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Gaseous air pollution and emergency hospital visits for hypertension in Beijing, China: a time-stratified case-crossover study

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Abstract

Background: A number of epidemiological studies have been conducted to research the adverse effects of air pollution on mortality and morbidity. Hypertension is the most important risk factor for cardiovascular mortality. However, few previous studies have examined the relationship between gaseous air pollution and morbidity for hypertension.

Methods: Daily data on emergency hospital visits (EHVs) for hypertension were collected from the Peking University Third Hospital. Daily data on gaseous air pollutants (sulfur dioxide (SO₂) and nitrogen dioxide (NO₂)) and particulate matter less than 10 μm in aerodynamic diameter (PM₁₀) were collected from the Beijing Municipal Environmental Monitoring Center. A time-stratified case-crossover design was conducted to evaluate the relationship between urban gaseous air pollution and EHVs for hypertension. Temperature and relative humidity were controlled for.

Results: In the single air pollutant models, a 10 μg/m³ increase in SO₂ and NO₂ were significantly associated with EHVs for hypertension. The odds ratios (ORs) were 1.037 (95% confidence interval (CI): 1.004-1.071) for SO₂ at lag 0 day, and 1.101 (95% CI: 1.038-1.168) for NO₂ at lag 3 day. After controlling for PM₁₀, the ORs associated with SO₂ and NO₂ were 1.025 (95% CI: 0.987-1.065) and 1.114 (95% CI: 1.037-1.195), respectively.

Conclusion: Elevated urban gaseous air pollution was associated with increased EHVs for hypertension in Beijing, China.

Background

Numerous recent studies have assessed the adverse effects of air pollution on population health, including mortality, hospital admissions, and emergency hospital visits (EHVs) for cardiovascular diseases, respiratory diseases, and other diseases [1-4]. Some studies found an elevated level of ambient air pollution increased the risks of mortality and morbidity [5,6], while others found inconsistent results [7]. Also, many studies focused on the relationship between ambient air pollution and subgroups of cardiovascular diseases such as

coronary disease [2,8], arrhythmia [5,9], myocardial infarction [10], and heart failure [11,12]. Our previous studies found a positive association between particulate air pollution and EHVs for hypertension and cardiovascular diseases [1,13]. However, less evidence is available to illustrate the effect of gaseous air pollution on acute events for hypertension.

Hypertension is not only one of the most serious risk factors for deaths and disease worldwide [14], but is also a major contributor to chronic heart failure, and a major risk factor for stroke and coronary heart disease, and their progression [15]. In the United States, the number of deaths caused by hypertension rose by 53% from 1991 to 2001 [16]. Ninety-one percent of people with heart failure had preceding hypertension, and half of all patients suffering a heart attack (and two-thirds of those having a first time stroke) have a blood pressure greater than systolic blood pressure (SBP) 140 mmHg

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and diastolic blood pressure (DBP) 90 mmHg [16]. Research conducted in Beijing, China shows that about 47% of investigated people had hypertension [17]. It is important to identify triggers and/or risk factors for hypertension. Air pollution may induce hypertension, so it is necessary to examine the relationship between air pollution and hypertension.

A few studies have examined the relationship between gaseous air pollution and blood pressure. Ibalid-Mulli et al. [18] carried out a study using a random population sample to assess the association between air pollution and blood pressure, and found that an increase of 80 $\mu\text{g}/\text{m}^3$ in SO_2 was linked with an increase in SBP of 0.74 mmHg (95% CI: 0.08-1.40). De Paula Santos et al. [19] found SO_2 had positive and statistically significant effects on blood pressure. SO_2 was associated with blood pressure in cold weather, as well as NO_2 in warm weather [20]. In addition, SO_2 and NO_2 also strengthened the association between particulate matter less than 2.5 μm in aerodynamic diameter and blood pressure [21].

In this study we used a time-stratified case-crossover design to examine the relationship between gaseous air pollution and EHV for hypertension.

Materials and methods

Data on emergency hospital visits

Data on EHV for hypertension were collected between Jan 1, 2007 and Dec 31, 2007 from the Peking University Third Hospital, located in the northwest of urban city of Beijing [1]. EHV were coded according to the International Classification of Disease, tenth revision (ICD-10) for hypertension (ICD-10: I10). The primary diagnoses were used in this study. The EHV for hypertension were diagnosed by the visits' symptoms, inquiries, and medical inspections so that we expect the misclassification rate to be relatively small. About 95% of visits for hypertension are diagnosed only with hypertension, while 5% have accompanying heart failure, myocardial infarction or other diseases. The cases in this study lived in the residential areas around the hospital or in the urban area in Beijing.

Data on air pollution and weather condition

We accessed daily data on urban sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and particulate matter less than 10 μm in aerodynamic diameter (PM_{10}) from the Beijing Municipal Environmental Monitoring Center. Air pollutants were monitored at eight fixed monitoring sites which were distributed in the urban area of Beijing. The site map was shown in our previous study [1]. Hourly pollutant data was recorded at each site, which we made into daily averages at each site and then for the whole city. If there were missing data from a monitoring station on a given day, then the values from the remaining

monitors were used to calculate the average concentration.

Daily data on temperature and relative humidity for the study period were obtained from the China Meteorological Data Sharing Service System.

Data analysis

Spearman's correlation coefficients were used to evaluate the inter-relations between air pollutants and weather conditions. The time-stratified case-crossover design was used to analyse the association of gaseous air pollution and EHV for hypertension. The case-crossover design compares the exposure in the case period when events occurred with exposures in nearby control periods to examine the differences in exposure which might explain the differences in the daily number of cases. In this study, the cases and controls were matched by day of the week to control for any weekly patterns in deaths or pollution. Controls were compared with cases using the time-stratified method with twenty-eight days strata. So the first stratum was Jan 01 to Jan 28, 2007, the second stratum was Jan 29 to Feb 25. For an EHV on Jan 31 the control days were Feb 7, 14 and 21. The case-crossover method controls for any long-term trends and seasonal patterns in hospital visits and air pollutants. Each case day had three matching control days. The model gives the odds ratio (OR) of an EHV due to an increase in air pollution. When using the case-crossover design, confounders related to individual characteristics such as age, sex, and education are inherently controlled for. Studies have demonstrated that the case-crossover gives unbiased estimates in the presence of strong seasonal confounding [22,23].

There may be a delay between exposure to pollution and onset of hypertension. To examine the hazard period of air pollution for hypertension, we used the polynomial distributed lag model to evaluate the possibly delayed effect of air pollutants [24]. The polynomial smoothing with four degrees of freedom for SO_2 and NO_2 was used in the models. The hazard period was defined the same day as the hospital visit to up to five days prior.

Daily data on temperature and relative humidity were included in all models as confounders [25], using the same polynomial spline as SO_2 and NO_2 . Single pollutant and multiple pollutants models were used to control the influence of other air pollutants. ORs and confidence intervals (CIs) were calculated for each air pollutant. All statistical tests were two-sided. The "season" package of R (version 2.10.1) was used to fit the time-stratified case-crossover [26,27].

Results

There were 1,491 EHV for hypertension in Peking University Third Hospital during the study period. The

descriptive statistics for air pollutants and weather conditions are shown in Table 1. The average concentrations of SO₂, NO₂ and PM₁₀ were 47.5 µg/m³, 66.6 µg/m³ and 149.3 µg/m³, respectively. The average levels of NO₂ and PM₁₀ air pollution were higher than the national secondary ambient air quality standard in China (40 µg/m³ and 100 µg/m³). The average concentration of SO₂ was lower than the national secondary ambient air quality standard in China (60 µg/m³). The average temperature and relative humidity were 14.1°C and 54.2%, respectively.

Figure 1 shows the time series of gaseous air pollutants and EHV_s for hypertension. The concentrations of both SO₂ and NO₂ were highest in winter. The number of EHV_s for hypertension was highest in November.

Table 2 shows the correlations between air pollutants, temperature and humidity. PM₁₀, SO₂ and NO₂ were significantly correlated with each other, e.g., between SO₂ and NO₂ ($r = 0.65, P < 0.01$), PM₁₀ and SO₂ ($r = 0.46, P < 0.01$), and PM₁₀ and NO₂ ($r = 0.64, P < 0.01$).

A 10 µg/m³ increase in SO₂ was significantly associated with EHV_s at lags of 0 and 2 days (Top-left of figure 2), while NO₂ was significantly associated with EHV_s at lags of 0, 2 and 3 days (Top-left of figure 3). According to the OR values and the 95% confidence intervals, we chose the current day and three days after exposure as the hazard period for SO₂ and NO₂, respectively. The ORs were 1.037 (95% CI: 1.004-1.071) for SO₂ and 1.101 (95% CI: 1.038-1.168) for NO₂ (Table 3).

The results of multiple pollutant models are shown in Figure 2, Figure 3 and Table 3. For SO₂, after controlling PM₁₀, NO₂, or both PM₁₀ and NO₂, the ORs were no longer statistically significant and the means were lower than the single pollutant model. After adjusting for PM₁₀, SO₂, or both SO₂ and PM₁₀, the mean effects of NO₂ at lag 3 days were higher than the single pollutant model.

Discussion

This is the first study to examine the association between gaseous air pollution and EHV_s for hypertension in Beijing, China. The time-stratified case-crossover

was used to examine the relationship between gaseous air pollution and EHV_s for hypertension. We found that gaseous air pollution had a significant impact on the EHV_s visits for hypertension. An increase of 10 µg/m³ in levels of SO₂ and NO₂ were associated with an increase of 3.7% (95% CI: 0.4% - 7.1%) and 10.1% (95% CI: 3.8% - 16.8%) in EHV_s for hypertension, respectively. Figure 1 illustrates that the concentrations of SO₂ and NO₂ were remarkably higher in winter, which may be due to the increased use of heating in winter. However, we controlled for this seasonal change using the case-crossover. The major sources of SO₂ and NO₂ in Beijing were heating and industrial sources. Besides, urban NO₂ levels are closely related to traffic emissions [28].

In order to understand if there were any short-term delays between gaseous air pollution and hypertension, the lags of 0 to 5 days were examined in the single air pollutant models. The results show that the adverse effects of SO₂ on EHV_s for hypertension were statistically significant at lags of 0 and lag 2 days, while the impact of NO₂ was significant at lags of 0, 2, and 3 days. In a previous study, we examined the relationship between air pollution and EHV_s for cardiovascular diseases from the same hospital between 2004 and 2006 and found that after adjusting for temperature and relative humidity, the ORs for EHV_s for cardiovascular diseases were 1.014 (95% CI: 1.004-1.024) and 1.016 (95% CI: 1.003-1.029) for a 10 µg/m³ increase in levels of SO₂ or NO₂ at lag 0 day, respectively [1]. This study suggests that the gaseous air pollution has a lagged effect on EHV_s for hypertension.

In the multiple pollutant models, the OR values of SO₂ were lower than the single pollutant models, particularly after controlling for NO₂. Conversely, the OR values of NO₂ were higher than the single pollutant models. Previous research [1,29] on the association between air pollution and mortality and EHV_s for cardiovascular diseases in Beijing showed the same results. There may be some co-linearity between SO₂ and NO₂ (Figure 1 and Table 2).

Studies have examined the potential biological mechanisms that help explain the effects of SO₂ and

Table 1 Summary statistics of daily air pollutants, weather condition and emergency hospital visits for hypertension in Beijing, 2007

	Minimum	25%	50%	75%	Maximum	Mean	SD
SO ₂ (µg/m ³)	6.0	15.0	26.0	64.0	247.5	47.3	48.6
NO ₂ (µg/m ³)	17.6	51.2	64.0	78.4	150.4	66.6	22.8
PM ₁₀ (µg/m ³)	15.0	96.0	140.0	184.0	600.0	149.3	85.7
Temperature (°C)	-6.1	3.7	14.5	25.0	30.7	14.1	10.7
Humidity (%)	15.0	37.0	54.0	74.0	97.0	54.2	20.9
EHV _s	0	2	4	5	19	4.1	0.1

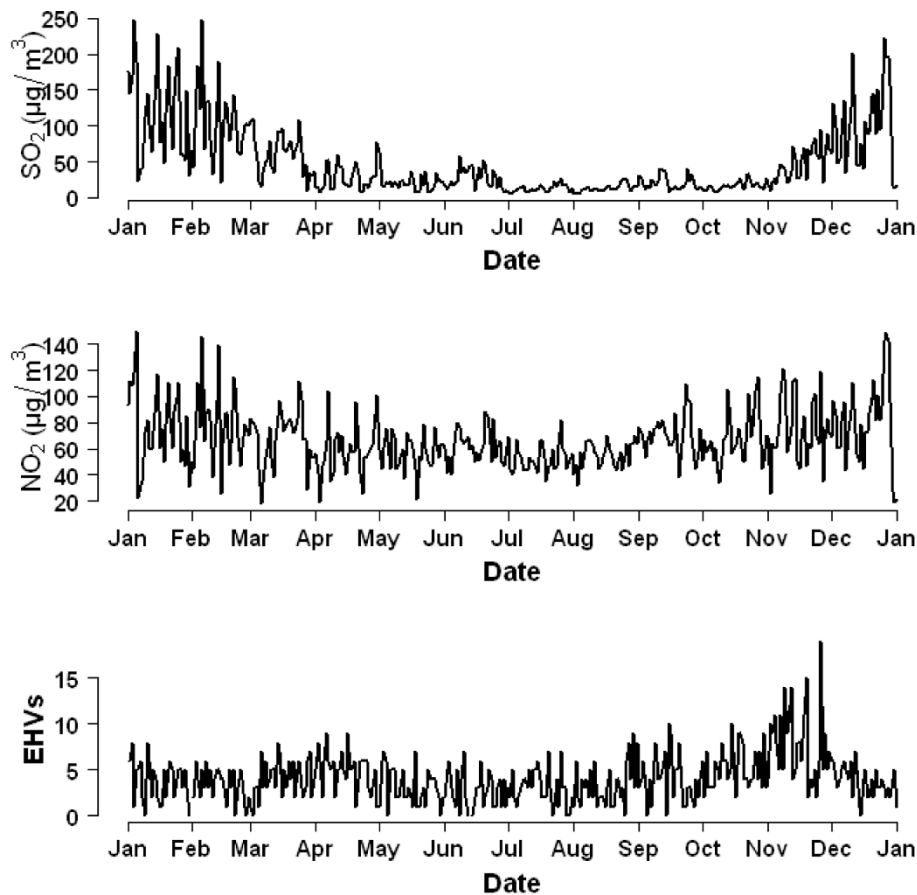


Figure 1 The time series of daily mean concentrations of gaseous air pollutants and number of emergency hospital visits for hypertension in Beijing during 2007.

NO₂ on the cardiovascular system. As ambient SO₂ concentrations rise, the SO₂ concentration in blood and other tissues of the body increase [30]. When SO₂ is inhaled the lipid peroxidation level in mice is raised. SO₂ at all tested concentrations significantly decreased activities of superoxide dismutase in mice of both sexes, as well as that of glutathione peroxidase from male mice [31].

Some studies suggest that elevated levels of NO₂ could increase the number of EHVs for cardiovascular disease [5,6]. Studies on the biological mechanism found that increased NO₂ is associated with increased plasma

fibrinogen, ventricular arrhythmia and ventricular tachycardia [32-34]. However, recently, Langrish et al. [35] found that inhalation of NO₂ did not impair vascular vasomotor or fibrinolytic function in man.

Previous studies found that gaseous air pollutants have adverse effects on heart rate variability (HRV) which reflects cardiac autonomic function. Min et al. [36] measured HRV among community residents in Korea to study the effects of PM₁₀, SO₂, and NO₂ on cardiac autonomic function, and found that exposure to PM₁₀, SO₂, and NO₂ resulted in reduced HRV, significant decreases in the standard deviation of the normal to normal interval (SDNN) and low frequency (LF) domain effect, and the effect was sustained for twelve hours. Routledge et al. [37] found that SO₂ exposure resulted in a significant reduction in HRV markers of cardiac vagal control at four hours in healthy people, but no changes were found in patients with stable angina. de Paula Santos et al. [19] observed that an inter-quartile range increase of 9.6 µg/m³ in level of SO₂ was negatively associated with SDNN of -7.93 ms (95% CI: -15.3, -0.6). Chan et al. [38] carried out a panel study to

Table 2 Spearman's correlations between daily air pollutants and weather conditions in Beijing 2007

	PM ₁₀	SO ₂	NO ₂	Temperature
SO ₂	0.46 ^a			
NO ₂	0.64 ^a	0.65 ^a		
Temperature	0.04	-0.68 ^a	-0.28 ^a	
Humidity	0.28 ^a	-0.12 ^a	0.25 ^a	0.21 ^a

^a P < 0.05.

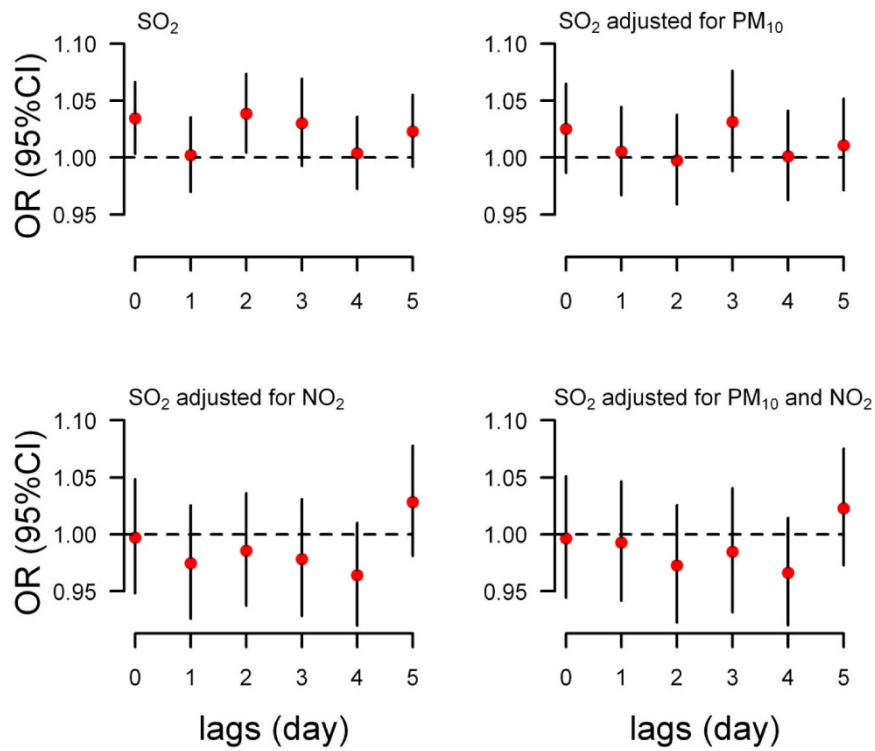


Figure 2 The association between a 10 $\mu\text{g}/\text{m}^3$ increase in SO_2 and daily emergency hospital visits for hypertension at lag days 0 to 5 in single pollutant and multiple pollutants models (time-stratified case-crossover controlling temperature and relative humidity).

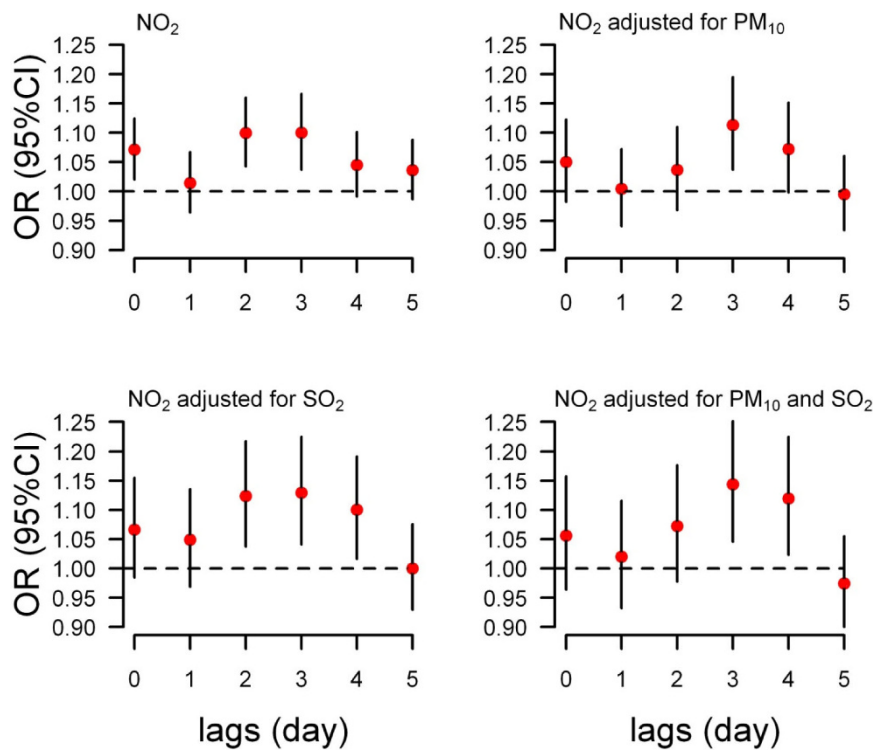


Figure 3 The association between a 10 $\mu\text{g}/\text{m}^3$ increase in NO_2 and daily emergency hospital visits for hypertension at lag days 0 to 5 in single pollutant and multiple pollutants models (time-stratified case-crossover controlling temperature and relative humidity).

examine the association between NO₂ and HRV in a susceptible population with coronary heart disease (CHD) or more than one major CHD risk factor, and found that an increase of 10 ppb in NO₂ was associated with 1.5-2.4% decreases in SDNN, and for each 10 ppb increase in NO₂ the LF was decreased by 2.2-2.5%.

Much research has been carried out to look at the influence of gaseous air pollution on mortality and morbidity for cardiovascular disease and shown that the increased concentrations of gaseous air pollution can impact on population health. Kan and Chen [39] applied a case-crossover to evaluate the relation between air pollution and daily mortality in Shanghai. The results showed that an increase of 10 µg/m³ in the levels of SO₂ and NO₂ had a relative risk of 1.017 (95% CI: 1.009-1.026) and 1.024 (95% CI: 1.011-1.036) for cardiovascular mortality. D'Ippoliti et al. [10] conducted a time-stratified case-crossover to explore the relationship between urban air pollutants and hospital admissions for acute myocardial infarction in Rome, and showed that each 10 µg/m³ increase of NO₂ was associated with a relative risk of 1.026 (95%CI: 1.002-1.052) for hospital admissions for acute myocardial infarction. Grazuleviciene et al. [40] carried out a population-based case-control study among men aged 25-64 years residing in Kaunas, to explore the relationship between long-term exposure to NO₂ and myocardial infarction. Results suggested that urban NO₂ pollution increased the risk of myocardial infarction.

This study has three strengths. Firstly, to our knowledge, it is the first epidemiological study which has specifically explored the relationship between gaseous air pollution and EHV for hypertension. Secondly, a relative large sample size was used, with a considerable

daily variance of exposure and outcomes. Finally, EHV are a good indicator of the acute effects of air pollution, because when people in China with cardiovascular disease feel uncomfortable, the first choice they usually make is to visit the hospital emergency department.

This study also has some limitations. The cases were only selected from one hospital. Although the patients lived near the hospital, it still cannot control their hospital selection. Outdoor average concentrations of PM₁₀, SO₂ and NO₂ were collected from fixed sites, but the data on individual exposure were unavailable. There might be misclassification bias for coding EHV, because the diagnosis of EHV for hypertension can never be 100% correct. Socioeconomic factors were not considered, as we could not get the patients' data on socioeconomics. The study was only conducted in the urban city of Beijing, and therefore the generalisability of the results is limited.

Conclusion

We found that elevated concentrations of gaseous air pollutants were associated with EHV for hypertension in Beijing, China. The findings provide additional information about the health effects of air pollution, and may have implications for planning local environmental protection and public health interventions.

Abbreviations

EHVs: emergency hospital visits; SBP: systolic blood pressure; DBP: diastolic blood pressure; ICD10: International Classification of Disease, tenth revision; SO₂: sulfur dioxide; NO₂: nitrogen dioxide; PM₁₀: particulate matter less than 10 µm in aerodynamic diameter; OR: odds ratio; CI: confidence interval; HRV: heart rate variability; SDNN: standard deviation of the normal to normal interval; LF: low frequency; CHD: coronary heart disease; SD: standard deviation;

Acknowledgements

We thank the Medical Record Department of Peking University Third Hospital, the Beijing Municipal Environmental Monitoring Center, and China Meteorological Data Sharing Service System for providing data. YMG is supported by the QUT Postgraduate Research Award (QUTPRA); SLT is supported by a NHMRC Research Fellowship (#290515); this study is funded by the National Natural Science Foundation of China (# 20637026).

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Authors' contributions

YMG conceived and coordinated the study, performed data analysis and drafted the manuscript; SLT and SSL contributed to study design, reviewed and edited the manuscript; AGB contributed to statistical analysis, reviewed and edited the manuscript; WWY and YSZ contributed to review and edit the manuscript. XCP provided air pollution information and health data, and edited the manuscript; All authors have read and approved the final manuscript.

Table 3 Odds ratios for daily emergency hospital visits for hypertension for a 10 µg/m³ increase in air pollutants (Results from a time-stratified case-crossover for single pollutant and multiple pollutants models) ^a

Air pollutants	OR	95% CI	
		lower	upper
SO ₂	1.037 ^b	1.004	1.071
+PM ₁₀	1.025	0.987	1.065
+NO ₂	0.997	0.949	1.048
+PM ₁₀ +NO ₂	0.997	0.945	1.051
NO ₂	1.101 ^b	1.038	1.168
+PM ₁₀	1.114 ^b	1.037	1.195
+SO ₂	1.130 ^b	1.041	1.225
+PM ₁₀ +SO ₂	1.144 ^b	1.046	1.251

^a ORs of SO₂ are at lag 0 day; ORs of NO₂ are at lag 3 day; OR: odds ratio; CI: confidence interval;

^b P < 0.05.

Competing interests

The authors declare that they have no competing interests.

Received: 10 August 2010 Accepted: 5 October 2010

Published: 5 October 2010

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doi:10.1186/1476-069X-9-57

Cite this article as: Guo et al.: Gaseous air pollution and emergency hospital visits for hypertension in Beijing, China: a time-stratified case-crossover study. *Environmental Health* 2010 **9**:57.