Environmental Science and Pollution Research
https://doi.org/10.1007/s11356-023-31098-z

RESEARCH ARTICLE

Long-term exposure to PM_1 is associated with increased prevalence of metabolic diseases: evidence from a nationwide study in 123 Chinese cities

Qin Zhou[1] · Xianfeng Li[2] · Jinglong Zhang[3] · Zhizhou Duan[4] · Shuyuan Mao[5] · Jing Wei[6] · Shichao Han[7] · Zhiping Niu[8]

Received: 16 August 2023 / Accepted: 14 November 2023

Abstract

Exposure to particulate matter (PM) has been linked to metabolic diseases. However, the effects of PM with an aerodynamic diameter ≤ 1.0 μm (PM_1) on metabolic diseases remain unclear. This study is aimed at assessing the associations of PM_1 with metabolic disease risk and quantifying the concentration–response (C-R) relationship of PM_1 with metabolic disease risk. A national cross-sectional study was conducted, including 12,495 middle-aged and older adults in 123 Chinese cities. The two-year average concentration of PM_1 was evaluated using satellite-based spatiotemporal models. Metabolic diseases, including abdominal obesity, diabetes, hypertension, dyslipidemia, and metabolic syndrome, were identified based on physical examination, blood standard biochemistry examination, and self-reported disease histories. Generalized linear models and C-R curves were used to evaluate the associations of PM_1 with metabolic diseases. A total of 12,495 participants were included in this study, with a prevalence of 45.73% for abdominal obesity, 20.22% for diabetes, 42.46% for hypertension, 41.01% for dyslipidemia, and 33.78% for metabolic syndrome. The mean ± standard deviation age of participants was 58.79 ± 13.14 years. In addition to dyslipidemia, exposure to PM_1 was associated with increased risks of abdominal obesity, diabetes, hypertension, and metabolic syndrome. Each 10 μg/m^3 increase in PM_1 concentrations was associated with 39% (odds ratio (OR) = 1.39, 95% confidence interval (CI) 1.33, 1.46) increase in abdominal obesity, 18% (OR = 1.18, 95%CI 1.12, 1.25) increase in diabetes, 11% (OR = 1.11, 95%CI 1.06, 1.16) increase in hypertension, and 25% (OR = 1.25, 95%CI 1.19, 1.31) in metabolic syndrome, respectively. C-R curves showed that the OR values of abdominal obesity, diabetes, hypertension, and metabolic syndrome were increased gradually with the increase of PM_1 concentrations. Subgroup analysis indicated that exposure to PM_1 was associated with increased metabolic disease risks among participants with different lifestyles and found that solid fuel users were more susceptible to PM_1 than clean fuel users. This national cross-sectional study indicated that exposure to higher PM_1 might increase abdominal obesity, diabetes, hypertension, and metabolic syndrome risk, and solid fuel use might accelerate the adverse effects of PM_1 on metabolic syndrome risk. Further longitudinal cohort studies are warranted to establish a causal inference between PM_1 exposure and metabolic disease risk.

Keywords Particulate matter · Obesity · Diabetes · Hypertension · Dyslipidemia · Metabolic syndrome

Introduction

Non-communicable diseases have been reported as the leading causes of global burden, accounting for 15 million premature deaths each year (Chong et al. 2023; Collaborators 2020). A large percentage of non-communicable diseases could be attributed to the increasing burden of metabolic diseases, such as obesity, diabetes, hypertension, dyslipidemia, and metabolic syndrome (Chew et al. 2023; Chong et al. 2023; Han et al. 2022). Genetic defects, physical inactivity, unhealthy behaviors, and lifestyles have been identified

Responsible Editor: Lotfi Aleya

Qin Zhou, Xianfeng Li, and Jinglong Zhang contributed equally to this work.

Extended author information available on the last page of the article

Published online: 28 November 2023

as significant risk factors for rising metabolic disease risks (Chong et al. 2023). However, those factors might not fully explain the high prevalence of metabolic diseases, environmental risk factors, such as particulate matter (PM), have attracted extensive attention from epidemiologists in recent years.

Numerous studies indicated that exposure to PM was associated with increased metabolic disease risks, especially PM with an aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$) and PM with an aerodynamic diameter ≤ 10 μm (PM_{10}). Many studies indicated that exposure to PM was associated with an increased risk of metabolic diseases, including obesity (Zheng et al. 2021), diabetes (Liu et al. 2019a, 2019b; Yitshak Sade et al. 2023), hypertension (Li et al. 2020; Niu et al. 2023; Weng et al. 2022), dyslipidemia (Mao et al. 2020a), and metabolic syndrome (Han et al. 2022; Letellier et al. 2022; Zhang et al. 2021a). For example, Zheng et al. conducted a cross-sectional study of 36,456 aged 7–19 years in Jiangsu province of China and found that each 10 μg/m^3 increase in $PM_{2.5}$ concentrations was associated with an 18.5% increase in obesity risk (odds ratio (OR) = 1.198, 95% confidence interval (CI) 1.054, 1.333) (Zheng et al. 2021). Liu et al. conducted a meta-analysis of 30 studies and indicated that long-term exposure to $PM_{2.5}$ and PM_{10} was all associated with increased diabetes. They found each 10 μg/m^3 increase in $PM_{2.5}$ and PM_{10} concentrations was associated with 9% (OR = 1.09, 95%CI 1.05, 1.13) and 12% (OR = 1.12, 95%CI 1.06, 1.19) increase in diabetes risk, respectively (Liu et al. 2019a). Weng et al. conducted a prospective study of 91,366 participants in the UK biobank and found each 10 μg/m^3 increase in $PM_{2.5}$ and PM_{10} was associated with 41% (hazard ratio (HR) = 1.41, 95%CI 1.29, 1.53) and 5% (HR = 1.05, 95%CI 1.00, 1.09) increase in hypertension incidence, respectively (Weng et al. 2022). Letellier et al. conducted cross-sectional study of 552 participants Hispanics/Latinos and non-Hispanics and indicated that higher dynamic exposure to $PM_{2.5}$ was associated with increased risk of obesity (RR = 1.23, 95% CI 1.10, 1.37), dyslipidemia (RR = 1.12, 95% CI 1.03, 1.22) and metabolic syndrome (RR = 1.17, 95% CI 1.07–1.28) (Letellier et al. 2022).

PM with an aerodynamic diameter ≤ 1.0 μm (PM_1), smaller size PM with a bigger surface-to-mass ratio than $PM_{2.5}$ and comprising over 80% of $PM_{2.5}$ (Li et al. 2019; Yang et al. 2018b), could carry more extensive toxins and easily penetrate deep into lung alveolar region and enter into the circulatory system (Guo et al. 2022; Yang et al. 2019). The health effects of PM_1 have drawn increasing attention from epidemiologists in recent years (Xu et al. 2022). However, compared with sufficient evidence of $PM_{2.5}$ and PM_{10}, the number of PM_1 is limited and results are still controversial. Several studies suggested that exposure to PM_1 was associated with increased metabolic disease risks (Hou et al. 2020; Li et al. 2019; Mao et al. 2020b; Yang et al. 2019; Yang et al. 2018a), while other studies reported in-significant associations (Zang et al. 2021; Zhang et al. 2021a). For example, Li et al. conducted a cross-sectional study of Chinese rural adults and found each 1 μg/m^3 increase in PM_1 was associated with a 4.3% (OR = 1.403, 95%CI 1.033, 1.053) increase in hypertension (Li et al. 2019). Yang et al. conducted a cross-sectional study of 15,477 Chinese adults and found each inter-quartile range (IQR, 15 μg/m^3) increase in PM_1 was associated with a 13% (OR = 1.13, 95%CI 1.04, 1.22) increase in diabetes prevalence (Yang et al. 2018a). However, Zhang et al. conducted a cross-sectional study of 9,897 Chinese children and adolescents and did not observe statistically significant associations between PM_1 and metabolic syndrome (OR = 1.20, 95%CI 0.99, 1.46) (Zhang et al. 2021a). Moreover, those studies focused on the association of long-term exposure to PM_1 with each metabolic disease (such as obesity, diabetes, hypertension, or metabolic syndrome) were published in isolation, comprehensive evidence of the potential adverse effects of PM_1 in on different metabolic diseases remains scarce, especially in rapidly increasing population of Chinese middle-aged and older adults (Niu et al. 2022a).

Therefore, to fill this gap, we used a two-stage analytic approach to investigate the associations of exposure to PM_1 with the four most common metabolic diseases among Chinese middle-aged and older adults, including obesity, diabetes, hypertension, dyslipidemia, and metabolic syndrome. Potential modification effects of health behaviors and lifestyles were also examined in this study.

Material and methods

Study population

The study population was drawn from the China Health and Retirement Longitudinal Study (CHARLS), a national cohort study of middle-aged and older adults in China. Briefly, participants were recruited from community-based populations in 28 provinces of China through a multistage probability proportionate sampling (PPS) method. The national baseline survey was carried out in 2011, and 3 waves of follow-ups were completed in 2013, 2015, and 2018, respectively. Details of the CHARLS are shown in previous studies (Chen et al. 2019; Zhao et al. 2014). Physical examination and blood-based biomarkers assessment were only conducted in 2011 and 2015, and estimated PM_1 concentration data was obtained after 2013; therefore, we conducted a cross-sectional study to investigate the association of PM_1 and metabolic diseases, using the health data of CHARLS wave 3 in 2015.

A total of 21,059 participants were recruited in the CHARLS 2015, with 16,406 participants participating in the physical examination. Subsequently, we excluded 2,055 study subjects without blood samples and 3,560 study subjects without metabolic biomarkers data. Finally, we excluded 351 participants in some regions of Xinjiang and Inner Mongolia where PM_1 exposure failed to be assessed. A total of 12,495 participants from 27 provinces were included in the study. The flowchart of participants' inclusion is presented in Figure S1.

Study ethics approval of the CHARLS study was obtained from Peking University (Reference Number: IRB00001052–11015). All participants and their guardians signed consent voluntarily.

PM_1 exposure assessment

Individual PM_1 exposure was estimated from space using space–time extremely randomized trees (STET) models. Briefly, surface PM_1 measurement (collected from the China Atmosphere Watch Network of the China Meteorological Administration), Multi-Angle Implementation of Atmospheric Correction aerosol optical depth (MAIAC AOD, collected from the National Aeronautics and Space Administration's (NASA) Terra and Aqua Moderate Resolution Imaging Spectroradiometer (MODIS) C6 MAIAC Level (L) 2 swath aerosol products (MCD19A2)) product, meteorological data (from ERA-Interim atmospheric reanalysis products), the multi-resolution emission inventory for China (MEIC) data, and other ancillary data (such as land cover, topography, traffic, and population) were combined in PM_1 assessments at 1 km spatial resolution. After data integration, all selected factors were input to the STET model, which took into account the spatial autocorrelation, adjacent pixels, and temporal difference. The average cross-validation R^2 and root-mean-square error (RMSE) were 0.83 and 9.50 μg/m^3 at daily levels, respectively. Detailed information on modeling methodology was shown in previous studies (Wei et al. 2019, 2023) and the CHAP website (https://weijing-rs.github.io/product.html). Annual PM_1 concentrations were assigned to each participant according to their residential cities. The two-year average concentration of PM_1 was calculated as long-term PM_1 exposure (Zheng et al. 2022).

Assessment of metabolic diseases

Metabolic disease is defined as a cluster of metabolic system disorders. To identify the most common and impactful metabolic diseases in humans, we conducted a careful literature review and found that obesity, diabetes, hypertension, dyslipidemia, and non-alcoholic fatty liver disease (NAFLD) were the most notable metabolic diseases (Azizi et al. 2019; Chew et al. 2023; Steenblock et al. 2021). In addition to NAFLD which was not available in the CHARLS dataset, our study included 4 above metabolic diseases (obesity, diabetes, hypertension, and dyslipidemia), which may effectively represent metabolic diseases. Moreover, we noticed that metabolic syndrome is defined as a cluster of metabolic disorders, including abdominal obesity, dyslipidemia, hypertension, and diabetes, has attracted wide attention from recent studies, and metabolic syndrome could reflect the metabolic disorders more comprehensively (Eckel et al. 2005; Han et al. 2022). Therefore, we included metabolic syndrome in our study.

Waist circumference (WC) was assessed for diagnosis of abdominal obesity and metabolic syndrome. Three times blood pressure measurements were completed for the diagnosis of hypertension and metabolic syndrome. Fasting blood glucose (FBG) and glycosylated hemoglobin, type A1C (HbA1C) were tested for diagnosis of diabetes and metabolic syndrome. Detailed information on WC, blood pressure, and blood-based biomarkers assessments was described in previous studies (Chen et al. 2019; Han et al. 2022; Niu et al. 2022a). In addition, self-reported clinical diseases and medication information were also collected to assist in the diagnosis of diabetes, hypertension, and metabolic syndrome via a face-to-face interview.

Abdominal obesity was defined as WC ≥ 85 cm for females and WC ≥ 90 cm for males (Cao et al. 2021). Diabetes cases were defined as FBG ≥ 7.0 mmol/L and/or HbA1C ≥ 6.5% and/or having self-reported clinically diagnosed diabetes and/or currently taking anti-diabetes medication (Association 2021; Chen et al. 2022). Hypertension was defined as SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg and/or having self-reported clinically diagnosed hypertension and/or currently taking anti-hypertension medication (Chew et al. 2023; Niu et al. 2022a). Dyslipidemia was defined as individual with one or more abnormal blood lipid concentrations (hypercholesterolemia: TC ≥ 6.22 mmol/L; hypertriglyceridemia: TG ≥ 2.26 mmol/L; hypoalphalipoproteinemia: HDL-C < 1.04 mmol/L; hyperbetalipoproteinemia LDL-C ≥ 4.14 mmol/L) (Mao et al. 2020b; Nie et al. 2023). Metabolic syndrome case was diagnosed as participant with WC ≥ 80 cm for females and WC ≥ 90 cm for males plus any two of the following components was defied as metabolic syndrome patient: (1) TG > 1.7 mmol/L, (2) HDL < 1.29 mmol/L in female or HDL < 1.03 mmol/L in male, (3) SBP ≥ 130 mmHg and/or DBP ≥ 85 mmHg and/or currently taking anti-hypertension medication, and (4) FBG ≥ 5.6 mmol/L and/or currently taking anti-diabetic medication (Han et al. 2022; Hou et al. 2020).

Covariates

Based on previous studies of particulate matter and metabolic diseases and directed acyclic graph analysis

(Figure S2), a series of confounders were identified in our study. These covariates included (1) climate condition: temperature and relative humidity (Han et al. 2022; Wang et al. 2023); (2) sociodemographic variables: age and sex (Liu et al. 2019b; Mao et al. 2020b; Zhang et al. 2021a); (3) socioeconomic factors: education status (Zhang et al. 2021a), marital status (Chen et al. 2022; Liu et al. 2019b), and annual household expenditure (Han et al. 2022); (4) health behaviors and lifestyles: smoking status (Yang et al. 2018b), drinking status (Mao et al. 2020b), cooking fuel use, and physical activity (PA) (Cao et al. 2021; Zhang et al. 2023). For smoking status, individuals were classified as smokers if they had ever engaged in tobacco chewing, pipe smoking, or self-rolled cigarettes, cigarettes, or cigars. Drinking status was determined by assessing participants' alcohol consumption behavior in the past year, including liquor, wine, or beer intake (Niu et al. 2022a). Physical activity was measured using the International Physical Activity Questionnaire. The physical activity score (PA score) was calculated using metabolic equivalent multipliers (MET) as follows: PA score (MET-hour/week) = 3.3 × total walking weekly duration score + 4.0 × total moderate activity weekly duration score + 8.0 × total vigorous activity weekly (Han et al. 2022; Zhang et al. 2023). For cooking fuel use, natural gas, marsh gas, and liquefied petroleum gas were classified as "clean fuel," while coal, crop residue, or wood burning were classified as "solid fuel" (Han et al. 2022; Wang et al. 2023). Covariates of temperature, relative humidity, age, and physical activity were included in models as continuous variables, while sex, education status, drinking status, and cooking fuel use were included in models as categorical variables. To avoid excessive loss of samples, missing data of covariates were input using the R package "mice" in this study (Wang et al. 2023).

Statistical analysis

Descriptive statistics of participants were conducted. Continuous variables were described as mean ± standard deviation (SD), and categorical variables were described as frequency (percentage, %). Student's *t*-test was used to examine the difference of continuous variables between patients and non-patients, and the Chi-square test was applied to assess the distribution discrepancy of categorical variables. Mean, SD, and interquartile range (IQR) were applied to summarize the descriptive chrematistics of metabolic biomarkers, PM_1 exposure, and climate conditions.

A two-stage analytic approach was performed to explore the relationship between PM_1 exposure and metabolic diseases. In the first stage, generalized linear models were performed to examine the associations of PM_1 concentrations with metabolic disease risks, including abdominal obesity, diabetes, hypertension, and metabolic syndrome. Results were shown as the odds ratio (OR) and its 95% confidence interval (CI) of metabolic disease risk with each 10 μg/m^3 increase in PM_1 concentrations. First, a crude model was performed without any adjustment. Second, temperature and relative humidity were included in adjusted model 1 to adjust potential climate condition confounders using the spline function. Third, sociodemographic and socioeconomic factors were additionally adjusted in adjusted model 2. Finally, health behaviors and lifestyle variables were additionally included in adjusted model 3. In the second stage, concentration–response (C-R) relationships of long-term exposure to PM_1 with the OR of metabolic diseases were also investigated in this study. Specifically, relationships of PM_1 with metabolic diseases were fitted as a smoothing term using restricted cubic spline (RCS) with 3 knots at the 10th, 50th, and 90th percentiles of 2-year average PM_1 concentration (reference is the 10th percentile) (Wang et al. 2023). Nonlinearity was tested using analysis of variance (ANOVA), and inflection points were identified using segmented regression (Hou et al. 2023).

Previous studies indicated that health lifestyles, such as smoking (Mao et al. 2020b), alcohol consumption (Mao et al. 2020b), cooking fuel use (Cao et al. 2021; Liu et al. 2021), and physical exercise (Hou et al. 2020; Zhang et al. 2023), could modify the adverse of air pollution on metabolic diseases; we conducted a series of subgroup analysis by different health lifestyles (smoking status, drinking status, cooking fuel use, and PA) to provide more evidence for reducing the adverse effect of exposure to PM_1 on metabolic diseases by choosing more healthy lifestyles.

Several sensitivity analyses were conducted to test the robustness of the results. First, we applied a generalized linear mixed-effect model to re-examine the association between PM_1 and metabolic diseases by including City ID as a random-effect intercept. This model allows participants in the same city to serve as their own control and could adjust for the spatial clustering of metabolic diseases (Ren et al. 2023; Wu et al. 2020). Second, we re-investigated the associations of PM_1 with metabolic diseases by using 1-year average concentration of PM_1 exposure before CHARLS Wave 3. Third, log-binomial Poisson regressions were performed, accounting for the high prevalence of metabolic disease (Barros and Hirakata 2003, Han et al. 2022). Fourth, participants who had ever changed their address from their last investigation in 2013 were excluded. Fifth, associations of PM_1 with hypertension and metabolic syndrome were re-examined by excluding participants currently taking anti-hypertension medication, accounting for the potential influence of anti-hypertension medication on blood pressure (Niu et al. 2022a). Finally, the associations of PM_1 with diabetes and metabolic syndrome were assessed by excluding participants currently taking anti-diabetic medication, accounting

for the potential influence of anti-diabetic medication on blood glucose.

Statistical analyses of this study were completed using R (version 4.2.1). "mice" package for the imputation of missing data, and "plotRCS" for C-R relationships plots. A two-sided P value < 0.05 was regarded as statistical significance.

Results

Descriptive statistic of study population characteristics, metabolic biomarkers, and ambient PM_1 exposure

A total of 12,495 participants were included in this study, and these participants were recruited from 435 communities in 123 cities. The spatial distribution of participants in 27 provinces of China is presented in Fig. 1. Of the 12,495 participants, 5,714 (45.73%) adults were identified as abdominal obesity cases, 2,526 (20.22%) adults were identified as diabetes cases, 5,303 (42.46%) adults were identified as hypertension cases, 5,124 (41.01%) adults were identified as dyslipidemia cases, and 4,221 (33.78%) adults were identified as metabolic syndrome cases, respectively. The mean age of participants was 58.79 ± 13.14 years. Table 1 summarizes the descriptive chrematistics of the participants.

Table 2 presents the descriptive chrematistics of metabolic biomarkers, PM_1 exposure, and climate conditions. In terms of metabolic biomarkers, the mean WC, FBG, HbA1c, SBP, DBP, TG, TC, HDL, and LDL were 85.24 ± 13.16 cm, 5.75 ± 1.95 mmol/L, 5.87 ± 1.00%, 128.33 ± 19.66 mmHg, 75.43 ± 11.19 mmHg, 1.61 ± 1.03 mmol/L, 4.75 ± 0.94 mmol/L, 1.32 ± 0.29 mmol/L, and 2.64 ± 0.74 mmol/L, respectively. The 2-year average PM_1 concentration of participants was 37.51 ± 10.04 μg/m^3.

Associations between exposure to PM_1 and metabolic diseases

Figure 2 presents the associations between PM_1 and four metabolic diseases. Both crude models and three adjusted models indicated that exposure to higher PM_1 concentrations was associated with increased risks of abdominal obesity, diabetes, hypertension, and metabolic syndrome. In fully-adjusted model (adjusted model 3), each 10 μg/m^3 increase in PM_1 concentrations was associated with 39% (OR = 1.39, 95% CI 1.33, 1.46) increase in abdominal obesity, 18% (OR = 1.18, 95%CI 1.12, 1.25) increase in diabetes, 11% (OR = 1.11, 95%CI 1.06, 1.16) increase in hypertension, and 25% (OR = 1.25, 95%CI 1.19, 1.31) in metabolic syndrome, respectively.

C-R relationships of exposure to PM_1 with metabolic diseases

Figure 3 presents the C-R relationships of exposure to PM_1 with metabolic diseases. We observed that the OR values of abdominal obesity, diabetes, hypertension, and metabolic syndrome were increased gradually with the increase of PM_1 concentrations (P for overall < 0.05), while a similar trend was not been observed in the relationship of PM_1 with dyslipidemia (P for overall > 0.05). In terms of the shapes of C-R curves, the C-R curves of PM_1 exposure with abdominal obesity, diabetes, and metabolic syndrome were nearly

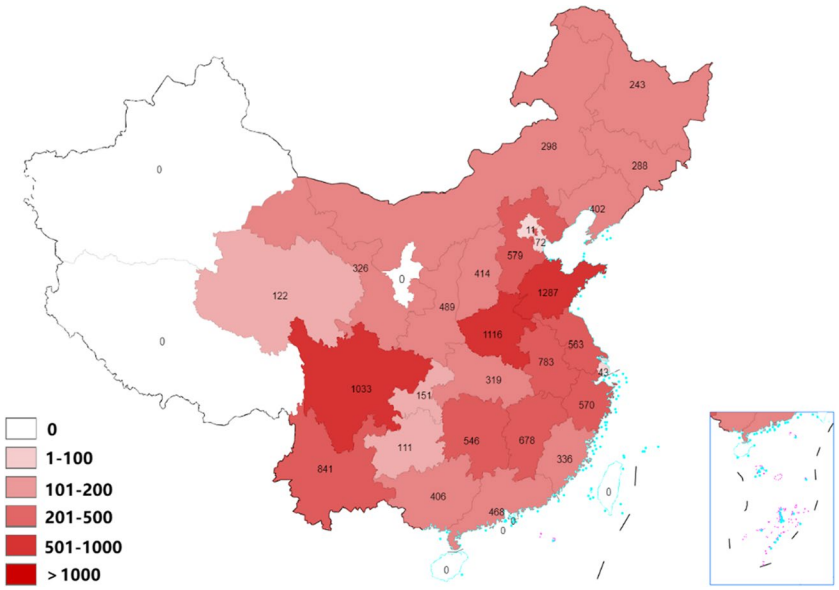

Fig. 1 The spatial distribution of 12,495 middle-aged and older adults in 27 provinces of China

Table 1 Basic characteristics of study participants

Characteristics	Total	Abdominal obesity		Diabetes		Hypertension		Dyslipidemia		Metabolic syndrome	
	(n=12,495)	Yes (n=5714)	No (n=6781)	Yes (n=2526)	No (n=9969)	Yes (n=5303)	No (n=7192)	Yes (n=5124)	No (n=7371)	Yes (n=4221)	No (n=8274)
Age (mean ± SD)	□										
Age (years)	58.79 ± 13.14	58.42 ± 12.68	59.10 ± 13.50*	60.72 ± 9.71	58.30 ± 12.98***	61.46 ± 13.61	56.81 ± 12.41***	59.65 ± 12.48	58.73 ± 13.18 *	58.94 ± 13.22	58.70 ± 13.01
Sex (n (%))											
Male	5732 (45.9)	2032 (35.6)	3700 (54.6)***	1118 (44.3)	4614 (46.3)	2511 (47.4)	3221 (44.8) **	2545 (49.7)	3187 (43.2)***	1313 (31.1)	4419 (53.4)***
Female	6763 (54.1)	3682 (64.4)	3081 (45.4)	1408 (55.7)	5355 (53.7)	2792 (52.6)	3971 (55.2)	2579 (50.3)	4184 (56.8)	2908 (68.9)	3855 (46.6)
Residence (n (%))											
Rural	7760 (62.1)	3199 (56.0)	4561 (67.3)***	1416 (56.1)	6344 (63.6)***	3189 (60.1)	4571 (63.6)***	3112 (60.7)	4638 (62.9)**	2290 (54.3)	5470 (66.1)***
Urban	4735 (37.9)	2515 (44.0)	2220 (32.7)	1110 (43.9)	3625 (36.4)	2114 (39.9)	2621 (36.4)	2012 (39.3)	2732 (37.1)	1931 (45.7)	2804 (33.9)
Education status (n (%))											
Elementary or below	8267 (66.2)	3704 (64.8)	4563 (67.3)*	1723 (68.2)	6544 (65.6)	3670 (69.2)	4597 (63.9)***	3361 (65.6)	4906 (66.6)	2842 (67.3)	5425 (65.6)*
Middle school or high	4228 (33.8)	2010 (35.2)	2218 (32.7)	803 (31.8)	3425 (34.4)	1633 (30.8)	2595 (36.1)	1763 (34.4)	2465 (33.4)	1379 (32.7)	2849 (34.4)
Marital status (n (%))											
Married	10,863 (86.9)	4986 (87.3)	5877 (86.7)	2154 (85.3)	8709 (87.4)**	4420 (83.3)	6443 (89.6)***	4443 (86.7)	6420 (87.1)	3632 (86.0)	7231 (84.7)*
Single, divorced, and widowed	1632 (13.1)	728 (12.7)	904 (13.3)	372 (14.7)	1260 (12.6)	883 (16.7)	749 (10.4)	681 (13.3)	951 (12.9)	589 (14.0)	1043 (12.6)
Household expenditure (mean ± SD)											
Household expenditure ($\times 10^3$ CNY)	14.22 ± 41.40	13.92 ± 35.11	14.48 ± 46.05	14.23 ± 34.03	14.22 ± 43.07	12.80 ± 32.63	15.27 ± 46.81***	14.82 ± 47.70	13.81 ± 36.39	14.12 ± 37.25	14.28 ± 43.38
Smoking status (n (%))											
Non-smoker	7234 (57.9)	3793 (66.4)	3441 (50.7)***	1461 (57.8)	5773 (57.9)	2973 (56.1)	4261 (59.2)***	2766 (54.0)	4468 (60.6)***	2916 (69.1)	4318 (52.2)***
Smoker	5261 (42.1)	1921 (33.6)	3340 (49.3)	1065 (42.2)	4196 (42.1)	2330 (43.9)	2931 (408)	2358 (46.0)	2903 (39.4)	1305 (30.9)	3956 (47.8)
Drinking status (n (%))											
Non-drinker	3262 (26.0)	1275 (22.3)	1977 (29.2)***	578 (22.9)	2674 (26.8)***	1388 (26.2)	1864 (25.9)	1331 (26.0)	1921 (26.1)	830 (19.7)	2422 (29.3)***
Drinker	9243 (74.0)	4433 (77.6)	4800 (70.8)	1947 (77.1)	7286 (73.1)	3909 (73.7)	5324 (74.0)	3789 (73.9)	5444 (73.9)	3388 (80.3)	5845 (70.6)
Cooking fuel use (n (%))											
Solid fuel	2700 (25.8)	3496 (61.2)	3822 (56.4)***	1527 (60.5)	5791 (58.1)*	3076 (58.0)	4242 (59.0)	3025 (59.0)	4293 (58.2)	2615 (62.0)	4703 (56.8)***
Clean fuel	3672 (35.1)	2218 (38.8)	2959 (43.6)	999 (39.5)	4178 (41.9)	2227 (42.0)	2950 (41.0)	2099 (41.0)	3078 (41.8)	1606 (38.0)	3571 (43.2)
Physical activity (mean ± SD)											
PA score (MET-hour/week)	125.89 ± 109.20	117.68 ± 105.30	132.82 ± 111.60***	119.00 ± 103.93	127.64 ± 110.21***	121.39 ± 107.58	129.22 ± 109.96*	117.67 ± 107.33	126.44 ± 109.12*	116.76 ± 103.97	130.56 ± 111.23***

MET, metabolic equivalent multipliers; *, **, and *** represented the P value of difference comparison for the characteristics between patients and non-patients; $^{*}P<0.05$, $^{**}P<0.01$, and $^{***}P<0.001$

Table 2 Descriptive statistics of metabolic biomarkers, PM_1, and meteorological factors

Variables	Mean ± SD	P25	P50	P75	IQR
Metabolic biomarkers					
WC (cm)	85.24 ± 13.16	78.80	86.00	93.00	14.20
FBG (mg/dL)	103.53 ± 35.04	88.29	95.50	106.30	18.01
SBP (mmHg)	128.33 ± 19.66	114.00	126.00	140.33	26.33
DBP (mmHg)	75.43 ± 11.19	67.33	74.67	82.33	15.00
TG (mmol/L)	1.61 ± 1.03	0.93	1.29	1.92	0.99
TC (mmol/L)	4.75 ± 0.94	4.10	4.67	5.30	1.20
HDL (mmol/L)	1.32 ± 0.30	1.12	1.29	1.49	0.37
LDL (mmol/L)	2.64 ± 0.74	2.13	2.59	3.09	0.96
Air pollutant					
PM_1 (μg/m^3)	37.51 ± 10.04	30.48	37.37	44.66	14.18
Climate condition					
Temperature (℃)	15.43 ± 4.03	14.10	16.10	17.85	3.75
Relative humidity (%)	67.84 ± 10.89	60.50	70.50	76.00	15.50

linear (P for nonlinear > 0.05). The C-R curves of PM_1 exposure with hypertension showed slight "J" shapes (P for nonlinear = 0.010), with inflection points of 37.5 μg/m^3.

Subgroup analysis by different health behaviors and lifestyles for the associations of PM_1 concentration with metabolic diseases

Figure 4 displays the subgroup analysis by different health behaviors and lifestyles for the associations of PM_1 with metabolic diseases. Positive and significant associations of PM_1 with abdominal obesity, diabetes, hypertension, and metabolic syndrome were observed in all subgroups. When comparing the estimated effects in different subgroups, we observed the OR value of metabolic syndrome with PM_1 was higher in solid fuel use participants than that of clean fuel use participants, with P-interaction < 0.05.

Sensitivity analysis

Sensitivity analysis showed that the positive associations of PM_1 with all four metabolic diseases were stable when City ID was included in a generalized linear mixed-effect model as a random-effect intercept, 1-year average PM_1 concentration, and log-binomial Poisson regression model were applied in models or participants who had ever changed their address from the last investigation were excluded (Table S2-S5). Sensitivity analysis by excluding anti-hypertension medication or anti-diabetic medication takers showed consistent associations, except for hypertension in adjusted model 1 and diabetes in the crude model (Table S6, Table S7).

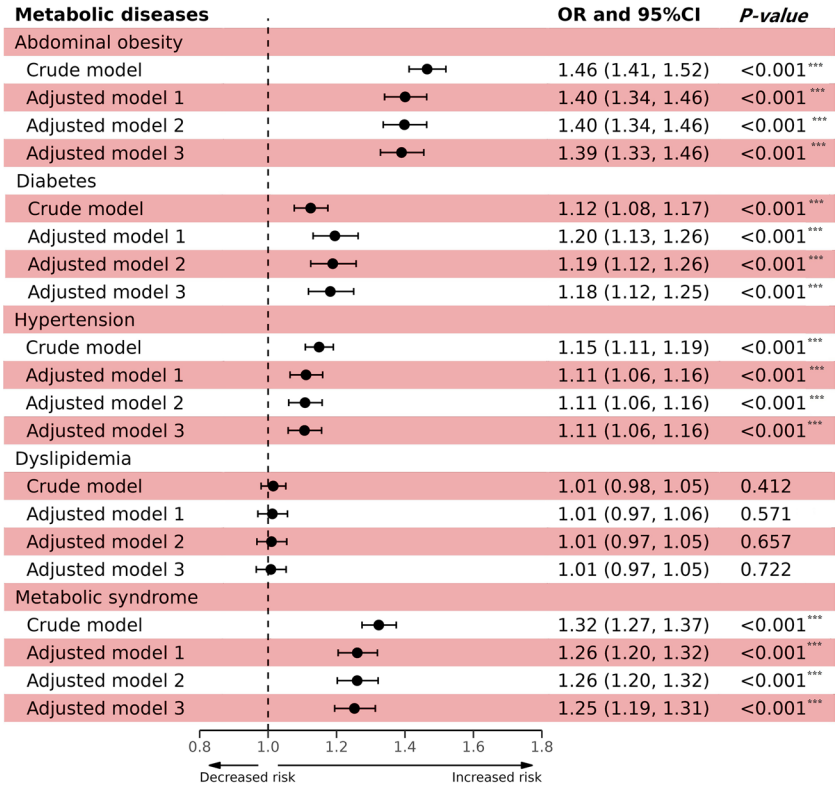

Fig. 2 OR and 95%CI of metabolic diseases associated with 10 μg/m^3 increase in PM_1. Notes: $^*P < 0.05$, $^{**}P < 0.01$, and $^{***}P < 0.001$

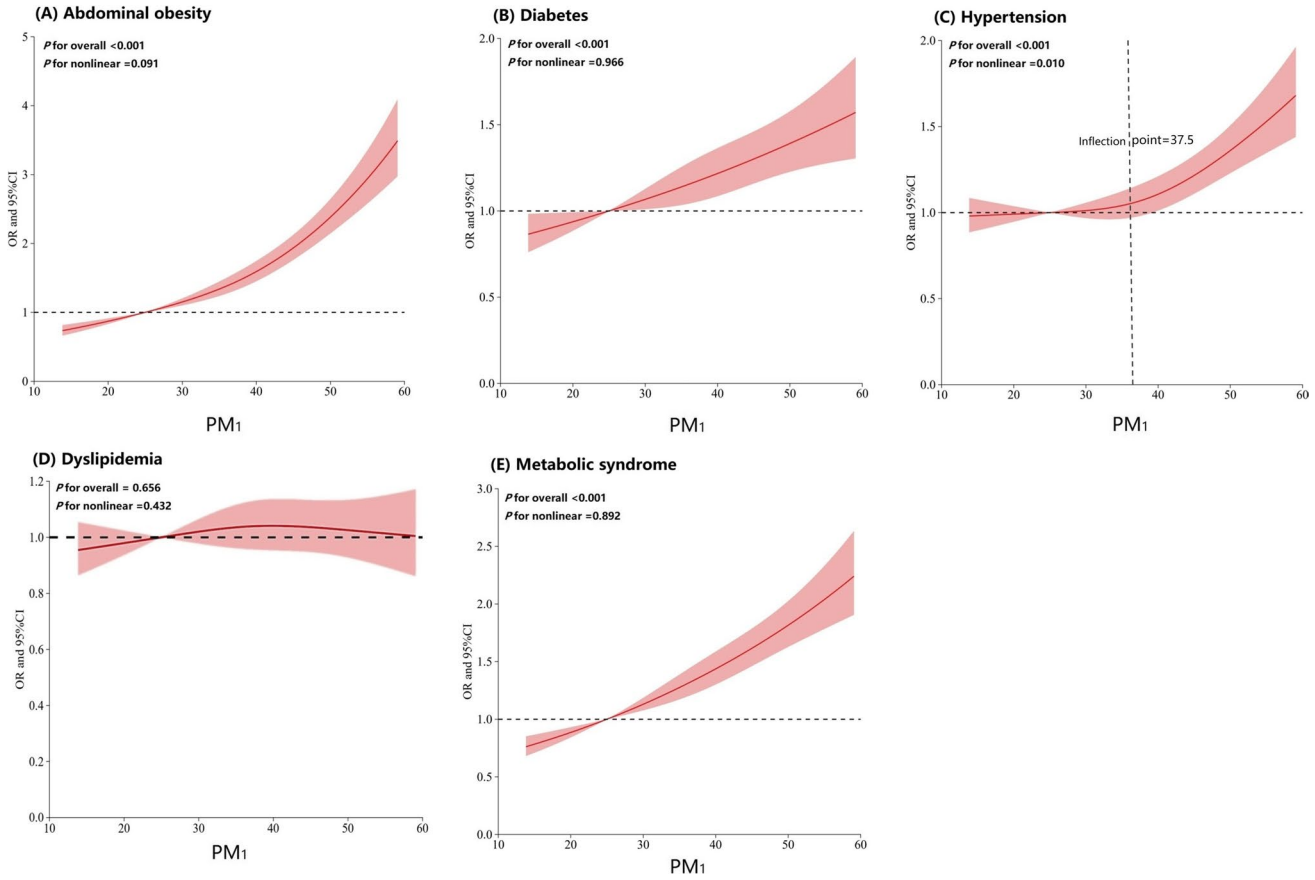

Fig. 3 Concentration–response relationships of long-term exposure to PM_1 with the OR of metabolic diseases. **A** abdominal obesity, **B** diabetes, **C** hypertension, **D** dyslipidemia, and **E** metabolic syndrome

Discussion

Key findings

In this Chinese national cross-sectional study, we found that exposure to higher PM_1 concentration was associated with an increased risk of metabolic diseases, including abdominal obesity, diabetes, hypertension, and metabolic syndrome. Moreover, the C-R curves of PM_1 with metabolic diseases showed that the OR values of metabolic diseases were increased gradually with the increase of PM_1 concentrations, with slight "J" shapes for hypertension and linear shapes for abdominal obesity, diabetes, and metabolic syndrome. To our knowledge, this is the most comprehensive study that has systematically examined the adverse effects of PM_1 on four common metabolic diseases.

Comparison with other studies and interpretations

While the associations of exposure to PM_1 with different metabolic diseases still have not been examined in one single study, several studies of long-term exposure to PM_1 with specific metabolic diseases could support our findings.

We found that exposure to higher PM_1 concentration was associated with an increased abdominal obesity risk. This finding was consistent with three similar designed studies (Liu et al. 2023; Zhang et al. 2021a, 2021b). In a national Chinese cross-sectional study, Liu et al. reported that each 10 μg/m^3 increase in PM_1 concentrations was associated with a 29.4% (OR = 1.294, 95%CI 1.225, 1.367) increase in the prevalence of central obesity (Liu et al. 2023). Moreover, two cross-sectional studies of children and adolescents also reported a positive association between PM_1 and abdominal obesity. They found that each 10 μg/m^3 increase in PM_1 concentrations was associated with a 42% (OR = 1.42, 95%CI 1.23, 1.64) (Zhang et al. 2021a) and 39% (OR = 1.39, 95%CI 1.23, 1.57) (Zhang et al. 2021b) increase in the prevalence of central obesity, which were similar to our findings (OR = 1.39, 95%CI 1.33, 1.46). The estimated effects of our study were similar to the studies conducted by Zhang et al. (2021a) and Zhang et al. (2021b), but higher than that of Liu et al.'s study, which might be due to different definition of obesity, PM_1 exposure estimation methods, different sample

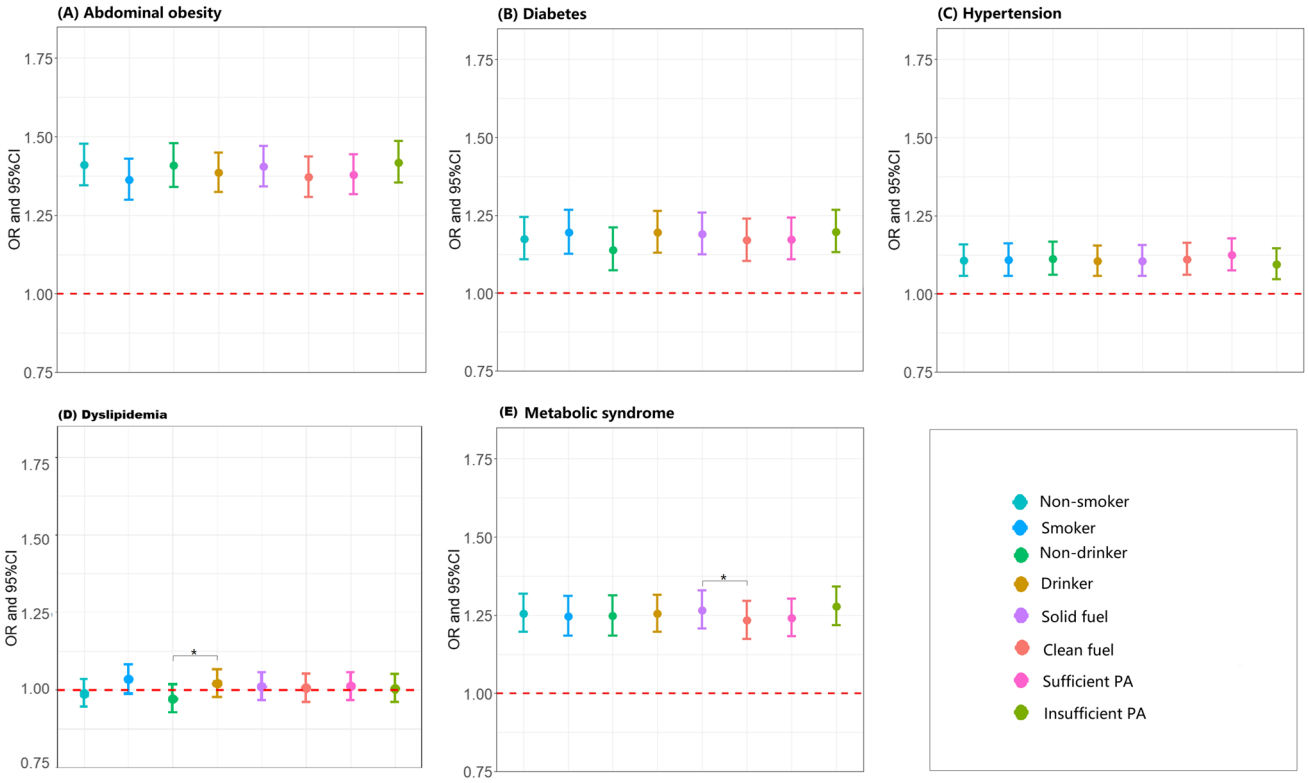

Fig. 4 Subgroup analysis by lifestyles for the associations of long-term exposure to PM_1 with metabolic diseases. **A** abdominal obesity, **B** diabetes, **C** hypertension, **D** dyslipidemia, and **E** metabolic syndrome. Notes: $^{*}P<0.05$, $^{**}P<0.01$, and $^{***}P<0.001$

size, and other study characteristics. In addition, we found that those studies were conducted in China; further studies are necessary to confirm the adverse effects of exposure to PM_1 on metabolic systems in other countries, especially low-polluted countries. Finally, we also examined the C-R of PM_1 with abdominal obesity and observed linear shapes of C-R curves for the relationship of PM_1 with abdominal obesity. To our knowledge, the C-R of PM_1 with abdominal obesity has not been reported previously, and our study might provide more comprehensive evidence for the adverse effects of long-term exposure to PM_1 on abdominal obesity risk.

This study indicated that long-term exposure to PM_1 was related to increased diabetes risk. Several previous studies have examined the association of exposure to PM_1 with diabetes, with some studies finding positive associations (Liu et al. 2019b; Liu et al. 2019c; Mei et al. 2023; Yang et al. 2018a) and other studies reporting nonsignificant association (Wang et al. 2022). For example, Mei et al. conducted a cross-sectional study and found that each 10 μg/m^3 increase in PM_1 was associated with a 14% (OR = 1.14, 95%CI 1.0, 1.29) increase in diabetes risk (Mei et al. 2023). Another cross-sectional study of the 33 Chinese communities indicated that each 15 μg/m^3 increase in PM_1 was associated with a 13% (OR = 1.13, 95%CI 1.04, 1.29) increase in diabetes risk (Yang et al. 2018a). The estimated effects of our study (OR = 1.18, 95%CI 1.12, 1.25), but higher than that of Mei et al.'s study and Yang et al.'s study, which may be explained by different study sample size, study period, and regression models. Conversely, a cross-sectional study from the China Multi-Ethnic Cohort reported an insignificant association between PM_1 and diabetes (Wang et al. 2022). The insignificant result of Wang et al.'s study may be explained by sample size, study population, and region. In addition, we examined the C-R of PM_1 with diabetes. It is worth noting that the C-R curve was nearly linear, suggesting that even exposure to low-level PM_1 might lead to increased diabetes risk (Meng et al. 2021).

Positive relationships between PM_1 and hypertension risk identified in this study were reported in previous studies (Li et al. 2019; Niu et al. 2023; Yang et al. 2019). For example, a cross-sectional study of Chinese rural adults found that each 1 μg/m^3 increase in PM_1 concentration was associated with a 4.3% (OR = 1.043, 95%CI 1.033, 1.053) increase in hypertension (Li et al. 2019). Two meta-analyses also examined the association of long-term exposure to PM_1 with the prevalence of hypertension (Niu et al. 2022b; Qin et al. 2021). Qin et al. conducted a meta-analysis of 2 studies and found no significant association of PM_1 with hypertension (Qin et al. 2021). The insignificant result of Qin et al.'s study

may be due to the limited original studies. After Qin's study, we also conducted a meta-analysis of 4 studies and found a positive association between PM_1 with hypertension (Niu et al. 2023). This meta-analysis showed that each 10 μg/m^3 increase in PM_1 was associated with a 27% (OR = 1.27, 95%CI 1.06, 1.52) increase in hypertension risk, which was significantly higher than that of this study (OR = 1.11, 95%CI 1.06, 1.16). The inconsistency of the estimated effect might be due to the limited number of included studies (only 4 studies) and significant heterogeneity among the included studies (Niu et al. 2023). Moreover, our study also observed a slight "J" shape C-R curve for the relationships of PM_1 with hypertension. While no study examined the C-R for the relationships between PM_1 with hypertension, our findings could be supported by Song et al.'s study (Song et al. 2021). They found the C-R functions between $PM_{2.5}$ concentration and hypertension risk were presented as a slight "J" shape, which was consistent with our findings.

Several previous studies have examined the association of exposure to PM_1 with dyslipidemia (Gui et al. 2020; Hu et al. 2023; Mao et al. 2020b; Sun et al. 2023; Wang et al. 2021). For example, a cross-sectional study of 67,305 adults in the China Multi-Ethnic Cohort (CMEC) study found that exposure to PM_1 was associated with increased dyslipidemia risk. They found that each 10 μg/m^3 increase in PM_1 was associated with a 13% (OR = 1.13, 95%CI 1.06, 1.21) increase in dyslipidemia risk (Wang et al. 2021). A longitudinal study of 6976 middle-aged and older adults found that exposure to PM_1 was associated with increased dyslipidemia incidence. They found that each 10 μg/m^3 increase in PM_1 was associated with a 3% (OR = 1.03, 95%CI 1.01, 1.23) increase in dyslipidemia incidence (Hu et al. 2023). However, a cross-sectional study of Chinese rural adults reported that exposure to PM_1 was associated with decreased dyslipidemia (Mao et al. 2020b). In this study, we did not observe a statistically significant association between exposure to PM_1 and dyslipidemia risk. The inconsistency of results may be explained by various causes, such as diversity in the samples, study geographies, and methodology. Moreover, as a comprehensive indicator of blood lipid disorder, dyslipidemia is defined as an individual with one or more abnormal blood lipid concentrations, including hypercholesterolemia, hypertriglyceridemia, hypoalphalipoproteinemia, and hyperbetalipoproteinemia. The inconsistency of association between PM_1 and dyslipidemia may be attributed to the different results of these four indicators. For example, a cross-sectional study of 12,814 children and adolescents found exposure to PM_1 was associated with increased hypercholesterolemia risk (OR = 2.15, 95%CI 1.27, 3.65), but the associations of PM_1 with hypertriglyceridemia, hypoalphalipoproteinemia, and hyperbetalipoproteinemia were non-significant (Gui et al. 2020). Similar inconsistent results of PM_1 with these four indicators were also reported by Mao et al. (Mao et al. 2020b). Finally, as a recent meta-analysis mentioned, although PM_1 was associated with increased dyslipidemia risk, PM_1 was weakly correlated with four indicators of dyslipidemia (Sun et al. 2023). Further studies with large-scale and more regions should be conducted to provide more convincing evidence on the associations between PM_1 and dyslipidemia risk.

Five previous studies have investigated the associations of PM_1 with metabolic syndrome (Hou et al. 2020; Liu et al. 2023; Yang et al. 2018b; Zang et al. 2021; Zhang et al. 2021a). However, the results are still controversial. Three studies of Chinese adults reported positive associations between PM_1 and metabolic syndrome risk (Hou et al. 2020; Liu et al. 2023; Yang et al. 2018b). For example, Hou et al. conducted a cross-sectional study of 39,089 rural adults and indicated that each 5 μg/m^3 increase in PM_1 concentration was associated with a 25.1% (OR = 1.251, 95%CI 1.199, 1.306) increase in metabolic syndrome (Hou et al. 2020). In our study, we found that each 10 μg/m^3 increase in PM_1 was associated with a 25% (OR = 1.25, 95%CI 1.19, 1.31) increase in metabolic syndrome. We found that the estimated effect of our study was lower than that of Hou et al.'s study, which may be explained by more serious PM_1 pollution (the average PM_1 concentration 57.45 μg/m^3 for Hou et al.'s study, and 37.51 μg/m^3 for our study). However, significant associations between PM_1 and the prevalence of metabolic syndrome have not been found in a meta-analysis (Zang et al. 2021), children, and adolescents (Zhang et al. 2021a). The in-significant result of Zang et al.'s study may be explained by only 2 original research included, and the in-significant result of Zhang et al.'s study may be due to the different age composition of participants, different diagnostic criteria of metabolic syndrome in participants of different age groups. In our study, we found that each 10 μg/m^3 increase in PM_1 was associated with a 25% (OR = 1.25, 95%CI 1.19, 1.31) increase in metabolic syndrome, and the C-R curve of PM_1 with metabolic syndrome risk was nearly linear, which might provide new epidemiological evidence of adverse effects of long-term exposure to PM_1 on metabolic syndrome risk.

In addition to the robust positive associations of PM_1 exposure with abdominal obesity, diabetes, hypertension, and metabolic syndrome in all subgroups, we also indicated that cooking fuel use can modify the adverse effects of PM_1 on metabolic syndrome. Specifically, for each 10 μg/m^3 increase in PM_1, the OR values of metabolic syndrome were 1.26 (95%CI 1.21, 1.33) in solid fuel use participants and 1.23 (95%CI 1.17, 1.29) in clean fuel use participants, with P-interaction < 0.05. Several potential explanations should be noted. First, incomplete combustion of solid fuels also could produce PM_1 (Clark et al. 2013; Li et al. 2021), resulting in higher actual PM_1 exposure concentrations than the estimated exposure concentrations. Second, in addition to

PM_1, solid fuel burning can release carbon monoxide, sulfur dioxide, and other air pollutants (Gordon et al. 2014); those air pollutants would also lead to increased metabolic syndrome risks. Third, solid fuel users tend to have lower socioeconomic levels and worse living conditions than clean fuel users (Gordon et al. 2014; Li et al. 2021), which would accelerate the adverse effects of PM_1 on metabolic syndrome.

Potential biological mechanisms

As PM_1 is a major constituent of PM (Mao et al. 2020b), the biological mechanisms of adverse effects of PM_1 on metabolic diseases might be interpreted by several studies of PM and metabolic diseases. Although the specific biological mechanisms underlying the impact of PM exposure on the human metabolic system have not been fully understood, several biological mechanisms, including oxidative stress (Hu et al. 2021; Rao et al. 2018), systematic inflammation (Hahad et al. 2020), dysfunction autonomic nerve system, and DNA damage, have been proposed in previous studies. First, the PM could be inhaled and deposited in the respiratory system, as well as entry into the circulatory system directly (Chen et al. 2015; Xu et al. 2022), which could trigger oxidative stress and produce a large number of reactive oxygen species (ROS). Excessive reactive oxygen species (ROS) could primarily alter the structure and function of biomacromolecules, such as lipids and proteins, leading to metabolic system disorder (Qiu et al. 2019, Wang and Tang 2019). In addition to numerous epidemiological studies (Hu et al. 2021), previous animal studies also have linked PM exposure to oxidative stress and metabolic system disorder (Piao et al. 2018; Ren et al. 2020). For example, Ren et al. conducted an experimental study in zebrafish embryos and found that the extractable organic matter from $PM_{2.5}$ induced excessive ROS production and resulted in DNA damage and apoptosis, leading to cardiovascular system injury and metabolic system disorders (Ren et al. 2020). Second, systematic inflammation is an important biological mechanism of metabolic system disorders induced by PM (Tang et al. 2020; Xie et al. 2023). The inhaled PM could stimulate abnormal expression of inflammatory factors within cells, and the excessive release of these factors is a major cause of inflammation. Macrophages, which are an important defense barrier in the organism, could produce interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), and these factors could participate in the inflammatory response, ultimately leading to metabolic system disorders (Tang et al. 2020; Xie et al. 2023). Third, inhalation of PM may lead to the dysfunction autonomic nervous system, which has been reported as the crucial pathway of PM-induced inflammatory responses and metabolic system disorder increase (Niu et al. 2020; Xie et al. 2023). Xie et al. conducted an animal study of diesel exhaust $PM_{2.5}$ with pulmonary and systemic inflammation and indicated that the autonomic nervous system may play an important role in inflammatory responses induced by $PM_{2.5}$ (Xie et al. 2023). Fourth, particulate matter-induced DNA methylation is indicated as an important physiological mechanism of the adverse effects of PM exposure on metabolic systems and diseases (Chen et al. 2016; Eze et al. 2020; Wang et al. 2023).

Strengths and limitations

To our knowledge, this is the first study that examined the associations of PM_1 with four common metabolic diseases, including abdominal obesity, diabetes, hypertension, and metabolic syndrome. This study provided comprehensive epidemiological evidence for the adverse effects of exposure to PM_1 on metabolic diseases, especially abdominal obesity and metabolic syndrome. Moreover, C-R relationships of PM_1 with metabolic diseases were examined in this study, the slight "J" shapes for hypertension, and linear shapes for abdominal obesity, diabetes, and metabolic syndrome expand current knowledge on the relationships of exposure to PM_1 with metabolic diseases. The large sample size, nationwide coverage, and high spatiotemporal resolution of PM_1 assessment provided convincing epidemiological evidence for the adverse effects of PM_1 on metabolic systems.

Several limitations should be acknowledged in the study. First, the primary limitation was the cross-sectional study design. Cause and effect of the relationship between PM_1 and metabolic diseases could not be established. Longitudinal cohort studies are warranted to establish causal inference between PM_1 exposure and metabolic disease incidence. Second, the basic characteristics of participants, history of clinically diagnosed diseases, and medication information were collected using self-reported questionnaires, thus reporting bias and recall bias might exist (Han et al. 2022). Third, diet and other health lifestyles were not available in CHARLS, which might induce metabolic biomarkers changes (Wang et al. 2023); however, smoking status, drinking status, cooking fuel use, and physical activity were adjusted in the study and results suggested that the association of PM_1 with metabolic diseases were all robust. Fourth, anti-hypertension or anti-diabetic medication intake would also influence blood pressure and blood glucose (Chew et al. 2023; Niu et al. 2022a). However, the sensitivity analysis of excluding anti-hypertension or anti-diabetic medication users showed consistent and robust results. Fifth, several participants changed their living cities, which might lead to erroneous estimates in PM_1 exposure assessment (Yao et al. 2022). However, we conducted a sensitivity analysis excluding 498 participants who changed their address after 2013 and still observed the positive associations of PM_1 with

all four metabolic diseases. Finally, dietary factors and other lifestyle factors were not collected in the CHARLS, which might modify the associations of PM1 with metabolic diseases. Further studies that include control for more potential confounders are required to confirm our results.

Conclusion

In summary, this national cross-sectional study provides robust evidence for the adverse effects of exposure to higher PM_1 on metabolic disease risks, including abdominal obesity, diabetes, hypertension, and metabolic syndrome. Further longitudinal cohort studies are warranted to establish a causal inference between PM_1 exposure and metabolic disease risk.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11356-023-31098-z.

Author contribution Z.N. and Q.Z. contributed to the study conception and design; X.L., J.Z., Z.D., S.M., and J.W. contributed to the data collection; Q.Z., X.L., and J.Z. performed the data analysis and drafted the manuscript. Z.N. and S.H. helped revise the manuscript. All authors read and approved the final manuscript.

Funding This work was supported by the National Natural Science Foundation of China (No. 82102322).

Data availability Data will be made available on request.

Declarations

Ethical approval Study ethics approval of the CHARLS study was obtained from Peking University (Reference Number: IRB00001052–11015).

Consent to participate All participants and their guardians signed consent voluntarily.

Consent for publication Not applicable.

Competing interests The authors declare no competing interests.

References

Association AD (2021) 2. Classification and diagnosis of diabetes: standards of medical care in diabetes-2021. Diabetes Care 44:S15-s33

Azizi F, Hadaegh F, Hosseinpanah F, Mirmiran P, Amouzegar A, Abdi H, Asghari G, Parizadeh D, Montazeri SA, Lotfaliany M, Takyar F, Khalili D (2019) Metabolic health in the Middle East and North Africa. Lancet Diabetes Endocrinol 7:866–879

Barros AJ, Hirakata VN (2003) Alternatives for logistic regression in cross-sectional studies: an empirical comparison of models that directly estimate the prevalence ratio. BMC Med Res Methodol 3:21

Cao S, Guo Q, Xue T, Wang B, Wang L, Duan X, Zhang JJ (2021) Long-term exposure to ambient PM(2.5) increase obesity risk in Chinese adults: a cross-sectional study based on a nationwide survey in China. Sci Total Environ 778:145812

Chen R, Zhao Z, Sun Q, Lin Z, Zhao A, Wang C, Xia Y, Xu X, Kan H (2015) Size-fractionated particulate air pollution and circulating biomarkers of inflammation, coagulation, and vasoconstriction in a panel of young adults. Epidemiology 26:328–336

Chen R, Meng X, Zhao A, Wang C, Yang C, Li H, Cai J, Zhao Z, Kan H (2016) DNA hypomethylation and its mediation in the effects of fine particulate air pollution on cardiovascular biomarkers: a randomized crossover trial. Environ Int 94:614–619

Chen X, Crimmins E, Hu PP, Kim JK, Meng Q, Strauss J, Wang Y, Zeng J, Zhang Y, Zhao Y (2019) Venous blood-based biomarkers in the China health and retirement longitudinal study: rationale, design, and results from the 2015 wave. Am J Epidemiol 188:1871–1877

Chen M, Qin Q, Liu F, Wang Y, Wu C, Yan Y, Xiang H (2022) How long-term air pollution and its metal constituents affect type 2 diabetes mellitus prevalence? Results from Wuhan Chronic Disease Cohort. Environ Res 212:113158

Chew NWS et al (2023) The global burden of metabolic disease: data from 2000 to 2019. Cell Metab 35(414–428):e3

Chong B et al (2023) The global syndemic of metabolic diseases in the young adult population: A consortium of trends and projections from the Global Burden of Disease 2000–2019. Metabolism 141:155402

Clark ML, Peel JL, Balakrishnan K, Breysse PN, Chillrud SN, Naeher LP, Rodes CE, Vette AF, Balbus JM (2013) Health and household air pollution from solid fuel use: the need for improved exposure assessment. Environ Health Perspect 121:1120–1128

Collaborators GRF (2020) Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet 396:1223–1249

Eckel RH, Grundy SM, Zimmet PZ (2005) The metabolic syndrome. Lancet 365:1415–1428

Eze IC, Jeong A, Schaffner E, Rezwan FI, Ghantous A, Foraster M, Vienneau D, Kronenberg F, Herceg Z, Vineis P, Brink M, Wunderli JM, Schindler C, Cajochen C, Röösli M, Holloway JW, Imboden M, Probst-Hensch N (2020) Genome-wide DNA methylation in peripheral blood and long-term exposure to source-specific transportation noise and air pollution: the SAPALDIA study. Environ Health Perspect 128:67003

Gordon SB et al (2014) Respiratory risks from household air pollution in low and middle income countries. Lancet Respir Med 2:823–860

Gui ZH, Yang BY, Zou ZY, Ma J, Jing J, Wang HJ, Dong GH, Ma YH, Guo YM, Chen YJ (2020) Exposure to ambient air pollution and blood lipids in children and adolescents: a national population based study in China. Environ Pollut 266:115422

Guo B, Guo Y, Nima Q, Feng Y, Wang Z, Lu R, Baimayangji MY, Zhou J, Xu H, Chen L, Chen G, Li S, Tong H, Ding X, Zhao X, Cohort C-E, collaborative g, (2022) Exposure to air pollution is associated with an increased risk of metabolic dysfunction-associated fatty liver disease. J Hepatol 76:518–525

Hahad O, Lelieveld J, Birklein F, Lieb K, Daiber A, Münzel T (2020) Ambient air pollution increases the risk of cerebrovascular and neuropsychiatric disorders through induction of inflammation and oxidative stress. Int J Mol Sci 21(12):4306

Han S, Zhang F, Yu H, Wei J, Xue L, Duan Z, Niu Z (2022) Systemic inflammation accelerates the adverse effects of air pollution on metabolic syndrome: findings from the China health and retirement longitudinal study (CHARLS). Environ Res 215:114340

Hou J, Liu X, Tu R, Dong X, Zhai Z, Mao Z, Huo W, Chen G, Xiang H, Guo Y, Li S, Wang C (2020) Long-term exposure to ambient air pollution attenuated the association of physical activity with metabolic syndrome in rural Chinese adults: a cross-sectional study. Environ Int 136:105459

Hou W, Chen S, Zhu C, Gu Y, Zhu L, Zhou Z (2023) Associations between smoke exposure and osteoporosis or osteopenia in a US NHANES population of elderly individuals. Front Endocrinol 14:1074574

Hu W, Wang Y, Wang T, Ji Q, Jia Q, Meng T, Ma S, Zhang Z, Li Y, Chen R, Dai Y, Luan Y, Sun Z, Leng S, Duan H, Zheng Y (2021) Ambient particulate matter compositions and increased oxidative stress: exposure-response analysis among high-level exposed population. Environ Int 147:106341

Hu M, Wei J, Hu Y, Guo X, Li Z, Liu Y, Li S, Xue Y, Li Y, Liu M, Wang L, Liu X (2023) Long-term effect of submicronic particulate matter (PM(1)) and intermodal particulate matter (PM(1–2.5)) on incident dyslipidemia in China: a nationwide 5-year cohort study. Environ Res 217:114860

Letellier N, Zamora S, Spoon C, Yang JA, Mortamais M, Escobar GC, Sears DD, Jankowska MM, Benmarhnia T (2022) Air pollution and metabolic disorders: dynamic versus static measures of exposure among Hispanics/Latinos and non-Hispanics. Environ Res 209:112846

Li N, Chen G, Liu F, Mao S, Liu Y, Hou Y, Lu Y, Liu S, Wang C, Xiang H, Guo Y, Li S (2019) Associations of long-term exposure to ambient PM(1) with hypertension and blood pressure in rural Chinese population: the Henan rural cohort study. Environ Int 128:95–102

Li N, Chen G, Liu F, Mao S, Liu Y, Liu S, Mao Z, Lu Y, Wang C, Guo Y, Xiang H, Li S (2020) Associations between long-term exposure to air pollution and blood pressure and effect modifications by behavioral factors. Environ Res 182:109109

Li C, Zhou Y, Ding L (2021) Effects of long-term household air pollution exposure from solid fuel use on depression: evidence from national longitudinal surveys from 2011 to 2018. Environ Pollut 283:117350

Liu F, Chen G, Huo W, Wang C, Liu S, Li N, Mao S, Hou Y, Lu Y, Xiang H (2019a) Associations between long-term exposure to ambient air pollution and risk of type 2 diabetes mellitus: a systematic review and meta-analysis. Environ Pollut 252:1235–1245

Liu F, Guo Y, Liu Y, Chen G, Wang Y, Xue X, Liu S, Huo W, Mao Z, Hou Y, Lu Y, Wang C, Xiang H, Li S (2019b) Associations of long-term exposure to PM(1), PM(2.5), NO(2) with type 2 diabetes mellitus prevalence and fasting blood glucose levels in Chinese rural populations. Environ Int 133:105213

Liu T et al (2019c) Gut microbiota partially mediates the effects of fine particulate matter on type 2 diabetes: evidence from a population-based epidemiological study. Environ Int 130:104882

Liu YH, Lu YK, Liu XT, Li YL, Hu LK, Gao HY, Yang K, Yan YX (2021) Association of household solid fuel use and long-term exposure to PM(2.5) with arthritis in middle-aged and older population in China: a cohort study. Ecotoxicol Environ Saf 230:113104

Liu F, Wang X, Pan M, Zhang K, Zhou F, Tong J, Chen Z, Xiang H (2023) Exposure to air pollution and prevalence of metabolic syndrome: a nationwide study in China from 2011 to 2015. Sci Total Environ 855:158596

Mao S, Chen G, Liu F, Li N, Wang C, Liu Y, Liu S, Lu Y, Xiang H, Guo Y, Li S (2020a) Long-term effects of ambient air pollutants to blood lipids and dyslipidemias in a Chinese rural population. Environ Pollut 256:113403

Mao S, Li S, Wang C, Liu Y, Li N, Liu F, Huang S, Liu S, Lu Y, Mao Z, Huo W, Chen G, Xiang H, Guo Y (2020b) Is long-term PM(1) exposure associated with blood lipids and dyslipidemias in a Chinese rural population? Environ Int 138:105637

Mei Y, Li A, Zhao J, Zhou Q, Zhao M, Xu J, Li R, Li Y, Li K, Ge X, Guo C, Wei Y, Xu Q (2023) Association of long-term air pollution exposure with the risk of prediabetes and diabetes: systematic perspective from inflammatory mechanisms, glucose homeostasis pathway to preventive strategies. Environ Res 216:114472

Meng X et al (2021) Short term associations of ambient nitrogen dioxide with daily total, cardiovascular, and respiratory mortality: multilocation analysis in 398 cities. BMJ 372:n534

Nie C, Li Z, Yang T, Zhong J, Liu Q, Mi F, Yu J, Pan Y, Kan H, Hong F (2023) Associations of long-term exposure to particulate matter with gallstone risks in Chinese adults: a large cross-sectional study. Ecotoxicol Environ Saf 252:114644

Niu Z, Liu F, Li B, Li N, Yu H, Wang Y, Tang H, Chen X, Lu Y, Cheng Z, Liu S, Chen G, Zhang Y, Xiang H (2020) Acute effect of ambient fine particulate matter on heart rate variability: an updated systematic review and meta-analysis of panel studies. Environ Health Prev Med 25:77

Niu Z, Duan Z, Wei J, Wang F, Han D, Zhang K, Jing Y, Wen W, Qin W, Yang X (2022a) Associations of long-term exposure to ambient ozone with hypertension, blood pressure, and the mediation effects of body mass index: a national cross-sectional study of middle-aged and older adults in China. Ecotoxicol Environ Saf 242:113901

Niu Z, Duan Z, Yu H, Xue L, Liu F, Yu D, Zhang K, Han D, Wen W, Xiang H, Qin W (2023) Association between long-term exposure to ambient particulate matter and blood pressure, hypertension: an updated systematic review and meta-analysis. Int J Environ Health Res 33:268–283

Niu Z, Duan Z, Yu H, Xue L, Liu F, Yu D, Zhang K, Han D, Wen W, Xiang H, Qin W (2022b) Association between long-term exposure to ambient particulate matter and blood pressure, hypertension: an updated systematic review and meta-analysis. Int J Environ Health Res 33(3):268–283

Piao MJ, Ahn MJ, Kang KA, Ryu YS, Hyun YJ, Shilnikova K, Zhen AX, Jeong JW, Choi YH, Kang HK, Koh YS, Hyun JW (2018) Particulate matter 2.5 damages skin cells by inducing oxidative stress, subcellular organelle dysfunction, and apoptosis. Arch Toxicol 92:2077–2091

Qin P, Luo X, Zeng Y, Zhang Y, Li Y, Wu Y, Han M, Qie R, Wu X, Liu D, Huang S, Zhao Y, Feng Y, Yang X, Hu F, Sun X, Hu D, Zhang M (2021) Long-term association of ambient air pollution and hypertension in adults and in children: a systematic review and meta-analysis. Sci Total Environ 796:148620

Qiu YN, Wang GH, Zhou F, Hao JJ, Tian L, Guan LF, Geng XK, Ding YC, Wu HW, Zhang KZ (2019) PM2.5 induces liver fibrosis via triggering ROS-mediated mitophagy. Ecotoxicol Environ Saf 167:178–187

Rao X, Zhong J, Brook RD, Rajagopalan S (2018) Effect of particulate matter air pollution on cardiovascular oxidative stress pathways. Antioxid Redox Signal 28:797–818

Ren F, Ji C, Huang Y, Aniagu S, Jiang Y, Chen T (2020) AHR-mediated ROS production contributes to the cardiac developmental toxicity of PM2.5 in zebrafish embryos. Sci Total Environ 719:135097

Ren M, Zhang C, Di J, Chen H, Huang A, Ji JS, Liang W, Huang C (2023) Exploration of the preterm birth risk-related heat event thresholds for pregnant women: a population-based cohort study in China. Lancet Reg Health West Pacif 37:100785

Song J, Gao Y, Hu S, Medda E, Tang G, Zhang D, Zhang W, Li X, Li J, Renzi M, Stazi MA, Zheng X (2021) Association of long-term exposure to PM(2.5) with hypertension prevalence and blood pressure in China: a cross-sectional study. BMJ Open 11:e050159

Steenblock C et al (2021) COVID-19 and metabolic disease: mechanisms and clinical management. Lancet Diabetes Endocrinol 9:786–798

Sun M, Li T, Sun Q, Ren X, Sun Z, Duan J (2023) Associations of long-term particulate matter exposure with cardiometabolic diseases: a systematic review and meta-analysis. Sci Total Environ 903:166010

Tang H, Cheng Z, Li N, Mao S, Ma R, He H, Niu Z, Chen X, Xiang H (2020) The short- and long-term associations of particulate

matter with inflammation and blood coagulation markers: a meta-analysis. Environ Pollut 267:115630
Wang Y, Tang M (2019) PM2.5 induces ferroptosis in human endothelial cells through iron overload and redox imbalance. Environ Pollut 254:112937
Wang L, Chen G, Pan Y, Xia J, Chen L, Zhang X, Silang Y, Chen J, Xu H, Zeng C, Wei J, Li S, Guo Y, Yang S, Hong F, Zhao X, Cohort C-E, collaborative g, (2021) Association of long-term exposure to ambient air pollutants with blood lipids in Chinese adults: the China Multi-Ethnic Cohort study. Environ Res 197:111174
Wang X, Guo B, Yang X, Li J, Baima Y, Yin J, Yu J, Xu H, Zeng C, Feng S, Wei J, Hong F, Zhao X (2022) Role of liver enzymes in the relationship between particulate matter exposure and diabetes risk: a longitudinal cohort study. J Clin Endocrinol Metab 107:e4086–e4097
Wang Y, Tan H, Zheng H, Ma Z, Zhan Y, Hu K, Yang Z, Yao Y, Zhang Y (2023) Exposure to air pollution and gains in body weight and waist circumference among middle-aged and older adults. Sci Total Environ 869:161895
Wei J, Li Z, Guo J, Sun L, Huang W, Xue W, Fan T, Cribb M (2019) Satellite-derived 1-km-resolution PM(1) concentrations from 2014 to 2018 across China. Environ Sci Technol 53:13265–13274
Wei J, Li Z, Chen X, Li C, Sun Y, Wang J, Lyapustin A, Brasseur G, Jiang M, Sun L, Wang T, Jung C, Qiu B, Fang C, Liu X, Hao J, Wang Y, Zhan M, Song X, Liu Y (2023) Separating daily 1 km $PM_{2.5}$ inorganic composition in China since 2000 via deep learning integrating ground, satellite, and model data. Environ Sci Technol 57:18282–18295
Weng Z, Liu Q, Yan Q, Liang J, Zhang X, Xu J, Li W, Xu C, Gu A (2022) Associations of genetic risk factors and air pollution with incident hypertension among participants in the UK Biobank study. Chemosphere 299:134398
Wu Y, Wang W, Liu C, Chen R, Kan H (2020) The association between long-term fine particulate air pollution and life expectancy in China, 2013 to 2017. Sci Total Environ 712:136507
Xie Y, Tao S, Pan B, Yang W, Shao W, Fang X, Han D, Li J, Zhang Y, Chen R, Li W, Xu Y, Kan H (2023) Cholinergic anti-inflammatory pathway mediates diesel exhaust PM(2.5)-induced pulmonary and systemic inflammation. J Hazard Mater 458:131951
Xu R, Wei J, Liu T, Li Y, Yang C, Shi C, Chen G, Zhou Y, Sun H, Liu Y (2022) Association of short-term exposure to ambient PM(1) with total and cause-specific cardiovascular disease mortality. Environ Int 169:107519
Yang BY et al (2018a) Ambient air pollution in relation to diabetes and glucose-homoeostasis markers in China: a cross-sectional study with findings from the 33 Communities Chinese Health Study. Lancet Planet Health 2:e64–e73
Yang BY, Qian ZM, Li S, Fan S, Chen G, Syberg KM, Xian H, Wang SQ, Ma H, Chen DH, Yang M, Liu KK, Zeng XW, Hu LW, Guo Y, Dong GH (2018b) Long-term exposure to ambient air pollution (including PM(1)) and metabolic syndrome: the 33 Communities Chinese Health Study (33CCHS). Environ Res 164:204–211
Yang BY et al (2019) Ambient PM(1) air pollution, blood pressure, and hypertension: insights from the 33 Communities Chinese Health Study. Environ Res 170:252–259
Yao Y, Lu T, Liu Y, Qin Q, Jiang J, Xiang H (2022) Association of depressive symptoms with ambient PM(2.5) in middle-aged and elderly Chinese adults: a cross-sectional study from the China health and Retirement Longitudinal Study wave 4. Environ Res 203:111889
Yitshak Sade M, Shi L, Colicino E, Amini H, Schwartz JD, Di Q, Wright RO (2023) Long-term air pollution exposure and diabetes risk in American older adults: a national secondary data-based cohort study. Environ Pollut 320:121056
Zang ST, Luan J, Li L, Wu QJ, Chang Q, Dai HX, Zhao YH (2021) Air pollution and metabolic syndrome risk: evidence from nine observational studies. Environ Res 202:111546
Zhang JS, Gui ZH, Zou ZY, Yang BY, Ma J, Jing J, Wang HJ, Luo JY, Zhang X, Luo CY, Wang H, Zhao HP, Pan DH, Bao WW, Guo YM, Ma YH, Dong GH, Chen YJ (2021a) Long-term exposure to ambient air pollution and metabolic syndrome in children and adolescents: a national cross-sectional study in China. Environ Int 148:106383
Zhang Z, Dong B, Chen G, Song Y, Li S, Yang Z, Dong Y, Wang Z, Ma J, Guo Y (2021b) Ambient air pollution and obesity in school-aged children and adolescents: a multicenter study in China. Sci Total Environ 771:144583
Zhang J, Zhang F, Xin C, Duan Z, Wei J, Zhang X, Han S, Niu Z (2023) Associations of long-term exposure to air pollution, physical activity with blood pressure and prevalence of hypertension: the China Health and Retirement Longitudinal Study. Front Public Health 11:1137118
Zhao Y, Hu Y, Smith JP, Strauss J, Yang G (2014) Cohort profile: the China Health and Retirement Longitudinal Study (CHARLS). Int J Epidemiol 43:61–68
Zheng H, Xu Z, Wang Q, Ding Z, Zhou L, Xu Y, Su H, Li X, Zhang F, Cheng J (2021) Long-term exposure to ambient air pollution and obesity in school-aged children and adolescents in Jiangsu province of China. Environ Res 195:110804
Zheng XY, Tang SL, Liu T, Wang Y, Xu XJ, Xiao N, Li C, Xu YJ, He ZX, Ma SL, Chen YL, Meng RL, Lin LF (2022) Effects of long-term PM(2.5) exposure on metabolic syndrome among adults and elderly in Guangdong, China. Environ Health 21:84

Authors and Affiliations

Qin Zhou[1] · Xianfeng Li[2] · Jinglong Zhang[3] · Zhizhou Duan[4] · Shuyuan Mao[5] · Jing Wei[6] · Shichao Han[7] · Zhiping Niu[8]

✉ Zhiping Niu
zhiping_niu@whu.edu.cn

1 Department of Orthodontics, College of Stomatology, Xi'an Jiaotong University, No. 98 XiWu Road, Xi'an 710004, Shaanxi, China

2 Department of Reproductive Service Technology, Urumqi Maternal and Child Health Hospital, No. 344 Jiefang South Road, Tianshan District, Urumqi 830000, China

3 Department of Cardiovascular Surgery, Xijing Hospital, The Fourth Military Medical University, 127 West Changle Road, Xi'an 710032, China

4 Preventive Health Service, Jiangxi Provincial People's Hospital, The First Affiliated Hospital of Nanchang Medical College, 152 Aiguo Road, Nanchang, Jiangxi, China

5 The Third Affiliated Hospital of Zhengzhou University, 7 Kangfu Road, Zhengzhou 450000, Henan, China

6 Department of Atmospheric and Oceanic Science, Earth System Science Interdisciplinary Center, University of Maryland, College Park, USA

7 Department of Urology, Xijing Hospital, The Fourth Military Medical University, 127 West Changle Road, Xi'an 710032, China

8 Department of Environmental Health, School of Public Health, Fudan University, 196 Xietu Road, Shanghai 200032, China