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Association between air pollution and non-accidental mortality in Guiyang, China: a time-series analysis (2013–2023)

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Background: Air pollution remains one of the leading environmental risk factors for human health globally, significantly contributing to the burden of disease and premature mortality. The relationship between air pollutants and non-accidental deaths in Guiyang was unclear.

Method: Descriptive analysis was conducted to characterize air pollutants and mortality trends during the study period. A time-stratified case-crossover design and a quasi-Poisson regression within a generalized additive model (GAM) framework were employed to examine the association between air pollution and mortality. The models incorporated a 7-day lag structure and adjusted for temporal trends, meteorological factors, and day of the week. Analyses were further stratified by cause-specific mortality, age group, sex, and seasonality as an endpoint for acute health effects, stratified by cause-specific mortality, age, sex and season.

Results: SO₂ exposure was associated with a 3.18% (95% CI: 1.95 to 4.42%) increase in excess risk (ER) for non-accidental mortality per 10 µg/m³ increase, with risks peaking at lag 1 and cumulative effects persisting beyond 7 days. NO₂ exposure resulted in a 2.87% (95% CI: 1.72 to 4.03%) ER increase, peaking at lag 1, with effects extending to lag 7. PM_{2.5} and PM₁₀ exposure resulted in ER increases of 1.42% (95% CI: 0.84 to 1.99%) and 0.86% (95% CI: 0.50 to 1.22%), respectively, peaking within 1–2 days. CO exposure was associated with a 1.64% (95% CI: 1.01 to 2.29%) ER increase per 0.1 mg/m³, with effects observed at lag 0. Females showed higher susceptibility to SO₂ (ER: 3.87, 95% CI: 2.05 to 5.72%), while males were more vulnerable to PM_{2.5} (ER: 1.80, 95% CI: 1.10 to 2.51%). Individuals aged ≥65 years exhibited elevated risks across all pollutants, particularly SO₂ (ER: 4.16, 95% CI: 2.73 to 5.61%). All pollutants contributed to excess mortality in the cold season.

Conclusion: Different pollutants exhibit different lag durations of excess risks and cumulative excess risks for non-accidental mortality and deaths related to circulatory and respiratory systems. Females showed greater susceptibility to SO₂, while males were more vulnerable to other pollutants. The individuals aged 65 years and older were particularly vulnerable populations.

KEYWORDS

air pollutants, daily mortality, excess risks, time-stratified case-crossover design, generalized additive model (GAM)

1 Introduction

Air pollution remains one of the leading environmental risk factors for human health globally, significantly contributing to the burden of disease and premature mortality. According to the World Health Organization (WHO), 99% of the world's population is exposed to air quality levels that exceed recommended limits. It is estimated that 8.7 million deaths worldwide result from breathing in particulate matter emitted into the atmosphere by fossil fuel combustion (1). In 2019, ambient air pollution was estimated to have caused 4.2 million premature deaths worldwide, primarily affecting cardiovascular and respiratory systems (2, 3). In 2021, particulate matter pollution, including both outdoor and indoor sources, was identified as a leading environmental risk factor for global health, according to the Global Burden of Disease Study (4). Air pollution is closely linked to cardiovascular and respiratory mortality. Fine particulate matter, due to its small particle size, can penetrate deeply into the lungs and bloodstream, causing significant damage to both cardiovascular and respiratory systems (5, 6). Gaseous pollutants like SO₂ and NO₂ are more associated with respiratory effects through airway irritation. Vulnerability varies by age and sex, with older adults (≥65 years) showing higher risks due to reduced physiological resilience, and females potentially more susceptible to gaseous pollutants due to greater airway reactivity (7, 8).

Guiyang, the capital of Guizhou Province, is located on the eastern edge of the Yunnan–Guizhou Plateau. The city features mountainous terrain, high forest coverage, and a humid subtropical climate with frequent rainfall, largely influenced by a quasi-stationary front. These unique geographical and meteorological characteristics can influence the generation, dispersion, and health impacts of air pollutants. Although Guiyang generally experiences lower pollution levels than many other Chinese cities, it remains a less-developed urban area with limited environmental management capacity. The uneven distribution of environmental risks across population raises concerns about environmental equity. Most existing studies in China have focused on heavily polluted or economically developed cities (8, 9). Evidence from less-developed inland cities, featuring unique geographic and climatic conditions as well as socioeconomic profiles, remains scarce. This study addresses this gap by investigating the associations between ambient air pollutants and mortality in Guiyang, providing region-specific evidence to inform public health interventions and promote environmental justice in under-resourced settings.

We employed a time-stratified case-crossover design combined with generalized additive models (GAMs) to evaluate the short-term effects of air pollutants on all-cause and cause-specific mortality in Guiyang. We also assessed potential effect modifications by age, sex, season, and cause of death to identify particularly vulnerable subgroups. Localized studies such as this are essential for capturing context-specific exposure–response relationships and informing the development of targeted air quality management strategies aimed at enhancing environmental health protections.

2 Materials and methods

2.1 Data sources

Mortality data were obtained from the Disease Surveillance Points System, managed by the Chinese Center for Disease Control and

Prevention, spanning January 1, 2013, to December 31, 2023. The data included a range of causes of death based on the primary diagnosis coded by ICD-10 (international classification of diseases, 10th revision), including non-accidental causes (A00–R99), respiratory cases (J00–J99), cardiovascular cases (I00–I99). We excluded the data from December 18, 2022, to January 28, 2023, due to the COVID-19 outbreak in Guiyang, which disrupted mortality patterns and caused an abnormal increase in deaths, potentially introducing significant bias into the analysis. Daily temperature and relative humidity data (2013–2023) were sourced from the Guiyang Meteorological Bureau. Daily pollutant concentrations (SO₂, NO₂, PM_{2.5}, PM₁₀, and CO) were calculated as the arithmetic mean across data from 12 air quality monitoring stations in Guiyang's main urban area, following standard time-series studies. All stations operational during the study period (2013–2023) were included. Missing values for air pollutant data, accounting for less than 1% at each station, were random and sporadic. Given the spatial averaging across multiple monitoring sites, these localized gaps had negligible impact on city-level daily means. Thus, the missing data were excluded from the analysis. Meteorological data had no significant missingness.

2.2 Statistics analysis

Descriptive statistics including the 25th percentile (P25), median (P50), 75th percentile (P75), minimum, and maximum were calculated for daily death counts, temperature, relative humidity, and pollutant concentrations (CO, PM_{2.5}, PM₁₀, SO₂, and NO₂). Spearman correlation analysis was applied to examine the relationships between pollutants and meteorological factors. Variance inflation factor (VIF) analysis was used to assess multicollinearity among pollutants.

We adopted a hybrid approach by integrating a GAM-based time-series framework with a time-stratified case-crossover design to assess the association between air pollution and mortality. The case-crossover design, validated in environmental epidemiology research (10). This design compares exposures during case periods (days preceding death) with matched control days (same weekday within the same month), effectively controlling for temporal trends and seasonality while estimating acute effects of air pollutants on mortality (11). Given the 11-year duration of our study, smooth term for time was included in the model to capture smooth, long-term variations over extended periods that case-crossover design may not fully captured (12).

Given that daily death counts relative to the total population represent a low to probability event, a generalized additive model with a quasi-Poisson distribution was employed. The formula is as follows:

$$\lg[E(Y_t)] = \beta Z_t + ns(time, df) + ns(temp, df) + ns(rhum, df) + Dow + stratum + intercept$$

Where Y_t represents number of deaths on day t . β represents the regression coefficient. Z_t represents concentration of a single pollutant on day t . $ns()$ is natural spline smoothing function. $Time$ is the variable of the temporal trend.; df is degrees of freedom. $Temp$ is daily average temperature. $Rhum$ is daily average relative humidity; Dow represents day to week effect; $Stratum$ is time stratification variable; $Intercept$ is model intercept.

Degrees of freedom were set to 7 per year for time, 3 for temperature, and 3 for relative humidity, optimized using the minimum quasi Akaike Information Criterion (QAIC).

Lag effects were analyzed for lag 0 to lag 7, and cumulative lag effects were assessed for lag 01 to lag 07. The acute mortality risk from air pollutants, while not always instantaneous, is predominantly concentrated within the first week following exposure (13, 14). The data were stratified into four subgroups: cause-specific mortality (overall, respiratory and cardiovascular), age (<65 years and ≥65 years), a stratification used to define older adults with greater vulnerability to air pollution and consistent with a large body of literature, sex (male and female) and season (defined October to March as cold season and April to September as warm season). Effects were considered statistically significant if the 95% confidence interval (CI) excluded zero. The analyses were performed primarily using the *Hmisc* and *mgcv* packages in R software (version 4.3.1). The significance level was set at $\alpha = 0.05$.

2.3 Sensitivity analysis

To test the robustness of our findings, we conducted sensitivity analyses by varying the degrees of freedom (df) for the time trend (6–8 per year), temperature (3–6), and humidity (3–6). We also fitted two-pollutant models, adjusting for one co-pollutant at a time, to assess potential confounding.

Furthermore, we evaluated the temporal stability of the association by stratifying the analysis into three periods: a baseline (2013–2019), the full period (2013–2023), and an adjusted period which excluded the acute COVID-19 outbreak phase. This comparison allowed us to assess the consistency of the effect estimates.

3 Results

3.1 Descriptive statistics

From 2013 to 2023, a total of 140,099 non-accidental deaths were recorded in Guiyang's main urban area, comprising 83,272 males (59.44%), and 56,827 females (40.56%). Deaths due to circulatory and respiratory system diseases totaled 59,830 and 21,028, respectively

(Table 1). The average temperature and relative humidity were 15.3°C and 80.3%, respectively. Daily average concentrations of CO, NO₂, SO₂, PM_{2.5}, and PM₁₀ were 0.60 µg/m³, 22.5 µg/m³, 13.8 µg/m³, 32.4 µg/m³ and 53.1 µg/m³, respectively (Table 2). The results for the entire study period are presented in Supplementary Tables S1, S2.

3.2 Correlations between air pollutants and meteorological factors

Spearman correlation analysis revealed negative correlations between temperature, relative humidity and most pollutants, except for CO, which was positively correlated with relative humidity ($r = 0.22$). The correlation coefficient between temperature and SO₂ was -0.60 . Positive correlations were observed among air pollutants, with the strongest association between PM_{2.5} and PM₁₀ ($r = 0.95$). Variance inflation factor analysis identified significant multicollinearity between PM_{2.5} and PM₁₀ (VIF = 11.2), while CO, NO₂, and SO₂ exhibited no multicollinearity, with VIF values of 2.4, 3.6, and 2.8, respectively (Tables 3, 4).

3.3 Lag effects of air pollutants on mortality

3.3.1 Effects of SO₂ on non-accidental mortality

No excess mortality risk from SO₂ exposure was observed in individuals under 65 years. For other groups, the risks persisted for 1–3 days, peaking at lag 1. A 10 µg/m³ increase in SO₂ concentration was associated with a 3.18% (95% CI: 1.95 to 4.42%) increase in non-accidental mortality risk, 3.23% (95% CI: 1.47 to 5.01%) in cardiovascular mortality risk, 5.89% (95% CI: 3.30 to 8.55%) in respiratory mortality risk. By sex, the risk increased by 2.72% (95% CI: 1.23 to 4.23%) for males, and 3.87% (95% CI: 2.05 to 5.72%) for females, while for individuals aged ≥65 years by 4.16% (95% CI: 2.73 to 5.61%). Cumulative lag effects showed no impact in the under 65 years group, while respiratory mortality and females risks peaked at lag 03 before gradually declining. Other groups exhibited increases at lags 06–07, with cumulative lag effects extending beyond 7 days (Figure 1).

TABLE 1 Distributions of non-accidental mortality in Guiyang, 2013–2023.

Variables	<i>n</i>	$\bar{x} \pm s$	Min	P25	P50	P75	Max
Number of non-accidental deaths	140,099	32.3 ± 16.2	1.0	21.0	37.0	48.0	100.0
Number of respiratory system deaths	21,028	5.3 ± 3.3	0.0	3.0	5.0	7.0	26.0
Number of circulatory system deaths	59,830	15.1 ± 7.7	0.0	9.0	15.0	20.5	45.0
Male	83,272	21.0 ± 9.9	0.0	13.0	21.0	28.0	58.0
Female	56,827	14.3 ± 7.5	0.0	8.0	14.0	20.0	42.0
Age 0–64 years	37,460	9.4 ± 4.8	0.0	6.0	9.0	13.0	28.0
Age ≥65 years	102,639	25.8 ± 12.7	0.0	15.0	26.0	36.0	73.0

Data exclude the period from December 18, 2022, to January 28, 2023, due to the COVID-19 outbreak.

TABLE 2 Daily air pollutants concentration and meteorological factors in Guiyang, 2013–2023.

Variables	$\bar{x} \pm s$	Min	P25	P50	P75	Max
Temp (°C)	15.3 ± 7.4	−4.4	9.4	16.4	21.8	28.2
RH (%)	80.3 ± 11.8	32.0	72.7	81.0	89.7	100.0
CO (mg/m ³)	0.6 ± 0.2	0.3	0.5	0.6	0.7	1.9
NO ₂ (μg/m ³)	22.5 ± 9.9	3.3	15.4	20.9	27.8	71.1
SO ₂ (μg/m ³)	13.8 ± 14.4	3.0	6.0	8.9	15.4	159.0
PM _{2.5} (μg/m ³)	32.4 ± 21.0	4.8	17.1	27.6	41.6	164.7
PM ₁₀ (μg/m ³)	53.1 ± 30.8	8.1	30.7	45.7	67.6	263.3

Data exclude the period from December 18, 2022, to January 28, 2023, due to the COVID-19 outbreak.

TABLE 3 Correlation between air pollutants and meteorological factors (*r*).

Variables	Temp	RH	CO	NO ₂	SO ₂	PM _{2.5}	PM ₁₀
Temp (°C)	1.00						
RH (%)	−0.34*	1.00					
CO (mg/m ³)	−0.47*	0.22*	1.00				
NO ₂ (μg/m ³)	−0.24*	−0.13*	0.65*	1.00			
SO ₂ (μg/m ³)	−0.60*	−0.09*	0.69*	0.70*	1.00		
PM _{2.5} (μg/m ³)	−0.38*	−0.25*	0.62*	0.72*	0.74*	1.00	
PM ₁₀ (μg/m ³)	−0.21*	−0.42*	0.52*	0.75*	0.68*	0.95*	1.00

**p* < 0.05.

TABLE 4 Variance inflation factor analysis.

Air pollutant	Variance inflation factor (VIF)
CO	2.4
NO ₂	3.6
SO ₂	2.8
PM _{2.5}	11.2
PM ₁₀	11.2

A VIF > 10 indicates significant multicollinearity.

3.3.2 Effects of NO₂ on non-accidental mortality

No excess mortality risk from NO₂ exposure was observed in individuals under 65 years of age. For other groups, the risk persisted for 2–3 days. Respiratory mortality risk was highest on the day of exposure (lag 0), while other groups showed peak risk at lag 1. With a 10 μg/m³ increase in NO₂ concentration, non-accidental mortality risk increased by 2.87% (95% CI: 1.72 to 4.03%), respiratory mortality risk by 3.46% (95% CI: 0.75 to 6.25%), cardiovascular mortality risk by 2.93% (95% CI: 1.28 to 4.61%), male mortality risk by 2.67% (95% CI: 1.27 to 4.10%), female mortality risk by 3.16% (95% CI: 1.48 to 4.87%), and mortality risk in individuals aged ≥65 years by 3.27% (95% CI: 1.94 to 4.61%). Cumulative lag risks, except for the <65 years age group, peaked at lag 2–3 before gradually declining. The cumulative lag duration was 5 days for respiratory mortality risk and exceeded 7 days for all other groups (Figure 2).

3.3.3 Effects of PM_{2.5} on non-accidental mortality

No excess mortality risk from PM_{2.5} exposure was observed in individuals under 65 years of age. For other groups, single-day effects persisted for 1–3 days, with the highest risk occurring on the day of exposure (lag 0). With a 10 μg/m³ increase in PM_{2.5} concentration,

non-accidental mortality risk increased by 1.42% (95% CI: 0.84 to 1.99%), cardiovascular mortality risk by 1.24% (95% CI: 0.42 to 2.08%), respiratory mortality risk by 2.78% (95% CI: 1.49 to 4.08%), male mortality risk by 2.72% (95% CI: 1.23 to 4.23%), female mortality risk by 1.25% (95% CI: 0.41 to 2.10%), and mortality risk in individuals aged ≥65 years by 1.93% (95% CI: 1.27 to 2.59%). Cumulative risks, except for the <65 years age group, peaked at lag 2 before gradually declining. The cumulative lag duration was 5 days for respiratory mortality risk, 4 days for female mortality risk, and exceeded 7 days for all other groups (Figure 3).

3.3.4 Effects of PM₁₀ on non-accidental mortality

No excess mortality risk from PM₁₀ exposure was observed in individuals under 65 years of age. For other groups, single-day effects persisted for 1–3 days, with the highest risk occurring on the day of exposure (lag 0). With a 10 μg/m³ increase in PM₁₀ concentration, non-accidental mortality risk increased by 0.86% (95% CI: 0.5 to 1.22%), cardiovascular mortality risk by 0.79% (95% CI: 0.2 to 1.37%), respiratory mortality risk by 1.62% (95% CI: 0.71 to 2.55%), male mortality risk by 0.86% (95% CI: 0.42 to 1.3%), female mortality risk by 0.87% (95% CI: 0.34 to 1.41%), and mortality risk in individuals aged ≥65 years by 1.17% (95% CI: 0.71 to 1.65%). Cumulative lag risks, except for the <65 years age group, peaked at lag 1–2 before gradually declining. The cumulative lag duration was 5 days for both cardiovascular and respiratory mortality risks, 4 days for female mortality risk, and exceeded 7 days for all other groups (Figure 4).

3.3.5 Effects of CO on non-accidental mortality

No excess mortality risk from CO exposure was observed in individuals under 65 years of age. For other groups, single-day lag effects persisted for 0–1 days, with the highest risk occurring on the day of exposure (lag 0). With a 0.1 mg/m³ increase in CO

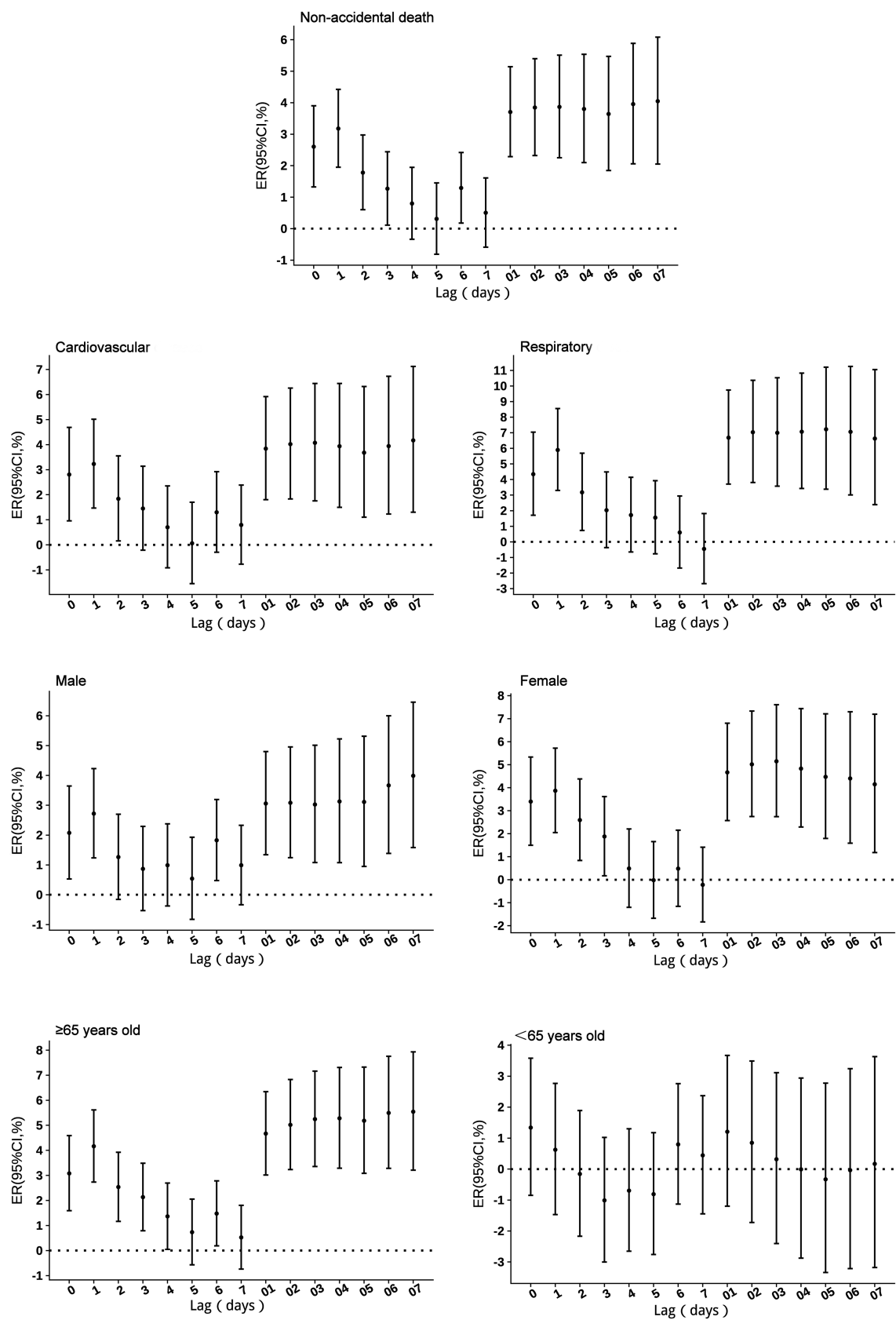


FIGURE 1
Excess risks of non-accidental mortality associated with SO_2 exposure among residents in Guiyang.

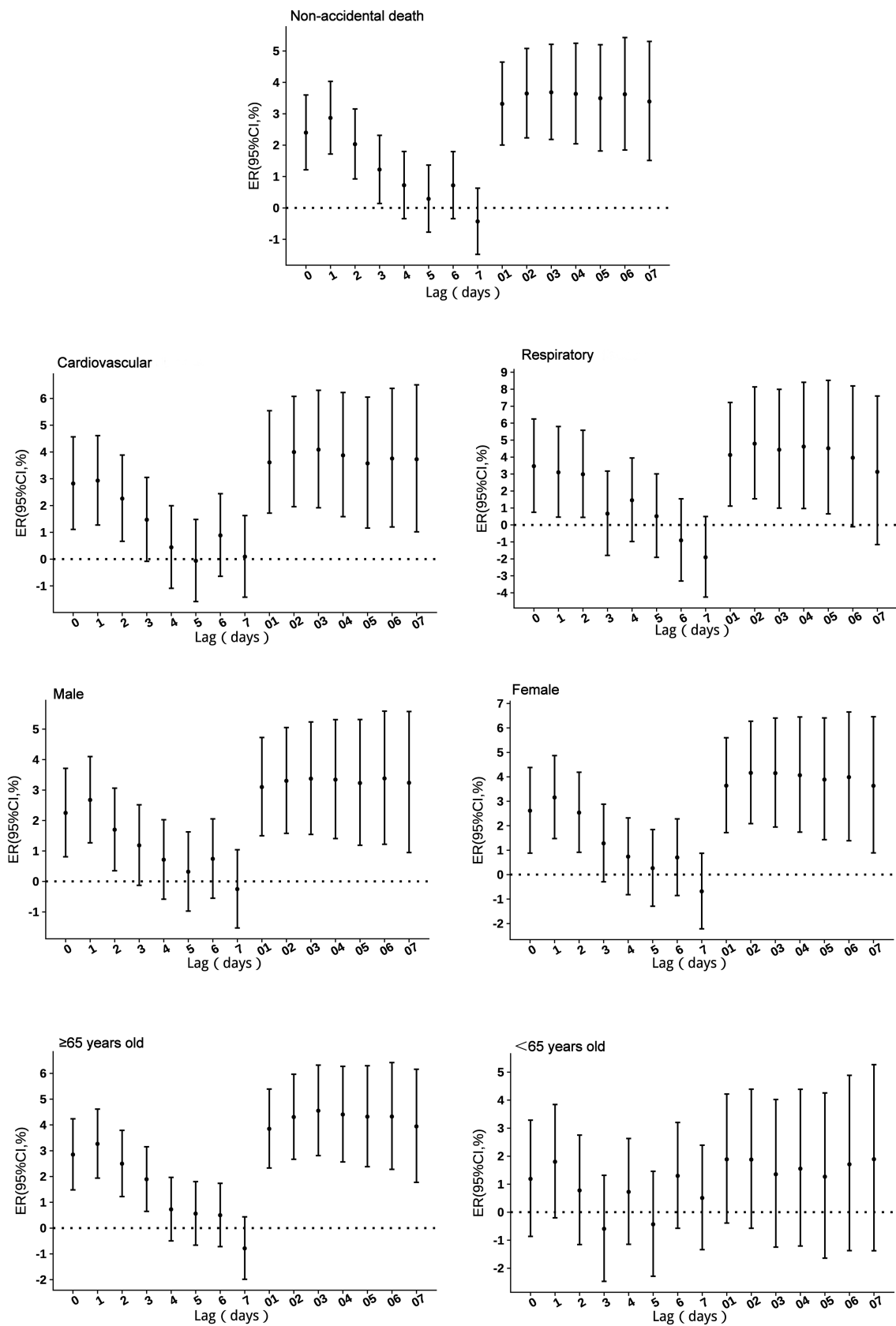


FIGURE 2
Excess risks of non-accidental mortality associated with NO_2 exposure among residents in Guiyang.

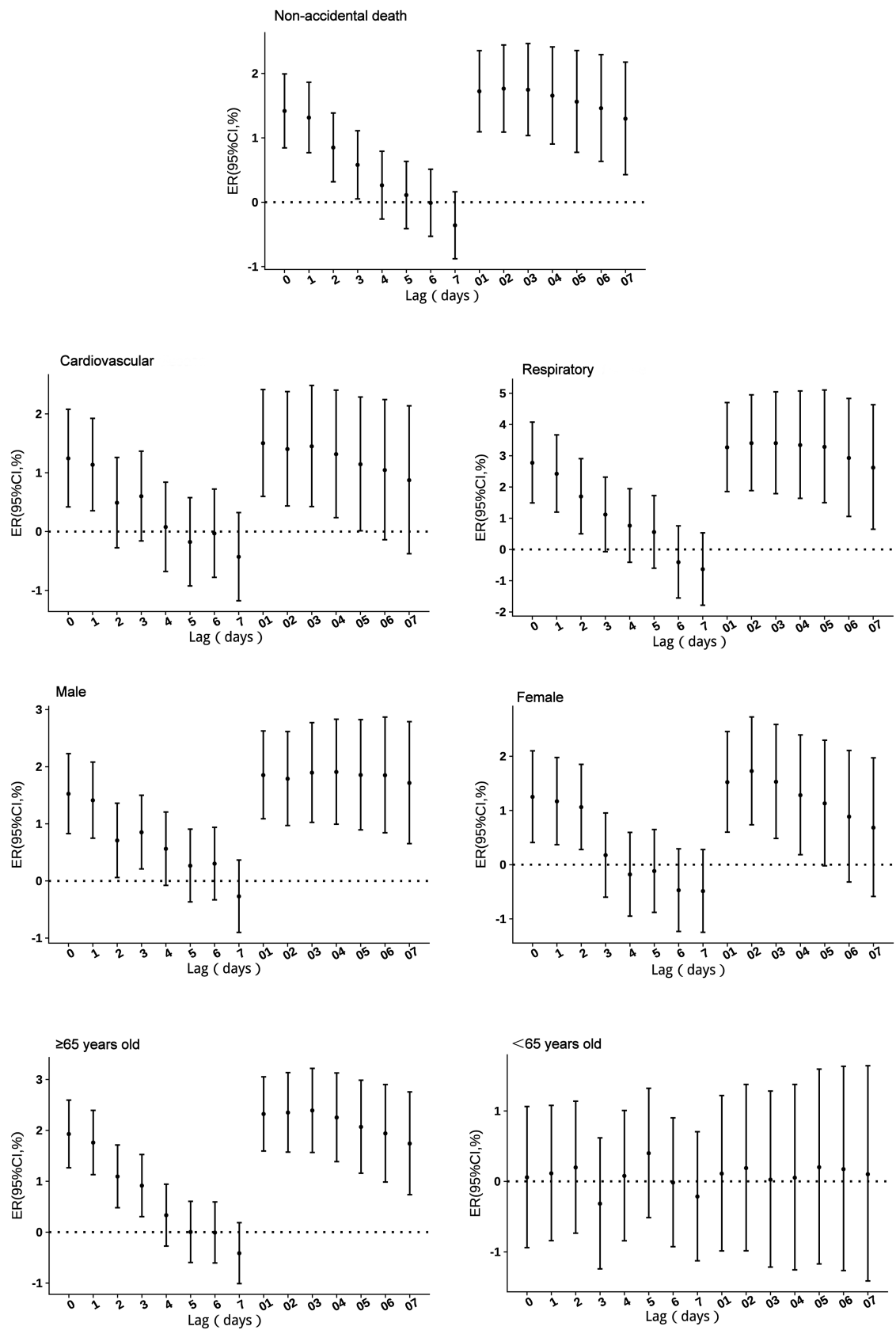


FIGURE 3
Excess risks of non-accidental mortality associated with $PM_{2.5}$ exposure among residents in Guiyang.

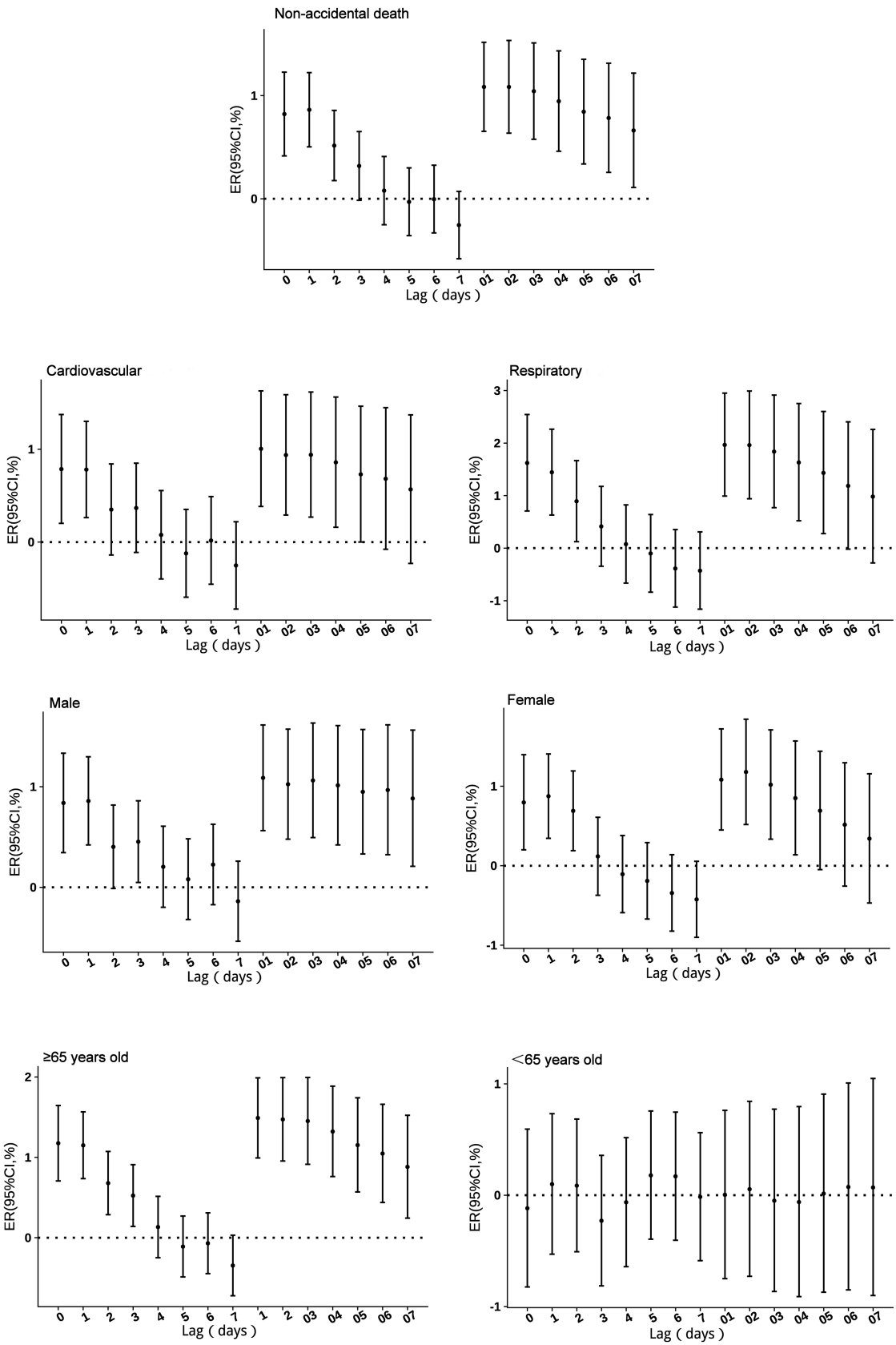


FIGURE 4
Excess risks of non-accidental mortality associated with PM_{10} exposure among residents in Guiyang.

concentration, non-accidental mortality risk increased by 1.64% (95% CI: 1.01 to 2.29%), cardiovascular mortality risk by 1.82% (95% CI: 0.89 to 2.75%), respiratory mortality risk by 2.38% (95% CI: 0.96 to 3.81%), male mortality risk by 1.81% (95% CI: 1.03 to 2.59%), female mortality risk by 1.39% (95% CI: 0.46 to 2.34%), and mortality risk in individuals aged ≥ 65 years by 1.90% (95% CI: 1.16 to 2.64%). Additionally, respiratory mortality risk showed an increase at lag 5. For cumulative lag risks, no effects were observed in the <65 years age group. In the respiratory mortality group, an increase in excess risk appeared at cumulative lag 5–6. For other groups, cumulative lag risks peaked at lag 1–2 before gradually declining, with cumulative effects persisting for 3–6 days (Figure 5).

3.3.6 Excess mortality risks from air pollutants in cold and warm seasons

The study period was divided into cold and warm seasons for analysis. During the cold season, all pollutants demonstrated excess mortality risks across all groups, except for individuals under 65 years of age. For non-accidental diseases, mortality risk increased by 2.42% (95% CI: 1.05 to 3.80%) for SO_2 , 3.19% (95% CI: 1.72 to 4.67%) for NO_2 , 1.46% (95% CI: 0.79 to 2.13%) for $\text{PM}_{2.5}$, 0.92% (95% CI: 0.44 to 1.41%) for PM_{10} , and 1.47% (95% CI: 0.95 to 2.45%) for CO. No significant mortality risks were observed during the warm season (Table 5).

3.3.7 Sensitivity analysis

The sensitivity analysis showed consistent results across different degrees of freedom (df) for time, humidity, and temperature, confirming the robustness of the model. For time (df = 6–8), humidity (df = 3–5), and temperature (df = 3–5), the excess mortality risks for SO_2 , NO_2 , $\text{PM}_{2.5}$, PM_{10} , and CO varied minimally, with only slight changes in effect estimates (Supplementary Table S3). In two-pollutant models, These findings indicate that the associations between air pollutants and mortality risks are stable under different model settings (Supplementary Table S4). The time-stratified sensitivity analysis revealed that the inclusion of the acute COVID-19 outbreak phase substantially altered the results (Supplementary Figure S1). The Baseline and Adjusted periods estimates remained broadly similar, whereas, whereas the results for the Full period differed substantially, particularly for CO, PM_{10} , SO_2 , and NO_2 . Therefore, to mitigate this potential source of bias, the results from the full period analysis were excluded from our main analysis.

4 Discussion

This study provides a comprehensive analysis of the acute mortality effects of air pollution in Guiyang, China. Our approach utilized a time-series analysis, specifically employing a time-stratified case-crossover design with generalized additive models (GAMs), to examine these associations across strata of cause-specific mortality, sex, age, and season. The study covered 140,099 non-accidental deaths, with 59.44% occurring among males and 73.26% among those aged ≥ 65 years. The average temperature and relative humidity in Guiyang were 15.3°C and 80.3%, respectively. Daily average pollutant concentrations were relatively low compared to other Chinese cities, reflecting Guiyang's minimal industrial activity. However, spearman correlation analysis revealed significant negative correlations between

temperature and air pollutants during the study period, meaning cold season may be a dangerous factor.

Our analysis revealed that each pollutant induced mortality through distinct temporal patterns and biological mechanisms. A $10 \mu\text{g}/\text{m}^3$ increase in SO_2 was associated with increases in non-accidental, respiratory, and cardiovascular mortality of 3.18% (95% CI: 1.95 to 4.42%), 5.89% (95% CI: 3.30 to 8.55%), and 3.23% (95% CI: 1.47 to 5.01%), respectively. As a water-soluble irritant gas, SO_2 is rapidly absorbed by the upper respiratory tract, triggering vagus nerve reflexes that cause bronchial smooth muscle contraction, airway narrowing, and respiratory inflammation (15, 16). These mechanisms explain the stronger association with respiratory mortality. Additionally, SO_2 may impact cardiovascular function through oxidative stress and interference with biological markers (17). Our findings align with studies linking short-term SO_2 exposure to increased respiratory-related medical visits (18) and long-term exposure to bronchitis and reduced lung function.

NO_2 exposure showed comparable acute health impacts, with single-day excess mortality risks peaking at lag 1 for most groups, except respiratory mortality, which was highest at lag 0. A $10 \mu\text{g}/\text{m}^3$ increase in NO_2 concentration increased non-accidental mortality risk by 2.87% (95% CI: 1.72 to 4.03%), cardiovascular mortality by 2.93% (95% CI: 1.28 to 4.61%), and respiratory mortality by 3.46% (95% CI: 0.75 to 6.25%). These results are consistent with an Italian cohort study by Chiusolo et al. (19), which reported mortality increments of 2.09% (95% CI: 0.96 to 3.24%) for all-cause deaths, 3.48% (95% CI: 0.75 to 6.29%) for respiratory mortality and 2.63% (95% CI: 1.53 to 3.75%) for cardiovascular mortality per $10 \mu\text{g}/\text{m}^3$ increase in NO_2 . Cumulative lag effects persisted beyond 7 days for cardiovascular mortality, indicating more severe and prolonged damage compared to respiratory effects, which ceased after day 6. A meta-analysis supports greater cardiovascular damage from long-term NO_2 exposure (20), with associations to coronary heart disease, stroke, and arrhythmia (21, 22).

Exposure to particulate matter was significantly associated with increased mortality, with the effects of $\text{PM}_{2.5}$ being consistently stronger than those of PM_{10} . A $10 \mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ was associated with a 1.42% (95% CI: 0.84 to 1.99%) increase in non-accidental mortality, a 1.24% (95% CI: 0.42 to 2.08%) increase in cardiovascular mortality, and a 2.78% (95% CI: 1.49 to 4.08%) increase in respiratory mortality. In comparison, the same increase in PM_{10} resulted in lower excess mortality effects, with increases of 0.86% (95% CI: 0.50 to 1.22%) for non-accidental, 0.79% (95% CI: 0.20 to 1.37%) for cardiovascular, and 1.62% (95% CI: 0.71 to 2.55%) for respiratory mortality. The stronger health impact of $\text{PM}_{2.5}$ aligns with its smaller particle size and higher reactivity, which enable deeper penetration into alveolar tissue and the absorption of toxic substances (23), resulting in both respiratory and cardiovascular damage as supported by prior research (24). Both pollutants exhibited similar acute effects, with single-day risks peaking at lag 0 and persisting for 1–3 days, while cumulative risks for both extended beyond 7 days for most groups. Furthermore, the strong correlation ($r = 0.95$) and significant multicollinearity ($\text{VIF} = 11.2$) observed between $\text{PM}_{2.5}$ and PM_{10} are noteworthy. This statistical relationship is expected, as $\text{PM}_{2.5}$ is a physical subset of PM_{10} . Additionally, the strong association suggests that both pollutants largely originate from common emission sources in Guiyang, such as vehicular traffic and coal combustion, highlighting the need for integrated control strategies (25).

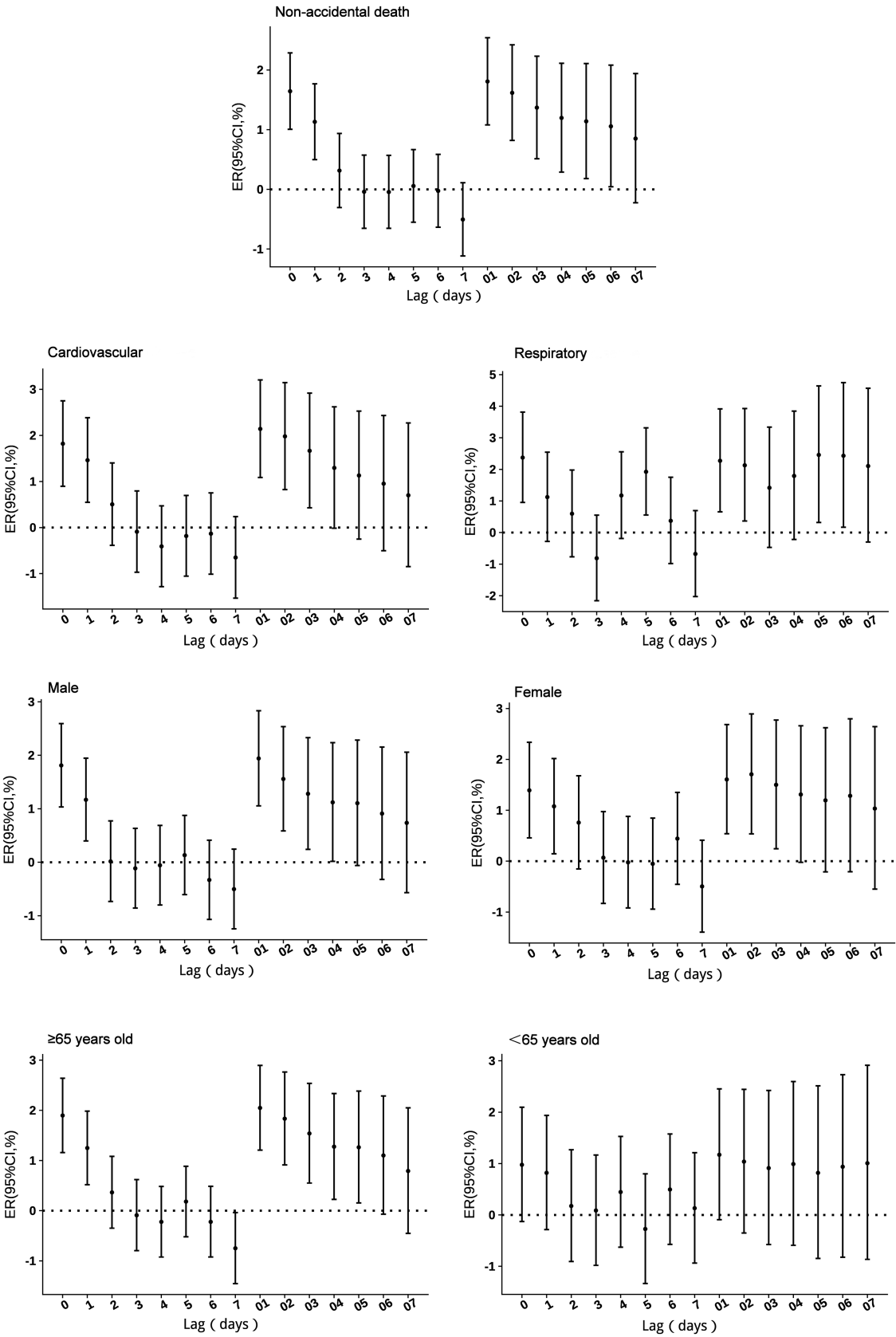


FIGURE 5
Excess risks of non-accidental mortality associated with CO exposure among residents in Guiyang.

TABLE 5 Excess risks (%; 95CI) from air pollutants in cold and warm seasons.

Groups	SO ₂	NO ₂	PM _{2.5}	PM ₁₀	CO
Cold season					
Non-accidental diseases	2.42% (1.05 to 3.80%)	3.19% (1.72 to 4.67%)	1.46% (0.79 to 2.13%)	0.92% (0.44 to 1.41%)	1.47% (0.95 to 2.45%)
Respiratory diseases	3.61% (0.79 to 6.51%)	4.13% (0.79 to 7.58%)	2.59% (1.10 to 4.10%)	1.45% (0.35 to 2.55%)	2.10% (0.44 to 3.79%)
Cardiovascular diseases	2.90% (0.93 to 4.91%)	3.52% (1.41 to 5.67%)	1.05% (0.10 to 2.02%)	0.75% (0.05 to 1.44%)	1.90% (0.82 to 2.99%)
Female	3.02% (1.04 to 5.05%)	3.15% (1.04 to 5.29%)	1.02% (0.06 to 1.99%)	0.70% (0.01 to 1.40%)	1.44% (0.36 to 2.53%)
Male	2.02% (0.36 to 3.71%)	3.22% (1.42 to 5.04%)	1.75% (0.94 to 2.57%)	1.07% (0.48 to 1.66%)	1.87% (0.95 to 2.79%)
≥65 years old	2.50% (0.94 to 4.08%)	3.78% (2.12 to 5.47%)	2.08% (1.33 to 2.84%)	1.31% (0.76 to 1.86%)	2.02% (1.17 to 2.88%)
<65 years old	2.18% (−0.20 to 4.61%)	1.54% (−1.03 to 4.17%)	−0.23% (−1.40 to 0.95%)	−0.13% (−0.98 to 0.74%)	0.82% (−0.49 to 2.14%)
Warm season					
Non-accidental diseases	1.71% (−2.92 to 6.56%)	0.13% (−1.82 to 2.13%)	0.93% (−0.21 to 2.08%)	0.33% (−0.41 to 1.08%)	0.83% (−0.44 to 2.12%)
Respiratory diseases	5.76% (−3.92 to 16.42%)	0.85% (−3.69 to 5.60%)	2.42% (−0.18 to 5.09%)	1.53% (−0.15 to 3.24%)	2.35% (−0.56 to 5.34%)
Cardiovascular diseases	−0.87% (−7.33 to 6.05%)	0.68% (−2.18 to 3.61%)	1.53% (−0.14 to 3.22%)	0.68% (−0.40 to 1.77%)	0.91% (−0.94 to 2.80%)
Female	1.97% (−5.02 to 9.46%)	0.69% (−2.23 to 3.70%)	1.41% (−0.30 to 3.16%)	0.68% (−0.44 to 1.81%)	0.72% (−1.18 to 2.66%)
Male	1.53% (−3.97 to 7.35%)	−0.24% (−2.59 to 2.18%)	0.61% (−0.77 to 2.00%)	0.10% (−0.79 to 1.00%)	0.91% (−0.63 to 2.48%)
≥65 years	3.87% (−1.71 to 9.77%)	0.00% (−2.29 to 2.34%)	0.90% (−0.43 to 2.25%)	0.48% (−0.39 to 1.36%)	0.69% (−0.80 to 2.20%)
<65 years	−3.38% (−10.65 to 4.47%)	0.46% (−2.91 to 3.94%)	0.95% (−1.01 to 2.95%)	−0.08% (−1.35 to 1.21%)	1.21% (−0.98 to 3.45%)

CO exposure also presented a significant acute risk, with a 1.64% (95% CI: 1.01 to 2.29%) mortality increase per 0.1 mg/m³ rise, peaking at lag 0. This immediate effect is explained by its primary toxic mechanism: the formation of carboxyhemoglobin, which impairs the blood's oxygen-carrying capacity and leads to systemic tissue hypoxia, primarily affecting cardiovascular and respiratory systems (26, 27).

Individuals aged ≥65 years exhibited higher vulnerability across all pollutants, with excess risks of 4.16% (95% CI: 2.73 to 5.61%) for SO₂, 3.27% (95% CI: 1.94 to 4.61%) for NO₂, 1.93% (95% CI: 1.27 to 2.59%) for PM_{2.5}, 1.17% (95% CI: 0.71 to 1.65%) for PM₁₀, and 1.90% (95% CI: 1.16 to 2.64%) for CO. This aligns with prior studies attributing increased susceptibility to compromised immune function and prevalent comorbidities in older adults (28, 29). Sex-stratified analyses revealed nuanced differences. Females showed greater susceptibility to SO₂ with an excess risk of 3.87% (95% CI: 2.05 to 5.72%), an effect that persisted for multiple days and may be linked to higher airway reactivity and hormonal influences (30). In contrast, males were more vulnerable to PM_{2.5}, showing an excess risk of 2.72% (95% CI: 1.23 to 4.23%) with extended cumulative effects, a finding possibly exacerbated by higher smoking rates which can amplify particulate matter toxicity (31). While risks for NO₂, PM₁₀, and CO were more comparable between sexes, these findings highlight how gender can modify susceptibility depending on the specific pollutant.

When dividing the study period into cold and warm seasons. All significant health risks were concentrated in the cold season, with no notable effects in the warm season, coinciding with higher pollutant concentrations during this period. This finding aligns with previous studies (32, 33). This pronounced seasonal difference is likely driven by two key factors: a surge in pollutant emissions from heating during winter, and adverse meteorological conditions to Guiyang. The city's climate, heavily influenced by a quasi-stationary front, leads to humid, calm, and rainy winters that trap pollutants near ground level and result in sustained high-concentration exposures. The strong negative correlation between temperature and pollutant levels further supports the role of the cold season as the primary period of risk.

Several limitations should be noted in this study. First, as an ecological study, our analysis may be subject to ecological fallacy, meaning population-level associations may not accurately reflect individual exposure concentration-mortality relationships. Second, we were unable to control for important socioeconomic factors, particularly income level and occupation, which may influence exposure and vulnerability. Third, despite strict quality control, potential misdiagnosis or coding errors in mortality data cannot be ruled out. Fourth, the lack of indoor environmental data may introduce bias to our results. Despite these limitations, our findings hold significant public health relevance. Future research should aim to address these gaps by employing more detailed individual-level studies and enhanced data collection methods. Furthermore, conducting source apportionment analyses would help identify the specific pollution sources most strongly associated with these health effects, thereby providing more targeted evidence for effective policy interventions.

5 Conclusion

This study provides compelling evidence that short-term exposure to ambient air pollutants in Guiyang significantly increases the risk of

non-accidental, cardiovascular, and respiratory mortality. The key public health message is that substantial health threats persist even at the city's relatively moderate pollution levels, particularly for vulnerable segments of the population during specific high-risk periods. Our findings pinpoint a clear profile of this vulnerability: the older adults (aged ≥65 years) are consistently the most affected subgroup, facing elevated risks from all measured pollutants, while specific gender-based susceptibilities were observed for SO₂ and PM_{2.5}. Based on these findings, we recommend targeted interventions during the cold season. Policymakers should consider reducing emissions from residential heating by encouraging the use of cleaner energy sources like natural gas or electric heating systems. Public health officials should also develop campaigns to promote protective measures for vulnerable groups, including the use of air purifiers and masks during high pollution periods.

Data availability statement

The data analyzed in this study is subject to the following licenses/restrictions: the datasets presented in this article are managed in strict accordance with the data security and confidentiality requirements imposed by the Guizhou Center for Disease Control and Prevention. Requests to access these datasets should be directed to gzcde_gws@163.com.

Ethics statement

Ethical approval was not required for the study involving humans in accordance with the local legislation and institutional requirements. Written informed consent to participate in this study was not required from the participants or the participants' legal guardians/next of kin in accordance with the national legislation and the institutional requirements.

Author contributions

XC: Conceptualization, Formal analysis, Funding acquisition, Methodology, Writing – original draft, Writing – review & editing. MS: Conceptualization, Data curation, Methodology, Writing – review & editing. MY: Data curation, Visualization, Writing – review & editing. ZJ: Data curation, Writing – review & editing. DY: Data curation, Writing – review & editing. RL: Data curation, Formal analysis, Writing – review & editing. HG: Funding acquisition, Supervision, Writing – review & editing. JZ: Conceptualization, Methodology, Supervision, Validation, Writing – review & editing.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Generative AI statement

The authors declare that no Gen AI was used in the creation of this manuscript.

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Supplementary material

The Supplementary material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fpubh.2025.1602900/full#supplementary-material>