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Long-term exposure to major constituents of fine particulate matter and sleep disorders among children and adolescents: A population-based survey in Guangdong province, China

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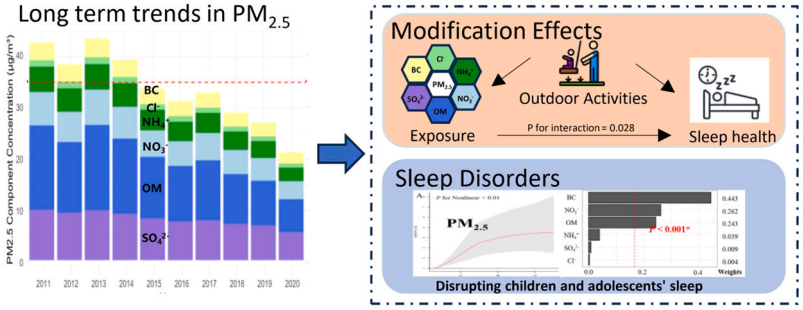
HIGHLIGHTS

- This study first explored the link between $PM_{2.5}$ constituents and children's sleep disorders.
- Exposure to $PM_{2.5}$ and its constituents increased the risk of sleep disorders.
- Black carbon contributed most to the observed association.
- Participants with shorter outdoor exercise duration time were more susceptible.

GRAPHICAL ABSTRACT



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ABSTRACT

Long-term exposure to $PM_{2.5}$ is associated with sleep health in adults, but its impact on children and adolescents remains unclear. This cross-sectional study analyzed 64,203 children and adolescents (6–18 years) from four cities in Guangdong, China, to assess the impact of $PM_{2.5}$ and its major constituents on sleep disorders. Sleep disorders were assessed using the Sleep Disturbance Scale for Children. Generalized linear mixed models and

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weighted quantile sum regression were applied to assess the joint effects of $PM_{2.5}$ constituents. The results indicated a positive association between $PM_{2.5}$ and sleep disorders. For example, the odds of sleep disorder increased with per interquartile range (IQR) increase in $PM_{2.5}$ concentrations [odds ratio (OR): 1.15, 95 % confidence interval (CI):1.06, 1.26). Black carbon (BC) and organic matter (OM) increase the odds by 16 % (95 % CI: 9, 24 %) and 23 % (95 % CI: 9, 39 %), respectively. Notably, combustion-related BC (44.3 %) and OM (24.3 %) contributed the key contributors to the mixture risk. Children and adolescents with limited outdoor activity (< 2 hours/day) were more vulnerable to $PM_{2.5}$ exposure (*P* for interaction = 0.028). These findings highlight that stricter air pollution regulations and targeted measures to reduce children's exposure to combustion-derived pollutants are essential for protecting sleep health and overall well-being.

1. Introduction

Healthy sleep is essential for children and adolescents' physical and mental health, cognitive development, and attention [1–3]. Growing evidence suggests that sleep disorders such as insufficient sleep, hypersomnia, and insomnia not only compromise these critical developmental domains during childhood but may also predispose individuals to health complications in adulthood [4,5]. Globally, epidemiological studies estimate that 20–30 % of children experience at least one sleep disorder [6,7], with China witnessing a concerning rise to approximately 40 % over the past two decades [8]. This escalating prevalence underscores the urgent need to identify modifiable risk factors, among which environmental determinants have emerged as significant contributors.

Ambient particulate matter($PM_{2.5}$) pollution represents a major environmental health challenge worldwide [9]. Epidemiological research has shown links between $PM_{2.5}$ exposure and sleep disorders in adults [10–12]. However, evidence regarding the impact in children or adolescents remains limited and inconsistent. Among six studies examining this association, demonstrated significant relationships between $PM_{2.5}$ exposure and pediatric sleep impairments, including sleep-disordered breathing and daytime sleepiness, while one Chilean multi-regional study e did not find this association (see in Table S1) [13–18]. Notably, existing research has primarily focused on the mass concentration of $PM_{2.5}$, with critical knowledge gaps regarding the effects of specific chemical constituents. Emerging evidence suggests that key constituents of $PM_{2.5}$, such as black carbon (BC) and organic matter (OM) may exert differential impacts on adult sleep health [19,20], yet their roles in pediatric populations remain largely unexplored.

Experimental studies have begun elucidating potential mechanisms through which $PM_{2.5}$ constituents may influence sleep physiology. BC and OM have been shown to disrupt neurochemical regulation by altering serotonin metabolism and melatonin balance [21]. Additionally, $PM_{2.5}$ deposition in the airways can trigger inflammatory responses, increase airflow restriction and obstruction, damage respiratory cells, and contribute to sleep-related breathing disorders [22]. Furthermore, emerging evidence suggests these components may induce calcium dysregulation and sympathetic activation, potentially disrupting cardiac rhythm during sleep phases [23].

The Pearl River Delta (PRD) region presents a unique context for investigating these associations, with ambient $PM_{2.5}$ concentrations demonstrating a downward trajectory from moderately polluted levels (42 $\mu g/m^3$ in 2014) to within China's National Ambient Air Quality Standard limits (19 $\mu g/m^3$ in 2022)[24]. While most previous investigations have concentrated on health impacts of higher pollution levels, the consequences of chronic exposure to lower $PM_{2.5}$ concentrations, particularly through constituent-specific pathways, remain poorly understood. To address these gaps, a large-scale study was conducted among children and adolescents in the PRD region to investigate the associations between long-term exposure to major constituents of $PM_{2.5}$ and sleep disorders. Our findings aim to inform targeted interventions by delineating the specific $PM_{2.5}$ constituents that most substantially impact pediatric sleep health.

2. Method

2.1. Study design and participants

This extensive cross-sectional study investigated environmental health impacts in Guangdong province, China, as previously described in our prior publication [25]. In brief, local residents were recruited through multistage random sampling of schools across four cities (Foshan, Shenzhen, Zhongshan, and Maoming) selected to represent geographic heterogeneity in air pollution profiles and socioeconomic characteristics. Eligibility criteria included an age range of 6–18 years and a minimum residency of two years at their current location. A total of 69,610 individuals were initially recruited, of whom 64,203 completed the questionnaire and had sufficient baseline data to be included in the analysis (Fig. S1). The data collection spanned from 2020 to 2021. All participants provided informed consent before enrollment, and the study was approved by the Human Studies Committee of Sun Yat-sen University.

2.2. Sleep disorders measurement

Sleep disorders were assessed using the validated Chinese version of the Sleep Disturbance Scale for Children (C-SDSC), a tool that is applicable to children and adolescents aged 3–18 years, and is widely used in clinical and epidemiological studies as a screening tool for sleep disorders [26]. The C-SDSC has been validated in Chinese children, demonstrating good internal consistency (Cronbach $\alpha = 0.81$) [27], and has been widely used in epidemiological studies on sleep disorders among Chinese children and adolescents [2,28].

The scale comprises 26 items assessing sleep quality over the past six months, with scores indicating overall sleep status. Among these, 24 items are assessed using a 5-point Likert scale, covering six subtypes of sleep disorders, while 2 items specifically evaluate sleep duration and sleep latency. The Likert scale ranges from 1 (never) to 5 (always, 6–7 times per week). Sleep duration is rated from 1 (> 9 hours) to 5 (< 5 hours), and sleep latency is rated from 1 (< 15 minutes) to 5 (> 1 hour).

Sleep disorders and their subtypes are evaluated based on the calculated scores. The total sleep disorder score ranges from 26 to 130, while the scores for the subtypes are as follows: sleep breathing disorders (SBD, 3–15), disorders of excessive somnolence (DOES, 5–25), disorders of arousal (DA, 3–15), disorders of initiating and maintaining sleep (DIMS, 7–35), sleep-wake transition disorders (SWTD, 6–30), and sleep hyperhidrosis (SHY, 2–10). Higher scores indicate a higher risk of sleep disorders. To facilitate comparisons, the raw scores were converted to T-scores, with scores > 70 indicating the presence of sleep disorder symptoms. Additionally, based on international consensus recommendations, sleep duration was classified as short (< 7 hours) and sleep latency as long (> 45 minutes).

2.3. Exposure assessment

Data on $PM_{2.5}$ and its major constituents were sourced from the ChinaHighAirPollutants (CHAP, https://weijing-rs.github.io/product.ht

ml) database, which provides nationwide coverage with a spatial resolution of 1 × 1 km. Considering the sources, composition, and previous research on $PM_{2.5}$, this study focuses on six major constituents: ammonium (NH_4^+), nitrate (NO_3^-), chloride (Cl^-), sulfate (SO_4^{2-}), OM, and BC, which are widely applied in epidemiological studies [29–31]. The development and validation of the dataset have been thoroughly documented in prior research [30,32]. The data were generated using the four-dimensional spatiotemporal deep forest (4D-STDF) model designed by Jing Wei and collaborators [33,34]. This model employs deep forest techniques to estimate real-time concentrations of $PM_{2.5}$ and its major constituents [30]. Inputs to the model include ground-based $PM_{2.5}$ chemical measurements, satellite-derived $PM_{2.5}$ data, CAMS emission inventories, ERA5 meteorological parameters, surface-related and demographic variables, as well as spatial-temporal terms. It was validated using tenfold cross-validation, demonstrating high data quality (annual R^2 values of 0.87–0.95). The annual average concentrations of $PM_{2.5}$ and its major constituents were matched to participants' residential addresses. To represent long-term exposure, the 5-year mean concentrations (2016–2020) preceding the survey were utilized.

2.4. Covariates

Participant data on socio-demographic characteristics, behavioral habits, and health-related factors were obtained through administration of a structured questionnaire. The collected information included age, gender (boys or girls), height, weight, residential addresses, annual household income (< 30,000 Yuan, 30,000–100,000 Yuan, ≥ 100,000 Yuan), parents' education level (defined by the highest attainment of either parent, categorized as less than high school or high school or greater), low birth weight (defined as under 2500 g, yes or no), premature (defined as less than 37 weeks, yes or no), breastfeeding (≤ 3months, yes or no), residential proximity to major roadways (classified as within 100 m or beyond), daily outdoor activity duration (< 2 hours/day or ≥ 2 hours/day), secondhand smoke exposure (yes or no), presence of respiratory diseases (defined based on a parental-reported questionnaire, including conditions such as asthma, chronic bronchitis, or other lung diseases, categorized as yes or no), and allergic diseases (defined based on a parental-reported questionnaire, including conditions such as allergic rhinitis and atopic dermatitis, categorized as yes or no). Additionally, childhood obesity and overweight status were assessed using Body mass index (BMI) z-scores, which were age- and gender-standardized according to the WHO growth reference. BMI was calculated by dividing weight (in kilograms) by the square of height (in meters).

Ambient temperature and relative humidity data were sourced from the ERA5 dataset, the fifth generation ECMWF atmospheric reanalysis of the global climate, which provides hourly estimates of various atmospheric parameters at a 31 km global resolution [35]. Data on other pollutants, including PM_{10}, SO_2, O_3, NO_2, and CO, were obtained from the CHAP dataset, which has been detailed in previous studies [36–38].

Following prior research, a directed acyclic graph (DAG) was created using DAGitty 3.1 software (Fig. S2) to determine the essential set of variables for adjustment. The selected covariates for adjustment included: 1) child-related factors: age, gender, obesity, outdoor activity duration, breastfeeding, premature, and low birth weight; 2) parental and household factors: parents' education level and annual household income; 3) environmental exposure factors: exposure to secondhand smoke, residential proximity to major roadways, ambient temperature, and relative humidity.

2.5. Statistical analysis

In this study, participants were screened for sleep disorders using the C-SDSC scale. Those with more than 10 missing values were excluded, while those with fewer missing values were imputed using the K-nearest neighbors (KNN) method. Continuous covariates were imputed using multiple imputation (MI) based on predictive mean matching, while categorical covariates were imputed using KNN to ensure data robustness and completeness. Continuous variables are presented as means with standard deviations (SD), and categorical variables as frequency counts.

Restricted cubic spline (RCS) functions with three knots were used to analyze the associations between $PM_{2.5}$ and its constituents with sleep disorders, capturing nonlinear exposure-outcome associations through smooth segmented functions. The optimal degrees of freedom were selected based on the Akaike Information Criterion (AIC), and model fit was assessed via likelihood ratio tests, with their p-values used to determine potential nonlinearity. Given the non-linear nature of most pollutant-outcome relationships in the RCS functions, generalized linear mixed models (GLMMs) were applied to estimate the odds ratios (ORs) and corresponding confidence intervals (CIs) for sleep disorders at higher $PM_{2.5}$ concentrations and its major constituents, compared to the lowest quartile. GLMMs have been widely used in risk assessment and statistical analysis due to their ability to account for both fixed and random effects in complex data structures [39]. In these models, city was treated as a random effect, while air pollutants and the adjusted covariates were considered fixed effects. Furthermore, given the differences in the distribution of $PM_{2.5}$ component concentrations and their direct impact on observed associations, as well as the linear relationship of certain constituents with sleep disorders, the ORs were calculated for the increase in $PM_{2.5}$ and its constituents at each interquartile range (IQR) increment.

To assess the combined effects of $PM_{2.5}$ constituents on sleep disorders, weighted quantile sum (WQS) regression and Quantile G-Computation (Qgcomp) model were employed, which are validated methods widely used for risk assessment in environmental mixture exposure studies [40,41]. In the WQS model, the same covariates as in the primary model were adjusted using a quartile-based approach, with the number of bootstrap samples set to 100 and a positive constraint enforced to ensure that the WQS index captures only adverse effects. To identify key constituents with notable contributions, an average weight threshold at 16.7 % was applied, corresponding to an equal distribution among the six constituents. Weights exceeding this threshold and showing a significant association were considered as contributing beyond what would be expected.

The Qgcomp model was employed to assess the joint effects of complex component exposures, which combines WQS regression and g-computation, applying a soft constraint on the direction of weights so that they can be both positive and negative simultaneously [41]. This method employs g-computation to estimate causal effects between exposures and health outcomes, and to evaluate the weights of complex component exposures.

Various sensitivity analyses were conducted to assess the robustness and reliability of our findings. First, the interaction effects of potential effect modifiers including children's age, gender, outdoor activity duration, annual household income, parents' education level and residential type (rural or urban, classified as rural if the GDP of the residential district/county was <100 billion Yuan and urban if the GDP was ≥100 billion Yuan), were examined. These variables were added to the primary model to assess the interaction effects, and stratified analysis provided information for further interpretation. Additionally, the following sensitivity analyses were performed: 1) Based on literature reporting the effects of asthma, pollen allergies, and other diseases on sleep quality, individuals with self-reported respiratory and allergic conditions were excluded to assess the robustness of the associations [42]; 2) The impact of different exposure periods was explored by using the average concentration of air pollutants over varying time frames; 3) The effect of school exposure was examined by using the average concentration of air pollutants matched to school addresses; 4) To evaluate city-specific influences, analyses were repeated by sequentially excluding individuals from one city at a time and assessing the effects in the remaining three cities; 5) Considering that including cities as

random effects may obscure within-city spatial correlations, an additional model with district/county as a random effect was used to examine the influence of finer spatial levels within cities; 6) To investigate the potential influence of other air pollutants, additional adjustments were made for gaseous pollutants (SO_2, CO, PM_{10}) to assess their impact on the results. All analyses were conducted utilizing R software (version 4.3.2), with statistical significance set at a two-tailed *P*-value < 0.05.

3. Results

3.1. Study characteristics

The socio-demographic characteristics of the study participants are detailed in Table 1. A total of 64,203 children and adolescents were included, with the mean (SD) age of 9.98 ± 2.89 years. Among them, 34,758 (54.1 %) were boys and 29,445 (45.9 %) were girls. Most participants had parents with at least a high school education (70.4 %), were breastfed (76.9 %), were not exposed to secondhand smoke (84.1 %), and had no respiratory diseases (98.4 %) or allergic conditions (90.7 %). A total of 2238 children and adolescents (3.5 %) were identified as having sleep disorders, with the prevalence of six subtypes reported as follows: 3.9 % (DIMS), 5.9 % (SBD), 6.6 % (DA), 3.4 % (SWTD), 5.6 % (DOES); 6.6 % (SHY). Regarding sleep quality, 9977 children and adolescents (15.5 %) experienced short sleep duration, whereas 2.0 % (1296 children and adolescents) reported prolonged sleep latency.

Table 1
Characteristics of the study participants stratified by children's gender (N = 64,203).

Variables	Overall	Boys	Girls	*P*
	(N = 64203)	(N = 34,758)	(N = 29,445)	
Age (years), (mean (SD))	9.98 (2.89)	9.99 (2.89)	9.97 (2.90)	0.373
Obesity, n (%)	5620 (8.8)	4234 (12.2)	1386 (4.7)	< 0.001
Annual household income, n (%)				
<30,000 Yuan	20,214 (31.5)	10,778 (31.0)	9436 (32.0)	0.014
30,000–100,000 Yuan	19,640 (30.6)	10,746 (30.9)	8894 (30.2)	
≥ 100,000 Yuan	24,349 (37.9)	13,234 (38.1)	11,115 (37.7)	
Parents' education level, n (%)				
Less than high School	19,003 (29.6)	10,423 (30.0)	8580 (29.1)	0.019
High school or greater	45,200 (70.4)	24,335 (70.0)	20,865 (70.9)	
Low birth weight, n (%)	3651 (5.7)	1790 (5.1)	1861 (6.3)	< 0.001
Premature, n (%)	3208 (5.0)	1821 (5.2)	1387 (4.7)	0.002
Breastfeeding, n (%)	49,366 (76.9)	26,902 (77.4)	22,464 (76.3)	0.003
Residential proximity to major roadways (<100 m), n (%)	24,150 (37.6)	13,310 (38.3)	10,840 (36.8)	< 0.001
Outdoor activity duration (<2 h/day), n (%)	36,308 (56.6)	20,359 (58.6)	15,949 (54.2)	< 0.001
Secondhand smoke exposure (SHS), n (%)	10,189 (15.9)	5576 (16.0)	4613 (15.7)	0.198
Respiratory diseases, n (%)	1043 (1.6)	602 (1.7)	441 (1.5)	0.021
Allergic diseases, n (%)	5946 (9.3)	3614 (10.4)	2332 (7.9)	< 0.001
Subtypes of sleep disorders				
Sleep disorder, n (%)	2238 (3.5)	1149 (3.3)	1089 (3.7)	0.007
DIMS, n (%)	2496 (3.9)	1181 (3.4)	1315 (4.5)	< 0.001
SBD, n (%)	3815 (5.9)	2327 (6.7)	1488 (5.1)	< 0.001
DA, n (%)	4268 (6.6)	2293 (6.6)	1975 (6.7)	0.587
SWTK, n (%)	2164 (3.4)	1122 (3.2)	1042 (3.5)	0.031
DOES, n (%)	3578 (5.6)	1730 (5.0)	1848 (6.3)	< 0.001
SHY, n (%)	4266 (6.6)	2848 (8.2)	1418 (4.8)	< 0.001
Shorter sleep duration, n (%)	9977 (15.5)	4810 (13.8)	5167 (17.5)	< 0.001
Longer sleep latency, n (%)	1296 (2.0)	630 (1.8)	666 (2.3)	< 0.001

Abbreviations: SD, standard deviation; DIMS, disorders of initiating and maintaining sleep; SBD, sleep breathing disorders; DA, disorders of arousal, SWTD, sleep-wake transition disorders; DOES, disorders of excessive somnolence; SHY, sleep hyperhidrosis.

The estimated 5-year average concentrations of $PM_{2.5}$ and its major constituents are shown in Table 2 and Fig. S3. $PM_{2.5}$ levels ranged from 21.29 to 37.67 μg/m^3, with a median concentration of 28.15 μg/m^3 (IQR 1.54 μg/m^3). The median concentrations of the six constituents were 3.37 μg/m^3 (NH_4^+), 4.25 μg/m^3 (NO_3^-), 6.99 μg/m^3 ($SO4^{2-}$), 9.51 μg/m^3 (OM), 2.63 μg/m^3 (BC), and 0.90 μg/m^3 (Cl^-). Significant positive correlations were observed between $PM_{2.5}$ and its major constituents, with Spearman correlation coefficients (ρ) ranging from 0.47 to 0.83.

3.2. Associations between $PM_{2.5}$ constituents and sleep disorders

Fig. S4 shows the dose-response relationship between $PM_{2.5}$ and its major constituents and sleep disorders after adjusting for covariates. Except for Cl^-, $PM_{2.5}$ and its major constituents exhibited a significant nonlinear relationship with sleep disorders. At low exposure levels, all six constituents showed a linear relationship with sleep disorders, but this association plateaued or weakened at higher concentrations. Therefore, the associations were assessed using both quartile and continuous exposure methods.

The adjusted models appropriately selected covariates, with variance inflation factors (VIFs) for all covariates being less than two, indicating no significant multicollinearity among them (Table S2). In the adjusted single-constituent models, exposure to $PM_{2.5}$ and its major constituents OM, and BC was significantly associated with an increased risk of sleep disorders (Table 3). For $PM_{2.5}$, an increase of per IQR in $PM_{2.5}$ mass concentration was significantly associated with an increased risk of sleep disorders (adjusted OR: 1.15 [95 % CI: 1.06, 1.26]). Additionally, BC and OM demonstrated relatively stronger associations with sleep disorder, with the odds of sleep disorders increasing by 16 % (95 % CI: 9, 24 %) for BC and 23 % (95 % CI: 9, 39 %) for OM with each IQR increase in their mass concentrations. Significant positive associations were observed for SBD, DOES, and shorter sleep duration within sleep disorder subtypes, with ORs for the constituents similar to those observed for total sleep disorders (Tables S3 – S10).

In the WQS analysis, each IQR increase in the $PM_{2.5}$ constituent mixture index was significantly linked to a higher risk of sleep disorders (OR = 1.08; 95 % CI: 1.03–1.13), SBD (OR = 1.10; 95 % CI: 1.02–1.17), DOES (OR = 1.04; 95 % CI: 1.00–1.08), and shorter sleep duration (OR = 1.14; 95 % CI: 1.07–1.20) (Table S11). Fig. 1 shows the contribution weights of $PM_{2.5}$ and its major constituents in the WQS index. BC played an influential critical role in the association with sleep disorders, SBD, DOES, and shorter sleep duration, with component weights of 0.443, 0.388, 0.195, and 0.455, respectively. Similar results were observed in the Qgcomp analysis, where BC was the key component with the highest positive weight in the associations with the above sleep disorders and subtypes (Fig. S5).

3.3. Stratified analysis

The results of the stratified analyses for the relationships between $PM_{2.5}$ and its major constituents and sleep disorders by potential effect modifiers are presented in Fig. 2 and Fig. S6–S13. A stronger links between air pollutants and sleep disorders in boys and younger children. Participants from families with lower annual incomes or parents with

Table 2
Five years (2016–2020) annual average concentration of $PM_{2.5}$ and its major constituents, and pairwise Spearman correlation.

Pollutants (μg/m3)	Summary statistics					Spearman correlation coefficients						
	Mean (SD)	Median	Minimum	Maximum	IQR	$PM_{2.5}$	NH_4^+	NO_3^-	SO_4^{2-}	OM	BC	Cl^-
$PM_{2.5}$	28.30 (2.49)	28.15	21.29	37.67	1.54	1.00	0.79 *	0.61 *	0.83 *	0.47 *	0.65 *	0.57 *
NH_4^+	3.48 (0.39)	3.37	2.86	5.01	0.31		1.00	0.90 *	0.42	0.03	0.86 *	0.86 *
NO_3^-	4.60 (0.93)	4.25	3.38	7.90	0.77			1.00	0.15	−0.21	0.85 *	0.96 *
SO_4^{2-}	6.97 (0.52)	6.99	5.78	8.56	0.78				1.00	0.78 *	0.28	0.10
OM	9.59 (1.07)	9.51	4.18	11.55	1.50					1.00	0.04	−0.30
BC	2.69 (0.31)	2.63	1.64	3.59	0.44						1.00	0.79 *
Cl^-	0.97 (0.24)	0.90	0.57	2.22	0.36							1.00

Abbreviations: $PM_{2.5}$, particle with aerodynamic diameter $\leq$ 2.5 μm; NH_4^+, ammonium; NO_3^-, nitrate; SO_4^{2-}, sulfate; OM, organic matter; BC, black carbon; Cl^-, chloride; SD, standard deviation.
*$P < 0.05$.

lower educational attainment showed a higher risk of shorter sleep duration when exposed to $PM_{2.5}$ and its major constituents, including OM, and BC. Additionally, associations between $PM_{2.5}$ and its major constituents, except for OM, and sleep disorders were more pronounced in participants with limited outdoor physical activity. However, no significant effect modification by residential type was observed, as shown in Table S12.

3.4. Sensitivity analysis

Sensitivity analyses were conducted by excluded participants with respiratory diseases (e.g., asthma, pneumonia) and allergic diseases (e.g., allergic rhinitis, pollen allergy) (Table S13), explored the impact of different exposure periods by using average concentrations of $PM_{2.5}$ and its major constituents from different years (Table S14–S17), used school-level exposure as an alternative exposure indicator (Table S18) and then sequentially excluded participants from each city and analyzed the associations between air pollutants and sleep disorders in the populations of the remaining three cities (Tables S19–S22). Additionally, sensitivity analyses were performed by replacing cities with districts/counties as the random effect to account for differences in living conditions across cities (Table S23) and by further adjusting for other gaseous pollutants to control for potential confounding effects (Tables S24–S26). These adjustments had minimal impact on the effect estimates and did not result in substantive changes to the study findings.

4. Discussion

4.1. Key findings

To our knowledge, this study is the first to investigate the impact of $PM_{2.5}$ constituents on sleep disorders in children and adolescents. Our findings indicate that prolonged exposure to $PM_{2.5}$ is significantly associated with an increased risk of sleep disorders in this population. Specifically, it was linked to higher scores for SBD and DOES subtypes, as well as shorter sleep duration in children and adolescents. Furthermore, BC and organic matter OM were identified as key contributors to these associations, offering valuable insights for prioritizing $PM_{2.5}$ control strategies to reduce its adverse impact on pediatric sleep health.

4.2. Comparison with previous studies

Previous studies have consistently reported an association between higher $PM_{2.5}$ exposure and poorer sleep quality in children and adolescents, as summarized in Table S1. A large cross-sectional study across various regions in China found that a 10 $\mu g/m^3$ rise in $PM_{2.5}$ concentration (mean $\pm$ SD: 32.77 $\pm$ 3.75 $\mu g/m^3$) was associated with an increased risk of sleep disorders (OR = 1.24, 95 % CI: 1.14–1.35), with significant associations observed in several sleep subtypes on the SDSC scale [15]. Similarly, a nationwide study in China involving 115,023 individuals found that for each IQR increase in postnatal $PM_{2.5}$ exposure (median: 50 $\mu g/m^3$, IQR: 42–58 $\mu g/m^3$), the risk of sleep disorders in children and adolescents increased (OR = 1.10, 95 % CI: 1.04–1.15), as measured by the Children's Sleep Habits Questionnaire (CSHQ) [14]. However, a cross-sectional study conducted in Chile among children aged 5–9 years (N = 564) found no significant association between higher $PM_{2.5}$ exposure concentrations (48.4 $\pm$ 4.2 $\mu g/m^3$) and pediatric sleep-related breathing disorders [18].

Notably, most previous studies have focused on the effects at higher $PM_{2.5}$ concentrations. Since 2014, $PM_{2.5}$ levels in China have steadily declined [24]; however, evidence on the health effects of lower $PM_{2.5}$ exposure levels on sleep disorders in children and adolescents remains scare. Our study fills this gap by demonstrating that even at relatively lower $PM_{2.5}$ concentrations (median: 28.15 $\mu g/m^3$, range: 21.29–37.67 $\mu g/m^3$) in Guangdong province, the risk of sleep disorders in children and adolescents remains significantly elevated. These findings align with a mother-child pregnancy cohort study in Mexico, which found a significant association between lower maternal prenatal $PM_{2.5}$ exposure (23.0 [21.1–24.3] $\mu g/m^3$) and reduced sleep quality and shorter sleep duration, as objectively measured [13]. The evidence underscores the potential health risks of long-term exposure to even lower levels of $PM_{2.5}$ for vulnerable populations such as children and adolescents. Future policies should implement targeted interventions to reduce $PM_{2.5}$ exposure and mitigate its impact on pediatric health.

The dose-response relationship between $PM_{2.5}$ exposure and sleep disorders revealed a potential plateau effect at higher concentrations. Behavioral adaptation, such as reduced outdoor activities or air filtration use in highly polluted areas, may contribute to this effect [43]. Additionally, the assessment of extreme exposure values in the study population may also contribute to this phenomenon. Further research is needed to clarify the underlying mechanisms, which may inform strategies to mitigate the impact of $PM_{2.5}$ and its constituents on sleep health.

4.3. Impact of $PM_{2.5}$ constituents

Previous studies primarily focused on the association between $PM_{2.5}$ mass concentration and sleep disorders in children, overlooking the effects of its chemical constituents. Given the complex effects of $PM_{2.5}$ and its constituents, our study applied multiple statistical models to comprehensively assess its impact on sleep disorders. GLMM was used to estimate single-pollutant effects but could not address multicollinearity among pollutants [39]. To capture mixture effects, WQS was applied under the assumption of uniform effect directions [40], while Qgcomp was used to allow for both positive and negative effects and detect nonlinear relationships [41]. The complementary use of these models enhances the robustness of our findings and provides a more comprehensive analytical perspective.

This study identified combustion-related BC and OM as key constituents influencing sleep disorders. As a key constituent of $PM_{2.5}$, originating from the incomplete combustion of fossil fuels, BC has been identified as a major contributor to sleep disturbances [44]. In

Table 3
The associations between sleep disorder and exposure to $PM_{2.5}$ and its major constituents.

Pollutants	OR (95 % CI)			
	Q1	Q2	Q3	Q4
$PM_{2.5}$, (μg/m^3)	21.29–27.14	27.14–28.15	28.15–28.68	28.68–37.67
Crude model	Ref	**1.32 (1.18, 1.48)**	**1.52 (1.31, 1.77)**	1.13 (0.92, 1.38)
Adjusted model[a]	Ref	**1.16 (1.02, 1.32)**	**1.35 (1.13, 1.61)**	1.04 (0.80, 1.33)
Per IQR increase[a]	**1.15 (1.06, 1.26)**	-	-	-
NH_4^+, (μg/m^3)	2.86–3.26	3.26–3.37	3.37–3.57	3.57–5.01
Crude model	Ref	**1.34 (1.18, 1.52)**	**1.28 (1.12, 1.46)**	1.02 (0.81, 1.29)
Adjusted model[a]	Ref	**1.24 (1.07, 1.42)**	**1.20 (1.04, 1.38)**	**1.23 (1.07, 1.41)**
Per IQR increase[a]	**1.03 (1.01, 1.05)**	-	-	-
NO_3^-, (μg/m^3)	3.38–3.98	3.98–4.25	4.25–4.75	4.75–7.90
Crude model	Ref	1.05 (0.92, 1.20)	**1.96 (1.75, 2.20)**	1.11 (0.98, 1.26)
Adjusted model[a]	Ref	0.95 (0.82, 1.10)	**1.37 (1.12, 1.69)**	**1.21 (1.06, 1.39)**
Per IQR increase[a]	**1.14 (1.03, 1.26)**	-	-	-
SO_4^{2-}, (μg/m^3)	5.78–6.49	6.49–6.99	6.99–7.27	7.27–8.56
Crude model	Ref	**1.25 (1.08, 1.45)**	**1.31 (1.07, 1.61)**	**1.33 (1.06, 1.66)**
Adjusted model[a]	Ref	1.15 (0.99, 1.33)	0.85 (0.68, 1.07)	0.87 (0.67, 1.12)
Per IQR increase[a]	**1.07 (1.02, 1.11)**	-	-	-
OM, (μg/m^3)	4.18–8.98	8.98–9.51	9.51–10.47	10.47–11.55
Crude model	Ref	**1.18 (1.06, 1.32)**	**1.32 (1.08, 1.61)**	**1.39 (1.13, 1.71)**
Adjusted model[a]	Ref	**1.11 (1.01, 1.25)**	**1.22 (1.01, 1.53)**	**1.20 (1.00, 1.54)**
Per IQR increase[a]	**1.23 (1.09, 1.39)**	-	-	-
BC, (μg/m^3)	1.64–2.48	2.48–2.63	2.63–2.92	2.92–3.59
Crude model	Ref	**1.40 (1.23, 1.59)**	**1.44 (1.26, 1.64)**	**1.71 (1.47, 1.99)**
Adjusted model[a]	Ref	**1.31 (1.15, 1.50)**	**1.35 (1.18, 1.54)**	**1.40 (1.23, 1.59)**
Per IQR increase[a]	**1.16 (1.09, 1.24)**	-	-	-
Cl^-, (μg/m^3)	0.57–0.78	0.78–0.90	0.90–1.14	1.14–2.22
Crude model	Ref	0.98 (0.85, 1.13)	1.06 (0.89, 1.27)	**1.24 (1.00, 1.53)**
Adjusted model[a]	Ref	0.93 (0.82, 1.07)	1.04 (0.90, 1.21)	**1.16 (1.01, 1.32)**
Per IQR increase[a]	**1.14 (1.06, 1.22)**	-	-	-

Abbreviations: $PM_{2.5}$, particle with aerodynamic diameter ≤ 2.5 μm; NH_4^+, ammonium; NO_3^-, nitrate; SO_4^{2-}, sulfate; OM, organic matter; BC, black carbon; Cl^-, chloride; IQR, interquartile range; OR, odds ratio; CI, confidence interval; Q1, quartile 1; Q2, quartile 2; Q3, quartile 3; Q4, quartile 4.
[a] Adjusted for children's age, gender, obesity, outdoor activity duration, feeding style, premature, low birth weight, Secondhand smoke exposure, parents' education level, annual household income, and residential proximity to major roadways.
Bold indicates statistical significance ($P < 0.05$).

Guangdong Province, China, combustion sources such as industrial and vehicle emissions account for 46 % of total $PM_{2.5}$ emissions, with BC being the predominant component [45]. Existing evidence includes two studies exploring $PM_{2.5}$ constituents, both of which focused on adults, while research on children and adolescents remains limited. A cross-sectional study conducted across five universities (ages 16–30 years) found a significant association between BC exposure and impaired sleep quality (OR = 1.26, 95 % CI: 1.11–1.43)[19]. Another large cohort study in southwestern China involving adults aged 30–79 years showed that exposure to $PM_{2.5}$ constituents increased the risk of sleep disorders (HR = 1.54, 95 % CI: 1.33–1.78), with OM identified as a critical factor [20]. Our findings demonstrate similar associations between BC and OM exposure and sleep disorders in children and adolescents as those observed in adults. Further studies specifically targeting children and adolescents are needed to validate these findings and better understand the underlying mechanisms in childhood.

Mechanistically, $PM_{2.5}$ constituents, particularly BC and OM, may contribute to sleep disorders through several pathways. First, BC and OM can directly reach the brain via the olfactory nerve, blood-brain barrier transport, or macrophage-mediated translocation, leading to increased reactive oxygen species (ROS) production [19,46]. This oxidative stress disrupts neurochemical homeostasis by altering serotonin metabolism and melatonin balance, ultimately impairing sleep regulation [21]. Second, $PM_{2.5}$ exposure, especially its carbonaceous components, can activate the NLRP3/NLRC4 inflammasome, triggering caspase-1/IL-1β activation and amplifying inflammatory response [46,47]. This chronic neuroinflammation may interfere with the sleep-wake cycle and contribute to sleep disturbances [19,48]. Third, BC tends to deposit in the upper respiratory tract, causing irritation and damage to nasal passages. This can lead to airflow restriction, increased respiratory resistance, and heightened susceptibility to sleep-related breathing disorders [10,22]. Additionally, emerging evidence suggests that $PM_{2.5}$ exposure may induce calcium dysregulation and sympathetic activation, potentially disrupting cardiac rhythm during sleep [23].

Given the multifaceted effects of BC and OM on both respiratory and neurochemical pathways, further research is needed to elucidate their precise mechanisms and develop targeted interventions. These findings underscore the critical role of BC and OM in children's sleep health, highlighting the need for stricter regulation of combustion sources and enhanced pediatric health interventions. Future policies should not only focus on air pollution control but also promote awareness among parents and schools in high-pollution areas to encourage preventive measures.

4.4. Modification effects of covariates

The heterogeneous effects of $PM_{2.5}$ and its major constituents were further examined across children and adolescents with different characteristics. Children and adolescents with less outdoor physical activity were more vulnerable to the effects of $PM_{2.5}$, leading to sleep disorders. This may be due to a lack of physical activity, which weakens the children's immune function [49]. Other studies have suggested that children and adolescents with more outdoor activity may experience increased fatigue, reduce nasal resistance, and enhance upper airway function, which improves sleep quality and stability [50].

It was also found that OM and BC had a more significant impact on the occurrence of sleep disorders, SBD, DOES, and shorter sleep duration in children under the age of 12, as detailed in Fig. 1, Fig. S7, S10, and S12. Compared to adults, younger children are particularly vulnerable to respiratory damage from air pollutants due to their developing respiratory systems and higher minute ventilation rates, which increases their susceptibility to sleep disorders [51]. Previous studies have also reported that $PM_{2.5}$ can cause upper airway cell damage, triggering mucosal inflammation or edema, leading to airway restriction and obstruction [21,52,53]. Additionally, exposure to $PM_{2.5}$ can induce oxidative stress, increase airway inflammation, and lead to upper airway narrowing and increased inspiratory resistance, ultimately contributing to SBD and other sleep disorders [54,55]. This study highlights the potential for differentiated effects of $PM_{2.5}$ and its major constituents on children and adolescents with different characteristics.

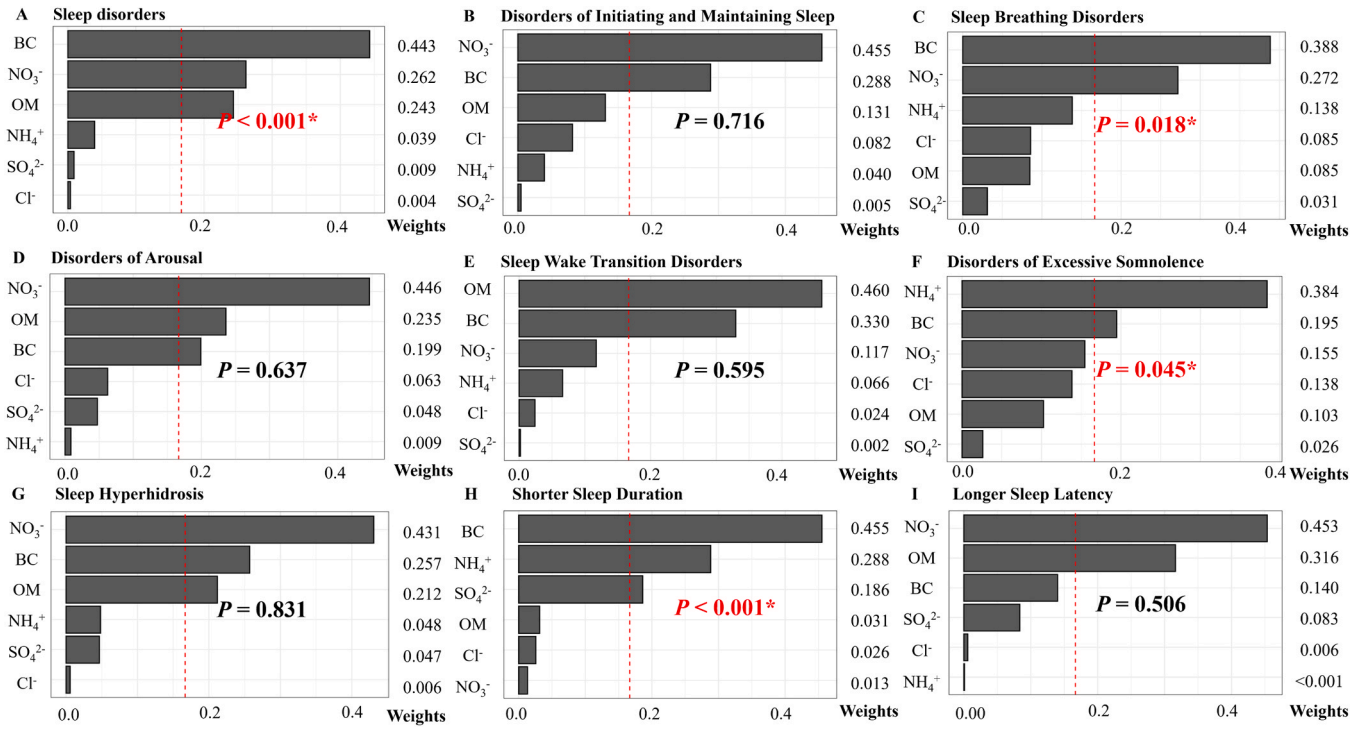

Fig. 1. The associations between $PM_{2.5}$ and its major constituents, and sleep disorders based on WQS regression analysis. Subplots represent sleep disorders (A), disorders of initiating and maintaining sleep (B), sleep breathing disorders (C), disorders of arousal (D), sleep-wake transition disorders (E), disorders of excessive somnolence (F), sleep hyperhidrosis (G), shorter sleep duration (H), and longer sleep latency (I). Abbreviations: NH_4^+, ammonium; NO_3^-, nitrate; SO_4^{2-}, sulfate; OM, organic matter; BC, black carbon; Cl^-, chloride; WQS, weighted quantile sum regression. * $P < 0.05$.

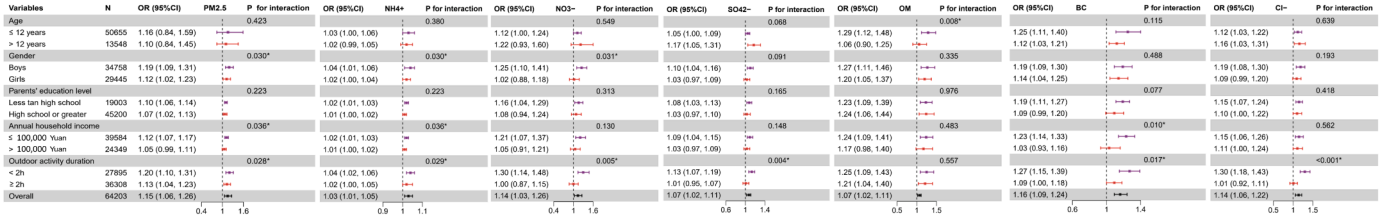

Fig. 2. The associations between $PM_{2.5}$ and its major constituent concentrations and the likelihood of sleep disorder among multiple subgroups. The subgroups include age, gender, parents' education level, annual household income, outdoor activity duration. The boxes represent ORs, with horizontal lines representing 95 % CI. Black boxes and horizontal lines represent the overall OR and 95 % CI. Purple and red boxes and horizontal lines represent the Subgroup OR and its 95 % CI. The black dashed line represents the reference line (OR = 1). Abbreviations: $PM_{2.5}$, particle with aerodynamic diameter ≤ 2.5 μm; NH_4^+, ammonium; NO_3^-, nitrate; SO_4^{2-}, sulfate; OM, organic matter; BC, black carbon; Cl^-, chloride; OR, odds ratio; CI, confidence interval. *$P < 0.05$, *P*-values were corrected for false discovery rate (FDR) using the Benjamini-Hochberg (BH) method, with significant results defined as $P < 0.05$.

4.5. Strengths and limitations

This study has several strengths. First, it is the first to investigate the impact of $PM_{2.5}$ constituents on sleep disorders in children and adolescents. Unlike most research focusing solely on $PM_{2.5}$ mass concentrations, this study provides a comprehensive assessment of not only the individual effects of $PM_{2.5}$ and its six constituents on sleep disorders but also their combined impact. Moreover, the study was conducted in a region where air pollution levels have declined to moderate ranges, allowing for a nuanced evaluation of $PM_{2.5}$ effects at relatively lower concentrations—an aspect that has been largely overlooked in previous studies. Lastly, the large sample size enhances the study's statistical power, ensuring robust and reliable findings that contribute meaningful insights to the field.

However, this study has certain limitations. First, the cross-sectional design limits the ability to infer causal relationships from the observed associations. However, as a large cross-sectional study, the evidence is relatively strong. Moreover, exposure levels from the five years prior to the questionnaire were used, which somewhat reduces the potential for reverse causality. Future cohort studies are needed to further explore causality. Second, pediatric sleep disorders were assessed using the parent-reported SDSC scale, which may introduce recall bias. Briefly, parents may overestimate observable disorders and underestimate nocturnal issues. Nevertheless, validated Chinese version (C-SDSC) demonstrates good reliability (Cronbach's α = 0.81) and is widely used in clinical and non-clinical research [26]. Third, despite adjusting for major confounders, residual confounding from unmeasured variables such as depression, anxiety, peer pressure, and other psychological factors cannot be ruled out. Future research should incorporate mental health assessments to better understand their role in the association between air pollution and sleep disorders. Additionally, the exposure assessment was based on residential addresses, which may overlook variations due to outdoor activities and commuting. However, a sensitivity analysis using school-based $PM_{2.5}$ exposure estimates (Table S18) showed consistent results. Given that most children and adolescents in China spend the majority of their time near their homes and schools, this

suggests that the impact of exposure misclassification is minimal. Lastly, it is acknowledged that in densely populated urban areas, the CHAP database may not fully capture local pollution variations due to its spatial resolution limitations. However, it remains one of the most comprehensive and validated sources for air pollution exposure assessment in China [30,33]. Future studies could incorporate higher-resolution models or personal exposure measurements to further refine exposure estimates.

5. Conclusions

This study is the first to comprehensively investigate the associations between $PM_{2.5}$, its major constituents, and sleep disorders in children and adolescents. Our findings reveal that prolonged exposure to $PM_{2.5}$ and its constituents is significantly associated with an increased risk of sleep disorders. Notably, combustion-related constituents, particularly BC and OM, emerged as key contributors to these associations, highlighting their potential role in sleep disorders. Outdoor activity may serve as a potential effect modifier in the relationship between $PM_{2.5}$ constituents and sleep disorders. Future research should further explore the biological mechanisms through which BC and OM contribute to sleep disorders. Current air quality standards may not be sufficient to fully protect children's sleep health. More stringent air pollution regulations and targeted control measures for specific $PM_{2.5}$ constituents, particularly combustion-derived pollutants, are needed. Strengthening monitoring efforts and implementing policies to reduce children and adolescents' exposure to harmful air pollutants could have important implications for safeguarding sleep health and overall well-being.

Environmental implication

Ambient particulate matter pollution is a major global public health issue. Emerging evidence suggests a significant association between $PM_{2.5}$ and sleep disorders, but little is known about the effects of its constituents on health. This study is the first to explore the relationship between $PM_{2.5}$ and its major constituents and sleep disorders in children and adolescents in China. This research provides important implication for the prevention of environmental particulate matter pollution and offers guidance for reducing air pollution exposure to improve public health.

CRediT authorship contribution statement

Zhao Bin: Writing – review & editing. **Wei Jing:** Resources, Methodology. **Dong Guang-Hui:** Writing – review & editing, Supervision, Conceptualization. **Jalava Pasi:** Writing – review & editing. **Oudin Anna:** Writing – review & editing. **Lv Yuan:** Writing – review & editing. **Dai Jian:** Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Conceptualization. **Zeng Qing-Guo:** Writing – original draft, Visualization, Methodology, Formal analysis, Data curation, Conceptualization. **Zeng Xiao-Wen:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Funding acquisition, Conceptualization. **Jalaludin Bin:** Writing – review & editing, Supervision, Methodology.

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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.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jhazmat.2025.138254.

Data availability

Data will be made available on request.

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