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Title page

Associations between PM_{2.5} and its chemical constituents and blood pressure: A cross-sectional study

Short title: PM_{2.5} constituents and blood pressure

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Abstract

Objectives: To investigate the associations between PM_{2.5} and its chemical constituents with blood pressure (BP), assess effects across BP quantiles, and identify the key constituent elevating BP.

Methods: A total of 36,792 adults were included in the cross-sectional study, representing 25 districts/counties of southeast China. Quantile regression models were applied to estimate the associations of PM_{2.5} and its chemical constituents (ammonium [NH₄⁺], nitrate [NO₃⁻], sulfate [SO₄²⁻], black carbon [BC], organic matter [OM]) with systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean artery pressure (MAP). A weighted quantile sum (WQS) index was used to estimate the relative importance of each PM_{2.5} chemical constituent to the joint effect on BP.

Results: The adverse effects of each interquartile range (IQR) increase in PM_{2.5}, NH₄⁺, NO₃⁻, SO₄²⁻, and BC on BP were found to be greater with elevated BP, especially when SBP exceeded 133 mmHg and DBP exceeded 82 mmHg. Each IQR increase in all five PM_{2.5} chemical constituents was associated with elevated SBP (β [95% CI]: 0.90 [0.75, 1.05]), DBP (β : 0.44 [0.34, 0.53]) and MAP (β : 0.57 [0.45,

68 0.69]), NH_4^+ (for SBP: weight=99.43%; for DBP: 12.78%; for MAP: 60.73%) and BC
69 (for DBP: 87.06%; for MAP: 39.07%) predominantly influencing these effects. The
70 joint effect of $\text{PM}_{2.5}$ chemical constituents on risks for elevated SBP and DBP
71 exhibited an upward trend from the 70th quantile (SBP exceeded 133 mmHg, DBP
72 exceeded 82 mmHg).

73

74 **Conclusion:** Long-term exposure to $\text{PM}_{2.5}$ and its chemical constituents was
75 associated with increased risk for elevated BP, with NH_4^+ and BC being the main
76 contributors, and such associations were significantly stronger at 70th to 90th quantiles
77 (SBP exceeded 133 mmHg, DBP exceeded 82 mmHg).

78

79 **Keywords:** hypertension; blood pressure; $\text{PM}_{2.5}$; chemical constituent; quantile
80 regression

1. Introduction

Hypertension affects 1.13 billion adults globally and is a major public health concern [1]. Elevated blood pressure (BP) is a primary contributor to cardiovascular diseases and increases the global disease burden, accounting for over 10 million deaths in 2019[2]. Hence, identifying modifiable risk factors for elevated BP can help prevent hypertension and even reverse the disease process at the population level. Fine particulate matter (PM_{2.5}), the most common air pollutant, has been associated with elevated BP in existing epidemiological studies[3,4].

PM_{2.5} is not a homogenous pollutant; rather, it is a complex mixture containing various chemical constituents[5], including secondary inorganic aerosols (i.e., ammonium [NH₄⁺], nitrate [NO₃⁻], sulfate [SO₄²⁻]), black carbon [BC], organic matter [OM], crustal elements, and water[6]. These PM_{2.5} chemical constituents display diverse physicochemical and toxicological characteristics, thereby potentially exerting different effect sizes on BP. For instance, per-interquartile range (IQR) increase in BC and SO₄²⁻ was associated with 17% and 11% higher odds of hypertension respectively[7]. However, previous studies have primarily focused on describing the effect of PM_{2.5} constituents on the presence or absence of hypertension, overlooking the impact of individual PM_{2.5} constituents on patients with varying baseline levels of BP. This is a crucial aspect because participants with higher BP may experience malfunctions in neurohumoral regulations[9], and were thereby more sensitive and susceptible to the effects of air pollution. Therefore, we hypothesize that the associations between PM_{2.5} constituents and BP levels are non-linear and are stronger at higher baseline BP values. This hypothesis is supported by previous studies[10,11], which have indicated non-linear health effects of systolic BP (SBP) and stronger associations between PM_{2.5} and systemic inflammatory markers at higher BP levels.

Notably, prior studies utilizing linear regression have primarily focused on the average effect of PM_{2.5} on BP, lacking insights into whether individuals with varying BP levels are equally affected[12]. Identifying these variations and potential thresholds where effects intensify is vital for pinpointing populations at increased risk and for crafting targeted interventions.

To address the aforementioned gaps, this study aimed to investigate how long-term exposure to PM_{2.5} and its chemical constituents affects BP both individually and in combination, referred to as the ‘joint effects’. Based on the baseline data of the Fujian Behavior and Disease Surveillance (FBDS) cohort, we applied quantile regression, a viable approach to estimate exposure-response relationships at different percentiles of the outcome distribution, and explored the independent and joint effects of PM_{2.5} constituents across BP quantiles[13]. Our findings would provide evidence for developing strategies to reduce the risk of hypertension by precisely mitigating air pollution.

2. Methods

2.1 Study population and data collection

The FBDS cohort, established between 2018 and 2020, aims to investigate health-related behaviors and chronic diseases among adults in Fujian Province, China. Utilizing baseline data of this cohort, collected by a three-stage cluster random sampling method detailed in previous studies[14-16], we initially selected 8 out of 9 prefecture-level cities in Fujian. Within each city, 3-6 districts/counties were chosen, followed by the selection of 4-6 neighborhoods/villages within each district/county,

depending on the size. Participants included those who: 1) were aged ≥ 18 years on the survey date; 2) had resided in the surveyed neighborhoods/villages for over six months within the past year; and 3) completed a face-to-face questionnaire interview, physical examination, and blood tests. We excluded participants with: 1) severe mental illnesses (e.g., schizophrenia and bipolar disorder); 2) significant physical illnesses (e.g., paraplegia or terminal illnesses); and 3) refusal to comply with FBDS protocols. From the original 54,961 participants, 9,318 who self-reported hypertension or were on blood pressure medications were excluded, avoid medicine effects on BP, and an additional 8,851 participants were excluded due to missing BP data. Consequently, 36,792 participants (66.94%) from 25 districts/counties in Fujian province were included in this cross-sectional study (**Figure S1**). The FBDS was approved by the Ethics Committee of the Fujian Provincial Center for Disease Control and Prevention (Number, 2018001, date June 27, 2018), and all participants provided informed consent.

2.2 Blood pressure

In this study, SBP, diastolic blood pressure (DBP), and mean artery pressure (MAP) were the health outcomes assessed. SBP and DBP were measured by trained medical personnel using an electronic sphygmomanometer (model: OMRON U10). Measurements were taken in a seated, upright position, three times, with a rest interval of 5 to 10 minutes between each reading. Participants were instructed to refrain from smoking, drinking, or exercising for at least 30 minutes before the measurement[17].

Hypertension was defined as SBP ≥ 130 mmHg or DBP ≥ 80 mmHg, in line with the 2017 ACC/AHA hypertension guideline recommendations[18]. To account for both cardiac output and systemic vascular resistance, we employed mean artery pressure (MAP), calculated as $(\text{SBP} + 2 \times \text{DBP}) / 3$ [19], and the final BP data included in the study was the average of all three measurements.

2.3 PM_{2.5} and its chemical constituents

Annual concentrations of PM_{2.5} and its chemical constituents (NH₄⁺, NO₃⁻, SO₄²⁻, BC, and OM) were obtained from the "Tracking Air Pollution in China" database (<http://tapdata.org.cn>). We derived these measurements at a spatial resolution of 10 km utilizing the Weather Research and Forecasting-Community Multiscale Air Quality modeling system, ground observations, a machine learning algorithm, and multisource-fusion PM_{2.5} data. We calculated the average concentrations of PM_{2.5} and each of its chemical constituents around the participants' residences over the past three years and used them as exposure variables. These constituents showed a strong correlation with the available observations, with daily correlation coefficients ranging from 0.67 to 0.80 from 2013 to 2020[20].

2.4 Covariates

Per prior studies[21,22], this research considered variables known to influence the relationships between air pollution and the risks for hypertension and BP as

covariates. These included sociodemographic characteristics, lifestyle factors, and body mass index (BMI). Sociodemographic characteristics included sex (male, female), age (years), ethnicity (Han, other minorities), marital status (unmarried, married, separated/divorced/widowed), educational level (illiteracy, elementary to high school, junior college or above), occupation (farmer/worker, others), and insurance type (non-commercial, commercial, no insurance). Lifestyle covariates consisted of smoking status (yes, no), alcohol consumption (yes, no), sleep quality (poor, fairly good, good), physical activity (< 2.5 hours/week, ≥ 2.5 hours/week), daily salt intake (grams/day), and Healthy Diet Score (HDS) and BMI (kg/m^2). The HDS, indicating adherence to five healthy dietary habits – daily consumption of fresh vegetables and fruits, eating red meat 1-6 days per week, consuming legumes ≥ 4 days per week, and eating fish ≥ 1 day per week – was scored from 0 to 5, with a higher score indicating a healthier diet[23]. BMI was calculated as weight in kilograms divided by the square of height in meters.

2.5 Statistical analysis

Continuous variables were presented as means and standard deviations (SD). Categorical variables were presented as frequencies and percentages. To compare differences between participants with and without hypertension, t-test or chi-square tests were used as appropriate. Pearson's correlation coefficients measured the correlations between $\text{PM}_{2.5}$ and its chemical constituents.

Quantile regression models were used to estimate the independent effects of

each IQR increase in PM_{2.5} and its chemical constituents (i.e., NH₄⁺, NO₃⁻, SO₄²⁻, BC, and OM) on the risks for elevated SBP, DBP, and MAP after adjusting for covariates, as BP had a non-normal distribution (**Figure S2, Table S1**). Compared to traditional linear regression, quantile regression is suitable for data with both normal and skewed distributions, allowing for the estimation of the effects of exposure variables on outcomes at various quantiles[24]. In the modeling framework, let Y be the outcome of interest (i.e., BP), and A be the exposure (i.e., PM_{2.5} and its chemical constituent). We modeled the τ quantile of Y conditional on $X = x_i$ using the quantile regression model given as

$$Q_{y_i|x_i}(\tau|X_i) = A^\tau \beta_{\tau a} + X_i^\tau \beta_\tau \quad (1)$$

where, $Q_{y_i|x_i}(\tau|X_i)$ is the conditional τ quantile outcome given X_i , and $\tau \in (0, 1)$ is the τ quantile of the outcome variable (BP). For example, $\tau=0.5$ means median BP regression. $X_i = (x_{i1}, x_{i2}, \dots, x_{ip})^\tau$ is the vector of covariates for each individual i , and $\beta_\tau = (\beta_{\tau 0}, \beta_{\tau 1}, \beta_{\tau 2}, \dots, \beta_{\tau p})^\tau$ is the vector of $(p + 1)$ regression coefficients which is calculated weighted least squares method at a known τ [25,26].

The weighted quantile sum (WQS) regression was used to investigate the joint effect and the relative contributions of multiple highly correlated exposures, effectively addressing collinearity and controlling for the confounding effect. In this study, we used β (95%CI) and weight to present the joint effect of each IQR increase in all five PM_{2.5} chemical constituents on BP and their independent relative importance using WQS regression[27,28]. Subsequently, the quantile regression model was used to assess the joint effect of each IQR increase in all five PM_{2.5}

chemical constituents on elevated SBP, DBP, and MAP across different quantiles (see details in the **supplementary material**).

To assess the robustness of our results, we performed the sensitivity analyses by:

- 1) excluding participants with a family history of hypertension due to potential genetic influences; 2) excluding participants who self-reported coronary heart disease, as medication taken to manage BP could render their measurements unreflective of natural levels; 3) using a 1-year exposure window instead of 3-years to assess the stability of the residential address.

All the analyses were conducted using R software (version 4.0.2), with statistical significance considered at $P < 0.05$ with two-sided tests.

3. Results

3.1 Characteristics of the study population

Among the 36,792 participants, the mean age was 53.16 ± 13.93 years, and 16,319 (44.35%) were males. The majority of the participants were Hans (98.23%), married (88.06%), and had non-commercial insurance (94.52%). A total of 18,314 participants were newly diagnosed with hypertension, resulting in a prevalence rate of 49.78%. Significant differences were observed between participants with hypertension and those with normal BP regarding age, sex, marital status, educational level, occupation, insurance type, smoking status, alcohol consumption, sleep quality, daily salt intake, HDS, and BMI, all with P values less than 0.05 (**Table 1**). Median values of SBP, DBP, and MAP were 124 mmHg, 77 mmHg, and 93 mmHg,

respectively (**Table S1**).

The median concentrations of PM_{2.5} and its chemical constituents NH₄⁺, NO₃⁻, SO₄²⁻, BC, and OM were 23.23 µg/m³, 3.24 µg/m³, 4.14 µg/m³, 4.83 µg/m³, 1.39 µg/m³, and 6.55 µg/m³, respectively (**Table S2**). The three-year average concentration of PM_{2.5} varied across the study area (**Figure S1**). High correlations were found among PM_{2.5} chemical constituents, with the Spearman correlation coefficients ranging from 0.49 to 0.97, and the highest correlation was observed between BC and OM ($r = 0.97$) (**Figure S3**).

3.2 Associations of PM_{2.5} and its chemical constituents with BP

We observed that the adverse effects of each IQR increase in PM_{2.5}, NH₄⁺, NO₃⁻, SO₄²⁻, and BC exposures increased with elevated quantiles of SBP, DBP, and MAP, while the adverse effect of OM exposure remained relatively stable. Specifically, we noted a substantial increase in the adverse effects of PM_{2.5}, NH₄⁺, NO₃⁻, SO₄²⁻, and BC exposures when SBP exceeded 133 mmHg and DBP exceeded 82 mmHg (i.e., from the 70th quantile to the 90th quantile). The most pronounced effects were observed for NH₄⁺ exposure on SBP (highest effect at the 90th percentile [β : 2.09, 95%CI: 1.61, 2.56]) and BC exposure on DBP (highest effect at the 90th percentile [β : 0.78, 95%CI: 0.51, 1.05]) (**Figures 1-3, Tables S3-S5**).

For the joint effect of PM_{2.5} chemical constituents, each IQR increase in all five PM_{2.5} chemical constituents was associated with elevated SBP (β [95% CI]: 0.90 [0.75, 1.05]), predominantly influenced by NH₄⁺ (weight: 99.43%). Similarly, the

joint effect of all PM_{2.5} chemical constituents was associated with risks for elevated DBP and MAP (β [95%CI] for DBP: 0.44 [0.34, 0.53]; β for MAP: 0.57 [0.45, 0.69]), primarily driven by BC (weight for DBP: weight=87.06%; for MAP: 39.07%) and NH₄⁺ (weight for DBP: 12.78%; for MAP: 60.73%) (**Figure 4**).

All PM_{2.5} chemical constituents exhibited associations with elevated SBP, DBP, and MAP across all quantiles, with effects increasing with higher SBP and MAP quantiles. Specifically, the joint effect on risks for elevated SBP and MAP increased from the 10th (β [95%CI] for SBP: 0.51 [0.32, 0.70]; β for MAP: 0.39 [0.25, 0.53]) to the 90th quantile (β for SBP: 1.54 [1.24, 1.83]; β for MAP: 0.84 [0.58, 1.10]), with the adverse joint effect of PM_{2.5} on DBP showing an upward trend from the 70th quantile (β : 0.46 [0.34, 0.58]) (**Figure 5, Table S6**).

3.3 Sensitivity analyses

The associations of PM_{2.5} and its chemical constituents (i.e., SO₄²⁻, NO₃⁻, NH₄⁺, OM, and BC) with the risks for elevated SBP, DBP, and MAP remained stable upon the exclusion of participants with a family history of hypertension (**Figures S4-S8, Tables S7-S10**), with NH₄⁺ and BC still emerging as the dominant contributors. Furthermore, robust results were observed when excluding participants with self-reported coronary heart disease or when shortening the PM_{2.5} and its chemical constituents exposure assessment from three years to one year (**Figures S9-S18, Tables S11-S18**).

4. Discussion

This study found that increased PM_{2.5} and its chemical constituents were associated with elevated SBP, DBP, and MAP. Notably, the impact of these pollutants intensified across higher BP quantiles. Specifically, exposure to NH₄⁺ emerged as the primary contributor to the joint effects of all PM_{2.5} chemical constituents on the risks for increased SBP. Additionally, exposure to BC and NH₄⁺ played a pivotal role in the risks for elevated DBP and MAP.

Our study found that the associations between PM_{2.5} and its chemical constituents and BP were significantly increased at the 70th to 90th quantile (SBP>133mmHg, DBP>82mmHg). Notably, the use of N95 facemasks[29,30] or air filters indoors[31] appeared to mitigate the adverse effects of air pollution on BP. For these populations, adopting more stringent protective measures, such as consistently wearing masks during poor air quality periods and utilizing air purifiers indoors, is advisable. The underlying mechanisms might involve inflammation and epigenetic changes in people with hypertension[32]. A panel study found that hypertension may exhibit enhanced associations between PM_{2.5} chemical constituents and systemic inflammatory markers, suggesting that PM-mediated toxicity would be stronger in individuals with hypertension[11]. Additionally, individuals with hypertension exhibit regulatory changes in DNA methylation[33], and the deleterious effects of air pollution appear more pronounced in those with elevated levels of DNA methylation[34].

We observed associations between PM_{2.5} and its chemical constituents with

305 elevated BP. Similar findings have been reported in prior studies conducted on
 306 different populations. A study in South Korea demonstrated that elevated
 307 concentrations of elemental BC and SO_4^{2-} in the $\text{PM}_{2.5}$ chemical constituents were
 308 associated with a higher risk for hypertension among the elderly population[35].
 309 Chinese college students showed that exposure to metal constituents of $\text{PM}_{2.5}$ was
 310 associated with elevated BP risk[36], and exposure to NH_4^+ was associated with
 311 elevated SBP risk[37]. The association between $\text{PM}_{2.5}$ and its chemical constituents
 312 and elevated BP risk could be explained by the oxidative stress that raises
 313 catecholamine levels or increases vascular resistance (especially for MAP)
 314 [38][39][40][19]. Furthermore, our findings indicated a more adverse effect on
 315 elevated SBP than DBP. This difference could be due to the activation of the renin-
 316 angiotensin system[41] and higher angiotensin II levels[42], which more significantly
 317 affect SBP than DBP. NH_4^+ , and BC were identified as the main chemical
 318 constituents affecting elevated BP. NH_4^+ , a water-soluble constituent in $\text{PM}_{2.5}$,
 319 contributed to early oxidative stress by promoting reactive oxygen species (ROS)
 320 formation, mitochondrial multiplication, and multilayer formation[43]. Besides, NH_4^+
 321 might act on lipid metabolism, indirectly affecting BP, and played a role in the
 322 aggregation of high-density lipoprotein apoA-I and degradation of low-density
 323 lipoprotein apo-B[44]. BC primarily originates from incomplete burning of fossil
 324 fuels or biomass[45]. A previous epidemiological study showed that every unit
 325 increase in BC exposure increased SBP and DBP by 0.53 (95%CI: 0.17, 0.89) mmHg
 326 and 0.37 (95%CI: 0.10, 0.65) mmHg, respectively[46]. BC might activate pulmonary

macrophages or down-regulating DNA methylation, inducing systemic inflammation and oxidative stress, resulting in cardiovascular system dysfunction and abnormal BP control[47,48], thus leading to adverse cardiovascular outcomes. Furthermore, BC had high aggregation and adsorption and could be used as a carrier of the water-soluble constituents NH_4^+ , continuously inducing an inflammatory response and causing cell damage[49], potentially explaining the elevated BP caused by NH_4^+ and BC as the main chemical constituents.

Our study has limitations. First, its cross-sectional design prevents establishing causality, despite exposure occurring before the outcomes. Second, some other possible confounding factors, including individuals' past medical or medication history (e.g. history of renal failure or using steroids), were not considered in this study due to unavailable data, which may affect the associations between $\text{PM}_{2.5}$ exposure and its chemical constituents and BP. However, the results remained robust when we excluded participants with a family history of hypertension, which may partly represent the heredity of hypertension in the sensitivity analysis. Third, the spatial resolution of $\text{PM}_{2.5}$ chemical constituents was 10 km, which is insufficient to reveal fine-scale variations in the concentrations of $\text{PM}_{2.5}$ chemical constituents, although it was the finest resolution exposure dataset available in China. Further studies using wearable devices may collect more precise exposure data to $\text{PM}_{2.5}$ constituents. Finally, findings from one Chinese province may not apply elsewhere. Nevertheless, this study had a strength. We used the quantile regression approach, which does not assume a normal distribution for outcomes, unlike traditional models.

This approach is more suitable for the natural distribution of BP and aids in accurately identifying susceptible populations.

In conclusion, Long-term exposure to $\text{PM}_{2.5}$ and its chemical constituents (NH_4^+ , NO_3^- , SO_4^{2-} , OM, and BC) was associated with elevated BP, and the associations were significantly increased at 70th to 90th quantiles, with NH_4^+ and BC as the main constituents affecting the risk for elevated BP. Our study calls for additional research into the association between $\text{PM}_{2.5}$ and its chemical constituents and elevated BP. Our findings provide evidence to accurately identify sensitive populations. Furthermore, this evidence is essential for developing targeted strategies to reduce emissions of specific harmful constituents, such as NH_4^+ and BC.

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Conflicts of interest

There are no conflicts of interest.

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Table 1. Characteristics for the study population

Variables	Number (%) or Mean $\pm$ SD			<i>P</i>
	All (<i>n</i> =36,792)	Hypertension		
		Without (<i>n</i> =18,478)	With (<i>n</i> =18,314)	
Sex				< 0.001
Male	16,319 (44.35)	7,291 (39.46)	9,028 (49.30)	
Female	20,473 (55.65)	11,187 (60.54)	9,286 (50.70)	
Age, years	53.16 (13.93)	49.97 (13.86)	56.38 (13.25)	< 0.001
Marital status				< 0.001
Unmarried	1,604 (4.36)	984 (5.33)	620 (3.39)	
Married	32,399 (88.06)	16,401 (88.76)	15,998 (87.35)	
Separated/divorced/widowed	2,789 (7.58)	1,093 (5.92)	1,696 (9.26)	
Ethnicity				
Han	36,139 (98.23)	18,154 (98.25)	17,985 (98.20)	0.785
minority	653 (1.77)	324 (1.75)	329 (1.80)	
Educational level				< 0.001
Illiteracy	13,967 (37.96)	6,092 (32.97)	7,875 (43.00)	
Elementary to high school	20,070 (54.55)	10,515 (56.91)	9,555 (52.17)	
Junior college or above	2,755 (7.49)	18,71 (10.13)	884 (4.83)	
Occupation				< 0.001
Farmers/workers	13,048 (35.46)	6,173 (33.41)	6,875 (37.54)	
Others	23,744 (64.54)	12,305 (66.59)	11,439 (62.46)	
Insurance type				< 0.001
Commercial	411 (1.12)	210 (1.14)	201 (1.10)	
Non-commercial	34,777 (94.52)	1,7319 (93.73)	1,7458 (95.33)	
No insurance	1,604 (4.36)	949 (5.14)	655 (3.58)	
Smoking status				< 0.001
No	26,567 (72.21)	13,962 (75.56)	12,605 (68.83)	
Yes	10,225 (27.79)	4,516 (24.44)	5,709 (31.17)	
Alcohol consumption				< 0.001
No	24,182 (65.73)	12,526 (67.79)	11,656 (63.65)	
Yes	12,610 (34.27)	5,952 (32.21)	6,658 (36.35)	
Sleep quality				< 0.001
Poor	4,979 (13.53)	2,376 (12.86)	2,603 (14.21)	
Fairly good	1,2729 (34.60)	6,311 (34.15)	6,418 (35.04)	
Good	19,084 (51.87)	9,791 (52.99)	9,293 (50.74)	
PA, hours/week				0.066
< 2.5	23,539 (63.98)	11,769 (63.69)	11,770 (64.27)	
$\geq$ 2.5	13,253 (36.02)	6,709 (36.31)	6,544 (35.73)	
Daily salt, g/day	5.87 (3.99)	5.80 (3.84)	5.94 (4.13)	< 0.001
HDS	2.44 (0.90)	2.51 (0.90)	2.36 (0.90)	< 0.001
BMI, kg/m²	23.53 (4.05)	22.91 (4.13)	24.15 (3.86)	< 0.001

BMI: body mass index; HDS: Health Diet Score; PA: physical activity

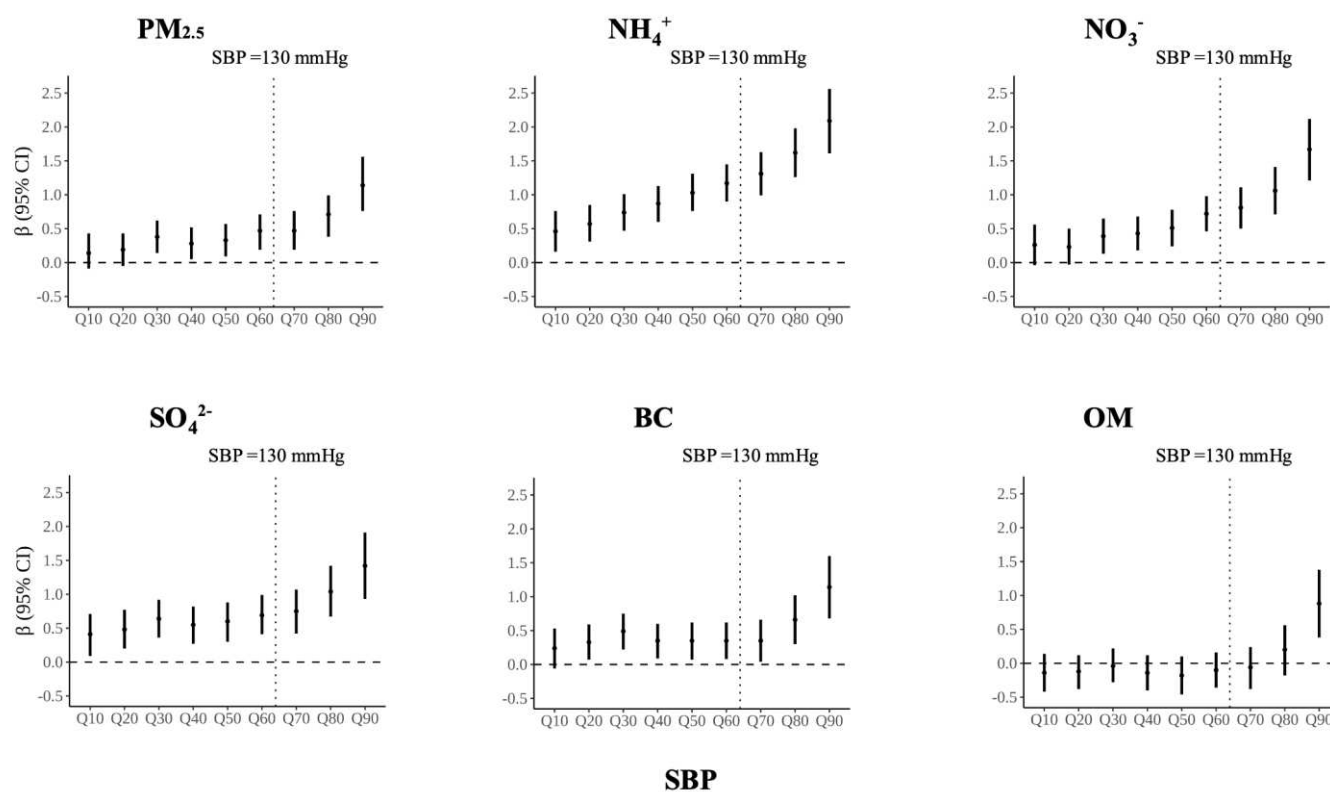


Figure 1. Associations between each IQR increase in concentrations of PM_{2.5} and its chemical constituents and SBP in quantiles

Covariates adjusted for age, sex, ethnicity, marital status, education level, occupation, insurance type, smoking status, alcohol consumption, sleep quality, physical activity, daily salt intake, Health Diet Score and BMI.

BMI: body mass index; IQR: interquartile range; SBP: systolic blood pressure; PM_{2.5}: particle with aerodynamic diameter $\leq 2.5 \mu\text{m}$; NH₄⁺: ammonium; NO₃⁻: nitrate; SO₄²⁻: sulfate; BC: black carbon; OM: organic matter.

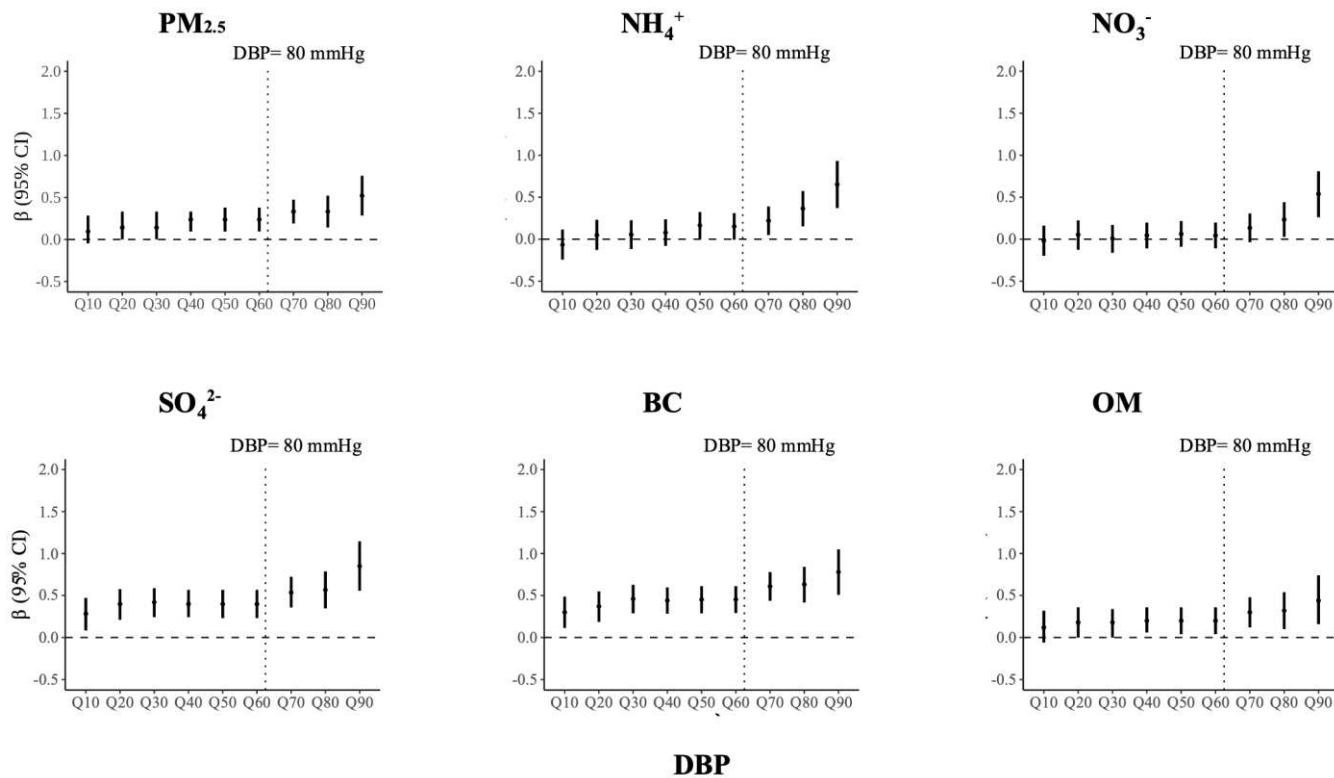


Figure 2. Associations between each IQR increase in concentrations of $\text{PM}_{2.5}$ and its chemical constituents and DBP in quantiles

Covariates adjusted for age, sex, ethnicity, marital status, education level, occupation, insurance type, smoking status, alcohol consumption, sleep quality, physical activity, daily salt intake, Health Diet Score and BMI.

BMI: body mass index; DBP: diastolic blood pressure; IQR: interquartile range; $\text{PM}_{2.5}$: particle with aerodynamic diameter $\leq 2.5 \mu\text{m}$; NH_4^+ : ammonium; NO_3^- : nitrate; SO_4^{2-} : sulfate; BC: black carbon; OM: organic matter.

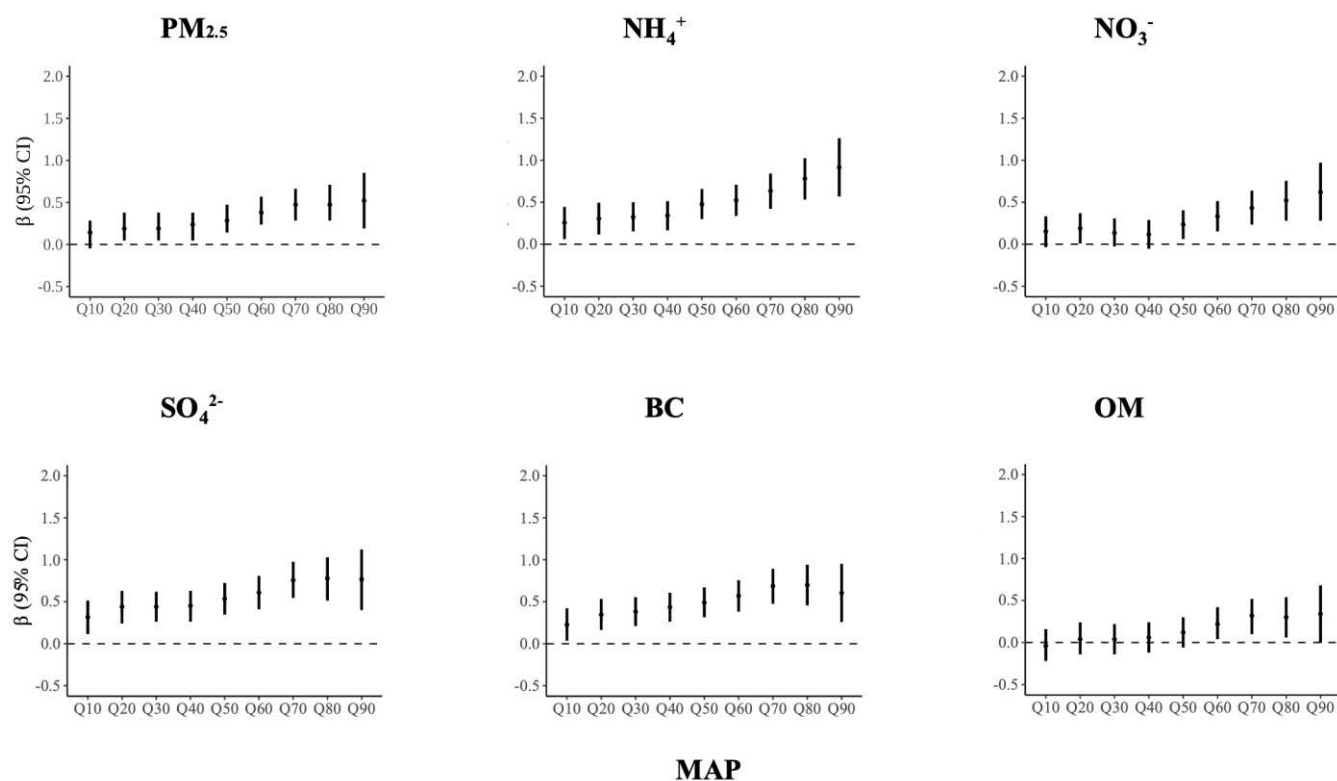


Figure 3. Associations between each IQR increase in concentrations of PM_{2.5} and its chemical constituents and MBP in quantiles

Covariates adjusted for age, sex, ethnicity, marital status, education level, occupation, insurance type, smoking status, alcohol consumption, sleep quality, physical activity, daily salt intake, Health Diet Score and BMI.

BMI: body mass index; IQR: interquartile range; MAP: mean artery pressure; PM_{2.5}: particle with aerodynamic diameter $\leq 2.5 \mu\text{m}$; NH₄⁺: ammonium; NO₃⁻: nitrate; SO₄²⁻: sulfate; BC: black carbon; OM: organic matter.

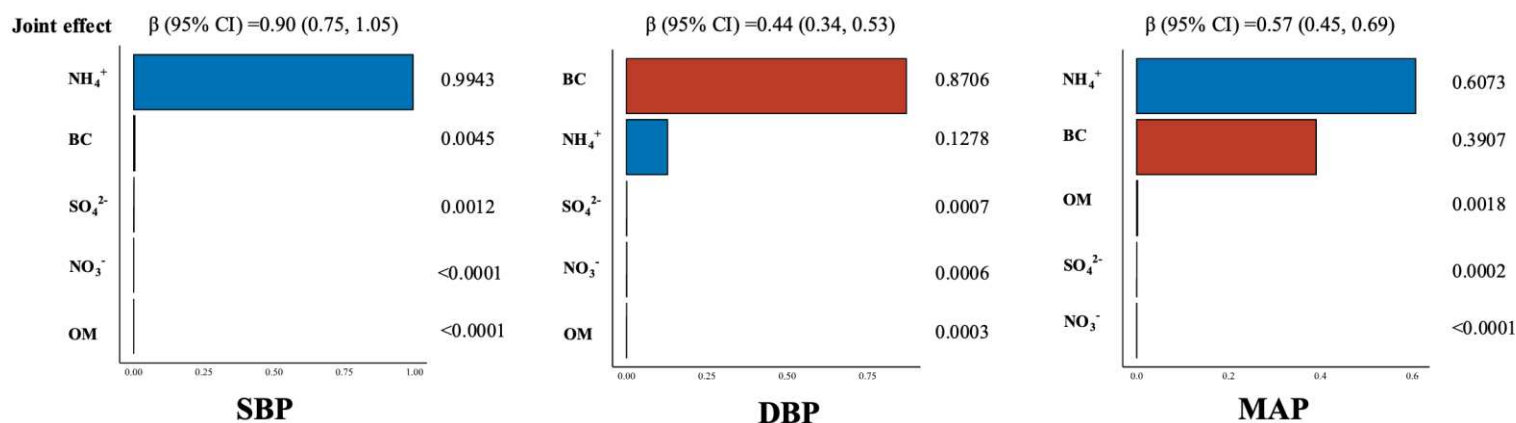


Figure 4. Joint effect and the relative importance of five $\text{PM}_{2.5}$ chemical constituents on the risk for elevated blood pressure

The blue bar represented weight of NH_4^+ , the red bar represented weight of BC.

Covariates adjusted for age, sex, ethnicity, marital status, education level, occupation, insurance type, smoking status, alcohol consumption, sleep quality, physical activity, daily salt intake, Health Diet Score and BMI.

BMI: body mass index; DBP: diastolic blood pressure; MAP: mean artery pressure; SBP:

systolic blood pressure; $\text{PM}_{2.5}$: particle with aerodynamic diameter $\leq 2.5 \mu\text{m}$; NH_4^+ :

ammonium; NO_3^- : nitrate; SO_4^{2-} : sulfate; BC: black carbon; OM: organic matter.

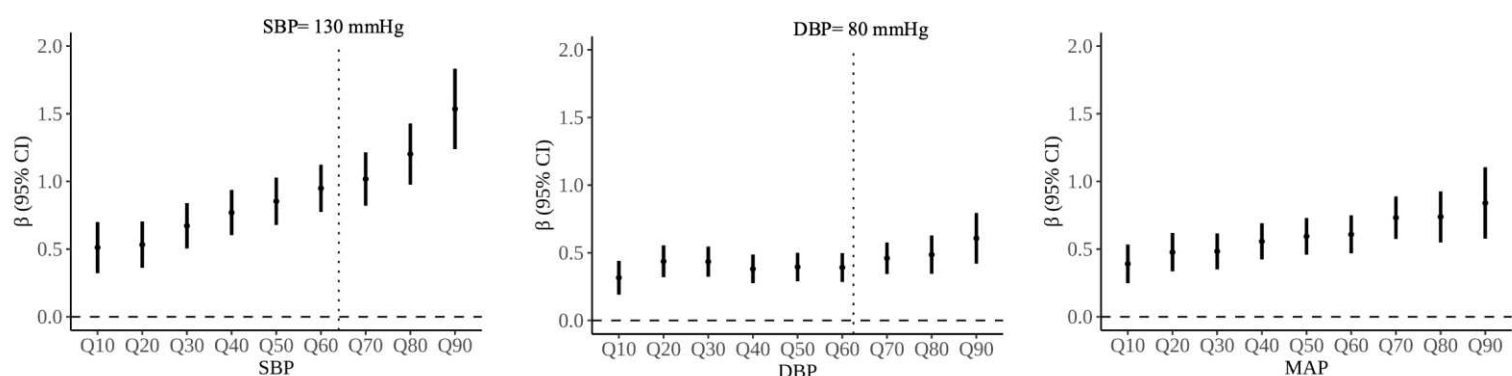


Figure 5. Associations between each IQR increase in concentrations of all PM_{2.5} chemical

constituents and blood pressure in quantiles

Covariates adjusted for age, sex, ethnicity, marital status, education level, occupation, insurance

type, smoking status, alcohol consumption, sleep quality, physical activity, daily salt intake,

Health Diet Score and BMI.

BMI: body mass index; DBP: diastolic blood pressure; IQR: interquartile range; MAP: mean

artery pressure; SBP: systolic blood pressure; PM_{2.5}: particle with aerodynamic diameter ≤ 2.5

μm ; NH_4^+ : ammonium; NO_3^- : nitrate; SO_4^{2-} : sulfate; BC: black carbon; OM: organic matter.