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Association of $PM_{2.5}$ chemical constituents with general, abdominal and visceral obesity and mediation roles of physical activity

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Abstract

Background The association between exposure to $PM_{2.5}$ chemical constituents and obesity remains to be elucidated, as most studies have used a single measure of obesity. This study aimed to investigate the associations of exposure to $PM_{2.5}$ chemical constituents with general, abdominal, and visceral obesity, and the mediation effect of physical activity (PA) in the associations.

Methods Based on a total of 49,819 adults from the baseline of the Yunnan Behavior and Disease Surveillance cohort (YBDS) in southern China in 2021, we used multiple linear regression, weighted quantile sum regression, and quantile regression to estimate independent and joint effects of $PM_{2.5}$ chemical constituents on elevated BMI, waist circumference (WC) and visceral adiposity index (VAI) and whether these effects changed in quantiles. Mediation analysis was used to examine whether physical activity acts as a mediator in these pathways.

Results Per IQR μg/m^3 increase in all $PM_{2.5}$ chemical constituents was significantly associated with the elevated BMI (β [95% CI]: 0.170 [0.127, 0.214]), WC (0.316 [0.217, 0.415]) and VAI (0.102 [0.075, 0.129]), with the largest weights from OM (53.89, 81.67, and 89.82%, respectively). The effects of $PM_{2.5}$ chemical constituents on obesity showed an overall upward trend from quantiles 1–4 of BMI, WC, and VAI, especially with a rapid upward trend from the sixth decile of VAI. Reduced PA mediated 3.16, 7.08, and 3.78% of the associations between $PM_{2.5}$ chemical constituents and elevated BMI, WC, and VAI, respectively.

Conclusions Exposure to $PM_{2.5}$ chemical constituents, especially OM, was significantly associated with increased risks for obesity in adults. The effects of associations increased with obesity severity, with PA playing a mediation role.

Keywords $PM_{2.5}$, Obesity, Physical activity, Weighted quantile sum regression

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Introduction

Obesity, a complex metabolic disorder, is a major risk factor for many non-communicable diseases such as diabetes, hypertension, and cardiovascular diseases [1, 2]. The prevalence of adult obesity was 13.1% globally and reached 28.6% in the Americas in 2016 according to the latest World Health Organization (WHO) statistics [3]. In China, 16.4% of adults were dealing with obesity based on the most recent national data in 2015, with one of the largest obese populations in the world [4]. According to the location and characteristics of fat distribution in the body, obesity can be assessed specifically as general, abdominal, and visceral obesity [5]. Abdominal obesity and visceral obesity are more likely to contribute to a variety of diseases such as hypertension, diabetes, and cardiovascular events, compared with general obesity denoted or defined by body mass index (BMI) [6]. A large number of studies have investigated modifiable environmental determinants of BMI-measured obesity, such as greenness and air pollutants [7, 8], and found that their effects on obesity differ by obesity type [9]. Among them, particulate matter with an aerodynamic diameter < 2.5 μm ($PM_{2.5}$) has been identified as the most persistent environmental risk factor for obesity in recent years [10, 11], but the associations between $PM_{2.5}$ exposure and non-BMI measured obesity, such as abdominal and visceral obesity, remain understudied.

$PM_{2.5}$ is a complex mixture of chemical constituents, including secondary inorganic aerosols (i.e., nitrate [NO_3^-], sulfate [SO_4^{2-}], ammonium [NH_4^+]), organic matter (OM), black carbon (BC), crustal elements, and water, which may have various toxicities and contributions in the development of obesity. A few studies have focused on the effects of $PM_{2.5}$ chemical constituents on obesity to find the predominant constituent. A previous study in children reported that long-term exposure to BC contributed to the risk for childhood general obesity [12]. A recent study in Western China also showed that long-term exposure to $PM_{2.5}$ and its constituents was associated with general obesity among adults [13]. However, these studies mainly focused on a singular dimension of obesity, namely general obesity defined by BMI, neglecting the other obesity dimensions, such as abdominal and visceral obesity. Besides, these studies often assumed that the association between $PM_{2.5}$ exposure and obesity was linear, which ignored the potential nonlinear effects of $PM_{2.5}$ on people with different levels of obesity. An animal experimental study showed that obesity may exacerbate susceptibility to the harmful effects of $PM_{2.5}$ [14], but failed to reveal the heterogeneous effects of $PM_{2.5}$ chemical constituents on different severity of obesity, which may hinder to identify sensitive populations to $PM_{2.5}$ chemical constituents.

The concentrations of $PM_{2.5}$ may affect people's regular health-related behaviors, such as reducing PA and prolonging sedentary behavior [15], which may lead to an increased risk for obesity [2]. Increased outdoor $PM_{2.5}$ may reduce the PA, and then increase the obesity risk. However, previous epidemiological studies only suggested the modification role of PA in the association between $PM_{2.5}$ and obesity risk [16, 17], and few investigated the mediation effects of PA on $PM_{2.5}$ chemical constituents and obesity associations. Besides, it is suggested that weight loss caused by lifestyle changes prioritizes the reduction of visceral adipose tissue compared to subcutaneous adipose tissue [18]. Thus, it is necessary to explore the mediation role of PA in the pathway linking $PM_{2.5}$ chemical constituents with risks for obesity.

To fill the aforementioned research gaps, based on a recently established representative cohort in southwest China. we aimed to investigate the independent and joint effects of long-term exposure to $PM_{2.5}$ chemical constituents on risk for obesity and the mediation effects of PA. Specifically, the effects of $PM_{2.5}$ and its chemical constituents on the risks for general, abdominal, and visceral obesity were examined, as well as the variations of these effects across the degrees of obesity severity. The findings of this study would provide a deeper understanding of atmospheric environmental influences on obesity, and scientific evidence for reducing adverse effects of air pollution.

Materials and methods

Study setting and participants

This is a cross-sectional study based on the baseline of the Yunnan Behavior and Disease Surveillance cohort (YBDS), established in 35 counties in Yunnan province of southwest China, between January and November 2021. The cohort was designed for research on chronic diseases and their risk factors. A multistage, cluster, and random sampling process was used to recruit participants from the general population. First, 1–4 districts/counties were randomly selected from each of the 16 prefecture-level cities in the province, and 35 districts/counties were selected in total. Second, eight communities were randomly selected from each of the selected districts/counties. Finally, at least 90 households were randomly chosen in each selected community, and only one family member was invited to participate in the study. A total of 51,480 participants from 35 districts/counties were randomly enrolled in the following eligibility criteria: (1) aged ≥ 18 years on the date of the survey, and (2) being permanent residents in the selected communities.

The excluded participants were participants who had severe physical and mental diseases that hindered them from participating in the survey, such as paraplegia, schizophrenia, and terminal illnesses. In this study, 1661 participants who had incomplete residential addresses, sociodemographic information, or missing information on general, abdominal, and visceral obesity were excluded. Further detailed information is shown in Fig. 1.

The YBDS was approved by the medical ethics committee of the Yunnan Center for Disease Prevention and Control (202017), and was carried out in accordance with the Helsinki Declaration of 1964. All participants signed informed consent before the survey.

Outcome variables

We used BMI, waist circumference (WC), and visceral adiposity index (VAI) to describe general, abdominal, and visceral obesity, respectively, which had been widely used in the literature [5, 19, 20]. Anthropometric and laboratory tests were performed by qualified medical practitioners according to standard protocols. All participants underwent a comprehensive anthropometric examination (i.e., body weight, body height, and WC) and laboratory tests. Measurements of body weight, height, and WC were taken in light clothing and without shoes. Weight was measured to a precision of 0.1 kg and height and WC were measured accurately to 0.1 cm. The mean value of three repetition measurements was used for analysis. For the blood biochemical test, fast venous blood was collected after an overnight fast of at least 8 h, using vacuum blood collection tubes and blood lancets. Biochemical indicators, including high-density lipoprotein cholesterol (HDL) and triglycerides (TG), were used for the measurement of VAI. Overall, BMI was calculated as the body weight (kg) divided by the height squared (m^2). WC was the average of three measurements recorded for each participant. VAI was calculated for women and men according to the formulas: $VAI = (WC\ [cm]/39.68 + 1.88 \times BMI\ [kg/m^2]) \times (TG\ [mmol/L]\ /\ 1.03) \times (1.31/HDL\text{-}C\ [mmol/L])$ for men; and $VAI = (WC\ [cm]/36.58 + 1.89 \times BMI\ [kg/m^2]) \times (TG\ [mmol/L]/0.81) \times (1.52/HDL\text{-}C\ [mmol/L])$ for women [5].

Concentrations of $PM_{2.5}$ and chemical constituents

The average concentrations of $PM_{2.5}$ and chemical constituents, including NO^{3-}, SO_4^{2-}, NH_4^+, OM, and BC, around participants' residences in the past three years (2019–2021) before the survey were considered as exposure variables, with a spatial resolution of 10 km. The annual average concentrations of $PM_{2.5}$ and chemical constituents were extracted from the "Tracking Air Pollution in China" database (http://tapdata.org.cn) during 2019–2021. These data were generated by a series of machine learning models (e.g., Weather Research and Forecasting—Community Multiscale Air Quality model, and extreme gradient boosting models)

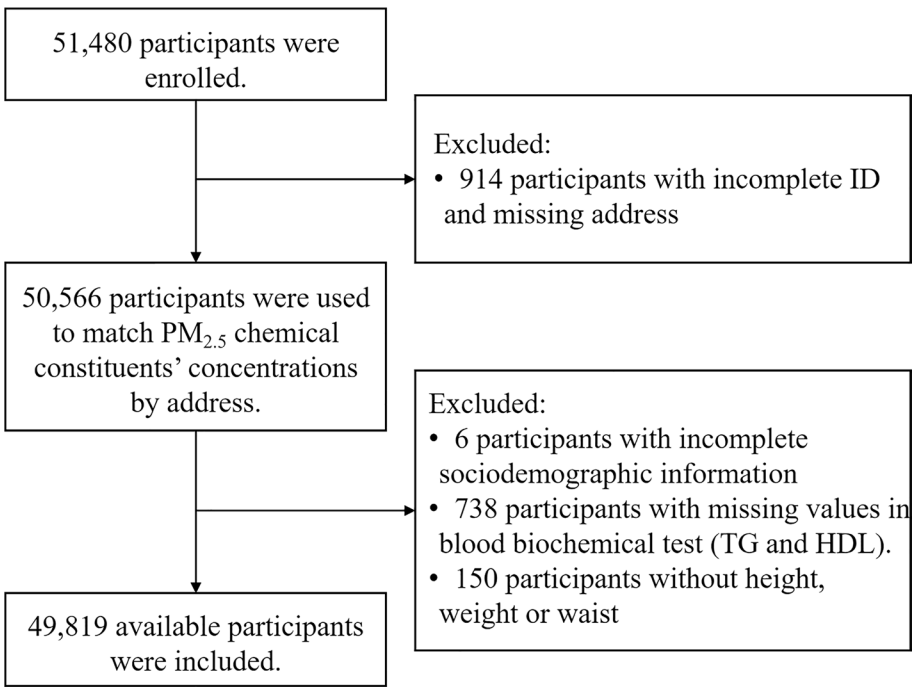

Fig. 1 Flowchart of the study population

based on satellite-derived aerosol optical depth (AOD), on-site monitoring data of $PM_{2.5}$ chemical constituents, and other ancillary data, such as elevation, land use data, population, and meteorological fields. The degree of agreement between the modeled values and actual observed daily concentrations was considered acceptable, with correlation coefficients ranging from 0.67 to 0.80 [21].

Mediator

Metabolic equivalent (MET) was used to quantify PA as the mediator in our study. The measurement of participants' PA is based on the International Physical Activity Questionnaire (IPAQ) [22], including occupation, transportation, housework, and leisure physical activity, and individual physical activities frequency and duration usually in a week were collected. MET is the ratio of the energy consumed by an individual during one hour of activity to the energy consumed during one hour of sitting, where one hour of sitting behavior equals one MET [23]. Different types and intensities of PA have different MET values. Based on the MET assignment of the IPAQ questionnaire [24], the daily MET for PA was calculated as ([Weekly frequency of high-intensity physical activity × Duration of each activity × 8] + [Frequency of moderate intensity physical activity × Duration of each activity × 4])/7.

Covariates

A series of covariates included sociodemographic characteristics, lifestyle behaviors, and temperature based on previous studies and Directed Acyclic Graphs [11, 25, 26]. All of these were selected based on knowledge of common causal precedents for both exposure and outcomes. Sociodemographic characteristics included sex (male, female), age (18–44, 45–59, 60–99 years), educational level (primary school or below, junior high school or above), occupation (farmer, employed, unemployed), and marital status (married, unmarried). Lifestyle behaviors included smoking status (yes, no), drinking status (yes, no), and dietary habits. Dietary habits were classified by median daily intake of vegetables, fruit, and meat, respectively. Additionally, the 3-year average of temporary (°C) before the survey was considered a covariate, which was obtained from the National Earth System Science Data Center, National Science & Technology Infrastructure of China (http://www.geodata.cn). When the outcome was WC, the models further included height.

Statistical analysis

Categorical data were summarized by number and percentage, while continuous data were shown as mean (standard deviation [SD]) or median (25th percentile, 75th percentile). Spearman's rank correlation coefficients were used to estimate the correlation between SO_4^{2-}, NO_3^-, NH_4^+, BC, and OM.

Multiple linear regression models were used to assess the independent effects of each $PM_{2.5}$ chemical constituent on the risks for elevated BMI, WC, and VAI, respectively. Then, weighted quantile sum (WQS) regression was further applied to test the joint effects of all $PM_{2.5}$ chemical constituents. Under the assumption that all $PM_{2.5}$ chemical constituents had positive effects on obesity, WQS regression could determine the relative importance of each $PM_{2.5}$ chemical constituent in its association with the risks for elevated BMI, WC, and VAI, and obtain a weighted index of each constituent [21]. The effect estimates were expressed as beta (β) and 95% confidence intervals (CIs) of per IQR $\mu g/m^3$ increase in the 3-year average concentration of $PM_{2.5}$ chemical constituents. Moreover, quantile regression models were used to assess the heterogeneous effects of $PM_{2.5}$ chemical constituents on increasing risks of obesity indicators in quantiles.

We further used mediation analysis to examine the potential mediation effect of PA in the association between $PM_{2.5}$ chemical constituents and elevated BMI, WC, and VAI. In this approach, the "total effect" can be decomposed into a "direct effect" (not mediated by PA) and an "indirect effect" (mediated by PA).

To assess the robustness of our results, we conducted a series of sensitivity analyses, including: (1) using quantile g-computation to estimate the joint effect of all constituents and the weight of each constituent. (2) excluding participants who had taken lipid-lowering medications, as the medication could render their measurements incapable of reflecting natural levels. (3) using obesity categorized by the criteria of China and the WHO, to be more correlated with general obesity. (4) adjusting the prefecture-level cities as a random intercept in multi-level linear regression models, to rule out potential influences of the variations across those sites. (5) performing mediation analysis stratified by sex (male vs female) g to identify potential modifying effects on the mediation effects of PA.

The significance level was 0.05 for two-sided tests. The R software (version 4.2.2) was used to conduct all analyses.

Results

Characteristics of study participants

Most of the 49,819 participants, aged 54.09 ± 13.58 years on average, were females (56.4%), married (89.8%), farmers (61.7%), and had an educational level of elementary school or below (66.5%). The 32.5% and 31.3%

of the participants were smokers and alcohol drinkers, respectively. There were significant differences in their sociodemographics, lifestyle behaviors, and obesity outcomes between exposure to higher and lower $PM_{2.5}$ concentrations, except for marital status and alcohol drinking (P value > 0.05) (Table 1). The mean BMI, WC, and VAI were 24.09 ± 3.77 kg/m^2, 81.68 ± 9.99 cm, and 2.43 ± 2.70, respectively.

The median concentrations of NO_3^-, SO_4^{2-}, NH_4^+, OM, and BC over the three years were 3.00, 4.41, 2.96, 6.02, and 1.31 μg/m^3, respectively (Table S1). High correlations were found between $PM_{2.5}$ chemical

Table 1 Baseline characteristics of study participants

Variables	Overall	Percentage (%) or mean (SD)		*P*-value
		$PM_{2.5}$ concentration, μg/m^3		
		[13.00–19.03][a]	[19.03–31.33]	
	n = 49,819	n = 24,774	n = 25,045	
Sociodemographics				
Age, years				
18–44	11,827 (23.7)	6,469 (26.1)	5,358 (21.4)	< 0.001
45 ~ 59	20,804 (41.8)	10,503 (42.4)	10,301 (41.1)	
60–99	17,188 (34.5)	7,802 (31.5)	9,386 (37.5)	
Sex				
Male	21,723 (43.6)	11,138 (45.0)	10,585 (42.3)	< 0.001
Female	28,096 (56.4)	13,636 (55.0)	14,460 (57.7)	
Education level				
Elementary school or below	33,125 (66.5)	16,609 (67.0)	16,516 (65.9)	0.010
Junior high school or above	16,694 (33.5)	8,165 (33.0)	8,529 (34.1)	
Marital status				
Unmarried	5,097 (10.2)	2,514 (10.1)	2,583 (10.3)	0.552
Married	44,722 (89.8)	22,260 (89.9)	22,462 (89.7)	
Occupation				
Unemployed	10,860 (21.8)	4,740 (19.1)	6,120 (24.4)	< 0.001
Employed	8,209 (16.5)	3,143 (12.7)	5,066 (20.2)	
Farmer	30,750 (61.7)	16,891 (68.2)	13,859 (55.4)	
Lifestyle behaviors				
Smoking				
No	33,652 (67.5)	16,854 (68.0)	16,798 (67.1)	0.023
Yes	16,167 (32.5)	7,920 (32.0)	8,247 (32.9)	
Alcohol drinking				
No	34,228 (68.7)	17,026 (68.7)	17,202 (68.7)	0.929
Yes	15,591 (31.3)	7,748 (31.3)	7,843 (31.3)	
MET (h/day)	25.03 (25.27)	26.94 (26.08)	23.14 (24.30)	< 0.001
Vegetable intake (g/day)	369.97 (340.24)	360.48 (351.86)	379.36 (328.07)	< 0.001
Fruit intake (g/day)	119.97 (233.32)	111.75 (194.52)	128.10 (265.95)	< 0.001
Meat intake (g/day)	153.19 (215.69)	163.09 (225.95)	143.38 (204.58)	< 0.001
3 year average temperature, °C	10.38 (6.58)	7.40 (7.27)	13.32 (4.04)	< 0.001
Height, cm	158.08 (8.29)	158.28 (8.37)	157.89 (8.22)	< 0.001
Outcome Variables				
BMI, kg/m^2	24.09 (3.77)	23.97 (3.82)	24.21 (3.72)	< 0.001
WC, cm	81.68 (9.99)	81.58 (10.03)	81.78 (9.95)	0.031
VAI	2.43 (2.70)	2.36 (2.58)	2.50 (2.81)	< 0.001

[a] The participants were divided into two groups based on the median concentration of exposure to $PM_{2.5}$

BC black carbon, *BMI* body mass index, NH_4^+ ammonium, NO_3^- nitrate, *OM* organic matter, $PM_{2.5}$ particulate matter with an aerodynamic diameter ≤ 2.5 μm, SO_4^{2-} sulfate, *VAI* visceral adiposity index, *WC* waist circumference

constituents, ranging from 0.60 to 0.94, with the strongest correlation observed between NO_3^- and NH_4^+ (0.94) (Fig. S1).

Associations between $PM_{2.5}$ chemical constituents and obesity

For the joint effects of $PM_{2.5}$ chemical constituents, per IQR μg/m^3 increase in all $PM_{2.5}$ chemical constituents was significantly associated with elevated BMI (β [95% CI]: 0.170 [0.127, 0.214]), WC (β: 0.316 [0.219, 0.413]) and VAI (β: 0.102 [0.075, 0.129]) (Table 2). The largest contribution to joint effects of $PM_{2.5}$ chemical constituents on elevated BMI (weight: 53.89%, β [95%CI]: 0.120 [0.085, 0.156]), WC (weight: 86.54%, β: 0.303 [0.211, 0.396]) and VAI (weight: 89.82%, β: 0.096 [0.071, 0.122]) was from OM. Besides, we also observed that NH_4^+ had a high weight contribution for the increased risks for elevated BMI (weight: 43.36%, β: 0.110 [0.074, 0.145]), and SO_4^{2-} for WC (weight: 13.07%, β: 0.215[0.122, 0.309]).

Table 2 Independent and joint effects of per IQR increase in $PM_{2.5}$ chemical constituents' concentrations on BMI, WC and VAI

Outcome	$PM_{2.5}$ chemical constituents	β (95%CI)	WQS index weight, %
BMI, kg/m^2	NO_3^-	0.090 (0.055, 0.124)***	0.43
	SO_4^{2-}	0.092 (0.056, 0.128)***	0.45
	NH_4^+	0.110 (0.074, 0.145)***	43.36
	OM	0.120 (0.085, 0.156)***	53.89
	BC	0.095 (0.061, 0.129)***	1.87
	Joint effect	0.170 (0.127, 0.214)***	100.00
WC, cm	NO_3^-	0.011 (– 0.079, 0.102)	0.15
	SO_4^{2-}	0.215 (0.122, 0.309)***	13.07
	NH_4^+	– 0.171 (– 0.264, – 0.078)***	0.13
	OM	0.303 (0.211, 0.396)***	86.54
	BC	0.113 (0.024, 0.202)**	0.11
	Joint effect	0.316 (0.219, 0.413)***	100.00
VAI	NO_3^-	0.047 (0.022, 0.072)***	3.61
	SO_4^{2-}	0.042 (0.016, 0.068)**	< 0.01
	NH_4^+	0.038 (0.013, 0.064)***	6.37
	OM	0.096 (0.071, 0.122)***	89.82
	BC	0.051 (0.026, 0.076)**	0.20
	Joint effect	0.102 (0.075, 0.129)***	100.00

Independent and joint effects were modeled by multiple linear regression and weighted quantile sum regression, respectively. The weights of $PM_{2.5}$ chemical constituents were estimated by the weighted quantile sum regression. Covariates adjust for sex, age, marital status, educational level, occupation, smoking, alcohol drinking, fruit intake, vegetable intake, meat intake and 3-year average temperature. When the outcome was WC, the models further included height

BC black carbon, *BMI* body mass index, NH_4^+ ammonium, NO_3^- nitrate, *OM* organic matter, $PM_{2.5}$ particulate matter with an aerodynamic diameter ≤ 2.5 μm, SO_4^{2-} sulfate, *VAI* visceral adiposity index, *WC* waist circumference

$^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$

The association between $PM_{2.5}$ chemical constituents and the risks for general, abdominal, and visceral obesity varied with quantiles of BMI, WC, and VAI (Fig. 2). Specifically, we observed that the effect of each $PM_{2.5}$ chemical constituents on the risks for general obesity gradually elevated with the raised quantiles of BMI, and the effect tended to be stable after increasing to the fifth decile, except for effect of BC that maintained a stable trend. Besides, the association between each PM2.5 chemical constituent and abdominal obesity showed a stable upward trend with raised quantiles of WC. The associations between $PM_{2.5}$ chemical constituents and quantiles of VAI were J-shaped curves, with a rapid upward trend from the sixth decile of VAI. The joint effect of $PM_{2.5}$ chemical constituents showed an upward trend with the raised quantiles of BMI, waist circumference, and VAI.

Mediation effect of PA

The results of mediation analysis showed that reduced MET of individual PA mediated 3.16%, 6.48% and 3.78% of the associations between joint of $PM_{2.5}$ chemical constituents and elevated BMI (β [95% CI]: 0.005 [0.003, 0.008]), WC (β: 0.020 [0.014, 0.027]) and VAI (β: 0.004 [0.003, 0.005]), respectively (Fig. 3). For the independent effects of each $PM_{2.5}$ chemical constituents (i.e., NO_3^-, SO_4^{2-}, NH_4^+, OM and BC) on elevated BMI, WC and VAI, reduced MET mediated 2.47% to 22.92% of the effects (Table S6).

Sensitivity analysis

In the QG-computation model, per IQR increase in all $PM_{2.5}$ chemical constituents was associated with

(See figure on next page.)

Fig. 2 Associations between exposure to $PM_{2.5}$ chemical constituents and quantiles of BMI, WC and VAI The trends of effects were modeled by quantile regression model. The solid line indicates the coefficient that per IQR μg/m3 increase of $PM_{2.5}$ chemical constituents associated with each percentile of BMI, WC and VAI. Covariates adjusted included sex, age, marital status, educational level, occupation, smoking, alcohol drinking, fruit intake, vegetable intake, meat intake and 3 year average temperature. When the outcome was WC, the models further included height. The colored areas represent the 95% confidence interval of the estimated value. *BC* black carbon, *BMI* body mass index, NH_4^+ ammonium, NO_3^- nitrate, *OM* organic matter, $PM_{2.5}$ particulate matter with an aerodynamic diameter ≤ 2.5 μm, SO_4^{2-} sulfate, *VAI* visceral adiposity index, *WC* waist circumference. $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$

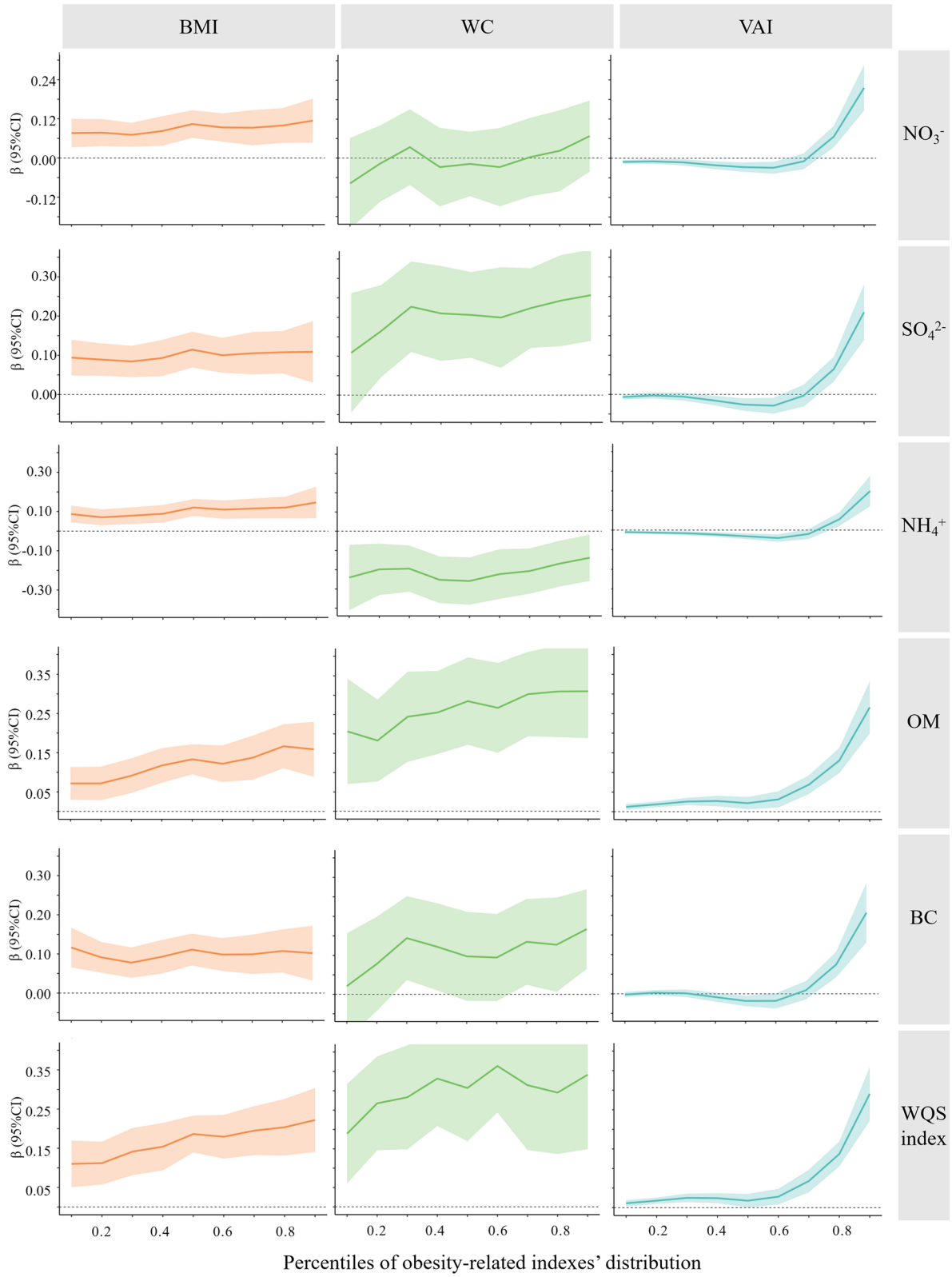

Fig. 2 (See legend on previous page.)

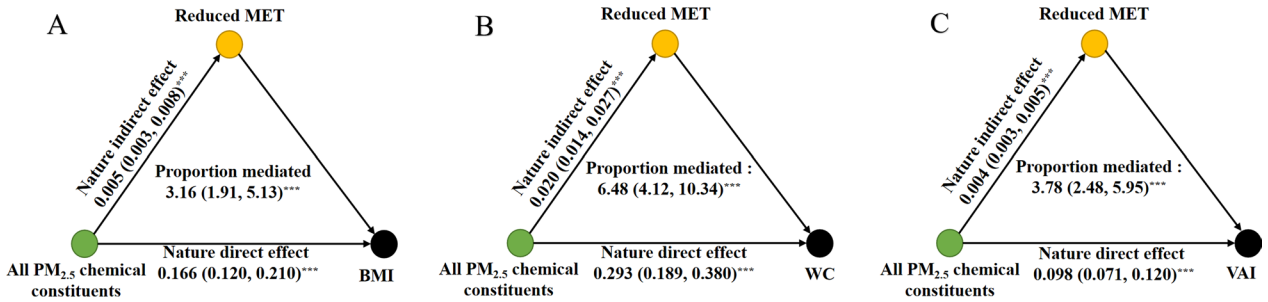

Fig. 3 Mediation effects of reduced MET on the associations between exposure to all $PM_{2.5}$ chemical constituents and BMI, WC and VAI WQS indexes obtained from the corresponding WQS regression model are used to represent the weighted total concentration of each component of $PM_{2.5}$. Figure **A** represents the reduced MET as a mediator on the causal pathway between the joint of $PM_{2.5}$ chemical constituents and BMI. Figure **B** represents the reduced MET as a mediator on the causal pathway between the joint of $PM_{2.5}$ chemical constituents and WC. Figure **C** represents the reduced MET as a mediator on the causal pathway between the joint of $PM_{2.5}$ chemical constituents and VAI. Covariates adjusted included sex, age, marital status, educational level, occupation, smoking, alcohol drinking, fruit intake, vegetable intake, meat intake and 3 year average temperature. When the outcome was WC, the models further included height. *BMI* body mass index, *MET* metabolic equivalent, *$PM_{2.5}$* particulate matter with an aerodynamic diameter ≤ 2.5 μm, *VAI* visceral adiposity index *WC* waist circumference $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$

elevated BMI (β [95% CI]: 0.186 [0.143, 0.230]), WC (β: 0.168 [0.055, 0.281]) and VAI (β: 0.100 [0.070, 0.132]), and OM contributed greatly to the association, which were robust with the main results (Table S2). After excluding participants who had had taken lipid-lowering medications, categorizing BMI into obesity, and accounting for the random effect of city-level, the results were still stable (Tables S3, TableS4, and Table S5). The mediation effects of PA remained robust in males and females (Table S7).

In the QG-computation model, per IQR increase in all PM2.5 chemical constituents was associated with elevated BMI (β [95% CI]: 0.186 [0.143, 0.230]), WC (β: 0.168 [0.055, 0.281]) and VAI (β: 0.100 [0.070, 0.132]), and OM contributed greatly to the association, which were robust with the main results (Table S2). After excluding participants who had had taken lipid-lowering medications, categorizing BMI into obesity, and accounting for the random effect of city-level, the results were still stable (Tables S3, TableS4, and Table S5). The mediation effects of PA remained robust in males and females (Table S7).

Discussion

To the best of our knowledge, this is the first study to reveal that long-term exposure to $PM_{2.5}$ chemical constituents increased the risks for general, abdominal, and visceral obesity in Chinese adults, and OM played a dominant role. The independent and joint effects of $PM_{2.5}$ chemical constituents were greater in participants with more severe obesity. Furthermore, reduced PA of individuals played a partial mediation effect in the association between $PM_{2.5}$ chemical constituents and obesity.

We found that exposure to $PM_{2.5}$ chemical constituents was associated with risks for general, abdominal, and visceral obesity in the general population. Growing evidence had shown that air pollutants were important risk factors for the development of obesity. A meta-analysis included 19 studies worldwide, and the combined results showed that per 10 μg/m^3 increase in $PM_{2.5}$ increased 1.15 odds of the risk for obesity [10]. A recent study found that long-term exposure to $PM_{2.5}$ and its constituents were positively associated with obesity in Chinese adults [13]. Although these studies mainly focused on general obesity defined by BMI, they also mentioned some possible reasons for explaining the effects of $PM_{2.5}$ on other measurements of obesity. First, long-term exposure to $PM_{2.5}$ could affect individual blood lipid levels, leading to a decrease in HDL and an increase in TG [27], which were important components of VAI [5]. Second, $PM_{2.5}$ regulated visceral adipose tissue inflammation, liver lipid metabolism, and glucose utilization in skeletal muscles through CC-chemokine receptor 2 (CCR2)-dependent and -independent pathways [28]. Besides, $PM_{2.5}$ could cause hepatic lipid metabolism abnormality through inducing circadian rhythm disturbance, and disrupt the balance of lipid metabolism [29], which might lead to visceral obesity.

We observed that NH_4^+ had a high weight contribution to the increased risks for general obesity, which was consistent with previous research findings in another adult populations [13]. Water-soluble molecules are more likely to dissolve in the moist alveolar walls. It was evidenced by animal and cell experiments that exposure to NH_4^+ leads to the accumulation of neutral lipid droplets and dysregulation of lipid metabolism [30]. We also found that OM played a dominant role in the risks

for general, abdominal, and visceral obesity, which may be the primary obesogenic constituent in adults. It has not been found in previous studies defining obesity by BMI. However, previous studies have found that OM shows a higher adverse effect on obesity (defined by BMI), dyslipidemia, and metabolic syndrome, which is consistent with our findings [13, 31, 32]. This suggests that OM, a fat-soluble substance, may have a more obvious effect on adipose tissue. The possible reasons may be explained by the following mechanisms. First, OM was mainly derived from the combustion emission of fossil fuels (e.g., oil and coil). Organic aerosols are complex mixtures, among which polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs) are known for their harmful health effects [33]. PAHs are lipophilic and may be absorbed and accumulated by organisms. PAHs directly promote adipocyte proliferation differentiation and endocrine function, thus leading to obesity [34]. Second, OM accumulates in adipose tissue induced inflammation-related genes and increases the secretion of chemokines CXLC8 and MMP1, exaggerating adipose tissue inflammation [35].

Besides, we found that the joint effect of $PM_{2.5}$ chemical constituents showed an upward trend with the raised quantiles of these obesity-related indexes, especially for WC and VAI, indicating that participants with obesity exhibited heterogeneity in the effects of exposure to $PM_{2.5}$ chemical constituents. Previous epidemiological studies have found that individuals with chronic diseases, including diabetes [36, 37], cardiovascular events [38], and hypertension [39], were more susceptible to the adverse effects of $PM_{2.5}$. The possible mechanism could be that the impaired neuroendocrine regulation of homeostasis would lead to the more susceptible to the impact of air pollution in the presence of other stressors, such as obesity [40]. For example, exposure to $PM_{2.5}$ caused vasoconstriction in obese mice, while such an effect was not observed in lean mice [14]. Besides, increased tidal volume among obese individuals might lead to an increase in the amount of particulate matter inhaled [41]. Our results indicated that people with obesity, especially those with abdominal obesity and visceral obesity, are more susceptible to the effects of $PM_{2.5}$ chemical constituents. Fat distribution more than overall body weight is a key determinant of the risk for cardiovascular disease [42]. Therefore, it seems to be unreasonable that we should continue to focus on general obesity defined by BMI in future research. This also provided evidence to accurately identify sensitive populations and intervene.

We found that reduced PA mediated the association between $PM_{2.5}$ chemical constituents and obesity. Air pollutants may reduce PA and increase sedentary behaviors [15], and thus lead to obesity. This pathway is consistent with some previous studies, which have focused on the interaction of $PM_{2.5}$ and PA on obesity, suggesting that increasing PA can reduce the effects of $PM_{2.5}$ on obesity [16, 17]. Besides, a cross-sectional study in China found that individuals' PA attenuated the effect of long-term exposure to ambient air pollutants on metabolic syndrome [43]. One meta-analysis found that each 1 $\mu g/m^3$ increase in $PM_{2.5}$ concentration was associated with an increase in the odds of physical inactivity by 1.1% [15]. The associations could be explained by respiratory symptoms caused by air pollutants [44], reduced outdoor activities due to smog, and PA decisions affected by media alerts [45, 46]. Our results suggested that PA may reduce or counteract the adverse effects of $PM_{2.5}$ chemical constituents on obesity. Previous studies had suggested similar conclusions, but this could be related to the concentration level of exposure to $PM_{2.5}$ [17, 47]. Therefore, future studies need to be conducted in areas at higher $PM_{2.5}$ concentrations to clarify the mediating role of PA in the causal pathway between $PM_{2.5}$ chemical constituents and obesity.

Several limitations should be noted. First, the cross-sectional design cannot draw causal inferences. Future research using follow-up data are expected to improve the strength of the evidence. Second, although the spatial resolution of $PM_{2.5}$ chemical constituents was 10 km, which was the highest resolution exposure dataset available in China. Besides, the average outdoor concentrations of $PM_{2.5}$ chemical constituents over participants' residences may not well represent the actual exposure level. More accurate measurements of individual exposure should be conducted in future research. Third, our study was conducted in southern China, and may not be well generalized to other parts of the world given the regional variation in $PM_{2.5}$ concentration levels, but it is a good reference for other developing countries. Fourth, we lacked a specific distinguishment between indoor and outdoor physical activities during the questionnaire survey stage, so the confounding bias of the mediating role of PA is inevitable. Further studies are needed to investigate the mediation effects of indoor and outdoor physical activity on the associations between $PM_{2.5}$ constituents and obesity.

In conclusion, we observed that long-term exposure to $PM_{2.5}$ chemical constituents (SO_4^{2-}, NO_3^-, NH_4^+, OM, and BC), particularly exposure to OM, were associated with increased risks for general, abdominal, and visceral obesity in Chinese adults, which were partly mediated by reduced PA. Participants with higher BMI, WC, and VAI showed high sensitivity to

the harmful effects of $PM_{2.5}$ chemical constituents. Our findings provide evidence to accurately identify sensitive populations and may help formulate tailored interventions for general, abdominal, and visceral obesity. Emission reduction of toxic constituents especially OM and increasing physical activity to reduce or counteract adverse effects of $PM_{2.5}$ chemical constituents on obesity.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12302-024-00935-4.

Additional file 1.

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Author contributions
Tingting Ye was responsible for conceptualization, methodology, formal analysis, data curation, writing the original draft, and editing. Ying Shao was responsible for investigation, data curation, and supervision. Changwei Cai was responsible for conceptualization, methodology, formal analysis, and writing the original draft. Yuchen Li was responsible for methodology, formal analysis, review and editing the manuscript. Bin Yu, Xu Qiao and Chuanteng Feng contributed to the formal analysis. Peng Jia conducted the writing of the original draft of the study. Shujuan Yang conducted the conceptualization, methodology, writing of the original draft, review, editing, and supervision of the study. All authors were involved in writing the paper and had final approval of the submitted and published versions.

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Availability of data and materials
Some or all datasets generated during and/or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by the medical ethics committee of the Yunnan Center for Disease Prevention and Control (202017) and was carried out in accordance with the Helsinki Declaration of 1964. All participants signed informed consent before the survey.

Consent for publication
Not applicable.

Competing interests
The authors have no relevant financial or non-financial interests to disclose.

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