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Effect of pregnancy and infancy exposure to outdoor particulate matter (PM_1, $PM_{2.5}$, PM_{10}) and SO_2 on childhood pneumonia in preschool children in Taiyuan City, China☆

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ABSTRACT

There is currently a paucity of research on the effects of early life exposure to particulate matter (PM) of various size fractions on pneumonia in preschool-aged children. We explored the connections between antenatal and postnatal exposure to atmospheric pollutants and diagnosed pneumonia among 4814 offspring children in Taiyuan City, northern China. Outdoor air pollutant concentrations and ambient temperature were collected. A machine learning-based model was utilized to compute daily mean concentrations of PM_{10}, $PM_{2.5}$, and PM_1 at the home address. Associations were calculated using generalized linear mixed models, and stratified analysis was used to detect sensitive subpopulations. We observed significant associations between prenatal exposure to atmospheric pollutants and the incidence of pneumonia in children. For every 10 μg/m^3 increase, the odds ratios (ORs) were 1.06 for PM_{10}, 1.15 for $PM_{2.5}$, 1.24 for PM_1, and 1.05 for SO_2 for the whole pregnancy period. In mid-pregnancy, the most vital connections were found for PM_{10}, $PM_{2.5}$, and PM_1 exposure. Girls showed higher sensitivity to exposure to $PM_{2.5}$ and PM_{10}. The most significant connections between PM and pneumonia were observed at high SO_2 exposure. Connections between PM_1, $PM_{2.5}$ and pneumonia were stronger in children without environmental tobacco smoke (ETS) at home. Associations between PM_{10} and pneumonia were stronger in children with ETS at home. The synthesis of the data suggests that exposure to PM_{10}, $PM_{2.5}$, PM_1, and SO_2 during pregnancy contributes to an elevated susceptibility to childhood pneumonia. The second trimester period is significant and represents a critical window of vulnerability. PM_1 may have the strongest impact. Exposure to SO_2 can further enhance the PM related risks of pneumonia. Gender and ETS exposure at home can modify associations between outdoor PM and pneumonia. Further reductions in outdoor PM, especially PM_1, are needed to reduce childhood pneumonia in China.

1. Introduction

Pneumonia continues to rank as a major contributor to childhood mortality globally, accounting for roughly 14% of yearly fatalities in children aged 5 and below (Liu et al., 2016). In China, children aged 0–4 years experienced a significantly higher occurrence of pneumonia (0.08 instances per child per year) contrasted to rates observed in developed nations (0.01–0.03 instances per child per year) (Rudan et al., 2013). Various microorganisms, including bacteria, viruses, and fungi, can contribute to pneumonia (Kennedy et al., 2018). Most fatalities attributed to pneumonia are concentrated in developing nations, particularly in regions such as sub-Saharan Africa and South Asia (Nguyen et al., 2019; Walker et al., 2013). The mortality rate in these areas has shown a continuous increase (UNICEF, 2016). Childhood pneumonia is highly

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prevalent in urban areas of China (Norbäck et al., 2018). Although there has been a decline in pneumonia-related deaths, the pace of progress towards meeting the Sustainable Development Goal of reducing avoidable fatalities within the neonatal and under-five age groups by 2030 is not as rapid as hoped (Health - United Nations Sustainable Development).

Globally, particulate matter (PM) not only poses a great threat to health, but also seriously pollutes the ambient environment. In the last five years, numerous researchers have highlighted the connections between PM, specifically PM_{10} and $PM_{2.5}$, and pneumonia among children (Liang et al., 2022; Pu et al., 2021; Shi et al., 2021). Nonetheless, the influence of PM_1 on childhood pneumonia remains limited. PM_1, a subset of $PM_{2.5}$, which is part of PM_{10}, could potentially pose greater health risks compared to other constituents of PM_{10} and $PM_{2.5}$, like $PM_{1–2.5}$ (Chen et al., 2017; Lin et al., 2016). Moreover, PM_1 particles, being smaller in diameter contrasted to $PM_{2.5}$ and PM_{10}, have the capability to penetrate deeper into the lungs, potentially causing damage to the lower lobes. Firstly, the micrometer-scale size of PM_1 enables them to pass through alveoli and enter the bloodstream. A model prediction study indicated that the deposition efficiency and distribution of 0.03 μm particles in the lungs were significantly higher than those of 0.3 μm particles (Park and Wexler, 2008). Research has shown that the smaller the particle size, the higher the deposition efficiency in the human respiratory system and the greater the oxidation potential per unit mass (Biswas et al., 2009). Secondly, the primary sources of PM_1 include industrial combustion and vehicle exhaust, and its surface is prone to adsorbing heavy metals (such as lead and nickel) and polycyclic aromatic hydrocarbons (PAHs). Studies have demonstrated that exposure to PM_1 is more significantly associated with an increased risk of hospitalization for respiratory diseases, especially among elderly patients (Zhong et al., 2025). Other research has revealed the high pollution levels of PM_1 and its mutagenicity and cytotoxicity (Goswami et al., 2024).

Additionally, research has found that exposure to PM_1 increases the diversity of the intestinal microbiota in newborns and alters its community structure. PM_1 exposure during pregnancy may impact newborns' health by influencing their intestinal microbiota (Cao et al., 2023). Furthermore, long-term exposure to PM_1 is significantly associated with an increased risk of preterm birth, particularly among young, rural, and less educated mothers (Wang et al., 2018). Research on the health impacts associated with PM_1 is still relatively restricted in epidemiological studies. Therefore, there is a crucial need for epidemiological investigations focusing on the relationship between PM_1 exposure and childhood pneumonia. Furthermore, previous research indicated that pollutants originating from traffic, such as nitrogen dioxide (NO_2), may increase the susceptibility of preschool children to pneumonia (Lu et al., 2014). A study conducted in Hefei, Anhui Province, indicated that PM_1 has a more pronounced immediate effect on pneumonia among children than PM_{10} and $PM_{2.5}$ (Wang et al., 2021). One recent study conducted in Hanoi demonstrated that PM_{10}, $PM_{2.5}$, PM_1, and NO_2 can be linked to hospital admissions for childhood pneumonia (Nguyen et al., 2024). In another study, it was observed that children exposed to high temperatures both before conception and during pregnancy showed heightened vulnerability to the impact of exposure to industrial pollutant sulfur dioxide (SO_2), resulting in a heightened likelihood of pneumonia, contrasted to those exposed to low temperatures (Lu et al., 2021).

Recent investigations indicated that early environmental exposures significantly influence lung health outcomes in preschool children (Deng et al., 2016, 2015; Jiang et al., 2018). In recent years, researchers have shown a rising interest in investigating the link between prenatal atmospheric pollutants exposure and the occurrence of pneumonia in children (Liu et al., 2020; Lu et al., 2021; Shi et al., 2021). A study undertaken in Changsha indicated that traffic pollutant NO_2 exposure throughout the entire gestational period, especially during the prior to conception and early gestational phases, was associated with a higher likelihood of preterm birth (Lu et al., 2019). A cellular study shows that ozone (O_3) inhalation triggers asthmatic airway hyper reactivity (AHR) (Ray et al., 2025). Another study also showed that O_3 exposure significantly affected bronchial barrier function, inflammatory response and cell viability, especially in high concentrations (400 ppb) (Huang et al., 2025). Although previous research has indicated an association between exposure to outdoor environmental factors and an elevated risk of childhood pneumonia, studies are needed to focus on the influence of early environmental factors during pregnancy and infancy, specifically PM_1, on childhood pneumonia.

Our investigation sought to explore the connection between pneumonia incidence among offspring children in Taiyuan, northern China, and their pregnancy and infancy exposure to ambient air pollution, including PM_{10}, $PM_{2.5}$, PM_1, O_3, SO_2, and NO_2. Additionally, stratified analyses were performed to identify vulnerable subgroups, categorized by gender, age, breastfeeding status, and delivery method were conducted to identify vulnerable subgroups.

2. Material and methods

2.1. Study subjects

2.1.1. Study location

Taiyuan is located in the Taiyuan Basin of Shanxi Province, China (111°30′–113°09′ East, 37°27′–38°25′ North), and features a warm temperate continental monsoon climate. The city spans an area of 6988 km^2 and is home to a cohort of 5.4 million. Taiyuan, nestled among mountains to the North, East, and West, is flanked by the Fenhe River. The region experiences significant day-and-night temperature variations, and its semi-closed geography restricts the dispersion of pollutants (Wang et al., 2023). The prevalence of coal in industrial and energy sectors, along with these geographical factors, amplifies outdoor air pollution and leads to declining air quality.

2.1.2. Study population

Taiyuan is among the seven cities participating in the subsequent stage of the multi-center China Children Homes Health (CCHH) study. In this study, children from stochastically selected daycare centers in these cities across China were invited to participate. In Taiyuan, the second phase of the CCHH study included 15 kindergartens in six urban areas of Taiyuan (Fig. S1). We invited all 5394 children attending those day care centers to join our study. The questionnaire was distributed to their guardians or parents of each child, with questions on health status, personal factors and home environment of the children. The questionnaire was answered by one adult from each family, who could be either a parent or another guardian of the children. Out of 5394 questionnaires received, each representing one child, 580 questionnaires had missing information on many questions and were excluded. Finally, this study encompassed 4814 valid questionnaires.

2.2. Health outcome

We identified children with pneumonia based on a corroborative response to a question: "Has the child received the diagnosis of pneumonia before?"

2.3. Covariates

The data for covariates were collected through questionnaires. Covariates considered in our study comprised: (1) personal characteristics: gender (boys/girls), breastfeeding (No breastfeeding/0–1/1–4/4–6/6–12/>12 months); (2) residential factors: environmental tobacco smoke (ETS), family heating methods in winter, and visible mold/damp stains.

2.4. Personal exposure to ambient air pollution and temperature

We employed a sophisticated machine learning method called the enhanced space-time extremely randomized trees model to predict the daily average concentrations of ambient PM_{10}, $PM_{2.5}$, and PM_1 throughout Taiyuan from January 2013 to December 2018. This method allowed for a spatial resolution of 1 km^2. We used residential addresses to quantify the ambient PM_{10}, $PM_{2.5}$, and PM_1 exposure. The PM concentration prediction model proposed in this paper integrates ground monitoring, satellite remote sensing and meteorological data, and achieves high-precision prediction through machine learning methods. The R^2 of the model during calibration and validation periods reached 0.81 and 0.79, respectively, with RMSE of 8.6 μg/m^3 and 9.1 μg/m^3, indicating a small prediction error. Meanwhile, the MAE was 5.8 μg/m^3 and 6.3 μg/m^3, respectively, further verifying the reliability of the model. The correlation coefficient between the predicted and actual values was 0.89 during the validation period, indicating a high correlation between the predicted and actual values, and the model has good performance (Wei et al., 2019; Wu et al., 2022). Data regarding ambient air pollutants, including O_3, SO_2, and NO_2, were obtained from the nearest national air quality monitoring site to the residential addresses of the participants (Fig. S1). This included daily averages for NO_2 and SO_2 and the highest 8 h average concentration of O_3 (O_3-8 h). The distribution of air pollution and temperature data are shown in Table 2.

2.5. Exposure time windows

In this study, we examined two distinct exposure periods: prenatal (during pregnancy) and postnatal (during infancy). Pregnancy was characterized as the duration starting from the mother's last menstrual period at conception and ending with the delivery of the child. Additionally, we calculated the average exposure concentrations of various particulate matter during each trimester of gestation. The first trimester was delineated as the period spanning from 1 to 13 weeks of gestation, while the second trimester spanned 14–26 weeks. The third trimester lasted from 27 weeks to delivery. Infancy was characterized as the time from the delivery of the child to their first year.

2.6. Statistical analysis

We carried out several analyses. First, we conducted a Spearman correlation analysis to analyze the connections between outdoor pollution factors during pregnancy and infancy. Second, we utilized single-pollution models to assess the relationships between outdoor environmental factors and childhood pneumonia during different time windows. Two-pollution models were employed to evaluate the combined impacts of multiple outdoor pollution factors on childhood pneumonia during pregnancy and infancy. Subsequently, we analyzed PM exposure during the three trimesters of gestation. These analyses primarily utilized generalized linear mixed models (GLMM), which are capable of handling issues related to non-normally distributed error structures or correlated data. Extending from linear mixed models (LMM), GLMM allows for the consideration of both fixed and random effects, accommodating situations where the dependent variable may not follow a normal distribution. The models primarily aimed to evaluate the relationships between ambient environmental conditions and childhood pneumonia while accounting for the relevant covariates. Additionally, we conducted stratified analyses to explore the connections between PM exposure and health outcomes across various subgroups (sex, age, duration of breastfeeding, and delivery method). The association was quantified using odds ratios (ORs) along with corresponding 95% confidence intervals (CIs), with a statistical significance level defined as a two-sided P-value < 0.05. We conducted all data processing and model construction using R software (version 4.2.2), with support from the 'lmerTest' package.

3. Results

3.1. General characteristics of study participants

Table 1 describes the demographic profile of the children, with the final analysis including 4814 children. Among them, 1169 (24.3%) children were diagnosed with pneumonia by doctors. The majority of the subjects (90.2%) were aged between 3 and 5 years. Approximately 50% of the research subjects are boys, and simultaneously, 60.1% of the studied children were born via cesarean section. Moreover, 52.6% of the participants were breastfed for over 12 months, and the predominant winter heating method utilized was central heating (87.8%). Notably, indoor factors such as dampness (3.9% during pregnancy and 3.9% during infancy) and exposure to ETS during pregnancy and infancy (24.9% and 24.4%, respectively) were also considered. In the table, the sex statistics account for 4789 cases, while the heating method statistics account for 4732 cases, both totaling 4814. The main reason is that parents did not answer the corresponding questions when answering the questionnaire, leading to the data missing.

3.2. Outdoor air pollution

Table 2 summarizes the levels of outdoor atmospheric pollutants and temperature, and the relationships among different pollutants and temperature are shown in Table 3. We observed that the connection between O_3 and temperature was high ($r = 0.745$) during pregnancy, which reflects that temperature affects ozone concentration. PM_1 were moderately correlated with SO_2 ($r = 0.696$). We also observed comparable findings for $PM_{2.5}$ ($r = 0.684$). The concentration of PM_1 showed a positive correlation with NO_2 ($r = 0.391$) and SO_2 but negatively with O_3 ($r = -0.287$) and temperature ($r = -0.337$) during pregnancy. However, PM_1 was weakly positively correlated with NO_2, SO_2, O_3 and temperature in infancy. We also observed analogous findings for $PM_{2.5}$

Table 1
Demographic characteristics of investigated children, covariates (N = 4814).

Variables	Value
Doctor-diagnosed pneumonia, N (%)	
Yes	1169 (24.3%)
No	3645 (75.7%)
Age, N (%)	
3	1265 (26.3%)
4	1187 (24.7%)
5	1918 (39.8%)
6	444 (9.2%)
Sex, N (%) [a]	
Boys	2407 (50.0%)
Girls	2382 (49.4%)
Delivery method, N (%)	
Vaginal delivery	2898 (60.1%)
Cesarean delivery	1921 (39.9%)
Duration of breastfeeding, N (%)	
No breast-feeding	485 (9.6%)
< 1 month	204 (4.1%)
1–4 months	337 (6.7%)
4–6 months	391 (7.8%)
6–12 months	967 (19.2%)
> 12 months	2648 (52.6%)
Heating method, N (%) [a]	
Unheating in winter	11 (0.2%)
Central heating	4231 (87.8%)
Single-family heating	54 (1.1%)
District heating	436 (9.0%)
Dampness at home during pregnancy, N (%)	191 (3.9%)
Dampness at home during infancy, N (%) [b]	191 (3.9%)
ETS at home during pregnancy, N (%)	1227 (24.9%)
ETS at home during infancy, N (%) [b]	1178 (24.4%)

[a] The sum of the numbers and percentages [N (%)] in the paired groups may not equal the total count or 100% due to missing values.
[b] The period from the child's date of birth to their first birthday.

Table 2
The descriptive statistics of atmospheric pollutants and temperature during various early life stages (N = 4814).

Variables	Exposure windows	Mean ± SD	Percentiles				
			Min	P_{25}	P_{50}	P_{75}	Max
PM_1 (μg/m^3)	The First Trimester	39.79 ± 12.26	7.03	28.58	38.10	50.61	120.77
	The Second Trimester	38.17 ± 11.26	7.01	28.53	34.67	48.14	106.95
	The Third Trimester	126.13 ± 24.55	44.44	108.00	122.70	146.11	367.37
	Pregnancy	38.48 ± 5.24	17.63	35.30	38.22	42.26	92.22
	Infancy	38.65 ± 4.51	16.59	35.52	38.86	42.28	60.14
$PM_{2.5}$ (μg/m^3)	The First Trimester	68.56 ± 17.99	17.82	52.90	66.33	83.89	201.79
	The Second Trimester	65.61 ± 15.93	23.29	52.47	60.77	78.84	188.20
	The Third Trimester	64.54 ± 16.72	19.30	51.14	58.64	78.28	172.96
	Pregnancy	66.21 ± 8.51	27.13	60.27	66.41	72.50	160.74
	Infancy	66.52 ± 7.71	28.21	61.25	67.07	72.29	104.78
PM_{10} (μg/m^3)	The First Trimester	131.31 ± 29.91	30.41	107.62	128.69	153.59	376.21
	The Second Trimester	126.13 ± 24.55	44.44	108.00	122.70	146.11	367.37
	The Third Trimester	123.65 ± 24.56	38.69	105.31	119.78	143.51	296.28
	Pregnancy	126.92 ± 17.20	46.71	112.19	129.63	140.28	307.82
	Infancy	124.47 ± 14.73	48.32	112.61	125.75	135.52	179.57
NO_2 (μg/m^3)	Pregnancy	38.74 ± 5.52	18.73	34.33	37.42	43.11	58.12
	Infancy	40.45 ± 7.48	18.06	34.35	37.00	46.12	68.90
SO_2 (μg/m^3)	Pregnancy	74.31 ± 20.91	18.51	60.02	70.98	86.93	152.95
	Infancy	76.70 ± 16.83	16.50	63.69	75.19	87.69	118.92
O_3 (μg/m^3)	Pregnancy	60.96 ± 13.53	23.58	51.19	59.49	68.03	119.89
	Infancy	63.12 ± 9.97	22.23	55.12	63.98	68.91	124.24
T (°C)	Pregnancy	11.03 ± 2.89	4.15	8.37	10.57	13.81	19.86
	Infancy	11.26 ± 0.28	10.90	11.05	11.16	11.41	12.01

Abbreviations: SD, standard deviation; PM_1, fine particulate matter (≤ 1 μm in diameter); $PM_{2.5}$, fine particulate matter (≤ 2.5 μm in diameter); PM_{10}, inhalable particulate matter (≤ 10 μm in diameter); NO_2, nitrogen dioxide; SO_2, sulfur dioxide; O_3, ozone; T, temperature.

Table 3
Spearman correlation coefficients depicting the relationships between atmospheric pollutants and temperature across various early life stages (N = 4814).

	Pregnancy							Infancy						
	PM_1	$PM_{2.5}$	PM_{10}	NO_2	SO_2	O_3	T	PM_1	$PM_{2.5}$	PM_{10}	NO_2	SO_2	O_3	T
PM_1	1.000							1.000						
$PM_{2.5}$	**0.963**	1.000						**0.970**	1.000					
PM_{10}	**0.817**	**0.860**	1.000					**0.899**	**0.873**	1.000				
NO_2	0.391	0.403	0.517	1.000				0.521	0.490	0.522	1.000			
SO_2	0.696	0.684	0.448	0.187	1.000			0.488	0.543	0.462	0.015	1.000		
O_3	−0.287	−0.206	−0.049	0.191	−0.418	1.000		0.235	0.258	0.139	0.414	−0.132	1.000	
T	−0.337	−0.224	−0.010	0.220	−0.509	**0.745**	1.000	0.366	0.383	0.246	0.525	0.177	0.519	1.000

Bold figures indicate $P > 0.7$.

and PM_{10}.

3.3. Associations between outdoor environmental factors and pneumonia

Table 4 presents the ORs indicating the association between childhood pneumonia and a 10 μg/m^3 rise in the concentrations of each outdoor pollutant during pregnancy and infancy. In single-pollutant models, childhood pneumonia exhibited a significant relationship with PM_{10}, $PM_{2.5}$, and PM_1 during pregnancy, with the strongest association noted for PM_1. As for SO_2, we found that it also affects the risk of pneumonia in children during pregnancy. However, no significant effects of outdoor environmental pollutants on childhood pneumonia were identified in infancy. In trimester-stratified analyses, PM_{10}, $PM_{2.5}$, and PM_1 showed significant associations with childhood pneumonia, but only for exposure during the second trimester (Table 5). For instance, a rise of 10 μg/m^3 in PM_1, $PM_{2.5}$, and PM_{10} exposure during the second trimester corresponded to odds ratios for childhood pneumonia of 1.21 (95% CI: 1.10–1.35), 1.14 (95% CI: 1.07–1.21), and 1.06 (95% CI: 1.02–1.09), respectively. Table 6 displays the associations between childhood pneumonia and particulate matter in dual-pollutant models. The relationships between PM exposure and pneumonia in children during pregnancy remained robust after controlling for NO_2 and O_3 during pregnancy. However, two-pollutant models of particulate matter and SO_2 weakened the PM associations.

Table 4
Odds ratios (95% CI) of pneumonia in children associated with ambient pollutants exposure throughout pregnancy and infancy using single-pollutant models [a] (N = 4814).

	Pregnancy		Infancy	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
PM_1	**1.24 (1.08, 1.44)** **	**0.002**	1.05 (0.89, 1.25)	0.551
$PM_{2.5}$	**1.15 (1.05, 1.25)** **	**0.001**	1.03 (0.93, 1.14)	0.550
PM_{10}	**1.06 (1.02, 1.11)** **	**0.005**	1.01 (0.96, 1.07)	0.663
NO_2	1.14 (0.97, 1.33)	0.099	0.90 (0.75, 1.08)	0.248
SO_2	**1.05 (1.01, 1.10)** *	**0.010**	1.04 (0.99, 1.09)	0.129
O_3	0.97 (0.88, 1.06)	0.477	0.92 (0.82, 1.03)	0.113

OR (95% CI) was calculated for every 10 μg/m^3 increase in ambient pollutants during each specific time window.
*$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$.
[a] Single-pollutant model: adjustments were made for sex, breastfeeding, dampness, heating method, ETS and outdoor temperature for each time window.

3.4. Subgroup analyses

Since the previous statistical analyses did not find significant results in infancy, only pregnancy exposure was considered in the subgroup analysis. Fig. 1 presents subgroup-specific OR estimates, depicting the relationships between childhood pneumonia and prenatal outdoor PM_{10}, $PM_{2.5}$, and PM_1 exposure. Risk estimates were similar between

Table 5
Odds ratios (95% CI) of pneumonia in childhood linked to exposure to various types of particulate matter during different trimesters of pregnancy using single-pollutant models [a] (N = 4814).

	OR (95% CI)	*P*-value
The First Trimester		
PM_1	1.03 (0.97, 1.10)	0.332
$PM_{2.5}$	1.03 (0.98, 1.07)	0.195
PM_{10}	1.02 (0.99, 1.05)	0.119
The Second Trimester		
PM_1	**1.21 (1.10, 1.35)** ***	**< 0.001**
$PM_{2.5}$	**1.14 (1.07, 1.21)** ***	**< 0.001**
PM_{10}	**1.06 (1.02, 1.09)** **	**0.001**
The Third Trimester		
PM_1	1.03 (0.96, 1.10)	0.345
$PM_{2.5}$	1.02 (0.98, 1.07)	0.278
PM_{10}	1.02 (0.99, 1.05)	0.260

OR (95% CI) was calculated for every 10 μg/m^3 increase in ambient pollutants during each specific time window.
*$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$.
[a] Single-pollutant model: adjustments were made for sex, breastfeeding, dampness, heating method, ETS and outdoor temperature at different stages of pregnancy.

Table 6
Odds ratios (95% CI) of pneumonia in children associated with atmospheric pollutants exposure throughout pregnancy and infancy using two-pollutant models [a] (N = 4814).

		OR (95% CI)	*P*-value
Pregnancy			
PM_1	$+NO_2$	**1.22 (1.01, 1.46)** *	**0.032**
PM_1	$+O_3$	**1.24 (1.05, 1.46)** **	**0.008**
PM_1	$+SO_2$	1.16 (0.96, 1.41)	0.122
NO_2	$+PM_1$	1.04 (0.88, 1.24)	0.629
O_3	$+PM_1$	0.97 (0.89, 1.06)	0.517
SO_2	$+PM_1$	1.03 (0.98, 1.09)	0.174
$PM_{2.5}$	$+NO_2$	**1.13(1.02, 1.26)** *	**0.022**
$PM_{2.5}$	$+O_3$	**1.14 (1.04, 1.26)** **	**0.005**
$PM_{2.5}$	$+SO_2$	1.10 (0.98, 1.23)	0.103
NO_2	$+PM_{2.5}$	1.05 (0.88, 1.24)	0.601
O_3	$+PM_{2.5}$	0.97 (0.88, 1.06)	0.474
SO_2	$+PM_{2.5}$	1.03 (0.98, 1.09)	0.216
PM_{10}	$+NO_2$	1.05 (0.99, 1.11)	0.077
PM_{10}	$+O_3$	**1.06 (1.01, 1.11)** *	**0.018**
PM_{10}	$+SO_2$	1.04 (0.99, 1.10)	0.161
NO_2	$+PM_{10}$	1.05 (0.87, 1.25)	0.587
O_3	$+PM_{10}$	0.97 (0.88, 1.06)	0.478
SO_2	$+PM_{10}$	1.04 (0.98, 1.09)	0.084
Infancy			
PM_1	$+NO_2$	1.07 (0.87, 1.30)	0.529
PM_1	$+O_3$	1.01 (0.83, 1.22)	0.924
PM_1	$+SO_2$	0.96 (0.78, 1.18)	0.681
NO_2	$+PM_1$	0.88 (0.73, 1.07)	0.202
O_3	$+PM_1$	0.92 (0.82, 1.03)	0.116
SO_2	$+PM_1$	1.04 (0.99, 1.10)	0.123
$PM_{2.5}$	$+NO_2$	1.04 (0.92, 1.16)	0.551
$PM_{2.5}$	$+O_3$	1.01 (0.90, 1.13)	0.866
$PM_{2.5}$	$+SO_2$	0.97 (0.86, 1.11)	0.657
NO_2	$+PM_{2.5}$	0.89 (0.73, 1.07)	0.211
O_3	$+PM_{2.5}$	0.92 (0.82, 1.03)	0.117
SO_2	$+PM_{2.5}$	1.04 (0.99, 1.10)	0.123
PM_{10}	$+NO_2$	1.03 (0.96, 1.09)	0.437
PM_{10}	$+O_3$	1.00 (0.95, 1.06)	0.918
PM_{10}	$+SO_2$	0.99 (0.93, 1.05)	0.750
NO_2	$+PM_{10}$	0.87 (0.71, 1.06)	0.176
O_3	$+PM_{10}$	0.92 (0.82, 1.03)	0.117
SO_2	$+PM_{10}$	1.04 (0.99, 1.10)	0.130

OR (95% CI) was calculated for every 10 μg/m^3 increase in ambient pollutants during each specific time window.
*$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$.
[a] Two-pollutant models: adjustments were made for sex, breastfeeding, dampness, heating method, ETS, outdoor temperature and other pollutants during each specific time window.

boys and girls overall, but girls showed a higher susceptibility to pneumonia associated with PM, particularly PM_{10} and $PM_{2.5}$.

Risk associations with PM_{10}, $PM_{2.5}$, and PM_1 were consistent across different age groups, showing no substantial differences. In the sex groups, the ratio of boys to girls is approximately 1:1. In terms of age groups, the proportions of younger children (aged 3–4 years) and older children (aged 5–6 years) are also nearly balanced at a ratio of 1:1. Regarding SO_2 concentration stratification, the proportion of high-concentration samples accounts for 66.6%, which is nearly double that of low-concentration samples. In the ETS during pregnancy group, 24.9% of individuals reported being exposed to ETS within their households. Children exposed to high concentrations of SO_2 (> 70.98 μg/m^3) had a higher pneumonia risk caused by particle matter (PM_{10}, $PM_{2.5}$ and PM_1) during pregnancy than those exposed to low concentrations of SO_2 (≤ 70.98 μg/m^3). Our study revealed that children without exposure to ETS faced an increased risk of childhood pneumonia when exposed to PM_1 and $PM_{2.5}$ during pregnancy as compared to those exposed to ETS. In contrast, the risk of PM_{10} exposure during pregnancy was higher for those exposed to indoor ETS (OR = 1.09, 95% CI: 1.01 to 1.18) (Table S1).

4. Discussion

In our study on childhood pneumonia data from Taiyuan, China (ages 3–6, N = 4814), we utilized GLMM to compare the impacts of different types of air pollution. Our findings revealed that PM had a notably more significant impact during pregnancy than in infancy. Significant associations were observed between childhood pneumonia and affected by outdoor pollutants (PM_{10}, $PM_{2.5}$, PM_1, and SO_2) during gestation. Additionally, we assessed the effects of particulate matter exposure across different pregnancy trimesters, noting the strongest PM associations during the second trimester. Subgroup-specific analyses by sex, age, SO_2, and ETS during pregnancy confirmed the impacts of PM_{10}, $PM_{2.5}$, and PM_1 in various subgroups, but the effects of PM_1 were stronger in some subgroups. Our results help enhance our comprehension of the influence of PM exposure on children's health.

The study findings are consistent with previous research, but there are differences in the prevalence rates. For instance, a study in Changsha, China, reported an increased prevalence of pneumonia in children at 38.6%, contrasting with our study's 24.3% (Jiang et al., 2018). This variance may be attributed to our greater sample size (N = 4814 vs. N = 2598). In the context of the high incidence of pneumonia, the significance of preventive interventions, such as vaccination, has become increasingly prominent. Recent studies indicate that pneumococcal conjugate vaccines (PCV), particularly PCV13, demonstrate substantial potential in preventing pneumococcal infections among children (Xiaofei et al., 2024). However, in China, the vaccination rate is only 22% (Liu et al., 2023), and widespread adoption faces significant cost challenges, with each dose costing approximately $100. Each year, close to 294000 deaths occur among children under 5 globally due to pneumococcal infections, with China ranking second globally in this age group, representing nearly 12% of global cases (Wahl et al., 2018; WHO Publication, 2012).

$PM_{2.5}$ levels in urban, suburban, and rural settings across numerous countries in Africa, the Eastern Mediterranean, and Southeast Asia surpassed the interim target of 35 μg/m^3 set by World Health Organization (WHO) (World Health Statistics 2023, 2023). In Taiyuan, daily average $PM_{2.5}$ and PM_{10} concentrations are below the interim target of 75 and 150 μg/m^3 set by the WHO, respectively, suggesting control over particulate matter pollution. NO_2 levels are below the interim target of 129 μg/m^3 set by WHO. Moreover, SO_2 and O_3 levels are below the interim target of 125 and 160 μg/m^3 set by WHO, respectively. Overall, Taiyuan's pollution levels generally meet WHO targets.

Our study revealed a notable positive correlation between PM_{10} exposure during gestation and childhood pneumonia, particularly in the second trimester. Furthermore, this effect was more pronounced in girls

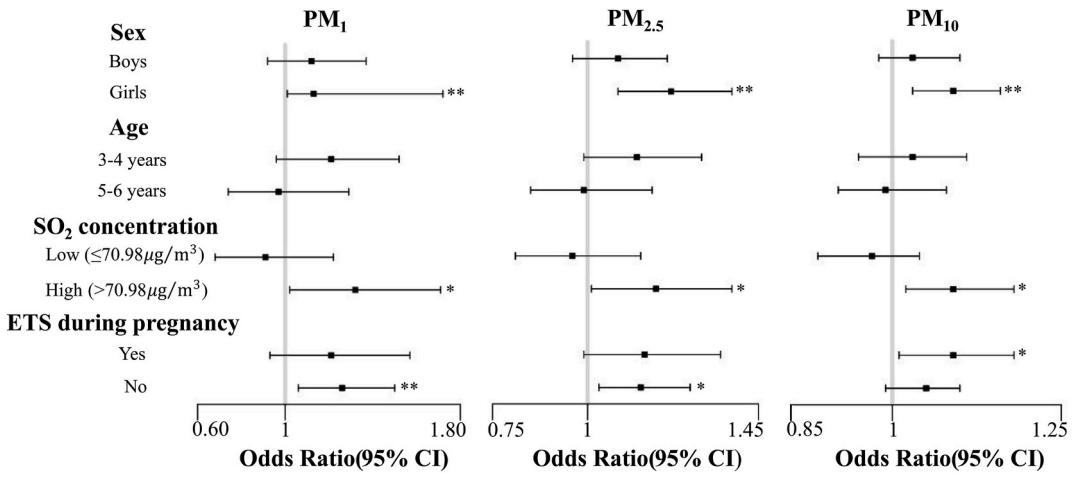

Fig. 1. Odds ratios (95% CI) for pneumonia in children for each 10 μg/m^3 rise in PM_1, $PM_{2.5}$, and PM_{10} levels during pregnancy, categorized by sex, age, SO_2 and ETS.

than in boys. One study showed a stronger negative correlation between air pollutants and lung function in girls, particularly reductions in FVC (forced lung capacity) and FEV_1 (forced expiratory volume in 1 s) (Clougherty, 2010). Girls' lung development was also different from boys' lung. Girls' airway diameters were smaller and their amounts per unit area were higher for the same pollutant concentration (Gauderman et al., 2015). Most studies have also shown that outdoor air pollution has a greater impact on pneumonia in boys than in girls (Chang et al., 2018; Huang et al., 2022). This gender difference may be related to lung function development and activity patterns, and will provide important references for formulating air pollution prevention and control strategies. Therefore, further research is needed. Our results agree with one previous study associating pregnancy PM_{10} exposure with childhood pneumonia. Significant associations between pneumonia in children and industrial PM_{10} exposure during pregnancy were identified in a study conducted in China (Yang et al., 2023). Two previous studies in China indicated that while early postnatal exposure to PM_{10} was significantly associated with the risk of pneumonia in children, especially in the first year of life, no significant association was observed between prenatal exposure and childhood pneumonia (Jiang et al., 2018; Liu et al., 2022). This is a different result from our study. The variations may stem from distinctions in research locales. The investigations above were conducted in urban areas in the southern region of China, with one in Changsha and the other in Shenzhen. The variation in pollutant types and particulate matter composition between different study regions in China may contribute to differences in research outcomes.

Our findings regarding $PM_{2.5}$ mirrored those of PM_{10}, albeit with pregnancy $PM_{2.5}$ showing a notably stronger impact on childhood pneumonia than PM_{10}. A study in Poland revealed a noteworthy connection between prenatal $PM_{2.5}$ exposure and the likelihood of repeated bronchopulmonary infections, including occurrences of pneumonia, among children aged between 1 and 7 years (Jedrychowski et al., 2013). A recent investigation conducted in Changsha, China, identified the infancy and second and third trimesters as critical periods for the influence of $PM_{2.5}$ exposure on pneumonia in children (Lu et al., 2023). In the two-pollutant model, we found that both NO_2 and $PM_{2.5}$ exposure during pregnancy and $PM_{2.5}$ and O_3 demonstrated a significant positive relationship with childhood pneumonia, similar to the association observed with $PM_{2.5}$ exposure alone. However, no associations between pregnancy $PM_{2.5}$ and pneumonia were observed with dual exposure to SO_2 and $PM_{2.5}$.

In contrast to other studies, we are among the few investigating the associations between PM_1 and childhood pneumonia. In our study, it is noteworthy that prenatal exposure to PM_1 showed the highest risk estimate among PM_{10}, $PM_{2.5}$ and PM_1. Additionally, our findings revealed that the risk estimates for PM_1 were higher in girls, similar to PM_{10} and $PM_{2.5}$. Due to the scarcity of studies conducted under similar research conditions, it is not easy to directly compare with previous research. However, a recent study has indicated that PM_1 had a more pronounced short-term effect on hospitalizations for childhood pneumonia in comparison to $PM_{2.5}$ and PM_{10} (Wang et al., 2021). Two studies indicate that PM_1 exerts a more immediate impact on pneumonia and respiratory mortality/incidence rates than larger particulate matter (Yang et al., 2018; Zhang et al., 2020). Our study found a significant positive link between pediatric pneumonia and concurrent exposure to PM_1 and NO_2 or PM_1 and O_3 throughout pregnancy. These findings are consistent with risk estimates observed in models examining individual exposures. As an ultra-fine particulate matter, PM_1 has the strongest toxic effect, but the current air quality standards have not included it in the monitoring scope. Our study provides a certain basis for the government to formulate the concentration limit standard of PM_1.

Our findings indicated an association between pneumonia in children and prenatal SO_2 exposure. However, in a study conducted in Changsha, China, between 2011 and 2012, involving 2598 children aged between 3 and 6 years, the analysis revealed a notable association between pneumonia in children and antenatal NO_2 exposure, with no significant links identified for SO_2 or PM_{10} (Jiang et al., 2018). We think that this is because Taiyuan mainly uses coal as an energy source, and the use of coal will release sulfur compounds. However, a subsequent study demonstrated a significant association between short-term exposure to SO_2 and diminished lung function and heightened airway inflammation in children (Barbone et al., 2019). Other recent studies have suggested that the concentrations of atmospheric pollutants, like SO_2, directly impact the distribution and makeup of microorganisms in the environment, indirectly influencing the risk of respiratory illnesses (Fu et al., 2021; Sun et al., 2022). Derivatives of SO_2 have been shown to mediate the induction of endoplasmic reticulum stress and inflammation alongside alterations in Histone-3 modification. Consequently, exposure to SO_2 has adverse effects on placental trophoblast cells, and prolonged exposure during pregnancy may result in detrimental outcomes (Singh et al., 2025). Furthermore, both SO_2 and NO_2 were positively associated with neurodevelopmental delays, with ORs (95% CI) of 1.15 (1.08, 1.22) and 1.05 (1.03, 1.07), respectively. This association appeared to be more pronounced during exposure in the first and second trimesters of pregnancy (Fu et al., 2024). Our study revealed that prenatal exposure to SO_2 significantly increased the risk of pneumonia in children, while no significant correlation was observed during infancy. This discrepancy may arise because postnatal infants are primarily exposed to SO_2 via direct inhalation, whereas fetal exposure involves long-term effects of maternal metabolic and oxidative stress responses, which can be more

harmful to developing organs. Nevertheless, a national-level study indicated that pre-birth exposure to SO_2 had a relatively weaker impact on respiratory diseases in children, whereas post-birth exposure exhibited a stronger association with respiratory conditions (Lu et al., 2022), which contradicts our findings. These inconsistencies might stem from variations in physiological mechanisms, developmental stages, and exposure patterns. Future research should aim to elucidate the specific mechanisms underlying SO_2 exposure during different time windows in early life and its implications for childhood pneumonia.

The inclusion of SO_2 in the two-pollutant model attenuated PM effect estimates, likely attributable to the high correlation between PM and SO_2 during pregnancy ($r = 0.696$) and their interactive effects, complicating the statistical disentanglement of their independent contributions. This aligns with findings from Zhengzhou, where SO_2 effects became nonsignificant in models co-adjusted for NO_2 or PM_{10} (Wang et al., 2023). While our results underscore the utility of multipollutant models for evaluating combined exposures, they simultaneously highlight the need for mechanistic investigations into pollutant correlations and interference. Stratified analyses revealed stronger PM_1-pneumonia associations at elevated SO_2 levels ($> 70.98\ \mu g/m^3$), suggesting SO_2 may potentiate PM toxicity via oxidative or inflammatory pathways. Supporting this, recent evidence demonstrates that SO_2 exacerbates Th1/Th2/Th17-mediated inflammation in allergic rhinitis patients and induces Th1/Th17 responses in healthy individuals (Ye et al., 2024). Furthermore, sulfate derivatives from SO_2 constitute 20–30% of PM_1 mass, potentially amplifying health risks through enhanced hygroscopicity and pulmonary deposition efficiency. Chemical speciation analyses of PM_1 corroborate this mechanism, indicating that sulfate-metal complexes (e.g., Fe, Zn) may synergistically exacerbate oxidative stress (Zhang et al., 2018). In summary, while multipollutant modeling diminishes the statistical independence of SO_2 effects, its biological role as an effect modifier, rather than a standalone risk factor, emerges as mechanistically plausible. Future studies should integrate PM component-resolved analyses (sulfate, organic carbon, metals) with in vitro toxicological assays to elucidate SO_2-PM interaction dynamics at molecular and cellular levels.

We found that prenatal PM exposure affects preschoolers' pneumonia risk more than infancy exposure. This could be because the pregnancy period is a sensitive time window in early life, and the second trimester is a sensitive window of concern among the various stages of pregnancy. Other studies have linked pregnancy PM exposure to respiratory illnesses. For instance, research conducted in the United States demonstrated a relationship between increased $PM_{2.5}$ exposure throughout the mid-pregnancy phase and the onset of asthma by the time boys reached 6 years old (Leon Hsu et al., 2015), while an INMA cohort study suggested mid-pregnancy exposures may impact lung function development (MacIntyre et al., 2014). PM contains a variety of heavy metals, which will also have a certain impact on children. A recent animal study showed significant long-term effects of prenatal arsenic exposure on lung and immune cell function (Rychlik et al., 2023). Wnt/β-catenin signaling is essential for normal lung development, and abnormal Wnt signaling contributes to the pathogenesis of bronchopulmonary dysplasia (BPD) and idiopathic pulmonary fibrosis (IPF). An animal study showed that post-translational modifications of β-catenin at tyrosine 489 and 654 are part of normal lung development, with peak signaling during the second trimester (Sucre et al., 2018). It can be seen that the second trimester is the critical period of lung development in children. The strong association between exposure to pollutants in the second trimester and childhood pneumonia reflects the high sensitivity of fetal lung development and placental function at this stage. In the future, multi-omics techniques and longitudinal studies should be combined further to analyze the molecular mechanism of this window period.

According to our study, PM_1 demonstrated the most significant impact on childhood pneumonia. PM_1 could consist of chloride, nitrate, ammonium, sulfate and primary organic aerosols (Niu et al., 2021). These constituents of PM_1 can come from cooking activities, traffic emissions, and others (Zhang et al., 2018). These ingredients can affect children's respiratory health. A large population study confirmed that PM_1 has a significantly greater negative impact on lung function in children than $PM_{2.5}$, especially in the FEV_1 and FVC indicators (Yang et al., 2020). A Korean study showed that in the lung region, PM_1 is the only particulate matter that significantly affects airway inflammation and that this effect has a cumulative effect, with an increase in PM_1 associated with a significant rise in FeNO levels by 15.9% (95% CI: 2.7–30.9) (Jeong et al., 2024). In addition, a toxicological study showed that PM_1 exposure had a significant dose-dependent toxic effect on bronchial cells and, at the same time, caused significant changes in the expression of specific genes. Some expression levels increased by more than two times (fold increase > 2), and some expression levels decreased by 50% (fold reduce > 0.5). These results indicated that PM_1 had a significant effect on the molecular mechanism of bronchial cells (Gualtieri et al., 2024). Future research into the effects of PM_1 on respiratory health could further validate and extend these findings with more sophisticated models and diverse exposure conditions.

According to our study, in stratification of ETS exposure during pregnancy, we found that ETS exposure showed a significant effect on childhood pneumonia only in PM_{10} exposure. Because there are few similar studies, it is not easy to find the same studies to compare, but some similar studies support our findings. A study from the Nutrition in Early Childhood Asthma (NELA) birth cohort found that environmental exposures, such as air pollution and ETS, can have a significant impact on lung function development in children, especially when benzene series are detected in high concentrations in exhaled gas (Sola-Martínez et al., 2025). A study in Argentina noted that tobacco burning significantly increased indoor concentrations of $PM_{2.5}$, a reliable marker of tobacco smoke, and that dynamic changes in the concentration of $PM_{2.5}$ are closely related to smoking behavior (Chaparro et al., 2024). This supports our findings that outdoor $PM_{2.5}$ exposure combined with ETS exposure has a weaker effect on children's lung health than indoor ETS exposure alone. While a study from Mexico showed that increased $PM_{2.5}$ was associated with decreased mtDNA content in models that included prenatal ETS exposure, suggesting increased sensitivity to PM-induced oxidative stress (OS) during this life stage (Rosa et al., 2017). These findings provide a basis for formulating targeted public health strategies, and future studies need to combine a multidisciplinary approach to promote the transformation from mechanism exploration to prevention policies.

This study offers a scientific foundation for the "Healthy China 2030" initiative and provides theoretical support for integrating the objective of children's respiratory health into the national environmental and health strategy. It also highlights the importance of achieving synergy between pollution control and health promotion through inter-sectoral collaboration (environmental protection, public health, and education). Future research should include cost-benefit analyses to evaluate the feasibility of these policies and establish long-term monitoring mechanisms to validate the effectiveness of interventions.

Indeed, our study has limitations. Firstly, the data on childhood pneumonia outcomes and demographic characteristics were gathered through questionnaire data, which may be affected by recall bias. Secondly, the data of NO_2, SO_2 and O_3 in our study were mainly obtained from monitoring sites, which were scattered and may be different from the actual exposure concentration of children. Again, we did not have the opportunity to investigate the effects of PM's chemical composition in our study. Finally, it is crucial to mention that our study was performed within a lone city, and therefore, the results may not be applied to other cities.

Our study also has some significant strengths. We used a mature machine learning-based method to estimate participants' exposure to PM, making the findings more precise. In addition, our study is among a limited number of investigations that have examined the connection between PM_1 exposure and pneumonia in children. Moreover, we

analyzed associations for different parts of pregnancy and postnatal exposure. Finally, our study analyzed the modifying effect of SO_2 on the relationships between PM and pneumonia in children, considering coal as the primary energy source in Taiyuan City.

5. Conclusions

Overall, prenatal PM_{10}, $PM_{2.5}$, PM_1, and SO_2 exposure can increase the hazard of pneumonia in pre-schoolers, with fine particulate matter, particularly PM_1, exhibiting a more pronounced effect. The second gestation period can be critical for particulate matter to influence the hazard of pneumonia in pre-schoolers. Additionally, SO_2 can further exacerbate the impact of PM on childhood pneumonia. Gender-specific susceptibility variations may exist in relation to the effects of particulate matter on pneumonia, possibly rendering girls more vulnerable. Moreover, household exposure to ETS might alter the connection between particulate matter and childhood pneumonia.

CRediT authorship contribution statement

Ying Wang: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Liu Yang:** Investigation, Data curation. **Jiyuan Shao:** Investigation, Data curation. **Huiyu Gao:** Investigation, Data curation. **Dan Norbäck:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Yunquan Zhang:** Project administration, Investigation. **Jing Wei:** Methodology. **Ling Zhang:** Project administration, Investigation. **Xin Zhang:** Writing – review & editing, Validation, Supervision, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envpol.2025.126303.

Data availability

Data will be made available on request.

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