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Short-Term Exposure to Air Pollution Associated with an Increased Risk of ST-Elevation and Non-ST-Elevation Myocardial Infarction Hospital Admissions: A Case-Crossover Study from Beijing (2013–2019), China

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Abstract: While air pollution is known as a risk factor for acute myocardial infarction (AMI) incidence, its impact on AMI subtypes—ST-elevation (STEMI) and non-ST-elevation myocardial infarction (NSTEMI)—remains incompletely understood. This study analyzed 149,632 AMI hospital admissions (70,730 STEMI and 69,594 NSTEM) in Beijing, China, from 2013 to 2019 using a time-stratified case-crossover design to evaluate the association between daily concentrations of six air pollutants ($PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3) and daily hospital admissions for total AMI, STEMI, and NSTEMI. Elevated levels of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were significantly associated with increased admission risk for total AMI, STEMI, and NSTEMI, with the strongest lag effects observed at lag0 for STEMI and at lag1 for NSTEMI. Subgroup analyses showed enhanced effects of $PM_{2.5}$, SO_2, and NO_2 for total AMI and SO_2 for NSTEMI among individuals with asthma. Additionally, a stronger effect of PM_{10} on STEMI was observed among individuals with stroke. These findings demonstrate that air pollutants differentially impact AMI subtypes through distinct temporal patterns and population vulnerabilities, underscoring the necessity of incorporating AMI subtype classification and individual susceptibility factors in environmental health risk assessments and related public health policies.

Keywords: air pollution; acute myocardial infarction; ST-elevation myocardial infarction; non-ST-elevation myocardial infarction; hospital admission; case-crossover

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Academic Editor: Daniele Contini

Received: 22 April 2025
Revised: 31 May 2025
Accepted: 10 June 2025
Published: 13 June 2025

Citation: Zhao, Y.; Chen, Y.; Liu, Y.; Tang, S.; Han, Y.; Fu, J.; Chang, Z.; Zhao, X.; Zhuang, Y.; Lei, J.; et al. Short-Term Exposure to Air Pollution Associated with an Increased Risk of ST-Elevation and Non-ST-Elevation Myocardial Infarction Hospital Admissions: A Case-Crossover Study from Beijing (2013–2019), China. *Atmosphere* **2025**, *16*, 715. https://doi.org/10.3390/atmos16060715

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1. Introduction

Cardiovascular disease (CVD), including acute myocardial infarction (AMI), is the leading cause of mortality and disability worldwide [1]. Epidemiological studies have indicated that short-term exposure to ambient air pollutants is associated with an increased risk of CVD morbidity and mortality [2–4]. Furthermore, there seems to be no safe threshold for the effects of air pollution on humans, as CVD risk can rise even at low pollutant concentrations. Therefore, mitigating and controlling the adverse impacts of air pollution on CVD are essential.

As two subtypes of AMI, ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI) exhibit some differences in their pathogenesis. Several studies showed that air pollution (primarily fine particulate matter) has different effects on the incidence of STEMI and NSTEMI [5–7], which might indicate potential mechanisms of air pollution triggering AMI. Considering the varying effects of air pollution on different AMI subtypes, subtype-specific analyses are essential for a more precise evaluation of air pollution's effects on AMI. However, most current research on air pollution and AMI either has not distinguished between AMI subtypes or has focused solely on STEMI [8,9]. There is limited research on the differences in air pollutants' effects on STEMI and NSTEMI, with most studies primarily examining particulate matter [5,6,10–12]. Therefore, further investigation into the impacts of particulate matter and gaseous pollutants on the incidence of different AMI subtypes is necessary.

Some studies suggested that certain demographic and health factors, such as age, sex, race, comorbidities (such as COPD, diabetes), and a history of CVD, may increase susceptibility to the adverse effects of air pollution on cardiovascular events [13–17]. Meanwhile, few studies have investigated the difference in susceptibility to air pollution's effects on different AMI subtypes among populations with varying sex, age, comorbidities, and CVD history. This highlights the need for further research to explore these disparities.

In our research, we examined the association between short-term exposure to six air pollutants [particulate matter < 2.5 μm in diameter ($PM_{2.5}$), particulate matter < 10 μm in diameter (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), and ozone (O_3)] and daily hospital admissions for total AMI, STEMI, and NSTEMI among permanent residents in Beijing, China. Subgroup analyses were also conducted by age, sex, comorbidities, and CVD history to identify susceptible populations.

2. Materials and Methods

2.1. Health Data Collection

Beijing (39°56′ N, 116°20′ E), the capital of China, is located in the northern China region and was chosen as our study region. This study encompassed all individuals aged 20 years and above who were permanent residents of Beijing and admitted to hospitals with a primary diagnosis of AMI (ICD-10, I21–I22) between 1 January 2013 and 31 December 2019. The Beijing Municipal Health Commission Information Center provided hospital admission data, which were extracted from the front page of the hospitalization medical records of all public and private hospitals in Beijing, including the patients' sex, age, current address, registration address, hospital admission date, diagnoses (coded by ICD-10), etc. To protect patient privacy, all population data were collected anonymously.

The subtype of AMI was categorized into STEMI (I21.0–I21.3), NSTEMI (I21.4), and unknown type. According to the patients' diagnoses, we collected information on comorbidities and previous cardiovascular diseases, which included asthma (J45–J46), chronic obstructive pulmonary disease (COPD) (J44), hypertension (I10–I15), diabetes (E10–E14), chronic kidney disease (CKD) (N18−N19), old myocardial infarction (OMI) (I25.2), previous percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) history (Z95.1, Z95.5, Z98.84), and stroke (I60–I64, I69).

2.2. Environmental Data Collection

Hourly concentrations of six air pollutants ($PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3) were obtained from the China National Environmental Monitoring Centre's 12 outdoor monitoring stations in Beijing [18]. Daily 24 h average concentrations of pollutants were calculated as exposure for each pollutant. Meteorological data, including daily mean temperature (Tmean), relative humidity (RH), and air pressure (AP), were also collected from

the Beijing Meteorological Service to control for the effects of meteorological conditions [9]. Influenza surveillance data were obtained from the Chinese National Influenza Center (CNIC) [https://ivdc.chinacdc.cn/cnic/ (accessed on 28 May 2021)] and adjusted for the potential confounding effects of influenza epidemics [19,20]. More specific information about the influenza epidemics can be found in our previously published articles [21]. Data on public holidays (e.g., New Year's Day, Spring Festival Holiday, National Day, etc.) and heating time were collected from open government news.

2.3. Study Design

The case-crossover (CCO) design, superior in controlling time-invariant confounders, is a prevalent method for investigating the health outcomes of acute air pollution exposure [22]. In our study, a time-stratified case-crossover design was utilized to evaluate the association between the daily concentrations of six air pollutants and the risk of hospital admission for AMI among permanent residents of Beijing. The days of AMI admissions were matched to the same day of the week within the same month and year. For instance, for the case day 1 January 2013 (Tuesday), control days were all other Tuesdays in January 2013 (8th, 15th, 22nd, 29th). Case and control days were compared for AMI admission counts and pollutant levels. This time-stratified approach establishes mutually non-overlapping strata defined by year, month, and day of the week, thereby controlling for both seasonal variations and weekday effects while preserving the self-matched nature of the case-crossover design [22,23].

2.4. Statistical Analyses

Continuous variables are shown as mean ± standard deviation (SD). Spearman correlation coefficients (*r*) were calculated to explore the correlations between air pollutants and meteorological factors.

The conditional Poisson regression generalized linear model (GLM), which could adjust for overdispersion and auto-correlation [23,24], was built to evaluate the association between short-term air pollution exposure and daily hospital admissions for total AMI, STEMI, and NSTEMI. The best-fit model was selected by sequentially adjusting all meteorological factors, influenza epidemics, and public holidays in the basic model. The final model, determined by the minimum Akaike information criterion for the quasi-Poisson (Q-AIC) value [25], included daily mean temperature, air pressure, public holidays, and heating as follows:

$$\begin{aligned} \text{Log}[E(Y_t)] = \beta Z_t + \text{ns}(\text{Tmean lag06}, 4) + \text{ns}(\text{AP lag06}, 4) + \text{factor}(\text{holiday}) \\ + \text{factor}(\text{heating}) + \text{factor}(\text{stratum}) + \text{intercept} \end{aligned} \quad (1)$$

where $E(Y_t)$ represents the number of hospital admissions for AMI on day t; Z_t indicates the daily average level of the air pollutant on day t; β is the vector of coefficients for Z_t, indicating the change in $\log[E(Y_t)]$ per unit change in the pollutant concentration on day t; Tmean lag06 and AP lag06 indicate the 7-day averages of daily mean temperature and air pressure on the same day and the previous 6 days; ns(, 4) refers to a natural spline with 4 degrees of freedom (dfs) to control for the nonlinearity of temperature and air pressure; factor(holiday) and factor(heating) indicate classification variables that adjust for public holidays and heating, respectively; factor(stratum) indicates time stratification; and intercept is the constant term.

The lag effect of air pollution on AMI admissions was investigated using single-day lags (from lag0 to lag5) and multi-day moving average lags (from lag01 to lag05). The lag effect with the strongest association (hereafter referred to as the strongest effect) was chosen as the main exposure for each air pollutant. Specifically, if more than half of the

relative risks (RRs) for an air pollutant were greater than 1, the lag with the maximum RR was selected as the strongest lag effect; if the majority of RRs were below 1 and statistically significant, the lag with the minimum or significant RR was selected.

Subgroup analyses were performed according to the following: (1) sex—male vs. female; (2) age—<65 years vs. ≥65 years; (3) comorbidities and previous history of CVDs—with or without asthma, COPD, hypertension, diabetes, CKD, OMI, previous PCI/CABG history, and stroke. In subgroup analyses, only the strongest effects in the single-day lag model were evaluated. The statistical differences between effect estimates for subgroup analyses were examined by a Z test [26].

2.5. Sensitivity Analysis

To account for the interaction effect between air pollutants, two-pollutant models were constructed to test the robustness of a pollutant's strongest effects in single-pollutant models, except when two pollutants were highly correlated (Spearman correlation coefficient > 0.7) [27]. Additionally, we varied the degrees of freedom (dfs) of the natural cubic spline function from 3 to 6 for meteorological factors (temperature and air pressure) and adjusted the lag effects of meteorological factors for lag0 and different multi-day moving average lags (lag01, lag03) to assess the robustness of our findings. Time-series analysis using generalized additive models (GAMs) was also applied for sensitivity analysis (see Supplementary Materials).

Statistical analyses were performed using the "glm", "mgcv", and "splines" packages in R statistical software (version 4.1.1). Results are presented as the relative risks (RRs) with 95% confidence intervals (CIs) of daily AMI hospital admissions associated with a per 10 μg/m^3 increase in $PM_{2.5}$, PM_{10}, SO_2, NO_2, and O_3 concentrations and a per 1 mg/m^3 increase in CO concentration, calculated as

$$RR = e^{\beta * increment} \quad (2)$$

where e is the base of the natural logarithm; β represents the regression coefficient of the air pollutant in Formula (1); and increment indicates the magnitude of pollutant concentration increase as mentioned above.

Statistical significance was defined as a two-tailed p-value < 0.05.

3. Results

During the study period, a total of 149,632 patients were admitted for AMI in Beijing, including 70,730 STEMI, 69,594 NSTEMI, and 9308 patients with an unspecific subtype of AMI. Table 1 presents the demographic and clinical data of the patients. The patients were predominantly male (69.31% for total AMI, 55.65% for STEMI, and 64.66% for NSTEMI) and aged ≥ 65 years (53.73% for total AMI and 61.36% for NSTEMI), while patients with STEMI were predominantly younger (55.6% aged < 65 years). Approximately half of the patients had hypertension (50.58% for total AMI, 48.4% for STEMI, and 53.73% for NSTEMI), followed by diabetes (26.3% for total AMI, 25.26% for STEMI, and 27.65% for NSTEMI). Compared with patients admitted for STEMI, the prevalence of comorbidities and CVD history was higher in those admitted for NSTEMI.

Table 1. Demographic and clinical data of patients admitted for total AMI, STEMI, and NSTEMI in Beijing from 2013 to 2019.

	Total AMI	STEMI	NSTEMI
Total, *n* (mean ± SD)	149,632 (59 ± 14)	70,730 (28 ± 7)	69,594 (27 ± 9)
Age, n (%)			
<65	69,231 (46.27)	39,359 (55.65)	26,893 (38.64)
≥65	80,401 (53.73)	31,371 (44.35)	42,701 (61.36)
Gender, *n* (%)			
Male	103,706 (69.31)	52,870 (74.75)	45,002 (64.66)
Female	45,926 (30.69)	17,860 (25.25)	24,592 (35.34)
Comorbidities, *n* (%)			
Asthma	970 (0.65)	413 (0.58)	482 (0.69)
COPD	2675 (1.79)	900 (1.27)	1490 (2.14)
Hypertension	75,687 (50.58)	34,234 (48.40)	37,394 (53.73)
Diabetes	39,360 (26.30)	17,864 (25.26)	19,245 (27.65)
CKD	8850 (5.91)	2768 (3.91)	5182 (7.45)
CVD history, *n* (%)			
OMI	17,705 (11.83)	5395 (7.63)	11,285 (16.22)
PCI/CABG history	19,259 (12.87)	7544 (10.67)	10,697 (15.37)
Stroke	15,250 (10.19)	6015 (8.50)	7786 (11.19)

AMI: acute myocardial infarction; STEMI: ST-segment-elevation myocardial infarction; NSTEMI: non-ST-segment-elevation myocardial infarction; SD: standard deviation; COPD: chronic obstructive pulmonary disease; CKD: chronic kidney disease; CVD: cardiovascular disease; OMI: old myocardial infarction; PCI/CABG: percutaneous coronary intervention/coronary artery bypass grafting.

Table 2 summarizes the descriptive statistics of air pollutant concentrations and meteorological factors. During the study period, the daily mean concentrations of $PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3 were 67.61 μg/m^3, 97.91 μg/m^3, 12.34 μg/m^3, 46.06 μg/m^3, 1.07 mg/m^3, and 67.87 μg/m^3, respectively. For more than half of the study period, the temperature was below 15.2 °C and the humidity was less than 52%.

Table 2. Descriptive statistics of air pollutants and meteorological factors in Beijing from 2013 to 2019.

	Mean ± SD	Min	P25	P50	P75	Max	IQR
$PM_{2.5}$ (μg/m^3)	67.61 ± 60.81	3.88	25.34	49.69	88.67	475.43	63.33
PM_{10} (μg/m^3)	97.91 ± 71.07	7.08	49.92	80.5	124.15	956.13	74.23
SO_2 (μg/m^3)	12.34 ± 16.05	1.94	3.25	6.28	13.55	138.37	10.30
NO_2 (μg/m^3)	46.06 ± 22.82	6.68	29.88	40.92	56.79	156.08	26.91
CO (mg/m^3)	1.07 ± 0.85	0.19	0.56	0.84	1.23	8.04	0.67
O_3 (μg/m^3)	67.87 ± 49.76	2.18	32.09	57.86	90.41	331.08	58.32
Tmean (°C)	13.78 ± 11.23	−14.3	2.9	15.2	24.2	32.6	-
RH (%)	51.45 ± 19.87	8	35	52	67	99	-
AP (hPa)	1012.9 ± 10.2	990	1004.2	1013	1021.1	1040	-

SD: standard deviation; Min: minimum; Max: maximum; P25: 25th percentile; P50: 50th percentile; P75: 75th percentile; IQR: interquartile range; Tmean: daily mean temperature; RH: relative humidity; AP: air pressure.

Figure 1 shows the Spearman correlations between air pollutants and meteorological factors. The concentrations of $PM_{2.5,}$ PM_{10}, SO_2, NO_2, and CO were positively correlated with each other. The concentrations of $PM_{2.5}$ and PM_{10} (r = 0.88) and $PM_{2.5}$ and CO (r = 0.87) showed strong positive correlations. In contrast, the concentrations of O_3 were negatively correlated with the concentrations of other pollutants (r = −0.11 to −0.48) and showed strong correlation with daily average temperature (r = 0.72) and air pressure (r = −0.65).

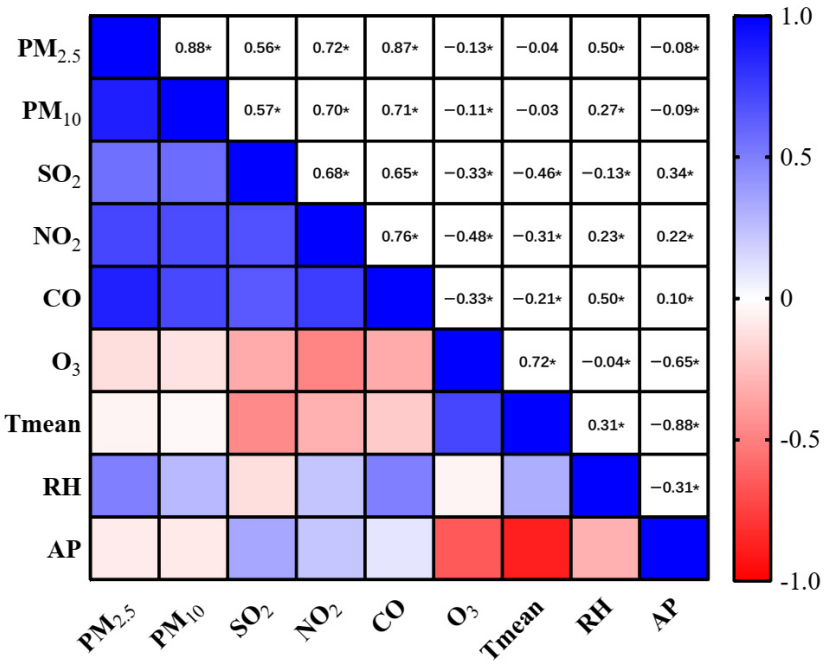


Figure 1. Spearman correlations between air pollutants and meteorological factors in Beijing from 2013 to 2019. Note: * indicates p-value < 0.05; Tmean: daily mean temperature; RH: relative humidity; AP: air pressure.

Figure 2 presents associations between air pollutants and hospital admissions for total AMI, STEMI, and NSTEMI on different lag days in the single-pollutant model. The strongest effects of each pollutant in the single- and multi-day lag models are shown in Table 3.

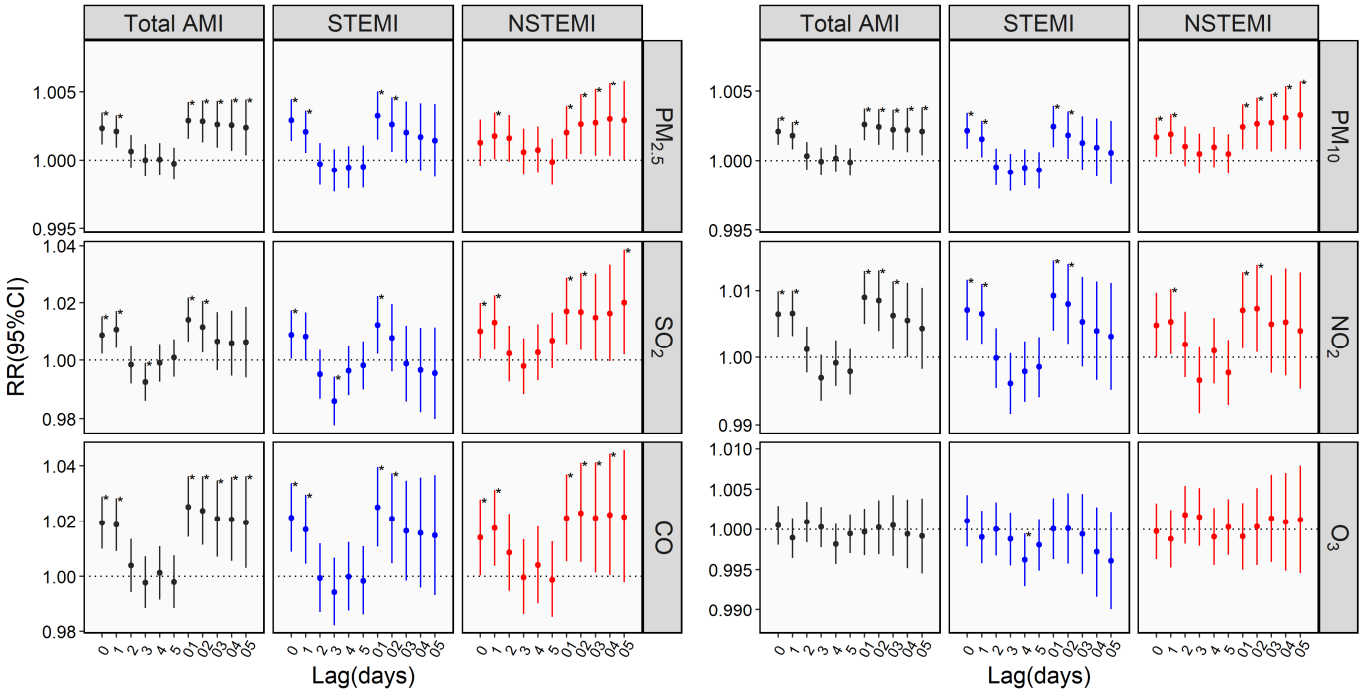


Figure 2. Associations between air pollutants and hospital admissions for total AMI, STEMI, and NSTEMI in the single-pollutant model. Relative risks (RRs) are for an increment of 10 μg/m^3 for $PM_{2.5}$, PM_{10}, SO_2, NO_2, and O_3 concentrations and 1 mg/m^3 for CO concentration. Note: * indicates $p < 0.05$.

Table 3. The strongest effects of air pollutants on hospital admissions for total AMI, STEMI, and NSTEMI in single- and multi-day lag models.

		Total AMI		**STEMI**		**NSTEMI**
$PM_{2.5}$	lag0	1.002 (1.001, 1.003) *	lag0	1.003 (1.001, 1.004) *	lag1	1.002 (1.000, 1.003) *
	lag01	1.003 (1.002, 1.004) *	lag01	1.003 (1.002, 1.005) *	lag04	1.003 (1.000, 1.006) *
PM_{10}	lag0	1.002 (1.001, 1.003) *	lag0	1.002 (1.001, 1.003) *	lag1	1.002 (1.000, 1.003) *
	lag01	1.003 (1.001, 1.004) *	lag01	1.002 (1.001, 1.004) *	lag05	1.003 (1.001, 1.006) *
SO_2	lag1	1.011 (1.004, 1.017) *	lag0	1.009 (1.000, 1.017) *	lag1	1.013 (1.004, 1.023) *
	lag01	1.014 (1.006, 1.022) *	lag01	1.012 (1.002, 1.022) *	lag05	1.020 (1.002, 1.039) *
NO_2	lag1	1.007 (1.003, 1.010) *	lag0	1.007 (1.003, 1.012) *	lag1	1.005 (1.000, 1.010) *
	lag01	1.009 (1.005, 1.013) *	lag01	1.009 (1.004, 1.015) *	lag02	1.007 (1.001, 1.014) *
CO	lag0	1.019 (1.010, 1.029) *	lag0	1.021 (1.009, 1.034) *	lag1	1.017 (1.004, 1.031) *
	lag01	1.025 (1.014, 1.036) *	lag01	1.025 (1.011, 1.040) *	lag02	1.023 (1.005, 1.041) *
O_3	lag2	1.001 (0.998, 1.003)	lag4	0.996 (0.993, 0.999) *	lag2	1.002 (0.998, 1.005)
	lag03	1.000 (0.997, 1.004)	lag05	0.996 (0.990, 1.002)	lag03	1.001 (0.996, 1.007)

* indicates $p < 0.05$. The effect estimates are presented as the relative risks (RRs) with 95% confidential intervals (CIs) of hospital admissions for total AMI, STEMI, and NSTEMI per 10 $\mu g/m^3$ increase in $PM_{2.5}$, PM_{10}, SO_2, NO_2, and O_3 concentrations and per 1 mg/m^3 increase in CO concentration.

In the single-pollutant model, higher levels of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were associated with increased risk of hospital admission for total AMI as well as STEMI and NSTEMI. According to the aforementioned definition of the strongest effect, for total AMI, the strongest effects were observed at lag0 for $PM_{2.5}$, PM_{10}, and CO and at lag1 for SO_2 and NO_2 in the single-day lag model. In the multi-day moving average lag model, the strongest effects for the above five pollutants were observed at lag01. For STEMI, the strongest effects for $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were observed at lag0 and lag01 in the single- and multi-day lag models, respectively. The effects of these five pollutants gradually decreased from lag02 to lag05, and there was no statistically significant effect on lag03 and later, which means that the lag effect lasted for a maximum of 3 days. For NSTEMI, the strongest effects for $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were observed at lag1 in the single-day lag model. In the multi-day lag model, the strongest effects for these five pollutants were observed at lag04, lag05, lag05, lag02, and lag02, respectively.

A higher concentration of O_3 at lag4 was significantly associated with a reduced risk of STEMI hospital admission (RR = 0.996 per 10 $\mu g/m^3$, 95%CI: 0.993–0.999), while O_3 showed no significant association with total AMI or NSTEMI. According to the previous definition, the strongest effect of O_3 was selected at lag2 for total AMI, lag4 for STEMI, and lag2 for NSTEMI.

Figure 3 and Tables S1–S10 show the associations of total AMI, STEMI, and NSTEMI hospital admissions with six air pollutants in different subgroups and the results of Z tests for between-group comparisons (presented as P_z). The effects of SO_2, NO_2, and CO were stronger in the elderly (aged $\geq$ 65 years) for total AMI and NSTEMI, while they were stronger in younger individuals (aged < 65 years) for STEMI. The effects of these three pollutants on total AMI and STEMI hospital admissions were stronger in women than in men. However, the differences between sex and age subgroups did not reach statistical significance ($P_z > 0.05$). The effects of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO on all types of AMI hospital admission were almost higher in the population with asthma or stroke than in those without asthma and stroke. Specifically, the effects of $PM_{2.5}$, SO_2, and NO_2 on total AMI and the effect of SO_2 on NSTEMI were significantly stronger in the asthma group than in the non-asthma group ($P_z < 0.05$). Similarly, the effect of PM_{10} on STEMI was significantly stronger in the stroke group than in the non-stroke group ($P_z < 0.05$). O_3 showed a significant protective effect in the aged < 65 group (RR = 0.993 per 10 $\mu g/m^3$, 95%CI: 0.989–0.998) and those without COPD (RR = 0.996 per 10 $\mu g/m^3$, 95%CI:

0.993–0.999), which was significantly different from those in individuals aged ≥ 65 years (RR = 1.000 per 10 μg/m^3, 95%CI: 0.995–1.005) and those with COPD (RR = 1.023 per 10 μg/m^3, 95%CI: 0.997–1.050) ($P_z < 0.05$). No significant difference was observed in the effects of all air pollutants between individuals with and without hypertension, diabetes, CKD, OMI, or a history of PCI/CABG ($P_z > 0.05$).

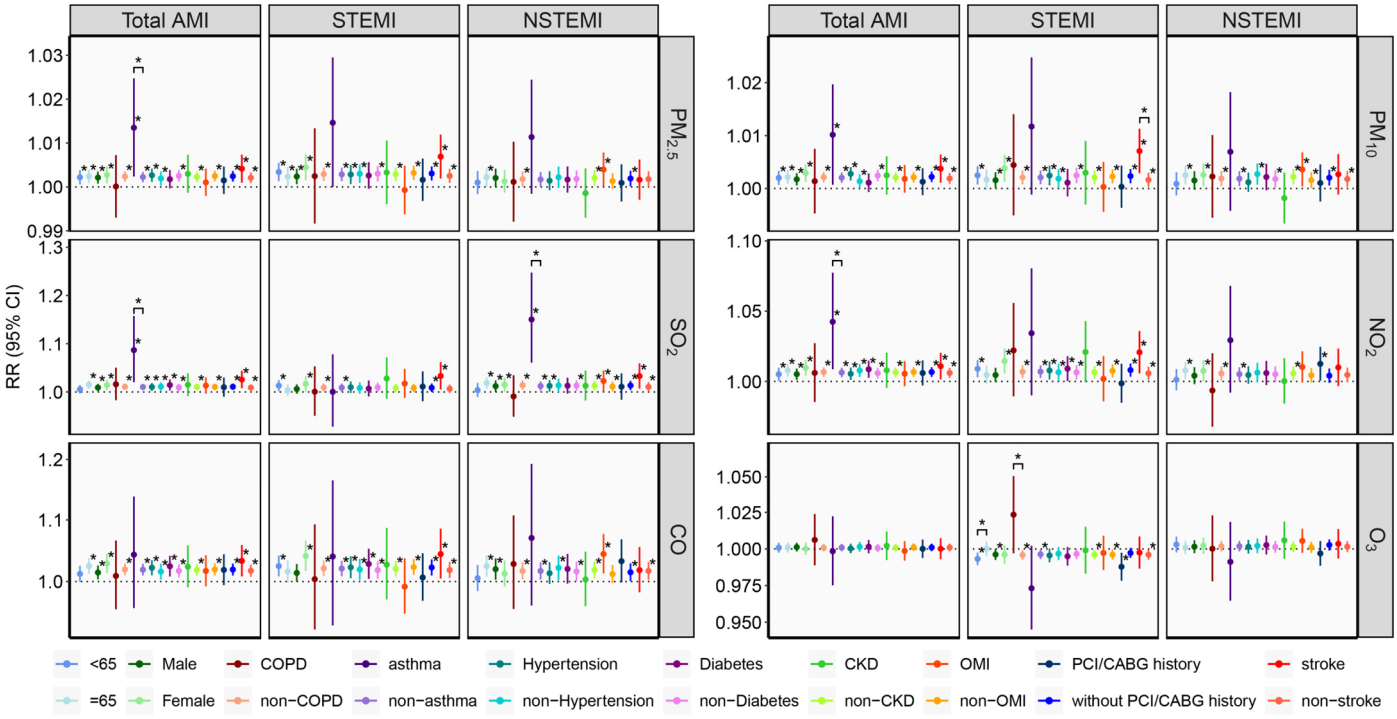


Figure 3. Associations between air pollutants and hospital admissions for total AMI, STEMI, and NSTEMI on strongest-effect lag days: stratified by age, sex, comorbidities, and cardiovascular disease history. Note: Relative risks (RRs) are for an increment of 10 μg/m^3 in $PM_{2.5}$, PM_{10}, SO_2, NO_2, and O_3 concentrations and 1 mg/m^3 in CO concentration. The strongest-effect lag days of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were lag0 for total AMI and STEMI and lag1 for NSTEMI. For O_3, the strongest-effect lag days were lag2 for total AMI and NSTEMI and lag4 for STEMI. * indicates *p*-value <0.05. Brackets and asterisks (*) indicate statistical significance of *Z* tests for between-group comparisons ($P_z < 0.05$). COPD: chronic obstructive pulmonary disease; CKD: chronic kidney disease; OMI: old myocardial infarction; PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting.

In the two-pollutant model (Table 4), $PM_{2.5}$, PM_{10}, NO_2, and CO remained positively associated with increased risks of total AMI admissions after adjusting for other pollutants with Spearman correlation coefficients < 0.7. However, the effect of SO_2 decreased and was no longer statistically significant after adjusting for $PM_{2.5}$ and CO. No significant association was observed between O_3 and total AMI admission risk in the two-pollutant model.

After adjusting the lag effects of meteorological factors (Figure S1), the effects of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO remained robust and statistically significant. In contrast, the effect of O_3 was sensitive to the lag effects of meteorological factors. The protective effect of O_3 at lag4 was unstable across the adjustment of meteorological factors' lag duration, transitioning from significant (lag0/lag03) to non-significant (lag06) with a longer lag duration of temperature and air pressure. No significant changes were observed after adjusting the dfs of the natural spline from 3 to 6 (Figure S2). The results of the time-series analysis were consistent with the findings from the CCO analysis (Figure S3).

Table 4. Associations between air pollutants and hospital admissions for total AMI in two-pollutant model.

Air Pollutants	Model	RR (95%CI)	Spearman Correlation
$PM_{2.5}$	unadjusted	1.002 (1.001, 1.003) *	-
	+SO_2	1.002 (1.001, 1.004) *	0.56
	+O_3	1.002 (1.001, 1.004) *	−0.13
PM_{10}	unadjusted	1.002 (1.001, 1.003) *	-
	+SO_2	1.002 (1.001, 1.003) *	0.57
	+O_3	1.002 (1.001, 1.003) *	−0.11
SO_2	unadjusted	1.011 (1.004, 1.017) *	-
	+$PM_{2.5}$	1.007 (1.000, 1.014) *	0.56
	+PM_{10}	1.007 (1.000, 1.014) *	0.57
	+NO_2	1.008 (1.001, 1.014) *	0.68
	+CO	1.007 (1.000, 1.014)	0.65
	+O_3	1.011 (1.004, 1.017) *	−0.33
NO_2	unadjusted	1.007 (1.003, 1.010) *	-
	+SO_2	1.006 (1.002, 1.009) *	0.68
	+O_3	1.006 (1.003, 1.010) *	−0.48
CO	unadjusted	1.019 (1.010, 1.029) *	-
	+SO_2	1.019 (1.007, 1.031) *	0.65
	+O_3	1.020 (1.011, 1.030) *	−0.33
O_3	unadjusted	1.001 (0.998, 1.003)	-
	+$PM_{2.5}$	1.001 (0.998, 1.003)	−0.13
	+PM_{10}	1.001 (0.998, 1.003)	−0.11
	+SO_2	1.001 (0.998, 1.003)	−0.33
	+NO_2	1.001 (0.999, 1.004)	−0.48
	+CO	1.001 (0.998, 1.003)	−0.33

* indicates *p*-value < 0.05. Relative risks (RRs) with 95% confidential intervals (CIs) are for an increment of 10 μg/m^3 in $PM_{2.5}$, PM_{10}, SO_2, NO_2, and O_3 concentrations and 1 mg/m^3 in CO concentration. According to the strongest effects in single-pollutant models, the lag days chosen were lag0 for $PM_{2.5}$, SO_2, NO_2, and CO and lag2 for O_3.

4. Discussion

Based on 7-year Beijing hospital admission data, we assessed the effects of short-term exposure to $PM_{2.5}$ and PM_{10}, SO_2, NO_2, CO, and O_3 on the risk of hospital admissions for total AMI and its subtypes (STEMI and NSTEMI). Higher concentrations of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were significantly associated with increased admission risks for total AMI as well as STEMI and NSTEMI, with the effects on STEMI occurring earlier and lasting for a shorter duration than those on NSTEMI. Individuals with asthma or stroke were particularly susceptible to the harmful effects of air pollution.

Particulate matter (especially $PM_{2.5}$) has received the most attention in previous studies on the association between air pollution and AMI. Most findings consistently indicated that short-term exposure to higher concentrations of fine particulate matter ($PM_{2.5}$) increased the risk of AMI morbidity [4,28], a conclusion supported by our study. However, studies on gaseous pollutants and AMI morbidity showed inconsistent conclusions [29,30]. In our study, higher concentrations of SO_2, NO_2, and CO were significantly associated with increased AMI admission risks, consistent with most previous studies. However, the effect of SO_2 was not robust and became non-significant after adjusting other pollutants in the two-pollutant model. The health effect of SO_2 as a single pollutant or with other atmospheric pollutants has long been controversial [1,31] and necessitates further research.

Previous studies on the relationship between O_3 exposure and AMI admission risk have produced conflicting findings. In most studies, the relative risk of O_3 was less than 1, indicating a protective effect on AMI, with a few studies reporting significant results [32]. However, a few studies have found adverse effects of O_3 on AMI incidence [33,34]. These

contradictory results for O_3 may be due to the fact that O_3 concentrations are highly influenced by temperature, sunshine, and other pollutant concentrations [35]. O_3 was consistently negatively correlated with other pollutants, suggesting that higher O_3 levels may indicate lower exposure to other pollutants [36]. In this study, O_3 concentration was negatively correlated with other pollutants and strongly correlated with daily mean temperature and air pressure. Sensitivity analysis revealed that the protective effect of O_3 on AMI admission risk was likely influenced by meteorological factors and was not robust, supporting our hypothesis.

Air pollution may adversely affect the cardiovascular system through multiple mechanisms, such as oxidative stress, inflammation, endothelial dysfunction, autonomic nervous imbalance, and thrombosis, leading to an elevated risk for AMI and other CVDs [37,38]. As previously mentioned, STEMI and NSTEMI exhibit differences in their pathogenic mechanisms. STEMI often results from plaque rupture or erosion, followed by thrombus formation and complete coronary artery occlusion [39,40]. Meanwhile, NSTEMI may result from plaque rupture or erosion without complete coronary obstruction or be triggered by other mechanisms, such as endothelial dysfunction, vasomotor dysfunction, and dysregulation of the autonomic nervous system. These differences in pathogenesis suggest that air pollutants may influence the occurrence of different AMI subtypes through distinct pathways, such as thrombosis and endothelial dysfunction.

In this study, higher concentrations of $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were significantly associated with increased risks of hospital admission for both STEMI and NSTEMI. However, there were differences in the magnitude and duration of lag effects for each pollutant between STEMI and NSTEMI. Compared with NSTEMI, the strongest effects of air pollutants on STEMI occurred earlier (lag0 vs. lag1) and lasted less time (2–3 days vs. 3–6 days). Studies by Rich et al. [41] and Gardner et al. [5] found that elevated $PM_{2.5}$ concentrations in the 24 h preceding emergency visits and 1 h prior to acute coronary syndrome onset were associated with an increased risk of transmural myocardial infarction (MI) and STEMI, respectively, but not non-transmural MI or NSTEMI. A study from Poland reported that the effect of particulate matter (especially PM_{10}) and SO_2 was more delayed in time in patients with NSTEMI than those with STEMI [42]. Similarly, a study on fine particulate matter and IHD in Beijing found that the effects of $PM_{2.5}$ on chronic and non-fatal cases lasted longer than those on acute and fatal cases [43].

The temporal variations in air pollution's impact on different AMI subtypes may arise from distinct time scales at which different mechanisms (e.g., rapid thrombogenicity within hours vs. delayed endothelial dysfunction over days) trigger AMI events. Furthermore, compared to NSTEMI, patients with STEMI experienced a shorter delay in seeking medical attention due to more severe symptoms. In our study, patients' admission time was used as a proxy for actual AMI onset time, which might also contribute to the observed differences in the lag effects of pollutants between STEMI and NSTEMI.

Most previous studies have shown that the elderly are more susceptible to air pollution than younger individuals [7,11,17]. In our study, the effects of gaseous pollutants on NSTEMI were greater in the elderly (age > 65 years), while the effects of these pollutants on STEMI were greater in younger individuals (age < 65 years), although these differences were not statistically significant. Previous studies have shown that the elderly are generally more likely to experience NSTEMI than STEMI, often exhibit more comorbidities, and are treated more conservatively [44,45]. Differences in baseline health profiles, the treatment of underlying diseases, and variations in air pollution's triggering mechanisms could explain the age-related differences in air pollution's effects on different AMI subtypes.

Our study found that the effect of some gaseous pollutants on AMI admissions was significantly higher in women than in men or follows this trend, consistent with some

previous studies [46]. However, other studies have found that males are more sensitive than females or that there is no significant difference in sensitivity between sexes [41,47,48]. Researchers believe that the differences in air pollution effects between sexes may be related to biological factors (e.g., hormone composition, bronchial structure, lung volume), individual behavior patterns (e.g., activity patterns, smoking), and psychosocial stress [49]. Some studies have shown that women and non-smokers are more likely to develop AMI when exposed to air pollutants compared to men and smokers [46,48], supporting this view. Additionally, variations in AMI mechanisms between males and females [50] may be another important reason for the sex-specific difference in susceptibility to the adverse effects of air pollution.

The associations between air pollution and AMI risk are stronger among individuals with certain comorbidities (e.g., hypertension, diabetes, dyslipidemia) or cardiopulmonary diseases, as shown in some studies [5,11,17,47]. In our study, stronger associations were observed in individuals with asthma and stroke. And O_3 exposure was associated with an elevated risk of STEMI admission in individuals with COPD, whereas an inverse association was observed in individuals without COPD. A Norwegian study found that active asthma and poorly controlled asthma were associated with an increased risk of AMI [51]. Air pollution may increase AMI incidence risk by exacerbating asthma symptoms [52,53]. A study from Tuscany, Italy, also found that individuals with COPD are more susceptible to the adverse effects of air pollutants in triggering AMI [54]. Patients with asthma or COPD are susceptible to the adverse effects of air pollution [52,55], and uncontrolled asthma or pre-existing COPD can increase the risk of AMI [51,56]. The common pathophysiological mechanisms of asthma, COPD, and AMI, such as a systemic inflammatory response, may explain why individuals with asthma and COPD are more vulnerable to the adverse effects of air pollution triggering AMI. A study from Taiwan, China, found that individuals with multiple AMI risk factors were more susceptible to the adverse effects of air pollutants in increasing AMI risk [47]. People with a history of stroke likely have more AMI risk factors, such as hypertension and diabetes, making them more susceptible to the adverse effects of air pollution.

This study has several strengths. Firstly, this is one of the limited number of studies analyzing the effect of air pollutants, including gaseous pollutants, on the risk of hospital admission for different AMI subtypes, while also examining the influence of comorbidities and cardiovascular disease history on susceptibility to air pollution. Secondly, based on 7-year high-quality hospitalization data covering all hospitals in Beijing, this study minimizes heterogeneity [28], enhancing the stability and generalizability of the findings. Thirdly, we utilized a time-stratified case-crossover design to account for long-term and seasonal trends and control for time-stable confounders such as genetics, smoking habits, nutrition, and socioeconomic status. Furthermore, the results were adjusted for confounders thought to influence the outcome, such as the interaction between different air pollutants and the lag effects of meteorological factors. And a time-series study design was utilized to verify the stability of the results as well.

There are a couple of study limitations. Firstly, using city-level average air pollutant concentrations as a proxy for personal exposure may lead to exposure misclassification [57,58]. Since AMI is a disease with rapid progression, using a daily time scale might cause exposure errors. Our study did not assess indoor air pollution (e.g., second-hand smoking, cooking) or further differentiate the source of pollution (e.g., vehicular vs. industrial), which should be further studied in future research. Secondly, using AMI admission data for analysis may miss pre-hospital deaths and asymptomatic patients who did not seek medical attention, and unmeasured confounders could affect the results. Additionally, patients' AMI subtypes, comorbidities, and previous diagnoses were collected from ICD-10-coded

discharge diagnoses, and uncertainties in diagnosis may influence the results. Further accurate research is needed to confirm our findings in subsequent studies.

5. Conclusions

This study suggests that short-term exposure to particulate matter ($PM_{2.5}$, PM_{10}) and gaseous pollutants (SO_2, NO_2, and CO) have adverse effects on AMI, with distinct temporal patterns and population vulnerabilities for STEMI and NSTEMI. These findings highlight the necessity of incorporating AMI subtype classification and individual vulnerability factors in both environmental health risk assessments and related public health policies. Future research is needed to explore the mechanisms underlying the differences in the effects of air pollutants on AMI subtypes.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/atmos16060715/s1, Tables S1–S10: Associations between air pollutants and hospital admissions for total AMI, STEMI, and NSTEMI on strongest-effect lag days: stratified by age, sex, COPD, asthma, hypertension, diabetes, CKD, stroke, OMI, and PCI/CABG history; Figure S1: Associations between air pollutants and hospital admissions for total AMI, with adjustment for different lag days of temperature and air pressure; Figure S2: Associations between air pollutants and total AMI hospital admissions, with adjustment for different degrees of freedom (dfs) of natural spline of daily mean temperature and air pressure; Figure S3: Associations between air pollutants and total AMI hospital admissions on different lag days: CCO design and time-series design [59–61].

Author Contributions: Conceptualization, Y.Z. (Yakun Zhao) and Z.F.; methodology, Y.Z. (Yakun Zhao) and Y.C.; validation, Y.C. and Z.F.; investigation, Y.Z. (Yakun Zhao) and Y.H.; formal analysis, visualization, and writing—original draft preparation, Y.Z. (Yakun Zhao); writing—review and editing, Y.Z. (Yakun Zhao), Y.C., Y.L., S.T., Y.H., J.F., Z.C., X.Z., Y.Z. (Yuansong Zhuang), J.L. and Z.F.; data curation, supervision, project administration, and funding acquisition, Z.F. All authors have read and agreed to the published version of the manuscript.

Funding: This work was funded by National High-Level Hospital Clinical Research Funding of Peking Union Medical College Hospital (2022-PUMCH-C-024, 2022-PUMCH-A-241, and 2022-PUMCH-B-030), Postdoctoral Fellowship Program of China Postdoctoral Science Foundation (GZC20230294), and National Natural Science Foundation (12126602 and 91643208).

Institutional Review Board Statement: This study was approved by the Peking Union Medical College Hospital (PUMCH) Institutional Review Board (protocol number: S-K1097).

Informed Consent Statement: Not applicable.

Data Availability Statement: The data presented in this study are available on request from the corresponding author. The data are not publicly available due to this dataset now being confidential.

Conflicts of Interest: The authors declare no conflicts of interest.

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