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Exposures to particulate matters and childhood sleep disorders—A large study in three provinces in China

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ARTICLEINFO	A B S T R A C T
Keywords: Particulate matter Sleep disorders Children Cross-sectional study Epidemiology	<i>Objectives</i> : Evidence on the link between long-term ambient particulate matter (PM) exposures and childhood sleep disorders were scarce. We examined the associations between long-term exposures to $PM_{2.5}$ and PM_1 (PM with an aerodynamic equivalent diameter <2.5 µm and <1 µm, respectively) with sleep disorders in children. <i>Methods</i> : We performed a population-based cross-sectional survey in 177,263 children aged 6 to 18 years in 14 Chinese cities during 2012–2018. A satellite-based spatiotemporal model was employed to estimate four-year annual average $PM_{2.5}$ and PM_1 exposures at residential and school addresses. Parents or guardians completed a checklist using the Sleep Disturbance Scale for Children. We estimated the associations using generalized linear mixed models with adjustment for characteristics of children, parents, and indoor environments. <i>Results</i> : Long-term $PM_{2.5}$ and PM_1 exposures were positively associated with odds of sleep disorders for almost all domains. For example, increments in $PM_{2.5}$ and PM_1 per 10 µg/m ³ were associated with odds ratios of global sleep disorder of 1.24 (95 % confidence interval [CI]: 1.14, 1.35) and 1.31 (95 %CI: 1.18, 1.46), respectively. Similar results were observed for subtypes of sleep disorder. These associations were heterogeneous regionally, with stronger associations among children residing in southeast region than in northeast and northwest regions. <i>Conclusion:</i> Long-term $PM_{2.5}$ and PM_1 exposures are independently associated with higher risks of childhood sleep disorders, and these associations vary by geographical region.

1. Introduction

Sleep disorders such as insufficient sleep, disturbed sleep, and

hypersomnia are common in pediatric populations and can occur alone or coincide with multiple neurological diseases (Maski and Owens, 2016). Globally, 25 % of children have had at least one sleep disorder

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(Maski and Owens, 2016). This prevalence in China has reached 37.6 % over the past two decades and continues to rise (Chen et al., 2021). While the cause of this trend remains unclear, environmental factors have gained growing attention in attempts to understand the determinants of the epidemic of childhood sleep disorders. Ambient particulate matter (PM) pollution is an environmental challenge that affects millions of people (GBD, 2017 Risk Factor Collaborators, 2018). Although China's air quality has improved in recent years, PM_{2.5} (PM with an aerodynamic equivalent diameter <2.5 µm) remains one of the most prevalent air pollutants (GBD, 2019 Diseases and Injuries Collaborators, 2020). Between 2016 and 2019, the annual level of PM_{2.5} in China was 6–9 times higher than the suggested limit (5 µg/m³) of the World Health Organization (Shi et al., 2021). Children are deemed one of the most sensitive populations to air pollution exposure (Heinrich and Slama, 2007).

Experimental studies suggest that inhaled PM affects sleep through oxidative stress, inflammatory response and neurochemicals imbalance (Liu et al., 2020, 2021). However, the epidemiological evidence of the associations between PM2.5 exposure and childhood sleep disorder are mixed. Childhood PM_{2.5} exposure has been linked to poorer sleep health in four previous studies (Bose et al., 2019; Cai et al., 2023; He et al., 2023; Lawrence et al., 2018), while another study did not find such an association (Sanchez et al., 2019). Variations in study results may stem from disparities in factors such as sample size, setting, exposure and outcome assessments (Table S1). While two large studies have been conducted in China (Cai et al., 2023; Lawrence et al., 2018), one used data on PM exposures at school addresses and neither of them investigated the associations in different regions. Because PM1 (particles with airborne diameters $<1 \mu m$) has stronger ability to penetrate the respiratory tract and a high amount of absorbed toxins (Meng et al., 2013), PM1 may have more severe effects in comparison with PM2.5. Nevertheless, the evidence for PM1 exposure and childhood sleep disorders was limited to one previous study (Lawrence et al., 2018). Identification of adverse effects of PM is crucial for developing effective intervention strategies to safeguard children's health. Furthermore, comparing the effects of PM1 and PM2.5 could offer valuable insights into potential public health benefits achieved by taking targeted clean air policies. Therefore, we estimated time-weighted average long-term PM2.5 and PM₁ exposures at both home and school locations by using a satellitebased spatiotemporal model. Our investigations focus on the associations with multiple dimensions of childhood sleep disorders across 14 cities from three provinces that are located in northeast, northwest and southeast region of China, respectively. We hypothesized that (1) associations between long-term exposures to PM2.5 and PM1 with elevated prevalence of sleep disorders were independent of regions; (2) associations between PM1 and sleep disorders were larger than estimates of PM_{2.5}.

2. Materials and methods

2.1. Study population and study design

This study was nested within the National Chinese Children Health Study (NCCHS), a large population-based cross-sectional survey of children, conducted from 2012–2018. The aim of this survey is to explore the associations between environmental factors and children's physical and neurodevelopmental health. Detailed descriptions of study design and population has been published elsewhere (Wang et al., 2022). Briefly, we selected 14 cities in Liaoning province (northeast China), Guangdong province (southeast China) and Xinjiang Uygur Autonomous Region (referred to as "Xinjiang"; northwest China). Within each city, 12–13 schools were randomly selected based on geographic district, school grade (elementary, junior high school and high school), and school size, yielding a total of 173 schools. The flowchart of study participants recruitment is displayed in Fig. S1. Children living in their current residential addresses for at least four years were eligible to participate in this study. We identified 190,771 children aged 6–18 years from 173 schools. We further excluded 13,508 children whose parents/guardians failed to complete the questionnaires or who were living at their current address less than four years, leaving a final sample size of 177,263 (response rate 92.9 %). This study was approved by the Ethical Review Committee for Biomedical Research, Sun Yat-sen University. We received written informed consent from parents/guardians of all children preceding data collection.

2.2. Sleep disorder measurement

We used the Sleep Disturbance Scale for Children (SDSC) to assess children's sleep disorders over the past six months (Huang et al., 2014). It is suitable for children aged 3–18 years (Bruni et al., 1996), which was completed by their parents or guardians. The SDSC has been applied extensively in both clinical and epidemiological settings as a screening tool for sleep disorders (Bruni et al., 1996). We used the Chinese version of SDSC that has been adapted to the cultural and socioeconomic conditions in China (Huang et al., 2014). For the sample in the NCCHS, the internal consistency of the SDSC scale is acceptable (Cronbach $\alpha = 0.81$; Huang et al., 2014). The SDSC consists of 24 items with a 5-point scale (from 1 = never to 5 = always) to assess six domains of sleep disorders: disorders of initiating and maintaining sleep (DIMS), sleep-wake transition disorders (SWTD), sleep hyperhidrosis (SHY), sleep breathing disorders (SBD), disorders of arousal (DA), and disorders of excessive somnolence (DOES). Each domain score is calculated by summing across items within that domain. A total score, representing the global sleep quality, is calculated by summing the six domains. All scores were converted into *t*-scores (see the formula below). Higher *t*-scores reflect more sleep problems. A *t*-score > 70 was classified as having a symptom of a sleep disorder.

T-score = 50 + (value - mean)/standard deviation \times 10

2.3. Air pollutants exposure assessment

We predicted four-year average ambient PM2.5 and PM1 concentrations on a daily basis at residential and school addresses using a satellitebased random forest model at a high resolution of $1 \text{ km} \times 1 \text{ km}$ (Chen et al., 2018a, 2018b). Briefly, this model was developed through combining ground-based observations of daily PM2.5 and PM1 concentrations as the dependent variable with the aerosol optical depth data, as well as other spatiotemporal predictors including meteorology, greenness, fractional urban cover, and elevation as the independent variables. The cross-validated R² (root mean squared error, RMSE) at annual level were 86 % (6.9 μ g/m³) for PM_{2.5} and 75% (8.8 μ g/m³) for PM₁ (Chen et al., 2018a, 2018b). We estimated residential and school PM exposures by assigning the predicted daily PM concentration to each child's residential and school addresses, respectively. We also derived combined PM exposures (home-school PM) by averaging residential and school PM concentrations given the assumption that children spend 2/3 of their time (16 h) at home and 1/3 (8 h) at school. We calculated four-year average school-home, school, and home PM levels as indicators of long-term exposures, encompassing the year of sleep assessment and three preceding years.

2.4. Covariates

Based on the previous literature (Cai et al., 2023; Chen et al., 2021) and the articles previously published by the NCCHS (Lawrence et al., 2018; Liu et al., 2023), we identified the following three groups of covariates: (1) child's factors, including age (years), sex (boys or girls), preterm (yes or no), caesarean (yes or no), breastfeeding \geq 4 months (yes or no), low birth weight (yes or no, defined as <2500 g), asthma (yes or no), eczema (yes or no), relatively weight (underweight, normal weight and overweight/obesity; de Onis et al., 2007), physical activity (\leq 1 or

>1 h/day), school assignment (light, moderate, or high) and season of assessment (warm or cold); (2) parents' factors, including parental education attainment (highest parental education: below senior high school, senior high school, college or above) and annual household income (\leq 10,000 or 10,001–30,000 or 30,001–100,000 or >100,000 RMB); (3) indoor environments, including current second-hand smoking exposure (yes or no), house renovation (yes or no), visible mold or dampness (yes or no), and per capita living area of the family (<20 m², 20–30 m²).

2.5. Statistical analyses

Continuous variables were presented as mean \pm standard deviation (SD) or median (interquartile range, IQR), and categorized variables were presented as number (percentage). Associations between homeschool, home, and school PM exposures with sleep disorders were tested using generalized linear mixed models. We included one PM in a model at a time because of strong collinearity (r = 0.99) between PM_{2.5} and PM₁. We started with the crude model (model 1) and sequentially added the covariates of children (model 2), parents (model 3), and indoor environments (model 4). City was treated as random intercept in all the models. Results were expressed as odds ratios (ORs) with their 95 % confidence intervals (CIs) for $10 \,\mu\text{g/m}^3$ increment for PM. We extended other analyses based on model 4 and used the single indicator of homeschool PM exposures. We further conducted stratified regression models, yielding province-specific estimates, to investigate regional variations in the associations. We did a set of stratified analyses by stratifying children based on factors such as preterm birth (Visser et al., 2021), cesarean section (Zhang et al., 2019), low birth weight (Young et al., 2019) and asthma (Ramirez et al., 2019), which might be likely to increase the vulnerability to environmental hazards (Gee and Payne-Sturges, 2004) and sleep disorders. Sensitivity analyses were conducted by excluding children with preterm birth, cesarean section, low birth weight and asthma. All analyses were performed with R software (version 4.3.2 with main packages *lmerTest*). Significance was set at a two-tailed P < 0.05.

3. Results

3.1. Participants characteristics

Characteristics of all participants and stratified by region are presented in Table 1. The mean age (SD) of participants was 11.7 (2.9) years, and half of children (52.5 %) were boys. Overall, 19.4 % children had at least one sleep disorder, including 5.5 % for DIMS, 4.6 % for SBD, 3.5 % for DA, 4.1 % for SWTD, 5.1 % for DOES, and 6.1 % for SHY.

3.2. Concentrations of ambient PM

As shown in Table 1, the mean (SD) home-school concentrations of $PM_{2.5}$ and PM_1 were 42.6 (20.0 µg/m³) and 33.4 (10.7 µg/m³), respectively. The ratio of $PM_{1/}PM_{2.5}$ was 0.81, suggesting that 81 % of $PM_{2.5}$ comprised PM_1 . PM concentrations showed significant spatial variation across regions. Xinjiang exhibited the highest average $PM_{2.5}$ concentrations (118.7 µg/m³) and PM_1 (55.0 µg/m³) with 0.46 for $PM_{1/}$ PM_{2.5} ratio. Guangdong had lowest $PM_{2.5}$ (32.8 µg/m³) and PM_1 (26.7 µg/m³) concentrations with 0.81 for $PM_{1/}PM_{2.5}$ ratio.

Fig. 1 displays the geographic locations of the study cities and distribution of $PM_{2.5}$ and PM_1 levels in 14 cities. Kashgar, which is close to one of the largest sand-shifting deserts (Taklimakan desert) in the world, exhibited the highest median $PM_{2.5}$ (121.0 µg/m³) and PM_1 (55.0 µg/m³) concentrations, followed by Dandong (63.3 µg/m³ for PM_{2.5}; 54.1 µg/m³ for PM₁) and Anshan (63.0 µg/m³ for PM_{2.5}; 53.9 µg/m³ for PM₁), two heavy industrial cities (Table S2). Shenzhen and Zhuhai, two coastal cities, had the lowest median PM_{2.5} (28.4 and 29.1 µg/m³, respectively) and PM₁ (24.1 and 24.4 µg/m³, respectively) concentrations.

3.3. Associations between exposures to $PM_{2.5}$ and PM_1 with sleep disorders

Table 2 shows the ORs and 95 %CIs for the associations between home-school, home and school PM2.5 and PM1 exposures with sleep disorders. The estimates from model 1 to model 4 (adjusted for characteristics of children, parents, and indoor environments) were stable, indicating that the results were robust. Each 10 μ g/m³ increment in PM_{2.5} concentration was positively associated with higher odds of global sleep disorder, DIMS, SBD, DA, SWTD, DOES, and SHY, with estimates ranging from 1.10 (95 %CI: 1.01, 1.19) to 1.29 (95 %CI: 1.20, 1.38). Likewise, each $10 \,\mu\text{g/m}^3$ increment in PM₁ concentration was positively associated with higher odds of global sleep disorder, DIMS, SBD, DA, SWTD, DOES, and SHY, with estimates ranging from 1.14 (95 %CI: 1.03, 1.26) to 1.37 (95 %CI: 1.26, 1.49). The estimates were analogous when using home exposures. The associations were diminished when using school exposures, which may be due to the nondifferential exposure misclassification based on school exposures. Several sensitivity analyses were performed to validate the robustness of the results. The effect estimates remained unchanged after excluding children with preterm birth, cesarean section, low birth weight, or asthma (Table S4 and Table S5). Additionally, the results did not differ greatly after excluding children with high school assignment (data not shown).

Fig. 2 and Table S3 show the associations between PM exposures in quartiles and sleep disorders. When $PM_{2.5}$ and PM_1 exposures were fitted as a categorized variable of quartiles, participants at higher quartiles (2nd, 3rd and 4th) showed higher odds of sleep disorders across all dimensions than those at the lowest quartile levels (all *P* for trend < 0.01).

Stratified analyses of the associations between PM exposures and sleep disorders by province are shown in Table 3. The associations varied considerably among the three provinces. We only detected positive associations between $PM_{2.5}$ and PM_1 exposures with DA and SWTD in Xinjiang, and with global sleep disorder, DIMS, SWTD, and DOES in Liaoning. In Guangdong, $PM_{2.5}$ and PM_1 exposures were associated with increased odds of sleep disorders for all dimensions. The associations of PM exposures in Guangdong were larger than in Liaoning. Moreover, larger estimates of PM_1 were found than that of $PM_{2.5}$ in Guangdong.

We also performed other stratified analyses. The magnitudes in associations between children with and without preterm birth, cesarean section, low birth weight or asthma were not consistent across multiple domains of sleep disorders (Table S4 and Table S5).

4. Discussion

Our large multicity study showed 10 %–29 % higher odds of sleep disorders across various domains in relation to long-term exposure to $PM_{2.5}$. We detected similar associations of PM_1 , with the effect estimates ranging from 1.14 to 1.37. These associations differed regionally. Children living in Guangdong were more likely to be sensitive than those living in Liaoning and Xinjiang. Of note, larger estimates of PM_1 exceeded that of $PM_{2.5}$ in Guangdong, whereas similar estimates between PM_1 and $PM_{2.5}$ were identified in Xinjiang and Liaoning. Stratified analyses did not show clear patterns of effects among children with and without preterm birth, cesarean section, low birth weight or asthma. To our knowledge, this is the largest epidemiologic study to date to examine the associations between individual-assessed exposures to two particle size classes of $PM_{2.5}$ and PM_1 in different regions of China and childhood sleep disorders.

Four out of five studies have found associations between higher $PM_{2.5}$ exposure and poorer sleep health (Bose et al., 2019; Cai et al., 2023; He et al., 2023; Lawrence et al., 2018), but one did not detect significant associations (Sanchez et al., 2019). A large cross-sectional study of 59,754 children aged 2–17 years in northeast China reported that higher satellite-based $PM_{2.5}$ exposure surrounding schools was linked with 26 %–50 % increased odds of global sleep disorder, DIMS, DA, SWTD, DOES and SHY screened by the SDSC (Lawrence et al.,

Table 1

Characteristics of children by region.

Variables ^a	Total population	Xinjiang	Liaoning	Guangdong	P-values
	(N = 177,263)	(N = 8,330)	(N = 46,365)	(N = 122,568)	
Children					
Age, mean (SD), years	11.68 (2.86)	11.70 (2.98)	11.61 (2.60)	11.71 (2.95)	< 0.001
Sex	92 995 (52 46)	4 225 (50 72)	23 262 (50 17)	65 508 (53 45)	< 0.001
Girls	84,268 (47.54)	4,105 (49.28)	23,103 (49.83)	57,060 (46.55)	
Preterm					
Yes	9,199 (5.19)	471 (5.65)	2,372 (5.12)	6,356 (5.19)	0.124
No	168,064 (94.81)	7,859 (94.35)	43,993 (94.88)	116,212 (94.81)	
Caesarean					
Yes	66,365 (37.44)	2,849 (34.20)	20,525 (44.27)	42,991 (35.08)	< 0.001
No	110,898 (62.56)	5,481 (65.80)	25,840 (55.73)	79,577 (64.92)	
× 11.1 11.					
Low birth weight Yes	9.079 (5.12)	991 (11.90)	1,778 (3.83)	6.310 (5.15)	< 0.001
No	168,184 (94.88)	7,339 (88.10)	44,587 (96.17)	116,258 (94.85)	
Breastfeeding within first 3 months	110110000		00 101 //0		
Yes	110,146 (62.14)	6,103 (73.27)	32,181 (69.41)	71,862 (58.63)	< 0.001
NO	07,117 (37.00)	2,227 (20.73)	14,104 (30.39)	30,700 (41.37)	
Asthma					
Yes	16,330 (9.21)	672 (8.07)	3,330 (7.18)	12,328 (10.06)	< 0.001
No	160,933 (90.79)	7,658 (91.93)	43,035 (92.82)	110,240 (89.94)	
Fczema					
Yes	20,297 (11.45)	537 (6.45)	6,123 (13.21)	13,637 (11.13)	< 0.001
No	156,966 (88.55)	7,793 (93.55)	40,242 (86.79)	108,931 (88.87)	
Relatively weight	00 100 (1(47)	1 001 (10 00)	0.000 (17.00)	10 700 (1(14)	< 0.001
Normal weight	29,190 (16.47) 125 101 (70 57)	1,081 (12.98)	8,326 (17.96) 32 705 (70 54)	19,783 (16.14) 85 570 (69 81)	
Overweight/obesity	22,972 (40.9)	423 (5.08)	5,334 (11.50)	17,215 (35.93)	
Physical activity ≥ 1 h per day					< 0.001
Yes	66,179 (37.33)	2,863 (34.37)	25,596 (55.21)	37,720 (30.77)	
School assignment	111,084 (02.07)	5,407 (05.03)	20,769 (44.79)	84,848 (09.23)	< 0.001
Light	20,884 (11.78)	846 (10.16)	7,110 (15.33)	12,928 (10.55)	
Moderate	140,393 (79.20)	6,746 (80.98)	35,411 (76.37)	98,236 (80.15)	
High	15,986 (9.02)	738 (8.86)	3,844 (8.29)	11,404 (9.30)	<0.001
Warm	151,581 (85.51)	8,314 (99.81)	40,873 (88.15)	102,394 (83.54)	<0.001
Cold	25,682 (14.49)	16 (0.19)	5,492 (11.85)	20,174 (16.46)	
Parents Parental education					<0.001
Below senior high school	58,193 (32.83)	3,043 (36.53)	13,919 (30.02)	41,231 (33.64)	<0.001
Senior high school	58,798 (33.17)	2,110 (25.33)	15,860 (34.21)	40,828 (33.31)	
College or above	60,272 (34.00)	3,177 (38.14)	16,586 (35.77)	40,509 (33.05)	
<pre>Yearly family income <10.000</pre>	38 296 (21 60)	2 044 (24 54)	10 401 (22 43)	25 851 (21 00)	< 0.001
10,001-30,000	41,035 (23.15)	1,925 (23.11)	18,186 (39.22)	20,924 (17.07)	
30,001–100,000	58,356 (32.92)	3,056 (36.69)	15,443 (33.31)	39,857 (32.52)	
>100,000	39,576 (22.33)	1,305 (15.67)	2,335 (5.04)	35,936 (29.32)	
Indoor anticonment					
Passive cigarette smoke exposure					< 0.001
Yes	67,344 (37.99)	2,929 (35.16)	22,167 (47.81)	42,248 (34.47)	
No	109,919 (62.01)	5,401 (64.84)	24,198 (52.19)	80,320 (65.53)	-0.001
Yes	85,640 (48.31)	4,131 (49,59)	17.068 (36 81)	64.441 (52.58)	< 0.001
No	91,623 (51.69)	4,199 (50.41)	29,297 (63.19)	58,127 (47.42)	
Visible mold or dampness					< 0.001
Yes	61,565 (34.73)	3,076 (36.93)	7,717 (16.64)	50,772 (41.42)	
Per capita living area of the family	113,098 (05.27)	5,254 (03.07)	əə,048 (83.3b)	/1,/90 (58.58)	<0.001
$\leq 20 \text{ m}^2$	69,064 (38.96)	2,191 (26.30)	21,207 (45.74)	45,666 (37.26)	
20–30 m ²	72,143 (40.70)	4,117 (49.42)	16,581 (35.76)	51,445 (41.97)	
				(continued	on next page)

Table 1 (continued)

Variables ^a	Total population	Xinjiang	Liaoning	Guangdong	P-values
	(N = 177,263)	(N = 8,330)	(N = 46,365)	(N = 122,568)	
>30 m ²	36,056 (20.34)	2,022 (24.27)	8,577 (18.50)	25,457 (20.77)	
Prevalence of sleep disorders					
Gross sleep disorder	7,584 (4.28)	279 (3.35)	1,638 (3.53)	5,667 (4.62)	< 0.001
DIMS	9,792 (5.52)	251 (3.01)	1,526 (3.29)	8,015 (6.54)	< 0.001
SBD	8,268 (4.66)	322 (3.87)	2,471 (5.33)	5,475 (4.47)	< 0.001
DA	6,248 (3.52)	634 (7.61)	1,527 (3.29)	4,087 (3.33)	< 0.001
SWTD	7,257 (4.09)	356 (4.27)	2,135 (4.60)	4,766 (3.89)	< 0.001
DOES	9,026 (5.09)	465 (5.58)	2,674 (5.77)	5,887 (4.80)	< 0.001
SHY	10,810 (6.10)	491 (5.89)	2,412 (5.20)	7,907 (6.45)	< 0.001
Particulate matters concentrations					
Home-school					< 0.001
PM ₁ , mean (SD), $\mu g/m^3$	33.36 (10.65)	54.97 (2.02)	47.01 (5.60)	26.73 (2.37)	
$PM_{2.5}$, mean (SD), $\mu g/m^3$	42.59 (19.99)	118.66 (6.03)	54.87 (6.16)	32.77 (3.75)	
PM ₁ :PM _{2.5}	0.81	0.46	0.86	0.81	
Home					< 0.001
PM ₁ , mean (SD), μg/m ³	33.15 (10.32)	51.08 (0.83)	47.01 (5.60)	26.69 (2.43)	
PM _{2.5} , mean (SD), μg/m ³	42.62 (20.19)	119.78 (4.41)	54.87 (6.16)	32.74 (3.87)	
PM ₁ :PM _{2.5}	0.81	0.42	0.86	0.81	
School					< 0.001
PM ₁ , mean (SD), μg/m ³	33.79 (11.51)	62.75 (5.85)	47.01 (5.60)	26.81 (2.39)	
$PM_{2.5}$, mean (SD), $\mu g/m^3$	42.52 (19.83)	116.42 (15.89)	54.87 (6.16)	32.82 (3.79)	
PM ₁ :PM _{2.5}	0.82	0.53	0.86	0.82	

Abbreviations: DA, Disorders of arousal, DIMS, Disorders of initiating and maintaining sleep; DOES, Disorders of excessive somnolence; PM_1 , particles with airborne diameters $<1 \mu$ m; $PM_{2.5}$, particles with airborne diameters $<2.5 \mu$ m; SBD, Sleep breathing disorders; SD, standard deviation; SHY, Sleep hyperhidrosis; SWTD, Sleep-wake transition disorders.

^bP-values were calculated from analysis of variance for continuous variables, and from chi-square test for categorized variables.

^a Data are presented as number (percentage) of children unless otherwise indicated.



Fig. 1. Location of study cities and home-school particulate matters exposures by city. A, Location of study cities and PM exposures by city. B, The horizontal line inside the boxes indicates the median PM concentrations, the lower and upper ends of the boxes the lower and upper quartiles of PM concentration, and the whiskers the minimum and maximum PM concentration. AS indicates Anshan; BX, Benxi; DD, Dandong; FoS, Foshan; FuS, Fushun; KS, Kashgar; LY, Liaoyang; MM, Maoming; SY, Shenyang; SZ, Shenzhen; ZH, Zhuhai; and ZS, Zhongshan.

2018). Similarly, a nationwide study in China of 115,023 preschool children aged 3–7 years found that prenatal and postnatal $PM_{2.5}$ exposures were associated with lower sleep quality and higher risks of SBD and daytime sleepiness identified by Children's Sleep Habits Questionnaire (Cai et al., 2023). Another two studies with small sample sizes adopted actigraphy to objectively measure sleep outcomes (Bose et al., 2019; He et al., 2023). Of them, a pregnancy cohort in Mexico City of 397 mother–child pairs reported that greater satellite-based $PM_{2.5}$ exposure during early and late gestation was associated with alteration in both sleep efficiency and sleep duration among preschoolers (Bose et al., 2019). A study of 246 adolescents in the US also found that short-term individual and intermediate-term residential $PM_{2.5}$ exposures were

adversely and synergistically associated with increased sleep variability (He et al., 2023). Aligning with the findings of four previous studies, we observed that greater ambient $PM_{2.5}$ exposure was associated with elevated odds of sleep disorders in 184,130 Chinese school-aged children.

Sanchez et al. conducted a cross-sectional study of 564 children aged 5–9 years in three cities in Chile (Sanchez et al., 2019). Using a questionnaire to assess wheezing and snoring and estimating school $PM_{2.5}$ concentrations from monitoring stations, the authors failed to find significant associations between them. The lack of observed association might be attributed to factors including the small sample size, the cross-sectional study design, the use of school address instead of residential

Table 2

Odds ratios of sleep disorders associated with exposures to ambient particulate matters concentrations $>10 \ \mu g/m^3$.

Odds ratios (95 % confidence intervals)	Home-school exposures		Home exposures		School exposures	
	PM_1	PM _{2.5}	PM ₁	PM _{2.5}	PM_1	PM _{2.5}
Global sleep disorder						
Model 1	1.46 (1.33, 1.60)*	1.37 (1.27, 1.47)*	1.50 (1.37, 1.64)*	1.39 (1.29, 1.50)*	1.28 (1.17, 1.39)*	1.14 (1.07, 1.22)*
Model 2	1.26 (1.14, 1.40)*	1.21 (1.11, 1.32)*	1.28 (1.16, 1.42)*	1.21 (1.11, 1.32)*	1.16 (1.05, 1.27)*	1.08 (1.01, 1.15)*
Model 3	1.27 (1.15, 1.41)*	1.31 (1.22, 1.42)*	1.30 (1.17, 1.43)*	1.23 (1.14, 1.34)*	1.16 (1.05, 1.27)*	1.07 (1.00, 1.15)*
Model 4	1.31 (1.18, 1.46)*	1.24 (1.14, 1.35)*	1.30 (1.17, 1.43)*	1.24 (1.14, 1.34)*	1.16 (1.05, 1.27)*	1.07 (1.00, 1.15)*
DIMS						
Model 1	1 24 (1 22 1 47)*	1 25 (1 17 1 24)*	1 27 (1 25 1 40)*	1 DE (1 17 1 24)*	1 01 (1 11 1 01)*	1 1 2 (1 06 1 10)*
Model 2	1.34(1.23, 1.47) 1.12(1.02, 1.24)	1.23 (1.17, 1.34)	1.37 (1.23, 1.49)	1.23(1.17, 1.34) 1.07(0.00, 1.16)	1.21(1.11, 1.31)	1.12(1.00, 1.19)
Model 2	1.13 (1.02, 1.24)"	1.10 (1.00, 1.07)*	1.15 (1.05, 1.25)"	1.07 (0.99, 1.10)	1.08 (0.98, 1.18)	1.06 (1.00, 1.13)*
Model 3	1.13 (1.03, 1.25)*	1.18 (1.09, 1.27)*	1.15 (1.04, 1.26)*	1.09(1.01, 1.18)*	1.07 (0.98, 1.18)	1.06 (1.00, 1.12)*
Model 4	1.14 (1.03, 1.26)*	1.10 (1.01, 1.19)*	1.14 (1.04, 1.26)*	1.09 (1.01, 1.18)*	1.07 (0.97, 1.17)	1.05 (0.99, 1.12)
SBD						
Model 1	1.13 (1.06, 1.20)*	1.10 (1.04, 1.16)*	1.15 (1.08, 1.22)*	1.10 (1.04, 1.17)*	1.08 (1.02, 1.15)*	1.05(1.01, 1.10)*
Model 2	1.14 (1.07, 1.21)*	1.09 (1.03, 1.15)*	1.15 (1.08, 1.23)*	1.09 (1.03, 1.15)*	1.10 (1.03, 1.16)*	1.05 (1.01, 1.10)*
Model 3	1.14 (1.07, 1.22)*	1.13 (1.06, 1.19)*	1.16 (1.09, 1.23)*	1.10 (1.04, 1.16)*	1.10 (1.04, 1.17)*	1.06 (1.02, 1.10)*
Model 4	1.16 (1.09, 1.24)*	1.11 (1.04, 1.17)*	1.16 (1.09, 1.23)*	1.10 (1.04, 1.16)*	1.11 (1.04, 1.18)*	1.06 (1.02, 1.11)*
DA						
	1 10 (1 10 1 0())		1 1 7 (1 00 1 05)		1 1 7 (1 10 1 0 ()	1 11 (1 05 1 1 0)
Model 1	1.18 (1.10, 1.26)*	1.12 (1.08, 1.16)	1.17 (1.09, 1.25)*	1.11 (1.07, 1.17)*	1.17 (1.10, 1.24)*	1.11 (1.07, 1.14)*
Model 2	1.15 (1.07, 1.23)*	1.11 (1.07, 1.15)*	1.13 (1.05, 1.21)*	1.09 (1.05, 1.14)*	1.14 (1.08, 1.22)*	1.10 (1.07, 1.14)*
Model 3	1.13 (1.05, 1.21)*	1.11 (1.07, 1.15)*	1.12 (1.04, 1.21)*	1.09 (1.05, 1.14)*	1.11 (1.04, 1.19)*	1.08 (1.05, 1.12)*
Model 4	1.14 (1.06, 1.23)*	1.11 (1.06, 1.15)*	1.13 (1.05, 1.22)*	1.10 (1.05, 1.14)*	1.12 (1.05, 1.20)*	1.08 (1.05, 1.13)*
SWTD						
Model 1	1.22 (1.14, 1.31)*	1.18 (1.10, 1.27)*	1.23 (1.15, 1.31)*	1.16 (1.08, 1.25)*	1.18 (1.11, 1.25)*	1.11 (1.05, 1.16)*
Model 2	1.15 (1.07, 1.23)*	1.13 (1.05, 1.22)*	1.20 (1.13, 1.28)*	1.11 (1.04, 1.19)*	1.17 (1.10, 1.25)*	1.10 (1.05, 1.15)*
Model 3	1.20 (1.12, 1.28)*	1.20 (1.11, 1.29)*	1.20 (1.13, 1.28)*	1.12 (1.04, 1.20)*	1.16 (1.09, 1.23)*	1.09 (1.04, 1.14)*
Model 4	1.21 (1.14, 1.30)*	1.14 (1.06, 1.22)*	1.21 (1.13, 1.29)*	1.13 (1.05, 1.21)*	1.17 (1.10, 1.24)*	1.09 (1.04, 1.14)*
DOEC						
Model 1	173 (161 197)*	1 52 (1 42 1 62)*	176(163 100)*	1 56 (1 46 1 65)*	1 54 (1 43 1 66)*	1 25 (1 18 1 22)*
Model 2	1.73(1.01, 1.07) 1.15(1.07, 1.22)	1.32(1.43, 1.02) 1.96(1.19, 1.25)*	1.70(1.03, 1.90) 1.22(1.02, 1.42)	1.30(1.40, 1.03) 1.25(1.17, 1.24)*	1.34(1.43, 1.00) 1.96(1.17, 1.25)	1.23(1.10, 1.32) 1.12(1.00, 1.10)
Model 2	1.13(1.07, 1.23) 1.22(1.22, 1.44)*	1.20(1.16, 1.33) 1.22(1.24, 1.41)*	1.32(1.22, 1.43) 1.22(1.22, 1.43)	1.23(1.17, 1.34) 1.27(1.10, 1.26)*	1.20(1.17, 1.33) 1.25(1.17, 1.24)	1.13(1.00, 1.10) 1.12(1.00, 1.10)
Model 4	1.33(1.22, 1.44) 1.27(1.26, 1.40)	1.32(1.24, 1.41)	1.33(1.23, 1.44) 1.24(1.24, 1.45)	1.27(1.10, 1.30)	1.23(1.17, 1.34) 1.26(1.10, 1.25)	1.13(1.00, 1.10)
Model 4	1.37 (1.26, 1.49)*	1.29 (1.20, 1.38)*	1.34 (1.24, 1.45)*	1.27 (1.19, 1.36)*	1.26 (1.18, 1.35)*	1.13 (1.08, 1.19)*
SHY						
Model 1	1.01 (0.95, 1.08)	1.04 (0.99, 1.09)	1.02 (0.96, 1.08)	1.03 (0.98, 1.08)	1.01 (0.95, 1.06)	1.03 (0.99, 1.07)
Model 2	1.17 (1.08, 1.26)*	1.14 (1.06, 1.22)*	1.18 (1.09, 1.28)*	1.15 (1.07, 1.23)*	1.11 (1.04, 1.20)*	1.07 (1.02, 1.12)*
Model 3	1.17 (1.08, 1.27)*	1.19 (1.11, 1.27)*	1.18 (1.10, 1.28)*	1.16 (1.08, 1.24)*	1.12 (1.04, 1.20)*	1.07 (1.02, 1.13)*
Model 4	1.18 (1.09, 1.28)*	1.15 (1.07, 1.23)*	1.19 (1.10, 1.28)*	1.16 (1.08, 1.24)*	1.12 (1.04, 1.20)*	1.07 (1.02, 1.13)*

Abbreviations: DA, Disorders of arousal, DIMS, Disorders of initiating and maintaining sleep; DOES, Disorders of excessive somnolence; PM_1 , particles with airborne diameters $< 1 \mu m$; $PM_{2.5}$, particles with airborne diameters $< 2.5 \mu m$; SBD, Sleep breathing disorders; SD, standard deviation; SHY, Sleep hyperhidrosis; SWTD, Sleepwake transition disorders.

Model 1: crude model; Model 2: adjusted for age, sex, preterm, caesarean, breastfeeding, asthma, eczema, relatively weight, physical activity, school assignment and season of assessment; Model 3: additionally adjusted for parental education attainment and annual household income; Model 4: additionally adjusted for current second-hand smoking exposure, house renovation, visible mold or dampness, and per capita living area of the family. City was treated as a random effect in all the models.

Statistically significant association (P < 0.05).

address for exposure estimate, and sources and components of $PM_{2.5}$ in study regions. Almost all of these few epidemiological studies on $PM_{2.5}$ and childhood sleep had some limitations, which hindered the ability to reach a definitive conclusion on the association of interest.

We found spatial heterogeneity in the associations between PM and sleep disorders across three provinces. This disparity could potentially be attributed to various characteristics of study sites, including indoor air pollution, weather patterns, population susceptibility, particle size, chemical constituents and sample size. In this study, Guangdong with lower PM levels presented higher effect estimates per increment of PM concentrations, compared with Liaoning and Xinjiang with higher PM levels. Previous studies have indicated that the concentration–response functions of PM exposure and mortality typically flatten out at higher concentrations (Liu et al., 2019). Moreover, because of cold climate in both northern regions, residents tend to prolong their indoor stays during winter and depend more extensively on solid fuels for indoor heating, thereby mitigating the impact of outdoor PM. Another possible explanation for variability in the associations is that the sources and toxicity of particles may vary regionally. A recent study in 31 Chinese cities has indicated that Guangzhou city (one of cities in Guangdong province in this study) had greater oxidative toxicity than Liaoning and Xinjiang cities (Zhang and Yao, 2022). In addition, the small sample size in Xinjiang might in part explain the less significant or null associations.

Particle size is one of the key factors determining the magnitude of hazardous effects of PM. However, the findings from previous human studies comparing the effects of size-segregated PM are controversial. Recent studies have shown that PM_1 is more harmful than $PM_{2.5}$ on childhood lung function (Yang et al., 2020), pneumonia (Wang et al., 2021), attention deficit hyperactivity disorder (Liu et al., 2023) and asthma and wheeze (Wu et al., 2022). Nevertheless, one previous study



Fig. 2. The odds ratios and 95 % confidence intervals of PM quartiles in relation to sleep disorders. The solid rectangles represent the effect estimates and the black line represents 95 % confidence intervals. Q1: below the 25th percentile of PM₁ concentration ($<25.4 \ \mu g/m^3$; the reference group) and PM_{2.5} concentration ($<31.0 \ \mu g/m^3$); Q2: the 25th to 50th percentile of PM₁ concentration ($\geq 25.4-28.7 \ \mu g/m^3$) and PM_{2.5} concentration ($31.0-35.7 \ \mu g/m^3$); Q3: the 50th to 75th percentile of PM₁ concentration ($\geq 28.7-41.2 \ \mu g/m^3$) and PM_{2.5} concentration ($35.7-48.8 \ \mu g/m^3$); Q4: above 75th percentile of PM₁ concentration ($\geq 41.2 \ \mu g/m^3$) and PM_{2.5} concentration ($\geq 28.7-41.2 \ \mu g/m^3$). Abbreviations: DA, Disorders of arousal, DIMS, Disorders of initiating and maintaining sleep; DOES, Disorders of excessive somnolence; SBD, Sleep breathing disorders; SHY, Sleep hyperhidrosis; SWTD, Sleep-wake transition disorders.

observed similar estimates of childhood hypertension in relation to PM1 and PM_{2.5} exposures (Wu et al., 2020). The associations between PM₁ exposure and childhood sleep disorders have been rarely examined. In the present study, PM₁ seems to have stronger associations with sleep disorders than PM25 in Guangdong, whereas similar associations between PM were detected in Xinjiang and Liaoning. Also, a study from northeast China observed that the effect sizes between PM1 and PM2.5 exposures with childhood sleep disorders were not heterogeneous (Lawrence et al., 2018). We speculated that the regional variation in the associations of PM1 and PM2.5 may be due in part to different particlesize distribution, components and toxicity of PM by region. For example, Xinjiang suffers from severe natural dust of PM (Zhu et al., 2018). Fossil fuel/biomass is an important source due to large amount of coal combustion for residential heating/field burning of crop residue in Liaoning, whereas vehicular exhaust emissions that contain more toxic components of PM make the greatest contribution to the concentration of ambient PM in Guangdong (Zhu et al., 2018). The lower toxicity of PM in Liaoning than in Guangdong (Zhang and Yao, 2022) might partially explain the similar associations of PM₁ and PM_{2.5} in Liaoning.

The magnitudes in associations between children with and without preterm birth, cesarean section, low birth weight or asthma were not consistent, contrary to what we expected based on the existing evidence (Gee and Payne-Sturges, 2002). We did not observe larger associations between PM exposures and sleep disorders in children having early-life risk factors than their counterparts. Despite this, our results were not unexplained because the former, facing multiple competitive causes of health, might be less affected by air pollution alone, while the latter are protected from several early-life risk factors and thus might be more affected by air pollution.

The adverse associations between PM exposures and sleep are biological plausible. It has been documented that PM interferes with sleep health by affecting respiratory and central nervous systems (Liu et al., 2020). Inhalation of PM, especially ultrafine particles, can directly access the central nervous system through blood circulation (Block and Calderon-Garciduenas, 2009; Schraufnagel et al., 2019) and through the olfactory nerve (Elder et al., 2006; Zanobetti et al., 2010), leading to dysregulated neurochemicals and/or physiological activity and impaired sleep health (Liu et al., 2021). In addition, PM has been documented to cause edema of mucous membranes by triggering tonsils/upper airway inflammation, which further increases restriction and obstruction of normal airflow, and leads to poor sleep quality and breathing-related sleep disorders (Liu et al., 2020, 2021).

This study has several strengths. First, we estimated PM concentrations based on both residential and school addresses and had a fine-scale

Table 3

Odds ratios of sleep disorders associated with home-school exposures to ambient particulate matters concentrations $>10 \ \mu g/m^3$ by region.

Odds ratios (95 % confidence intervals) ^a	Xinjiang	Liaoning	Guangdong
Global sleep disorder			
PM ₁	1.41 (0.76.	1.12 (1.01.	4.00 (3.10.
	2.72)	1.24)*	5.16)*
PM2 5	1.10 (0.90,	1.11 (1.01.	2.06 (1.77.
2.0	1.37)	1.21)*	2.38)*
DIM			
DIMS	1.25 (0.72	1 01 (0 00	1 00 (1 60
PM1	1.35(0.72, 0.64)	1.01 (0.90,	1.98 (1.00,
DM	2.04)	1.12)	2.44)
P1012.5	1.10 (0.89,	1.02 (0.92,	1.42 (1.20,
	1.36)	1.12)	1.01)
SBD			
PM_1	1.06 (0.58,	1.07 (0.99,	3.80 (2.94,
	2.01)	1.17)	4.91)*
PM _{2.5}	1.04 (0.85,	1.07 (0.99,	2.03 (1.74,
	1.29)	1.15)	2.36)*
DA			
PM_1	1.90 (1.18,	1.07 (0.96,	1.44 (1.09,
	3.15)*	1.18)	1.89)*
PM _{2.5}	1.20 (1.03,	1.06 (0.97,	1.16 (1.01,
	1.41)*	1.16)	1.37)*
SWTD			
PM	1.93 (1.07,	1.16 (1.07,	3.01 (2.29.
-	3.69)*	1.26)*	3.96)*
PM _{2.5}	1.26 (1.03,	1.16 (1.07,	1.69 (1.44,
	1.56)*	1.25)*	1.98)*
DOES			
DOES	1 10 (0 60	1 10 (1 00	2 OF (2 28
Pivi1	1.10 (0.09,	1.19 (1.09,	2.01)*
DM	1.70)	1.30)	3.91) 1.94 (1.EO
P1012.5	1.03 (0.88,	1.19 (1.09,	2 1 2)*
	1.21)	1.20)	2.13)
SHY	1 10 (0 (1	1 05 (0 05	0 50 (0 10
PM_1	1.12 (0.61,	1.05 (0.97,	2.72 (2.18,
DM	2.18)	1.15)	3.38)*
PIM _{2.5}	1.10 (0.90,	1.06 (0.97,	1.70 (1.49,
	1.371	1.14)	1.93)*

Abbreviations: DA, Disorders of arousal, DIMS, Disorders of initiating and maintaining sleep; DOES, Disorders of excessive somnolence; PM₁, particles with airborne diameters <1 μ m; PM_{2.5}, particles with airborne diameters <2.5 μ m; SBD, Sleep breathing disorders; SD, standard deviation; SHY, Sleep hyperhidrosis; SWTD, Sleep-wake transition disorders.

^a Adjusted for age, sex, preterm, caesarean, breastfeeding, low birth weight, asthma, eczema, relatively weight, physical activity, school assignment, season of assessment, parental education attainment, annual household income, current second-hand smoking exposure, house renovation, visible mold or dampness, and per capita living area of the family, city was treated as a random effect.

Statistically significant association (P < 0.05).

spatiotemporal assessment for PM pollutants, which enabled precision of the present findings. Second, we assessed multiple types of sleep disorders using a validated questionnaire. Third, we considered a multitude of potential confounders in the model to calculate adjusted associations. Fourth, our findings provide evidence of both $PM_{2.5}$ and PM_1 exposures with sleep disorders in school-aged children from a large, multicity population across three provinces. Our province-specific findings can be extrapolated to other similar ambient PM pollution contexts, such as other areas of northwest, northeast and southeast China, which suffer from severe natural dust, fossil fuel/biomass combustion emissions and higher PM toxicity, respectively. Nevertheless, the generalization of our findings requires caution due to the diversity of environmental conditions and populations. Additional region-specific studies are warranted to explore between-region differences in PM–sleep associations.

Our study also has some limitations. First, the cross-sectional nature of this study might be unable to provide evidence of causal relationships between PM exposures and childhood sleep disorders. We excluded children who had not lived in their addresses for the last four years, which might reduce the likelihood of inverse causality. Second, recall bias is unavoidable because ascertainment of childhood sleep disorders was acquired by parents-reported questionnaires. Nevertheless, the SDSC has previously been validated and suitable for large-scale epidemiological studies (Bruni et al., 1996). Third, as the same exposure level was assigned to all a school when estimating school PM concentration, exposure misclassification may occur. Moreover, we used ambient home-school PM concentration as an exposure indicator, omitting the contribution of exposure in other microenvironments such as indoor PM concentration as well as during commutes and elsewhere, which may not directly reflect individual exposure level. These exposure assessment errors, known as Berkson's error, might introduce little to no bias in point estimates. The best solution would be for individuals to wear samplers, but this approach is not feasible for a study with a large sample size. We plan to develop evaluation method suitable for joint exposure of indoor, commuting, and ambient exposures surrounding home and school in our future work. Fourth, because PM composition data were not available, we were unable to analyze the associations between the source and chemical composition of PM with childhood sleep disorders. Fifth, we did not adjust for other potential covariates such as nighttime noise exposure, shared living space, peer pressure and parental support, providing opportunity for residual confounding.

5. Conclusion

Our large multicity study of school-aged Chinese children shows that long-term exposures to PM_1 and $PM_{2.5}$ were associated with higher risks of sleep disorders. These associations were regionally heterogenous, with stronger associations of PM_1 and $PM_{2.5}$ in Guangdong province, and stronger PM_1 associations than $PM_{2.5}$ in Guangdong. These findings add new evidence on the relationship between PM exposures, PM_1 in particular, and childhood sleep disorders. Furthermore, our findings suggest that policy makers should consider not only PM mass concentrations and sizes, but also PM composition and sources when further carrying out air purification actions. More well-designed multi-center longitudinal investigations are essential to validate our finding.

CRediT authorship contribution statement

Zhao-Huan Gui: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Methodology, Formal analysis, Data curation, Conceptualization. Joachim Heinrich: Writing – review & editing, Conceptualization. Zhengmin (Min) Qian: . Mario Schootman: Writing – review & editing. Tian-Yu Zhao: Writing – review & editing. Shu-Li Xu: Investigation. Nan-Xiang Jin: Resources. He-Hai Huang: Investigation. Wan-Ting He: Investigation. Qi-Zhen Wu: Investigation. Jing-Lin Zhang: Investigation. Dao-Sen Wang: Investigation. Mo Yang: Investigation. Ru-Qing Liu: Writing – review & editing. Xiao-Wen Zeng: Writing – review & editing. Guang-Hui Dong: Writing – review & editing, Supervision, Resources, Project administration, Funding acquisition. Li-Zi Lin: Writing – review & editing.

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.

Data availability

Data supporting the findings of this study are available in the Supplementary Information. Data requests should be sent to the corresponding author and will be responded to within 30 days.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi. org/10.1016/j.envint.2024.108841.

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