



OPEN Short-term effect of PM_{2.5} exposure on pediatric neurological outpatient visits in Shijiazhuang China 2013–2021

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In this study, outpatient data of pediatric neurological diseases together with air pollutants data and meteorological factors were collected in SJZ from 2013 to 2021 to study the correlation between exposure to PM_{2.5} and pediatric neurological diseases. A Poisson regression GLM was employed to assess the risk associated with both a single time lag and a moving average time lag of 0–7 days. The Poisson regression GLM was evaluated with respect to sex, age, seasons, and disease types. For per IQR (75.2 µg/m³) increase in PM_{2.5} concentration over a 7-day period (lag07) was correlated with a 2.047% increase in the overall count of pediatric outpatient visits for nervous system-related issues. As PM_{2.5} concentration increased, its impact was more obvious among girls or children aged < 6 years. Moreover, the adverse effects on nervous systems of children were more pronounced during the warm season (ER: 4.875%). A preliminary exposure assessment revealed a significant correlation between PM_{2.5} and both the overall outpatient visits for pediatric neurological diseases and the number of various outpatient visits for neurological disorders in children. Additionally, girls < 6 years old were identified as a group particularly sensitive to PM_{2.5} exposure.

Keywords PM_{2.5} exposure, Pediatric neurological outpatient visits, Time series analysis

Abbreviations

SJZ	Shijiazhuang
GLM	Generalized linear model
NSDs	Nervous system diseases
CNS	Central nervous system
BBB	Blood–brain barrier
SO ₂	Sulfur dioxide
NO ₂	Nitrogen dioxide
O ₃	Ozone
GLM	Generalized linear model
ER	Excess risk
ANOVA	Analysis of variance
CI	Confidence interval
IQR	Interquartile range

The global burden of diseases, injuries, and risk factors study recognizes NSDs as significant contributors to morbidity and mortality worldwide¹. NSDs encompass a wide range of conditions that include communicable neurological disorders, stroke, headache, neurodegenerative diseases, demyelinating diseases, brain, and CNS cancers. NSDs also encompass other less common neurological disorders, such as tetanus and meningitis, idiopathic epilepsy, encephalitis, and migraine, which are the primary causes of pediatric neurologic disorders².

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Environmental pollution is a notable significant external factor that contributes to the development of mental and neurological diseases that manifest across all age groups.

PM_{2.5}, one of six standard air pollutants, poses serious risks to human health^{3–7}. Acute exposure to PM_{2.5} can result in rapid systemic effects because of its ability to penetrate the alveoli and enter the bloodstream, leading to various physiological responses^{8–10}. In particular, PM_{2.5} exposure can exacerbate inflammation and oxidative stress, two key biological mechanisms that have been implicated in neurological disorders. Acute neuroinflammatory responses may be triggered as PM_{2.5} prompts the release of inflammatory cytokines at entry points such as the lungs, and it can subsequently reach the CNS through the peripheral circulatory system¹¹.

Although PM_{2.5} may not directly enter the CNS, its acute inflammatory and oxidative effects can compromise the BBB or blood–cerebrospinal fluid barriers, thus facilitating secondary neurotoxicity^{12,13}. These mechanisms have been linked to the acute exacerbation of neurological diseases. Recent studies have highlighted the association between PM_{2.5} exposure and acute neurological diseases^{14–18}, including stroke, cognitive decline, and the aggravation of pre-existing conditions, such as epilepsy. A case-crossover study conducted in eastern China revealed a significant association between short-term PM_{2.5} exposure and an increased risk of childhood epilepsy exacerbation¹⁹. It is crucial to further explore the acute effects of PM_{2.5} on pediatric neurological health, as children are especially vulnerable to environmental hazards because of their ongoing neurodevelopment^{20–24}.

SJZ, a city located in northern China, has historically experienced severe air pollution²⁵. Although numerous studies have assessed the health risks to adults^{26,27}, the impact of air pollution on children's nervous systems remains understudied. The aims of this study are: (1) to investigate the association between PM_{2.5} exposure and pediatric neurologic disorders in SJZ; (2) to explore the effect of PM_{2.5} exposure on the number of outpatient visits for pediatric neurologic disorders using a time-series model; (3) to identify populations who are sensitive to PM_{2.5} exposure in SJZ; and (4) to evaluate the potential health risk to pediatric nervous systems.

Methods

Study area and data collection

SJZ is located southwest of Hebei Province, China, at 114°30'E and 38°02'N. The region experiences a typical temperate monsoon climate that is characterized by distinct seasonal fluctuations.

Daily records of pediatric outpatient visits were collected from a local children's hospital from January 1, 2013, to December 31, 2021. All of the cases involved individuals aged ≤ 14 years, and all of the patients were residents of urban districts. This children's hospital is the only tertiary grade A pediatric specialty hospital located in the urban area of SJZ, and it has the highest volume of pediatric consultations in the city. Further, the hospital is distinguished by its focus on the diagnosis, treatment, and rehabilitation of neurological disorders. Consequently, the daily outpatient volume of this hospital serves as a valuable indicator of the effects of PM_{2.5} concentration fluctuations on pediatric health outcomes in the primary urban area of SJZ, thus possessing a degree of representativeness. Demographic data were recorded for every patient, including the diagnosis date, age, sex, and International Classification of Diseases 10th Revision (ICD-10) codes. NSDs were categorized based on ICD-10 codes (G00–G99), and further classified and summarized according to their pathophysiological mechanisms.

Central infectious encephalopathy includes meningitis, encephalitis, and myelitis. Noninfectious acute encephalopathy includes demyelinating diseases, epilepsy, migraine, and hydrocephalus. Chronic brain dysfunction disease includes systemic atrophy, Parkinson's disease, and cerebral palsy. The specific classifications of neurological diseases are shown in (Table 1).

The meteorological data (e.g., daily average temperature, relative humidity, atmospheric pressure, sunshine duration, and wind speed) during the study period were obtained from the National Meteorological Science Data Center (<http://data.cma.cn/>, accessed 15 May 2021). Air pollution data of SJZ during the study period, which included the daily 24-hour average concentration of PM_{2.5}, PM₁₀, SO₂, NO₂, CO and the maximum 8-hour mean concentration of O₃, were obtained from the China National Urban Air Quality Real-time Publishing Platform (<https://air.cnemc.cn:18007/>). There are seven monitors (Xinan Gaojiao, Xibei Shuiyuan, Renmin Huitang, Zhi Gong Yiyuan, Gaoxinqu, Shiji Gongyuan, 22 Zhong Nanxiaoqu) in SJZ main urban area. We used the average air pollutant concentrations calculated as the mean values from 7 monitors for following analysis. Missing environmental and meteorological data points were imputed using the mean of adjacent values.

Statistical analysis

Time-series analyses have been extensively used to examine the immediate impacts of ambient pollutants and meteorological conditions on human health²⁸. In this study, we used a GLM to quantify the associations²⁹. The primary exposure variables consisted of the daily averages of individual air pollutants. We used a time-series regression analysis based on the Poisson distribution family and natural splines, to estimate the short-term relationship between PM_{2.5} and pediatric outpatient visits for neurological disorders.

Disease type	ICD coding range
Central infectious encephalopathy	G00–G09
Noninfectious acute encephalopathy	G35–G37; G40–G47; G91–G96
Chronic brain dysfunction disease	G10–G14; G20–G26; G80–G83

Table 1. Classification of neurological diseases based on ICD-10 codes.

Adjustments for factors in the meteorological data and the daily average concentration of PM_{2.5} were introduced into the GLM in order to control for potential confounding effects. Factor variables were used to adjust for weekdays and holidays. The model is described by Eq. (1):

$$\text{Log}[E(Y_t)] = \beta Z_t + ns(\text{time}, df) + ns(X_t, df) + DOW + \text{holiday} + \text{intercept} \quad (1)$$

where $E(Y_t)$ represents the daily expected outpatient visits on day t ; β signifies the regression model coefficients; Z_t represents the daily concentration of PM_{2.5} in the single pollutant model and the concentrations of both PM_{2.5} and the respective co-pollutant (O₃, SO₂, or NO₂) in the two-pollutant model; DOW and holiday are dummy variables representing the weekdays and holidays, respectively; ns refers to the natural smooth spline function; time corresponds to the date; df represents the degrees of freedom; and X_t encompasses the daily weather factors, including daily average temperature, relative humidity, atmospheric pressure, wind speed, and sunshine duration. Natural smooth spline functions were adopted to control for the time trend in the dates, with seven degrees of freedom per year³⁰. For the seasonal analysis, three degrees of freedom per year was used. The daily average temperature was adjusted with six degrees of freedom, while the other meteorological factors were adjusted with three degrees of freedom each.

Single-day lags (lag0 to lag7) and moving average lags (lag01 to lag07) were included to determine the lag day with the most significant effect, and this was identified as the optimal effect period. The effect estimate was defined as the ER and its 95% confidence interval (95% CI) for daily outpatient visits resulting from an increase in daily ambient pollutant concentrations. In addition, stratified analyses were used to investigate potential effect modifiers based on sex, age, disease type, and season to assess the effect of exposure to PM_{2.5} on neurological outpatient visits. The cold season was defined as November to April of the subsequent year, while the warm season spanned from May to October. We conducted one-way ANOVA tests to examine significant differences between the effect estimates from stratified groups.

Informed consent

The study was approved by the Shijiazhuang Center for Disease Control and Prevention Institutional Ethical Review Board Committee (#2024-06). All methods were carried out in accordance with guidelines and regulations, including the Declaration of Helsinki. The dataset included age, sex, the date of visit, and ICD-10 diagnosis code for each outpatient visit. There was no personal identifiers such as names, identification numbers, or residential addresses collected or used in the analysis. Therefore, the data of pediatric neurological outpatient visits were anonymous, and the requirement for informed consent was therefore waived.

Sensitivity analysis

A two-pollutant model was constructed to evaluate the robustness of the single-pollutant model that was developed in order to account for potential interaction effects in the model. Co-pollutants with absolute values of the Spearman's correlation coefficient < 0.7 were included in the two-pollutant model in this study³¹.

All of the analyses were conducted using the program R 4.2.0.

Results

Descriptive statistics

Table 2 summarizes the air contaminant statistics, meteorological parameters, and outpatient visits for NSDs of children. A total of 154,348 records for NSDs were collected between January 1, 2013, and December 31, 2021, with a median daily outpatient visit count of 43 cases. During the study period, the daily PM_{2.5} concentrations ranged from 6.3 µg/m³ to 771.3 µg/m³, and the IQR was 75.2 µg/m³.

The percentages of boys and girls with NSDs were 59.3% and 40.7%, respectively. The percentages of patients aged 0–6 and 7–14 years with NSDs were 71.4% and 28.6%, respectively. The total daily pediatric neurological outpatient visits ranged from 0 to 153, with a median (P25, P75) of 43 (31, 61). The medians (P25, P75) of the daily pediatric neurological outpatient visits for boys and girls were 25 (17, 37) and 18 (12, 24), respectively. The medians (P25, P75) of the 0–6- and 7–14-year-old groups were 31 (18, 47) and 13 (9, 17), respectively. The volume of central infectious encephalopathy, noninfectious acute encephalopathy, and chronic brain dysfunction disease outpatient visits ranged from 0 to 69, 0 to 89, and 0 to 14, with medians (P25, P75) of 6 (2, 13), 32 (24, 43), and 1 (0, 2), respectively. Figure 1 shows a strong correlation ($r=0.92$) between the daily concentrations of PM_{2.5} and PM₁₀. PM_{2.5} exhibited positive correlations with carbon monoxide (CO) ($r=0.80$), NO₂ ($r=0.66$), and SO₂ ($r=0.63$), as well as with air pressure ($r=0.22$) and humidity ($r=0.22$), and a negative correlation with O₃ ($r=-0.35$).

Associations between the PM_{2.5} concentrations and pediatric neurological outpatient visits

We investigated with effect of air pollution concentration on the daily number of outpatients with NSDs with a single-pollutant model. We considered how different lag times impacted these effects. Figure 2 shows lag effects for PM_{2.5} on total neurological outpatient visits among children were statistically significant for single-day lag times of 3 to 6 days, as well as moving average of 4 days and 7 days. The largest lag effect was observed at a moving average of 7 days, with a per IQR (75.2 µg/m³) increase in PM_{2.5} correlated with a 2.047% increase in the total pediatric neurological outpatient visits (95% CI 0.66%–3.45%).

Table 3 shows the stratified analysis results. For girl outpatients, at lag07, every IQR increase in PM_{2.5} was associated with a 3.96% increase (95% CI 1.77%–6.20%). For boy outpatients, at lag4 every IQR increase in PM_{2.5} was associated with a 1.00% increase (95% CI 0.14%–1.87%). The difference between the sex groups was statistically significant at lag5 and lag07, and girls were more sensitive to PM_{2.5} ($P<0.05$).

Variable	Mean(SD)	Min	Median(P25, P75)	Max
Kind-specific diseases				
All	47(21.9)	0	43(31, 61)	153
Central infectious encephalopathy	9.3(10.5)	0	6(2, 13)	69
Noninfectious acute encephalopathy	33.8(13.8)	0	32(24, 43)	89
Chronic brain dysfunction disease	1.42(1.71)	0	1(0, 2)	14
Sex				
Male	27.9(13.9)	0	25(17, 37)	93
Female	19.1(9.2)	0	18(12, 24)	73
Age (years)				
0–6	33.5(19.2)	0	31(18, 47)	120
7–14	13.4(6.2)	0	13(9, 17)	44
Air pollution exposure*				
PM _{2.5} (µg/m ³)	96.9 (78.6)	6.3	61 (35.9, 111.1)	771.3
PM ₁₀ (µg/m ³)	159.9 (121)	13	124.9 (78.6, 200.3)	867.3
SO ₂ (µg/m ³)	38.5 (46.7)	2	20.7 (11, 43.7)	319.4
NO ₂ (µg/m ³)	49.3 (25.8)	7.4	44.7 (30.7, 62.3)	188
O ₃ (µg/m ³)	93.6 (59.3)	1.9	84.7 (47.3, 132.4)	309.6
CO (mg/m ³)	1.3 (1.1)	0.1	1 (0.7, 1.4)	10.5
Meteorological conditions				
Temperature (°C)	14.8 (10.7)	-10.2	15.9 (5, 24.5)	34.7
Air pressure (kpa)	100.0 (10.0)	98.2	100.5 (99.6, 101.3)	103.4
Relative humidity (%)	56.1 (20.3)	4.9	57 (40, 72.3)	100
Wind speed (m/s)	1.9 (0.8)	1.3	1.8 (1.3, 2.2)	6.5
Sunshine duration (h/d)	5.8 (4.4)	0	7 (0, 9.4)	13.8

Table 2. Descriptive characteristics of daily pediatric neurological outpatient visits, air pollution, and meteorological data. ALL = Total daily pediatric neurological outpatient visits. *Air pollution concentrations represent the 24-hour average concentrations for PM_{2.5}, PM₁₀, SO₂, NO₂, CO, and maximum 8-hour mean concentration for O₃.

As shown in Table 3, in the children aged 0–6 years group, the association between PM_{2.5} and outpatient visits was the highest at lag07 (ER: 2.66%, 95% CI 1.05–4.30%). There was no statistically significant excess risk of daily outpatient visits associated with PM_{2.5} for children aged 7–14 for lag07. An analysis of the inter-group differences by age group was statistically significant at lag3, and 0–6-year-old were more sensitive to PM_{2.5} ($P < 0.05$).

The results of the analysis of the different NSD types are presented in (Fig. 2). For central infectious encephalopathy diseases, the lag impacts of PM_{2.5} were statistically significant for lag0–lag3 and lag01–lag07. The largest lag effect was discovered for lag03, with every IQR increase in PM_{2.5} corresponding to a 4.19% increase in outpatient visits (95% CI: 1.73%–6.71%). The lag effects of PM_{2.5} were statistically significant for lag4–lag6 and lag07 for noninfectious acute encephalopathy diseases. The largest lag effect was observed for lag07, with every IQR increase in PM_{2.5} corresponding to a 1.69% increase in outpatient visits (95% CI: 0.07%–3.33%). For chronic brain dysfunction diseases, the lag effects of PM_{2.5} were statistically significant for lag1 and lag01–lag04. The largest lag effect was observed for lag03, with every IQR increase in PM_{2.5} corresponding to a 7.84% increase in outpatient visits (95% CI: 1.65%–14.41%).

Seasonal differences in the effect of PM_{2.5} exposure on pediatric neurological outpatient visits

As shown in Table 3, the annual data were categorized into cold and warm seasons for a seasonal differences analysis in order to further analyze the effect of PM_{2.5} on neurological outpatient visits of children across various seasons. In the cold season, at lag07 every IQR (114 µg/m³) increase in PM_{2.5} was associated with a 2.84% increase (95% CI: 0.29%–5.46%). However, the effect of PM_{2.5} on NSDs was more pronounced during the warm season compared to the cold season. Every IQR (47 µg/m³) increase in PM_{2.5} corresponded to a 4.88% increase in outpatient visits (95% CI: 3.37%–6.41%) at lag07 during the warm season. The study of the seasonal differences between groups indicated that during the warm season, changes in the PM_{2.5} concentration had a greater impact on the number of outpatient visits for pediatric neurological diseases, and this was statistically significant ($P < 0.05$).

Sensitivity analysis

Based on the single-pollutant model, pollutants with an absolute value of Spearman's coefficient < 0.7 were included in the two-pollutant model. In the two-pollutant models, after adjusting for O₃, SO₂ and NO₂, the effects of PM_{2.5} on pediatric neurological outpatient visits remained statistically significant, indicating stable

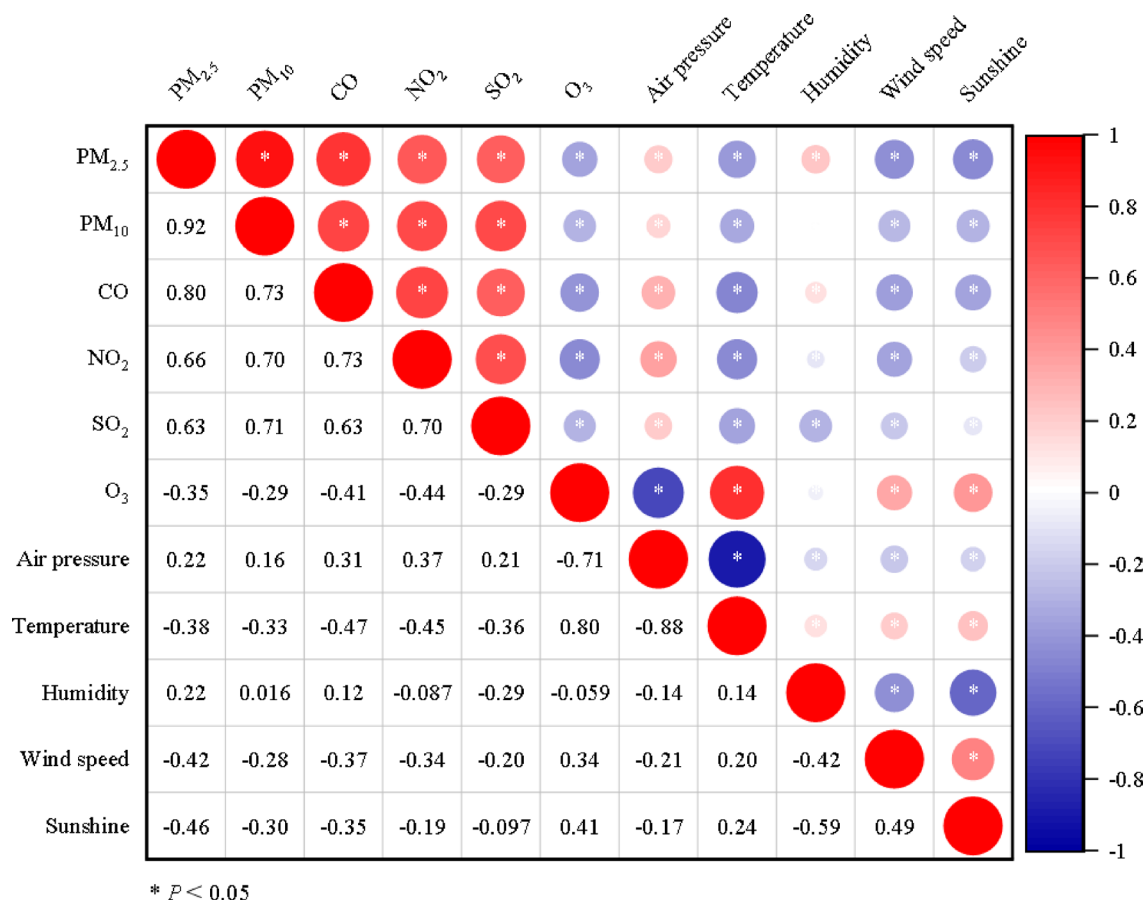


Fig. 1. Spearman's correlations between daily concentration of PM_{2.5} and other pollutants (PM₁₀, CO, NO₂, SO₂, O₃), meteorological condition in SJZ, 2013–2021.

results (Table 4). After adjusting for NO₂, the ER decreased to 1.43%, though the association remained statistically significant.

Discussions

In this study, we assessed the effects of short-term PM_{2.5} exposure on pediatric outpatient NSD visits based on data from pediatric outpatient visits, air pollutants, and meteorological factors spanning from 2013 to 2021. As a representative component of air pollution, studies have shown that PM_{2.5} can enter the bloodstream through the gas–blood barrier, reach the brain, and then invade the CNS through the BBB, potentially causing damage through oxidative stress, inflammation, and other mechanisms, and this may lead to the occurrence of NSDs^{32–35}. Our findings revealed a positive association between the PM_{2.5} concentration and outpatient visits for pediatric neurological diseases. The results also indicated that the effect estimates of the single-day lagged model were lower compared to those of the lagged model using moving averages. This indicated that PM_{2.5} negatively impacts children's nervous systems, with a particular emphasis on the accumulative effects of PM_{2.5} on children's nervous systems. This highlights the importance of understanding the short-term health impacts of PM_{2.5} exposure and the necessity for increased awareness regarding the health effects associated with prolonged pollutant exposure.

A stratified analysis revealed that girls showed higher sensitivity to PM_{2.5} exposure than boys. This result could have been due to differences in particle deposition between sexes³⁶ and variations in physiological structures and hormone levels³⁷, making girls' nervous systems more vulnerable. In addition, children aged 0–6 years were more sensitive to PM_{2.5} exposure than those aged 7–14 years, likely due to the rapid development of the nervous system during early childhood when the brain reaches approximately 90% of its adult size by age five³⁸, also as the study of 1,967 mother–child dyads from three pregnancy cohorts in the US found ambient air pollution were associated with impaired behavioral functioning and cognitive performance at 4 to 6 years of age³⁹. PM_{2.5} also has potential developmental toxicity⁴⁰, making younger children more susceptible. Therefore, the period from birth to six years is particularly critical, and parents should intensify efforts to protect their children from PM_{2.5} during this vulnerable stage.

According to association analysis between PM_{2.5} concentrations and the three NSD types, we found that PM_{2.5} exposure positively correlated with outpatient visits for central infectious encephalopathy, noninfectious acute encephalopathy, and chronic brain dysfunction, thus suggesting a broad impact on children's nervous

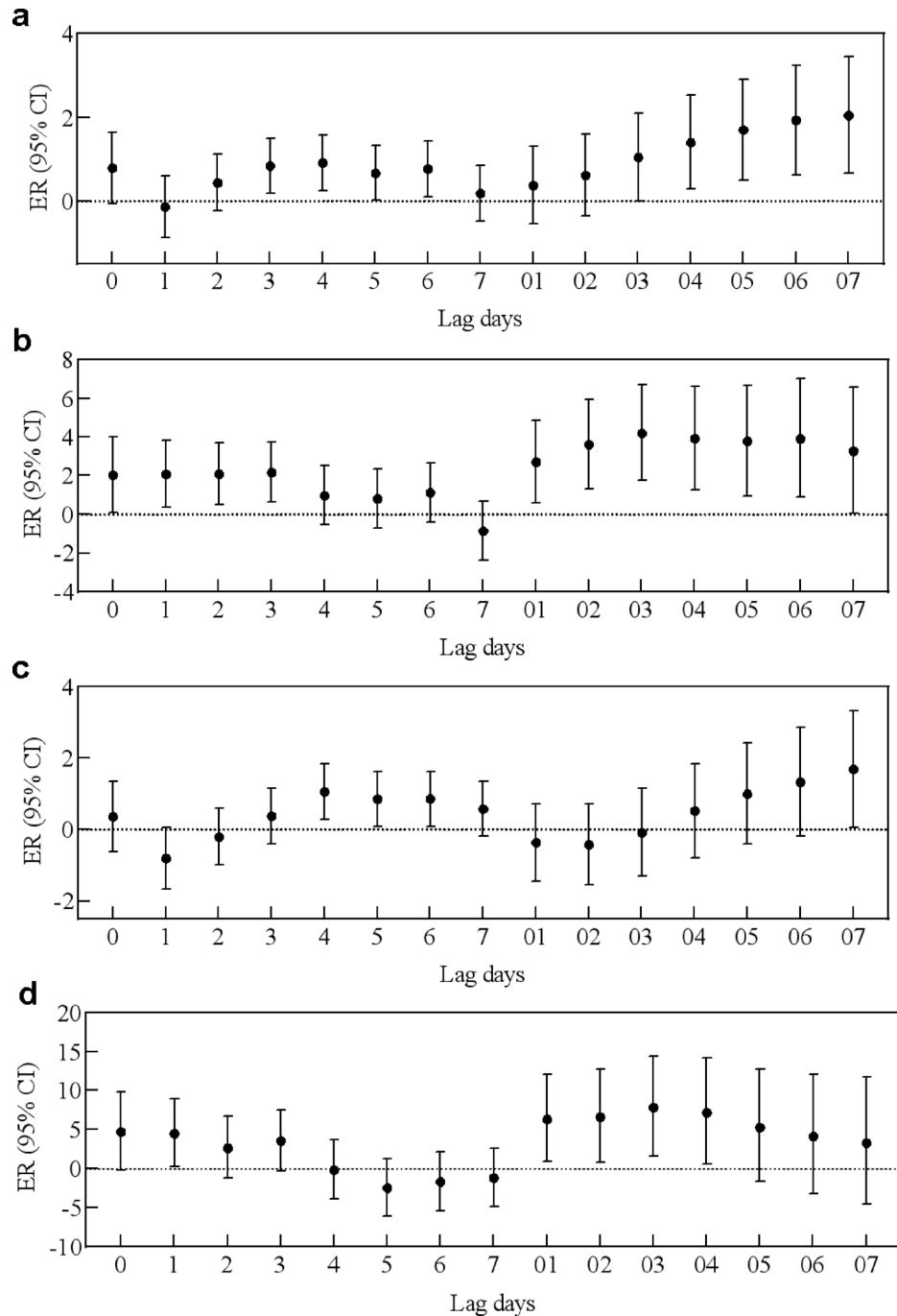


Fig. 2. ER (95% CI) of daily pediatric neurological outpatient visits associated with IQR increase in $PM_{2.5}$ concentrations by different lag days. (a) Total neurological outpatient visits; (b) Central infectious encephalopathy; (c) Noninfectious acute encephalopathy; (d) Chronic brain dysfunction disease.

systems^{15,23,41}. Meanwhile, neurological disorders showed varying sensitivities to $PM_{2.5}$ exposure. Extensive data in the previous studies supported that $PM_{2.5}$ could go through the BBB or otherwise breathe into the brain via the olfactory nerve, and other pathways, causing neurological inflammation, oxidative stress or damage to affect the nervous system with varying intensity^{42–44}. In addition, for the chronic brain dysfunction diseases, the study of Calderón-Garcidueñas⁴⁵ indicated that children in Mexico City exposed to $PM_{2.5}$ show systemic inflammation, blood-brain barrier disruption, cognitive deficits, and early Alzheimer's and Parkinson's pathology compared to those in clean-air environments. $PM_{2.5}$ could contribute to the pathological development of Alzheimer's disease in early life, offering further mechanistic support for the associations observed in our study. Additionally, pediatric outpatient visits for chronic brain dysfunction may also represent an exacerbation of health conditions resulting from chronic brain dysfunction caused by $PM_{2.5}$. Understanding these mechanisms can help tailor

Days of lag	Sex		Age		Season (IQR)	
	Female	Male	0–6 years	7–14 years	Warm season (47 µg/m ³)	Cold season (114 µg/m ³)
lag0	1.79 (0.47 ~ 3.12)	0.08 (-1.01 ~ 1.18)	0.56 (-0.42 ~ 1.54)	1.42 (-0.24 ~ 3.11)	3.15 (2.17 ~ 4.13)*	-0.12 (-1.64 ~ 1.42)
lag1	0.02 (-1.13 ~ 1.19)	-0.26 (-1.22 ~ 0.71)	-0.09 (-0.94 ~ 0.78)	-0.37 (-1.84 ~ 1.11)	1.75 (0.87 ~ 2.63)*	-0.78 (-2.08 ~ 0.54)
lag2	0.45 (-0.60 ~ 1.52)	0.42 (-0.46 ~ 1.31)	0.60 (-0.19 ~ 1.38)	-0.12 (-1.46 ~ 1.24)	1.37 (0.55 ~ 2.20)*	0.48 (-0.72 ~ 1.70)
lag3	0.61 (-0.42 ~ 1.65)	1.00 (0.14 ~ 1.87)	1.24 (0.47 ~ 2.01)*	-0.34 (-1.65 ~ 0.99)	1.60 (0.80 ~ 2.41)*	1.29 (0.10 ~ 2.48)
lag4	1.22 (0.19 ~ 2.26)	0.69 (-0.16 ~ 1.56)	1.06 (0.30 ~ 1.83)	0.53 (-0.79 ~ 1.85)	1.52 (0.72 ~ 2.32)	1.58 (0.41 ~ 2.77)
lag5	1.70 (0.67 ~ 2.74)*	-0.06 (-0.91 ~ 0.80)	0.74 (-0.02 ~ 1.50)	0.41 (-0.90 ~ 1.73)	1.41 (0.61 ~ 2.22)*	1.16 (-0.01 ~ 2.35)
lag6	1.54 (0.51 ~ 2.58)	0.22 (-0.63 ~ 1.08)	1.00 (0.24 ~ 1.77)	0.03 (-1.27 ~ 1.35)	0.75 (-0.05 ~ 1.55)	1.54 (0.37 ~ 2.72)
lag7	0.88 (-0.15 ~ 1.91)	-0.31 (-1.16 ~ 0.55)	0.23 (-0.53 ~ 0.99)	0.05 (-1.25 ~ 1.37)	1.24 (0.45 ~ 2.04)*	-0.21 (-1.37 ~ 0.97)
lag01	1.09 (-0.34 ~ 2.55)	-0.14 (-1.32 ~ 1.07)	0.29 (-0.78 ~ 1.36)	0.56 (-1.25 ~ 2.40)	3.20 (2.12 ~ 4.28)*	-0.58 (-2.20 ~ 1.07)
lag02	1.12 (-0.40 ~ 2.67)	0.25 (-1.02 ~ 1.53)	0.67 (-0.47 ~ 1.81)	0.36 (-1.55 ~ 2.32)	3.31 (2.17 ~ 4.48)*	0.06 (-1.67 ~ 1.81)
lag03	1.32 (-0.31 ~ 2.98)	0.84 (-0.52 ~ 2.22)	1.33 (0.12 ~ 2.56)	0.14 (-1.91 ~ 2.23)	3.71 (2.49 ~ 4.95)*	0.99 (-0.87 ~ 2.89)
lag04	1.90 (0.15 ~ 3.68)	1.05 (-0.40 ~ 2.52)	1.74 (0.44 ~ 3.06)	0.38 (-1.81 ~ 2.62)	4.11 (2.81 ~ 5.42)*	1.71 (-0.31 ~ 3.77)
lag05	2.76 (0.87 ~ 4.69)	0.95 (-0.61 ~ 2.53)	2.08 (0.68 ~ 3.50)	0.51 (-1.84 ~ 2.92)	4.50 (3.12 ~ 5.90)*	2.29 (0.11 ~ 4.52)
lag06	3.40 (1.35 ~ 5.48)	0.90 (-0.77 ~ 2.60)	2.48 (0.97 ~ 4.01)	0.29 (-2.23 ~ 2.87)	4.58 (3.14 ~ 6.05)*	2.84 (0.47 ~ 5.27)
lag07	3.96 (1.77 ~ 6.20)*	0.72 (-1.06 ~ 2.53)	2.66 (1.05 ~ 4.30)	0.24 (-2.44 ~ 3.00)	4.88 (3.37 ~ 6.41)*	2.84 (0.29 ~ 5.46)

Table 3. ER (95% CI) of daily pediatric neurological outpatient visits associated with every IQR increase in PM_{2.5} concentrations in different subgroups. Warm season = April to October; Cold season = November to March of the subsequent year. *The difference between groups was statistically significant ($P < 0.05$).

Co-pollutants	ER (95% CI)
Main model	2.05 (0.66 ~ 3.45)
PM _{2.5} +O ₃	2.12 (0.73 ~ 3.53)
PM _{2.5} +SO ₂	2.09 (0.70 ~ 3.49)
PM _{2.5} +NO ₂	1.43 (0.04 ~ 2.84)

Table 4. ER (95% CI) of daily pediatric neurological outpatient visits associated with every IQR increase in PM_{2.5} concentrations by the multi-pollutant model.

disease management strategies to specific vulnerabilities. These findings underscore the need for targeted public health interventions and policies to reduce PM_{2.5}-related neurological harm in children.

The results of seasonal difference suggested a stronger association between PM_{2.5} exposure and pediatric neurological diseases during warmer seasons, likely due to the synergistic effects of high temperatures and air pollution, that exacerbate physiological stress, inflammation, and oxidative damage. While PM_{2.5} concentrations are typically higher during winter⁴⁶, our findings indicated a stronger correlation during the warm season, consistent with Qiu et al.⁴⁷. Notably, the impact of PM_{2.5} on pediatric neurological visits peaked at lag07 during the warm months, suggesting a cumulative effect. Temperature plays a crucial role in this association, with high temperatures linked to increased risk of neurological disorders^{48,49}. Lee found that air pollution could trigger migraines on hot days, suggesting a synergistic effect between PM_{2.5} and temperature⁵⁰. However, other studies have reported different seasonal impacts, such as increased migraines during colder months⁵¹ and no seasonal effect on epilepsy hospitalizations⁵². Further research should explore the combined effects of PM_{2.5} and temperature and consider additional meteorological factors, such as sunshine duration and humidity.

In addition to the PM_{2.5} single-pollutant model, we explored two-pollutant models to investigate the effects of incorporating other pollutants (O₃, SO₂, and NO₂ for PM_{2.5}). As indicated in Table 4, the findings from the PM_{2.5} model remained statistically significant even after the addition of O₃, NO₂, and SO₂ as risk factors, indicating that these co-pollutants did not modify PM_{2.5}'s effect on neurological visits. This could have been due to the same source of these pollutants (e.g., vehicle exhaust or the burning of fossil fuels), and thus no additive effect was

detected⁵³. Additionally, when adjusting for NO₂, the ER for PM_{2.5} decreased to 1.43%, as compared to 2.05% in the main model, suggesting potential confounding or interaction effects.

In this study, we focused on pediatric neurological diseases, specifically examining the effects of short-term PM_{2.5} exposure on neurological diseases in children, thus filling a gap in research on the neurological health risks of air pollutants in this vulnerable population. In addition, we conducted sex and age stratified analyses, providing a basis for targeted health interventions. Utilizing outpatient and environmental data from SJZ from 2013 to 2021, the study provides long-term data support and examines the associations between PM_{2.5} exposure and pediatric neurological diseases across different seasons. This approach helps to understand the combined effects of temperature and pollutants on children's health, provides insights for future multifactorial research, and offers a data reference for public health interventions.

Strengths and limitation

This study has several limitations. First, there may be heterogeneity in the diagnosis of NSDs that could potentially lead to differences in disease classifications, and the limited number of chronic brain dysfunction diseases visits constrains the generalizability of the analysis results, necessitating further research to substantiate these findings. Second, we relied on pollutant concentration data from urban environmental monitors rather than accurate individual exposure values; this means that there may be disparities between these results and the real-life effects of PM_{2.5}. Meanwhile, due to data accessibility constraints, socioeconomic factors were not included in the model to be analysed.

Conclusion

In this study, we found a significant association between PM_{2.5} exposure and pediatric neurological disorders, especially in girls and children aged 0–6 years, thus highlighting the need for targeted public health interventions. Parents and caregivers should take proactive measures, such as reducing outdoor activities and using air purifiers during high-pollution periods, especially during warmer seasons. Local and national governments should prioritize air quality improvements in urban areas, especially during summer when PM_{2.5} has more severe health impacts. Policies aimed at reducing emissions from vehicles and industries combined with public education are essential to protect children's neurological health.

In summary, in this study, we revealed that PM_{2.5} exposure was linked to increased outpatient visits for pediatric neurological disorders in SJZ, with a stronger association observed during the warm season. The identification of high-risk groups, such as girls and young children, underscores the importance of protective measures to safeguard children's neurological health during periods of high pollution and temperature.

Data availability

Ambient air quality data during the study period (PM_{2.5}, PM₁₀, O₃, CO, SO₂, NO₂) were obtained from the China National Urban Air Quality Real-time Publishing Platform (<https://air.cnemc.cn:18007/>). The meteorological data (e.g., daily average temperature, relative humidity, atmospheric pressure, sunshine duration, and wind speed) during the study period were obtained from the National Meteorological Science Data Center (<http://data.cma.cn/>). The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

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Author contributions

Z. L. and A. G. conducted relevant research and wrote the paper, Y. Z. was responsible for determining the research direction, conducting academic review and revision of the manuscript, and provided guidance on experimental design and data analysis methods. H. K., F. C., H. M., X. Z., X. S., X. Y., Y. S. assisted in conducting research and revised the paper together.

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Declarations

Competing interests

The authors declare no competing interests.

Additional information

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