

AIR POLLUTION

Dietary Pattern and Long-Term Effects of Particulate Matter on Blood Pressure: A Large Cross-Sectional Study in Chinese Adults

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ABSTRACT: Previous experimental studies have identified specific foods or nutrients are capable of mitigating adverse effects induced by air pollution. However, whether the dietary pattern can modify the associations between long-term particulate matter (PM) and increasing blood pressure (BP) among adults has not yet been assessed. We assessed whether the dietary pattern and various foods modify the associations between long-term exposure to PM (PM₁, PM_{2.5}, and PM₁₀), hypertensive BP (BP \geq 140/90 mmHg, HBP), and BP in Chinese adults. This study included 61 081 participants from China Multi-Ethnic Cohort. PM was assessed through satellite-based random forest approaches. Outcomes were analyzed with logistic regression models and linear regression models. The dietary approaches to stop hypertension (DASH) diet was calculated for each participant. This study founds long-term exposure to PM was associated with HBP, systolic BP, and pulse pressure. The DASH diet modified the associations between PM, HBP, and some BP components. For each 10 $\mu\text{g}/\text{m}^3$ increase in PM₁, PM_{2.5}, and PM₁₀, the participants with the lowest quintile of DASH score had HBP risks with odds ratios (95% CI) of 1.196 (1.084–1.319), 1.145 (1.09–1.202), and 1.080 (1.045–1.117), whereas those with the highest quintile of DASH score had lower HBP risks with odds ratios (95% CI) of 1.063 (0.953–1.185), 1.074 (1.017–1.133), and 1.038 (1.000–1.077). Consuming more fresh fruits, vegetables, dairy, and whole grains would reduce the risk of raised BP caused by PM. In conclusion, the DASH diet rich in antioxidant compounds may be a wide-reaching intervention to reduce the deleterious impact of PM. (*Hypertension*. 2021;78:184–194. DOI: 10.1161/HYPERTENSIONAHA.121.17205.)

• Data Supplement

Key Words: air pollution ■ blood pressure ■ dietary approaches to stop hypertension ■ nutrients ■ particulate matter

Hypertension has been recognized as the leading cause of cardiovascular diseases and mortality worldwide.^{1,2} Indeed, high systolic blood pressure (SBP) has become the leading factor in attributable deaths globally, contributing to 10.8 million deaths (accounting for 19.2% of total deaths) in 2019.³ Findings from previous studies suggested that long-term exposure to air pollution was associated with

an increased risk of hypertension development.^{4–10} Air pollution has been recognized as the fourth-leading cause of mortality globally, especially in low- and middle-income countries where air pollution concentrations continuously rise.³ Despite the large-scale emissions-control policies used by the government and regulators, the global disease burden of air pollution has still increased over the past 20 years.¹¹ To reduce

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Novelty and Significance

What Is New?

- Whether dietary pattern can modify the associations between long-term air pollution exposure and blood pressure among adults has not been previously investigated.

What Is Relevant?

- The dietary approaches to stop hypertension diet modified the associations of long-term exposure to three kinds of particulate matter (PM₁, PM_{2.5}, and PM₁₀) with hypertensive blood pressure, systolic blood pressure, and diastolic blood pressure.
- Consuming more fruits, vegetables, dairy, and whole grains may reduce the risk of high blood pressure caused by particulate matter.

Summary

The dietary approaches to stop hypertension dietary pattern may have the ability to reduce the adverse effect of air pollution on cardiovascular health, even for the population that is most highly exposed. Adequate intake of foods rich in antioxidant compounds may be an effective and wide-reaching intervention to reduce the deleterious impact of air pollution on health.

Nonstandard Abbreviations and Acronyms	
BP	blood pressure
CMEC	China Multi-Ethnic Cohort
DASH	dietary approaches to stop hypertension
DBP	diastolic blood pressure
HBP	hypertensive BP
PM	particulate matter
SBP	systolic blood pressure

this disease burden, individual-level interventions may be used to complement emissions-control policies.

Air pollution causes harm to the cardiovascular system through inflammatory and oxidative stress pathways.¹² Previous experimental studies have identified that dietary antioxidants and anti-inflammatory substances could ameliorate various cardiovascular effects of air pollution by reducing oxidative stress and inflammation.^{13,14} These findings provide biological plausibility for the application of diet intervention to reduce the health effects of air pollution. However, current epidemiological evidence of dietary modification effects is based mainly on a few specific food items (such as vegetables and fruits) and lacks studies on other common foods, such as dairy products and whole grains.^{15–18} Studying a few specific foods may generate confounding effects due to the synergistic correlations among multiple food items. Moreover, instead of eating isolated food, people intake multiple food items in combination.¹⁹ The dietary pattern, defined as the quantity and combination of different foods in a diet, is more closely related to real-world situations.²⁰ It seems more reasonable to investigate the modification effect of dietary patterns on air pollution-related health effects, in which the results can be more easily translated into practical advice for the public.^{19,21,22} To date, no study has investigated the modification of

usual dietary patterns on the associations among long-term air pollution and BP.

In this study, we assessed whether the dietary eating pattern and various food items modify the associations between long-term exposure to ambient particulate matter (PM, including PM₁, PM_{2.5}, and PM₁₀) and hypertensive BP (BP≥140/90 mm Hg, HBP) and BP in Chinese adults, whose PM exposure concentrations are relatively higher than the limit recommended by the WHO guidelines. Research aimed at better understanding the potential role of diet in modifying the air pollution risk of cardiovascular disease. Research also provided insight into individual-level interventions to reduce the health impacts of air pollution worldwide, particularly for populations that are most highly exposed.

METHODS

Study Population

The data that support the findings of this study are available on reasonable request to the corresponding author. We studied participants from the baseline data of the CMEC study (China Multi-Ethnic Cohort), which has been previously presented.²³ Briefly, the cohort recruited participants aged 30 to 79 years from 5 provinces of Southwest China using a multistage, stratified cluster sampling method. The baseline survey was conducted from May 2018 to September 2019. Overall, a total of 99, 556 participants were enrolled in this cohort. All the participants signed an informed consent form before data collection. Ethical approval was received from the Sichuan University Medical Ethical Review Board.

This study was based on the CMEC baseline survey, which consisted of an electronic questionnaire with face-to-face interviews, medical examinations, and clinical laboratory tests. The electronic questionnaire included demographic characteristics, socioeconomic characteristics, health behaviors, physician-diagnosed diseases, medication history, family history of diseases, fertility history, physical activity, habitual diet, and psychological conditions. Medical examinations were conducted mainly using resources and personnel at local clinical centers.

Before the investigation, standardized training for the doctors and nurses was implemented. The medical examinations provided information including height, weight, BP, heart rate, ultrasound, X-rays, blood, and urine tests. We excluded residents in Abo because they lived nomadically and had no fixed residence. We also excluded residents in Tibet. Tibet has a special environment of high altitude (generally >3500 meters above sea level), low air pollution, low atmospheric pressure, hypoxia, and extreme cold, which has an important influence on the cardiovascular system and BP regulation.²⁴ Tibetans have a unique dietary habit, consisting of Zamba mixed with buttery tea or highland barley wine as the staple food, which is quite different from those at lower altitudes. Moreover, the pathogenesis of systemic hypertension contains important differences between highlanders and lowlanders.^{25,26} Because of these heterogeneities, there is no comparison between Tibetans and lowlanders. In addition, participants were excluded from this study if they (1) had an incomplete address; (2) resided at their present address for fewer than 3 years at the time of the investigation; (3) had any physician-diagnosed hypertension (considering that hypertensive patients may change eating patterns); or (4) had no available information on any outcome, exposure, or adjusted covariates. After these exclusions, 61 081 participants aged 30 to 79 years on the day of the investigation were included in the analyses (Figure S1 in the [Data Supplement](#)).

Air Pollution Exposure Assessment

Air pollution data during the study period were obtained from the ChinaHighAirPollutants (CHAP) data set (<https://weijing-rs.github.io/product.html>, accessed data: July 9, 2020). Daily PM₁, PM_{2.5}, and PM₁₀ concentrations were predicted by Wei et al^{27–31} at a 1 km×1 km spatial resolution using the Moderate Resolution Imaging Spectroradiometer Multiangle Implementation of Atmospheric Correction AOD product, meteorology, land use information, pollution emissions, and other spatial and temporal predictors. A space-time extremely randomized trees model was used for the estimation. A detailed description of the estimation has been described in previous studies.^{27–31} The results showed a high predictive ability with 10-fold cross-validation R² (root mean square error) values for the daily predictions of PM₁, PM_{2.5}, and PM₁₀ being 0.77 (14.6 μg/m³), 0.90 (10.01 μg/m³), and 0.86 (24.28 μg/m³), respectively. According to the geocoded residential address, average concentrations of PM₁, PM_{2.5}, and PM₁₀ during the 3 years before the baseline survey were calculated for each participant as the estimated surrogate of exposure.

Outcome Assessment

The BP measurements of study participants were taken by trained medical personnel using electronic sphygmomanometers at the baseline survey. All electronic sphygmomanometers were factorially calibrated before measurement. The American Heart Association's standardized protocol for the measurement of BP was strictly followed.³² All BP measurements were performed in a seated, upright position 3 times. Participants were told not to smoke, drink alcohol, drink coffee or tea, or exercise at least 30 minutes before the measurement. The 3 measurements were averaged to calculate the SBP and diastolic BP (DBP). Pulse pressure (PP) was calculated as the difference between SBP and DBP.³³ In this analysis, HBP was defined

as having an average measured SBP≥140 mmHg or DBP≥90 mmHg (BP≥140/90 mmHg).^{34,35}

Dietary Intake Assessment

The dietary approaches to stop hypertension (DASH) diet was developed to prevent and control hypertension without medication and has shown beneficial effects on decreasing SBP and DBP among hypertensive and normotensive individuals.^{36,37} The DASH diet emphasizes the consumption of vegetables, fruits, dairy products, and whole grains, providing a diet highly enriched in antioxidants and anti-inflammatory compounds.³⁸ Therefore, we hypothesized that higher adherence to the DASH diet may mitigate the adverse effects of PM on hypertension.

In this study, we calculated the DASH score for each participant based on information in CMEC baseline survey. Participants completed a food frequency questionnaire to assess their dietary intake in the baseline survey. The food frequency questionnaire was calibrated through preliminary investigation, and further validation was performed through a 24-hour dietary survey of the subsample. For each food item, there was a standard portion size and 4 kinds of frequency of consumption response in the food frequency questionnaire, ranging from how many times per year to how many times per day. The DASH score focuses on 7 kinds of food: vegetables, fresh fruits, legumes, whole grains, red and processed meat, dairy, and sodium salt. We assigned each kind of food a score of 1 to 5 according to the quintile of the average food intake.^{39,40} Higher intakes of whole grains, fresh fruits, fresh vegetables, legumes, and dairy products yielded higher scores, whereas higher levels of red meat and products and sodium intake yielded lower scores. Then, we summed all the food scores to obtain an overall DASH score ranging from 7 to 35.

Statistical Analysis

Multivariable logistic regression models were used to evaluate the long-term effects of exposure to PM on HBP as a dichotomous outcome. Multivariable linear regression models were used to evaluate long-term effects between PM and BP as a continuous measure. The estimated effect takes the form of odds ratios for HBP and changes in mmHg for BP as PM concentrations increase per 10 μg/m³.

Based on the previous literature on air pollution and BP, fully adjusted models included the following covariates: age, sex (male and female), annual family income (<12 000, 12 000–19 999, 20 000–59 999, 60 000–99 999, and ≥100 000 yuan), education level (illiteracy, primary school, junior high school, high school, junior college, and above), marital status (married or cohabiting, widowed, separated or divorced, and never married), region, ethnic group (Han and minority), alcohol status (never, occasionally, and regularly), smoking status (never, smoking, or quit smoking), secondary smoking (yes and no), indoor air pollution, physical activity, DASH score, body mass index, hypertension family history (yes, no, and not sure), and 2 meteorologic characteristics (annual temperature and humidity). For alcohol status, occasional drinking was defined as drinking an average of 1 to 2 days per week in the past year, and regular drinking was defined as drinking an average of 3 to 5 days per week in the past year. Body mass index was calculated as the weight (kg) divided by the square of the height (m). Indoor air pollution, a summary of cooking behavior, fuel types,

and ventilation equipment, was divided into 3 levels. The low level was defined as occasionally or no cooking at home. The moderate level was defined as frequently cooking at home and satisfaction of only one of the following conditions: (1) using unclean fuel or (2) not using ventilation equipment. The high level was defined as frequently cooking at home using unclean fuel without ventilation equipment. Physical activity was qualified by metabolic equivalent tasks per day, including participants' occupational, traffic, chores, and leisure time activities and then divided into 4 levels based on the quartile range of metabolic equivalent tasks per day.

Each potential effect modification of DASH diet was assessed in separate models by adding multiplicative interaction terms between PM and the DASH score. To test the statistical significance of each interaction, we used *P* values of the likelihood ratio statistic to compare model fit with and without interaction terms. Then, we divided DASH score into 5 levels based on the quintile range of DASH score and assessed the associations between long-term exposure to PM, HBP, and BP by each level. Higher quintiles of DASH score represent more adherence to DASH diet. In addition, we also considered the effect modifiers of various food items, including vegetables, fresh fruits, legumes, whole grains, red and processed meat, dairy, and sodium salt. Each kind of food was incorporated into the model in the form of food score.

Sensitivity Analyses

We performed a series of sensitivity analyses. (1) We additionally adjusted for preexisting diseases (diabetes, hyperlipidemia, and cardiovascular disease) to minimize the influence of comorbidities. (2) We assessed the linearity assumption of the exposure-response relationship using penalized spline models. (3) To examine the robustness of the results, we used average concentrations of PM for 1, 3 months and 1, 2, 4 years before the baseline survey to evaluate the long-term effects of PM exposure.

In addition, because DASH diet is recommended for patients with hypertension, we also added the following sensitivity analyses to complement the modifying effect of the DASH diet on the risk of PM-induced hypertension: (1) We evaluated the modifying effect of DASH diet on PM-induced risk of elevated BP among participants diagnosed with hypertension. (2) We included the participants diagnosed with hypertension into this study and then estimated the modifying effect of DASH diet on PM-induced risk of elevated BP. In this sensitivity analysis, we used an alternative definition of HBP, including both $BP \geq 140/90$ mm Hg and hypertensives as cases. In the above 2 sensitivity analyses, we additionally adjusted the hypertensive medication variable (regular use of antihypertensive drugs; irregular use; no use).

All statistical analyses were used using R (version 3.6.1).

RESULTS

The general characteristics of the study participants are summarized in Table 1. Additionally, we presented the basic characteristics of the participants by quintiles of DASH score in Table S1. Among the 61 081 participants included in the study, the mean age was 50.65 years (range from 30

to 79 years), and 61.2% of the participants were women. Overall, 20.0% of the participants were measured as HBP ($BP \geq 140/90$ mm Hg) by this survey. Participants with HBP were statistically older than normotensive participants (55.76 versus 49.37 years), had a higher body mass index (24.81 versus 23.49 kg/m²), and had greater exposure to alcohol and smoking. Normotensive participants were more likely to be married, have a higher educational level, and have a higher physical activity level (27.90 versus 25.88 metabolic equivalent tasks/d). In addition, participants with HBP had lower DASH score (19.87 versus 20.74) and lower consumption of dairy, fruit, whole grains, and legumes.

The 3-year average concentrations of PM_{10} , $PM_{2.5}$, and PM_{10} for the overall study were 28.62 $\mu\text{g}/\text{m}^3$ (SD: 6.70), 42.79 $\mu\text{g}/\text{m}^3$ (SD: 16.00), and 73.36 $\mu\text{g}/\text{m}^3$ (SD: 23.88), respectively. In general, participants with HBP tended to have higher PM_{10} and $PM_{2.5}$ exposures than normotensive participants. The HBP participants had similar PM_{10} exposure levels to normotensive participants (73.31 and 73.55 $\mu\text{g}/\text{m}^3$).

Table 2 shows that long-term exposure to PM was significantly associated with HBP. For each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} , $PM_{2.5}$, and PM_{10} , the odds ratios of HBP were 1.039 (95% CI, 1.009–1.070), 1.013 (95% CI, 1.000–1.025), and 1.004 (95% CI, 0.996–1.013) in the crude model, respectively. After fully adjusting for confounders (adjusted model 1), the effect estimates were higher, with 1.109 (95% CI, 1.027–1.198), 1.101 (95% CI, 1.055–1.148), and 1.053 (95% CI, 1.022–1.084) per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} , $PM_{2.5}$, and PM_{10} , respectively. The results remained similar after adjusting for various scores of food items instead of the DASH score (adjusted model 2).

Ambient PM was also associated with increased SBP and PP (Table S2). For SBP, each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} , $PM_{2.5}$, and PM_{10} concentration was associated with SBP elevations of 0.253 (95% CI, –0.182 to 0.689) mm Hg, 0.272 (95% CI, 0.107–0.436) mm Hg, and 0.637 (95% CI, 0.397–0.878) mm Hg in adjusted model 1, respectively. For PP, each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} , $PM_{2.5}$, and PM_{10} corresponded to 0.513 (95% CI, 0.211–0.815) mm Hg, 0.333 (95% CI, 0.218–0.447) mm Hg, and 0.666 (95% CI, 0.500–0.833) mm Hg increases, respectively. The results were still robust after adjusting for various scores of food items (adjusted model 2). We did not find a significant association between PM and DBP in either crude or fully adjusted models.

Figure 1 (and Table S3) displays the PM-HBP associations by quintiles of DASH score. The results suggested that the DASH score could modify the PM-HBP associations ($P_{\text{interaction}} < 0.01$). We observed decreasing associations of PM_{10} , $PM_{2.5}$, and PM_{10} with HBP as the DASH score increased. For BP (Figure S3 and Table S3), the associations of PM_{10} and $PM_{2.5}$ with SBP were significantly reduced with higher DASH score quintiles. A significant effect modification of DASH score between the 3 kinds of PM and DBP was also found, although the PM-DBP

Table 1. Basic Characteristics of Study Participants

Characteristics*	Normotension (n=48878)	HBP (n=12 203)	P value
Systolic blood pressure, mm Hg (SD)	116.84 (11.47)	147.65 (13.38)	<0.001
Diastolic blood pressure, mm Hg (SD)	74.72 (7.78)	90.62 (9.65)	<0.001
Pulse pressure, mm Hg (SD)	42.12 (9.37)	57.03 (15.05)	<0.001
3-y average PM ₁ , µg/m ³ (SD)	28.58 (6.74)	28.76 (6.55)	0.01
3-y average PM _{2.5} , µg/m ³ (SD)	42.73 (16.12)	43.05 (15.54)	0.045
3-y average PM ₁₀ , µg/m ³ (SD)	73.31 (24.08)	73.55 (23.06)	0.321
DASH score (SD)	20.74 (4.46)	19.87 (4.51)	<0.001
Dairy score (SD)	2.68 (1.61)	2.44 (1.59)	<0.001
Fruits score (SD)	3.06 (1.30)	2.80 (1.34)	<0.001
Vegetables score (SD)	2.93 (1.39)	2.99 (1.41)	<0.001
Whole grains score (SD)	2.69 (1.45)	2.52 (1.47)	<0.001
Read and processed meats score (SD)	3.27 (1.29)	3.28 (1.32)	0.483
Legumes score (SD)	3.09 (1.39)	2.99 (1.43)	<0.001
Sodium score (SD)	3.01 (1.38)	2.86 (1.40)	<0.001
Age, y (SD)	49.37 (10.75)	55.76 (10.61)	<0.001
Education level, n (%)			<0.001
Illiteracy	9777 (20.0)	3173 (26.0)	
Primary school	11 878 (24.3)	3332 (27.3)	
Junior high school	14 073 (28.8)	3227 (26.4)	
High school	6360 (13.0)	1467 (12.0)	
Junior college and above	6790 (13.9)	1004 (8.2)	
Sex, n (%)			<0.001
Female	31 118 (63.7)	6234 (51.1)	
Male	17 760 (36.3)	5969 (48.9)	
Marital status, n (%)			<0.001
Married or cohabiting	44 322 (90.7)	10 673 (87.5)	
Widowed	1911 (3.9)	451 (3.7)	
Separated or divorced	2182 (4.5)	966 (7.9)	
Never married	463 (0.9)	113 (0.9)	
Ethnic			<0.001
Han	31 861 (65.2)	7736 (63.4)	
Minority	17 017 (34.8)	4467 (36.6)	
Annual family income, yuan (%)			<0.001
<12 000	7562 (15.5)	2411 (19.8)	
12 000–19 999	8069 (16.5)	2169 (17.8)	
20 000–59 999	17 975 (36.8)	4399 (36.0)	
60 000–99 999	7797 (16.0)	1743 (14.3)	
≥100 000	7475 (15.3)	1481 (12.1)	
BMI, kg/m ² (%)			<0.001
[0,24]	28 710 (58.7)	5074 (41.6)	
[24,28]	16 021 (32.8)	5114 (41.9)	
[28,Inf]	4147 (8.5)	2015 (16.5)	
Alcohol drinking status, n (%)			<0.001
Never	27 047 (55.3)	6073 (49.8)	
Occasionally	16 085 (32.9)	3545 (29.1)	

(Continued)

Table 1. Continued

Characteristics*	Normotension (n=48878)	HBP (n=12 203)	P value
Regularly	5746 (11.8)	2585 (21.2)	
Smoking status			<0.001
Never	37 172 (76.1)	8413 (68.9)	
Smoking or quit smoking	11 706 (23.9)	3790 (31.1)	
Secondary smoking, n (%)			<0.001
Yes	25 892 (53.0)	5977 (49.0)	
No	22 986 (47.0)	6226 (51.0)	
Indoor air pollution, n (%)			<0.001
High	7786 (15.9)	2075 (17.0)	
Moderate	38 778 (79.3)	9487 (77.7)	
Low	2314 (4.7)	641 (5.3)	
Physical activity, METs/d (%)			<0.001
Quintile 1 [0,13.5]	11 635 (23.8)	3628 (29.7)	
Quintile 2 [13.5,23.8]	12 339 (25.2)	2957 (24.2)	
Quintile 3 [23.8,38.8]	12 420 (25.4)	2832 (23.2)	
Quintile 4 [38.8,142]	12 484 (25.5)	2786 (22.8)	
Hypertension family history, n (%)			<0.001
No	28 057 (57.4)	6412 (52.5)	
Not sure	6423 (13.1)	2065 (16.9)	
Yes	14 398 (29.5)	3726 (30.5)	

BMI indicates body mass index; DASH, dietary approaches to stop hypertension; HBP, hypertensive BP; METs, metabolic equivalent tasks; and PM, particulate matter.

*Data are the mean (SD) for continuous variables and number (percentage) for categorical variables.

risks were almost not statistically significant. We did not observe significant effect modification by DASH score in the associations between the 3 kinds of PM and PP.

We additionally evaluated the effect modifiers of various food items. For HBP (Figure 2), the associations between the 3 kinds of PM and HBP risks were significantly reduced among participants with higher dairy intake, whereas the PM_{2.5}- and PM₁₀-HBP risk associations were significantly lower among those who consumed more whole grains. We also found downward trends of HBP risk induced by the 3 kinds of PM when vegetable intake increased, but these trends were almost not statistically significant. For BP (Figure S4), we observed that the health effects of PM on DBP decreased among subjects who consumed more dairy and whole grains. We also observed that the adverse effects of the 3 kinds of PM on PP were significantly reduced among subjects who consumed more fresh fruits and vegetables.

For the sensitivity analysis, we observed that the results of the 3 kinds of PM on HBP and BP measurements remained robust when using average concentrations of PM for a series of exposure windows (Table S4). The results were also similar after adjusting for preexisting diseases (Table S5). According to the results of the penalized spline models, the linearity assumption of the exposure-response

Table 2. Odds Ratios and 95% CI of HBP Risk Associated With Each 10 $\mu\text{g}/\text{m}^3$ Increase in Long-Term Exposure to Ambient PM

PM	Crude model	Adjust model 1	Adjust model 2
PM ₁	1.039 (1.009–1.070)	1.109 (1.027–1.198)	1.112 (1.029–1.201)
PM _{2.5}	1.013 (1.000–1.025)	1.101 (1.055–1.148)	1.100 (1.055–1.147)
PM ₁₀	1.004 (0.996–1.013)	1.053 (1.022–1.084)	1.052 (1.022–1.083)

Crude Model: no adjustment. Adjusted Model 1: adjusted for age, sex, annual family income, education level, marital status, provinces, ethnicities, smoking status, alcohol drinking status, secondary smoking, indoor air pollution, physical activity, DASH score, BMI, hypertension family history, temperature, and humidity. Adjusted Model 2: adjusted for age, sex, annual family income, education level, marital status, provinces, ethnicities, smoking status, alcohol drinking status, secondary smoking, indoor air pollution, physical activity, dairy score, fruits score, vegetables score, whole grains score, red and processed meats score, legumes score, sodium score, BMI, hypertension family history, temperature, and humidity. PM₁, PM with aerodynamic diameter $\leq 1.0 \mu\text{m}$; PM_{2.5}, PM with aerodynamic diameter $\leq 2.5 \mu\text{m}$; and PM₁₀, PM with aerodynamic diameter $\leq 10 \mu\text{m}$. BMI indicates body mass index; DASH, dietary approaches to stop hypertension; HBP, hypertensive BP; and PM, particulate matter.

relationship was appropriate, except for the associations between PM and DBP, which deviated from linearity (Figure S5). In addition, among those participants diagnosed with hypertension, we also found the effect modification of DASH diet between PM and BP components ($P_{\text{interaction}} < 0.05$), although the PM-BP risks were almost not statistically significant among hypertensives (Figure S6). After including the participants diagnosed with hypertension into analyses, we still found the DASH diet modified the associations between PM, HBP, and BP components (including SBP, DBP, and PP; Figures S7 and S8).

an increased prevalence of HBP and elevated levels of SBP and PP in China, a highly polluted area. The DASH diet generally modified these relationships, as those with a higher DASH score had significantly lower PM-related risks of HBP, SBP, and DBP after adjusting for the usual confounders. In addition, consuming more fresh fruits, vegetables, dairy products, and whole grains reduced the risk of high BP caused by atmospheric particulates. The results provide an opportunity to understand the mechanistic underpinnings of PM-related health effects and to develop novel individual-level interventions.

DISCUSSION

To our knowledge, this is the first study to examine whether usual dietary patterns can modify the associations between PM and HBP as well as BP among adults aged 30 to 79 years. Our study found that long-term exposure to ambient PM₁, PM_{2.5}, and PM₁₀ was associated with

Potential Mechanism

Attenuation of hypertension risk and elevated BP risk associated with PM exposure by dietary pattern is consistent with the hypothesized biological mechanism of the adverse health effects of PM exposure, which was mainly related to inflammation and oxidative stress.

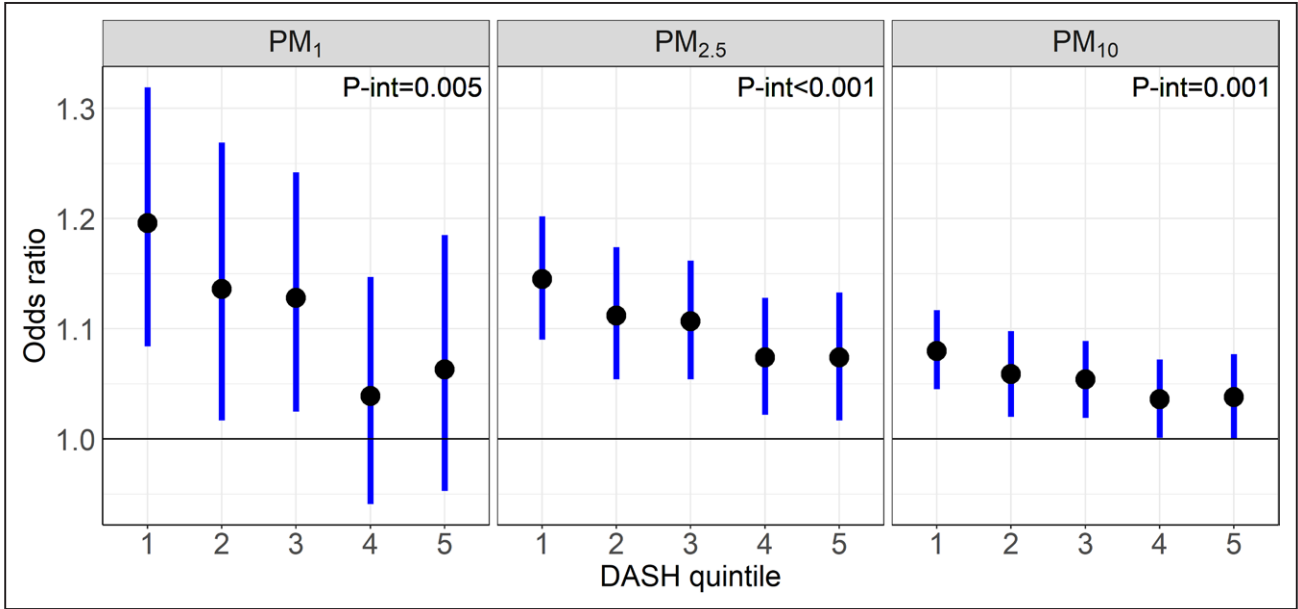


Figure 1. Odds ratio and 95% CI of hypertensive blood pressure (HBP) risk associated with ambient particulate matter (PM) by quintiles of dietary approaches to stop hypertension (DASH) score. $P_{\text{interaction}}$ indicates the P values of the modifier effects of DASH score. At the horizontal coordinate, the values 1, 2, 3, 4, and 5 denote the quintiles of DASH score.

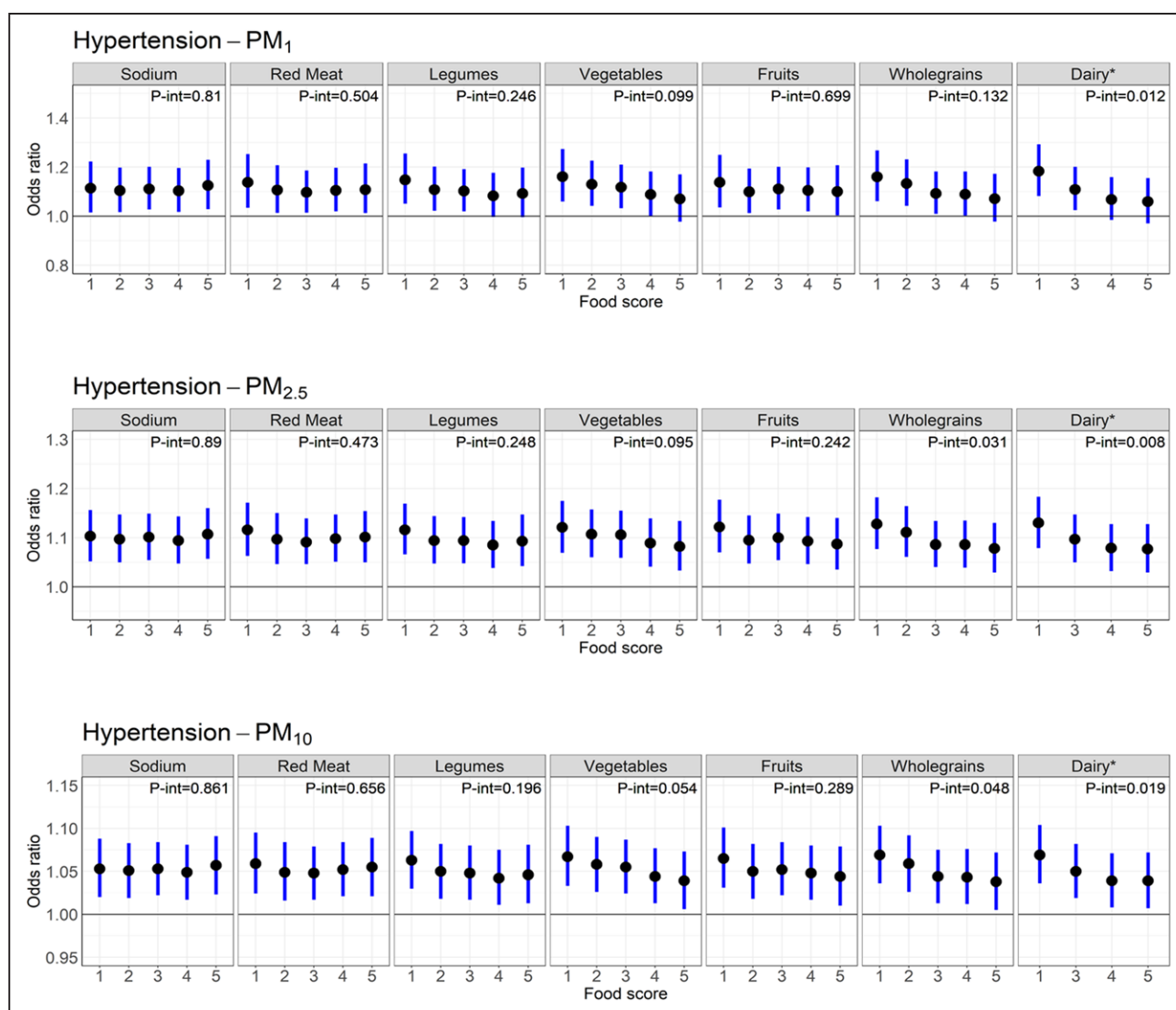


Figure 2. Odds ratio and 95% CI of hypertensive blood pressure (HBP) risk associated with ambient particulate matter (PM) by food score.

$P_{interaction}$ indicates the P values of the modifier effects of each food score. At the horizontal coordinate, the values 1, 2, 3, 4, and 5 denote the quintiles of food score. *The first and second quintiles of dairy intake are both equal to 0, so the lowest and second fifth of dairy score are all assigned a 1 value.

When particles are inhaled into the respiratory tract, oxidative reactions can be triggered and then lead to oxidative stress, systematic inflammation, autonomic nervous system imbalance, endothelial dysfunction, plasma viscosity increases, and DNA methylation,^{41–44} resulting in elevated BP. The DASH dietary pattern, characterized by a high intake of vegetables, fruits, whole grains, and low-fat dairy products, is rich in antioxidant and anti-inflammatory compounds, such as B vitamins, vitamin C, vitamin E, vitamin D, and omega-3 polyunsaturated fatty acids. These antioxidant and anti-inflammatory compounds play a modulating role in defending against inflammatory oxidative stress induced by PM.⁴⁵ For example, vitamins C and E can protect against oxidants and oxidant-mediated damage by scavenging a variety of free radicals and oxidants.⁴⁶ B vitamin supplementation can prevent DNA

methylation changes implicated in inflammation and oxidative stress.⁴⁷ Micronutrients, such as zinc and vitamin A, can influence the function of macrophages in innate immunity and inflammation.⁴⁸ In addition, for the modification effects of specific food items on the associations of air pollution BP, we discovered the attenuation of air pollution BP associations when increasing the consumption of foods rich in antioxidant and anti-inflammatory compounds, including dairy, whole grains, fresh fruits, and vegetables, which supports this hypothesis.

Comparison With Other Studies

Whether the DASH dietary pattern alters the associations between long-term exposure to air pollution and elevated BP has not yet been epidemiologically assessed among

adults. Our research explored this crucial question and made the first discovery that the DASH dietary pattern has the ability to alleviate the adverse effects of PM on HBP, SBP, and DBP. In this study, for exposure to PM₁, PM_{2.5}, and PM₁₀, the participants with the lowest quintile of the DASH score (minimal adherence to the DASH diet) had risks of HBP with odds ratios (95% CI) of 1.196 (1.084–1.319), 1.145 (1.09–1.202), and 1.080 (1.045–1.117), whereas those with the highest quintile of the DASH score (maximal adherence to the DASH diet) had lower risks of HBP with odds ratios (95% CI) of 1.063 (0.953–1.185), 1.074 (1.017–1.133), and 1.038 (1.000–1.077; Table S3). Similar large epidemiological studies relating to the modification effect of dietary patterns as observed here were limited. One analysis of the National Institutes of Health–American Association of Retired Persons (NIH-AARP) Diet and Health Study in less polluted areas found that participants with higher compliance with the Mediterranean Diet had a lower risk of cardiovascular disease mortality associated with long-term PM_{2.5} and NO₂ exposure,⁴⁹ which is in line with our results. It is worth mentioning that our study had a wide range of pollutant concentrations (with an average PM_{2.5} ranging from 18.27 to 105.29 µg/m³), which addressed the gap of whether a healthy dietary pattern can still attenuate air pollution-induced adverse cardiovascular health effects, even when extended to higher levels of air pollution exposure. Moreover, the outcomes of this study were HBP and elevated levels of BP, which are the leading causes of cardiovascular disease.^{1,2} Our findings implied that a healthy dietary pattern may be the fundamental individual-level intervention for the prevention of ambient PM-induced cardiovascular disease at an early stage.

Past studies have sought to examine which specific food items or nutrients have the ability to blunt the association between air pollution exposure and health outcomes. In this study, we observed that higher consumption of dairy and whole grains would reduce the risk of elevated BP induced by ambient PM after adjusting for other food items. Although epidemiological evidence for the modification effects of dairy and whole grains is lacking, our findings are consistent with some intervention studies of nutrients. Dairy products are one of the important sources of protein, calcium, phosphorus, and vitamins A, D, and B2. Whole grains are not only rich in B vitamins, magnesium, and Fe but also contain polyphenols, vitamin E, carotenoids, and other common antioxidants. A crossover trial showed that B vitamin supplementation could avert the DNA methylation changes induced by fine particle exposure.⁴⁷ A study evaluated the profile of oxidative stress markers in the blood before and after supplementation with vitamins E and C for 6 months and found that antioxidant intervention was able to protect against the oxidative insult associated with PM derived from coal burning.⁴⁶ We also found that increasing vegetable intake may reduce the risk of HBP induced by

PM, although the difference was not statistically significant. High vegetable intake led to an improvement in micronutrient status (vitamins B, C, E, and carotenoids), which may play an important role in reducing the deleterious impact of air pollution on health.¹³ Our finding is consistent with the findings from a 12-week randomized clinical trial, which found that intervention with broccoli sprouts enhanced the detoxication of some airborne pollutants and may attenuate their associated long-term health risks.⁵⁰ A previous epidemiological study conducted in China (mainly in eastern and southern China) found that the associations of PM_{2.5} with hypertension were counteracted by high consumption of fruit other than vegetables. This discrepancy may be caused by the differences in the sample size, living habits, and local characteristics. In addition, our study used the quantity of food consumption, rather than just the frequency of consumption, and we adjusted for a sufficient number of food items to avoid synergies between various foods.

This study also observed that a higher consumption of fresh fruit and vegetables could mitigate the health effects of ambient PM on PP elevation. Multiple observational studies and controlled trials have shown that PP is an important risk factor for cardiovascular disease and the main pulsatile force leading to vascular aging after middle age. The European Society for Hypertension has recognized that broadening PP is a distinct risk factor that separates it from increased SBP in the elderly.⁵¹ Longer human lives have led to a global burden of late-life disease,⁵² and air pollution has caused a huge global public health and associated economic burden. How to mitigate the broadening PP caused by air pollution is a neglected direction but worth researching. The findings of our results have the potential to inform the important role of diet in preventing vascular aging from air pollution-related PP broadening. Additional epidemiological studies are needed to supplement corresponding evidence.

Additional Findings on BP and PM

The positive association between long-term exposure to ambient PM_{2.5} and hypertension has been well established. However, few studies have evaluated the hypertension risk of long-term exposure to PM₁, which is a major part of PM_{2.5} and may induce more extensive toxic effects than PM_{2.5}.⁵³ Our study simultaneously examined the potential health effects of 3 size-specific PM concentrations (PM₁, PM_{2.5}, and PM₁₀) on HBP and BP in highly polluted areas. We found that all 3 kinds of PM were associated with an increased risk of HBP, and the harmful effect may be elevated with smaller particle sizes. It has been reported that the size and surface area of particles are pivotal characteristics that determine the potential biological effects. Most toxic metals tend to accumulate in smaller particles, which have higher surface-to-volume ratios, so PM₁ may contain more toxins and be inhaled more deeply into the airways of the respiratory tract, eliciting stronger biological effects.^{54,55} We

also found associations between PM and SBP as well as PP, which were consistent with some, but not all, previous literature.^{7,18,56–58} The reason for heterogeneity among studies may be that BP can fluctuate because it is easily affected by many unmeasured factors, whereas the effect sizes of PM on the 3 BP parameters are relatively weak. What is more, the exploration of the exposure windows leading to raising BP is an important issue. It may not only hint at the underlying biological mechanism of PM on the cardiovascular system but also better assist policy makers in developing measures to achieve population protection. The associations between PM and BP in different exposure windows implied that PM concentrations within 1-year may be most closely associated with SBP and DBP, especially for PM₁ (Table S4), which is similar to the findings of a study on the associations of PM and BP from an adult cohort study in India.⁵⁹

Strengths of This Study

This study has some major advantages. First, the air pollution exposure concentrations have sufficiently large ranges of variation in Southwest China, which is beneficial for the construction of the exposure-response function. Second, the study participants were from the baseline survey of the CMEC study, which collected extensive individual-level information, such as detailed physical activity and indoor air pollution information. This helped us more fully adjust to confounding variables. Third, this study comprehensively revealed the modification effect of diet on the associations between air pollution and BP from 2 dimensions of dietary pattern and food items, which makes it easier to translate the findings into public health guidelines.

Limitations of This Study

However, several limitations also exist in this study. First, the cross-sectional design does not allow for a causal interpretation between PM exposure and HBP. To compensate for this deficiency, patients diagnosed with hypertension at baseline were excluded, and people who had lived at their current address for fewer than 3 years were also excluded. These measures may have alleviated the problem of cause-effect inversion to some extent. Second, the 3-year average satellite-based PM concentrations may not directly reflect actual PM exposure; thus, exposure misclassification may not be avoided. Third, questionnaires were used to collect demographic information and lifestyle characteristics, so there may have been recall bias. Fourth, other potential confounders, including ozone, NO₂, and traffic noise, were not evaluated in this analysis. Furthermore, there may be potentially unmeasured confounding factors associated with BP, such as genetic information. Considering this problem, we also included an index of family history of hypertension in the analysis, which may have served as a proxy for genetic information.

Perspectives

This study suggests that long-term exposure to ambient PM was significantly associated with an increased risk of HBP and increased BP. The DASH dietary pattern enriched in antioxidant foods and compounds can mitigate the associations between air pollution exposure and increased risk of HBP as well as increased BP. These results can provide epidemiological evidence about the potential role of dietary patterns in protecting public health from the adverse effects of air pollution. Our findings may also provide complementary approaches to protect health against the harmful effects of air pollution through individual-level prevention strategies of healthy dietary patterns.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Materials

Data Supplement Tables I–VI
Data Supplement Figures I–VIII

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