The relationship between particulate air pollution and emergency hospital visits for hypertension in Beijing, China

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Abstract

Background: a number of epidemiological studies have examined the adverse effect of air pollution on mortality and morbidity. Also, several studies have investigated the associations between air pollution and specific-cause diseases including arrhythmia, myocardial infarction, and heart failure. However, little is known about the relationship between air pollution and the onset of hypertension. **Objective:** to explore the risk effect of particulate matter air pollution on the emergency hospital visits (EHVs) for hypertension in Beijing, China. **Methods:** we gathered data on daily EHVs for hypertension, fine particulate matter less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$), particulate matter less than 10 μm in aerodynamic diameter (PM_{10}), sulfur dioxide, and nitrogen dioxide in Beijing, China during 2007. A time-stratified case–crossover design with distributed lag model was used to evaluate associations between ambient air pollutants and hypertension. Daily mean temperature and relative humidity were controlled in all models. **Results:** there were 1,491 EHVs for hypertension during the study period. In single pollutant models, an increase in 10 $\mu g/m^3$ in $PM_{2.5}$ and PM_{10} was associated with EHVs for hypertension with odds ratios (overall effect of five days) of 1.084 (95% confidence interval (CI): 1.028, 1.139) and 1.060% (95% CI: 1.020, 1.101), respectively. **Conclusion:** elevated levels of ambient particulate matters are associated with an increase in EHVs for hypertension in Beijing, China.

Key Words: Particulate air pollution; Emergency hospital visit; Hypertension; Time-stratified case–crossover;

Introduction

Air pollution is a very serious issue for human health, particularly in urban areas in developing countries. Outdoor particulate air pollution was estimated to be responsible for about 0.8 million premature deaths and 6.4 million years of lost life worldwide (Cohen et al., 2005). Recently, numerous epidemiological studies have examined the effect of air pollution on mortality, hospital admissions, and emergency hospital visits (EHVs) for cardiovascular diseases, respiratory diseases, and cardiopulmonary diseases (Guo et al., 2009; Halonen et al., 2009; Oudin et al., 2009; Simkhovich et al., 2009). Additionally, some studies have shown that the ambient air pollution increased the risks of cause-specific cardiac disease e.g. coronary disease (Kan et al., 2008; Simkhovich et al., 2009), arrhythmia (Chiu and Yang, 2009; Santos et al., 2008), myocardial infarction (D'Ippoliti et al., 2003), and heart failure (Lee et al., 2008; Wellenius et al., 2006). However, few data are available on the potential impact of particulate air pollution on acute events for hypertension.

Research conducted in Beijing, China shows that about 47% of people have hypertension, which is defined by systolic blood pressure greater than 140 mmHg and diastolic blood pressure greater than 90 mmHg (Xu et al., 2008). In the United States, the number of deaths caused by hypertension rose by 53% from 1991 to 2001 (Wexler, 2007). 91 percent of people with heart failure had preceding hypertension, and half of all patients who suffered a heart attack (and two thirds of those who had a first-time stroke) had blood pressure greater than systolic blood pressure 140 mmHg and diastolic blood pressure 90 mmHg (Wexler, 2007).

Human exposure, animal experiments and epidemiological studies have illustrated that particulate air pollution potentially raises blood pressure (Brook, 2005), even in healthy

74 individuals (Urch et al., 2005). At present, however, it is unknown whether particulate air
75 pollution induces acute hypertension events in persons with preexisting hypertension.
76 Therefore, we used a time-stratified case–crossover design to analyse the relationship
77 between ambient air pollution and EHVs for hypertension, and aimed to discover whether a
78 short-term increase in ambient air pollution is associated with the onset of hypertension.

79 **Materials and methods**

80 1. Data on emergency hospital visits

81 Data on EHVs for hypertension were gathered between Jan and Dec 2007 from the
82 Peking University Third Hospital, located in the northwest part of Beijing (Guo et al., 2009).
83 The information of each case includes: age, gender, occupation, diagnostic code, living
84 address and primary diagnosis. The primary diagnoses of daily EHVs were coded according
85 to the International Classification of Disease, 10th revision for hypertension (ICD10: I10).
86 The EHVs for hypertension were diagnosed by the visiting patients' symptoms, inquiries,
87 and medical inspections. Therefore, we expect the misclassification rate to be relatively
88 small. About 95% of visits for hypertension are diagnosed only with hypertension, while 5%
89 have accompanying heart failure, myocardial infarction or other diseases. The cases enrolled
90 in this study had lived in the resident areas around the hospital or in urban areas of Beijing.

91

92 2. Data on air pollution and weather condition

93 Data on particulate matter less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) was
94 monitored on the campus of Peking University, which is about four kilometres away from
95 the Peking University Third Hospital. Daily average concentration of $PM_{2.5}$ was transformed
96 from hourly values. Daily records of particulate matter less than 10 μm in aerodynamic
97 diameter (PM_{10}), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2) were obtained from the
98 Beijing Public Net for Environmental Protection of Beijing Municipal Environmental

Monitoring Center.

Information on daily mean temperature and relative humidity were obtained from the China Meteorological Data Sharing Service System during the study period.

3. Data analysis

In this study, the time-stratified case–crossover design was used to analyse the relationship between particulate air pollution and EHVs 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 can be used to explain the differences in the daily number of cases. The case–crossover design can control confounders related to individual characteristics, e.g. age, sex, and smoking. The secular trends and seasonal patterns in the disease and exposures can also be successfully controlled by case–crossover design. The cases and controls were matched by the day of the week to control any weekly patterns in hospital visits and pollution levels. The length of the time strata was 28 days, so each case had three matching control days. Simulations have shown that this approach gives unbiased estimates in the presence of strong seasonal confounding (Basu et al., 2005; Lee and Schwartz, 1999).

To examine the delayed effect of exposure to air pollution on the onset of hypertension, we used a polynomial distributed lag model (Armstrong, 2006; Schwartz, 2000). The reason for using the distributed lag model is that air pollution can affect not merely health events occurring on the same day, but on several subsequent days. Therefore, EHVs for hypertension today would depend on the "same day" effect of the current day's pollution levels and the lag effects of the previous days' pollution concentrations. The polynomial distributed lag model (PDL (q, d) with q lags and degree d), the most common approach to constrain the shape of the variation of the $[beta]_q$'s with lag number, was used to gain more

efficiency and more insight into the shape of the distributed effect of air pollution on EHVs for hypertension over time (Schwartz, 2000). We examined the lag effect of exposure to air pollution on EHVs for hypertension up to four days prior to the hospital visit, because there were no apparent effects of $PM_{2.5}$ and PM_{10} on EHVs for hypertension when the lags were more than five days. The polynomial smoothing with four degrees of freedom for $PM_{2.5}$ and PM_{10} was used in the models (Santos et al., 2008). The overall effects (over all five days) on EHVs for hypertension associated with a 10 $\mu g/m^3$ increase, as well as an inter-quartile range (IQR) increase, in $PM_{2.5}$ and PM_{10} were calculated in this study.

Daily mean temperature and relative humidity were controlled in all models (Braga et al., 2001). Single and multiple air pollutants models were used to examine the independent effects of air pollutants. Odds ratios (Ors) and confidence intervals (CIs) were calculated for each pollutant, and all statistical tests were two-sided. Values of $P<0.05$ were considered statistically significant, and spearman correlation coefficients were used to summarise the correlations between weather conditions and air pollutants. The "season" package of the R software (version 2.10.1) was used to fit the time-stratified case–crossover (Barnett, 2010), and the "dlnm" package was used to fit the polynomial distributed lag model in the time-stratified case–crossover design (Armstrong, 2006).

Results

During the study period, there were 1,491 EHVs for hypertension in the Peking University Third Hospital. The patients' average age was 61 years. 51 percent of the cases were aged <65 and 35.4% were males.

The descriptive statistics for air pollutants and weather conditions are shown in Table 1. The IQRs for $PM_{2.5}$ and PM_{10} were 76.3 $\mu g/m^3$ and 88.0 $\mu g/m^3$, respectively. The levels of PM_{10} and NO_2 were higher than the national secondary ambient air quality standard in China

(100 μg/m^3 and 40 μg/m^3 respectively), while the level of SO_2 did not surpass the national secondary ambient air quality standard in China (60 μg/m^3). The standard for $PM_{2.5}$ air pollution has not been set in China, but the mean concentration of $PM_{2.5}$ air pollution was higher than the ambient air quality standard in the United States (35 μg/m^3, National Ambient Air Quality Standards of United States).

Table 2 presents Spearman correlation coefficients between air pollutants and weather conditions. $PM_{2.5}$, SO_2, and NO_2 were positively correlated with each other, as well as PM_{10}, SO_2 and NO_2 ($P<0.05$). Temperature was positively correlated with $PM_{2.5}$ ($r=0.22$, $P<0.05$). A positive correlation was found between humidity and $PM_{2.5}$ ($r=0.34$, $P<0.05$) and PM_{10} ($r=0.28$, $P<0.05$).

Figure 1 shows the distributed lag structures of effects on EHVs for hypertension caused by a 10 μg/m^3 increase in $PM_{2.5}$ and PM_{10} using fourth-degree polynomials ranging from lag 0 to lag 4. Both $PM_{2.5}$ and PM_{10} were significantly associated with EHVs for hypertension on the two-day lag, even after adjusting for SO_2 and NO_2.

Table 3 presents the overall effects (over all five days) of air pollutants on EHVs for hypertension associated with a 10 μg/m^3 increase and an IQR increase in single and multiple pollutant models. In the single pollutant models, the overall effects on EHVs for hypertension associated with a 10 μg/m^3 increase in $PM_{2.5}$ and PM_{10} were 1.084 (95%CI: 1.028, 1.139) and 1.060 (95%CI: 1.015, 1.106), respectively. After controlling SO_2 and NO_2, the overall effects of both $PM_{2.5}$ and PM_{10} were lower than the single air pollutant models.

Figure 2 shows that the adverse effects were greatest for the highest concentrations of $PM_{2.5}$ and PM_{10}, and the peak in EHVs for hypertension was one day after exposure.

Discussion

In this study, the time-stratified case–crossover design with polynomial smoothing was

used to analyse the impact of particulate air pollution on EHVs for hypertension in Beijing, China. We found a positive relationship between particulate air pollution and EHVs for hypertension. The statistically significant two-day lag effects were found for both $PM_{2.5}$ and PM_{10}.

Because studies of air pollution effects on hypertension morbidity or specifically EHVs for hypertension are unusual, it is hard to compare results with previous research. However, some studies have found that increased concentrations of air pollution can elevate blood pressure. Johnson and Parker (2009) used data from a large, nation-wide survey in the United States in which subjects were 30 years or older. This study found that $PM_{2.5}$ was associated with a small elevated risk of hypertension. Auchincloss et al. (2008) examined cross-sectional associations between short-term ambient $PM_{2.5}$ and systolic and diastolic blood pressure, mean arterial pressure, and pulse pressure, and found that systolic blood pressure and pulse pressure were positively associated with ambient levels of $PM_{2.5}$ and the associations were stronger in the presence of roadway traffic. Brook et al. (2009) found that particulate matter was responsible for increasing diastolic blood pressure, probably by instigating an acute autonomic imbalance.

Several biologic mechanisms shown in previous studies also support the results in our study. Hazari et al. (2009) found that a single exposure to particulate matter or gaseous pollutant increases the risk of aconitine-induced cardiac arrhythmia in hypertensive rats. Bartoli et al. (2009) used dogs and concentrated ambient air particles to investigate the effect of ambient particles on systemic hemodynamics, and found that exposure to concentrated ambient air particles ranging from 94.1 to 1557.0 $\mu g/m^3$ increased systolic blood pressure by an average of 2.7 mmHg, diastolic blood pressure by 4.1 mmHg, mean arterial pressure by 3.7 mmHg, heart rate by 1.6 beats per minute, and decreased pulse pressure by 1.7mmHg. Sun et al. (2008) found that when rats were exposed to $PM_{2.5}$ or filtered air for 10 weeks, air

198 pollution exposure potentiates hypertension through reactive oxygen species-mediated
199 activation of Rho/ROCK. Schins et al. (2004) found that exposure to $PM_{2.5}$ was significantly
200 associated with endotoxin content, interleukin (IL)-8 and TNF alpha from the whole blood of
201 rats. Ito et al. (2008) used mRNA expression and correlation analyses to examine how $PM_{2.5}$
202 induced the oxidative stress (a key cause of cardiovascular disease), and found that
203 chemicals in concentrated ambient particles might be transmitted to the heart where they
204 induce oxidative stress and activate endothelin signaling, resulting in an increase in the blood
205 pressure. Other research has found associations between air pollution and acute arterial
206 vasoconstriction (Brook et al., 2002), C reactive protein (Pekkanen et al., 2000; Pope et al.,
207 2004; Schwartz, 2001), and heart rate variability (Cavallari et al., 2008; Fang et al., 2009).
208 Because the aconitine-induced cardiac arrhythmia, blood pressure, reactive oxygen
209 species-mediated activation, endotoxin, interleukin (IL)-8 and TNF alpha, arterial
210 vasoconstriction, and heart rate variability are directly or indirectly related to the function of
211 cardiovascular system, the increase in particle air pollution may induce the onset of
212 hypertension.

213 The results of our study suggested that exposure to $PM_{2.5}$ has a greater effect than PM_{10}
214 on EHVs for hypertension per 10 $\mu g/m^3$ increase, as well as an increase in IQR (Table 3).
215 Compared with PM_{10}, $PM_{2.5}$ can stay in the air for a longer time. $PM_{2.5}$ can also be breathed
216 into the respiratory tract and directly inhaled into the pulmonary alveoli from where blood
217 circulation can take $PM_{2.5}$ to the cerebral-vascular system. Besides, $PM_{2.5}$ has a larger
218 superficial area than PM_{10}, thus adsorbing more types of chemical elements than PM_{10}.
219 Therefore, $PM_{2.5}$ might be more harmful on population health than PM_{10} (Yang, 2003).

220 In the multiple pollutants models, the ORs of $PM_{2.5}$ and PM_{10} were lower than single
221 pollutant models. Previous research (Guo et al., 2009; Yang, 2008) on air pollution and
222 mortality and EHVs for cardiovascular diseases in Beijing found similar associations to

those shown here. These results may suggest that air pollutants share health effects. The lower effect of PM pollution on EHVs for hypertension in the multiple pollutants models may be caused by their statistical co-linear effect.

The time stratified case–crossover design has the ability to control many confounders by design, such as age, gender, smoking, drinking, living custom, and so on. By matching on the day of the week, we avoid confounding due to generally higher pollutant levels on weekdays. The case–crossover also successfully controls seasonal patterns and long-term trends (Basu et al., 2005; Lee and Schwartz, 1999; Navidi, 1998; Neas et al., 1999). We checked the model sensitivity by moving monthly data, and found similar outcomes. Although our sample size is not large, the results already show a statistically significant relationship between particulate air pollution and EHVs for hypertension. Therefore, the results are reliable in this study.

The strength of this study is: EHVs reflect the association between air pollution and acute events of diseases more accurately than hospital admission, because when people in China with cardiovascular diseases feel uncomfortable, the first choice they usually make is to visit the hospital emergency department.

This study also has some limitations. The health data was collected from only one hospital and data on $PM_{2.5}$ was gathered from only one monitor station. Therefore, the selecting confounding might exist. Ambient air pollution was used rather than individual exposure. The diagnosis for EHVs for hypertension could not be 100% correct. It might cause the misclassification confound.

Conclusion

In this case–crossover study, we found that elevated concentrations of ambient particulate matter air pollutants were associated with an increase in the EHVs for

hypertension in Beijing during 2007. The findings provide additional information about the health effects of air pollution in Beijing, China, and may have implications for local environmental and public health.

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Abbreviations: EHVs: emergency hospital visits; ICD10: International Classification of Disease, 10th revision; $PM_{2.5}$: particulate matter less than 2.5 μm in aerodynamic diameter; SO_2: sulfur dioxide; NO_2: nitrogen dioxide; PM_{10}: particulate matter less than 10 μm in aerodynamic diameter; OR: odds ratio; CI: confidence interval; T: temperature; RH: relative humidity; SD: standard deviation;

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386 **Tables:**

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388 Table 1：Summary of air pollutants and weather conditions in the study period

	Frequency distribution					Mean	SD
	Minimum	25%	50%	75%	Maximum		
$PM_{2.5}$ (μg/m^3)	12.0	54.8	81.2	131.1	328.0	102.4	65.5
PM_{10} (μg/m^3)	15.0	96.0	140.0	184.0	600.0	149.3	85.7
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	78.0	150.4	66.6	22.8
Temperature (°C)	–6.1	3.7	14.5	25.0	30.7	14.1	10.7
Relative humidity (%)	15.0	37.0	54.0	74.0	97.0	54.2	20.9

389 **Abbreviations:** $PM_{2.5}$: particulate matter less than 2.5 μm in aerodynamic diameter; PM_{10}:
390 particulate matter less than 10 μm in aerodynamic diameter; SO_2: sulfur dioxide; NO_2:
391 nitrogen dioxide; SD: standard deviation;

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Table 2: Spearman correlation coefficients between air pollutants and weather conditions in the study period

	$PM_{2.5}$	PM_{10}	SO_2	NO_2	Temperature
PM_{10}	0.73*				
SO_2	0.24*	0.46*			
NO_2	0.38*	0.64*	0.65*		
Temperature	0.22*	0.04	–0.67*	–0.28*	
Relative humidity	0.34*	0.28*	–0.12*	0.25*	0.21*

*P<0.05

Abbreviations: $PM_{2.5}$: particulate matter less than 2.5 μm in aerodynamic diameter; PM_{10}: particulate matter less than 10 μm in aerodynamic diameter; SO_2: sulfur dioxide; NO_2: nitrogen dioxide;

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426 Table 3: The overall effects (over all five days) of air pollutants and emergency hospital

427 visits for hypertension (Results from single pollutant and multiple pollutants models)

Air pollutants	OR (95%*CI*)	
	10 μg/m^3	IQR[a]
$PM_{2.5}$	1.084 (1.028, 1.139)*	1.846(1.424, 2.269)*
+ SO_2	1.080 (1.024, 1.136)*	1.796 (1.368, 2.223)*
+ NO_2	1.053 (0.992, 1.109)	1.454 (1.008, 1.900)*
+ SO_2+ NO_2	1.035 (0.976, 1.094)	1.297 (0.847, 1.747)
PM_{10}	1.060 (1.020, 1.101)*	1.676 (1.316, 2.036)*
+ SO_2	1.060 (1.015, 1.106)*	1.677 (1.273, 2.080)*
+ NO_2	1.020 (0.968, 1.073)	1.195 (0.734, 1.656)
+ SO_2+ NO_2	1.010 (0.957, 1.063)	1.089 (0.623, 1.554)

428 *P<0.05; [a] IQRs for $PM_{2.5}$ and PM_{10} were 76.3 μg/m^3 and 88.0 μg/m^3, respectively;

429 **Abbreviations:** $PM_{2.5}$: particulate matter less than 2.5 μm in aerodynamic diameter; PM_{10}:

430 particulate matter less than 10 μm in aerodynamic diameter; SO_2: sulfur dioxide; NO_2:

431 nitrogen dioxide; OR: odds ratio; CI: confidence interval; IQR: Inter-quartile range;

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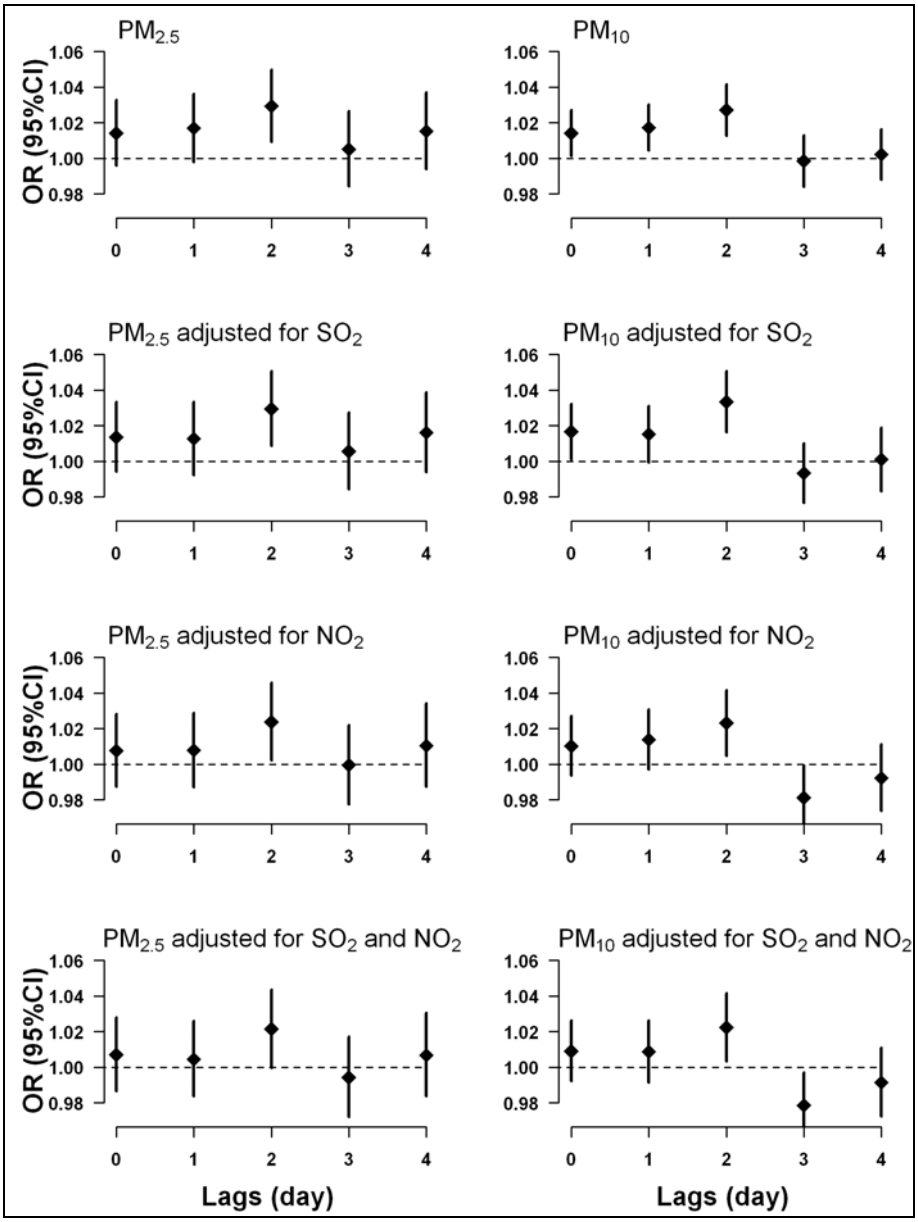

Figure 1: Association between a 10 μg/m^3 increase in particulate air pollution and emergency hospital visits for hypertension over five days (in single pollutant models and multiple pollutants models). Diamonds show the mean odds ratios, and the vertical lines are the 95% confidence intervals.

Abbreviations: $PM_{2.5}$: particulate matter less than 2.5 μm in aerodynamic diameter; PM_{10}: particulate matter less than 10 μm in aerodynamic diameter; OR: odds ratio; CI: confidence interval;

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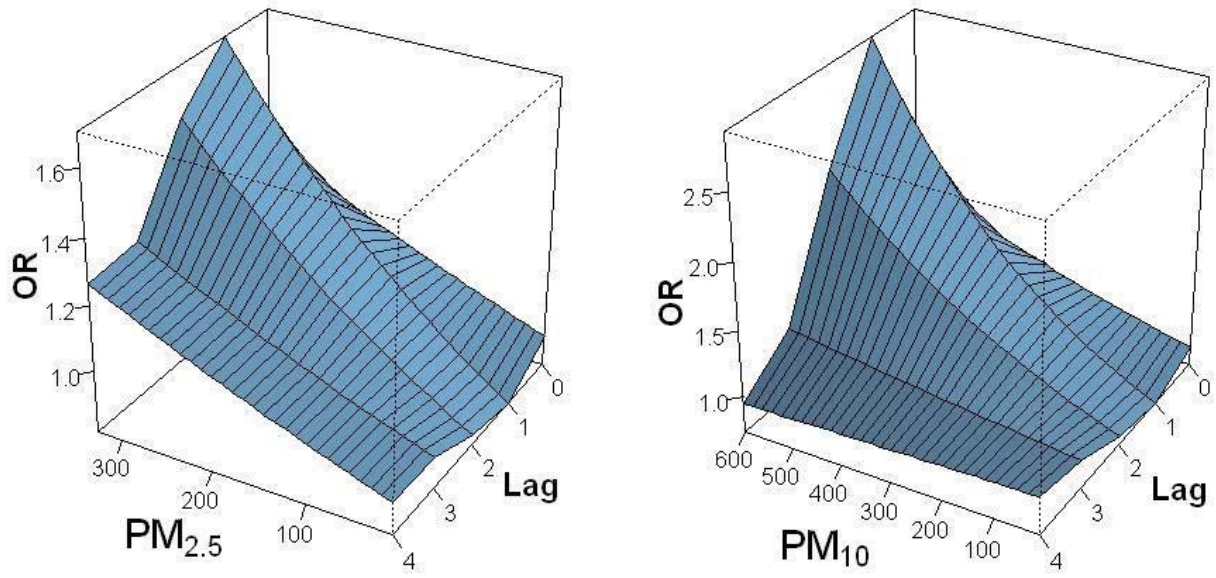

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451 Figure 2: Association between particulate air pollution and emergency hospital visits for
452 hypertension at lags from 0 to 4 days (in the single pollutant models).

453 **Abbreviations:** $PM_{2.5}$: particulate matter less than 2.5 μm in aerodynamic diameter; PM_{10}:
454 particulate matter less than 10 μm in aerodynamic diameter; OR: odds ratio;

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