

Association Between Long-Term Air Pollution and Increased Blood Pressure and Hypertension in China

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Abstract—Several studies have investigated the short-term effects of ambient air pollutants in the development of high blood pressure and hypertension. However, little information exists regarding the health effects of long-term exposure. To investigate the association between residential long-term exposure to air pollution and blood pressure and hypertension, we studied 24 845 Chinese adults in 11 districts of 3 northeastern cities from 2009 to 2010. Three-year average concentration of particles with an aerodynamic diameter $\leq 10 \mu\text{m}$ (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxides (NO_2), and ozone (O_3) were calculated from monitoring stations in the 11 districts. We used generalized additive models and 2-level logistic regressions models to examine the health effects. The results showed that the odds ratio for hypertension increased by 1.12 (95% confidence interval [CI], 1.08–1.16) per $19 \mu\text{g}/\text{m}^3$ increase in PM_{10} , 1.11 (95% CI, 1.04–1.18) per $20 \mu\text{g}/\text{m}^3$ increase in SO_2 , and 1.13 (95% CI, 1.06–1.20) per $22 \mu\text{g}/\text{m}^3$ increase in O_3 . The estimated increases in mean systolic and diastolic blood pressure were 0.87 mmHg (95% CI, 0.48–1.27) and 0.32 mmHg (95% CI, 0.08–0.56) per $19 \mu\text{g}/\text{m}^3$ interquartile increase in PM_{10} , 0.80 mmHg (95% CI, 0.46–1.14) and 0.31 mmHg (95% CI, 0.10–0.51) per $20 \mu\text{g}/\text{m}^3$ interquartile increase in SO_2 , and 0.73 mmHg (95% CI, 0.35–1.11) and 0.37 mmHg (95% CI, 0.14–0.61) per $22 \mu\text{g}/\text{m}^3$ interquartile increase in O_3 . These associations were only statistically significant in men. In conclusion, long-term exposure to PM_{10} , SO_2 , and O_3 was associated with increased arterial blood pressure and hypertension in the study population. (*Hypertension*. 2013;61:578-584.)

Key Words: ambient air pollutants ■ blood pressure ■ Chinese ■ hypertension

As the major risk factor for cardiovascular disease and other vascular diseases,^{1–3} high blood pressure (BP) and hypertension have been identified as the most important causes of disability and the leading risk factors for death in the world, causing an estimated 7.5 million deaths per year (13% of all deaths).⁴ Because chronically elevated BP is an important determinant for the development and progression of atherosclerosis, the general trend in most developing countries of the increasing prevalence of hypertension is of public health concern.⁵ It has been suggested that this increase cannot be explained by genetic factors and changes in lifestyle and diet structure alone but that environmental factors, including ambient air pollution, may play a role as well.⁶

In recent years, several studies have investigated the short-term effects of ambient air pollutants in the development of high BP and hypertension among humans, but the results of these studies are inconstant. Evidence from studies conducted in the United States,^{7–10} Brazil,¹¹ and Taiwan¹² indicated that short-term increases in particulate matter (PM), organic

carbon, black carbon, and ozone (O_3) air pollution lead to acute but transient increase in arterial BP and hypertension prevalence. However, results from 1 recent study conducted on 9238 nonsmoking adults >30 years of age indicated that short-term exposure to particles with an aerodynamic diameter $\leq 10 \mu\text{m}$ (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxides (NO_2), CO, and O_3 consistently reduced the systolic BP (SBP) and pulse pressure, whereas the diastolic BP (DBP) was increased by SO_2 , NO_2 , and O_3 .¹³ Stronger associations existed among men, participants >60 years of age, those with hypertension, and those living in an industrial township. Also, Mordukhovich et al¹⁴ observed positive associations not between BP and PM, but between BP and black carbon. Another recent controlled human inhalation study reported that exposure to diesel exhaust was associated with a rapid and measureable increase in SBP but not DBP.¹⁵ Furthermore, some authors even reported that there was a small significant decrease in DBP and SBP and hypertension in association with particulate air pollution.¹⁶ The reasons for inconsistent effects of ambient air

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pollution on BP in different parts of the world are unclear but may result from spatial and temporal variability in pollution sources and composition.^{17,18}

Compared with short-term effects of air pollution, there is little information on the relation between chronic exposure to air pollution and prevalent hypertension. Inconsistent results have also been reported on the association between incident hypertension and air pollution. For instance, Coogan et al¹⁹ showed in a 10-year follow-up for incident hypertension and diabetes mellitus of black women from Los Angeles that NO_x (but less PM_{2.5}) borderline increased the risk of becoming hypertensive. However, Sørensen et al²⁰ reported that long-term exposure (1 and 5 years) to NO_x was not associated with incident self-reported hypertension in a Danish cohort. We hypothesized that at the population level, long-term exposure to ambient air pollution is associated with higher BP and higher prevalence of hypertension among humans. In this study, we tested this hypothesis in the 11 Districts Chinese Study, a cross-sectional study of air pollution and adult health in a large, well-characterized population-based sample, residing in northeast China, where there are wide differences in inter- and intracity gradients and ambient pollutant levels, offering a valuable opportunity to assess the associations between exposure and response.

Methods

Study Cities Selection and Subject Recruitment

More than 20 million people reside in 14 cities in Liaoning province in northeast China. To maximize the inter- and intracity gradients of the pollutants of interest and minimize the correlation between district-specific ambient pollutants, in April 2009, we selected 3 cities (Shenyang, Anshan, and Jinzhou) based on the results of air pollution measurements between 2006 and 2008.

There are 5, well-identified geographic districts in Shenyang, and 3 districts in Anshan and Jinzhou, respectively. Three communities within 1 km of air-monitoring sites were randomly chosen from these districts resulting in 33 locales, and from each of these, 700 to 1000 households were randomly identified. One participant, aged 18 to 74 years, was selected from each household without replacement. Our entry criterion was that the subject should have lived at that place for at least 5 years. The design and conduct of this investigation was reviewed and approved by Human Studies Committee of China Medical University. Before data collection, a written informed consent form was obtained from each participant.

Ambient Air Pollution

In each of the selected study districts, there was only 1 municipal air pollution monitoring station, which was located 1 mile from the participants' homes. Measurements of PM₁₀, SO₂, NO₂, and O₃ concentrations from 2006 to 2008 were obtained at the stations. The measurements were subject to uniform criteria for monitoring, siting, instrumentation, and quality assurance. These monitoring stations were mandated to be away from major roads, industrial sources, buildings, or residential sources of emissions from the combustion of coal, waste, or oil so that the measurements from these monitors were more likely to reflect the background air pollution level in a city rather than those from local sources, such as traffic or industrial combustion. These measurements were used to develop surrogates of long-term exposure for the participants. The measurements strictly followed the methodological standards set by the State Environmental Protection Administration of China.²¹ Concentrations of each pollutant were measured continuously and reported hourly; PM₁₀ by β -attenuation, SO₂ by ultraviolet fluorescence, NO₂ by chemiluminescence, and O₃ by ultraviolet photometry.²² We calculated

daily average concentrations of PM₁₀, SO₂, NO₂, and O₃ (8-hour average) based on data from days for which at least 75% of the 1-hour values were available after excluding abnormal values in the hourly data collected from each monitoring station. Exposure parameters in the current study were 3-year average (2006–2008) concentrations calculated from the 24-hour PM₁₀, SO₂, and NO₂ concentrations, and 10:00 AM to 6:00 PM 8-hour O₃ concentrations in each district.

BP Measurement

All investigators and staff of the study were required to successfully complete a training program based on the procedures formulated by the American Heart Association,²³ designed to facilitate the administration of the study questionnaire and a standardized protocol to measure BP. At the end of the training program, each trainee was required to take a qualifying examination and those who passed were given a BP observer certificate. Participants were advised not to smoke, drink alcohol, coffee, or tea, and to abstain from exercising for at least 30 minutes before having their BP measured. BP cuffs came in 4 sizes: large adult cuff, pediatric cuff, regular adult cuff, and thigh cuff. Four BP cuffs were placed on the participant to check for appropriate cuff size. BP was measured 3 times by trained and certified observers, using standardized mercuric-column sphygmomanometer on the participant in a sitting position after 5 minutes of rest, and the time interval between successive pairs of BP measurements was 2 minutes. We followed the procedures recommended by Turner et al²⁴ to calibrate the mercury-column sphygmomanometers in the current study.

Diagnostic Criteria

The hypertension status of the participants was assessed based on the US Seventh Joint National Committee report on the prevention, detection, evaluation, and treatment of high BP.²⁵ Hypertension was defined as an average SBP ≥ 140 mmHg, or an average DBP ≥ 90 mmHg. Participants who self-reported current treatment for hypertension with antihypertensive medication within the 2 weeks before the interview were also classified as hypertensive. This definition specifically excludes those hypertensive patients whose BP has been reduced to a nonhypertensive range solely by the use of nonpharmacological measures.

Statistical Analysis

Data were tested for normality (using Shapiro–Wilks W test) and homogeneity (using Bartlett test for unequal variances). For each group, the values of mean \pm SD were calculated for continuous variables, and relative frequencies were calculated for categorical variables. Contingency tables and χ^2 test were used to calculate the association between categorical variables. We assessed the association of ambient air pollutants with BP using generalized additive models. To investigate the relation between hypertension and ambient air pollution, we considered a 2-level binary logistic regression model with participants being the first-level units and districts being the second-level units. At the participant level, we modeled the logit of the prevalence of hypertension as a function of k covariates ($X_1 \dots X_k$) as follows:

$$\text{logit}[\text{Probability}(Y_{ij})] = \alpha_j + \beta_1 X_{1ij} + \dots + \beta_k X_{kij} + e_{ij} \quad (1)$$

where the outcome variable (Y) is hypertension based on the BP classification described earlier, the subscript j identifies the district ($j=1, \dots, 25$), the subscript i identifies the participant ($i=1, \dots, n_j$), α_j are the intercepts at the district level, $\beta_1 \dots \beta_k$ are the regression coefficients of the covariates, and e_{ij} are the random errors, assumed to follow a normal distribution with mean of zero and constant variance. The α_j are assumed to vary across districts and so are random coefficients. In general, a district with a higher α_j is predicted to have higher prevalence rates than a district with a lower α_j .

At the district level, we regressed the district-specific intercepts α_j on the district-specific pollutant level (Z_j) to explain the variations of α_j , as follows:

$$\alpha_j = \alpha + \gamma_1 Z_j + u_j \quad (2)$$

Equation 2 predicts the prevalence in a district by Z_j . If γ_j is positive, then adjusting for covariates, the overweight and obesity prevalence are higher in districts with a higher pollutant level. Conversely, if γ_j is negative, then adjusting for covariates, the prevalence is lower in districts with a higher pollutant level. The u_j -terms are random errors at the district level, assumed to be independent and follow a normal distribution with mean of zero and constant variance. These random errors characterize the between-district variation and are assumed to be independent from e_{ij} at the participant level. Note that α , β_1, \dots, β_k , and γ_1 are not assumed to vary across districts. They, therefore, have no subscript j to indicate to which district they belong. They are referred to as fixed effects as they apply to all districts.

The above models can be written as a single regression equation by substituting equation 2 into equation 1:

$$\text{logit}[P(Y_{ij})] = (\alpha + \gamma_1 Z_j + \beta_1 X_{1ij} + \dots + \beta_k X_{kij}) + (u_j + e_{ij}) \quad (3)$$

The terms in the first and second parentheses in equation 3 are often called the fixed (or deterministic) and random (or stochastic) parts of the model, respectively.

All analyses were conducted using the GLIMMIX procedure in SAS 9.2. Statistical significance was assessed using a 2-sided test at the 5% level of significance.

Results

There were a total of 28 830 individuals randomly selected from 33 communities of the 3 cities, of whom 24 845 completed the survey and examination to give an overall response rate of 86.2%. Among the 24 845 participants analyzed, the average age was 45.59 years (SD =13.31 years) and 12 661 (50.1%) were men. For all participants, the prevalence rate of hypertension was 34.84%. The characteristics of the survey participants in this study, stratified by sex, are shown in Table 1. Men and women were found to be statistically different across all demographical and behavioral variables except age and race.

Table 2 presents the arithmetic mean for all 4 pollutants PM_{10} , NO_2 , SO_2 , and O_3 measured between 2006 and 2008, and compares them with the World Health Organization (WHO) guidelines and Chinese National Ambient Air Quality Standards. The table shows that PM_{10} and SO_2 levels exceed WHO guidelines in all districts, whereas 90.9% of all districts exceed PM_{10} levels and 27.3% of all districts exceed SO_2 levels dictated by the Chinese National Ambient Air Quality Standards; 18.2% of all districts exceed WHO guidelines for NO_2 . Additionally, 18.2% of all 11 Chinese districts exceed NO_2 levels dictated by both WHO guidelines and those of the Chinese National Ambient Air Quality Standards. O_3 levels in all districts were below the guidelines set by the WHO and those of the Chinese National Ambient Air Quality Standards.

A GLIMMIX model was used to assess the association between air pollution and hypertension. The results are displayed in Table 3. For all participants, we observed significant associations between prevalence rate of hypertension and PM_{10} (odds ratio [OR], 1.12; 95% confidence interval [CI], 1.08–1.16), SO_2 (OR, 1.11; 95% CI, 1.04–1.18), and O_3 (OR, 1.13; 95% CI, 1.06–1.20). However, when stratified by sex, these relations were obtained only in men, and none of the pollutants was found to be associated with prevalence of hypertension among women. For instance, in the single pollutant model analysis, positive associations were identified for hypertension with interquartile range (IQR) of PM_{10} (OR, 1.17; 95% CI, 1.06–1.30), SO_2 (OR, 1.19; 95% CI, 1.05–1.34),

Table 1. Characteristics of the Study Population in the 33 Communities of 3 Cities, China

Characteristics	Men (n=12 661)	Women (n=12 184)	Total (n=24 845)
Age, y (mean±SD)	45.35 (±14.12)	45.85 (±12.40)	45.59 (±13.31)
Race			
Han nationality	11 945 (94.34)	11 525 (94.59)	23 470 (94.47)
Other nationality	716 (5.66)	659 (5.41)	1375 (5.53)
Education*			
Junior college or higher	3405 (26.89)	2070 (16.99)	5475 (22.04)
Middle school	7694 (60.77)	7239 (59.41)	14 933 (60.10)
Primary school	1334 (10.54)	2112 (17.33)	3446 (13.87)
No school	228 (1.80)	763 (6.26)	991 (3.99)
Income*			
≤5000 RMB†	1117 (8.82)	1107 (9.09)	2224 (8.95)
5000–10 000 RMB†	1461 (11.54)	2076 (17.04)	3537 (14.24)
10 000–30 000 RMB†	6296 (49.73)	6052 (49.67)	12 348 (49.70)
>30 000 RMB†	3787 (29.91)	2949 (24.20)	6736 (27.11)
Smoking status*			
Nonsmoker	6171 (48.74)	11 372 (93.34)	17 543 (70.61)
Smoker	6490 (51.26)	812 (6.66)	7302 (29.39)
Drinking status*			
Nondrinking	7235 (57.14)	11 847 (97.23)	19 082 (76.80)
Drinking	5426 (42.86)	337 (2.77)	5763 (23.20)
Exercise*			
No	8506 (67.18)	8692 (71.34)	17 198 (69.22)
Yes	4155 (32.82)	3492 (28.66)	7647 (30.78)
Diet*			
Never	7740 (61.13)	6726 (55.20)	14 466 (58.22)
Almost never	1275 (10.07)	1203 (9.87)	2478 (9.97)
Occasionally	781 (6.17)	932 (7.65)	1173 (6.89)
Frequently	2270 (17.93)	2637 (21.64)	4907 (19.75)
Every day	595 (4.70)	686 (5.63)	1281 (5.16)
Sugar*			
≥5 days per week	560 (4.42)	372 (3.05)	932 (3.75)
2–4 days per week	1211 (9.56)	763 (6.26)	1974 (7.95)
<1 day per week	10 890 (86.01)	11 049 (90.68)	21 939 (88.30)
Family history of hypertension*			
No	8516 (67.26)	7096 (58.24)	15 612 (62.84)
Yes	4145 (32.74)	5088 (41.76)	9233 (37.16)
Hypertension*			
No	7517 (59.37)	8671 (71.17)	16 188 (65.16)
Yes	5144 (40.63)	3513 (28.83)	8657 (34.84)

*Significant difference exists between men and women by χ^2 test, $P < 0.05$.

†RMB, Chinese Yuan.

and O_3 (OR, 1.21; 95% CI, 1.04–1.38) only in men, but not in women. We found no association of NO_2 with prevalence of hypertension in either men or women.

We also found significant associations of long-term exposure of ambient air pollutants with arterial BP (Table 4). For all subjects, all the ambient air pollutants except for NO_2 were significantly associated with SBP and DBP levels. When

Table 2. Air Pollution Levels in the 11 Districts of 3 Cities, China, From 2006 to 2009 (mg/m3)

Air Pollution Levels	PM ₁₀	SO ₂	NO ₂	O ₃
Mean (±SD)	123.06 (±14.61)	54.42 (±14.33)	35.28 (±5.45)	49.4 (±14.07)
Median	123	48	33	50
Minimum	93	36	27	27
Maximum	145	78	45	71
Interquartile range*	19	20	9	22
NS†	100	60	40	160
% of > NS	90.9	27.3	18.2	0.0
WHO guideline‡	20	20	40	100
% of > WHO	100.0	100.0	18.2	0.0

NO₂ indicates nitrogen dioxide; NS, national standard; O₃, ozone; PM₁₀, particle with aerodynamic diameter ≤10 μm; SO₂, sulfur dioxide; and WHO, World Health Organization.

*Interquartile range (IQR): range from 25th to 75th percentile of district-specific concentrations.

†China national ambient air quality standard.

‡WHO 2005 air quality guidelines.

stratified by sex, these significant associations were limited to men. For example, in the single pollutant model, DBP and SBP increased by 0.44 mmHg (95% CI, 0.10–0.78 mmHg) and 0.98 mmHg (95% CI, 0.45–1.52 mmHg) per 19 μg/m³ increase in 3-year mean PM₁₀, 0.50 mmHg (95% CI, 0.20–0.79) and 1.13 mmHg (95% CI, 0.67–1.60 mmHg) per 20 μg/m³ increase in 3-year mean SO₂, 0.58 mmHg (95% CI, 0.24–0.91 mmHg) and 1.05 mmHg (95% CI, 0.52–1.58 mmHg) per 22 μg/m³ increase in 3-year mean O₃ in men but not in women, respectively. The estimated ORs varied by age group (Table 5). The ORs among participants ≥65 years of age seemed larger than those in the other 2 age groups with an exception of NO₂. The insignificant effects in the ≥65 years of age group might be attributable to the smaller sample size (2234) compared with the sample size (18698) in the <55 years of age group.

Discussion

The findings confirmed our hypothesis of the presence of an adverse association between long-term ambient air pollution and hypertension, and more generally a positive association with arterial SBP and DBP. These effects were seen with certain pollutants (PM₁₀, SO₂, and O₃) and observed only in men, suggesting that sex may be an effect modifier.

Table 3. Adjusted OR and 95% CI for the Prevalence of Hypertension of Long-term Exposure to the Air Pollutant (n=24 845)*

Pollutant	Men		Women		Total	
	OR†	95% CI	OR†	95% CI	OR†	95% CI
PM ₁₀ , μg/m ³	1.17	1.06–1.30	1.06	0.95–1.18	1.12	1.08–1.16
SO ₂ , μg/m ³	1.19	1.05–1.34	1.03	0.91–1.89	1.11	1.04–1.18
NO ₂ , μg/m ³	1.09	0.93–1.27	1.10	0.94–1.29	1.09	1.00–1.20
O ₃ , μg/m ³	1.21	1.04–1.38	1.07	0.91–1.18	1.13	1.06–1.20

CI indicates confidence interval; NO₂, nitrogen dioxide; O₃, ozone; OR, odds ratio; PM₁₀, particle with aerodynamic diameter ≤10 μm; and SO₂, sulfur dioxide.

*Adjusted for age, race, education, income, smoke, drink, exercise, diet, sugar, family history of hypertension, and district.

†OR were scaled to the interquartile range (IQR) for each pollutant (19 μg/m³ for PM₁₀, 20 μg/m³ for SO₂, 9 μg/m³ for NO₂, and 22 μg/m³ for O₃).

In contrast to an abundance of data demonstrating associations between short-term exposure to ambient air pollution and arterial BP, there is little published information on the relation between long-term exposure to ambient pollutants and measured BP or frank hypertension. We are aware of only 3 relevant human studies examining the effect of long-term air pollutants exposure on prevalence of hypertension and arterial BP.^{12,26,27} The results of these studies are inconsistent. In the Heinz Nixdorf Recall Study, a population-based prospective cohort in Germany, Fuks et al²⁷ investigated the cross-sectional association of residential long-term PM_{2.5} and PM₁₀ exposure with arterial BP and hypertension on 4291 participants aged 45 to 75 years, taking short-term variations of PM and long-term road traffic noise exposure into account. The results indicated that an IQR increase in PM₁₀ (3.9 μg/m³) was associated with estimated increase in mean SBP and DBP of 1.1 mmHg (95% CI, 0.2–2.0 mmHg) and 0.8 mmHg (95% CI, 0.3–1.2 mmHg), respectively. However, no association of PM with prevalence of hypertension was found.²⁷ Using the Taiwanese Survey on Prevalence of Hyperglycemia, Hyperlipidemia, and Hypertension study data, Chuang et al¹² conducted secondary analyses to investigate changes in BP associated with changes in exposure to ambient air pollutant by applying generalized additive models among 7578 participants aged 16 to 90 years. The results of that study showed no significant relation between air pollution and BP except for O₃, which revealed a 0.37 mmHg (95% CI, 0.04–0.69 mmHg) elevation for an IQR increase of 12.15 ppb. However, analysis of data gathered in Taiwan since 1989 among 1023 participants in Social Environment and Biomarkers of Aging Study demonstrated significant increase in BP for an IQR increase in 1-year average air pollution, where per IQR change in PM₁₀ of 48 μg/m³, SBP increased by 16.34 mmHg (95% CI, 12.27–20.42 mmHg) and DBP increased by 14.87 mmHg (95% CI, 12.73–17.02 mmHg).²⁶ Compared with these investigations, air pollutants in the current study included not only PM but also other air pollutants, such as SO₂, NO₂, and O₃, and furthermore, our study observed significant relation among PM₁₀, SO₂, and O₃ and the prevalence of hypertension. The findings in our study may be explained by the differences in spatial and temporal variability of air pollutant sources and components between

Table 4. Estimated Absolute Increase in Arterial Blood Pressure (mm Hg) With 95% CI per Interquartile Range of Long-Term Exposure to the Air Pollutant (n=24 845)*

Pollutant, $\mu\text{g}/\text{m}^3$	Men		Women		Total	
	Estimate†	95% CI	Estimate†	95% CI	Estimate†	95% CI
Diastolic blood pressure						
PM ₁₀	0.44	0.10–0.78	0.08	–0.25 to 0.40	0.32	0.08–0.56
SO ₂	0.50	0.20–0.79	0.01	–0.28 to 0.29	0.31	0.10–0.51
NO ₂	–0.02	–0.41 to 0.36	0.35	–0.02 to 0.73	0.22	–0.05 to 0.49
O ₃	0.58	0.24–0.91	0.02	–0.29 to 0.34	0.37	0.14–0.61
Systolic blood pressure						
PM ₁₀	0.98	0.45–1.52	0.45	–0.11 to 1.00	0.87	0.48–1.27
SO ₂	1.13	0.670–1.60	0.15	–0.33 to 0.63	0.80	0.46–1.14
NO ₂	–0.17	–0.78 to 0.43	0.50	–0.13 to 1.14	0.23	–0.21–0.68
O ₃	1.05	0.52–1.58	0.04	–0.50 to 0.58	0.73	0.35–1.11

CI indicates confidence interval; NO₂, nitrogen dioxide; O₃, ozone; PM₁₀, particle with aerodynamic diameter $\leq 10 \mu\text{m}$; and SO₂, sulfur dioxide.

*Adjusted for age, race, education, income, smoke, drink, exercise, diet, sugar, family history of hypertension, and district.

†Estimate was scaled to the interquartile range for each pollutant (19 $\mu\text{g}/\text{m}^3$ for PM₁₀, 20 $\mu\text{g}/\text{m}^3$ for SO₂, 9 $\mu\text{g}/\text{m}^3$ for NO₂, and 22 $\mu\text{g}/\text{m}^3$ for O₃).

the different regions, difference in the age, ethnicity, and lifestyle of the study populations, differences in exposure assessment, and so on.

Interpretation of our findings requires caution. First, we only observed marginally significant associations between NO₂ and BP and prevalence of hypertension, which is different from those reported from recent studies. Sørensen et al²⁰ reported that long-term exposure of NO_x was associated with a lower prevalence of baseline self-reported hypertension. The authors used nitrogen oxides (NO_x) as ([NO] + [NO₂]) because NO_x is a more constant and stable measure of exposure. The differences in the reported results might be attributable to the different markers of air pollution exposure. Compared with NO_x, NO₂ is not stable, which may lead to significant exposure uncertainty. In our study, ORs for PM₁₀, SO₂, and O₃ in men were very comparable. NO₂ was the only exception. The correlation analyses show that PM₁₀, SO₂, and O₃ are significantly, highly correlated with each other, whereas the correlations between NO₂ and PM₁₀, SO₂, and O₃ are much weaker. The stronger correlations among PM₁₀, SO₂, and O₃ would lead to much more similar impacts on the outcomes

than NO₂. In addition, there remains some doubt whether O₃ increases SBP and prevalent hypertension and even more whether O₃ increases DBP. O₃ concentrations are highly correlated with temperature. Hoffmann et al²⁸ observed that O₃ and higher temperature were associated with decreases in SBP in a study of the short-term effects of ambient air pollution and temperature on arterial BP in 70 people with type 2 diabetes mellitus. Although the combined effects of these 2 factors are not clear, the effect of O₃ exposure on SBP in the study might be masked by an independent temperature effect on SBP. Weinhold²⁹ also speculated that the opposing effects of air pollutants and heat exposures could plausibly exist in independent ways and might not cancel one another out. Furthermore, the current study is a cross-sectional study of associations between air pollution and prevalence of BP and hypertension. We could not address temporal relation between them. Sørensen et al²⁰ assessed the associations between incidence of hypertension and long-term air pollution. The authors did not observe the associations between long-term exposure to air pollution and incident hypertension. The different study designs (prevalence versus incidence) might be the reason for the difference in observed results, in addition to the differences in sociodemographic variables, lifestyle indicators, healthy history, health care access, prescription drug use, tobacco smoke, and season.

The mechanisms by which the constituents of ambient air pollution may elevate or modify BP are not yet understood. Possible causal pathways include the elicitation of oxidative stress, systemic proinflammatory responses and the activation of pulmonary reflexes in turn leading to arterial remodeling.^{26,30–33} Acute effects of PM on BP may occur via activation of pulmonary reflexes that decrease parasympathetic tone while increasing sympathetic activity in the autonomic nervous system, whereas later-onset effects of PM are probably mediated via inflammation-induced vascular reactivity.³⁰ Other investigators have also reported a link between inflammatory markers (reflected by percent neutrophils and interleukin-6) and elevated BP after long-term air pollution exposure.²⁶ Elevations in fasting glucose, ApoB, and HbA1C

Table 5. Adjusted OR and 95% CI for the Prevalence of Hypertension per Interquartile Range of Long-Term Exposure to Air Pollutant, by Age*

Pollutant	Age <55, y (n=18 698)		55≤Age<65, y (n=3913)		Age≥ 65, y (n=2234)	
	OR†	95% CI	OR†	95% CI	OR†	95% CI
PM ₁₀ , $\mu\text{g}/\text{m}^3$	1.12	1.07–1.17	1.05	0.96–1.14	1.14	0.99–1.32
SO ₂ , $\mu\text{g}/\text{m}^3$	1.10	1.04–1.17	1.04	0.95–1.15	1.17	0.99–1.38
NO ₂ , $\mu\text{g}/\text{m}^3$	1.10	1.02–1.20	1.05	0.94–1.17	1.04	0.84–1.29
O ₃ , $\mu\text{g}/\text{m}^3$	1.13	1.07–1.20	1.02	0.92–1.14	1.15	0.96–1.39

CI indicates confidence interval; NO₂, nitrogen dioxide; O₃, ozone; OR, odds ratio; PM₁₀, particle with aerodynamic diameter $\leq 10 \mu\text{m}$; and SO₂, sulfur dioxide.

*Adjusted for age, race, education, income, smoke, drink, exercise, diet, sugar, family history of hypertension, and district.

†OR were scaled to the interquartile range (IQR) for each pollutant (19 $\mu\text{g}/\text{m}^3$ for PM₁₀, 20 $\mu\text{g}/\text{m}^3$ for SO₂, 9 $\mu\text{g}/\text{m}^3$ for NO₂, and 22 $\mu\text{g}/\text{m}^3$ for O₃).

were associated with progression of atherosclerosis and mortality attributable to cardiovascular disease.

Other investigators have also reported a link between long-term air pollution exposure and elevations in fasting glucose, ApoB, and HbA1C—all associated with progression of atherosclerosis and mortality attributable to cardiovascular disease—and immune system activation marked by increased levels of percent circulating neutrophils and interleukin-6, thought to contribute to acute coronary syndromes.^{6, 26} In smaller vessels in the arterial tree, raised intravascular pressure increases wall stress, stimulating the activation of medial smooth muscle in arterioles and subsequent hypertrophic remodeling of resistance vessels potentiating a rise in BP as a result of increased total peripheral resistance.³¹

There is growing epidemiological evidence of the differing associations between air pollution and health for men and women, but the literature is far from consistent. Our study showed that the effect of ambient air pollution exposure on BP and prevalence rates of hypertension was evident only in men. These findings are not entirely unexpected as men may have greater pulmonary exposure to ambient air pollutants because they engage in more frequent or intense outdoor activity than women.^{34–36}

Finally, some limitations of the current study should be noted. First, the findings cannot establish a cause-and-effect relation among long-term ambient air pollution, BP, and hypertension because of the nature of a cross-sectional study design. We do not have any baseline readings available, nor could any temporality in the association be ascertained. Selection and information bias are possible. However, we have attempted to reduce these biases as much as possible through strict quality control and assurance procedures. Second, possible misclassification of the behavioral risk factors (smoking, drinking, and exercising) could exist because of the nature of the survey questions (yes or no). Third, we develop a variable of 3-year average concentrations to serve as surrogates for the long-term exposure to PM₁₀, SO₂, NO₂, and O₃. As stated in the method section, the measurements are more likely to reflect the background air pollution levels in a city. Traffic-related air pollution exposure data are not available to this study. This approach may lead to significant uncertainty of long-term exposure. Detailed spatial and temporal information is needed to develop a better long-term exposure. Addressing spatial variations of traffic-related air pollution effects would be one of the future directions. Fourth, there is still some doubt on whether the described associations between air pollutants and SBP or hypertension are causal. Exposure to PM_{2.5} and NOx does not infrequently go together with noise, and exposure to noise (especially traffic noise) has also been incriminated as a causal factor for incident hypertension. Therefore, it would be ideal to adjust for noise when air pollution effects on SBP or hypertension are assessed. Unfortunately, no noise data are available to this study. Nevertheless, these factors are not likely to vary significantly across the 7 studied cities because these are big cities in the northeast China, where city landscapes, traffic density and patterns, and time activity patterns are quite similar. Finally, we have found a strong intercorrelation between the 3 pollutants PM₁₀, SO₂, and O₃ and a moderate correlation among NO₂, PM₁₀, and O₃. Therefore, it would

be inappropriate to fit a model including all 4 pollutants. We are not able to assess multiple pollutants' effect simultaneously in this study.

Perspectives

In this current study, we found an association of long-term exposure to PM, SO₂, and O₃ with increased arterial BP and established hypertension in a population-based sample. This finding supports the belief that long-term ambient air pollution exposure may promote atherosclerosis and other arterial damage with air-pollution-induced increases in BP being one possible biological pathway. However, the mechanisms by which the constituents of ambient air pollution may elevate or modify BP needs to be investigated in prospective studies.

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Disclosures

None.

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Novelty and Significance

What Is New?

- This is a prospective study of air pollution and adult health in a large, well-characterized population-based sample, residing in northeast China, where there are wide differences in inter- and intracity gradients and ambient pollutant levels, offering a valuable opportunity to assess the associations between exposure and response.

What Is Relevant?

- There is a significant association of long-term exposure to ambient air pollutants with increased arterial blood pressure and established hypertension in adults.

Summary

Long-term exposure to air pollutants was associated with increased arterial blood pressure and hypertension in men but not in women.

Association Between Long-Term Air Pollution and Increased Blood Pressure and Hypertension in China

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