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Systemic inflammation accelerates the adverse effects of air pollution on metabolic syndrome: Findings from the China health and Retirement Longitudinal Study (CHARLS)

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ABSTRACT

Long-term exposure to air pollution and systemic inflammation are associated with increased prevalence of metabolic syndrome (MetS); however, their joint effects in Chinese middle-aged and older adults is unknown. In this cross-sectional study, 11,838 residents aged 45 years and older from the China Health and Retirement Longitudinal Study (CHARLS) Wave 3 in 2015 were included. MetS was diagnosed using the Joint Interim Societies' definition. C-Reactive Protein (CRP) was assessed to reflect systemic inflammation. Individual exposure to air pollutants (particulate matter with a diameter $\leq$2.5 μm ($PM_{2.5}$) or $\leq$ 10 μm (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), and carbon monoxide (CO)) was evaluated using satellite-based spatiotemporal models according to participant residence at county-level. Generalized linear models (GLMs) were applied to examine the association between air pollution and MetS, and the modification effects of CRP between air pollution and MetS were estimated using interaction terms of CRP and air pollutants in the GLM models. The prevalence of MetS was 32.37%. The adjusted odd ratio (OR) of MetS was 1.192 (95% confidence interval (CI): 1.116, 1.272), 1.177 (95% CI: 1.103, 1.255), 1.158 (95% CI: 1.072, 1.252), 1.303 (95% CI: 1.211,1.403), 1.107 (95% CI: 1.046, 1.171) and 1.156 (95% CI:1.083, 1.234), per inter-quartile range increase in $PM_{2.5}$ (24.04 μg/m^3), PM_{10} (39.00 μg/m^3), SO_2 (19.05 μg/m^3), NO_2 (11.28 μg/m^3), O_3 (9.51 μg/m^3) and CO (0.46 mg/m^3), respectively. CRP was also associated with increased prevalence of MetS (OR = 1.049, 95% CI: 1.035, 1.064; per 1.90 mg/L increase in CRP). Interaction analysis suggested that high CRP levels enhanced the association between air pollution exposure and MetS. Long-term exposure to air pollution is associated with increased prevalence of MetS, which might be enhanced by systemic inflammation. Given the rapidly aging society and heavy burden of MetS, measures should be taken to improve air quality and reduce systemic inflammation.

1. Introduction

Metabolic syndrome (MetS) is defined as a cluster of metabolic disorders, including abdominal obesity, dyslipidemia, hypertension, and diabetes (Alberti et al., 2005; Cornier et al., 2008), which has become a major public health challenge worldwide. The prevalence of MetS is about 20–25% globally and the prevalence has continued to rise in recent years (O'Neill and O'Driscoll, 2015; Pucci et al., 2017). While genetic defects, low physical activity, and an unhealthy diet have been well-demonstrated as crucial risk factors for MetS (Wang et al., 2022;

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Yang et al., 2020), other hazardous factors, such as air pollutants might also induce MetS (Wang et al., 2022; Zang et al., 2021).

Exposure to ambient air pollution is the most common environmental risk factor for MetS-related diseases, including hypertension, diabetes, obesity, and dyslipidemia (Li et al., 2020; Liu et al., 2019b; Mao et al., 2020a; Niu et al., 2022a, 2022b). However, evidence on the long-term effects of exposure to air pollution on MetS is limited (Hou et al., 2020; Wang et al., 2022). A recent meta-analysis of nine studies found no significant association between air pollution (particulate matter with a diameter $\leq$2.5 μm or 10 μm in diameter, $PM_{2.5}$, PM_{10}; nitrogen dioxide, NO_2) and MetS (Zang et al., 2021). A recent study in China found that higher $PM_{2.5}$, PM_{10}, and ozone (O_3) levels were associated with elevated MetS prevalence, whereas negative and insignificant association were observed for NO_2 and sulfur dioxide (SO_2) (Wang et al., 2022). The Heinz Nixdorf Recall study in Germany reported a significant association of NO_2 with MetS prevalence, whereas no significant association was observed for $PM_{2.5}$ and PM_{10} (Matthiessen et al., 2018). In addition to the uncertainty in epidemiological findings, the majority of studies focused on the effects of particulate matter and NO_2, with little attention being paid to SO_2, O_3, and carbon monoxide (CO).

Systemic inflammation has been identified as an important predictor of MetS and MetS-related diseases (Dabass et al., 2018). Moreover, systemic inflammation has been demonstrated as a crucial mechanism of air pollution-induced adverse health effects, including many cardiovascular, respiratory, and metabolic diseases (Dadvand et al., 2014; Liu et al., 2019d; Puett et al., 2019). While no study has examined the effect modification of systemic inflammation in the associations of air pollution with MetS, those previous studies suggested that systemic inflammation might modify the association between air pollution and MetS.

In this national cross-sectional study, we aimed to investigate associations of long-term exposure to air pollution or systemic inflammation with MetS and their interaction with MetS using the China Health and Retirement Longitudinal Study (CHARLS).

2. Methods

2.1. Study population

The study participants were from the China Health and Retirement Longitudinal Study (CHARLS), which is a national cohort study among residents aged 45 years and older. The CHARLS survey has covered about 150 county-level cities in 28 provinces of China and was established to collect a wide range of high-quality microdata of middle-aged and older adults. In brief, multi-stage stratified probability proportionate sampling methods were employed to recruit participants. After the CHARLS 2011 baseline assessment, the CHARLS proceeded to follow-up with participants in three waves in 2013, 2015, and 2018, respectively. As the CHARLS is dynamic cohort, a small number of new respondents are recruited in the follow-ups (Zhang et al., 2021). Detailed information of CHARLS has been published previously (Zhao et al., 2014). Blood lipid, blood pressure, fasting plasma glucose, and other physical examinations were only conducted in CHARLS 2011 and 2015, and the assessment of air pollution in China was mainly performed after 2013; therefore, we conducted a national cross-sectional study of CHARLS in 2015 in this study. A total of 16,406 middle-aged and older adults who participated in physical examinations were enrolled. After excluding participants without blood lipid, blood pressure, and fasting plasma glucose results and those participants in the very few regions where air pollution exposure failed to be assessed, we enrolled 11,838 middle-aged and older adults from 27 provinces of China in the present study (Fig. S1). Informed consent was obtained from all participants and CHARLS was approved by the Institutional Review Board of Peking University (Code: IRB00001052-11015).

2.2. Assessment of waist circumference, blood pressure, blood liquids, plasma glucose, and systemic inflammation

Waist circumference (WC) and blood pressure of each participant were measured in a free physical examination. Briefly, WC at the level of the navel was evaluated using a tape measure, and participants were asked to keep a standing position and hold their breath at end of the exhale. The blood pressure of each individual was measured three times using an electronic monitor (HEM-7200 Monitor, Omron, Kyoto, Japan) on the left arm. Participants were asked not to smoke, exercise, drink alcohol, and take food within 30 min before the blood pressure test. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were calculated by averaging the results of three examinations.

A fasting venous blood sample (6 mL) was collected from each participant for the assessments of blood liquids, plasma glucose, and systemic inflammation. Briefly, after the venous blood was separated, plasma from each participant was stored in two 1 mL cryovials and then shipped to the KingMed Diagnostics laboratory to assay plasma glucose and systemic inflammation. Triglycerides (TG), high-density lipoprotein (HDL), and fasting plasma glucose (FPG) were examined for the diagnosis of MetS. High-sensitivity C-reactive protein (hsCRP) was assessed to reflect systemic inflammation levels. More detailed descriptions of the process of blood collection and the methods for blood-based bioassays are described in our previous study (Chen et al., 2019).

2.3. Diagnosis of MetS

The diagnosis of MetS was carried out according to the Joint Interim Societies' definition of MetS, using their categorizations of WC for Chinese people (Alberti et al., 2005). In brief, an individual with WC $\geq$ 80 cm for females and WC $\geq$ 90 cm for males plus who met any two of the following criteria were diagnosed as MetS patient: (1)TG > 150 mg/dL (1.7 mmol/L); (2) HDL <40 mg/dL (1.03 mmol/L) in men or < 50 mg/dL (1.29 mmol/L) in women; (3) SBP $\geq$130 mm Hg or DBP $\geq$85 mm Hg or use of anti-hypertensive medicine; (4) FPG $\geq$100 mg/dL (5.6 mmol/L) or the use of anti-diabetic medicine (Hou et al., 2020).

2.4. Assessments of air pollution exposure

Full-coverage ground-level air pollution concentrations ($PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3 and CO) for each individual was assessed by artificial intelligence at 0.1° (≈10 km) gridded spatial resolution from 2013 to 2015, which were collected from the CHAP dataset (available at https://weijing-rs.github.io/product.html). Briefly, ground-based measurements, remote sensing products, atmospheric reanalysis, and model simulations were all employed and the space-time extremely randomized trees (STET) model was used to estimate the daily concentrations of ambient $PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3. The 10-fold cross-validation R^2 and Root-Mean-Square Error of the estimated daily averages of all air pollutants showed high consistency with surface measurements (Table S1). The detailed data on air pollution estimation are described in previous studies (Wei et al., 2019, 2021a, 2021b, 2022a, 2022b). The annual air pollution exposure of each participant was estimated according to their residential addresses at the county-level and three-year average air pollution concentrations before the CHARLS wave 3 were calculated as long-term air pollution exposure in the main effects analyses, while two-year average air pollution concentrations were used in the sensitivity analysis (Mao et al., 2020b).

2.5. Covariates

Numerous potential confounders were enrolled in this study, including meteorological factors, sociodemographic, socioeconomic variables, health behaviors, and lifestyles. Meteorological factors (temperature, relative humidity) were assessed by meteorological monitoring stations and the data was obtained from the China Meteorological

Administration (http://www.cma.gov.cn/). Sociodemographic variables included age (years) and sex (male or female). Socioeconomic variables included residence (rural or urban), education status ("elementary school or below" or "middle school or above"), marital status ("married and living with a spouse", "married but living without a spouse", or "single, divorced, and widowed"), and annual household expenditure. Health behaviors and lifestyles included smoking status ("non-smoker" or "smoker"), drinking status ("non-drinker" or "drink less than once a month" or "drink more than once a month"), physical activity (PA), and cooking fuel use ("solid fuel" or "clean fuel") (Li et al., 2019). For smoking status, a smoker was defined as an individual who had ever chewed tobacco, smoked a pipe, or self-rolled cigarettes, cigarettes, or cigars. Drinking status was assessed by investigating the alcoholic behavior of each participant in the past year and alcohol types contained liquor, wine, or beer. Physical activity was assessed by the International Physical Activity Questionnaire (IPAQ). The time range in CHARLS was converted to an intermediate value: "≥ 4 h" to 240 min, "≥ 2 h and <4 h" to 180 min, "≥ 30 min and <2 h" to 75 min, and "≥10 min and <30 min" and the physical activity score (PA score) was evaluated using metabolic equivalent multipliers as follows: PA score = 8.0 × total vigorous activity weekly duration score + 4.0 × total moderate activity weekly duration score + 3.3 × total walking weekly duration score (Bai et al., 2021; Garrett et al., 2019). Indoor air pollution was evaluated by whether a participant used solid fuel for cooking (Luo et al., 2021). Coal, crop residue, or wood burning were categorized as "solid fuel" and natural gas, marsh gas, and liquefied petroleum gas were categorized as "clean fuel" (Luo et al., 2021).

2.6. Statistical analysis

Descriptive analyses were conducted. Continuous variables were described as the mean ± standard deviation (SD) and categorical variables were represented as count data (percentage). The difference in continuous variables and categorical variables between non-MetS and MetS participants was tested using Student's t-test and a chi-squared test (Wang et al., 2022). Pearson correlation was applied to examine the correlations among different air pollutants.

Generalized linear models (GLM) were employed to examine the effects of air pollution and CRP on MetS, and their interactions on MetS. The effect estimates and 95% confidence intervals (95% CI) were presented as the odds ratio (OR) for MetS per interquartile range (IQR) increase in air pollutants. We first developed a crude model without any adjustment. Then, adjusted model 1 (Model 2) was employed to adjust for meteorological factors (temperature and relative humidity), sociodemographic (age and sex) and socioeconomic variables (residence, education, marital status, and annual household expenditure) (Butland et al., 2017). Finally, health behaviors and lifestyles (smoking status, drinking status, physical activity and cooking fuel use) were also adjusted in adjusted model 2 (Model 3). McFadden's pseudo R^2 was used to assess model fit (Park et al., 2022). In addition, we also included standardized concentrations of air pollution into model and compared the OR values of air pollutants to identify which air pollution component is more responsible for increased prevalence of MetS than the others.

To evaluate the effects of CRP on MetS and its modification effects in the association of air pollution with MetS, three models were carried out based on fully-adjusted models. Firstly, we assess the association of CRP with MetS, without any air pollutant (Model 4). Then, an additive term of CRP and each air pollutant were included in the same model (Model 5). Finally, an interaction term of CRP and each air pollutant were applied to test the interactive effects of CRP and individual air pollutant (Model 6) (Hou et al., 2020).

A series of sensitivity analyses were performed to test the robustness of the results. First, we re-performed GLM models and interactive analysis using the two-year average concentration of air pollutants before 2015. Second, we additionally adjusted for regional categories ("East", "Midland" and "West") considering the level of economic development with geographical difference. Third, we excluded participants who had changed their address between 2011 and 2015. Fourth, log-binomial Poisson regressions were performed to examine the associations of air pollution and CRP with MetS, and the modification effects of CRP in the associations of air pollution with MetS, accounting for the high prevalence of the binary outcomes of interest (Barros and Hirakata, 2003; Ning et al., 2022). Fifth, we categorized participants into a "Low CRP group (<1 mg/L)" and "Average or high CRP group (≥1 mg/L)" and performed a sensitivity analysis using an interaction between categorized CRP and air pollutants (Luttmann-Gibson et al., 2010; Pearson et al., 2003). Finally, two-pollutant models with adjustment for O_3 were developed, accounting for the weak or moderate co-linearity of O_3 and other air pollutants (Liu et al., 2019a).

All statistical analyses were completed using the R software (Version 4.2.1) and the imputation of missing data for several covariates were completed by using the "mice" package (Tan et al., 2018). A two-tailed P value < 0.05 was regarded as statistically significant for the association of CRP/air pollution and MetS, and a two-tailed P value < 0.10 was set as the statistical significance threshold for their interactions of CRP and air pollution (Hou et al., 2020).

3. Results

3.1. Descriptive statistics

A total of 11,838 middle-aged and older adults were included in our study, with only 179 new respondents (1.51% of all participants). Participants were recruited from 437 communities in 123 county-level cities in 27 Chinese provinces. The geographical distribution of the participants is shown in Fig. 1 and the flowchart for participant inclusion is shown in Fig. S1. The basic characteristics of participants are shown in Table 1. The mean age of participants was 60.63 ± 9.67 years. The mean levels of WC, TG, HDL, FBG, SBP, DBP, and CRP were 85.78 cm, 1.61 mmol/L, 1.32 mmol/L, 128.61 mmHg, 123.66 mmHg, and 2.69 mg/L, respectively. There were 3832 middle-aged and older adults diagnosed as having MetS, with a prevalence of 32.37%.

Table 2 presents the average concentrations of six air pollutants, temperature, and humidity. A three-year average ambient $PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3, and CO exposure were 62.99 ± 18.88 μg/m^3, 104.97 ± 32.71 μg/m^3, 34.29 ± 14.19 μg/m^3, 32.29 ± 7.63 μg/m^3, 84.82 ± 7.18 μg/m^3 and 1.29 ± 0.38 mg/m^3, respectively. We found that the annual $PM_{2.5}$ and PM_{10} exposure were far greater than the standards of the World Health Organization (AQG 2021; $PM_{2.5}$: 5 μg/m^3, PM_{10}:10 μg/m^3) and the secondary standard of Chinese ambient air quality guideline (GB 3095–2012, $PM_{2.5}$: 35 μg/m^3, PM_{10}:70 μg/m^3). The mean temperature and humidity were 15.24 ± 4.04 °C and 67.75 ± 8.38%. In addition to O_3, Pearson correlation analyses suggested that $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO were highly correlated with each other, with the correlation coefficient (r) ranging from 0.748 to 0.952 (Table S2).

3.2. Association between air pollution and the prevalence of MetS

The associations between each air pollutant and the prevalence of MetS are shown in Fig. 2. Long-term exposures to ambient $PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3, and CO were all associated with the increased prevalence of MetS in the crude model (Model 1) and the two adjusted models (Model 2 and Model 3). After adjusting for temperature, relative humidity, age, sex, residence, education, marital status, annual household expenditure, smoking status, drinking status, physical activity, and indoor air pollution, each IQR increase in $PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3, and CO exposure was associated with a 19.2%, 17.7%, 15.8%, 30.3%, 10.7%, and 15.6% increase in the prevalence of MetS, respectively. Moreover, the MC Fadden R^2 of associations of $PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3 and CO with MetS were 0.086, 0.085, 0.085, 0.087, 0.085, and 0.085, respectively (Table S3). Our results also found that the air pollutants most responsible for increased MetS risk are NO_2 and particulate matter

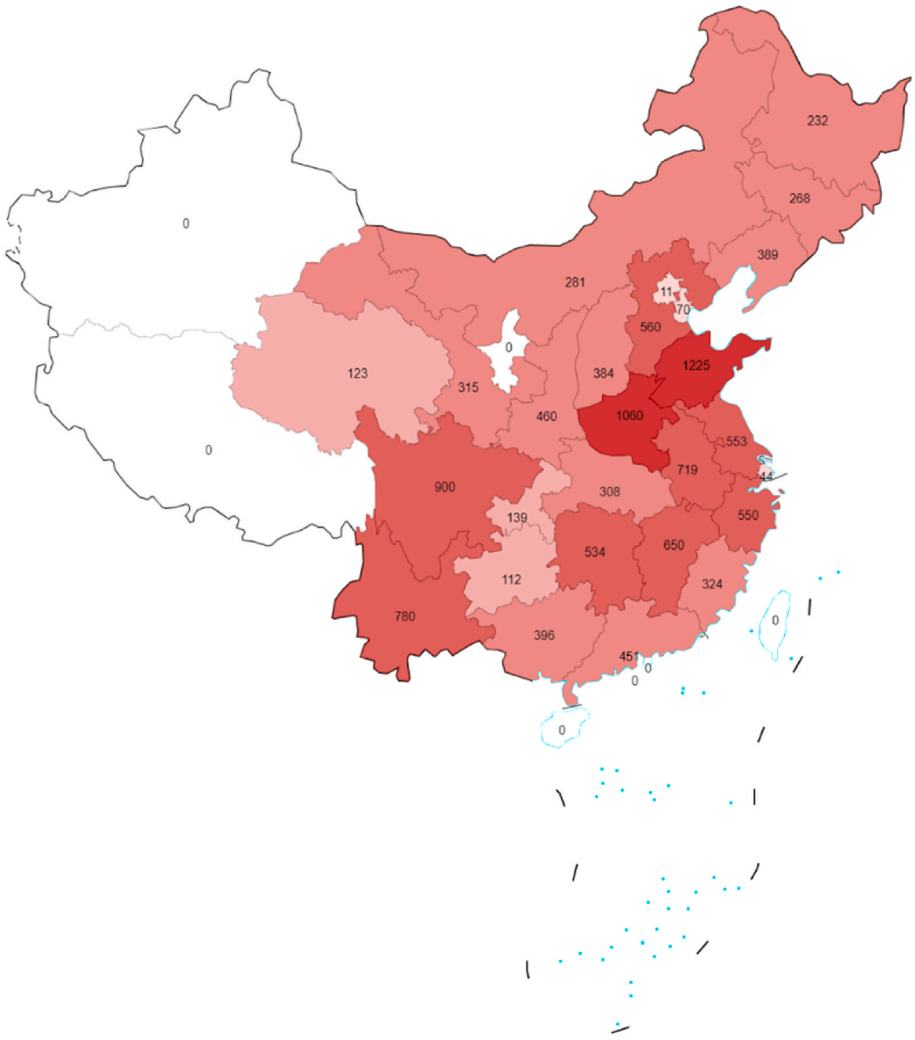

Fig. 1. The geographical distribution of 11,838 middle-aged and older adults in 27 provinces of China.

($PM_{2.5}$, PM_{10}), followed by CO and SO_2, and finally O_3 (Table S4).

3.3. Association between CRP and MetS, and the modification effects of CRP in the association of air pollution and MetS

We first enrolled CRP into the GLM model separately and found positive and significant associations between CRP and MetS risk (Model 4, Table 3). Each IQR (1.90 mg/L) increase in CRP was associated with an 4.9% (OR = 1.049, 95% CI: 1.035, 1.064) increase in prevalence of MetS (Model 4, Table 3). When CRP and one type of air pollutant were employed in the same model, the effect estimates of both CRP and air pollutants did not change substantially (Model 5, Table 3).

After introducing an interaction term of CRP and each air pollutant, we found positive and significant associations of both CRP and air pollutants with MetS. Significant interactions of CRP and air pollutants on MetS were observed (*P-interaction* < 0.10). Moreover, the results showed that the associations of all air pollutants with MetS were enhanced by increased CRP levels (Model 6, Table 3).

3.4. Sensitivity analysis

For the associations of air pollution with the prevalence of MetS, all sensitivity analyses showed similar effect values and consistent statistical significance, except for the association of 2-year exposure to SO_2 with MetS (Table S5, Table S7, Table S9, Table S11, Tables S13–14). For the association of CRP with MetS, the effect modification of CRP in the association of air pollution with MetS, both the univariate model (only CRP was applied in the model, Model 4) and the additive model (with CRP plus an air pollutant, Model 5) showed consistent results. When we introduced the interactions of CRP and air pollution, their effects on MetS showed similar results in most of sensitivity analysis (CRP-MetS: except for the interaction effect of CRP and O_3 in the sensitivity analysis of applying 2-year air pollution, log-binomial regression and adjustment for regional categories. Air pollution-MetS: except for the interaction effect of CRP and O_3, and CRP and SO_2 in sensitivity analysis of adjustment for regional categories). While a significant interaction effect was not observed in several relationships, all sensitivity analyses found a consistent trend: the effect estimates of CRP and air pollution on MetS increased when we introduced the interactions of CRP and air pollution (Table S6, Table S8, Table S10 and Table S12).

4. Discussion

In this nationally representative study of middle-aged and older Chinese adults, we found that long-term exposure to ambient air pollution ($PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3, and CO) is significantly associated with the increased prevalence of MetS. Moreover, the air pollutants most responsible for the increased prevalence of MetS were NO_2 and particulate matter ($PM_{2.5}$, PM_{10}), followed by CO and SO_2, and finally by O_3. High systemic inflammation levels could enhance the adverse effects of all pollutants on MetS. To the best of our knowledge, this is the first study to explore the interaction effects of long-term exposure to ambient

Table 1
Basic characteristics of participants.

Characteristics [a]	Total (n = 11,838)	Non-MetS (n = 8006)	MetS (n = 3832)	*P-value*
Age, years	60.63 ± 9.67	60.25 ± 9.65	61.43 ± 9.65	< 0.001
BMI , kg/m^2	23.87 ± 3.66	23.01 ± 3.41	25.68 ± 3.49	< 0.001
Sex				< 0.001
Male	5587 (47.2)	4529 (56.57)	1058 (27.6)	
Female	6251 (52.8)	3477 (43.43)	2774 (72.4)	
Residence				< 0.001
Rural	7350 (62.1)	5170 (64.58)	2180 (56.9)	
Urban	4488 (37.9)	2836 (35.42)	1652 (43.1)	
Marital status				< 0.001
Married and living with a spouse	9778 (82.6)	6719 (83.92)	3059 (79.8)	
Married but living without a spouse	491 (4.1)	329 (4.11)	162 (4.2)	
Single, divorced, and windowed	1566 (13.2)	955 (11.93)	611 (15.9)	
Education Status [b]				
Elementary school or blow	6259 (52.9)	4094 (51.14)	2165 (56.6)	< 0.001
Middle school or above	2816 (23.8)	1983 (24.77)	833 (21.7)	
Smoking Status [b]				< 0.001
Non-smoker	6680 (56.4)	3893 (48.63)	2787 (72.7)	
Smoker	766 (6.5)	591 (7.38)	175 (4.6)	
Drinking Status [b]				
Non-drinker	3147 (26.6)	2615 (32.66)	532 (13.9)	< 0.001
Drink but less than once a month	1045 (8.8)	770 (9.62)	275 (7.2)	
Drink more than once a month	7631 (64.5)	4608 (57.56)	3023 (78.9)	
Regional Categories				< 0.001
East	4529 (38.3)	2951 (36.9)	1578 (41.2)	
Midland	4436 (37.5)	2999 (37.5)	1437 (37.5)	
West	2873 (24.3)	2056 (25.7)	817 (21.3)	
Cooking Fuel Use [b]				0.001
Solid fuel	2958 (25.0)	2069 (25.8)	889 (23.2)	
Clean fuel	4194 (35.4)	2782 (34.8)	1412 (36.9)	
Physical Activity [b]	7592 ± 6519	8117 ± 6665	6434 ± 6028	< 0.001
Annual Household Expenditure [b]	13288 ± 46025	13771 ± 50920	12267 ± 33384	0.134
Waist circumference, cm	85.78 ± 12.13	81.89 ± 11.89	93.09 ± 7.86	
Triglycerides (TG), mmol/L	1.61 ± 1.03	1.37 ± 0.87	2.12 ± 1.15	< 0.001
High-density lipoprotein (HDL), mmol/L	1.32 ± 0.29	1.39 ± 0.30	1.19 ± 0.24	< 0.001
Fasting blood glucose (FBG), mmol/L	5.76 ± 1.94	5.46 ± 1.53	6.46 ± 2.45	< 0.001
Systolic blood pressure (SBP), mmHg	128.61 ± 19.70	125.46 ± 19.00	135.19 ± 19.51	< 0.001
Diastolic blood pressure (DBP), mmHg	123.66 ± 75.55	74.23 ± 11.08	78.29 ± 11.09	< 0.001
C-Reactive Protein (CRP), (mg/l)	2.69 ± 5.86	2.46 ± 6.06	3.18 ± 5.39	< 0.001
Anti-hypertensive medicine use				< 0.001
Yes	2881 (24.3)	1337 (16.7)	1544 (40.3)	
No	8952 (75.6)	6665 (83.3)	2287 (59.7)	
Anti-Diabetes medicine use				< 0.001
Yes	717 (6.1)	194 (2.4)	523 (13.7)	
No	11114 (93.9)	7806 (97.5)	3308 (86.3)	

[a] For continuous variables, numbers represent the mean ± standard deviation and for categorical variables, numbers represent count (percentage).
[b] For variables, missing values existed. *P-value* for significance test between non-MetS participants and hypertension MetS.

Table 2
Descriptive statistics 3-years average levels of air pollution, temperature and humidity.

	Mean	SD	P25	P50	P75	IQR
Air pollution						
$PM_{2.5}$ (μg/m^3)	62.99	18.81	50.52	60.36	74.56	24.04
PM_{10} (μg/m^3)	104.97	32.71	81.84	100.21	120.84	39.00
SO_2 (μg/m^3)	34.29	14.19	24.18	29.13	43.23	19.05
NO_2 (μg/m^3)	32.29	7.63	26.15	30.77	37.43	11.28
O_3 (μg/m^3)	84.82	7.18	79.05	82.82	88.56	9.51
CO (mg/m^3)	1.29	0.38	1.02	1.14	1.48	0.46
Temperature and humidity						
Temperature (°C)	15.24	4.04	13.55	15.60	17.65	4.10
Humidity (%)	67.75	8.38	62.50	69.50	74.50	12.00

Abbreviations: $PM_{2.5}$, particle with aerodynamic diameter ≤2.5 μm; PM_{10}, particle with aerodynamic diameter ≤10 μm; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbonic oxide; O_3, ozone; SD, standard deviation; P25, P50, P75, Lower, median and upper quartiles of variables; IQR, inter-quartile range.

air pollution and systemic inflammation on the risk of MetS.

Previous studies have evaluated the long-term effects of air pollution on MetS (Eze et al., 2015; Hou et al., 2020; Wang et al., 2022; Yang et al., 2018). Consistent with our study, a cross-sectional study (the 33 Communities Chinese Health Study (33CCHS)) reported that the prevalence of MetS increased by 9%, 13%, 10%, 33%, and 10% per 10 μg/m^3 increase in $PM_{2.5}$, PM_{10}, SO_2, NO_2, and O_3 exposure, respectively (Yang et al., 2018). Hou et al. reported that each 5 μg/m^3 increase in the $PM_{2.5}$, PM_{10}, and NO_2 concentrations was associated with a 42.4%, 22.87%, and 40.8% increased MetS risk in 39,089 adults from rural China (Hou et al., 2020). Wang et al. conducted a similar study and found that each IQR increase in $PM_{2.5}$ (6.7 μg/m^3), PM_{10} (12.2 μg/m^3), and O_3 (6.7 μg/m^3) was associated with a 19.3%, 21.6%, 7.4% increased prevalence of MetS, while no significant association of SO_2 or NO_2 with MetS was observed (Wang et al., 2022). The non-significant results for SO_2 and NO_2 might be attributed to the low variability of SO_2 and NO_2 concentrations in one city (Wuhan). There is little research available regarding the long-term effects of exposure to CO on MetS; however, several studies have reported that long-term exposure to CO was positively associated with MetS-related diseases, including hypertension (Dong et al., 2014; Lee et al., 2016)diabetes (Shin et al., 2019), and dyslipidemia (Shin et al., 2019).

Our study found that high systemic inflammation levels could enhance the long-term effects of all pollutants on MetS. To the best of our knowledge, no study has examined the interactive effect of systemic inflammation in the association between air pollution and MetS. However, the results of several studies could support our findings. First, studies reported that exposure to air pollution was associated with increased systemic inflammation (Azzouz et al., 2022; Liu et al., 2019d; Rich et al., 2012). For example, a meta-analysis found that each 10

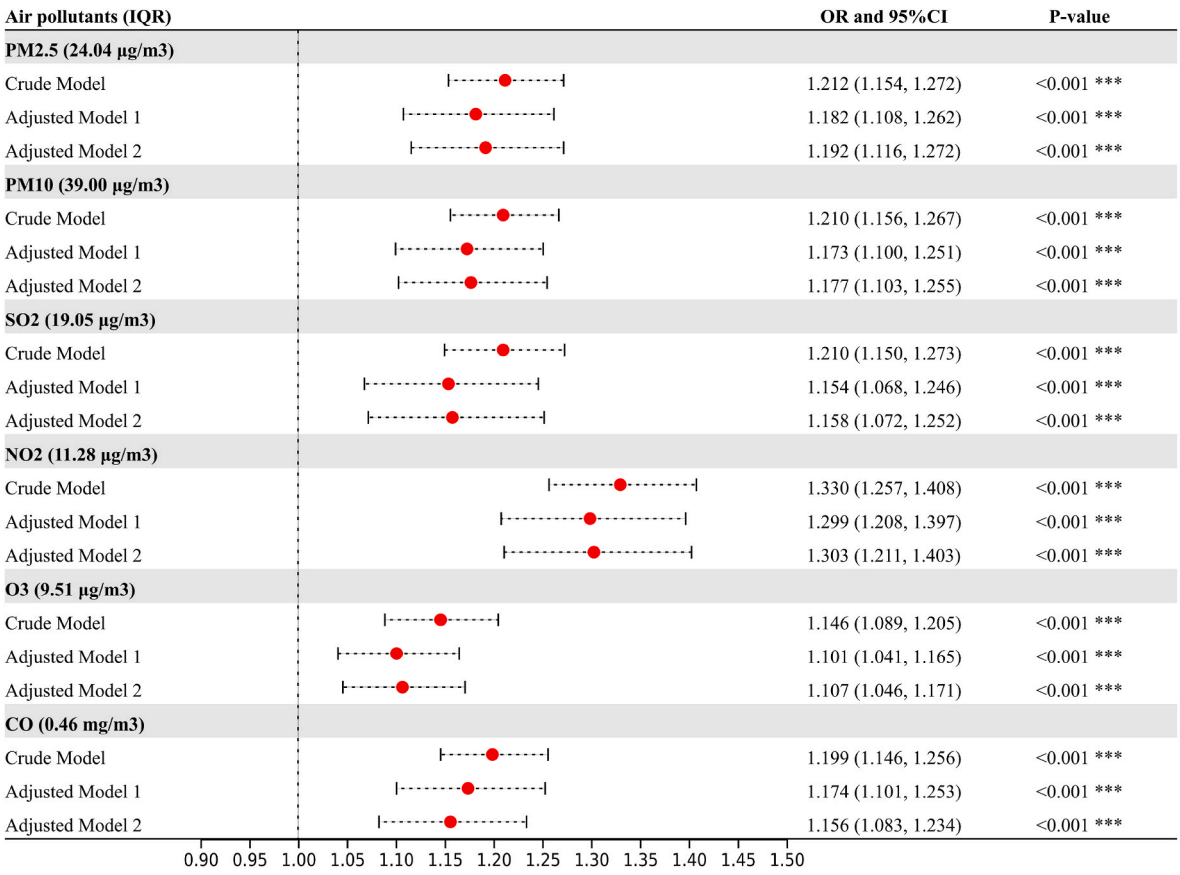

Fig. 2. Associations between air pollution and metabolic syndrome (MetS), per IQR increment in air pollutants. Notes: Model 1, crude model, without adjustment; Model 2, adjusted for temperature, relative humidity, age, sex, residence, education, marital status, and annual household expenditure; Model 3, adjusted for temperature, relative humidity, age, sex, residence, education, marital status, annual household expenditure, smoking status, and drinking status, physical activity, and cooking fuel use; *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

μg/m^3 increase in long-term exposure to $PM_{2.5}$ and PM_{10} were associated with a 18.1% (95%CI: 5.96%, 30.06%) and 5.61% (95%CI: 0.79%, 10.44%) increase in CRP levels (Liu et al., 2019d). Second, the relationship between systemic inflammation and MetS is well established (Hong et al., 2020; Ma et al., 2020; Park and Zhang, 2021; Ren et al., 2018). For example, a cross-sectional study from the Wuhan-Zhuhai cohort indicated that each 1-unit increase in log-transformed CRP was significantly associated with an 11% increased MetS prevalence (OR = 1.11, 95%CI: 1.03, 1.20) (Ma et al., 2020). In addition, people with MetS-related diseases might be vulnerable to the adverse effects of air pollution exposure. For example, Dabass et al. conducted a national cross-sectional study in the US to examine the associations of exposure to $PM_{2.5}$ with inflammatory markers and found that participants with MetS showed a stronger positive CRP response than those without MetS (Dabass et al., 2018).

The mechanism by which exposure to air pollution affects MetS remains unclear (Hou et al., 2020; Wang et al., 2022). Several potential biological pathways have been proposed for the relationship between air pollution and MetS-related diseases, including hypertension, diabetes, obesity, and dyslipidemia (Aryal et al., 2021; Yang et al., 2019). First, ambient air pollutants could migrate into the circulatory system through the alveolar membrane, leading to systematic inflammation and oxidative stress, consequently resulting in elevated blood pressure, increased body weight, insulin resistance (IR), and dyslipidemia (Mao et al., 2020b; Niu et al., 2022b; Wang et al., 2022). The significant associations between air pollution and CRP, and the interactive effect of CRP in the relationship between air pollution and MetS revealed in our study are consistent with these hypotheses. Second, dysfunction of the autonomic nervous system has also been identified as the key pathway contributing to air pollution-related hypertension, diabetes, and other diseases (Niu et al., 2021; Yang et al., 2019). Third, air pollution-induced insulin resistance (IR) and dysfunction of the renin-angiotensin-aldosterone system (RASS) are indicted to be an important physiological mechanism by which air pollution promotes MetS-related diseases, especially diabetes, obesity, and dyslipidemia (Kim et al., 2019; Liu et al., 2019c; Rich et al., 2018). Another important mechanism is abnormal epigenetic changes (Chen et al., 2016; Mao et al., 2020b). A randomized, double-blind crossover trial indicated that exposure to $PM_{2.5}$ was associated with a rapid reduction in DNA methylation, consequently contributing to CVD (Chen et al., 2016).

Our study has several strengths. First, we examined the associations between ambient air pollutants ($PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3, and CO) and the prevalence of MetS. This study contributes comprehensive epidemiological evidence compared with the limited studies of air pollution on the metabolic system, especially for SO_2, O_3, and CO (Ning et al., 2021; Zang et al., 2021). Moreover, our study evaluated the modification effect of systemic inflammation in air pollution-induced MetS for the first time, which suggested that one effective preventive measure for air pollution-induced MetS is to reduce systemic inflammation. Furthermore, the concentration of air pollutants was estimated using satellite remote sensing and novel statistical models at a 0.1° (≈10 km) gridded spatial resolution, which might have several advantages for accuracy and resolution compared with traditionally used fixed environmental monitoring stations and land use regression models without satellite data (Chen et al., 2018). Finally, a series of sensitivity analyses were conducted, the results of which indicated that our findings are robust.

There are also several limitations in our study. Firstly, because of the

Table 3
Association between CRP and MetS, and the modification effects of CRP in the association of air pollution with MetS.

Models	CRP-MetS		Air pollution-MetS		P-interaction
	OR and 95%CI	*P*	OR and 95%CI	*P*	
Model 4					
CRP	1.049 (1.035, 1.064)	<0.001 ***	–	–	–
Model 5					
$PM_{2.5}$ + CRP	1.049 (1.035, 1.064)	<0.001 ***	1.191 (1.115, 1.271)	<0.001 ***	–
PM_{10} + CRP	1.049 (1.035, 1.064)	<0.001 ***	1.176 (1.102, 1.255)	<0.001 ***	–
SO_2 + CRP	1.049 (1.035, 1.063)	<0.001 ***	1.156 (1.069, 1.249)	<0.001 ***	–
NO_2 + CRP	1.049 (1.035, 1.064)	<0.001 ***	1.303 (1.211, 1.403)	<0.001 ***	–
O_3 + CRP	1.049 (1.035, 1.064)	<0.001 ***	1.106 (1.045, 1.171)	<0.001 ***	–
CO + CRP	1.048 (1.035, 1.063)	<0.001 ***	1.152 (1.080, 1.080)	<0.001 ***	–
Model 6					
$PM_{2.5}$ + CRP + $PM_{2.5}$ × CRP	1.089 (1.040, 1.141)	<0.001 ***	1.215 (1.134, 1.303)	<0.001 ***	0.091 *
PM_{10} + CRP + PM_{10} × CRP	1.083 (1.037, 1.133)	<0.001 ***	1.197 (1.118, 1.282)	<0.001 ***	0.084 *
SO_2 + CRP + SO_2 × CRP	1.081 (1.042, 1.122)	<0.001 ***	1.183 (1.090, 1.283)	<0.001 ***	0.074 *
NO_2 + CRP + NO_2 × CRP	1.112 (1.053, 1.175)	<0.001 ***	1.343 (1.242, 1.453)	<0.001 ***	0.027 **
O_3 + CRP + O_3 × CRP	1.213 (1.034, 1.419)	<0.001 ***	1.131 (1.064, 1.204)	<0.001 ***	0.067 *
CO + CRP + CO × CRP	1.084 (1.037, 1.133)	<0.001 ***	1.172 (1.095, 1.256)	<0.001 ***	0.077 *

Notes: Results were presented as OR and 95%CI of MetS per IQR increase in CRP (1.9 mg/L) and air pollution ($PM_{2.5}$, 24.04 μg/m^3; PM_{10}, 39.00 μg/m^3; SO_2,19.05 μg/m^3; NO_2, 11.28 μg/m^3; O_3, 9.51 μg/m^3; CO, 0.46 mg/m^3) Model 4, univariate model, only CRP was applied in the model; Model 5, additive models, CRP and an air pollutant were applied in the model, with CRP plus air pollutants; Model 6, interaction model, CRP and an air pollutant were applied in the model, with CRP + air pollutants + CRP × air pollutants. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$ for the association of CRP/air pollution and MetS; *$P < 0.10$; **$P < 0.05$ for interactions of CRP and air pollution.
Abbreviations: CRP, C-Reactive Protein; $PM_{2.5}$, particle with aerodynamic diameter ≤2.5 μm; PM_{10}, particle with aerodynamic diameter ≤10 μm; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbonic oxide; O_3, ozone; OR, odd ratio; 95%CI, 95% confidence intervals.

cross-sectional design of this study, the causal relationship between air pollution, systemic inflammation, and MetS could not be established. Secondly, anti-hypertensive history, anti-diabetic medicine history, and potential confounders were obtained using a self-reported questionnaire, thus recall bias and reporting bias could not be avoided. Thirdly, a multi-pollutant model could not be constructed because the air pollutants ($PM_{2.5}$, PM_{10}, SO_2, NO_2 and CO) were highly correlated (Chen et al., 2017; Li et al., 2020; Mao et al., 2020b). However, sensitivity analysis using a two-pollutant model with adjustment for O_3 suggested that the associations of air pollution with MetS were robust. Fourthly, as a McFadden's pseudo R^2 ranging from 0.2 to 0.4 indicates very good model fit (McFadden et al., 1979; Hemmert et al., 2016), the McFadden's pseudo R^2 values of our study showed that the models were not particularly strong. Finally, salt intake and other life characteristics were not obtained in the CHARLS, which might modify the associations of air pollution with MetS. Further studies with controls for more potential confounders are needed to confirm our findings.

5. Conclusion

In summary, our study suggested that long-term exposure to ambient air pollution was associated with increased prevalence of MetS. The adverse effects of ambient air pollution on MetS were enhanced by increased systemic inflammation levels in Chinese middle-aged and older adults. Immediate measures should be taken to improve air quality and reduce systemic inflammation, which could contribute to decreasing the burden of MetS.

Author contribution statement

Conceptualization: ZP N, ZZ D, SC H; Data curation: SC H, F Z, HM Y; Formal analysis: SC H, ZP N; Funding acquisition: SC H; Investigation: SC H, F Z, HM Y, LN X; Methodology: ZP N, SC H, J W; Project administration: ZP N, ZZ D; Resources: SC H; Software: SC H, F Z; Supervision: ZP N, ZZ D; Validation: YM Y, LN X; Visualization: SC H, F Z, ZP N; Roles/Writing - original draft: SC H, F Z; Writing - review & editing: ZP N, ZZ D.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2022.114340.

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