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ORIGINAL RESEARCH

Short-Term Effects of Particle Size and Constituents on Blood Pressure in Healthy Young Adults in Guangzhou, China

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BACKGROUND: Although several studies have focused on the associations between particle size and constituents and blood pressure, results have been inconsistent.

METHODS AND RESULTS: We conducted a panel study, between December 2017 and January 2018, in 88 healthy university students in Guangzhou, China. Weekly systolic blood pressure and diastolic blood pressure were measured for each participant for 5 consecutive weeks, resulting in a total of 440 visits. Mass concentrations of particles with an aerodynamic diameter of ≤2.5 μm ($PM_{2.5}$), ≤1.0 μm ($PM_{1.0}$), ≤0.5 μm ($PM_{0.5}$), ≤0.2 μm ($PM_{0.2}$), and number concentrations of airborne particulates of diameter ≤0.1 μm were measured. Linear mixed-effect models were used to estimate the associations between blood pressure and particles and $PM_{2.5}$ constituents 0 to 48 hours before blood pressure measurement. PM of all the fractions in the 0.2- to 2.5-μm range were positively associated with systolic blood pressure in the first 24 hours, with the percent changes of effect estimates ranging from 3.5% to 8.8% for an interquartile range increment of PM. $PM_{0.2}$ was also positively associated with diastolic blood pressure, with an increase of 5.9% (95% CI, 1.0%–11.0%) for an interquartile range increment (5.8 μg/m^3) at lag 0 to 24 hours. For $PM_{2.5}$ constituents, we found positive associations between chloride and diastolic blood pressure (1.7% [95% CI, 0.1%–3.3%]), and negative associations between vanadium and diastolic blood pressure (–1.6% [95% CI, –3.0% to –0.1%]).

CONCLUSIONS: Both particle size and constituent exposure are significantly associated with blood pressure in the first 24 hours following exposure in healthy Chinese adults.

Key Words: air pollution ■ blood pressure ■ constituents ■ panel study ■ particulate matter

Short-term exposures to ambient particulate air pollution are closely linked to increased risks of morbidity and mortality from cardiovascular diseases[1–3]; however, the exact mechanisms underlying these associations remain unclear. High blood pressure (BP) is one of the major risk factors for cardiovascular diseases, and even young adults with elevated BP might have increased risks of cardiovascular events in later life.[4] In air pollution epidemiology, high blood pressure is usually hypothesized to be a mediator for the relationships between ambient particulate air pollution and cardiovascular diseases.[5] Although numerous epidemiological studies have explored the associations between particulate matter (PM) and blood pressure, the results are still inconsistent.[6,7] For example, while some studies reported that short-term PM exposures were significantly associated with increased blood pressure,[8–14] others showed a null or inverse association.[15–20]

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CLINICAL PERSPECTIVE

What Is New?

- Size-fractionated particulate matter with a wide range of smaller diameters (5 cumulative size fractions with upper limits from 0.1 to 2.5 μm) and various particulate matter 2.5-μm constituents were measured in a region with high air pollution levels, adding more evidence to the understanding of the association between particulate matter size and constituents and blood pressure.

What Are the Clinical Implications?

- Our results could add evidence to the association of particulate matter–related blood pressure in the young population, help us to more deeply understand the physiological mechanisms of the development of hypertension, and also provide healthcare professionals with suggestions or treatments for people to take preventive measures, and ultimately to mitigate the effects of air pollution on hypertension.

Nonstandard Abbreviations and Acronyms

DBP	diastolic blood pressure
EC	elemental carbon
IQR	interquartile range
OC	organic carbon
PM	particulate matter
$PM_{0.2}$	particles with an aerodynamic diameter of ≤0.2 μm
$PM_{0.5}$	particles with an aerodynamic diameter of ≤0.5 μm
$PM_{1.0}$	particles with an aerodynamic diameter of ≤1.0 μm
$PM_{2.5}$	particles with an aerodynamic diameter of ≤2.5 μm
$PN_{0.1}$	airborne particulates of diameter ≤0.1 μm
SBP	systolic blood pressure

The associations between PM and blood pressure changes could be influenced by many factors. First, PM pollution could differ in different cities because of its spatiotemporal variation and socioeconomic drivers, leading to varying degrees of health effects.[21] PM pollution has long been a major public health concern in China, but most previous studies were based on heavily polluted areas in northern China,[12,22,23] with fewer studies conducted in southern China where particulate air pollution levels are lower compared with northern China. Furthermore, aerodynamic diameter and chemical composition are the main physical characteristics of particles, and different sizes and composition of particles may have different toxicological and physicochemical properties,[24,25] thus leading to the heterogeneous findings of PM health impact. Because most of these studies have been conducted among patients or specific populations,[11,26–28] evidence from the general population is still scarce.

Therefore, in this study, our objective was to determine the acute effects of size-fractionated PM as well as chemical composition on blood pressure in healthy young adults in Guangzhou in southern China.

METHODS

Data Availability

All the data presented in this study are available from the authors upon request.

Study Participants

We carried out a panel study between December 2017 and January 2018 in healthy young adults aged 20 to 26 years. Participants were recruited from the North campus of Sun Yat-Sen University, which covers only an area of ≈0.39 km^2 in Guangzhou, China, and met the following criteria: (1) lived and studied in North campus during the study period; (2) no previous smoking or no alcohol consumption during the study period; (3) no cardiovascular, pulmonary, or other chronic diseases, and (4) free of medications that may influence cardiovascular function. We scheduled 5 weekly visits for each of the study participants during the study period, from December 7, 2017 to January 5, 2018. Finally, 88 students participated in our study. The demographic information of each participant, including name, sex, ethnicity, exercise frequency, and medical history or health status were collected at the first measurement. Height and weight of each participant were also measured, and body mass index was calculated as the weight (kg) divided by the square of the height (meters). All study participants provided written informed consent at the beginning of the study. The Human Studies Committee of the School of Public Health, Sun Yat-Sen University approved this study (Ethics Approval Number: L2017024).

BP Measurements

In order to obtain a BP observer certificate, a qualifying examination was taken for trainees involved in the study, and all investigators were trained according to the guideline of the European Society of Hypertension.[29]

Before BP measurements, study participants were advised not to exercise for at least 30 minutes, and not to drink coffee or tea on the visit day. With their back and right arm supported, participants sat in a quiet room for at least 5 minutes, feet on the floor and cubital fossa at heart level. The upper right arm brachial artery BP of participants, including systolic blood pressure (SBP) and diastolic blood pressure (DBP), was measured on 3 consecutive occasions using automated oscillometric monitors. The final reported BP value was the average of the 3 BP measurements and was used for analyses. Pulse pressure was calculated as the SBP minus the DBP, and mean arterial pressure (MAP) was calculated as of the DBP plus one third of the pulse pressure.

Five weekly study visits were taken for each participant over the entire study in a school building at the campus. In every repeated measurement on the 88 participants, half of them were measured on Thursday at 6:00 to 10:00 PM to collect their BP information, and the rest of the participants were measured on Friday at the same time and place.

Environmental Data

Using standard methods and quality controls, data of real-time PM mass and number concentration as well as meteorological exposure were measured on the roof of a 10-story building from December 1, 2017 to January 5, 2018 at the same campus where participants were recruited. A measurement site was established that was clear from any structures that would obstruct the air flow. At the site, minute-to-minute mass concentrations of PM with an aerodynamic diameter of ≤2.5 μm ($PM_{2.5}$), ≤1.0 μm ($PM_{1.0}$), ≤0.5 μm ($PM_{0.5}$), and ≤0.2 μm ($PM_{0.2}$) were measured. In addition, minute-to-minute number concentrations of PM with an aerodynamic diameter of ≤0.1 μm ($PN_{0.1}$) were measured. The measurements used a combination of an Optical Particle Sizer Spectrometer (Model OPS3330; TSI, USA) and a NanoScan Scanning Mobility Particle Sizer (Model 3910; TSI, USA), and the optical diameter was then converted into an aerodynamic diameter through the method suggested by Alas et al.[30] A Vaisala Weather Transmitter (Model WXT530; Vaisala, Finland) was used to continuously measure temperature and relative humidity.

At the same time and site of the real-time PM sampling, daily $PM_{2.5}$ sampling was also performed for further measurement of $PM_{2.5}$ constituents. The high-volume samplers (TH-1000H; Tianhong Co. Ltd., China) were used with a sampling rate of 1 m^3/min and the collection time of each sample was 24 hours. Quartz microfiber filters and Teflon filters were used. After sampling, the filter-based samples were placed in a desiccator for initial weighing and gravimetric quantification and then stored in a refrigerator (−20°C) for further chemical composition analysis. Using a semicontinuous OC/EC analyzer (Model 4G; Sunset Laboratory, OR) and thermo/optical transmission method, organic carbon (OC), elemental carbon (EC), total carbon, and water-soluble organic carbon were measured in quartz-fiber filters. Ten water-soluble inorganic ions, including ammonium (NH_4^+), sodium (Na^+), potassium (K^+), calcium (Ca^{2+}), magnesium (Mg^{2+}), chloride (Cl^-), fluoride (F^-), sulfate (SO_4^{2-}), nitrite (NO_2^-), and nitrate (NO_3^-) were measured by ion chromatography (model ICS-2000; Dionex Corp., Sunnyvale, CA). Fifteen trace elements, including sodium (Na), potassium (K), calcium (Ca), magnesium (Mg), aluminum (Al), phosphorus (P), vanadium (V), copper (Cu), chromium (Cr), manganese (Mn), iron (Fe), nickel (Ni), zinc (Zn), barium (Ba), and lead (Pb) were measured by inductively coupled plasma mass spectrometry (ICP-MS; Thermo Scientific, USA).

Statistical Analysis

Because all the outcome variables (SBP, DBP, pulse pressure, and MAP) were not normally distributed, we applied a log transformation. Spearman correlations were used to evaluate the relationship between study pollutants before including them in the regression analyses. Minute-to-minute pollutant concentrations, measured 0 to 48 hours before BP measurement every week, were used to calculate the lag concentrations in the following time windows: lag 0 to 6 hours, lag 7 to 12 hours, lag 13 to 24 hours, lag 0 to 24 hours, lag 25 to 48 hours, and lag 0 to 48 hours.

We used linear mixed models to estimate the associations between PM and BP, and random intercept that included each participant was included in each model to adjust for the within- participant correlations in repeated measurements. Covariates included as fixed-effect terms in these models were: age, sex, body mass index, exercise times per week, average temperature, and humidity.[31] To adjust for any possible weekly time trends, we also included "week" of BP measurements as an indicator variable. In the weekly repeated measurements, each participant was measured on the same day of week and at the same time of day in the same place, so that potential confounding because of day of week, time of measurement, and site was minimized. In addition, the study was conducted in 1 season with less changing in climate, so that seasonal factors were not considered as confounding factors.

We use 3 models to investigate the effects and robustness of the associations between $PM_{2.5}$ chemical composition and BP changes.[12,32] First, particles or $PM_{2.5}$ constituents were incorporated one at a time with adjustment for the above-mentioned potential confounders. In addition, we built the constituent-$PM_{2.5}$ joint model in which $PM_{2.5}$ total mass was introduced in the single-constituent model to account for potential confounding by $PM_{2.5}$ and all other constituents that covary with $PM_{2.5}$. Furthermore, for the constituent-residual model, we first created the residual of each constituent by establishing a linear regression model between the constituent and $PM_{2.5}$ total mass, and then replaced the constituent in the single-constituent model by its residual. This model could be regarded as a crude measure of the independent contribution of each constituent because the residual of each constituent was uncorrelated with $PM_{2.5}$ total mass.[33–35]

We calculated the percent changes of effect estimates in BP variables for an interquartile range (IQR) increase of particle or a constituent as $[10^{(\beta\times IQR)}-1]\times100\%$, and the 95% CIs were $[10^{[IQR\times(\beta\pm1.96\times SE)]}-1]\times100\%$, where β and SE were the estimated regression coefficient and its SE, respectively.[36]

All analyses in this study were conducted in R software (Version 3.6.1, R Foundation for Statistical Computing, Vienna, Austria) and the "lme4" package was used. Statistical significance level was defined as *P*<0.05 (two sided).

RESULTS

Descriptive Statistics

Overall, a total of 88 participants completed all of the 5 weekly control visits, resulting in a total of 440 visits. Characteristics of the study participants are shown in Table 1. The average age of study participants was 21.5±1.1 years old and their average body mass index was 21.4±2.9 kg/m^2. Fifty-four participants were female (n=54), and more than half of the participants exercised at least once a week. During the study period, all participants did not have any disease that would affect the study result and did not take any medication, dietary supplements, or alcoholic beverages. Sex-stratified descriptive statistics of study participants are shown in Table S1.

Table 1. Basic Characteristics of the Study Participants

Characteristics	Total (n=88)*
Age, y	21.5±1.1
Female (%)	54 (61.4%)
BMI, kg/m^2	21.4±2.9
Exercise time (%)	
0 time per wk	41 (46.6%)
1 time per wk	19 (21.6%)
2 times per wk	20 (22.7%)
≥3 times per wk	8 (9.1%)

BMI indicates body mass index.

*In this column, for some parameters an arithmetic mean is given and for some the number of occurrences.

Table 2 shows descriptive statistics on BP, average concentrations of air pollutants, weather conditions, and $PM_{2.5}$ composition during the study period. The mean SBP, DBP, pulse pressure, and MAP were 112.5±14.1 mm Hg, 71.0±8.9 mm Hg, 41.5±11.7 mm Hg, and 84.8±9.4 mm Hg, respectively. On average, daily $PM_{2.5}$ mass concentration was 72.7±35.1 μg/m^3 (mean±SD), and $PN_{0.1}$ number concentration was 20.2±11.7 (1000 particles/cm^3) (mean±SD). Correlations among PM metrics were highest for $PM_{2.5}$ and $PM_{1.0}$ (Table S2). Correlations between PM mass or number concentrations and temperature were low (*r* values between 0.27 and 0.46), and it was the same for relative humidity (*r* values between −0.14 and 0.16).

Regression Results

The relationship between different lag times of PM exposure and BP are shown in Figures 1 and 2. PM of all the fractions in the 0.2 to 2.5 μm range were positively associated with SBP in the first 24 hours. For an IQR increment of particles between 0.2 and 2.5 μm, this positive effect ranged from 3.5% to 8.8% at different lag times. Furthermore, $PM_{0.2}$ was also positively associated with DBP and MAP. These associations were first seen at 7 to 12 hours, but were not significant after lag 25 to 48 hours and lag 0 to 48 hours. At lag 0 to 24 hours, an IQR increase in $PM_{2.5}$ (45.9 μg/m^3) was significantly associated with an increase of 8.8% (95% CI, 1.5%–16.6%) in SBP, and an IQR increase in $PM_{0.2}$ (5.8 μg/m^3) was associated with an increase of 5.9% (95% CI, 1.0%–11.0%) in DBP and an increase of 5.1% (95% CI, 1.4%–8.8%) in MAP. The results for $PN_{0.1}$ were mixed, which had both positive and negative associations in SBP, DBP, and MAP, and was not similar to the results of particles between 0.2 and 2.5 μm. More specifically, for an IQR increase in $PN_{0.1}$ (12.8 [1000 particles/cm^3]), the estimated percent changes showed no statistically significant in $PN_{0.1}$ and SBP at lag 0 to 24 hours (0.9% [95% CI, −5.2% to 7.5%]), showed negative association with DBP at lag 0 to 6 hours (−5.4% [95% CI, −10.3% to −0.3%]), and showed positive association with DBP at lag 13 to 24 hours (5.4% [95% CI, 0.8%–10.2%]). These results were similar for both the crude and adjusted models.

For $PM_{2.5}$ constituents, significant positive associations were found between chloride and DBP in all 3 models, and in the single-constituent model, the increment was 1.7% (95% CI, 0.1%–3.3%) in DBP. In addition, significant negative associations were found between DBP and vanadium, and between MAP and fluoride in all 3 models, with estimated percent changes of −1.6%

Table 2. Descriptive Statistics on Blood Pressure, Average Concentrations of Air Pollutants, Weather Conditions, and $PM_{2.5}$ Constituents During Study Period

Exposures	Mean±SD	Min	P_{25}	Median	P_{75}	Max	IQR
Seated blood pressure							
SBP, mm Hg	112.5±14.1	86	102	110	121	157	19
DBP, mm Hg	71.0±8.9	49	65	71	77	99	12
PP, mm Hg	41.5±11.7	18	33	40	48	79	15
MAP, mm Hg	84.8±9.4	64.3	77.7	83.7	91.3	112	13.7
PM mass concentration							
$PM_{2.5}$, μg/m^3*	72.7±35.1	5.3	48.1	70.2	94.0	220.5	45.9
$PM_{1.0}$, μg/m^3	68.8±33.6	5.1	45.2	66.4	88.7	208.3	43.5
$PM_{0.5}$, μg/m^3	53.2±25.5	4.3	35.0	50.7	68.0	155.9	33.0
$PM_{0.2}$, μg/m^3	9.9±5.0	1.4	6.3	9.1	12.0	45.1	5.8
PM count concentration							
$PN_{0.1}$ (1000 particles/cm^3)	20.2±11.7	2.6	12.2	17.3	25.0	133	12.8
Meteorology							
Temperature, ℃	17.3±3.4	6.9	15.6	17.6	19.4	26.3	3.8
Relative humidity, %	44.4±13.8	14.6	33.2	44.7	56.2	76.2	23
Constituent							
Na, ng/m^3	930.1±255.2	435.3	799.4	917.6	1070.9	1419.2	271.5
Mg, ng/m^3	71.4±22.2	30.3	53.4	78.3	87.3	120.3	33.9
Al, ng/m^3	37.5±31.5	0.04	6.8	38.5	60.4	102.9	53.6
P, ng/m^3	26.8±15.9	1.4	11.6	26.0	37.2	60.3	25.7
K, ng/m^3	897.2±352.9	229.6	589.3	886.5	1144.8	1636.1	555.5
Ca, ng/m^3	1128.2±366.5	538.0	811.7	1109.9	1399.3	2057.5	587.6
V, ng/m^3	1.7±2.0	0.2	0.6	0.8	2.1	8.5	1.6
Cr, ng/m^3	1.2±0.8	0.3	0.7	1.0	1.4	3.5	0.7
Mn, ng/m^3	17.9±6.2	4.6	14.2	18.5	21.5	28.6	7.3
Fe, ng/m^3	44.9±27.2	2.2	19.6	44.5	58.4	97.5	38.8
Ni, ng/m^3	1.0±0.8	0.2	0.5	0.6	1.3	3.5	0.8
Cu, ng/m^3	17.4±12.6	3.0	8.7	12.6	24.0	56.6	15.3
Zn, ng/m^3	204.2±103.2	7.6	113.4	204.9	267.7	412.0	154.3
Ba, ng/m^3	7.9±2.5	3.0	6.0	7.2	9.6	12.8	3.6
Pb, ng/m^3	16.5±12.4	1.6	6.4	14.7	24.1	44.3	17.7
Na^+, μg/m^3	0.4±0.1	0.2	0.4	0.4	0.6	0.6	0.2
NH_4^+, μg/m^3	3.3±1.4	0.8	2.1	3.5	4.4	5.7	2.4
K^+, μg/m^3	0.6±0.2	0.1	0.4	0.6	0.8	1.0	0.4
Mg^{2+}, μg/m^3	0.1±0.02	0.02	0.1	0.1	0.1	0.1	0.02
Ca^{2+}, μg/m^3	0.6±0.2	0.3	0.5	0.6	0.7	1.1	0.3
F^-, μg/m^3	0.02±0.02	0	0.01	0.01	0.02	0.1	0.02
Cl^-, μg/m^3	0.6±0.4	0.2	0.3	0.5	0.7	2.0	0.4
NO_2^- μg/m^3	0.03±0.03	0	0.01	0.03	0.1	0.1	0.04
NO_3^-, μg/m^3	6.3±2.5	1.3	4.3	6.7	8.0	10.3	3.7
SO_4^{2-}, μg/m^3	6.2±2.4	2.3	3.9	6.3	7.9	10.2	4.0
WSOC, μg/m^3	4.3±1.7	0.8	3.1	4.7	5.7	7.4	2.6
OC, μg/m^3	14.8±5.3	5.4	10.9	14.7	19.1	23.3	8.2
EC, μg/m^3	1.0±0.3	0.6	0.8	1.0	1.1	1.6	0.3
TC, μg/m^3	15.8±5.5	6.1	11.6	15.7	20.1	24.5	8.4

DBP indicates diastolic blood pressure; EC, elemental carbon; IQR, interquartile range (computed by subtracting the first quartile from the third quartile); MAP, mean arterial pressure; Max, maximum; Min, minimum; OC, organic carbon; P_{25}, the 25th percentile; P_{75}, the 75th percentile; $PM_{0.2}$, particle with aerodynamic diameter ≤0.2 μm; $PM_{0.5}$, particle with aerodynamic diameter ≤0.5 μm; $PM_{1.0}$, particle with aerodynamic diameter ≤1.0 μm; $PM_{2.5}$, particle with aerodynamic diameter ≤2.5 μm; PP, pulse pressure; SBP, systolic blood pressure; TC, total carbon; and WSOC, water-soluble organic carbon.

*World Health Organization Guideline for $PM_{2.5}$ is 10 μg/m^3.

(95% CI, −3.0% to −0.1%) and −1.5% (95% CI, −2.8% to −0.3%) in the single-constituent model, respectively. Other constituents, such as OC, EC, and total carbon, were significantly associated with SBP based on single-constituent models (the estimated percent changes were 4.2% [95% CI, 0.6%–7.9%], 3.2% [95% CI, 0.8%–5.6%], and 4.3% [95% CI, 0.8%–8.0%], respectively), but these associations became nonsignificant in adjusted models (Figures 3 through 5).

DISCUSSION

Summary

We measured the effects of size-fractionated particulate matter (5 cumulative size fractions with upper limits from 0.1 to 2.5 μm) and 29 constituents of $PM_{2.5}$ (4 carbonaceous components, 10 inorganic ions, and 15 trace elements) on BP in 88 healthy young adults in Guangzhou, China. In the first 24 hours following exposure, positive associations were found between size-fractionated PM (particles between 0.2 and 2.5 μm) and SBP, and between $PM_{0.2}$ and DBP and MAP. As for $PM_{2.5}$ constituent concentrations, positive associations were found between DBP and chloride, and negative associations were found between DBP and vanadium and between MAP and fluoride.

Effect of Particle Size

Several previous studies have observed that short-term PM exposure may be related to BP changes; however, the results across these studies are inconsistent. In our study, we observed positive association between size-fractionated PM and SBP; similarly, previous studies have also reported that $PM_{2.5}$ exposure is

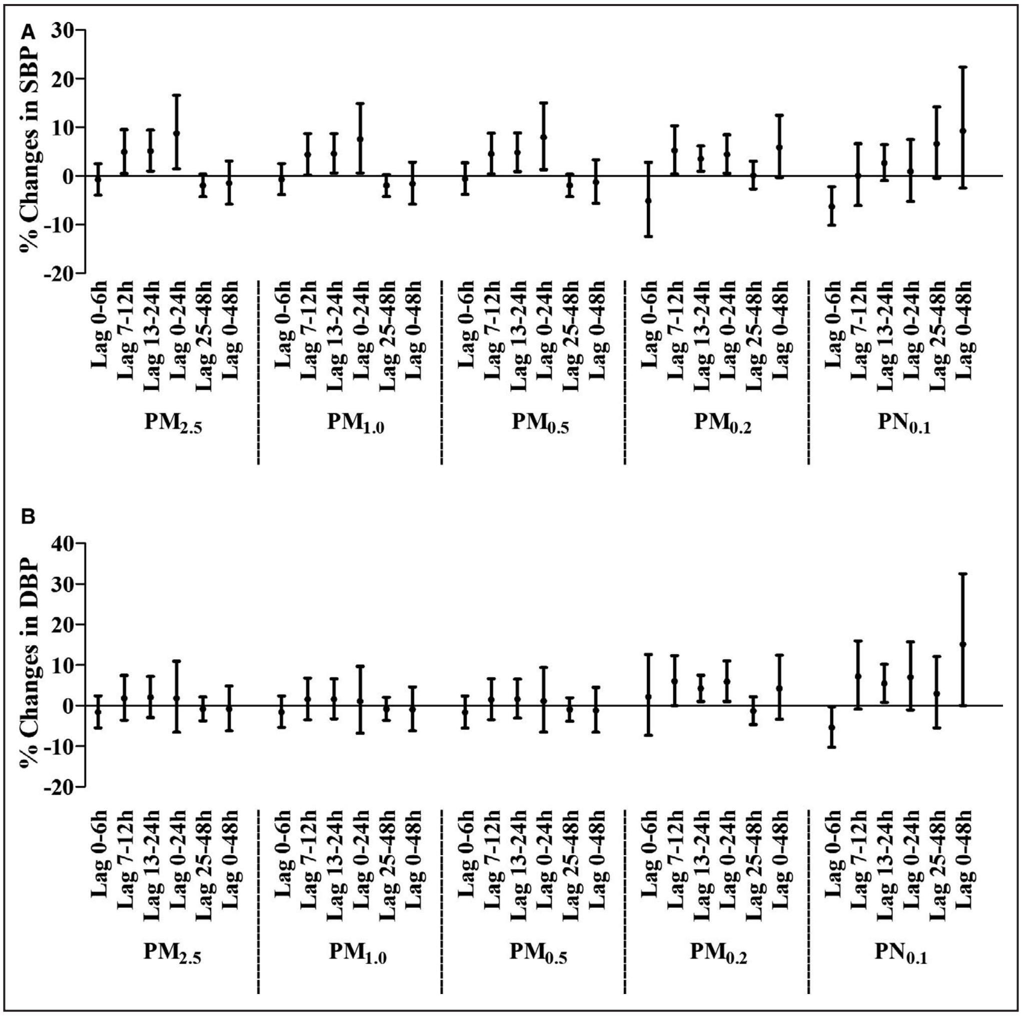

Figure 1. Percent changes (mean and 95% CIs) in blood pressure associated with an interquartile range increase in size-fractionated particulate matter.
A, SBP indicates systolic blood pressure; (**B**) DBP indicates diastolic blood pressure. $PM_{2.5}$ indicates particle with aerodynamic diameter ≤2.5 μm; $PM_{1.0}$, particle with aerodynamic diameter ≤1.0 μm; $PM_{0.5}$, particle with aerodynamic diameter ≤0.5 μm; $PM_{0.2}$, particle with aerodynamic diameter ≤0.2 μm; and $PN_{0.1}$, particle with aerodynamic diameter ≤0.1 μm.

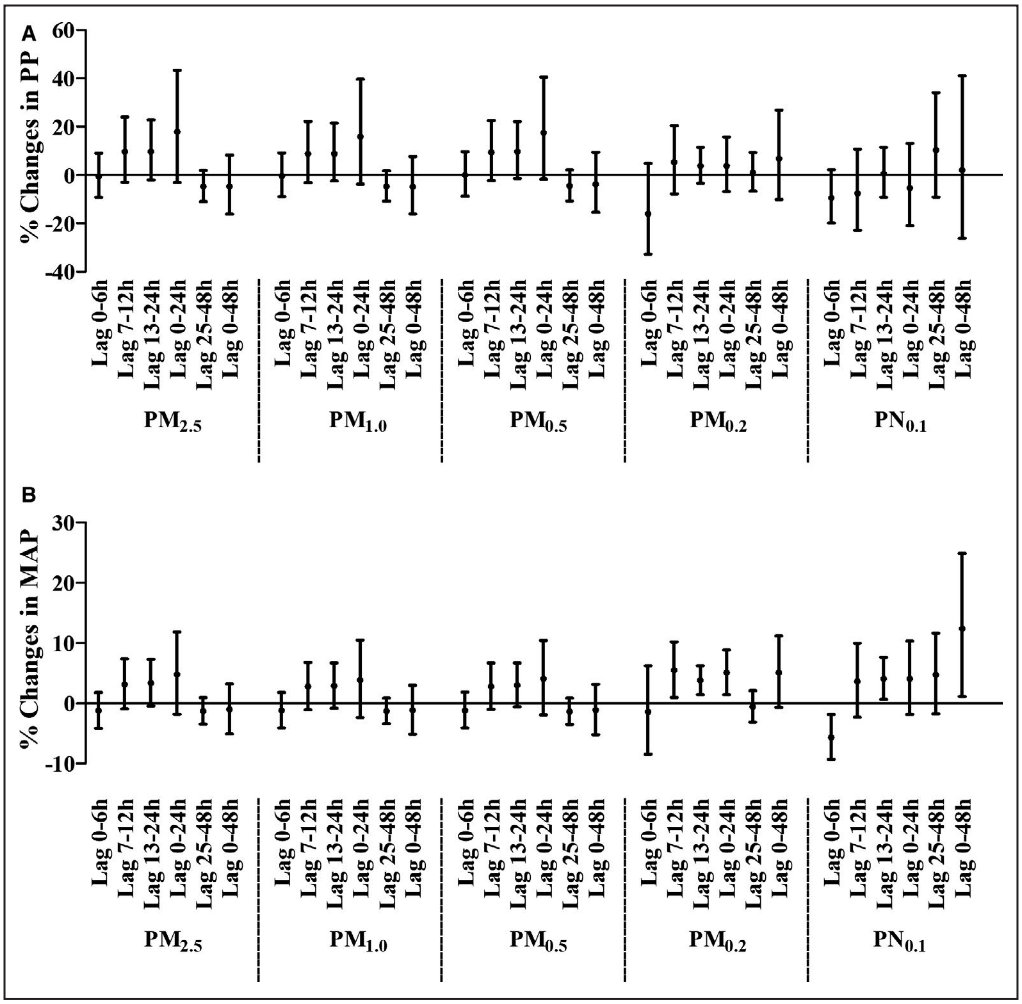

Figure 2. Percent changes (mean and 95% CIs) in blood pressure associated with an interquartile range increase in size-fractionated particulate matter.
A, PP indicates pulse pressure; (**B**) MAP indicates mean arterial pressure. $PM_{2.5}$ indicates particle with aerodynamic diameter ≤2.5 μm; $PM_{1.0}$, particle with aerodynamic diameter ≤1.0 μm; $PM_{0.5}$, particle with aerodynamic diameter ≤0.5 μm; $PM_{0.2}$, particle with aerodynamic diameter ≤0.2 μm; and $PN_{0.1}$, particle with aerodynamic diameter ≤0.1 μm.

related to increased SBP.[9,11,12,14,26,37,38] For example, Lin et al found that a 19.1 μg/m^3 (IQR) increment of $PM_{2.5}$ was related to an increase of 1.9 mm Hg (95% CI, 0.7–3.1) in SBP.[11] Zhao et al showed in a panel study that an increase in SBP corresponded to size-fractionated particulate matter among patients with type 2 diabetes mellitus.[26] Conversely, other studies have reported inverse or null associations.[15,16,19,39] For instance, Scheers et al failed to observe significant associations between PM_{10}, $PM_{2.5}$ variations, and BP.[39]

In this panel study, we also found that $PM_{0.2}$ were positively associated with DBP and MAP. Studies showed that results for PM-related DBP changes are mixed.[10–12,14,37,38,40] For instance, Yang et al reported that a 10 μg/m^3 increment on 0 to 6-day mean of $PM_{2.5}$ was associated with an increase of 0.49 mm Hg (95% CI, 0.45–0.53) in DBP among 6- to 17-year-old children in Suzhou.[14] However, Ibald-Mulli et al did not find significant positive associations for $PM_{2.5}$ and DBP among 131 patients with coronary heart disease.[17] MAP could be regarded as a strong predictor of cardiovascular events and mortality.[41,42] Consistent with our study, Wang et al found that an IQR increment (26.78 μg/m^3) in a 24-hour mean of $PM_{2.5}$ was associated with increments of 0.66 mm Hg in DBP and 0.82 mm Hg in MAP.[38] Therefore, short-term exposure to PM may be also related to changes in DBP and MAP, and further studies are still needed to clarify these associations.

Effects of $PM_{2.5}$ Constituents

Evidence linking $PM_{2.5}$ chemical composition and cardiovascular outcomes has been growing, and short-term exposure to various $PM_{2.5}$ constituents

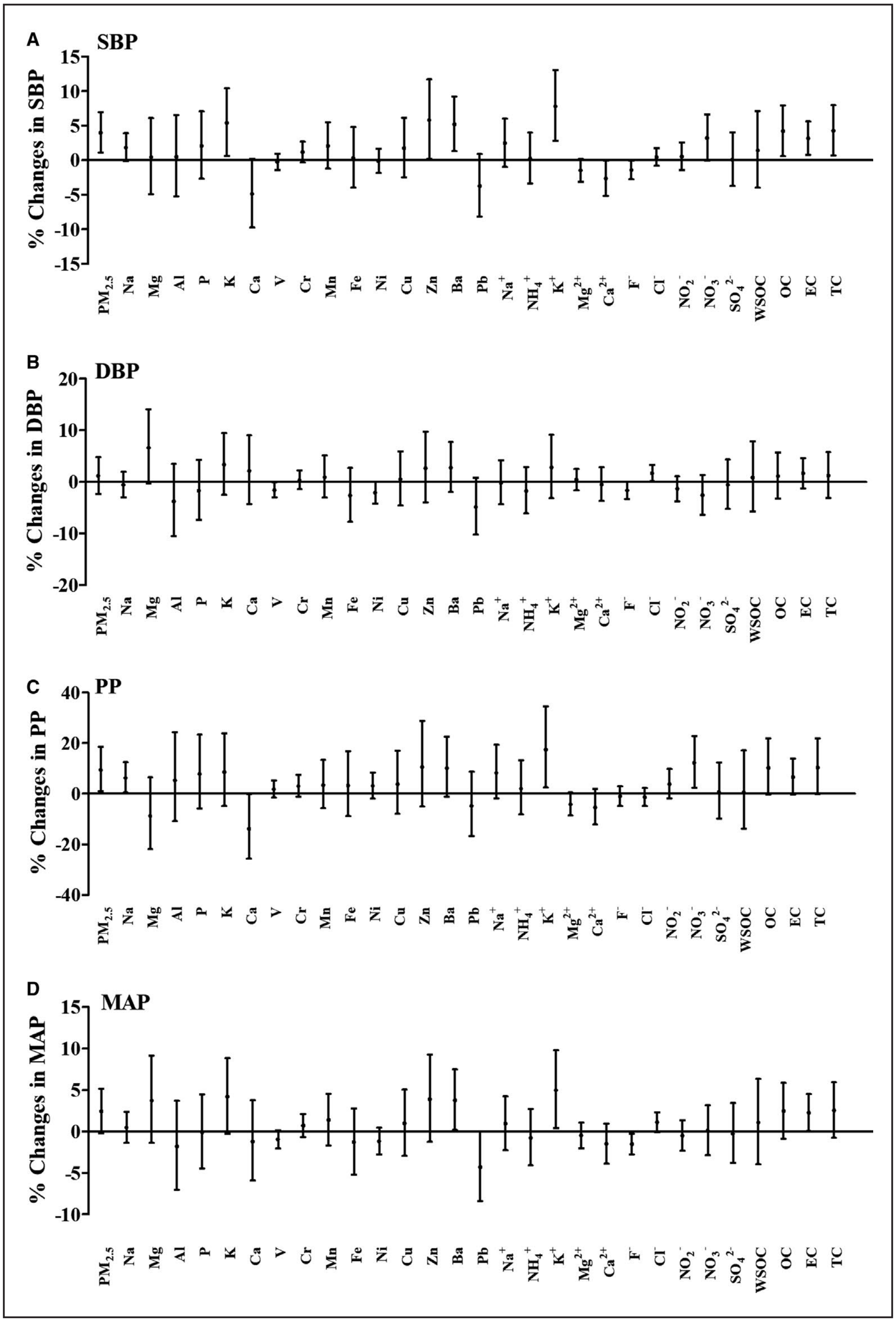

Figure 3. Percent changes (mean and 95% CIs) in blood pressure associated with an interquartile range increase in 24-hour average concentrations of $PM_{2.5}$ constituents in the single-constituent model.

A, SBP indicates systolic blood pressure; (**B**) DBP indicates diastolic blood pressure; (**C**) PP indicates pulse pressure; (**D**) MAP indicates mean arterial pressure. EC indicates elemental carbon; OC, organic carbon; $PM_{2.5}$, particle with aerodynamic diameter ≤2.5 μm; TC, total carbon; and WSOC, water-soluble organic carbon.

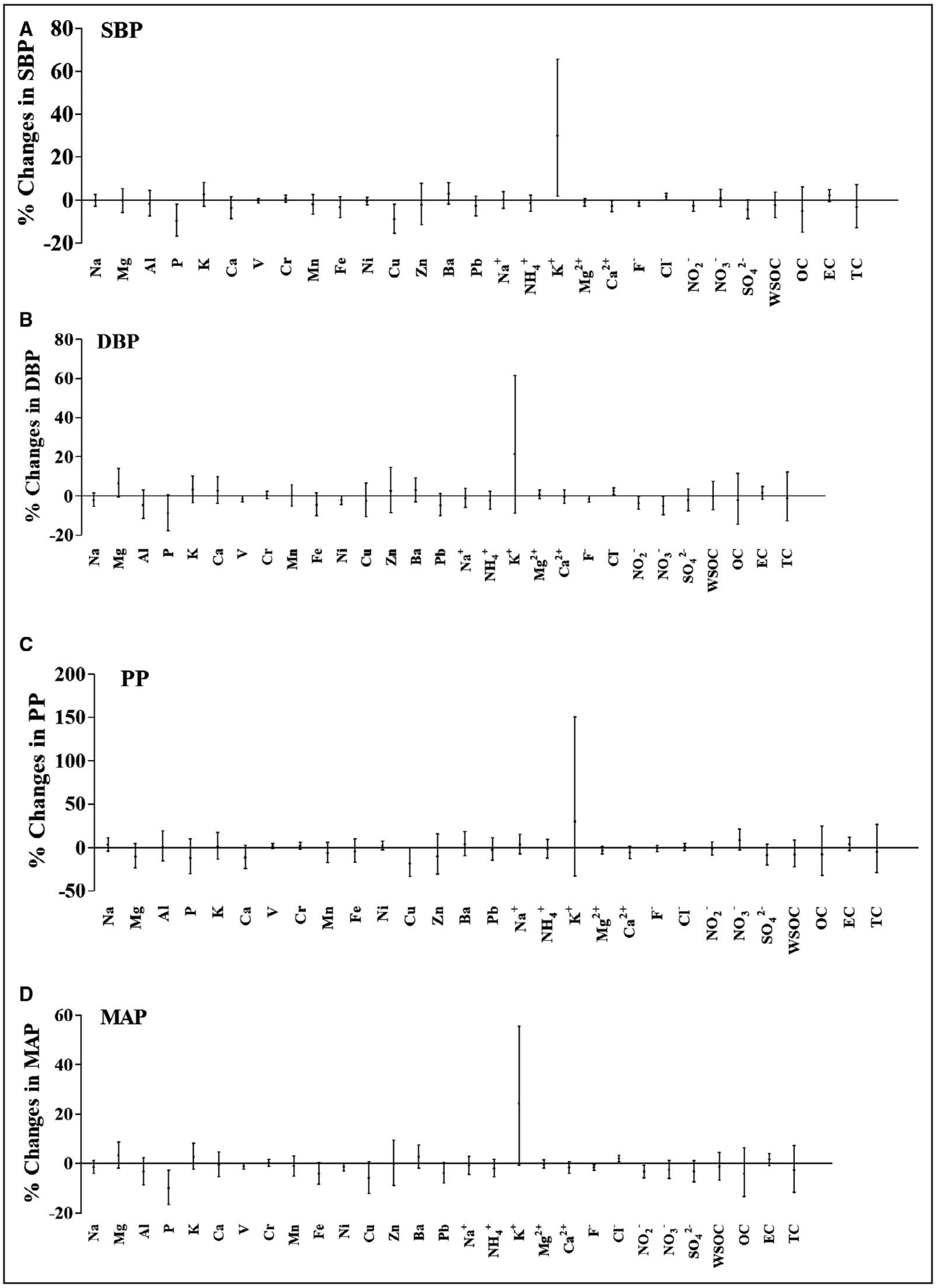

Figure 4. Percent changes (mean and 95% CIs) in blood pressure associated with an interquartile range increase in 24-hour average concentrations of $PM_{2.5}$ constituents in the constituent-$PM_{2.5}$ joint model.

A, SBP indicates systolic blood pressure; (**B**) DBP indicates diastolic blood pressure; (**C**) PP indicates pulse pressure; (**D**) MAP indicates mean arterial pressure. EC indicates elemental carbon; OC, organic carbon; $PM_{2.5}$, particle with aerodynamic diameter ≤2.5 μm; TC, total carbon; and WSOC, water-soluble organic carbon.

was reported to have an association with BP in several studies.[10,12,43] For instance, Wu et al found positive associations among zinc, nickel, magnesium, strontium, lead, arsenic, and SBP and DBP, whereas chromium, manganese, and molybdenum had significant inverse associations with SBP or DBP.[12] In our study, we found consistent positive associations between chloride and DBP but negative associations between fluoride and MAP in all 3 models. Airborne chloride in urban areas might mainly

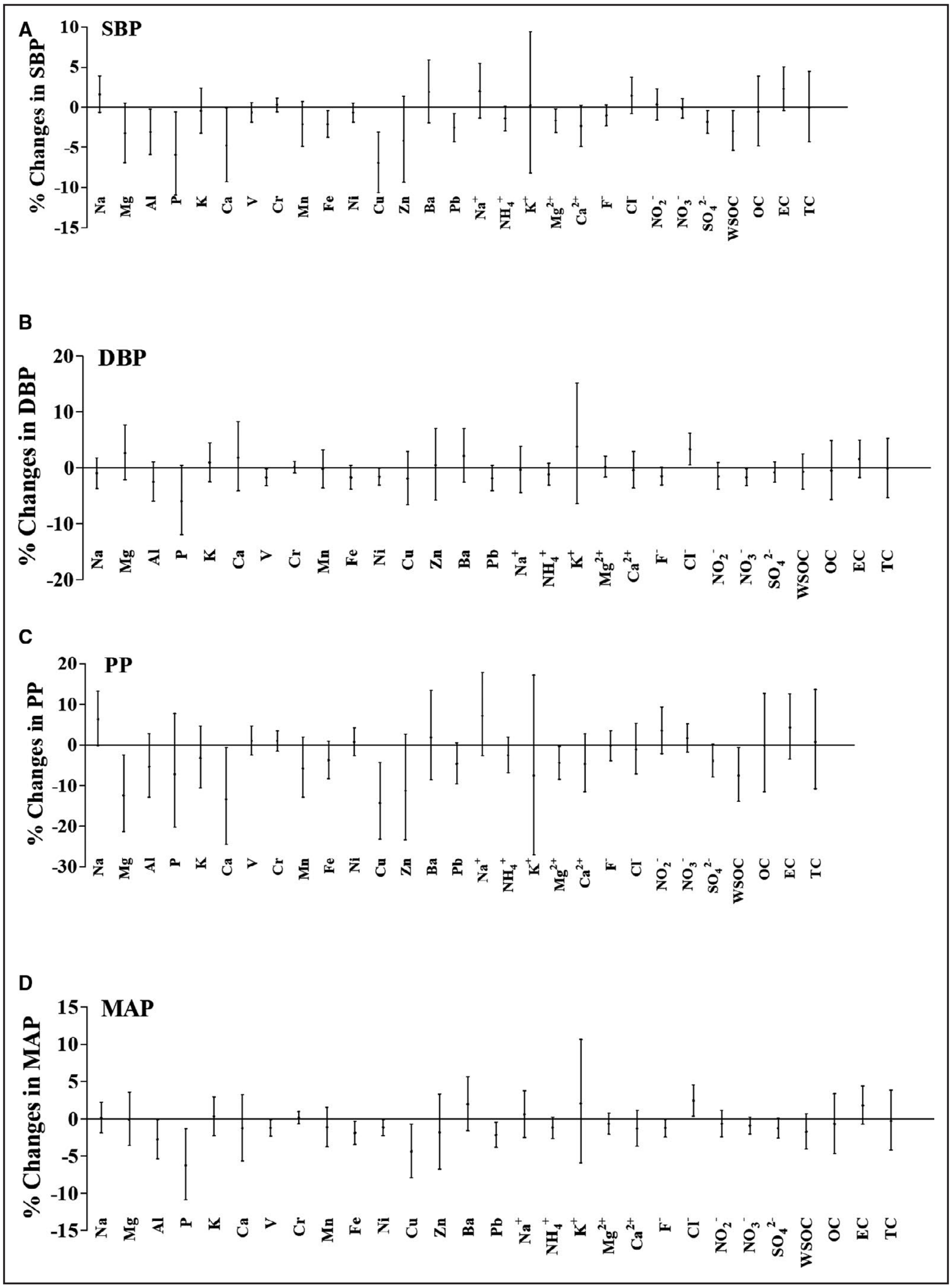

Figure 5. Percent changes (mean and 95% CIs) in blood pressure associated with an interquartile range increase in 24-hour average concentrations of $PM_{2.5}$ constituents in the constituent–residual model.

A, SBP indicates systolic blood pressure; (**B**) DBP indicates diastolic blood pressure; (**C**) PP indicates pulse pressure; (**D**) MAP indicates mean arterial pressure. EC indicates elemental carbon; OC, organic carbon; $PM_{2.5}$, particle with aerodynamic diameter ≤2.5 μm; TC, total carbon; and WSOC, water-soluble organic carbon.

come from polyvinyl chloride plastic burning in refuse dumps, and pollutants such as fluoride might also come from trash compacting or incineration.[44] Consistent with our findings, epidemiologic study found that chloride was slightly but robustly associated with SBP and DBP.[11] Previous animal experimental studies have demonstrated that dietary intake of chloride could be associated with increased BP in rats,[45] which indicates that chloride might be associated with increased BP. Wu et al found smaller consistent associations between fluoride and DBP.[12] Evidence for the association between airborne ions such as chloride and fluoride and BP still requires further investigation.

We also found that increased BP is associated with OC, EC, and total carbon in single-constituent models, but the result became inconsistent in adjusted models. Studies have demonstrated that carbonaceous particles (such as OC and EC) might come from traffic emissions.[44] Lin et al found that ambient OC and EC exposure had significant associations with increased risk of cardiovascular disease mortality in Guangzhou,[46] and another panel study in Shanghai found that OC and EC had associations with BP increment among people with chronic obstructive pulmonary disease.[11]

However, we also found negative associations between vanadium and DBP, in which vanadium can be generated from oil combustion.[47,48] Similarly, Jacobs et al found negative associations between DBP and vanadium,[10] and Sorensen et al found that vanadium was associated with oxidative stress,[49] which may have influenced BP.

Plausible Biological Mechanisms

We found consistent associations among $PM_{2.5}$, $PM_{1.0}$, $PM_{0.5}$, $PM_{0.2}$, and BP except for $PN_{0.1}$. $PM_{2.5}$ and $PM_{1.0}$ were highly correlated in our study, which was consistent with a previous study showing that that $PM_{1.0}$ is the major component of $PM_{2.5}$ in China.[50] Associations between $PM_{2.5}$ and $PN_{0.1}$ were inconsistent, and one possible biological mechanism for this result could be the weak correlation between $PM_{2.5}$ and $PN_{0.1}$ (r=0.18). Similarly, Rich et al found that ultrafine particles and $PM_{2.5}$ were weakly correlated (r=0.11),[51] and some studies have found that ultrafine particles and $PM_{2.5}$ have a weak inverse correlation (r=−0.18).[52] De Jesus et al found that particle number concentration and $PM_{2.5}$ measurements show little correlation, and they are not representative of each other.[53] That may be because particles sized smaller than 0.1 μm do not contribute very much to $PM_{2.5}$ because the ultrafine particles mass is low compared with those of the larger fractions. Gong et al found that PM-induced pathophysiological pathways affected by the fine and ultrafine size fraction of $PM_{2.5}$ might be independently.[52] Therefore, although some studies have found that PM-related health effects may be greater with smaller-sized PM,[26] other studies have shown that fine and coarse particles, instead of ultrafine particles, might be responsible for the associations between PM and health.[54,55] The differences in health effects between size-fractionated particles might reflect differences in clearance, deposition, and translocation after inhalation.[24,52]

Possible mechanistic explanations for the observed association include autonomic nervous system imbalance, systemic inflammation and oxidative stress, and the increase in plasma endothelin-1.[10] Sympathovagal balance of cardiovascular regulation is regarded as the major determinant of BP variability.[56] Inhaled particles stimulate the nerve endings and receptors in the airways, resulting in an imbalance of the autonomic nervous system[57] and vasoconstriction.[58,59] This may help explain the rapid changes of BP in response to short-term exposure to PM in our study. Second, systemic inflammation and oxidative stress induced by PM may enhance vasoconstriction and play a vital role in the cardiovascular diseases pathophysiologic process such as hypertension.[60] Short-term exposure to PM can lead to measurable systemic inflammation and oxidative stress in adults.[61,62] Third, short-term PM exposure may be associated with acute endothelial response and artery vasoconstriction.[63]

Strengths and Limitations

Our study has several strengths. First, we recruited nonsmoking, healthy college students rather than a susceptible population, that is, our participants had good compliance, were free of any cardiovascular disease, and did not take antihypertensive medication. Compared with the elderly, young people are less likely to be chronically exposed to various environmental confounders that may have cumulative effects on health. Second, size-fractionated PM with a wide range of smaller diameters (5 cumulative size fractions with upper limits from 0.1 to 2.5 μm) and various $PM_{2.5}$ constituents were measured in a region where air pollution levels are much higher than those in developed countries, adding more evidence to the understanding of the relationship between PM size and constituents and BP.

There are also several limitations to our study. First, PM measurement data were obtained on the rooftop of the school building rather than personal exposure measurements; thus a potential exposure measurement error cannot be fully excluded in this study. However, the measurement site was at the same campus where participants were recruited and the campus only covers an area of ≈0.39 km^2, so that the exposure misclassification may be quite low. Second, ambient gaseous pollutants and noise, both of which may contribute to the BP, were not monitored in this study, and their effect on BP should not be ignored. Third, the strong correlations among different particle-size fractions meant that we were not able to identify the independent effects of the different-sized PM. Fourth, we assessed the associations between 5 individual PMs with 6 different lag times and 4 BP outcomes, leading to a large number of statistical tests that might have inflated the amount of type I errors, leading to false-positive results, or results by chance. However, we did not correct for multiple testing in order to maximize our ability to detect and confirm modest effects of PM exposures by future investigations. Fifth, because study participants are young and nonhypertensive, there are limits to applying this result to other populations such as older or hypertensive adults. Finally, other information, such as dietary intake and detailed daily activities, which may have an influence on BP, were unfortunately not collected in this study and therefore we were not able to account for them.

Perspectives

In summary, this study suggests that short-term PM exposure is positively associated with SBP, DBP, and MAP in the first 24 hours following exposure and some $PM_{2.5}$ constituents also seem to be related to BP in China. The observed PM–BP associations should not only focus on $PM_{2.5}$, but also on smaller particles as well as $PM_{2.5}$ constituents. It should be noted that the prevalence of hypertension in younger adults is steadily rising,[64] and young adults with elevated BP may have an increased risk of cardiovascular events later in life.[4] Our results could add evidence to the association of PM-related BP in the young population, help us more deeply understand the physiological mechanisms of the development of hypertension, and also provide healthcare professionals with suggestions or treatments for people to take preventive measures, and ultimately to mitigate the effects of air pollution on hypertension. Further studies will help to clarify the role of particle size and chemical composition on BP.

ARTICLE INFORMATION

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Disclosures

None.

Supplementary Material

Tables S1–S2

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SUPPLEMENTAL MATERIAL

Table S1. Basic main characteristics of study participants (n = 88).

Characteristics	Male (n = 34)	Female (n = 54)	Total (n = 88)
Age, mean ± SD, years	21.35 ± 1.12	21.61 ± 1.09	21.51 ± 1.10
BMI, kg/m^2, mean ± SD	21.65 ± 1.67	21.3 ± 3.53	21.43 ± 2.94
Exercise time, n (%)			
0 time per week	13 (31.71%)	28 (68.29%)	41 (46.59%)
1 time per week	8 (42.11%)	11 (57.89%)	19 (21.59%)
2 times per week	10 (50.00%)	10 (50.00%)	20 (22.73%)
⩾3 times per week	3 (37.50%)	5 (62.50%)	8 (9.09%)
Seated blood pressure			
SBP, mean ± SD, mmHg	120.38 ± 12.80	107.45 ± 12.50	112.45 ± 14.07
DBP, mean ± SD, mmHg	73.83 ± 8.52	69.20 ± 8.69	70.99 ± 8.90
PP, mean ± SD, mmHg	46.55 ± 11.62	38.25 ± 10.57	41.46 ± 11.68
MAP, mean ± SD, mmHg	89.35 ± 8.54	81.95 ± 8.81	84.81 ± 9.40

SD, standard deviation; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.

Table S2. Spearman correlations of air pollutants and meteorological conditions*

Exposures	$PM_{2.5}$	$PM_{1.0}$	$PM_{0.5}$	$PM_{0.2}$	$PN_{0.1}$	Temperature (℃)	Relative humidity (%)
$PM_{2.5}$	1.00						
$PM_{1.0}$	1.00	1.00					
$PM_{0.5}$	0.99	0.99	1.00				
$PM_{0.2}$	0.71	0.71	0.75	1.00			
$PN_{0.1}$	0.18	0.17	0.20	0.54	1.00		
Temperature (℃)	0.27	0.29	0.33	0.46	0.40	1.00	
Relative humidity (%)	0.12	0.14	0.16	0.10	-0.14	0.17	1.00

$PM_{2.5}$, particle with aerodynamic diameter ≤ 2.5 µm; $PM_{1.0}$, particle with aerodynamic diameter ≤ 1.0 µm; $PM_{0.5}$, particle with aerodynamic diameter ≤ 0.5 µm; $PM_{0.2}$, particle with aerodynamic diameter ≤ 0.2 µm; $PN_{0.1}$, particle with aerodynamic diameter ≤ 0.1 µm;

* All correlations are statistically significant.