

# Associations of long-term exposure to ambient air pollution with blood pressure and hypertension defined by Chinese and ACC/AHA Guideline: The CHCN-BTH cohort study

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**Research**

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# Abstract

**Background** The association between long-term air pollutants exposure with blood pressure and hypertension defined by 2017 American College of Cardiology (ACC)/American Heart Association (AHA) Hypertension Guideline is still conflicting. This study was designed to investigate the associations between long-term exposure to air pollutants, blood pressure and hypertension defined by Chinese and ACC/AHA guideline in Chinese adults.

**Methods:** Our study was based on the baseline survey of the Cohort Study on Chronic Disease of Communities Natural Population in Beijing, Tianjin and Hebei (CHCN-BTH) from 2017 to 2019. A spatial statistical model was used to assess three-year (2014-2016) average pollutant concentrations for  $PM_{2.5}$ , and other pollutants concentration ( $PM_{10}$ ,  $SO_2$ ,  $NO_2$ ) was assessed by data of air monitoring stations.

**Results:** A total of 32,135 adults aged 18-80 years were included, each interquartile range (IQR) increment of  $PM_{2.5}$ ,  $PM_{10}$ ,  $SO_2$  and  $NO_2$  was associated with increases of 0.66mmHg (95%CI: 0.29, 1.03), 0.40mmHg (0.00, 0.81), 1.38mmHg (0.92, 1.84) and 0.54mmHg (0.16, 0.91) in SBP, respectively.  $SO_2$  was associated with increases 0.42mmHg (0.11, 0.72) in DBP.  $PM_{2.5}$ ,  $PM_{10}$ ,  $SO_2$  and  $NO_2$  was associated with an 14% (Odds ratio [OR]:1.14, 95%CI:1.06-1.23), 6% (1.06, 1.00-1.13), 9% (1.09, 1.02-1.17) and 8% (1.08, 1.01-1.16) increase of hypertension defined by Chinese guideline.  $PM_{2.5}$  and  $SO_2$  was associated with 16% (1.166, 1.093-1.245) and 10% (1.10, 1.03-1.18) increase of hypertension defined by ACC/AHA guideline. Two-pollutants model, traffic-related pollution model and stratified analysis yielded similar results.

**Conclusions:** We found that long-term exposure to  $PM_{2.5}$ ,  $NO_2$  and  $SO_2$  is associated with increase of blood pressure and hypertension defined by both Chinese and ACC/AHA guideline.  $PM_{10}$  is associated with higher SBP and risk of hypertension defined by Chinese guideline.

## 1. Background

Hypertension is a main risk factor for cardiovascular disease that contributed to 17.7 million deaths in the world in 2017[1, 2]. Particularly in China, approximately 27.9% of adults have hypertension, and higher prevalence of hypertension was found in large and medium-sized cities[3].

In recent years, mounting epidemiological studies have shown that exposure to air pollution was related to higher blood pressure and risk of hypertension[4–7], a recent meta-analysis reviewed the existing researches and reported globally significant associations of long-term exposure to  $PM_{2.5}$  with hypertension and  $PM_{2.5}/NO_2$  with DBP[8]. A cross-sectional study in China found  $PM_{2.5}$  and  $PM_{10}$  concentration was significantly associated with increases of SBP[9]. Another cross-sectional study in India found positive association between  $PM_{2.5}$  and SBP only in men[10]. However, the result is inconsistent, a Korean study reported no significant association between air pollution and SBP[11], a few studies even found a negative association between  $NO_2$  or  $O_3$  and blood pressure[12, 13].

In addition, compared with SBP and DBP, much less evidence has been available for air pollution and mean arterial pressure (MAP) as well as pulse pressure (PP). Moreover, rapid economic growth and related emissions have led to increases in air pollution of Beijing-Tianjin-Hebei (BTH) metropolitan region in China recent years[14]. Many previous studies conducted in North America or Europe, where the average PM<sub>2.5</sub> levels were much lower than those in BTH region.

The American College of Cardiology (ACC)/American Heart Association (AHA) Task Force on Clinical Practice Guidelines recently released the 2017 hypertension guideline. One new feature of this guideline is defining a systolic blood pressure(SBP) of 130 to 139 mmHg or diastolic blood pressure (DBP) of 80 to 89 mmHg as stage 1 hypertension[15], which has been evolving from an SBP/DBP of 140/90 mmHg or higher in 1997[16], and a further decrease to 130/80 mm Hg in 2017. However, as we known, there is no evidence to prove the association of air pollution and hypertension which defined by AHA/ACC guideline.

On the other hand, the types of ambient air pollutants are complex, the evidence of association between multi-air pollutants and blood pressure or hypertension was still lack. A retrospective cross-sectional study in Japan showed multi-pollutants, which included suspended particulate matter, SO<sub>2</sub>, NO<sub>2</sub> and photochemical oxidants, could increase blood pressure and the risk of hypertension[17]. Another study in Pakistan also found multi-pollutants were associated with high risk of CVD[18]. We are not found study of multi-pollutants in BTH region, and our study can fill this gap and provide strong evidence of the association between multi-pollutants and blood pressure in BTH region. The aim of the present study was to investigate the association between long-term and high-level exposure of ambient air pollution with hypertension and BP among participants from the baseline survey of the Cohort Study on Chronic Disease of Communities Natural Population in Beijing, Tianjin and Hebei (CHCN-BTH). We further investigated and compared the association of long-term exposure to ambient air pollution and incident of hypertension which were defined by both Chinese guidelines and AHA/ACC guideline. Moreover, proximity to roadways is considered as a substitute for long-term exposure to traffic-related air pollution, which can better capture exposure to both particle matter and gaseous mixtures. We also investigated several potential effect modifications by demographic characteristics and health behaviors.

## 2. Methods

### 2.1 Study population

Our study was based on the CoHort Study on CHronic Disease of Communities Natural Population in Beijing, Tianjin and Hebei (CHCN-BTH, Registration number: ChiCTR1900024725), details of the cohort has been introduced elsewhere[19]. Generally, a multistage stratified cluster sampling method was used to select participants from 41 communities, 36 townships and 7 functional units in five cities (i.e., Beijing, Tianjin, and Chengde, Baoding and Shijiazhuang of Hebei Province) from 2017 to 2019. The survey sites covered the central core functional area, the southern expansion area and the northwestern ecological conservation area. We included adults aged 18 to 80, and excluded people had psychiatric disorder or disabled and pregnant women. All participants lived in survey site for more than 3 years. At baseline,

participants underwent some physical examinations (e.g., height, weight and blood pressure measurements), questionnaire survey and blood tests. All the participants signed a consent form. This study was reviewed and approved by the ethical committees of Center of Disease Control and Capital Medical University.

## 2.2 Air pollution exposure assessment

A machine learning method was used for estimating the annual concentrations of particles with an aerodynamic diameter of  $\leq 2.5\mu\text{m}$  ( $\text{PM}_{2.5}$ ) from 2014 to 2016 (three years prior to baseline survey) at a resolution of  $10\text{km}\times 10\text{km}$ , using high dimensional expansion of numerous predictors (including ground monitored  $\text{PM}_{2.5}$  data, satellite-derived aerosol optical depth (AOD) and other satellite covariates, meteorological variables and chemical transport model simulations)[20]. The estimates were found to be in good agreement with in-situ observations, with determination coefficients ( $R^2$ ) of 0.77.  $\text{PM}_{2.5}$  concentrations was estimated according to geocode residential address of each participant. Then, we calculated the three-year average concentration before the baseline survey (2017) for each participant as the long-term exposure concentration of  $\text{PM}_{2.5}$ .

Air pollutant concentration data is from China National Environmental Monitoring Centre ([www.cnemc.cn](http://www.cnemc.cn)). We collected air pollution concentration data in 103 monitoring stations of Beijing, Tianjin and Hebei province, included  $\text{PM}_{10}$ ,  $\text{SO}_2$  and  $\text{NO}_2$ . The air pollution data is from January 1st 2014 to December 31st 2016. For the calculation of 24-hour mean concentrations of  $\text{PM}_{10}$ ,  $\text{SO}_2$  and  $\text{NO}_2$ , at least 75% of the one-hour values must be available on that particular day. If a station had more than 25% of the values missing for the whole period of analysis, the entire station was excluded from the analysis.

We collected residential address of every participant, and ArcGis5.0 was used to calculate ambient air pollution exposure by the nearest monitoring station data. Considering the cumulative effect of air pollution exposure, we defined 2016 ambient air pollution exposure as lag0, 2014 and 2015 ambient air pollution exposure as lag1 and lag2, and we calculated 2 and 3-years average concentration [ $\text{lag01}=(\text{lag0} + \text{lag1})/2$ ], [ $\text{lag02} = \text{lag01}/2+(\text{lag1} + \text{lag2})/2$ ], respectively.

## 2.3 Proximity to major roadway

We calculated residential distance to major roadway based on residential address for neighborhood analysis, which could indirectly represent the traffic-related pollution. The major roadways, were classified as national, provincial and county roadways according to the National Highway Classification in China. We examined the proximity to roadways in categories of proximity  $< 100\text{m}$ ,  $100$  to  $< 200\text{m}$ ,  $200$  to  $< 400\text{m}$ , and  $400$  to  $\leq 1000\text{m}$ . Residential addresses further than  $1000$  m from a major roadway are not likely an indicator of traffic-related exposure[21]. Therefore, we excluded these addresses ( $> 1000$  m, 8632 participants), which left 23,503 (73.1%) participants for the association analyses of proximity to a major roadway and blood pressure or hypertension.

## 2.4 Outcome assessment

Blood pressure was measured using an electronic sphygmomanometer (OMRON HEM-907, Japan) in the sitting position for three times and the cuff was placed around 2cm above the right arm elbow. Participants were advised not to take hypotensive drugs, smoke, drink alcohol, coffee, or tea, and to abstain from exercising on the test day. Additionally, they were not allowed to talk during the measurement. The average value of three measurements was used as the blood pressure measurement for this study. MAP was calculated as  $DBP + 1/3 (SBP - DBP)$ ; PP was calculated as the difference between SBP and DBP. According to the 2018 Chinese guidelines for the management of hypertension[22], hypertension in our analysis was defined as having a measured SBP  $\geq 140$  mm Hg or DBP  $\geq 90$  mm Hg, and/or having a self-reported physician-diagnosed hypertension or anti-hypertension treatment. We also defined prevalent hypertension as having the measured SBP  $\geq 130$  mm Hg or DBP  $\geq 80$  mm Hg according to the AHA/ACC Hypertension Practice Guidelines 2017[23].

## 2.5 Covariates

We controlled for potential confounders based on the previous literature on air pollution and blood pressure. Demographic covariates included age (years, as a continuous covariate), ethnicity (Han/other), sex (male/female), and BMI (as a continuous covariate). Socioeconomic covariates included education level (no school/primary school/middle school/junior college or higher), marital status (“married/cohabitating” or “widowed/single/divorced/separation”), residential area (“rural” or “urban”) and average monthly income (“ $\leq 500$  RMB”, “500–1000 RMB” or “ $\geq 1000$  RMB”). Health behavior covariates included smoking status (“never smoking” or “ever smoking”), alcohol drinking status (“never drinking” or “ever drinking”), exercise (regular/seldom), family history of hypertension (yes/no), type 2 diabetes (yes/no) and antihypertensive treatment (yes/no). The other continuous covariates included population density and per-capita gross domestic product (GDP), which was obtained from the Beijing, Tianjin, Chengde, Baoding and Shijiazhuang Statistical Yearbooks (<https://www.yearbookchina.com/>).

## 2.6 Statistical analysis

A generalized linear mixed models (GLMMs) was used to estimate the associations between 3-year air pollutants exposure and the blood pressure (SBP, DBP, MAP, PP), covariates including age, sex, BMI, ethnicity, education, marital status, family history of hypertension, self-reported smoking and drinking, physical activity, monthly income, residential area and ambient temperature were included into the models to modify the confounding bias. When air pollutants were considered as a continuous variable, regression coefficient ( $\beta$ ) and 95% confidence interval (CI) of BP was calculated by each IQR increment in the 3-year average air pollutants concentration. When the participants were categorized into 4 groups based on the quartiles of pollutants exposure,  $\beta$  value and 95%CI of BP was calculated using the first quartile as a reference.

A two-level logistic regression model to assess the associations between 3-year air pollutants exposure and hypertension defined by both Chinese and ACC/AHA guideline, where participants were considered as the first-level unit and the community as the second-level unit in the model[24]. Odds ratios (ORs) and 95%CI of incident hypertension was reported for each IQR increment in the 3-year average air pollutants concentration. We also estimated the associations of proximity to major roadway with blood pressure and

hypertension to representing the traffic-related air pollution. Spearman rank correlation coefficients were performed to assess the relationship between air pollutants, meteorological data and proximity to a major roadway.

We examined whether the association between air pollutants exposure and blood pressure or hypertension was modified by age (< 65y and  $\geq$  65y), gender (male and female), residence (rural and urban), smoking (never and ever smoker), alcohol drinking (never and ever drinking), physical exercise (usual and seldom), then reported the *p* values for interaction terms.

We also performed several sensitivity analyses. First, we used average air pollutants concentrations for 1–3 years before the survey. Second, participants with no information of hypertensive medication were excluded. Third, we excluded the participants with cardiovascular diseases. Statistical analyses were conducted using SAS version 9.4 (SAS Institute, Inc., Cary, NC). A two-tailed *P* value of < 0.05 was defined as statistically significant.

## 3. Results

### 3.1 Descriptive statistics

A total of 33,391 participants were measured BP, and 1,256 participants were excluded due to missing information on important potential confounders and hypertension history. The basic characteristics of the 32,135 study subjects are summarized in Table 1. Participants were on average 50.0 years and 55.4% of them were women. Most participants were Han, 72.1% of them live in urban, 30.7% of them have been a smoker and 42.7% of them used to drinking. There are 40.8% and 58.5% (Table A1) of the participants diagnosed as hypertension according to Chinese and AHA Hypertension Guideline, respectively. There were significant differences in all variables between hypertension defined by Chinese or AHA/ACC guideline and non-hypertension group.

Table 2 shows the 3-year average concentrations of six air pollutants exposure for the 32,135 participants. The average concentration of SO<sub>2</sub> was 22.9 $\mu$ g/m<sup>3</sup>, which were lower than the National Ambient Air Quality Standards of China (60 $\mu$ g/m<sup>3</sup>), however, the 3-year average concentration of PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub> were 73.5 $\mu$ g/m<sup>3</sup>, 123.0 $\mu$ g/m<sup>3</sup> and 52.2 $\mu$ g/m<sup>3</sup>, respectively, which exceeded the National Ambient Air Quality Standards of China (35 $\mu$ g/m<sup>3</sup>, 70 $\mu$ g/m<sup>3</sup> and 40 $\mu$ g/m<sup>3</sup>). The temperature of testing day was 14.7 $\pm$ 12.2 $^{\circ}$ C and the humidity was 55.5 $\pm$ 21.5%. The correlations of all air pollutants were moderate with each other. The mean of proximity to a major roadway was 408.5 $\pm$ 282.3m was negatively correlated with all air pollutants.

### 3.2 Associations of air pollutants with blood pressure and hypertension

The associations of long-term exposures to air pollutants with blood pressure are presented in **Table 3**. Totally, the IQR increment in PM<sub>2.5</sub>, PM<sub>10</sub>, SO<sub>2</sub> and NO<sub>2</sub> were significantly associated with an elevation of 0.66mmHg (95%CI: 0.29, 1.03), 0.40mmHg (95% CI: 0.00, 0.81), 1.38mmHg (95% CI: 0.92, 1.84),

0.54mmHg (95%CI: 0.16, 0.91) in SBP, and PM<sub>2.5</sub>, SO<sub>2</sub> and NO<sub>2</sub> can increase PP by 0.42mmHg (95% CI: 0.09, 0.74), 1.07mmHg (95%CI: 0.71, 1.42), 0.49mmHg (95%CI: 0.20, 0.79), respectively (Table 3). Furthermore, PM<sub>2.5</sub> and SO<sub>2</sub> were associated with increases of 0.37 mmHg (0.09, 0.65) and 0.65 mmHg (95%CI:0.33, 0.98) in MAP, SO<sub>2</sub> were associated with increases of 0.42mmHg (95%CI: 0.11, 0.72) in DBP.

Participants with PM<sub>2.5</sub> and SO<sub>2</sub> values in the fourth quartiles had much higher SBP, DBP, MAP, PP than those in the lowest quartile, and the air pollutants values in the second, third, and fourth quartiles had increased BP levels which compared with the lowest quartile (*P* for trend<0.01, Table 3). We found similar results between 3-y average NO<sub>2</sub> and SBP or PP. No significant association was found between PM<sub>10</sub> and any kinds of blood pressure.

The associations for air pollutants and Chinese/AHA hypertension guideline are shown in Table 4.

For Chinese hypertension guideline, compared with the lowest quintile (Q1) of PM<sub>2.5</sub>, SO<sub>2</sub> and NO<sub>2</sub>, the risk of hypertension with the highest quintile (Q4) was increased by 57% (OR=1.57, 95%CI: 1.36, 1.8), 33% (OR=1.33,95CI%: 1.15, 1.53) and 13% (OR=1.13, 95CI%: 1.02, 1.26), respectively. When air pollutants were treated as continuous variables, all pollutants can significantly increase hypertension. For every interquartile range (IQR) increase in PM<sub>2.5</sub>, PM<sub>10</sub>, SO<sub>2</sub> and NO<sub>2</sub> exposure, the risk of hypertension increased by 14% (OR=1.14, 95%CI: 1.06, 1.23), 6% (1.06, 1.00-1.27), 9% (1.09, 1.02-1.17) and 8% (1.08, 1.00-1.16), respectively. The similar results were found in PM<sub>2.5</sub> and SO<sub>2</sub> with hypertension defined by AHA/ACC guideline, compared with the lowest quintile of PM<sub>2.5</sub> and SO<sub>2</sub>, the risk of hypertension with the highest quintile was increased by 77% (OR=1.77, 95%CI: 1.54, 2.03) and 92% (OR=1.93, 95CI%: 1.21, 2.64), respectively. For every interquartile range (IQR) increase in PM<sub>2.5</sub> and SO<sub>2</sub> exposure, the risk of hypertension defined by AHA/ACC guideline increased by 16% (OR=1.17, 95%CI: 1.09, 1.25) and 10% (OR=1.10, 95%CI: 1.03, 1.18). However, no significantly associations were found between PM<sub>10</sub> and NO<sub>2</sub> exposure with the risk of AHA/ACC hypertension.

In two-pollutants model of air pollutants and blood pressure (Table A5), we found similar results as the single-pollutant model. The combined effects of SO<sub>2</sub> with other pollutants can increase blood pressure (SBP, DBP, MAP and PP), PM<sub>2.5</sub> combined with other pollutants can increase blood pressure except DBP, PM<sub>10</sub> combined with SO<sub>2</sub> can increase SBP and PP, NO<sub>2</sub> combined with other pollutants can increase SBP. In two-pollutants model of air pollutants and hypertension (Table A6), we found PM<sub>2.5</sub> combined with other pollutants and PM<sub>10</sub> combined with SO<sub>2</sub> can increase hypertension defined by both Chinese and AHA/ACC guideline. SO<sub>2</sub> combined with PM<sub>10</sub> can only increase hypertension defined by AHA/ACC guideline. The combined effect of NO<sub>2</sub> and other pollutants showed non-significant results.

### 3.3 Associations of proximity to roadway with blood pressure and hypertension

Participants whose residential address was  $\geq 1000$  m to a roadway were excluded in this association analyses, and 23,503 (73.1%) participants were remained. The main characteristics of this study participants were similar to Tabe1 (Supplementary materials Table A2). The result for proximity to

roadway and blood pressure are shown in **Figure 1**. Participants who live within 100m from major roadway are associated with significantly higher SBP and PP, and the effect decreased with the distance of proximity to roadway ( $P$  for trend=0.004 and 0.01, respectively). Participants who live within 100m from major roadway are associated with an increasing risk of Chinese/AHA hypertension and the OR decreased with the distance of proximity to roadway ( $P$  for trend=0.03 and 0.02).

### 3.4 Stratified analyses for the associations of air pollutants with blood pressure and hypertension

The results of the stratified analysis of air pollution exposure and blood pressure were shown in **Figure 2**. The associations between  $PM_{2.5}$ ,  $SO_2$ ,  $NO_2$  and PP in the age group  $\geq 65$  years were more significant.  $PM_{10}$  was associated with significantly higher DBP and MAP in the age group  $\geq 65$  years. We also found higher associations of  $SO_2$  and all four blood pressure values among participants in rural area.

The results of stratified analysis for hypertension are shown in **Figure 3**. The associations between  $PM_{2.5}$  and  $SO_2$  and hypertension were significantly higher in the age group  $< 65$  years in both Chinese and AHA/ACC hypertension guideline. We also found stronger associations between  $SO_2$  and hypertension in rural participants.

### 3.5 Sensitivity analyses

The results of sensitivity analysis are presented in Supplementary materials. The associations of blood pressure and hypertension to long-term air pollution exposure did not substantially alter in the sensitivity analyses by using 1- to 3-year average concentrations (**Table A3, A4**). When we excluded the patients with antihypertensive therapy (**Figure A1, A2**) and cardiovascular cases (**Figure A3, A4**) from the data, the results remained robust.

## Discussion

To our best knowledge, this is the first study to investigate long-term air pollution exposure and hypertension defined by Chinese and AHA/ACC guideline. We found that that 3-year average exposure to  $SO_2$  could significantly increase SBP, DBP, MAP and PP;  $PM_{2.5}$  was associated with elevated SBP, MAP and PP;  $NO_2$  could increase SBP and PP;  $PM_{10}$  could only significantly increase SBP in full adjusted single-pollutant model. All four pollutants were associated with the increment of hypertension defined by Chinese guideline, only  $PM_{2.5}$  and  $SO_2$  could increase the risk of hypertension defined by AHA/ACC guideline. In two-pollutants model we found  $PM_{2.5}$  and  $SO_2$  combined with other pollutants could significantly increase blood pressure; and  $PM_{2.5}$  could increase hypertension defined by both Chinese and AHA/ACC guideline with other pollutants.

$PM_{2.5}$ ,  $NO_2$  (only SBP and PP),  $SO_2$  was significantly associated with elevated blood pressure (SBP, DBP, MAP, PP) and hypertension which were defined by both Chinese and AHA guideline. Elder people had larger associations between  $PM_{2.5}$ ,  $NO_2$  and  $SO_2$  exposures and blood pressure (SBP and PP).

Our findings of a positive association between PM<sub>2.5</sub>, SO<sub>2</sub> and NO<sub>2</sub> exposure and blood pressure are in line with some previously study. Several studies found positive association between PM<sub>2.5</sub> exposure and blood pressure in China[25], India[10] and America[26]. An American cohort study reported that each IQR(3.91µg/m<sup>3</sup>) increase of 1-year average PM<sub>2.5</sub> exposure was associated with SBP (0.93mmHg; 95CI%,0.05-1.80) and PP (0.89mmHg; 95CI%,0.21-1.58) increased[27]. A large Sister Study in America also found a positive association between each 10µg/m<sup>3</sup> increase in PM<sub>2.5</sub> and SBP (1.4mmHg; 95CI%, 0.6-2.3) as well as PP (1.0mmHg; 95CI%, 0.4-1.7)[28]. And some studies also found positive association between long term SO<sub>2</sub> exposure and blood pressure[29, 30]. A Chinese cross-sectional study found 0.80mmHg (95CI%, 0.46-1.14) increase of SBP and 0.31mmHg (95CI%, 0.10-0.51) increase of DBP associated with each IQR (20µg/m<sup>3</sup>) increase in SO<sub>2</sub>[31]. In addition, a few studies found a positive association between short-term SO<sub>2</sub> exposure and blood pressure which can supplement our results[32, 33]. Several studies found similar association between NO<sub>2</sub> exposure and blood pressure as we found in our study[25, 34, 35]. A recent study found no-linear association between PM<sub>2.5</sub> with SBP and DBP[5], the population (n= 137,809) of this study was from 21 countries and there may remain ecological residual confounding in the analyses to lead no significantly results. The results of multi-pollutants model were robust, previous study also showed similar association between particular matter and blood pressure in multi-pollutants model, short-term exposure to PM was significantly associated with an increase of SBP and DBP[36].

All four air pollutants were significantly associated with hypertension defined by Chinses guideline. Some previous study also defined hypertension by SBP≥140 mm Hg or DBP≥ 90 mm Hg as Chinses guideline. The results in the China-PAR project showed an increased risk of incident hypertension (HR=1.77, 95% CI=1.56-2.00) among participants in the highest quartile exposure of PM<sub>2.5</sub> compared with the lowest quartile<sup>[6]</sup>.The Andhra Pradesh Children and Parents Study reported that each 1µg/m<sup>3</sup> increase of PM<sub>2.5</sub> was associated 4% higher odds of hypertension[10]. A Canadian study showed a weakly association between each IQR increase in NO<sub>2</sub> and incident hypertension (HR=1.01, 95% CI:1.00, 1.02)[37]. A study in America showed that gestational hypertension risk was significantly increased by 18% for SO<sub>2</sub> during the 3 months before conception[38]. Conversely, inconsistent associations were reported in several studies. A cross-sectional study in Taipei showed that 1-year PM<sub>2.5</sub> and NO<sub>2</sub> exposures were not associated with hypertension[39]. Black Women's Health Study in US found an inconsistent association between increasing of NO<sub>2</sub> exposure per IQR (9.7ppb) and hypertension (HR= 0.92, 95%CI: 0.86, 0.98)[12]. In addition, the results of PM<sub>2.5</sub> and SO<sub>2</sub> remain stable when we changed the definition to SBP≥130 mm Hg or DBP≥ 80 mm Hg as AHA/ACC guideline, PM<sub>10</sub> and NO<sub>2</sub> showed a non-significant association. Few previous study reported air pollution and hypertension defined by AHA/ACC, this is the first study to investigate air pollution and AHA/ACC hypertension in China, the result suggested that whichever definition is used, PM<sub>2.5</sub> and SO<sub>2</sub> is the major risk factor of hypertension.

In each city, the location of monitoring stations was not in the direct vicinity of traffic or of industrial sources, and were mandated not to be influenced by local pollution sources and should also avoid

buildings, or housing large emitters such as coal-, waste-, or oil-burning boilers, furnaces, and incinerators. Thus, we estimated air pollution exposure based on monitoring stations data, at the same time, we considered proximity to a major roadway as traffic-related pollution. Some studies found similar results as we did, a recently study among  $\geq 65$  years old Chinese people reported that residential proximity to major roadways was negative associated with hypertension, the ORs for participants living 50 to 100, 101 to 200, and  $\geq 200$  meters from major roads were 1.17, 1.21 and 1.22 respectively [40]. We found non significantly association between  $PM_{10}$  with blood pressure or hypertension, which could be explained that the association of particular matters and increasing of blood pressure is mainly affected by  $PM_{2.5}$  rather than  $PM_{10}$ . We also found the associations of  $PM_{2.5}$  and  $SO_2$  on PP appeared to be stronger consistently for participants  $\geq 65$  years old, which may be caused by vascular sclerosis in the  $\geq 65$  years group[41].

The biological mechanisms through which air pollutants can raise blood pressure are not fully understood. One potential pathway is that air pollutants may raise blood pressure and lead to hypertension by instigating autonomic imbalance[36]. The BP increase was a marker of augmented sympathetic tone[42], air pollutants especially PM likely altered autonomic balance via the activation of afferent pulmonary autonomic reflexes[1]. Other hypothesized pathway including systemic inflammation, oxidative stress reaction and endothelial dysfunction[1] and DNA methylation[43]. Our previous study and others have reported positive association between PM exposure and inflammatory cytokines[44, 45].

Our study has several strengths. First, this is the first study to investigate the air pollution and hypertension defined by both Chinese and AHA/ACC guideline, and we found a stable correlation between  $PM_{2.5}$  and  $SO_2$  with hypertension, even though the definition was changed to  $SBP \geq 130$  mm Hg or  $DBP \geq 80$  mm Hg. Second, the concentrations of all air pollutants were relatively high in our study regions rather than the low level of air pollution exposures in other countries, for example, the average concentration of  $PM_{2.5}$   $\mu g/m^3$  America[46]19.6 $\mu g/m^3$  in Danish[47] and 73.5 $\mu g/m^3$ , therefore, our results could represent the association between high-level and long-term exposures with blood pressure and incident hypertension. Third, we analyzed the association between residential proximity to major roadways with blood pressure values and incident hypertension, which presents the effects of traffic-related pollution. Fourth, the air pollution of our study included not only particular matters but also gaseous pollutants, furthermore we used two-pollutants model to explore the combination effect of air pollutants. Finally, a large set of covariates were considered in our comprehensive statistical analyses to minimize the impact of confounding variables, including BMI, education attainment, monthly income, gross domestic product, and population density.

There are several limitations in the present study. First, similar to previous cross-sectional studies, we could not establish a causal relationship between air pollutants exposure and blood pressure increasing or hypertension. Second, except for the concentrations of  $PM_{2.5}$ , where individual levels were estimated by a spatial statistical model, the exposure levels of  $PM_{10}$ ,  $SO_2$ ,  $NO_2$  were assigned using data from the nearest air monitoring station, which resulted in an exposure misclassification. Third, some covariates

were not available, such as indoor air pollution and diet information, we cannot exclude the possible confounding bias by unmeasured factors. Finally, the information of drugs taking was not included in the questionnaire, which was an unmeasured confounding factor in our study.

## Conclusion

In conclusion, we found that long-term exposure to higher levels of PM<sub>2.5</sub>, NO<sub>2</sub> and SO<sub>2</sub> is associated with increase of blood pressure values and hypertension defined by both Chinses and ACC/AHA guideline adjusting for a wide range of covariates. PM10 is associated with higher SBP and risk of hypertension defined by Chinese guideline. Participant who live within 100m from major roadway is associated with significantly higher SBP, PP and risk of hypertension. We also found robust association in stratified analysis and sensitivity analysis. Our study provides evidence of a role for high levels air pollutants in the blood pressure increase and development of hypertension. However, further longitudinal studies are warranted to confirm our findings.

## Declarations

### Ethical Approval and Consent to participate

Each of the participants provided a written informed consent at their enrollment, and the ethical committees of Capital Medical University (Beijing, China), Chaoyang District Center for Disease Prevention and Control (Beijing, China), and Hebei Provincial Center for Disease Prevention and Control (Shijiazhuang, Hebei) approved this study.

### Consent for publication

Written informed consent for publication was obtained from all participants.

### Competing interests

None

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### Authors' contributions

Bingxiao Li: Investigation, Methodology, Data curation, Formal analysis, Visualization, Writing original draft.

Han Cao: Investigation, Data curation, Formal analysis, Visualization. Kuo Liu: Data curation.

Li Pan, Ze Cui, Wei Zhao, Han Zhang, Kaijun Niu, Naijun Tang, Jixin Sun, Xiaoyan Han,

Zhengfang Wang, Juan Xia, Huijing He, Yajing Cao, Zhiyuan Xu, Ge Meng, Anqi Shan, Chunyue Guo, Yanyan Sun, Wenjuan Peng, Xiaohui Liu, Yunyi Xie, Fuyuan Wen, Fengxu Zhang: Investigation.

Guangliang Shan: Conceptualization, Investigation, Data curation, Validation.

Ling Zhang: Conceptualization, Investigation, Writing -review & editing, Supervision.

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## Abbreviations

ACC, American College of Cardiology; AHA, American Heart Association; CHCN-BTH, the Cohort Study on Chronic Disease of Communities Natural Population in Beijing, Tianjin and Hebei; CI, confidence interval; GLMMs, generalized linear mixed models; IQR, interquartile range; OR, odds ratio; PM<sub>2.5</sub>, particulate matter  $\leq 2.5\mu\text{m}$  in aerodynamic diameter; PM<sub>10</sub>, particulate matter  $\leq 10\mu\text{m}$  in aerodynamic diameter; SO<sub>2</sub>, sulfur dioxide; NO<sub>2</sub>, Nitrogen dioxide; SBP, systolic blood pressure; DBP, diastolic blood pressure

## References

1. Brook RD, Rajagopalan S, Pope CA, 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA *et al*: **Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association.** *Circulation* 2010, **121**(21):2331-2378.
2. **Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016.** *Lancet (London, England)* 2017, **390**(10100):1345-1422.
3. **Guidelines for the Prevention and Treatment of Hypertension in China (Revised 2018).** *Chinese Journal of Cardiology* 2019, **24**(01):24-56.
4. Liu WT, Lee KY, Lee HC, Chuang HC, Wu D, Juang JN, Chuang KJ: **The association of annual air pollution exposure with blood pressure among patients with sleep-disordered breathing.** *The Science of the total environment* 2016, **543**(Pt A):61-66.
5. Arku RE, Brauer M, Ahmed SH, AlHabib KF, Avezum Á, Bo J, Choudhury T, Dans AM, Gupta R, Iqbal R *et al*: **Long-term exposure to outdoor and household air pollution and blood pressure in the Prospective Urban and Rural Epidemiological (PURE) study.** *Environmental pollution (Barking, Essex : 1987)* 2020, **262**:114197.
6. Huang K, Yang X, Liang F, Liu F, Li J, Xiao Q, Chen J, Liu X, Cao J, Shen C *et al*: **Long-Term Exposure to Fine Particulate Matter and Hypertension Incidence in China.** *Hypertension (Dallas, Tex : 1979)* 2019,

73(6):1195-1201.

7. Zhang Z, Guo C, Lau AKH, Chan TC, Chuang YC, Lin C, Jiang WK, Yeoh EK, Tam T, Woo KS *et al*: **Long-Term Exposure to Fine Particulate Matter, Blood Pressure, and Incident Hypertension in Taiwanese Adults.** *Environmental health perspectives* 2018, **126**(1):017008.
8. Yang BY, Qian Z, Howard SW, Vaughn MG, Fan SJ, Liu KK, Dong GH: **Global association between ambient air pollution and blood pressure: A systematic review and meta-analysis.** *Environmental pollution (Barking, Essex : 1987)* 2018, **235**:576-588.
9. Zhang Z, Dong B, Li S, Chen G, Yang Z, Dong Y, Wang Z, Ma J, Guo Y: **Exposure to ambient particulate matter air pollution, blood pressure and hypertension in children and adolescents: A national cross-sectional study in China.** *Environment international* 2019, **128**:103-108.
10. Curto A, Wellenius GA, Milà C, Sanchez M, Ranzani O, Marshall JD, Kulkarni B, Bhogadi S, Kinra S, Tonne C: **Ambient Particulate Air Pollution and Blood Pressure in Peri-urban India.** *Epidemiology (Cambridge, Mass)* 2019, **30**(4):492-500.
11. Lee DH, Kim SH, Kang SH, Kwon OK, Park JJ, Yoon CH, Cho YS, Heo J, Yi SM, Youn TJ *et al*: **Personal exposure to fine particulate air pollutants impacts blood pressure and heart rate variability.** *Scientific reports* 2020, **10**(1):16538.
12. Coogan PF, White LF, Yu J, Brook RD, Burnett RT, Marshall JD, Bethea TN, Rosenberg L, Jerrett M: **Long-Term Exposure to NO<sub>2</sub> and Ozone and Hypertension Incidence in the Black Women's Health Study.** *American journal of hypertension* 2017, **30**(4):367-372.
13. Liu C, Fuertes E, Tiesler CM, Birk M, Babisch W, Bauer CP, Koletzko S, von Berg A, Hoffmann B, Heinrich J: **The associations between traffic-related air pollution and noise with blood pressure in children: results from the GINIplus and LISApplus studies.** *International journal of hygiene and environmental health* 2014, **217**(4-5):499-505.
14. Li J, Liao H, Hu J, Li N: **Severe particulate pollution days in China during 2013-2018 and the associated typical weather patterns in Beijing-Tianjin-Hebei and the Yangtze River Delta regions.** *Environmental pollution (Barking, Essex : 1987)* 2019, **248**:74-81.
15. Whelton PK, Carey RM, Aronow WS, Casey DE, Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW *et al*: **2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines.** *Journal of the American College of Cardiology* 2018, **71**(19):2199-2269.
16. **Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy.** *American journal of obstetrics and gynecology* 2000, **183**(1):S1-s22.
17. Ishii M, Seki T, Sakamoto K, Kaikita K, Miyamoto Y, Tsujita K, Masuda I, Kawakami K: **Association of short term exposure to Asian dust with increased blood pressure.** *Scientific reports* 2020, **10**(1):17630.
18. Lu Y, Lin S, Fatmi Z, Malashock D, Hussain MM, Siddique A, Carpenter DO, Lin Z, Khwaja HA: **Assessing the association between fine particulate matter (PM<sub>2.5</sub>) constituents and cardiovascular**

- diseases in a mega-city of Pakistan. *Environmental pollution (Barking, Essex : 1987)* 2019, **252**(Pt B):1412-1422.
19. Cao H, Li B, Peng W, Pan L, Cui Z, Zhao W, Zhang H, Tang N, Niu K, Sun J *et al*: **Associations of long-term exposure to ambient air pollution with cardiac conduction abnormalities in Chinese adults: The CHCN-BTH cohort study.** *Environment international* 2020, **143**:105981.
20. Xue T, Zheng Y, Tong D, Zheng B, Li X, Zhu T, Zhang Q: **Spatiotemporal continuous estimates of PM(2.5) concentrations in China, 2000-2016: A machine learning method with inputs from satellites, chemical transport model, and ground observations.** *Environment international* 2019, **123**:345-357.
21. Ljungman PLS, Li W, Rice MB, Wilker EH, Schwartz J, Gold DR, Koutrakis P, Benjamin EJ, Vasan RS, Mitchell GF *et al*: **Long- and short-term air pollution exposure and measures of arterial stiffness in the Framingham Heart Study.** *Environment international* 2018, **121**(Pt 1):139-147.
22. **2018 Chinese Guidelines for Prevention and Treatment of Hypertension-A report of the Revision Committee of Chinese Guidelines for Prevention and Treatment of Hypertension.** *Journal of geriatric cardiology : JGC* 2019, **16**(3):182-241.
23. Whelton PK, Carey RM, Aronow WS, Casey DE, Jr., Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW *et al*: **2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines.** *Hypertension (Dallas, Tex : 1979)* 2018, **71**(6):1269-1324.
24. Lin H, Guo Y, Zheng Y, Di Q, Liu T, Xiao J, Li X, Zeng W, Cummings-Vaughn LA, Howard SW *et al*: **Long-Term Effects of Ambient PM(2.5) on Hypertension and Blood Pressure and Attributable Risk Among Older Chinese Adults.** *Hypertension (Dallas, Tex : 1979)* 2017, **69**(5):806-812.
25. Li N, Chen G, Liu F, Mao S, Liu Y, Liu S, Mao Z, Lu Y, Wang C, Guo Y *et al*: **Associations between long-term exposure to air pollution and blood pressure and effect modifications by behavioral factors.** *Environmental research* 2020, **182**:109109.
26. Adar SD, Chen YH, D'Souza JC, O'Neill MS, Szpiro AA, Auchincloss AH, Park SK, Daviglius ML, Diez Roux AV, Kaufman JD: **Longitudinal Analysis of Long-Term Air Pollution Levels and Blood Pressure: A Cautionary Tale from the Multi-Ethnic Study of Atherosclerosis.** *Environmental health perspectives* 2018, **126**(10):107003.
27. Honda T, Pun VC, Manjourides J, Suh H: **Associations of long-term fine particulate matter exposure with prevalent hypertension and increased blood pressure in older Americans.** *Environmental research* 2018, **164**:1-8.
28. Chan SH, Van Hee VC, Bergen S, Szpiro AA, DeRoo LA, London SJ, Marshall JD, Kaufman JD, Sandler DP: **Long-Term Air Pollution Exposure and Blood Pressure in the Sister Study.** *Environmental health perspectives* 2015, **123**(10):951-958.
29. Stanković A, Nikolić M: **Long-term ambient air pollution exposure and risk of high blood pressure among citizens in Nis, Serbia.** *Clinical and experimental hypertension (New York, NY : 1993)* 2016,

- 38(1):119-124.
30. Dong GH, Qian ZM, Trevathan E, Zeng XW, Vaughn MG, Wang J, Zhao Y, Liu YQ, Ren WH, Qin XD: **Air pollution associated hypertension and increased blood pressure may be reduced by breastfeeding in Chinese children: the Seven Northeastern Cities Chinese Children's Study.** *International journal of cardiology* 2014, **176**(3):956-961.
  31. Dong GH, Qian ZM, Xaverius PK, Trevathan E, Maalouf S, Parker J, Yang L, Liu MM, Wang D, Ren WH *et al.*: **Association between long-term air pollution and increased blood pressure and hypertension in China.** *Hypertension (Dallas, Tex : 1979)* 2013, **61**(3):578-584.
  32. Liu L, Kauri LM, Mahmud M, Weichenthal S, Cakmak S, Shutt R, You H, Thomson E, Vincent R, Kumarathasan P *et al.*: **Exposure to air pollution near a steel plant and effects on cardiovascular physiology: a randomized crossover study.** *International journal of hygiene and environmental health* 2014, **217**(2-3):279-286.
  33. Chen SY, Su TC, Lin YL, Chan CC: **Short-term effects of air pollution on pulse pressure among nonsmoking adults.** *Epidemiology (Cambridge, Mass)* 2012, **23**(2):341-348.
  34. Foraster M, Künzli N, Aguilera I, Rivera M, Agis D, Vila J, Bouso L, Deltell A, Marrugat J, Ramos R *et al.*: **High blood pressure and long-term exposure to indoor noise and air pollution from road traffic.** *Environmental health perspectives* 2014, **122**(11):1193-1200.
  35. Foraster M, Basagaña X, Aguilera I, Rivera M, Agis D, Bouso L, Deltell A, Marrugat J, Ramos R, Sunyer J *et al.*: **Association of long-term exposure to traffic-related air pollution with blood pressure and hypertension in an adult population-based cohort in Spain (the REGICOR study).** *Environmental health perspectives* 2014, **122**(4):404-411.
  36. Brook RD, Urch B, Dvonch JT, Bard RL, Speck M, Keeler G, Morishita M, Marsik FJ, Kamal AS, Kaciroti N *et al.*: **Insights into the mechanisms and mediators of the effects of air pollution exposure on blood pressure and vascular function in healthy humans.** *Hypertension (Dallas, Tex : 1979)* 2009, **54**(3):659-667.
  37. Bai L, Chen H, Hatzopoulou M, Jerrett M, Kwong JC, Burnett RT, van Donkelaar A, Copes R, Martin RV, Van Ryswyk K *et al.*: **Exposure to Ambient Ultrafine Particles and Nitrogen Dioxide and Incident Hypertension and Diabetes.** *Epidemiology (Cambridge, Mass)* 2018, **29**(3):323-332.
  38. Zhu Y, Zhang C, Liu D, Ha S, Kim SS, Pollack A, Mendola P: **Ambient Air Pollution and Risk of Gestational Hypertension.** *American journal of epidemiology* 2017, **186**(3):334-343.
  39. Chen SY, Wu CF, Lee JH, Hoffmann B, Peters A, Brunekreef B, Chu DC, Chan CC: **Associations between Long-Term Air Pollutant Exposures and Blood Pressure in Elderly Residents of Taipei City: A Cross-Sectional Study.** *Environmental health perspectives* 2015, **123**(8):779-784.
  40. Yao Y, Cao K, Zhang K, Zhu T, Yue D, Zhang H, Zhang J, Jin X, Zeng Y: **Residential Proximity to Major Roadways and Prevalent Hypertension Among Older Women and Men: Results From the Chinese Longitudinal Healthy Longevity Survey.** *Frontiers in cardiovascular medicine* 2020, **7**:587222.
  41. Cheungpasitporn W, Nasr SH, Thongprayoon C, Mao MA, Qian Q: **Primary IgA nephropathy in elderly patients.** *Nephrology (Carlton, Vic)* 2015, **20**(6):419-425.

42. Palatini P: **Role of elevated heart rate in the development of cardiovascular disease in hypertension.** *Hypertension (Dallas, Tex : 1979)* 2011, **58**(5):745-750.
43. Wang C, Chen R, Cai J, Shi J, Yang C, Tse LA, Li H, Lin Z, Meng X, Liu C *et al*: **Personal exposure to fine particulate matter and blood pressure: A role of angiotensin converting enzyme and its DNA methylation.** *Environment international* 2016, **94**:661-666.
44. Sun Y, Huang J, Zhao Y, Xue L, Li H, Liu Q, Cao H, Peng W, Guo C, Xie Y *et al*: **Inflammatory cytokines and DNA methylation in healthy young adults exposure to fine particulate matter: A randomized, double-blind crossover trial of air filtration.** *Journal of hazardous materials* 2020, **398**:122817.
45. Tang H, Cheng Z, Li N, Mao S, Ma R, He H, Niu Z, Chen X, Xiang H: **The short- and long-term associations of particulate matter with inflammation and blood coagulation markers: A meta-analysis.** *Environmental pollution (Barking, Essex : 1987)* 2020, **267**:115630.
46. McGuinn LA, Ward-Caviness CK, Neas LM, Schneider A, Diaz-Sanchez D, Cascio WE, Kraus WE, Hauser E, Dowdy E, Haynes C *et al*: **Association between satellite-based estimates of long-term PM2.5 exposure and coronary artery disease.** *Environmental research* 2016, **145**:9-17.
47. Cramer J, Jørgensen JT, Hoffmann B, Loft S, Bräuner EV, Prescott E, Ketzel M, Hertel O, Brandt J, Jensen SS *et al*: **Long-Term Exposure to Air Pollution and Incidence of Myocardial Infarction: A Danish Nurse Cohort Study.** *Environmental health perspectives* 2020, **128**(5):57003.

## Tables

Table 1. Main characteristics of the study participants according to Chinese hypertension guideline.

Characteristics	Hypertension	Non-hypertension	Total
Number (%)	13,106 (40.8)	19,029 (59.2)	32,135 (100.0)
Age, mean $\pm$ SD, years	57.4 $\pm$ 11.8	44.9 $\pm$ 13.6	50.0 $\pm$ 14.3
Body mass index, mean $\pm$ SD, kg/m <sup>2</sup>	26.6 $\pm$ 3.6	24.4 $\pm$ 3.6	25.3 $\pm$ 3.8
Blood pressure, mean $\pm$ SD, mmHg			
Systolic blood pressure	145.7 $\pm$ 16.4	118.1 $\pm$ 11.4	129.3 $\pm$ 19.2
Diastolic blood pressure	85.6 $\pm$ 13.0	72.2 $\pm$ 8.4	77.7 $\pm$ 12.4
Mean arterial pressure	105.6 $\pm$ 12.0	87.5 $\pm$ 8.6	94.9 $\pm$ 13.5
Pulse pressure	60.1 $\pm$ 16.3	45.9 $\pm$ 8.6	51.7 $\pm$ 14.2
Gender, N (%)			
Male	6252 (47.7)	8078 (42.5)	14,330 (44.6)
Female	6854 (52.3)	10,951 (57.5)	17,805 (55.4)
Ethnicity, N (%)			
Han	11,933 (91.1)	16,886 (88.7)	28,819 (89.7)
Other	1173 (8.9)	2143 (11.3)	3316 (10.3)
Residence, N (%)			
Urban	8927 (68.1)	14,237 (74.8)	23,164 (72.1)
Rural	4179 (31.9)	4792 (25.2)	8971 (27.9)
Highest educational attainment, N (%)			
Junior college or higher	2303 (17.6)	7700 (40.5)	10,003 (31.1)
Middle school	8026 (61.2)	9280 (48.8)	17,306 (53.9)
Primary school	2222 (17.0)	1738 (9.1)	3960 (12.3)
No school	555 (4.2)	311 (1.6)	866 (2.7)
Marital status, N (%)			
Married or cohabiting	11,689 (89.2)	15,986 (84.0)	27,675 (86.1)
Single/Widowed/divorced/separation	1417 (10.8)	3043 (16.0)	4460 (13.9)
Average monthly income, N (%)			
$\leq$ 1000 RMB/month	3570 (27.2)	3437 (18.0)	7007 (21.8)
1001-5000 RMB/month	8503 (64.9)	12,513 (65.8)	21,016 (65.4)
5001-9999 RMB/month	819 (6.3)	2313 (12.2)	3132 (9.7)
$\geq$ 10000 RMB/month	214 (1.6)	766 (4.0)	980 (3.1)
Smoking status, N (%)			
Never	8492 (64.8)	13,777 (72.4)	22,269 (69.3)
Ever	4614 (35.2)	5252 (27.6)	9866 (30.7)
Alcohol drinking status, N (%)			
Never	7369 (56.2)	11,050 (58.1)	18,419 (57.3)
Ever	5737 (43.8)	7979 (41.9)	13,716 (42.7)
Regular exercise, N (%)			
Yes	9603 (73.3)	12,444 (65.4)	22,047 (68.6)
No	3503 (26.7)	6585 (34.6)	10,088 (31.4)
Family history of hypertension, N (%)	8645 (66.0)	9952 (52.3)	18,597 (57.9)
Diabetes, N (%)	3054 (23.3)	1410 (7.4)	4464 (13.9)

Note: Data are the mean (standard deviation) for continuous variables and number (percentage) for categorical variables.

Table 2. Ambient air pollutant concentrations and pairwise correlations

Variable	3-year average pollutant concentrations and proximity to a major roadway		Spearman rank correlation coefficients			
	Mean $\pm$ SD or N (%)	Median (IQR)	PM <sub>2.5</sub>	PM <sub>10</sub>	SO <sub>2</sub>	NO <sub>2</sub>
<b>Ambient air pollutant</b>						
PM <sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ )	73.5 $\pm$ 14.3	73.2 (10.2)	1	0.22*	0.25*	0.26*
PM <sub>10</sub> ( $\mu\text{g}/\text{m}^3$ )	123.0 $\pm$ 13.3	122.3 (14.9)	NA	1	0.47*	0.25*
SO <sub>2</sub> ( $\mu\text{g}/\text{m}^3$ )	22.9 $\pm$ 8.9	17.5 (13.7)	NA	NA	1	0.40*
NO <sub>2</sub> ( $\mu\text{g}/\text{m}^3$ )	52.2 $\pm$ 6.5	52.4 (11.5)	NA	NA	NA	1
<b>Proximity to a major roadway</b>						
Residential proximity, m	408.5 $\pm$ 282.3	376.5 (445.5)	-0.02*	-0.04*	-0.09*	-0.02*
<b>Residential proximity, m</b>						
<100	3826 (16.3)	NA	NA	NA	NA	NA
100 to <200	2869 (12.2)	NA	NA	NA	NA	NA
200 to <400	5628 (24.0)	NA	NA	NA	NA	NA
400 to $\leq$ 1000	11,180 (47.5)	NA	NA	NA	NA	NA
<b>Meteorological data</b>						
Tem, °C	14.7 $\pm$ 12.2	19.1 (23.3)	0.18*	0.10*	0.12*	-0.32*
Hum, %	55.5 $\pm$ 21.5	57.2 (38.0)	0.19*	0.20*	0.23*	-0.45*

PM<sub>2.5</sub>, particulate matter  $\leq 2.5\mu\text{m}$  in aerodynamic diameter; PM<sub>10</sub>, particulate matter  $\leq 10\mu\text{m}$  in aerodynamic diameter; SO<sub>2</sub>, sulfur dioxide; NO<sub>2</sub>, Nitrogen dioxide; IQR, interquartile range; Residential proximity; the distance of residential proximity to a major roadway; Tem, temperature of the test day; Hum, humidity of the test day.

Table 3. Associations between long-term exposures to air pollutants and blood pressure.

Pollutant	SBP	DBP	MAP	PP
PM <sub>2.5</sub> an IQR (19µg/m <sup>3</sup> )	<b>0.66 (0.30, 1.03)**</b>	0.23 (-0.07, 0.53)	<b>0.37 (0.09, 0.65)*</b>	<b>0.47 (0.09, 0.74) *</b>
	Reference	Reference	Reference	Reference
	0.62 (-0.11, 1.34)	<b>0.70 (0.16, 1.24) *</b>	<b>0.677 (0.13, 1.22) *</b>	-0.03 (-0.59, 0.54)
	<b>0.97 (0.05, 1.89) *</b>	0.04 (-0.67, 0.75)	0.36 (-0.34, 1.06)	<b>0.90 (0.16, 1.64) *</b>
	<b>4.35 (3.25, 5.44) **</b>	<b>2.05 (1.18, 2.91) **</b>	<b>2.81 (1.96, 3.66) **</b>	<b>2.36 (1.46, 3.26) **</b>
Linear trend	<0.01	<0.01	<0.01	<0.01
PM <sub>10</sub> an IQR (87µg/m <sup>3</sup> )	<b>0.40 (0.00, 0.81)*</b>	0.15 (-0.14, 0.43)	0.23 (-0.06, 0.53)	0.25 (-0.04, 0.55)
	Reference	Reference	Reference	Reference
	3.06 (-8.82, 14.95)	<b>10.57 (2.17, 18.97)*</b>	8.07 (-0.71, 16.85)	-7.50 (-16.25, 1.24)
	7.31 (-7.13, 21.74)	2.78 (-7.42, 12.98)	4.29 (-6.38, 14.96)	4.52 (-6.09, 15.14)
	4.52 (-10.45, 19.49)	5.75 (-4.83, 16.32)	5.34 (-5.73, 16.40)	-1.23 (-12.24, 9.78)
Linear trend	0.39	0.64	0.41	0.52
PM <sub>2.5-10</sub> an IQR (72µg/m <sup>3</sup> )	<b>1.38 (0.92, 1.84) **</b>	<b>0.42 (0.11, 0.72) *</b>	<b>0.65 (0.33, 0.98) **</b>	<b>1.07 (0.71, 1.42) **</b>
	Reference	Reference	Reference	Reference
	-0.52 (-1.15, 0.12)	<b>0.84 (0.38, 1.29) *</b>	0.39 (-0.08, 0.87)	<b>-1.39 (-1.88, -0.90) *</b>
	<b>1.79 (1.14, 2.43) *</b>	<b>0.57 (0.11, 1.03) *</b>	<b>0.98 (0.50, 1.46) *</b>	<b>1.19 (0.70, 1.69) *</b>
	<b>3.48 (2.56, 4.40) **</b>	<b>1.42 (0.99, 1.86) **</b>	<b>2.13 (1.47, 2.80) *</b>	<b>1.93 (1.21, 2.64) *</b>
Linear trend	<0.01	<0.01	<0.01	<0.01
PM <sub>2.5</sub> an IQR (50µg/m <sup>3</sup> )	<b>0.54 (0.16, 0.91) *</b>	0.14 (-0.18, 0.45)	0.24 (-0.08, 0.56)	<b>0.49 (0.20, 0.79) *</b>
	Reference	Reference	Reference	Reference
	-0.31 (-0.87, 0.24)	-0.43 (-0.88, 0.03)	-0.47 (-0.93, 0.01)	0.13 (-0.30, 0.55)
	-0.40 (-1.18, 0.39)	-0.14 (-0.75, 0.47)	-0.31 (-0.94, 0.31)	-0.25 (-0.85, 0.36)
	<b>0.94 (0.34, 1.54) *</b>	-0.38 (-0.86, 0.11)	-0.02 (-0.52, 0.47)	<b>1.32 (0.86, 1.79) *</b>
Linear trend	<0.01	0.42	0.20	<0.01

A GLMMs model was used and adjust for age, gender, BMI, ethnicity, place, marital, education level, income, smoke, alcohol, exercise, diabetes, hypertension medication, family history of hypertension, temperature, GDP. The regression coefficient ( $\beta$ ) was calculated by each IQR increment of pollutants exposure.

Q1, 1st quartile; Q2, 2nd quartile; Q3, 3rd quartile; Q4, 4th quartile

\*,  $P < 0.05$ ; \*\*,  $P < 0.01$ .

Table 4. Associations between long-term exposures to air pollutants and hypertension.

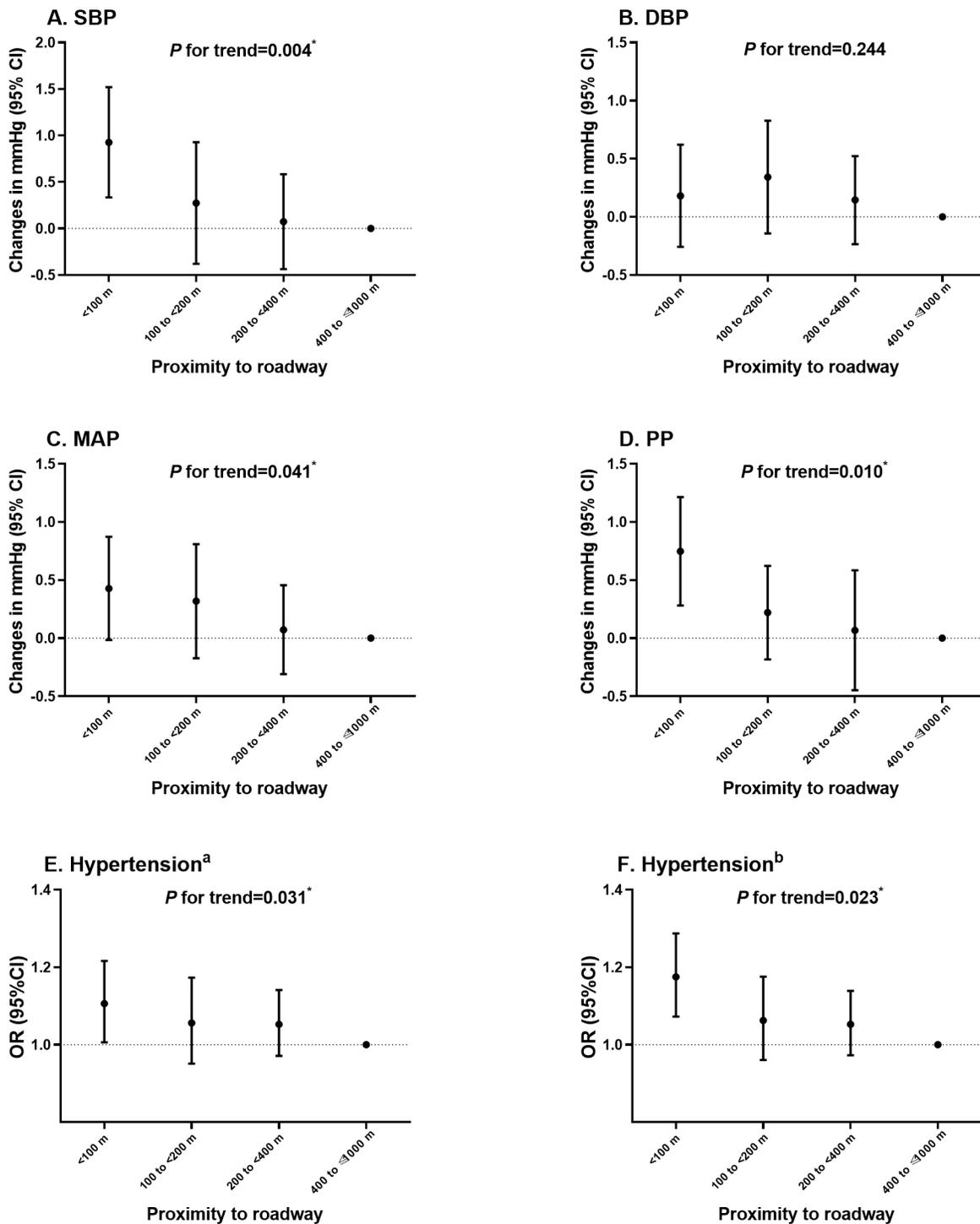
Air pollutant	Chinese Hypertension guideline	AHA Hypertension guideline
<b>N (%)</b>		
<b>PM<sub>2.5</sub></b>		
Per an IQR (10.2µg/m <sup>3</sup> )	<b>1.14 (1.06, 1.23)**</b>	<b>1.17 (1.09, 1.25) **</b>
Q1	Reference	Reference
Q2	1.05 (0.95, 1.15)	1.09 (0.99, 1.20)
Q3	<b>1.30 (1.18, 1.44)*</b>	<b>1.25 (1.14, 1.37) *</b>
Q4	<b>1.57 (1.36, 1.82)*</b>	<b>1.77 (1.54, 2.03) *</b>
P for trend	<0.01	<0.01
<b>PM<sub>10</sub></b>		
Per an IQR (14.9µg/m <sup>3</sup> )	<b>1.06 (1.00, 1.13)*</b>	1.03 (0.98, 1.09)
Q1	Reference	Reference
Q2	4.14 (0.77, 22.30)	0.55 (0.11, 2.76)
Q3	0.61 (0.08, 4.78)	0.13 (0.02, 1.91)
Q4	1.89 (0.23, 15.86)	0.58 (0.08, 4.36)
P for trend	0.07	0.08
<b>SO<sub>2</sub></b>		
Per an IQR (13.7µg/m <sup>3</sup> )	<b>1.09 (1.02, 1.17)*</b>	<b>1.10 (1.03, 1.18) *</b>
Q1	Reference	Reference
Q2	1.00 (0.90, 1.11)	1.10 (0.99, 1.21)
Q3	<b>1.29 (1.16, 1.43)*</b>	<b>1.36 (1.23, 1.50) *</b>
Q4	<b>1.33 (1.15, 1.53)*</b>	<b>1.33 (1.16, 1.53) *</b>
P for trend	<0.01	<0.001
<b>NO<sub>2</sub></b>		
Per an IQR (11.5µg/m <sup>3</sup> )	<b>1.08 (1.00, 1.16)*</b>	1.02 (0.96, 1.09)
Q1	Reference	Reference
Q2	0.99 (0.89, 1.09)	0.95 (0.87, 1.05)
Q3	0.95 (0.83, 1.09)	0.94 (0.83, 1.07)
Q4	<b>1.13 (1.02, 1.26)*</b>	1.05 (0.95, 1.16)
P for trend	<0.01	0.051

A two-level logistic model was used and adjust for age, gender, BMI, ethnicity, place, marital, education level, income, smoke, alcohol, exercise, diabetes, hypertension medication, family history of hypertension, temperature, GDP. The odds ratio (OR) was calculated by each IQR increment of pollutants exposure.

Q1, 1st quartile; Q2, 2nd quartile; Q3, 3rd quartile; Q4, 4th quartile

\*: P <0.05; \*\*: P <0.01.

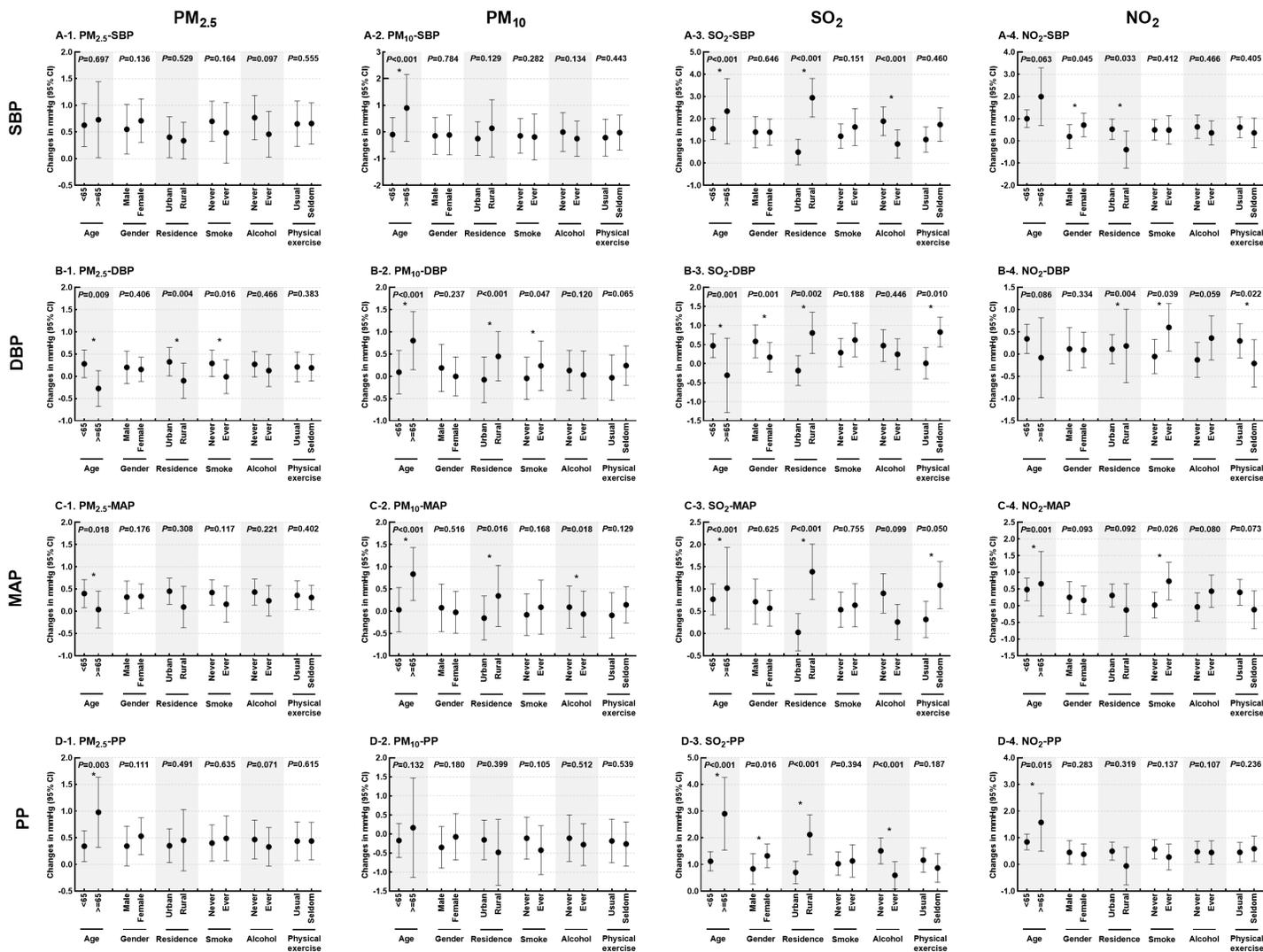
## Figures



**Figure 1**

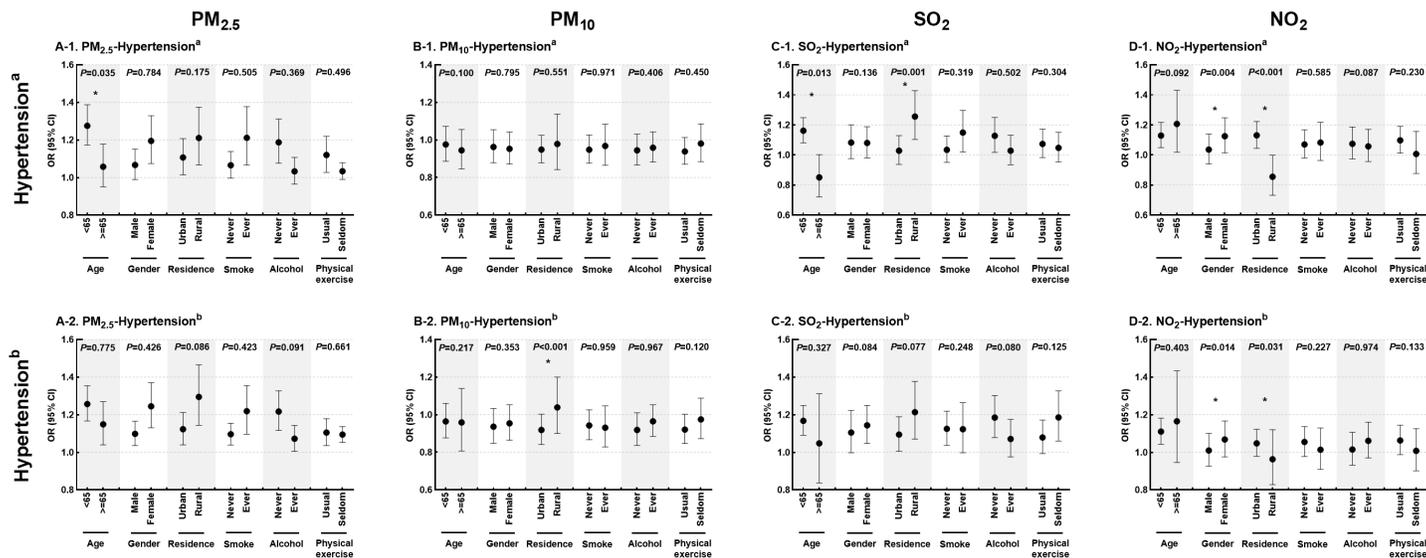
Associations between proximity to roadway and SBP, DBP, MAP, PP and hypertension defined by both Chinses (Hypertension a) and ACC/AHA guideline (Hypertension b). A GLMMs model was used between proximity to roadway and SBP, DBP, MAP, PP and adjust for age, gender, BMI, ethnicity, place, marital, education level, income, smoke, alcohol, exercise, diabetes, hypertension medication, family history of hypertension, temperature, GDP. The regression coefficient ( $\beta$ ) was calculated by each IQR increment of

pollutants exposure. A two-level logistic model was used between proximity to roadway and hypertension defined by both Chinses (Hypertension a) and ACC/AHA guideline (Hypertension b), and adjust for age, gender, BMI, ethnicity, place, marital, education level, income, smoke, alcohol, exercise, diabetes, hypertension medication, family history of hypertension, temperature, GDP. The odds ratio (OR) was calculated by each IQR increment of pollutants exposure.



**Figure 2**

Stratified analysis on associations between long-term air pollutants exposure (each IQR increments) and SBP, DBP, MAP and PP. Effect estimates (coefficients) are derived from GLMMs model, and bars cover 95% confidence intervals. Adjust for age (not in age-stratified analysis), gender (not in gender-stratified analysis), BMI (not in age-stratified analysis), ethnicity, residence (not in residence-stratified analysis), marital, education level, income, smoke (not in smoke-stratified analysis), alcohol (not in alcohol-stratified analysis), exercise (not in physical exercise-stratified analysis), diabetes, hypertension medication, family history of hypertension, temperature, GDP. p-Values for interaction terms between air pollutants (continuous variable) and each potential modifier (dichotomous variable) are presented in the figure.



**Figure 3**

Stratified analysis on associations between long-term air pollutants exposure (each IQR increments) and hypertension defined by both Chinse (Hypertension a) and ACC/AHA guideline (Hypertension b). Effect estimates (Odd ratios) are derived from two-level logistic regression model, and bars cover 95% confidence intervals. Adjust for age (not in age-stratified analysis), gender (not in gender-stratified analysis), BMI (not in age-stratified analysis), ethnicity, residence (not in residence-stratified analysis), marital, education level, income, smoke (not in smoke-stratified analysis), alcohol (not in alcohol-stratified analysis), exercise (not in physical exercise-stratified analysis), diabetes, hypertension medication, family history of hypertension, temperature, GDP. p-Values for interaction terms between air pollutants (continuous variable) and each potential modifier (dichotomous variable) are presented in the figure.

## Supplementary Files

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- [supplementalmaterial.docx](#)