₂. The highest O₃ levels were recorded around the Rocklea station which is surrounded by light industry and residential areas. The spatial features of SO₂ appeared to differ from those of NO₂ and O₃ (Fig. 2C). A negative correlation was also found ($r = -0.29$; $P < 0.01$) between NO₂ and SO₂. The highest SO₂ levels were observed around the Pinkenba and Eagle Farm stations, which are located in a light to moderately developed industrial area.

There was a strong spatial pattern of cardio-respiratory mortality across SLAs (Fig. 2D). The average ASM of CRD was 321.2 per 100,000 person-years

and the highest ASM of CRD (i.e. 1256 per 100,000 person-years) was found in Sandgate during the study period. Sandgate, with 11% of the population aged over 75 years, is one of the oldest suburbs in Brisbane and situated on the coastline, along Moreton Bay. The lowest ASM of CRD (0 per 100,000 person-years) was found in three SLAs, i.e. Gumdale, Riverhills and Tingapla. Less than 4% of the population is aged over 75 years in these areas.

The results of single pollutant and multiple pollutants models were similar and indicated that there was a positive relationship between the annual average SO₂ and cardio-respiratory mortality. After controlling for potential confounding factors (i.e. age, sex, population size, calendar year, spatial autocorrelation, SEIFA and other gaseous air pollutants), a 4.7% (95% confidence interval (CI) = 0.7-8.9%) increase in cardio-respiratory mortality per 1 ppb increase in the annual average SO₂ concentrations was estimated. However, there was no significant association of the annual average concentrations of NO₂ or O₃ with cardio-respiratory mortality (Table 2). The residual analyses indicated that there was no significant spatial autocorrelation of residuals for either of the single pollutant (Moran's $I = 0.079$, $P > 0.05$) and multiple pollutant model (Moran's $I = 0.075$, $P > 0.05$). This outcome suggests that the model provided a reasonable fit to the data.

Discussion

This study found a positive association between long-term exposure to SO₂ and cardio-respiratory mortality at the SLA level in the town of Brisbane, on the east coast of Australia. This significant association remained even after simultaneously adjusting for confounding effects such as age, sex, calendar year, SEIFA, population size and other gaseous air pollutants. However, there was no significant association of annual average concentrations of NO₂ or O₃ with cardio-respiratory mortality.

In the study, we assumed that annual average concentrations of O₃, NO₂ and SO₂ are appropriate indicators of long-term exposure to air pollution. As

Table 2. Relative risk for cardio-respiratory mortality from long-term exposure to gaseous air pollutants.

Pollutant	Relative risk	
	Single pollutant (95% CI) ^a	Multiple pollutant (95% CI) ^b
NO ₂	0.995 (0.970-1.020)	0.997 (0.971-1.022)
O ₃	1.002 (0.989-1.015)	0.999 (0.986-1.012)
SO ₂	1.047 (1.006-1.090)	1.047 (1.007-1.089)

^aAdjusted for the confounding effects of age, sex, population size, calendar year and SEIFA

^bAdjusted for the confounding effects of age, sex, population size, calendar year, SEIFA and other gaseous air pollutants.

gaseous pollutants often share the same source, or are subject to the same environmental influences, levels tend to be highly correlated over time. A number of other studies have used a similar approach to estimating long-term exposure to ambient air pollution levels (Vedal et al., 2003; Scoggins et al., 2004). In order to simulate the urban air quality of Brisbane at the unit of the SLA, the IDW method was used to estimate the gaseous air pollutant concentrations of O₃, NO₂ and SO₂ for each SLA (O'Sullivan and Unwin, 2003). This method provides a more complete spatial picture of air quality than monitoring data which is only available from a few stations. This is necessary for examining the effects of long-term exposure to air pollution because no single site can represent air quality of a whole city.

Exposure to air pollution affects mortality and the number of hospital admissions. However, relatively few studies have addressed the long-term effects of air pollution on mortality, and most of them have shown an association for NO₂ (Scoggins et al., 2004) and particulate matter (Pope et al., 2002). The levels of air pollution in Brisbane were found to be lower than most metropolitan cities in the world. The results of this spatial analysis still show a statistically significant association between the annual mean of SO₂ and cardio-respiratory mortality, even after controlling for potential confounding factors at the SLA level. However, we did not find significant effects of long-term exposure to NO₂ and O₃ on cardio-respiratory mortality.

In our opinion, the current study has three major strengths. Firstly, it systematically examined the spatial distribution of cardio-respiratory mortality associated with long-term exposure to gaseous air pollution in a metropolitan setting. Secondly, most of the known confounding factors have been controlled for, including age, sex, population size, calendar year, spatial auto-correlation and SEIFA at the SLA level. Finally, the inclusion of SEIFA as a composite index of socio-economic status also reflects the influence of some unmeasured factors such as smoking habits and physical inactivity spelled out in

the report on "Chronic Conditions and Health Risk Factors" published by the South Australian Department of Health.

Limitations are also encountered in this study. Firstly, seasonal differences in air pollution and mortality patterns were not examined as we only used annual average of data. Secondly, as this is an ecological study, exposure misclassification bias is inevitable to some extent. For example, exposure at people's homes and the place where they work may differ. However, most of the deaths occurred in the elderly who were likely to stay at home most of the time. Hence, the extent of misclassification bias is regarded as limited.

In conclusion, this study used a geospatial approach to analyse the relationship between long-term exposure to gaseous air pollutants and cardio-respiratory mortality. The results of our study indicate that long-term exposure to SO₂ in Brisbane, even at the levels lower than most other metropolitan cities, is associated with cardio-respiratory mortality. Furthermore, this association appears to vary with geographic area. Therefore, the spatial features of air pollution and health outcomes should be considered when modelling air pollution and health relationships, particularly for large cities.

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