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Moderating effect of a sodium-rich diet on the association between long-term exposure to fine particulate matter and blood lipids in children and adolescents

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Abstract

Background Several studies reported that exposure to higher levels of fine particulate matter ($PM_{2.5}$) was associated with deteriorated lipid profiles in children and adolescents. However, whether a sodium-rich diet could modify the associations remains unknown. We aimed to examine the associations of long-term exposure to $PM_{2.5}$ with blood lipids in children and adolescents, and further examine the effect modification by dietary and urinary sodium levels based on a multi-community population in China.

Methods The 3711 study participants were from a cross-sectional study, which interviewed children and adolescents aged 6 to 17 years across Sichuan Province, China between 2015 and 2017. Blood lipid outcomes including blood total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG) were assessed. Information on daily dietary sodium consumption was estimated with a semi-quantitative food frequency questionnaire (FFQ), and urinary sodium was used as an internal exposure biomarker. A linear regression model was applied to estimate the associations of prior 2-years' average exposure to ambient $PM_{2.5}$ with blood lipids. The effect modification by dietary and urinary sodium was examined by stratified analyses.

Results The participants from rural areas had higher levels of daily sodium consumptions. The results of multivariable regression analysis indicated that per 10 μg/m^3 incremental change in $PM_{2.5}$ was associated with a 1.56% (95% confidence interval 0.90%—2.23%) and a 2.26% (1.15%—3.38%) higher blood TC and LDL-C levels, respectively. Among the study participants with higher levels of dietary sodium or urinary sodium, exposure to higher levels of $PM_{2.5}$ was significantly associated with deteriorated lipid profiles. For example, each 10 μg/m^3 incremental change in exposure to $PM_{2.5}$ was correlated with a 2.83 (-4.65 to -0.97) lower percentage decrease in blood HDL-C levels among the participants who were from the highest quartile of urinary sodium levels. While, these associations changed to be nonsignificant in the participants who were from the lowest quartile of dietary sodium levels.

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Conclusion Exposure to higher levels of $PM_{2.5}$ was associated with deteriorated blood lipid levels in children and adolescents. It is noteworthy that these associations might be ameliorated through the adoption of a low-sodium dietary regimen.

Keywords $PM_{2.5}$, Blood lipids, Sodium-rich diet, Urinary sodium, Children, Adolescents

Background

Atherosclerotic cardiovascular disease (ASCVD) is a well-documented risk for the global population. The Global Burden of Disease study provided evidence to indicate that approximately 32% of global deaths were attributable to prevalent ASCVDs [1]. The existing evidence supported the strong associations of abnormal blood lipids, such as too low levels of blood HDL-C, and high levels of blood LDL-C, with elevated risk of incident ASCVDs in adults [2, 3]. Recently, the blood lipid abnormalities have been increasingly reported in children and adolescents, with a worldwide incidence ranging from 10 to 30% [4–6]. Those with deteriorated lipid profiles in childhood and adolescence are at increased risk of having ASCVDs in their adulthood [7–9]. Therefore, identifying the risk factors associated with deteriorated lipid profiles in children and adolescents is essential to bring down the globally increasing morbidity and disease burden related to ASCVDs.

Exposure to higher levels of ambient $PM_{2.5}$ has been widely documented as an important risk associated with deteriorated lipid profiles in adults [10–12]. However, the evidence in children and adolescents is limited. Mechanism evidence has emphasized the role of inflammatory response underlying the deteriorated effects of $PM_{2.5}$ exposure on lipid profiles in the general population [13–15]. On the other hand, increasing evidence have documented the relationship between higher blood sodium levels with greater inflammation risks [16]. Meanwhile, the increasing rodent evidence have reported that a high-sodium exposure could induce the inflammation [17, 18]. Generally, the accumulation of sodium in human body is largely from the daily dietary consumptions [19]. Epidemiological evidence highlighted the association between a high-sodium diet and elevated inflammation risks [20, 21]. Therefore, a sodium-rich diet might have an role in inducing the individual-level inflammation responses. Since the pro-oxidant and pro-inflammatory responses are the key mechanisms linking $PM_{2.5}$ exposure to dysregulation of lipid metabolism, it is plausible that a high-sodium diet could amplify the association of exposure to $PM_{2.5}$ with deteriorated lipid profiles. However, population evidence which focused on examining the effect modification of a high-sodium diet on the associations between $PM_{2.5}$ exposure and blood lipids, especially in children and adolescents, is still limited.

Therefore, we investigated the associations of exposure to $PM_{2.5}$ with blood lipids in children and adolescents, and further examined the effect modifications of sodium levels on these associations based on a cross-sectional multi-community Chinese population.

Methods

Population and study design

We enrolled the participants aged from 6 to 17 years from a multi-community cross-sectional study, which were conducted between November 2015 and December 2017, covering 5 urban districts and 9 rural counties in Sichuan Province, China. In each region, two communities from urban districts or two towns from rural counties were selected using the systematic sampling (SS) method. In each community or town, one primary school and one middle school were selected by the simple random sampling (SRS) method. Limited by the population size and economic status, many communities or towns did not have senior high schools, therefore we did not randomly select senior high schools at the community or town level. Alternatively, we selected the study senior high schools at the district or county level by the SRS method. Finally, one class was randomly selected from each grade, and 28 students were SS selected from each class to participate in the study. A face-to-face interview was used to acquire information on the participants' socio-economic and demographic characteristics, diet, physical activity, and health status. Anthropometric characteristics such as weight and height, and blood pressure were measured by physicians through standardized procedures.

Initially, 3784 participants were included. The participants missing the blood lipids measurements were excluded ($n=73$). Initially, 3711 participants were included in the primary association analyses. Then, in the further stratified analyses, the participants missing dietary sodium information ($n=76$), or urinary sodium information ($n=80$) were also excluded, respectively. Finally, the stratified analysis using dietary sodium data and urinary sodium data included 3635 and 3631 participants, respectively. The details about the selection procedures were reported in the Supplementary Material Figure S1.

Measurements of lipid outcomes

We took venous blood samples from all subjects. The plasma and serum samples were separated by centrifugation within 0.5–1.0 h and were dispensed into cryotubes according to a standardized protocol. Blood lipid biomarkers, including blood TC, blood HDL-C, blood LDL-C, and blood TG, were examined by experienced clinical laboratorians according to the standard operating procedures of the instruments in the laboratory of the study hospitals.

Evaluation of $PM_{2.5}$ exposures

Information on participants' address for schools were obtained through a face-to-face interview. Annual average $PM_{2.5}$ exposures, which covered the study areas, were assessed based on the global surface $PM_{2.5}$ V4.CH.02 product of the Dalhousie University Atmospheric Composition Analysis Group (DUACAG) with a spatial resolution of 1 km x 1 km [22]. Briefly, the DUACAG applied a geographically weighted regression model combined with ground level $PM_{2.5}$ monitors, remotely sensed aerosol optical depth (AOD), and multiangle imaging spectroradiometer (MISR) data, and outputs from the CEOS-Chem chemical transport model. Each participant's school address was geocoded, and then the past 2-years' average value of gridded $PM_{2.5}$ data within a 1-km buffer area surrounding the school was calculated to indicate the long-term $PM_{2.5}$ exposure for the study participants.

Measurements of dietary sodium levels

A standardized semi-quantitative Food Frequency Questionnaire (FFQ) recommended by the Chinese CDC, which is a reliable and valid measurement for the Chinese population, was used to assess the individual-level dietary information [23]. The FFQ included dietary information on131 food items, which were specifically categorized into 28 food groups. Details of the frequently consumed food items of the FFQ were summarized in Supplementary Material Table S1. Information of each food group on the consumption frequency corresponding to the participants' past 12 months was recorded as never, daily, weekly, monthly, or yearly for. Then, if the participants responded that they had consumed the specific food group, information on consumption amount for average intake per time was recorded by a Chinese unit of weight or capacity of Liang (50 g) or per cup (100 ml). We transformed all food consumption frequency into daily times by weekly times dividing 7, monthly times dividing 30, and yearly times dividing 365, respectively. Then, the daily amount of consumption for each food group was calculated by multiplying daily times and average intake amount per time. We estimated the daily dietary sodium intake according to the Chinese food composition database (2018), which was published by the Chinese CDC [24]. Additionally, we used the subjects' urinary sodium levels as an internal biomarker to represent the dietary sodium levels. A random urine sample of 8–10 ml was collected from each study participant. Experienced clinical laboratorians examined the urinary sodium levels according to the standard operating procedures of the equipment in the study laboratory.

Covariates

Participants' socio-economic and demographic characteristics were collected, including residential region (urban, rural), as well as father's ethnicity (minority, Han), education (0–6, 7–9, 10–12, and ≥ 13 years), and occupation (farmer/ retired worker, community/social service occupations, and skilled/professional/administrative jobs). The children's demographic information, including age (years) and sex (male, female) were also collected. The body mass index (BMI), which was calculated by dividing height (kg) by the square of height (meters), was used to represent a participant's physical fitness. Behavioral characteristics that could potentially affect children, such as passive smoking status (no, yes), and daily outdoor activity duration (hours), were collected.

Statistical analysis

We summarized the distribution of demographic, social-economic, and behavioral characteristics of the study participants across urinary sodium strata by count and proportion. The results of significant tests for these characteristics across urinary sodium strata were estimated by chi-square tests or Fisher exact tests.

Linear regression models were applied to explore the correlations of per 10 ug/m^3 increase in $PM_{2.5}$ exposure with blood lipid outcomes. We further categorized the $PM_{2.5}$ exposure into quartiles, and estimated the associations of the 2rd to highest quartiles of $PM_{2.5}$ exposure with blood lipids, compared with the lowest $PM_{2.5}$ exposure, to examine the potential dose–response relationships of the associations. The results of "*P* for trend" for blood lipid outcomes associated with $PM_{2.5}$ quartiles were calculated by modeling the median values of $PM_{2.5}$ within each quartile stratum as a continuous variable. Additionally, we examined the potential non-linear associations of $PM_{2.5}$ exposure with blood lipids using the restricted cubic spline methods. In the multivariable-adjusted analysis, a direct acyclic graphic (DAG) method was introduced to determine the potential confounders (Supplementary Material Figure S2). To assess the effect modification attributed to dietary sodium levels on the associations of $PM_{2.5}$

exposure with blood lipid outcomes, we conducted stratified analyses by dietary sodium quartiles and urinary sodium quartiles. In each stratum, a linear regression model which adjusted the covariates in the DAG diagram was fitted in order to explore the study associations. To obtain an overall assessment of the effect modification, we applied the log-likelihood ratio test by comparing the difference between the model with the interaction term and the model without that interaction term. We performed a number of sensitivity analyses to evaluate the robustness of the study associations. We first evaluated the dietary variations on the study associations by additionally adjusting for the dietary intake of total fat and total energy. In addition, we excluded the participants whose parents have been diagnosed with cardiovascular diseases to evaluate the genetic variations on the study associations. Finally, we conducted a sensitivity analysis by replicating the stratified analyses based on creatinine-adjusted urinary sodium concentrations, to allow for the random urine samples' variation in dilution.

All statistical analyses in this study were performed using SAS 9.4. The significance level was determined by a two-sided test with a "P value < 0.05".

Results

Table 1 summarized the distribution of demographic, social-economic, and behavioral characteristics for the study participants across urinary sodium quartiles. The participants who were from rural areas, had fathers belonging to ethnical minorities or accepting poor education, or being farmers/retirees, were more likely to have higher urinary sodium levels (Table 1). The children who had a higher BMI and were older preferred a high-sodium diet. The distribution of long-term $PM_{2.5}$ exposure levels ranged from 22.4 to 65.0 μg/m^3 among the study participants (Table 1). Those exposed to the highest quartiles of $PM_{2.5}$ exposure had higher levels of blood LDL-C (Table 2).

The results of association analyses between $PM_{2.5}$ exposure and blood lipids were presented in Table 3. After adjusted for confounders, per 10 μg/m^3 incremental change in $PM_{2.5}$ was related to a 1.56% (95% confidence

Table 1 The demographic and socio-economic characteristics for study participants stratified by urinary sodium levels. (N = 3631)

Covariates	Childhood urinary sodium levels, quartile				*P*- value
	Quartile 1	Quartile 2	Quartile 3	Quartile 4	
Region, n (%)					< 0.001
Urban	359 (27.32)	371 (28.23)	338 (25.72)	246 (18.72)	
Rural	549 (23.69)	541 (23.35)	576 (24.86)	651 (28.10)	
Father's ethnicity, n(%)					< 0.001
Minority	29 (13.06)	32 (14.41)	75 (33.78)	86 (38.74)	
Han	879 (25.78)	880 (25.81)	839 (24.61)	811 (23.79)	
Father's educational years, n (%)					< 0.001
0~6	206 (22.06)	183 (19.59)	254 (27.19)	291 (31.16)	
7~9	399 (23.71)	434 (25.79)	419 (24.90)	431 (25.61)	
10~12	175 (27.47)	187 (29.36)	149 (23.39)	126 (19.78)	
≥13	128 (34.04)	107 (28.46)	92 (24.47)	49 (13.03)	
Father's occupation, n (%)					< 0.001
Farmer/retired	141 (22.93)	136 (22.11)	161 (26.18)	177 (28.78)	
Community/social services occupations	629 (24.35)	666 (25.78)	648 (25.09)	640 (24.78)	
Skilled/professional/administration occupations	138 (32.09)	109 (25.35)	105 (24.42)	78 (18.14)	
Child BMI, kg/m^2, median(IQR)	16.36 (14.83- 18.46)	16.54 (15.02- 18.76)	17.22 (15.30- 19.64)	18.01 (15.85- 20.39)	< 0.001
Child's age, median (IQR)	10 (8- 13)	10 (8- 13)	11 (9- 13)	12 (10- 15)	< 0.001
Child sex, n (%)					< 0.001
Male	349 (19.06)	442 (24.14)	520 (28.40)	520 (28.40)	
Female	559 (31.06)	470 (26.11)	394 (21.89)	377 (20.94)	
Daily outdoor activity duration, hours, median (IQR)	0.29 (0.11- 0.64)	0.29 (0.14- 0.71)	0.29 (0.14- 0.71)	0.29 (0.11- 0.64)	0.468
Passive smoking, n (%)					0.375
No	486 (25.19)	484 (25.09)	466 (24.16)	493 (25.56)	
Yes	422 (24.91)	428 (25.27)	445 (26.27)	399 (23.55)	

interval 0.90%—2.23%), 2.26% (1.15—3.38) higher level of blood TC and LDL-C, and a 1.49% (-2.35% to -0.62%) lower level of blood HDL-C. Similar patterns persisted when $PM_{2.5}$ exposures were modeled as changes in quartiles with blood TC, and LDL-C levels. The results of restricted cubic spline regression analyses did not support non-linear associations of $PM_{2.5}$ exposure with blood lipids (Supplementary Figure S3). No associations were found between long-term exposure to $PM_{2.5}$ and blood TG.

We examined the effect modification by dietary sodium intake and urinary sodium excretion on the relationships between exposure to $PM_{2.5}$ and blood lipid outcomes. The results of the effect modification analyses by dietary sodium and urinary sodium are shown in Fig. 1 a, b, and Supplementary Material Table S2. We found strong evidence to indicate that a high-sodium diet modified the study associations (*P* for interaction < 0.05). For the participants who were in the highest dietary solidum strata, per 10 μg/m^3 increase in $PM_{2.5}$ exposure was related to a 1.42 (95% *CI* 0.04 to 2.83) higher percentage increase in blood TC levels, a 2.60 (95% *CI* 0.26 to 5.00) higher percentage increase in blood LDL-C levels, and a 2.35 (95% *CI* -4.07 to -0.60) lower percentage decrease in blood

Table 2 The distributions of blood lipid outcomes in children and adolescents stratified by $PM_{2.5}$ exposures. (*N* = 3711)

Blood lipids, mmol/L	$PM_{2.5}$ exposures, μg/m^3				*P*-value
Median (Q1-Q3)	Quartile 1 (22.4-42.8)	Quartile 2 (42.9-54.4)	Quartile 3 (54.5-59.0)	Quartile 4 (59.1-65.0)	
TC	3.58 (3.24- 3.98)	3.68 (3.34- 4.11)	3.72 (3.33- 4.15)	3.72 (3.35- 4.14)	< 0.0001
HDL-C	1.53 (1.31- 1.76)	1.53 (1.31- 1.80)	1.47 (1.27- 1.70)	1.52 (1.31- 1.75)	< 0.0001
LDL-C	1.84 (1.58- 2.15)	1.98 (1.67- 2.31)	1.93 (1.62- 2.32)	2.03 (1.68- 2.38)	< 0.0001
TG	0.77 (0.62- 1.00)	0.79 (0.61- 1.04)	0.80 (0.62- 1.06)	0.78 (0.63- 0.99)	0.0704

Table 3 The associations of exposure to $PM_{2.5}$ with blood lipid outcomes in children and adolescents.[a] (*N* = 3711)

$PM_{2.5}$ exposures	Adjusted percentage changes (95% CI)			
	TC	HDL-C	LDL-C	TG
Per 10 μg/m^3	1.56 (0.90, 2.23)*	-1.49 (-2.35, -0.62)*	2.26 (1.15, 3.38)*	0.08 (-1.43, 1.62)
Quartiles, quartile 1	Reference	Reference	Reference	Reference
Quartile 2	3.20 (1.50, 4.92)*	0.75 (-1.47, 3.03)	4.97 (2.11, 7.91)*	-0.16 (-3.94, 3.78)
Quartile 3	4.43 (2.73, 6.15)*	-3.32 (-5.43, -1.16)*	5.39 (2.56, 8.30)*	3.93 (0.04, 7.97)
Quartile 4	4.46 (2.52, 6.43)*	-1.00 (-3.47, 1.53)	5.95 (2.70, 9.30)*	-1.07 (-5.29, 3.34)
P for trend	< 0.0001	0.0044	< 0.0001	0.8323

* *P* < 0.05

[a] Adjusted for region, father's ethnicity, father's occupation, father's education, child's age, child's sex, child's BMI, childhood daily average outdoor physical activity duration and passive smoking

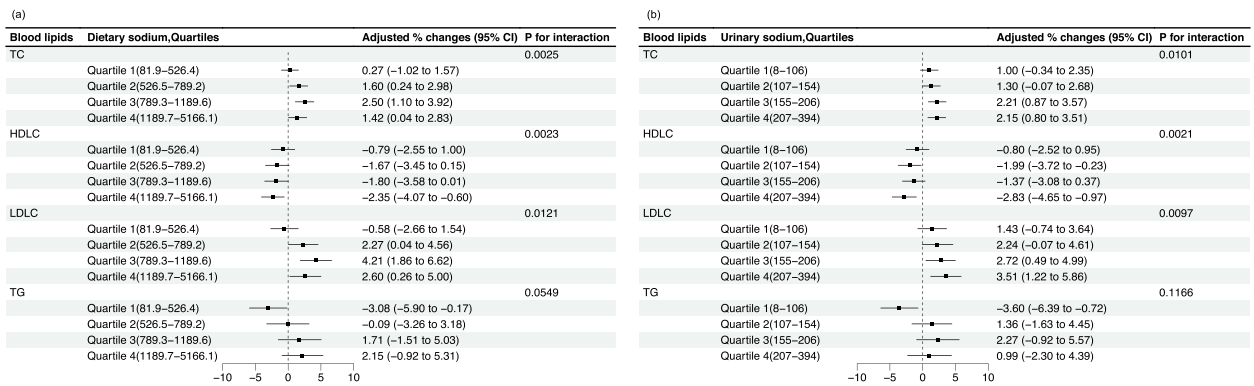

Fig. 1 The associations of $PM_{2.5}$ with blood lipids stratified by: (**a**) dietary sodium(*N* = 3635), (**b**) urinary sodium (*N* = 3631)

HDL-C levels. However, these relationships between $PM_{2.5}$ exposure and deteriorated lipid profiles changed to non-significant among the participants who were from the lowest quartile of dietary sodium levels. The similar associations were observed in replicating the main analyses by different urinary sodium quartiles, which yield pronounced associations of exposure to higher $PM_{2.5}$ levels with deteriorated blood TC, HDL-C, and LDL-C in the participants who were from the highest urinary sodium quartile strata. The associations changed to be nonsignificant in the participants who were from the lowest quartile level of urinary sodium. Although the stratified analyses documented negative associations of $PM_{2.5}$ exposure with blood TG levels among the participants from the lowest quartile of sodium level, the results of significant tests did not support the difference (*P* for interaction > 0.05).

In the sensitivity analyses, when dietary fat intake and dietary energy intake were included in the model, the results remained little changes (Fig. 2 and Supplementary Material Table S3). When excluded the participants whose parents had diabetes or hypertension, there were also little changes in the results (Fig. 2 and Supplementary Material Table S4). When we considered the variations caused by urinary creatinine, the results of association analyses of $PM_{2.5}$ with blood lipids stratified by creatinine-adjusted urinary sodium levels were similar to the main results (Supplementary Material Figure S4).

Discussion

By employing a cross-sectional data with multi-community population, we explored the relationships of long-term $PM_{2.5}$ exposure with blood lipids in children and adolescents. The results showed that exposure to higher levels of ambient $PM_{2.5}$ was related to higher blood TG, LDL-C, and lower HDL-C levels. Most importantly, we provided consistent evidence to support the effect modification of a high-sodium diet on the association between $PM_{2.5}$ exposure and blood lipids in children and adolescents.

Our findings that $PM_{2.5}$ exposure may lead to the deteriorated lipid profiles were consistent with existing evidence. A study in Iran involving 1413 adolescents found that air quality index was positively correlated with blood TC, blood LDL-C and blood TG, and negatively correlated with blood HDL-C. [25]. Another Iranian study which involved 186 children and adolescents from 10 to 18 years of age showed positive correlations between $PM_{2.5}$ exposure levels and blood LDL-C or TG levels [26]. Similarly, a cross-sectional survey of China involving 12,814 children aged from 7 to 18, showed that the children and adolescents who were chronically exposure to $PM_{2.5}$, had higher blood TC levels [14]. In our study, featured by a multi-ethnic Chinese population, we also demonstrated that there were associations between exposure to higher levels of ambient $PM_{2.5}$ surrounding school and deteriorated lipid profiles, including higher blood TC, higher blood LDL-C, and lower blood HDL-C levels in children and adolescents,. Our study highlighted the importance of reducing the school-level $PM_{2.5}$ exposure on benefit blood lipids in children and adolescents.

Although several studies have highlighted the modification of diet on the associations of air pollution situation and prevalence and mortality of cardiovascular disease in adults and elderly [27, 28], few studies have examined the role of dietary sodium underlying the association between atmospheric pollutants and blood lipids in children and adolescents. In this study, based on dietary estimates and urinary biomarkers, we provided consistent evidence that a lower sodium diet could reduce the toxic association of $PM_{2.5}$ exposure on blood lipids in children and adolescents.

The modifcations of sodium levels on the association between $PM_{2.5}$ and blood lipids can be explained by several mechanisms. It is well known that a high-sodium diet could induce the oxidative stress responses and inflammatory that lead to increased risk of cardiovascular

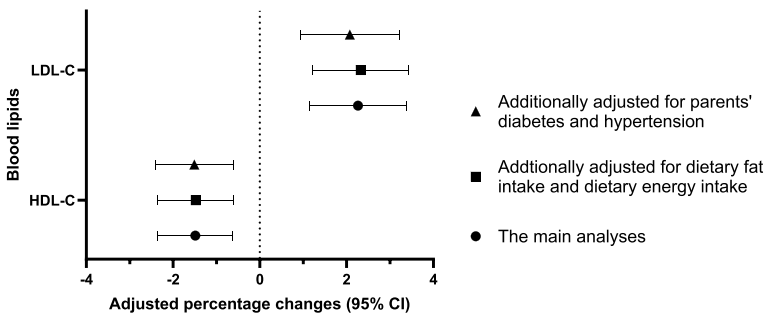

Fig. 2 Results of sensitivity analyses for $PM_{2.5}$ and lipids[a]. **a** The main analyses (*N*=3711), additionally adjusted for dietary fat intake and dietary energy intake (*N*=3711), and additionally adjusted for parents' diabetes and hypertension prevalence (*N*=3475)

diseases [24]. A number of studies have explained that exposure to high concentrations of $PM_{2.5}$ may disrupt lipid metabolism through pro-oxidative and pro-inflammatory effects [29–31]. Therefore, we can speculate that a lower sodium diet modulated the correlations between $PM_{2.5}$ exposure and blood lipids by reducing the $PM_{2.5}$ exposure related pro-oxidative and pro-inflammatory effects. Since the important role of abnormal lipid levels in childhood in predicting the subsequent risks of incident ASCVDs in adulthood, our findings provide a new insight to treat the low-sodium diet as a potential preventive method to reduce the risk of incident ASCVDs attributed to $PM_{2.5}$ exposure in children and adolescents.

There are some strengths and limitations in our study. We explored the relationships between long-term $PM_{2.5}$ exposure and blood lipid outcomes in children and adolescents based on 3711 participants who provided measurements of four blood lipid outcomes. We adjusted for many confounders such as family-socio-economic differences, passive smoking status, child's BMI, and outdoor physical activity. We estimated the effect modification of dietary sodium on the study associations using semi-quantitative FFQ methods and urinary internal biomarkers related to sodium consumption, and the results indicated strong dose–response patterns. However, there are still several limitations that need to be declared. First, the nature of cross-sectional study prevented us from establishing causal relationships for the study associations. Second, limited by the data availability, we only evaluated the long-term exposure of $PM_{2.5}$ based on schools, which missed the exposures from residence. In addition, a family's cooking and eating habits may affect a child's dietary sodium intake, and we did not collected these information, which may influenced the conclusions.

Conclusions

Exposure to higher levels of $PM_{2.5}$ is associated with deteriorated lipid profiles in children and adolescents. These associations became null in the participants who maintained a low-sodium diet. A low dietary sodium diet could mitigate the $PM_{2.5}$ related damage on blood lipids in children and adolescents.

Abbreviations

$PM_{2.5}$	Fine particulate matter
TC	Total cholesterol
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
TG	Triglycerides
FFQ	Food frequency questionnaire
ASCVDs	Atherosclerotic cardiovascular diseases
SS	Systematic sampling
SRS	Simple random sampling
CDC	Center for Disease Control and Prevention
DUACAG	Dalhousie University Atmospheric Composition Analysis Group
AOD	Aerosol optical depth
MISR	Multiangle imaging spectroradiometer
BMI	Body mass index
DAG	Direct acyclic graphic

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12887-024-04896-8.

Supplementary Material 1.

Supplementary Material 2.

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Authors' contributions

YH C completed the main analyses of the article and wrote the manuscript. YJ L, R Z, YD J and YR C collated the data and the results of the analyses. JR C checked and revised the manuscript. MT M, MM L and L Y collected data on the research participants. ZM L and JQ L proposed research ideas and provided guidance and assistance during the research process. All authors read, revised, and approved the final manuscript.

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Availability of data and materials

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

Declarations

Ethics approval and consent to participate

All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The protocol was reviewed and approved by the Ethics Committee of the Chinese Center for Disease Control and Prevention (CDC). A well-written informed consent form to participate in the study was signed by the guardian of each study participant.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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