BMC Public Health

RESEARCH

Open Access

Effect modifications of parents' age at childbirth on association between ambient particulate matter and children obesity

Xianzhi Li[1,2,3†], Bin Yu[4†], Yajie Li[5†], Haorong Meng[6], Zonglei Zhou[7], Shunjin Liu[1,2,3], Yunyun Tian[2,3], Xiangyi Xing[1,3,8*], Yingchao Lei[9*] and Li Yin[1,2,3*]

Abstract

Background There is limited evidence regarding the modifying effects of parents' age at childbirth on the relationship between air pollution and obesity in plateau areas. This study aimed to explore the association between particulate matter (PM) and child obesity, specifically investigating whether parents' age at childbirth could modify this relationship in the Tibetan plateau, China.

Methods Satellite-based random forest models were used to estimate the concentrations of $PM_{2.5}$ (particulate matter with aerodynamic diameters ≤ 2.5 μm), PM_c (particulate matter with aerodynamic diameters between 2.5 μm and 10 μm), and PM_{10} (particulate matter with aerodynamic diameters ≤ 10 μm). Linear and logistic regression models were employed to assess associations between PM exposure and obesity indicators, and effect estimates of PM across different particle sizes were compared.

Results The study comprised 2,015 children under five years old. Postnatal exposure to PM was positively associated with overweight and obesity (OWO), waist-to-hip ratio (WHR) and body mass index (BMI). Among these pollutants, PM_{10} exhibited the strongest association with BMI and OWO, whereas PM_c showed the strongest association with WHR. An interquartile range (IQR) increase in $PM_{2.5}$ (5.67 μg/m^3), PM_c (5.25 μg/m^3), and PM_{10} (11.06 μg/m^3) was positively associated with OWO (odd ratio [OR] for $PM_{2.5}$ = 1.52, 95% confidence interval [CI] for $PM_{2.5}$ = 1.24 to 1.85; OR for PM_c = 1.50, 95% CI for PM_c = 1.19 to 1.88; OR for PM_{10} = 1.56, 95% CI for PM_{10} = 1.25 to 1.96), respectively. Stratified analysis by parents' age at childbirth indicated that the effects of PM on obesity indicators were more pronounced in the advanced age group.

†Xianzhi Li, Bin Yu and Yajie Li contributed equally to this work.

*Correspondence:
Xiangyi Xing
xianzhi_scu@163.com
Yingchao Lei
752875498@qq.com
Li Yin
425281415@qq.com

Full list of author information is available at the end of the article

Conclusions Long-term exposure to PM was positively associated with OWO, WHR, and BMI. Our findings also underscore the importance of examining the effects of ambient PM exposure on OWO, particularly in parents of advanced age at childbirth.

Keywords Particulate matter, Child obesity, Maternal age, Paternal age, Tibet

Background

In recent decades, there has been a notable rise in overweight and obesity (OWO) among children globally, posing a substantial health burden [1]. The World Health Organization (WHO) reports a nearly threefold increase in obesity prevalence since 1975, with an estimated 38.9 million children under five being overweight or obese in 2020 [2]. According to the China Chronic Disease and Nutrition Surveillance survey (2015–2019), the prevalence of OWO among children under six was 6.8% and 3.6%, respectively [3]. Despite lower overall prevalence compared to adults, preschool children exhibit a significantly accelerated rate of OWO development [4]. Obesity not only constitutes a noncommunicable disease but also serves as a modifiable risk factor for various other conditions, such as hypertension, fatty liver disease, and myocardial infarction [5]. Furthermore, childhood obesity often persists into adulthood and is linked to increased risks of type 2 diabetes, cardiovascular diseases, cancer, and mental health disorders [6].

Multiple factors can influence childhood OWO, including genetic, metabolic, dietary, behavioral and environmental factors [7, 8]. Among these, behavioral and environmental factors are prominent contributors to obesity occurrence [5]. Recently, there has been an increasing focus on the impact of air pollution and its potential health implications, particularly its association with obesity. Several studies have investigated the link between air pollution and OWO [7, 9, 10]. However, significant gaps persist in our understanding of the relationship between air pollution and childhood OWO. Firstly, most epidemiological studies have primarily focused on school-aged children, adolescents and adults, with limited evidence available for preschool-aged children. Infants and young children are particularly sensitive to air pollution due to ongoing organ development and higher air intake per body weight, increasing their vulnerability to air pollutants [11]. Secondly, current evidence regarding the association between air pollution and OWO in children remains ambiguous. While many studies have reported an increased risk of childhood obesity with higher levels of ambient air pollution exposure [4, 7], others have found null or negative associations [12]. For instance, research in Catalonia suggests that early-life exposure to air pollution may slightly elevate the risk of childhood OWO [13], whereas studies in Hong Kong indicate a potentially protective effect of ambient air pollution exposure against childhood obesity [14]. Thirdly, the health impacts of air pollutants are influenced by their composition. Particulate matter (PM), a major air pollutant, varies in particle size and chemical composition across different regions and countries [15]. Most previous studies have focused solely on participants in plain regions, limiting our understanding of the broader impacts of ambient air pollution on childhood OWO in high-altitude areas.

The Qinghai-Tibet Plateau (QTP), often referred to as “the Third Pole” and “the Roof of the World”, is characterized by unique geographical features, including numerous areas exceeding 4,000 m in elevation. This region experiences significantly lower temperatures compared to its surroundings, with annual average temperatures below 0℃. Moreover, due to its very low population density and minimal human activities, the QTP is widely considered one of the cleanest areas globally [16]. However, despite minimal local anthropogenic emissions, the ambient air quality in the QTP is strongly influenced by exogenous atmospheric pollution transported from surrounding regions, particularly under the influence of monsoons and westerlies [17]. Consequently, air pollution levels over the QTP exhibit pronounced seasonal variations, typically peaking in spring and decreasing in summer [16]. Researchers have highlighted that the health impacts of air pollutants are closely linked to ambient meteorological conditions such as temperature, altitude, and human activities. Interestingly, even at low concentrations, these pollutants may pose significant health risks [18]. Given the distinctive environmental and geographical characteristics of the QTP, it is imperative to investigate the relationship between air pollution and childhood obesity in this region and develop targeted policies to safeguard children’s health.

The trend of women delaying pregnancy has become increasingly pronounced in recent years [19]. Research indicates that advancing maternal age is associated with adverse perinatal outcomes and childhood health issues, including stillbirth, miscarriage, fetal growth restriction (FGR), preterm birth, and malnutrition [20]. However, the relationship between maternal and paternal age and the risk of OWO in offspring remains debatable. A study conducted in the United States suggested that there is a 14% increase in the risk of obesity in girls for every five-year increase in maternal age at childbirth [21]. In contrast, other studies have reported either a negative correlation or no significant association between maternal age and offspring OWO risk [22–24]. Furthermore, there is limited evidence regarding the impact of paternal age on the development of OWO in children. Eriksen et al. [25] observed a cohort of 346,609 children and found an increased risk of OWO in offspring when the father's age at childbirth exceeded 50 years. Notably, no studies have investigated whether parents' age at childbirth modifies the association between air pollution and obesity among preschoolers, particularly in high-altitude areas.

To address these gaps, our study aimed to investigate the association between ambient PM — specifically $PM_{2.5}$ (particles ≤ 2.5 μm in diameter), PM_{10} (particles ≤ 10 μm), and PM_c (particles between 2.5 and 10 μm) — and various obesity indicators among preschool children in Tibet, Southwest China. These indicators include Z-scores of weight-for-height (WFH), waist-to-hip ratio (WHR), BMI, Z-scores of BMI, and the prevalence of OWO. Additionally, we explored whether parents' age at childbirth could modify the association between PM exposure and obesity indicators. Finally, we compared the effect estimates of PM across different particle sizes.

Methods

Study population

The detailed information regarding the study design and participant selection has been previously described [26]. Briefly, a three-stage, stratified cluster sampling approach was employed from July to October 2020. Initially, eight areas (counties) in Tibet were selected based on a proportional representation of altitude and population size. Subsequently, five towns or subdistricts from each county were chosen as primary sampling units (PSUs). Within each of the 40 PSUs, four villages or communities were randomly selected. The study included children aged 0–60 months and their parents residing in the selected villages or communities, who were interviewed using structured questionnaires. Ultimately, 2,559 children and their parents participated in the interviews.

The exclusion criteria for this study were as follows: (1) non-Tibetan children; (2) children with missing information on critical variables; and (3) exclusion of children under 12 months of age to better control for the effects of dietary factors and feeding practices based on previous literature [27–29]. A total of 2,015 children were included in the final analysis, resulting in an enrollment rate of 78.7% (Figure S1).

Data collection

Data collection involved face-to-face interviews with guardians using an electronic questionnaire and included medical examinations. The electronic questionnaire encompassed children's demographic characteristics (age, sex, low birth weight, drinking water source, annual household income), medical history (asthma, anemia, recent illness within the past two weeks, dental history), feeding practices (early initiation of breastfeeding within one hour of birth, exclusive breastfeeding until 6 months, continued breastfeeding at 1 year, introduction of complementary foods between 6 and 8 months, dietary diversity, meal frequency, consumption of iron-rich or iron-fortified foods), exposure to secondary smoking, parents' demographics (maternal education level, maternal height, maternal weight, maternal history of anemia during pregnancy, parents' age at childbirth), and residential address. The Global Positioning System (GPS) was utilized to record the geographical coordinates (latitude and longitude) of children's residential addresses.

Earlier publications have provided detailed definitions and measurements of covariates [26]. In these studies, birth weight less than 2500 g was categorized as low birth weight. Additionally, wealth status was categorized into five groups based on yearly family income: poorest, poorer, middle, richer, and richest. Water sources were classified as either improved (such as piped water and protected wells) or unimproved (including springs, lakes, ponds, unprotected wells, rivers, and dams) [30]. Children were assessed for a history of asthma, anemia, or dental caries based on diagnosed conditions. Optimal feeding practices were evaluated using structured questionnaires according to the *WHO infant and young children feeding practice guidelines* [31]. Minimum dietary diversity was achieved if a child consumed 4 or more of the seven food groups (including grains, roots and tubers, legumes and nuts, dairy products, flesh foods, eggs, vitamin-A-rich fruits and vegetables, and other fruits and vegetables), the previous day. Adequate meal frequency was defined as consuming meals more than 3 times a day. Additional data collected included (i) whether the child was breastfed within 1 h of birth, (ii) whether the child was fed exclusively with breast milk until 6 months of age, (iii) whether the child was given complementary foods between 6 and 8 months of age, (iv) whether the child was consistently breastfed until 12 months of age, and (v) whether the child was fed an iron-rich food or iron-fortified food on the previous day. Each item was scored as 0 or 1, and a composite optimal feeding practice score was derived by summing these scores. Scores were then categorized as low (< median feeding practice scores) or high (≥ median feeding practice scores) based on the median value. Further details on these scoring methods can be found in our previously published paper [26].

The anthropometric measurements of obesity included children's waist circumference (WC), hip circumference, height, and weight. Height measurements were taken with shoes off, in a recumbent position for children younger than 2 years and standing for older children. Weight was measured with bare feet and light clothing using a weight measurement device. Waist circumference was measured above the navel, and hip circumference at the widest part of the hip, both with light clothing. Each measurement was performed three times and then averaged to ensure accuracy and reliability.

Outcome assessment

OWO was the primary outcome in this study, and secondary outcomes comprised Z-scores of WFH, WHR, BMI and Z-scores of BMI. According to the WHO *Growth Standard* [32], Z-scores were calculated by dividing the difference between the observed value and the mean value of the reference population by its standard deviation (SD). WHR was determined by dividing WC (in cm) by hip circumference (HC in cm), and BMI was calculated by dividing weight (kg) by height squared (m^2). Childhood OWO was defined as WFH greater than 2 SDs above the median of the WHO Child Growth Standards (Z-scores of WFH > 2) [32, 33].

Air pollution exposure assessment

PM data for the study period were obtained from the ChinaHighAirPollutants (CHAP) dataset (https://weijing-rs.github.io/product.html). Wei et al. [34–36] utilized monitoring data, satellite remote sensing, temperature, humidity, land use information, and other spatial and temporal predictors to predict daily concentrations of $PM_{2.5}$ and PM_{10} at a spatial resolution of 1 km × 1 km using a space-time extremely randomized trees model. The model demonstrated high predictive ability, with 10-fold cross-validation R^2 (root mean square error) values of 0.92 (10.76 μg/m^3) for $PM_{2.5}$ and 0.90 (21.12 μg/m^3) for PM_{10}. Individual exposure estimates of $PM_{2.5}$ and PM_{10} were derived by geocoding each participant's residential address using GPS coordinates. Subsequently, one-year averaged concentrations of $PM_{2.5}$ and PM_{10} at those coordinates in the year prior to the survey date were computed for each participant as proxies for their exposure. PM_c concentrations were obtained by calculating the difference between $PM_{2.5}$ and PM_{10} concentrations [27, 37].

Parents' age at childbirth assessment

Mothers of the children were categorized into two groups based on maternal age at childbirth: < 35 years old (younger maternal age) and ≥35 years old (advanced maternal age) [38, 39]. Similarly, fathers were categorized based on paternal age at childbirth into < 40 years old (younger paternal age) and ≥40 years old (advanced paternal age) [40, 41].

Statistical analysis

Multivariable logistic regression models were utilized to investigate the association between long-term exposure to PM and OWO, with odds ratios (OR) and 95% confidence intervals (CI) reported. Multivariable linear regression models were used to assess the effect of long-term PM exposure on Z-scores of WFH, WHR, BMI and Z-scores of BMI as continuous measures, with effect estimates presented as beta (β) (coefficient of exposure variables based on multiple linear regression models) and 95% CIs. Additionally, natural cubic spline analysis was applied to explore potential non-linear relationships between ambient PM concentrations and obesity indicators. Based on the previous literature, the adjusted models included the following covariates: age (< 36 vs. ≥36), sex (female vs. male), low birth weight (categorized as: yes, no and not sure), asthma history (categorized as: yes, no and not sure), anemia history (categorized as: yes, no and not sure), history of dental caries (categorized as: yes, no and not sure), recent illness in the last two weeks (yes vs. no), optimal feeding scores (high scores vs. low scores), secondary smoke exposure (yes vs. no), residence (urban vs. rural), maternal education level (categorized as: illiteracy, primary school, and junior high school and above), maternal height (categorized as: <160.0, 160.0-169.9, and ≥170.0), maternal weight (categorized as: <50.0, 50.0-59.9, and ≥60.0), maternal anemia during pregnancy (categorized as: yes, no and not sure), parents' age at childbirth (childbearing age vs. advanced age), wealth category (categorized as: poorest, poorer, middle, richer, and richest), drinking water source (improved vs. unimproved), relative humidity (continuous variable), mean temperature (continuous variable), and altitude (continuous variable). All estimations were reported per 10 μg/m^3 increase in PM concentrations and per interquartile range (IQR) increase of PM (μg/m^3). Reporting estimations per 10 μg/m^3 increase allows for comparison of our results with those of other studies, while reporting per IQR μg/m^3 increase enables comparison of the effects of PM across different particle sizes.

We conducted subgroup analyses stratified by children's age (< 36 months vs. ≥36 months), sex (male vs. female), wealth status (low income vs. high income), optimal feeding scores (high scores vs. low scores), drinking water source (improved vs. unimproved), maternal age at childbirth (childbearing age vs. advanced age), and paternal age at childbirth (childbearing age vs. advanced age). *P value for difference* was calculated by Z-test to examine the differences between subgroups. We further calculated *P value for interaction* to explored interaction between parent's age at childbirth (binary variable) and PM by adding interaction term into multivariable regression models.

Sensitivity analyses were performed as follows: (1) We assessed the robustness of our findings by using average PM concentrations over 3, 6, and 9 months before the survey to evaluate long-term effects. (2) We examined the association between PM exposure and OWO after excluding participants with a history of asthma, anemia, and dental caries. (3) We reanalyzed the relationship between PM exposure and OWO, considering modifications by parents' age, using BMI Z-scores recalculated according to the Center for Disease Control (USA) reference population [42]. This analysis was restricted to children older than 23 months, given the age limitations of the reference data.

All statistical analyses were conducted using R 4.1.0. Statistical significance was defined as $P<0.05$. Multivariable logistic regression models, multivariable linear regression models, and natural cubic spline analyses were performed using the stats and *rms* packages in R, respectively.

Results

Demographic characteristics

Table 1 describes the characteristics of 2,015 children aged 0–60 months in this study. Among them, there were 1,814 non-OWO children and 201 OWO children, respectively. Compared to non-OWO children, those with OWO were more likely to be female, younger, suffer from anemia, breastfed within 1 h after delivery, given complementary foods between 6 and 8 months of age, and consistently breastfed until 12 months. Mothers of OWO children were found to have lower education levels, shorter height, and were more likely to be of advanced childbearing age compared to mothers of non-OWO children. Fathers of OWO children also tended to be of advanced childbearing age. Non-OWO children were more likely to suffer from dental caries, have higher dietary diversity, and have access to cleaner drinking water sources.

Table 2 presents the one-year average concentrations of $PM_{2.5}$, PM_c and PM_{10} at the locations where the children resided. The IQRs for $PM_{2.5}$, PM_c, and PM_{10} were 5.67 μg/m^3, 5.25 μg/m^3, and 11.06 μg/m^3, respectively. The concentrations of ambient PM exposure were higher in OWO children compared to non-OWO children ($P<0.05$).

Associations between ambient PM exposure and obesity indicators

Table 3 presents the effect estimates and 95% CI for the association between obesity indicators and a 10 μg/m^3 increase in average one-year $PM_{2.5}$, PM_c and PM_{10} exposure according to adjusted models. Significant statistical changes were observed in WHR, BMI and OWO per 10 μg/m^3 increments in $PM_{2.5}$, PM_c and PM_{10} concentrations. For instance, each 10 μg/m^3 increase in $PM_{2.5}$ was associated with an increase in WHR of 0.06 (95% CI, 0.04 to 0.08), BMI of 0.51 kg/m^2 (95% CI, 0.14 to 0.87), and an OR for OWO of 2.08 (95% CI, 1.46 to 2.97).

Table 4 shows the effect estimates and 95% CI for the association between obesity indicators and a per IQR μg/m^3 increase in average one-year $PM_{2.5}$, PM_c and PM_{10} exposure according to adjusted models. Generally, PM_{10} showed the largest absolute changes in BMI (β=0.38; 95% CI, 0.13 to 0.63) and the largest OR of OWO (OR=1.56; 95% CI, 1.25 to 1.96), while PM_c exhibited the largest absolute changes in WHR (β=0.08; 95% CI, 0.07 to 0.10). The effect estimates of $PM_{2.5}$ on WHR (β=0.03; 95% CI, 0.02 to 0.04) and BMI (β=0.29; 95% CI, 0.08 to 0.50) were the smallest among the three air pollutants. Similarly, the effect estimates of PM_c on OWO (OR=1.50; 95% CI, 1.19 to 1.88) were the smallest among the three air pollutants.

We observed non-linear relationships between long-term PM exposure and obesity indicators (Z-scores of WFH, WHR, BMI, Z-scores of BMI, and OWO), as illustrated in Figure S2-S6.

Stratified analyses

The stratified analyses by parents' age at childbirth for $PM_{2.5}$, PM_c and PM_{10} exposure are presented in Tables 5 and 6, respectively. Overall, a stronger effect of ambient PM was observed in the advanced age group. For Z-scores of WFH, WHR, BMI, Z-scores of BMI, and OWO, the associations were more pronounced in the advanced maternal age group compared to the childbearing maternal group, with significant differences in effect estimates between the two groups. Similarly, the stratified analyses by paternal age at childbirth showed comparable results. For instance, the association between OWO and a 10 μg/m^3 increase in $PM_{2.5}$ was significantly higher among the advanced paternal age group (OR=3.52; 95% CI, 2.92 to 4.24) than the childbearing paternal group (OR=1.74; 95% CI, 1.15 to 2.62).

Tables S1 to S5 (supplementary material) present the results of stratification analyses excluding parents' age at childbirth. Greater effects of $PM_{2.5}$ on Z-scores of WFH, WHR, BMI and Z-scores of BMI were observed in children from households with high income and high feeding scores. Similar effects were also observed for PM_c and PM_{10}. However, for OWO, the differences in effect estimates between different groups were not statistically significant.

Interaction between parent's age at childbirth and PM

Table 7 presents the results of interaction analyses between PM exposure and parent's age at childbirth on obesity indicators. Significant interactions were observed between PM exposure and maternal age group for all obesity indicators. For instance, the interaction effect values between $PM_{2.5}$ and maternal age group were 0.04 (95% CI, 0.01 to 0.07) for WHR, 1.61 (95% CI, 0.91 to 2.31) for BMI, 1.43 (95% CI, 1.01 to 1.85) for Z-scores of

Table 1 Basic characteristics of study participants

Variables	Total	Non-OWO	OWO	*P* value
Sample sizes	***N*** **= 2,015**	***N*** **= 1,814**	***N*** **= 201**	
Sex, ***n*** (%)				
Female	1,057 (52.5%)	927 (51.1%)	130 (64.7%)	< 0.001
Male	958 (47.5%)	887 (48.9%)	71 (35.3%)	
Age, **months**, **n (%)**				
<36	963 (47.79%)	850 (46.9%)	113 (56.2%)	0.014
≥ 36	1,052 (52.21%)	964 (53.1%)	88 (43.8%)	
Low birth weight, **n (%)**				
Yes	123 (6.1%)	111 (6.1%)	12 (6.0%)	0.860
No	1,701 (84.4%)	1,533 (84.5%)	168 (83.6%)	
Not sure	191 (9.5%)	170 (9.4%)	21 (10.4%)	
Asthma history, **n (%)**				
Yes	26 (1.3%)	24 (1.3%)	2 (1.0%)	0.110
No	1,835 (91.1%)	1,659 (91.5%)	176 (87.6%)	
Not sure	154 (7.6%)	131 (7.2%)	23 (11.4%)	
Anemia history, **n (%)**				
Yes	39 (1.9%)	35 (1.9%)	4 (2.0%)	0.020
No	1,826 (90.6%)	1,654 (91.2%)	172 (85.6%)	
Not sure	150 (7.4%)	125 (6.9%)	25 (12.4%)	
Suffering from dental caries, **n (%)**				
Yes	96 (4.8%)	89 (4.9%)	7 (3.5%)	0.010
No	1,806 (89.6%)	1,633 (90.0%)	173 (86.1%)	
Not sure	113 (5.6%)	92 (5.1%)	21 (10.4%)	
Being ill for the last two weeks, **n (%)**				
Yes	229 (11.4%)	202 (11.1%)	27 (13.4%)	0.420
No	1,786 (88.6%)	1,612 (88.9%)	174 (86.6%)	
Child breastfed within 1 h after delivery, **n (%)**				
Yes	184 (9.1%)	162 (8.9%)	22 (10.9%)	0.008
No	732 (36.3%)	642 (35.4%)	90 (44.8%)	
Not sure	1,099 (54.5%)	1,010 (55.7%)	89 (44.3%)	
Exclusive breast feeding until 6 months, **n (%)**				
Yes	810 (40.2%)	728 (40.1%)	82 (40.8%)	0.220
No	1,180 (58.6%)	1,066 (58.8%)	114 (56.7%)	
Not sure	25 (1.2%)	20 (1.1%)	5 (2.5%)	
Child given complementary foods between 6 and 8 months of age, **n (%)**				
Yes	564 (28.0%)	506 (27.9%)	58 (28.9%)	0.021
No	1,395 (69.2%)	1,264 (69.7%)	131 (65.2%)	
Not sure	56 (2.8%)	44 (2.4%)	12 (6.0%)	
Child consistently breastfed until 12 months, **n (%)**				
Yes	1,140 (56.6%)	1,009 (55.6%)	131 (65.2%)	0.011
No	875 (43.4%)	805 (44.4%)	70 (34.8%)	
Dietary diversity, **n (%)**				
< 4	427 (21.2%)	368 (20.3%)	59 (29.4%)	0.005
≥ 4	1,588 (78.8%)	1,446 (79.7%)	142 (70.6%)	
Fe-rich or Fe-fortified food, **n (%)**				
Yes	106 (5.3%)	97 (5.3%)	9 (4.5%)	0.740
No	1,909 (94.7%)	1,717 (94.7%)	192 (95.5%)	
Meal frequency, **times**, **n (%)**				
< 3	1,965 (97.5%)	1,770 (97.6%)	195 (97.0%)	0.630
≥ 3	50 (2.5%)	44 (2.4%)	6 (3.0%)	
Secondary smoke, **n (%)**				
Yes	506 (25.1%)	462 (25.5%)	44 (21.9%)	0.300
No	1,509 (74.9%)	1,352 (74.5%)	157 (78.1%)	

Table 1 (continued)

Variables	Total	Non-OWO	OWO	*P* value
Maternal education level, n (%)				
Illiteracy	1,035 (51.4%)	907 (50.0%)	128 (63.7%)	< 0.001
Primary school	344 (17.1%)	309 (17.0%)	35 (17.4%)	
Junior high school and above	636 (31.6%)	598(33.0%)	38(18.9%)	
Maternal height, cm, n (%)				
< 160.0	586 (29.1%)	512 (28.2%)	74 (36.8%)	0.040
160.0 ~ 169.9	1,329 (66.0%)	1,212 (66.8%)	117 (58.2%)	
≥ 170.0	100 (5.0%)	90 (5.0%)	10 (5.0%)	
Maternal weight, kg, n (%)				
< 50.0	407 (20.2%)	365 (20.1%)	42 (20.9%)	0.920
50.0 ~ 59.9	1,054 (52.3%)	948 (52.3%)	106 (52.7%)	
≥ 60.0	554 (27.5%)	501 (27.6%)	53 (26.4%)	
Mother suffering from anemia during pregnancy, n (%)				
Yes	191 (9.5%)	171 (9.4%)	20 (10.0%)	0.630
No	1,532 (76.0%)	1,384 (76.3%)	148 (73.6%)	
Not sure	292 (14.5%)	259 (14.3%)	33 (16.4%)	
Wealth status				
Poorest	360 (17.9%)	324 (17.9%)	36 (17.8%)	0.270
Poorer	861 (42.7%)	63 (42.1%)	98 (48.8%)	
Middle	456 (22.6%)	415 (22.8%)	41 (20.4%)	
Richer	137 (6.8%)	124 (6.8%)	13 (6.5%)	
Richest	201 (10.0%)	188 (10.4%)	13 (6.5%)	
Drinking water source				
Improved	1,455 (72.2%)	1,335 (73.6%)	120 (59.7%)	< 0.001
Unimproved	560 (27.8%)	479 (26.4%)	81 (40.3%)	
Maternal age				
Childbearing age	1,785 (88.6%)	1,651 (91.0%)	134 (66.7%)	< 0.001
Advanced age	230 (11.4%)	163 (9.0%)	67 (33.3%)	
Paternal age				
Childbearing age	1,861 (92.4%)	1,713 (94.4%)	148 (73.6%)	< 0.001
Advanced age	154 (7.6%)	101 (5.6%)	53 (26.4%)	

Notes: OWO, overweight and obesity

Table 2 Descriptive one-year average concentrations of PM by residence

Variables	Total ($n = 2,015$)	OWO[a] ($n = 201$)	Non-OWO ($n = 1,814$)	*P* value
	Median (25%, 75%)	Median (25%, 75%)	Median (25%, 75%)	
$PM_{2.5}$ (μg/m^3)	12.68 (11.19, 16.86)	16.10 (11.40, 17.60)	12.70 (11.20, 16.80)	< 0.001
PM_c (μg/m^3)	15.86 (13.70, 18.95)	16.50 (14.20, 19.50)	15.90 (13.70, 18.90)	0.001
PM_{10} (μg/m^3)	28.43 (25.01, 36.07)	33.90 (25.40, 36.70)	27.50 (25.00, 35.50)	< 0.001

Notes: PM, particulate matter; $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 μm; PM_c, particulate matter with an aerodynamic diameter of 2.5 to 10 μm, PM_{10}, particulate matter with an aerodynamic diameter of 10 μm; OWO, overweight and obesity

[a]OWO: Z-scores of weight-for-height ≥ 2

Table 3 Associations of obesity indicators with per 10 μg/m^3 increase of ambient air pollution

-	$PM_{2.5}$	PM_c	PM_{10}
Main analysis[a]	**β(95%CI)**	**β(95%CI)**	**β(95%CI)**
Z-scores of WFH	-0.02 (-0.39, 0.34)	-0.16 (-0.61, 0.30)	-0.05 (-0.27, 0.18)
WHR	0.06 (0.04, 0.08)	0.16 (0.14, 0.18)	0.06 (0.05, 0.07)
BMI	0.51 (0.14, 0.87)	0.59 (0.14, 1.05)	0.34 (0.11, 0.57)
Z-scores of BMI[b]	0.10 (-0.12, 0.32)	0.15 (-0.12, 0.43)	0.08 (-0.06, 0.21)
-	**OR(95%CI)**	**OR(95%CI)**	**OR(95%CI)**
OWO[c]	2.08 (1.46, 2.97)	2.15 (1.40, 3.32)	1.50 (1.22, 1.83)

Notes: $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 μm; PM_c, particulate matter with an aerodynamic diameter of 2.5 to 10 μm; PM_{10}, particulate matter with an aerodynamic diameter of 10 μm; WFH, weight-for-height; WHR, waist-to-hip ratio; BMI, body mass index; OWO, overweight and obesity

[a]Main analysis: adjusted for age, sex, low birth weight, asthma history, anemia history, history of dental caries, being ill for the last two weeks, optimal feeding scores, secondary smoke, residence, maternal education level, maternal height, maternal weight, mother suffering from anemia during pregnancy, wealth category, drinking water source, relative humidity, mean temperature, altitude, maternal age and paternal age

[b]Z-scores of BMI: calculated according to WHO reference population

[c]OWO: Z-scores of WFH ≥ 2

Table 4 Associations of obesity indicators with per IQR μg/m^3 increase of ambient air pollution

	$PM_{2.5}$ (IQR = 5.67 μg/m^3)	PM_c (IQR = 5.25 μg/m^3)	PM_{10} (IQR = 11.06 μg/m^3)
Main analysis[a]	**β(95%CI)**	**β(95%CI)**	**β(95%CI)**
Z-scores of WFH	-0.01 (-0.22, 0.19)	-0.08 (-0.32, 0.16)	-0.05 (-0.30, 0.20)
WHR	0.03 (0.02, 0.04)	0.08 (0.07, 0.10)	0.07 (0.06, 0.08)
BMI	0.29 (0.08, 0.50)	0.31 (0.07, 0.55)	0.38 (0.13, 0.63)
Z-scores of BMI[b]	0.06 (-0.07, 0.18)	0.08 (-0.06, 0.23)	0.08 (-0.07, 0.24)
	OR(95%CI)	**OR(95%CI)**	**OR(95%CI)**
OWO[c]	1.52 (1.24, 1.85)	1.50 (1.19, 1.88)	1.56 (1.25, 1.96)

Notes: $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 μm; PM_c, particulate matter with an aerodynamic diameter of 2.5 to 10 μm; PM_{10}, particulate matter with an aerodynamic diameter of 10 μm; WFH, weight-for-height; WHR, waist-to-hip ratio; BMI, body mass index; OWO, overweight and obesity

[a]Main analysis: adjusted for age, sex, low birth weight, asthma history, anemia history, history of dental caries, being ill for the last two weeks, optimal feeding scores, secondary smoke, residence, maternal education level, maternal height, maternal weight, mother suffering from anemia during pregnancy, wealth category, drinking water source, relative humidity, mean temperature, altitude, maternal age and paternal age

[b]Z-scores of BMI: calculated according to WHO reference population.

[c]OWO: Z-scores of WFH ≥ 2

Table 5 Associations between PM exposure and obesity indicators stratified by maternal age at childbirth

-	$PM_{2.5}$			PM_c			PM_{10}		
-	Childbearing age	Advanced age	P for difference	Childbearing age	Advanced age	P for difference	Childbearing age	Advanced age	P for difference
Main analysis[a]	**β(95%CI)**		-	**β(95%CI)**		-	**β(95%CI)**		-
Z-scores of WFH	-0.35 (-0.58, -0.12)	-0.08 (-0.66, 0.50)	< 0.001	-0.35 (-0.60, -0.11)	-0.21 (-0.83, 0.41)	< 0.001	-0.19 (-0.31, -0.07)	-0.08 (-0.39, 0.23)	< 0.001
WHR	0.00 (-0.04, 0.03)	0.04 (0.03, 0.05)	< 0.001	0.00 (-0.03, 0.03)	0.08 (0.07, 0.09)	< 0.001	0.00 (-0.02, 0.02)	0.03 (0.03, 0.04)	< 0.001
BMI	0.13 (-0.09, 0.36)	1.83 (1.18, 2.49)	< 0.001	0.38 (0.13, 0.63)	1.70 (0.98, 2.42)	< 0.001	0.13 (0.01, 0.26)	0.96 (0.60, 1.31)	< 0.001
Z-scores of BMI[b]	-0.04 (-0.17, 0.10)	1.41 (1.03, 1.78)	< 0.001	0.02 (-0.13, 0.17)	1.62 (1.23, 2.01)	< 0.001	0.00 (-0.08, 0.07)	0.81 (0.62, 1.01)	< 0.001
	OR(95%CI)		-	**OR(95%CI)**		-	**OR(95%CI)**		-
OWO[c]	1.63 (1.06, 2.51)	3.39 (2.23, 5.15)	0.002	1.42 (0.86, 2.34)	4.42 (4.00, 4.88)	< 0.001	1.26 (0.99, 1.60)	3.22 (2.00, 5.17)	0.001

Notes: PM, particulate matter; $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 μm; PM_c, particulate matter with an aerodynamic diameter of 2.5 to 10 μm, PM_{10}, particulate matter with an aerodynamic diameter of 10 μm; WFH, weight-for-height; WHR, waist-to-hip ratio; BMI, body mass index; OWO, overweight and obesity

[a]Main analysis: adjusted for age, sex, low birth weight, asthma history, anemia history, history of dental caries, being ill for the last two weeks, optimal feeding scores, secondary smoke, residence, maternal education level, maternal height, maternal weight, mother suffering from anemia during pregnancy, wealth category, drinking water source, relative humidity, mean temperature, altitude, and paternal age

[b]Z-scores of BMI: calculated according to WHO reference population

[c]OWO: Z-scores of WFH ≥ 2

BMI, and 2.65 (95% CI, 1.78 to 3.95) for OWO. Similar findings were observed for the paternal age group.

Sensitivity analyses

Tables S6 to S7 show comparable effect estimates for obesity indicators using average ambient PM concentrations from different exposure periods. For example, a 10 μg/m^3 increase in $PM_{2.5}$ over a nine-month average concentration was associated with the following changes: a β of 0.02 (95% CI, -0.40 to 0.45) for Z-scores of WFH, a β of 0.07 (95% CI, 0.05 to 0.09) for WHR, a β of 0.66 (95% CI, 0.23 to 1.08) for BMI, a β of 0.15 (95% CI, -0.11 to 0.41) for Z-scores of BMI, and an OR of 2.44 (95% CI, 1.60 to 3.73) for OWO. These findings underscore the robustness of our results across varying exposure durations. Tables S8 to S9 demonstrate that our results on the association between PM exposure and obesity indicators remained consistent after adjusting for three preexisting diseases (asthma, anemia, and dental caries). Tables S10 to S14 indicate that when redefining OWO and recalculating Z-scores of BMI according to the Center for Disease Control and Prevention (USA) reference population, similar results were observed. Specifically, ambient PM exposure was significantly associated with obesity indicators, and there was a notable interaction between PM exposure and maternal or paternal age on these indicators.

Table 6 Associations between PM exposure and obesity indicators stratified by paternal age at childbirth

-	$PM_{2.5}$			PM_c			PM_{10}		
Main analysis[a]	**Childbearing age**	**Advanced age**	**P value for difference**	**Childbearing age**	**Advanced age**	**P value for difference**	**Childbearing age**	**Advanced age**	**P value for difference**
	β(95%CI)		-	**β(95%CI)**		-	**β(95%CI)**		-
Z-scores of WFH	-0.38 (-0.60, -0.16)	0.15 (-0.57, 0.88)	< 0.001	-0.38 (-0.62, -0.13)	0.06 (-0.66, 0.78)	< 0.001	-0.20 (-0.32, -0.08)	0.06 (-0.32, 0.43)	< 0.001
WHR	-0.02 (-0.04, 0.01)	0.04 (0.03, 0.05)	< 0.001	-0.01 (-0.04, 0.01)	0.08 (0.07, 0.09)	< 0.001	-0.01 (-0.02, 0.00)	0.03 (0.03, 0.04)	< 0.001
BMI	0.19 (-0.04, 0.41)	1.99 (1.21, 2.77)	< 0.001	0.44 (0.19, 0.69)	1.77 (0.98, 2.56)	< 0.001	0.16 (0.04, 0.29)	1.00 (0.60, 1.40)	< 0.001
Z-scores of BMI[b]	0.00 (-0.14, 0.13)	1.53 (1.05, 2.01)	< 0.001	0.07 (-0.07, 0.22)	1.59 (1.12, 2.07)	< 0.001	0.02 (-0.06, 0.09)	0.83 (0.58, 1.07)	< 0.001
	OR(95%CI)		-	**OR(95%CI)**		-	**OR(95%CI)**		-
OWO[c]	1.74 (1.15, 2.62)	3.52 (2.92, 4.24)	0.001	1.57 (0.96, 2.55)	3.65 (2.62, 5.08)	0.002	1.32 (1.04, 1.66)	2.65 (2.05, 3.43)	0.001

Notes: PM, particulate matter; $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 μm; PM_c, particulate matter with an aerodynamic diameter of 2.5 to 10 μm, PM_{10}, particulate matter with an aerodynamic diameter of 10 μm; WFH, weight-for-height; WHR, waist-to-hip ratio; BMI, body mass index; OWO, overweight and obesity

[a]Main analysis: adjusted for age, sex, low birth weight, asthma history, anemia history, history of dental caries, being ill for the last two weeks, optimal feeding scores, secondary smoke, residence, maternal education level, maternal height, maternal weight, mother suffering from anemia during pregnancy, wealth category, drinking water source, relative humidity, mean temperature, altitude, and maternal age

[b]Z-scores of BMI: calculated according to WHO reference population

[c]OWO: Z-scores of WFH ≥ 2

Table 7 The interaction between age and PM exposure on obesity indicators

Main analysis[a]	$PM_{2.5}$		PM_c		PM_{10}	
Maternal age*PM						
-	**$β_{int}$(95%CI)**	**P value for interaction**	**$β_{int}$(95%CI)**	**P value for interaction**	**$β_{int}$(95%CI)**	**P value for interaction**
Z-scores of WFH	0.33(-0.37, 1.04)	0.351	0.14(-0.62, 0.90)	0.717	0.13(-0.25, 0.51)	0.497
WHR	0.04(0.07, 0.01)	0.018	0.07(0.11, 0.04)	< 0.001	0.03(0.05, 0.01)	0.001
BMI	1.61(0.91, 2.31)	< 0.001	1.16(0.40, 1.92)	0.003	0.76(0.38, 1.14)	< 0.001
Z-scores of BMI[b]	1.43(1.01, 1.85)	< 0.001	1.54(1.08, 1.99)	< 0.001	0.80(0.57, 1.03)	< 0.001
-	**OR_{int}(95%CI)**	**P value for interaction**	**OR_{int}(95%CI)**	**P value for interaction**	**OR_{int}(95%CI)**	**P value for interaction**
OWO[c]	2.65(1.78, 3.95)	0.002	2.97(1.52, 5.80)	< 0.001	2.55(1.50, 4.34)	0.001
Paternal age*PM						
-	**$β_{int}$(95%CI)**	**P value for interaction**	**$β_{int}$(95%CI)**	**P value for interaction**	**$β_{int}$(95%CI)**	**P value for interaction**
Z-scores of WFH	0.50(-0.37, 1.38)	0.260	0.35(-0.53, 1.22)	0.440	0.22(-0.23, 0.68)	0.330
WHR	0.05(0.09, 0.01)	0.016	0.09(0.12, 0.05)	< 0.001	0.03(0.05, 0.01)	0.001
BMI	1.86(0.98, 2.74)	< 0.001	1.31(0.43, 2.19)	0.004	0.84(0.39, 1.30)	< 0.001
Z-scores of BMI[b]	1.54(1.01, 2.07)	< 0.001	1.48(0.95, 2.01)	< 0.001	0.80(0.53, 1.08)	< 0.001
-	**OR_{int}(95%CI)**	**P value for interaction**	**OR_{int}(95%CI)**	**P value for interaction**	**OR_{int}(95%CI)**	**P value for interaction**
OWO[c]	3.10(2.09, 4.60)	0.001	3.46(2.1, 5.70)	0.002	2.77(1.49, 5.14)	0.001

Notes: PM, particulate matter; $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 μm; PM_c, particulate matter with an aerodynamic diameter of 2.5 to 10 μm, PM_{10}, particulate matter with an aerodynamic diameter of 10 μm; WFH, weight-for-height; WHR, waist-to-hip ratio; BMI, body mass index; OWO, overweight and obesity

[a]Main analysis: adjusted for age, sex, low birth weight, asthma history, anemia history, history of dental caries, being ill for the last two weeks, optimal feeding scores, secondary smoke, residence, maternal education level, maternal height, maternal weight, mother suffering from anemia during pregnancy, wealth category, drinking water source, relative humidity, mean temperature, altitude, maternal age and paternal age

[b]Z-scores of BMI: calculated according to WHO reference population

[c]OWO: Z-scores of WFH ≥ 2

Discussion

This study found significant correlations between increases in ambient $PM_{2.5}$, PM_c and PM_{10} and enhancements in WHR, BMI, and OWO among children under 5 years old residing in high-altitude areas of China. Specifically, PM_{10} showed the most substantial absolute changes in BMI and OR of OWO, while PM_c was associated with the most significant absolute changes in WHR. A more pronounced impact of ambient $PM_{2.5}$, PM_c and PM_{10} on obesity indicators was observed in mothers aged ≥ 35 years and fathers aged ≥ 40 years compared to younger parents. Demographic and socio-economic factors appeared to influence the relationship between PM exposure and obesity indicators in children, with the exception of OWO. These findings highlight the potential of PM control as a policy-level intervention for early prevention of childhood OWO in high-altitude regions, particularly among children of older parents.

A previous review established that exposure to air pollution contributes to an increased risk of childhood obesity [43]. Our study similarly found that elevated levels of PM were positively associated with OWO among children residing in high-altitude areas, consistent with findings from studies in Spain [44], Korea [45], and low-altitude regions of China [46, 47]. Specifically, the OR for the risk of weight gain or obesity due to $PM_{2.5}$ exposure in Asia was 1.11, while data on the effects of PM_{10} in Asia remains limited [43]. Our study contributes uniquely to the Asian region by identifying the impact of PM on childhood obesity in high-altitude areas, particularly highlighting the influence of PM_{10}. Interestingly, we observed heterogeneity in the effects of different pollutants on various obesity indicators. PM_{10}, for instance, showed the most significant absolute changes in BMI and OWO, whereas PM_c (specify the composition of PM_c) was associated with the largest absolute changes in WHR. These findings contrast with previous studies focusing on PM's impact on lung health, such as one study suggesting that PM_1 had greater effects on children relative to $PM_{2.5}$ and PM_{10}, varying by sex, age, and season [48]. This highlights the need to consider both the health and economic impacts of different diseases when prioritizing PM control strategies based on particle size. Further, population-based studies and empirical evidence are necessary to validate the impact of air pollution on different obesity indicators.

An important finding of our study is the modifying effect of parental age on the association between PM exposure and childhood obesity. A previous study involving 6,693 pregnant women compared childbearing age groups with advanced age groups and found that PM_{10} exposure was significantly associated with an increased risk of preterm birth only in the advanced age group during pregnancy, with this association modified by advanced maternal age [49]. In contrast to this previous focus solely on maternal age [49], our study identified that paternal childbearing age may also interact with PM exposure, showing stronger impacts on obesity indicators in the advanced maternal age group compared to the childbearing maternal group. Theoretically, an age-related increase in susceptibility to the negative effects of PM exposure may explain this stronger association in older mothers [48]. Younger mothers may have higher detoxification and excretion capacities for toxic compounds compared to older women. Specifically, hypoxia or toxic components in air pollution may directly affect maternal health [50, 51], and such physiological mechanisms may be more pronounced in older women due to vasoconstriction in umbilical arteries [52, 53]. Our findings suggest that policies promoting childbearing at younger ages may mitigate the health impacts of PM on offspring. However, research on the modifying effects of parental age at birth in the association between air pollution and child health outcomes is limited, necessitating cross-regional studies with large samples.

Several other modifiers warrant further discussion from our study. Firstly, we identified differences in the associations between PM exposure and obesity indicators across sex and age groups. Specifically, males and younger individuals showed greater susceptibility to the association between air pollution and BMI and its Z-scores compared to their counterparts, whereas females and older individuals were more affected in terms of WHR and Z-scores of WFH. These findings suggest that considering subtypes of obesity rather than relying solely on BMI may be crucial in identifying vulnerable populations. Unexpectedly, our study also found a stronger effect of $PM_{2.5}$ on Z-scores of WFH, WHR, BMI, and BMI Z-scores in children from households with high income and high feeding scores compared to those from lower-income households with lower feeding scores. These findings contradict previous research suggesting that better economic and dietary conditions may mitigate the impact of air pollution on obesity [13, 54]. The relatively unique lifestyle, including dietary patterns with high sodium intake, among the indigenous population at our study site may contribute to increased vulnerability to childhood weight gain and obesity [55]. Therefore, our findings suggest that interventions targeting PM-related childhood obesity should prioritize children from wealthier families.

Existing studies suggest that the association between air pollution and childhood OWO can be attributed to two primary mechanisms. The first involves biological pathways, including oxidative stress, systemic inflammation, hypothalamic-pituitary-adrenal axis dysregulation, and epigenetic modifications [56]. For instance, a study in Mexico City demonstrated variations in leptin, endothelin-1, glucagon-like peptide-1, ghrelin, and glucagon levels between children exposed to PM and those who were not exposed [54]. Experimental studies have also shown that PM exposure may disrupt metabolic regulation and contribute to weight gain through inflammation, oxidative stress, and hormonal disruptions [57, 58]. The second mechanism involves behavioral factors. Several studies indicate that air pollution is associated with reduced physical activity levels, potentially leading to increased sedentary behaviors and childhood obesity [59, 60]. However, some studies have reported non-significant associations [12, 56, 59]. For example, research in Italy found no significant association between pollutant exposure and obesity [12], while a study in Korea reported a negative association with weight in children aged 12–60 months [56]. These discrepancies may be influenced by factors such as sample size, overall pollutant concentrations, and characteristics of the study population.

To the best of our knowledge, this study is among the few that specifically investigate the impact of air pollution on preschool obesity in high-altitude areas of Asia, offering several strengths. Firstly, our study comprehensively assessed a range of obesity-related indicators, including WHR and z-scores of WFH, providing a detailed understanding of how these indicators are influenced by air pollution. Secondly, we explored the effect modification of parental age at birth, a factor seldom studied in this context. Thirdly, we analyzed the non-linear relationship between PM exposure and obesity indicators, identifying potential thresholds of greatest risk and informing timely intervention strategies. Lastly, we conducted rigorous sensitivity analyses, examining the impact of different study years and excluding specific cases, to ensure the robustness and stability of our findings.

However, several limitations should be acknowledged. Firstly, due to the inherent constraints of cross-sectional studies, establishing the chronological order between exposure to PM and the onset of childhood obesity is not possible. Therefore, caution is warranted in concluding that PM exposure is positively associated with childhood obesity. Future longitudinal or quasi-experimental studies are essential to provide stronger evidence and establish causal links. Secondly, our study was unable to adjust for all potential confounding factors. For example, while we assessed optimal feeding scores based on breastfeeding and food consumption frequency, we did not account for specific food or breast milk intake. Additionally, important mediators such as physical activity and methylation status were not measured and, therefore, not included in our analysis. Moreover, due to data limitations, we were unable to adjust for maternal gestational hypertension, diabetes variables, and indoor air pollution, which could potentially impact childhood obesity outcomes. Nonetheless, we conducted sensitivity analyses, including E-value calculations (Table S15), which suggested that our results are relatively robust despite these limitations. Thirdly, PM exposure measurements were based on participants' residential addresses, without accounting for potential exposure variations during outdoor activities or mobility across areas with different pollution levels. Lastly, our study was conducted specifically in Chinese high-altitude areas, and therefore, the generalizability of our findings to high-altitude regions in Western countries may be limited.

Conclusion

In conclusion, our study demonstrates that ambient PM exposure is positively associated with OWO, WHR, and BMI, with these associations further modified by parents' age at childbirth. These findings highlight the potential of PM control as a policy-level intervention for early prevention of childhood OWO, particularly in high-altitude areas. Targeting interventions toward parents of advanced childbearing age could substantially contribute to creating health-promoting environments.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-024-20598-3.

Supplementary Material 1

Acknowledgements

We are grateful to the data support from "National Earth System Science Data Center, National Science & Technology Infrastructure of China. (http://www.geodata.cn)".

Author contributions

Xianzhi Li: Data analysis, Validation, Writing-Original original draft preparation and Editing. Yajie Li and Bin Yu: Study design, Data collection, Validation, and Writing-Original original draft preparation. Haorong Meng, Zonglei Zhou, Shunjin Liu, and Yunyun Tian: Data cleaning and Data collection. Xiangyi Xing, Yingchao Lei and Li Yin: Study design, Validation, Supervision, and Writing-Reviewing and Editing.

Funding

This work was supported by Scientific Research Project of Panzhihua Central Hospital (grant numbers 202102, 202201, 202315, 202406), Special Basic Cooperative Research Programs of Yunnan Provincial Undergraduate Universities' Association (grant numbers 202101AO070272, 202101BA070001-220), Health Commission of Sichuan Province Medical Science and Technology Program (grant numbers 24WSXT004), Research Programme of Panzhihua Science and Technology Bureau (grant number 2021ZX-5-1, 2022ZD-S-12, 2022ZD-S-24, 2022ZD-S-26, 2023ZD-C-1, 2024ZD-S-29), Natural Science Foundation of Tibet Autonomous Region (grant numbers XZ202101ZR0103G, XZ2019ZR G-142), and Sichuan Province key clinical specialty construction Project, China.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was approved by the ethics committee of Tibet Center for Disease Control and Prevention (2020-003). Informed consent was obtained from all participants, and all methods were performed in accordance with the relevant guidelines and regulations. The records of participants were anonymized and deidentified before analysis.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

[1]Meteorological Medical Research Center, Panzhihua Central Hospital, Panzhihua, China

[2]Clinical Medical Research Center, Panzhihua Central Hospital, Panzhihua, China
[3]Dali University, Dali, China
[4]Institute for Disaster Management and Reconstruction, Sichuan University - Hong Kong Polytechnic University, Chengdu, China
[5]Tibet Center for Disease Control and Prevention, Lhasa, China
[6]Yunnan Center for Disease Control and Prevention, Kunming, China
[7]Department of Epidemiology, School of Public Health, Fudan University, Shanghai, China
[8]Department of Pharmacy, Panzhihua Central Hospital, Panzhihua, China
[9]Panzhihua University, Panzhihua, China

Received: 23 January 2024 / Accepted: 1 November 2024
Published online: 07 November 2024

References

1. Lister NB, Baur LA, Felix JF, Hill AJ, Marcus C, Reinehr T, Summerbell C, Wabitsch M. Child and adolescent obesity. Nat Reviews Disease Primers. 2023;9(1):24.
2. World Health Organization. Obesity and overweight, June. 14, 2021. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. Accessed Sep 20, 2023.
3. The State Council Information Office of the People's Republic of China. Press briefing for the Report on Chinese Residents' Chronic Diseases and Nutrition 2020. December 24, 2020 (in Chinese). http://www.gov.cn/xinwen/2020-12/24/content_5572983.html. Accessed Sep 20, 2023.
4. Zheng H, Xu Z, Wang Q, Ding Z, Zhou L, Xu Y, Su H, Li X, Zhang F, Cheng J. Long-term exposure to ambient air pollution and obesity in school-aged children and adolescents in Jiangsu Province of China. Environ Res. 2021;195:110804.
5. Shi X, Zheng Y, Cui H, Zhang Y, Jiang M. Exposure to outdoor and indoor air pollution and risk of overweight and obesity across different life periods: a review. Ecotoxicol Environ Saf. 2022;242:113893.
6. Lindberg L, Hagman E, Danielsson P, Marcus C, Persson M. Anxiety and depression in children and adolescents with obesity: a nationwide study in Sweden. BMC Med. 2020;18(1):30.
7. Su W, Song Q, Li N, Wang H, Guo X, Liang Q, Liang M, Ding X, Qin Q, Chen M, et al. The effect of air pollution and emotional and behavioral problems on preschoolers' overweight and obesity. Environ Sci Pollut Res Int. 2022;29(50):75587–96.
8. Aris IM, Perng W, Dabelea D, Padula AM, Alshawabkeh A, Vélez-Vega CM, Aschner JL, Camargo CA Jr., Sussman TJ, Dunlop AL, et al. Associations of Neighborhood Opportunity and Social Vulnerability with trajectories of Childhood Body Mass index and obesity among US children. JAMA Netw open. 2022;5(12):e2247957.
9. Jiang N, Bao WW, Gui ZH, Chen YC, Zhao Y, Huang S, Zhang YS, Liang JH, Pu XY, Huang SY, et al. Findings of indoor air pollution and childhood obesity in a cross-sectional study of Chinese schoolchildren. Environ Res. 2023;225:115611.
10. Ye L, Zhou J, Tian Y, Cui J, Chen C, Wang J, Wang Y, Wei Y, Ye J, Li C, et al. Associations of residential greenness and ambient air pollution with overweight and obesity in older adults. Obes (Silver Spring Md). 2023;31(10):2627–37.
11. Brumberg HL, Karr CJ. Ambient Air Pollution: Health hazards to Children. Pediatrics 2021, 147(6).
12. Fioravanti S, Cesaroni G, Badaloni C, Michelozzi P, Forastiere F, Porta D. Traffic-related air pollution and childhood obesity in an Italian birth cohort. Environ Res. 2018;160:479–86.
13. de Bont J, Díaz Y, de Castro M, Cirach M, Basagaña X, Nieuwenhuijsen M, Duarte-Salles T, Vrijheid M. Ambient air pollution and the development of overweight and obesity in children: a large longitudinal study. Int J Obes. 2021;45(5):1124–32.
14. Huang JV, Leung GM, Schooling CM. The association of air pollution with body mass index: evidence from Hong Kong's children of 1997 birth cohort. Int J Obes. 2019;43(1):62–72.
15. Cho CC, Hsieh WY, Tsai CH, Chen CY, Chang HF, Lin CS. In Vitro and in vivo experimental studies of PM(2.5) on Disease Progression. Int J Environ Res Public Health 2018, 15(7).
16. Huang T, Liu M, Xing R, Men Y, Shen G. Threat of air pollution in the cleanest plateau. Innov (Cambridge (Mass)). 2023;4(2):100390.
17. Shen G, Xiong R, Cheng H, Tao S. Rural residential energy carrier structure and primary PM2.5 emissions from the Qinghai-Tibet Plateau. Chin Sci Bull. 2021;66:1900–11.
18. Tian Y, Liu H, Wu Y, Si Y, Song J, Cao Y, Li M, Wu Y, Wang X, Chen L, et al. Association between ambient fine particulate pollution and hospital admissions for cause specific cardiovascular disease: time series study in 184 major Chinese cities. BMJ. 2019;367:l6572.
19. Jefferson L, Bloor K, Spilsbury K. Exploring gender differences in the working lives of UK Hospital consultants. J R Soc Med. 2015;108(5):184–91.
20. Seoud MA, Nassar AH, Usta IM, Melhem Z, Kazma A, Khalil AM. Impact of advanced maternal age on pregnancy outcome. Am J Perinatol. 2002;19(1):1–8.
21. Patterson ML, Stern S, Crawford PB, McMahon RP, Similo SL, Schreiber GB, Morrison JA, Waclawiw MA. Sociodemographic factors and obesity in pre-adolescent black and white girls: NHLBI's growth and health study. J Natl Med Assoc. 1997;89(9):594–600.
22. Robson JO, Verstraete SG, Shiboski S, Heyman MB, Wojcicki JM. A risk score for childhood obesity in an urban latino cohort. J Pediatr. 2016;172:29–e3421.
23. Weng SF, Redsell SA, Nathan D, Swift JA, Yang M, Glazebrook C. Estimating overweight risk in childhood from predictors during infancy. Pediatrics. 2013;132(2):e414–421.
24. Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, Steer C, Sherriff A. Early life risk factors for obesity in childhood: cohort study. BMJ. 2005;330(7504):1357.
25. Eriksen W, Sundet JM, Tambs K. Paternal age at birth and the risk of obesity in young adulthood: a register-based birth cohort study of Norwegian males. Am J Hum Biol. 2013;25(1):29–34.
26. Li X, Li Y, Xing X, Liu Y, Zhou Z, Liu S, Tian Y, Nima Q, Yin L, Yu B. Urban-rural disparities in the association between long-term exposure to high altitude and malnutrition among children under 5 years old: evidence from a cross-sectional study in Tibet. Public Health Nutr. 2022;26(4):1–10.
27. Li X, Li Y, Yu B, Nima Q, Meng H, Shen M, Zhou Z, Liu S, Tian Y, Xing X, et al. Urban-rural differences in the association between long-term exposure to ambient particulate matter (PM) and malnutrition status among children under five years old: a cross-sectional study in China. J Global Health. 2023;13:04112.
28. Wang Z, Dang S, Yan H. Nutrient intakes of rural tibetan mothers: a cross-sectional survey. BMC Public Health. 2010;10:801.
29. Dang SN, Yan H, Wang XL, Zeng LX, Xie H. [The introduction of complementary food of children under the age of three years in Tibet of China]. Zhonghua Liu Xing Bing Xue Za Zhi = Zhonghua liuxingbingxue zazhi 2003, 24(8):674–7.
30. WHO, UNICEF. 2006a. Core questions on drinking-water and sanitation for household surveys, 2023.
31. WHO, W.H.O. 2010. Indicators for Assessing Infant and Young Child Feeding Practices Part 3: Country Profiles, 2023.
32. WHO, W.H.O. 2006b. WHO child growth standards: length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: methods and development, 2023.
33. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. Bull World Health Organ. 2007;85(9):660–7.
34. Wei J, Li Z, Xue W, Sun L, Fan T, Liu L, Su T, Cribb M. The ChinaHighPM(10) dataset: generation, validation, and spatiotemporal variations from 2015 to 2019 across China. Environ Int. 2021;146:106290.
35. Wei J, Li Z, Lyapustin A, Sun L, Peng Y, Xue W, Su T, Cribb M. Reconstructing 1-km-resolution high-quality PM2.5 data records from 2000 to 2018 in China: spatiotemporal variations and policy implications. Remote Sens Environ. 2021;252:112136.
36. Wei J, Co-authors. Improved 1 km resolution PM. Estimates across China using enhanced space-time extremely randomized trees. Atmos Chem Phys. 2020;20(6):3273–89.
37. Shi W, Li Y, Zhao JV. Long-term exposure to ambient air pollution with sarcopenia among middle-aged and older adults in China. J Nutr Health Aging. 2024;28(2):100029.
38. Flenady V, Koopmans L, Middleton P, Frøen JF, Smith GC, Gibbons K, Coory M, Gordon A, Ellwood D, McIntyre HD, et al. Major risk factors for stillbirth in high-income countries: a systematic review and meta-analysis. Lancet (London England). 2011;377(9774):1331–40.
39. Fall CH, Sachdev HS, Osmond C, Restrepo-Mendez MC, Victora C, Martorell R, Stein AD, Sinha S, Tandon N, Adair L, et al. Association between maternal age at childbirth and child and adult outcomes in the offspring: a prospective

study in five low-income and middle-income countries (COHORTS collaboration). Lancet Glob Health. 2015;3(7):e366–377.
40. Brown HK. Paternal factors in preconception care: the case of paternal age. BMJ. 2018;363:k4466.
41. Svensson E, Rogvin M, Hultman CM, Reichborn-Kjennerud T, Sandin S, Moger TA. Schizophrenia susceptibility and age of diagnosis–a frailty approach. Schizophr Res. 2013;147(1):140–6.
42. National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention. BMI: body mass index. 2002 Atlanta, GA Centers for Disease Control and Prevention. http://www.cdc.gov/nccdphp/dnpa/bmi. Accessed January 27, 2023.
43. Parasin N, Amnuaylojaroen T, Saokaew S. Effect of Air Pollution on obesity in children: a systematic review and Meta-analysis. Children (Basel, Switzerland) 2021, 8(5).
44. de Bont J, Casas M, Barrera-Gómez J, Cirach M, Rivas I, Valvi D, Álvarez M, Dadvand P, Sunyer J, Vrijheid M. Ambient air pollution and overweight and obesity in school-aged children in Barcelona. Spain Environ Int. 2019;125:58–64.
45. Kim E, Park H, Park EA, Hong YC, Ha M, Kim HC, Ha EH. Particulate matter and early childhood body weight. Environ Int. 2016;94:591–9.
46. Tong J, Ren Y, Liu F, Liang F, Tang X, Huang D, An X, Liang X. The impact of PM2.5 on the growth curves of children's obesity indexes: a prospective cohort study. Front Public Health. 2022;10:843622.
47. Wu QZ, Xu SL, Tan YW, Qian Z, Vaughn MG, McMillin SE, Dong P, Qin SJ, Liang LX, Lin LZ, et al. Exposure to ultrafine particles and childhood obesity: a cross-sectional analysis of the Seven Northeast Cities (SNEC) study in China. Sci Total Environ. 2022;846:157524.
48. McMahon TF, Birnbaum LS. Age-related changes in toxicity and biotransformation of potassium cyanide in male C57BL/6 N mice. Toxicol Appl Pharmcol. 1990;105(2):305–14.
49. Han Y, Jiang P, Dong T, Ding X, Chen T, Villanger GD, Aase H, Huang L, Xia Y. Maternal air pollution exposure and preterm birth in Wuxi, China: effect modification by maternal age. Ecotoxicol Environ Saf. 2018;157:457–62.
50. Horner JM. Anthropogenic emissions of carbon monoxide. Rev Environ Health. 2000;15(3):289–98.
51. Kurmi OP, Gaihre S, Semple S, Ayres JG. Acute exposure to biomass smoke causes oxygen desaturation in adult women. Thorax. 2011;66(8):724–5.
52. Martens DS, Cox B, Janssen BG, Clemente DBP, Gasparrini A, Vanpoucke C, Lefebvre W, Roels HA, Plusquin M, Nawrot TS. Prenatal air Pollution and newborns' predisposition to Accelerated Biological Aging. JAMA Pediatr. 2017;171(12):1160–7.
53. Ion R, Bernal AL. Smoking and Preterm Birth. Reproductive sciences (Thousand Oaks, Calif) 2015, 22(8):918–926.
54. Calderón-Garcidueñas L, Franco-Lira M, D'Angiulli A, Rodríguez-Díaz J, Blaurock-Busch E, Busch Y, Chao CK, Thompson C, Mukherjee PS, Torres-Jardón R, et al. Mexico City normal weight children exposed to high concentrations of ambient PM2.5 show high blood leptin and endothelin-1, vitamin D deficiency, and food reward hormone dysregulation versus low pollution controls. Relevance for obesity and Alzheimer disease. Environ Res. 2015;140:579–92.
55. Agyemang C, Bhopal R. Hypertension and cardiovascular disease endpoints by ethnic group: the promise of data linkage. Heart. 2013;99(10):675–6.
56. Seo MY, Kim SH, Park MJ. Air pollution and childhood obesity. Clin Experimental Pediatr. 2020;63(10):382–8.
57. Sun Q, Yue P, Deiuliis JA, Lumeng CN, Kampfrath T, Mikolaj MB, Cai Y, Ostrowski MC, Lu B, Parthasarathy S, et al. Ambient air pollution exaggerates adipose inflammation and insulin resistance in a mouse model of diet-induced obesity. Circulation. 2009;119(4):538–46.
58. Haberzettl P, O'Toole TE, Bhatnagar A, Conklin DJ. Exposure to fine Particulate Air Pollution causes vascular insulin resistance by inducing pulmonary oxidative stress. Environ Health Perspect. 2016;124(12):1830–9.
59. An R, Ji M, Yan H, Guan C. Impact of ambient air pollution on obesity: a systematic review. International journal of obesity (2005) 2018, 42(6):1112–1126.
60. An R, Zhang S, Ji M, Guan C. Impact of ambient air pollution on physical activity among adults: a systematic review and meta-analysis. Perspect Public Health. 2018;138(2):111–21.

Publisher's note