AMBIENT AIR POLLUTION AS A MEDIATOR IN THE PATHWAY LINKING RACE/ETHNICITY TO HYPERTENSION: THE MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS (MESA)

by

Lanxin Song

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ABSTRACT

Background: Racial/ethnic disparities in blood pressure and hypertension have been evident in previous studies, as were associations between race/ethnicity with ambient air pollution and those between air pollution with hypertension, which suggests that air pollution may have mediating effects linking race/ethnicity to hypertension.

Objective: To assess the potential mediating effects of ambient air pollution on the association between race/ethnicity and blood pressure/hypertension.

Methods: We studied 6,463 White, Black, Hispanic and Chinese adults enrolled between 2000 and 2002 across 6 US cities. Systolic (SBP) and diastolic blood pressure (DBP) were measured at Exam 1 (2000-2002) and Exam 2 (2002-2004). Household-level annual average concentrations of fine particulate matter ($PM_{2.5}$), oxides of nitrogen (NO_X), and ozone(O_3) for the year 2000 were estimated for participants.

Results: The difference in SBP levels by race/ethnicity that was related to higher $PM_{2.5}$ concentrations compared with White men ("indirect associations") was 0.3 (95% CI: 0.1, 0.5) mmHg for Black men, 0.3 (95% CI: 0.1, 0.6) mmHg for Hispanic men and 1.0 (95% CI: 0.2, 1.8) mmHg for Chinese men. Findings were similar although not statistically significant for women. $PM_{2.5}$ did not mediate racial/ethnic differences in DBP. Indirect associations were significant for ozone for both SBP and DBP among men and women. In contrast, racial/ethnic disparities were attenuated due to exposure to NO_X. Associations with blood pressure levels were stronger among participants with hypertension. Among the 3,089 participants without hypertension at baseline, 422 developed incident hypertension. For racial/ethnic disparities in incident hypertension, only indirect associations for ozone among men were marginally significant.

Conclusion: Racial disparities in blood pressure were reduced after accounting for $PM_{2.5}$ and ozone while increased after accounting for NO_X, but mediating effects of air pollution on the pathway linking race/ethnicity to incident hypertension were barely found.

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Table of Contents

List of Tables

INTRODUCTION

Hypertension is a highly prevalent condition relevant to many cardiovascular events in the United States[1,2]. Racial/ethnic differences in hypertension prevalence and risk, have been shown in various studies[3-6] even after controlling for clinical risk factors of hypertension. In the National Health and Nutrition Examination Survey (NHANES, 21,489 adults aged >20 years)[3], non-Hispanic Blacks had 90% higher odds of poorly controlled blood pressure compared to non-Hispanic Whites after adjustment for sociodemographic and clinical characteristics ($p<0.001$), and among hypertensive subjects, non-Hispanic Blacks and Mexican-Americans had 40% higher odds of uncontrolled blood pressure compared to non-Hispanic Whites after adjustment for sociodemographic and clinical characteristics ($p <0.001$). The Multi-Ethnic Study of Atherosclerosis (MESA, 6,814 adults aged 45-84 years) [4] found that after adjustment for age, body mass index, prevalence of diabetes mellitus, and smoking, African American and Chinese race/ethnicity were significantly associated with hypertension compared to Whites. The Chicago Area Sleep Study (CASS, 494 adults aged 35-64 years)[5], a cross-sectional study randomly sampling Whites, Blacks, Hispanics and Chinese from Chicago, found that the prevalence of hypertension was highest in Blacks (36%), followed by Hispanics (14%), Asians (8%), and Whites (5%). Similarly among 1,476 adults aged ≥20 years who participated in the 2013–2014 New York City Health and Nutrition Examination Survey, non-Hispanic Black and Asian adults had higher odds of hypertension than non-Hispanic Whites[6]. Various factors have been suggested to account for the racial/ethnic disparities in hypertension, including genetic predisposition, awareness and management, obesity, sleep characteristics, SES, smoking, alcohol, social support, and chronic stress[5,7-12]. Besides these factors, air pollution exposure was indicated to be of importance, while still under-explored.[5,13-15]

Previous studies have accorded importance to the role of fine particulate matter (particles< 2.5μm in aerodynamic diameter [$PM_{2.5}$]) and nitrogen oxides (NO_X) in the development of hypertension, and there is a paucity of evidence that exposure to ozone (O_3) also elevate blood

pressure[16-23]. In a 2014 meta-analysis, exposure to $PM_{2.5}$ was positively associated with blood pressure levels (Combined mean difference per 10 μg/m3 increase in $PM_{2.5}$: 1.393 mmHg [95% CI: 0.874, 1.912] for systolic blood pressure and 0.895 mmHg [95% CI: 0.49, 1.299] for diastolic blood pressure); with the strongest associations with long-term exposure[19]. In a study of 4,291 adults 45-75 years of age from the Heinz Nixdorf Recall Study, a population-based cohort in Germany, long-term exposure to fine particulate matter ($PM_{2.5}$) was associated with increased blood pressure levels[20]. A study of White, Black and Hispanic adults ≥30 years of age who participated in the 1999-2005 National Health Interview Survey found that $PM_{2.5}$ was associated with higher odds of prevalent self-reported hypertension (OR: 1.05 [95% CI: 1.00, 1.10] for a 10 μg/m3 increase); this association differed by race/ethnicity and was statistically significant in White but not Black or Hispanic adults[22]. In a cohort of 33,771 Black women in the Black Women's Health Study (BWHS), long-term exposure to ozone was associated with increased hypertension incidence[18] . Among Black women in the BWHS in Los Angeles, annual exposure to ambient $PM_{2.5}$ and NOx was associated with increased risk of incident hypertension (Incidence rate ratio was 1.48 [95% CI: 0.95, 2.31] for a 10 μg/m3 increase in $PM_{2.5}$ and 1.14 [95% CI: 1.03, 1.25] for an interquartile range (12.4 parts per billion) increase in NOx[17] .

Exposure to air pollution has been shown to differ by race/ethnicity[24-26]. In MESA, living in majority White neighborhoods was associated with lower air pollution exposures, and living in majority Hispanic neighborhoods was associated with higher air pollution exposures.[26] A study of 151,709 children in Orange County, Florida indicated a consistent pattern of racial inequity in the spatial distribution of air pollution sources, with Black children facing the highest relative levels of potential exposure at both school and home locations.[27] There is an absence of published studies that have examined the mediating effect of air pollution on the relationship between race/ethnicity and hypertension. Therefore, the objective of this study was to examine the extent that exposure to air pollutants ($PM_{2.5}$, NO_X and O_3) contribute to racial/ethnic differences in blood pressure levels and incident

hypertension.

METHODS

Study Population

Between 2000 and 2002, the Multi-Ethnic Study of Atherosclerosis (MESA), a prospective cohort study of cardiovascular risk factors and subclinical atherosclerosis enrolled 6,814 participants aged 45-84 years who self-reported their racial/ethnic group as white or Caucasian, black or Black, Chinese, or Spanish/Hispanic/Latino, from 6 major metropolitan areas—Los Angeles, CA, St. Paul, MN, Chicago, IL, New York, NY, Baltimore, MD and Winston-Salem, NC.[28] Racial/ethnic composition of participants differed across the six study sites. Whites were enrolled in all six sites, Blacks were enrolled in all sites except for St. Paul, Hispanics were only enrolled in New York, Los Angeles, and St. Paul, and Chinese were only enrolled in Chicago and Los Angeles. From the 6,814 participants, we excluded 258 participants without information on exposure to air pollution ($PM_{2.5}$, ozone and NO_X) at baseline, 3 without measures of systolic and diastolic blood pressure at baseline, 5 participants who were enrolled from sites with few participants of the same race/ethnicity and 85 participants with missing data on potential covariates, leaving 6,463 participants for this analysis. To assess the development of incident hypertension, we further excluded 3,374 participants without blood pressure measures at Exam 2 (2002- 2004) or having hypertension at baseline.

Demographics

Participant race/ethnicity was assessed by self-report and then categorized as non-Hispanic White ("White"), non-Hispanic Black ("Black"), Chinese, and Hispanic. Self-reported educational attainment and annual family income at baseline were used as primary measures of socioeconomic status (SES). Participant education was measured as the highest level completed and categorized as less than high school, high school, some college/technical school, and college/graduate degree.

Annual household income was collected in 13 categories, and was further categorized into unknown, less than $24,999, $25,000–$49,999, $50,000–$74,999, and $75,000 and greater for our analyses.

Ambient Air Pollution Concentrations

Concentrations of $PM_{2.5}$, NO_X and O_3 from the MESA Air study along with national monitoring data from US Environmental Protection Agency (EPA) Air Quality System (AQS) were integrated into hierarchical spatiotemporal models to predict annual average long-term exposure at each participant residence for the year 2000. Details of the MESA Air monitoring campaign and adopted spatiotemporal models were described elsewhere.[29-32] In summary, for each participant, $PM_{2.5}$, NO_X were measured twice at the neighborhood, household and individual levels for two-week periods within 18 months after joining in the study. $PM_{2.5}$ was measured on Harvard Personal Environmental Monitor impactors using Teflon filters, and Ogawa passive samplers were used to measure NOx, and O_3 using ion chromatography and ultraviolet spectroscopy. In most of the regions, ozone data were collected in warm seasons between April and September. In Los Angeles, ozone data were continuously recorded throughout the entire study period at all monitoring sites. AQS stations that contained data for less than two years were excluded in order to get reliable estimate of main time trends by the spatiotemporal model. Ambient air pollution exposure was characterized using annual average concentrations of $PM_{2.5}$, NO_X and O_3 for the year 2000 that were estimated for each participant based on the location(s) lived in during that year. Concentrations were right-skewed and log-transformed in our analyses.

Blood Pressure Levels and Incident Hypertension

At each exam visit, resting systolic and diastolic blood pressure was measured in the right arm after five minutes in the seated position. An automated oscillometric method (Dinamap) and appropriate cuff size were used. Three readings were taken; the second and third readings were averaged to

characterize blood pressure levels in analyses.

At each study visit, hypertension was defined as systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg, according to JNC-VII,[33] or the use of any antihypertensive medication. Among participants without hypertension at baseline and with complete information at Exam 2 (N=3,089), we further defined incident hypertension as hypertension at Exam 2.

Other Variables

Cardiovascular disease risk factors collected at the baseline exam included cigarette smoking, alcohol intake, diabetes status, body mass index (BMI), waist circumference, cholesterol and medication use (statins and antihypertensives) . Cigarette smoking was categorized as current, former or never. Alcohol intake was categorized into never, former, and current drinkers. Statin use was dichotomized as currently using and not using. Diabetes status was classified as normal (fasting glucose <100 mg/dL), impaired (fasting glucose 100–125 mg/dL) or diabetes (fasting glucose ≥126 mg/dL with or without hypoglycaemic medication use). BMI (kg/m^2) was calculated using measured height and weight at baseline examination. Waist circumference was measured at the umbilicus using a Gullick II 150 cm anthropometric steel measuring tape with standard 4-ounce tension. Cholesterol levels were obtained from fasting blood measurements of total and high-density lipoprotein (HDL). Physical activity was assessed using the MESA Typical Week Physical Activity Survey adapted from the Cross-Cultural Activity Participation Study[34] and contained 28 detailed questions on time and frequency of activities during a typical week in the previous month. The total minutes of moderate and vigorous exercise during a typical week were estimated from the questionnaire and physical activity was categorized as poor (no exercise), intermediate (1-149 min of moderate exercise or 1-74 min of vigorous exercise per week) or ideal (≥150 min of moderate exercise or ≥75 min of vigorous exercise per week)[35,36].

Statistical Analysis

As suggested by previous studies[3,9,37,38], racial/ethnic differences in hypertension outcomes differ for men and women, therefore all analyses were stratified by sex.

To assess the extent that ambient air pollution concentrations explain racial/ethnic differences in systolic and diastolic blood pressure levels, we used generalized structural equation modeling (GSEM) to examine the association between race/ethnic and blood pressure levels ("total association"), the effect of race/ethnicity on blood pressure levels after adjusting for ambient air pollution concentrations ("direct association"), and the extent that concentrations of air pollutants explain racial/ethnic differences in blood pressure levels ("indirect association"),separately for men and women. Two equations within each GSEM model were specified: 1) ambient air pollution concentrations (log-transformed) were modeled as a function of race/ethnicity (comparing Black, Chinese, Hispanic, and White participants with Whites as the reference group), age, BMI, waist circumference, total cholesterol, HDL cholesterol, education, household income, antihypertensive medication use (dichotomous), statin use, diabetes, alcohol intakes (categorical), smoking status(categorical) and physical activity (categorical); 2) systolic and diastolic blood pressure levels were modeled as a function of race/ethnicity, adjusted for ambient air pollution exposure and all covariates as above.

The estimated indirect association, namely the mediation effect of interest, is defined as the mean difference in blood pressure levels in Blacks, Chinese and Hispanics compared to Whites that is related to racial/ethnic differences in air pollution exposures that are in turn associated with blood pressure levels. Point estimates and 95% confidence intervals for all the total and indirect association were estimated by nonlinear combinations of estimators following GSEM. Additionally, stratification by hypertensive status at baseline was applied to examine whether hypertension status could be an effect measure modifier in the association between race/ethnicity, air pollution exposure and blood pressure levels.

To examine the extent that concentrations of air pollutants explain racial/ethnic differences in the development of incident hypertension among participants without hypertension at baseline (N=3,089), we used the GSEM approach with the second equation specified as a logistic regression sub-model modeling incident hypertension on race/ethnicity, and conducted the same adjustment as above, except for antihypertensive medication use.

Participant recruitment was not consistent by race/ethnicity at each study site. To evaluate the potential influence of study site, we conducted sensitivity analyses further adjusting all GSEM models for study sites.

All statistical analyses were performed using STATA version 14.0. All statistical tests were 2-sided and confidence intervals were set at 95%.

RESULTS

Participant characteristics

Characteristics of study participants at baseline are shown in Table 1. Among a total of 6,463 participants, 38.9% were White, 27.7% were Black, 21.9% were Hispanic, and 11.4% were Chi. 52.8% of participants were women, 47.2% were men. Participant mean age within each race/ethnicity and gender stratum varied no more than 1 year from the overall mean age of 62 years. Educational attainment and family income were higher among Whites and lower among Hispanics. BMI was highest among Hispanics and Blacks, lowest among Chinese. Physical activity was highest among Whites and lowest among Hispanics. Smoking was highest among Blacks and lowest among Chinese. Alcohol intake was highest among Whites and lowest among Chinese. Diabetes was highest among Blacks and Hispanics and lowest among Whites. Statin use was lower among Hispanics and Chinese. Antihypertensive medication use was highest among Blacks and lowest among Chinese.

Systolic blood pressure was higher among Blacks, with a mean of 132.9 mmHg and 130.2

mmHg respectively for women for men, and lower among Whites, with a mean of 122.9 mmHg and 124.1 mmHg for women and men. Similarly, the average diastolic blood pressure was higher among Blacks (72.5 and 77.0 mmHg for women and men) and lower among Whites (66.9 and 73.8 mmHg for women and men). The prevalence of hypertension at baseline was higher among Blacks (60.8% and 59.9% for women and men), and lower among Chinese (40.3% and 40.8% for women and men), Hispanics (46.6% and 42.0% for women and men), and Whites (43.2% and 44.4% for women and men). Such patterns of participant characteristics were similar after excluding participants with prevalent hypertension at Exam 1.

Association between race/ethnicity and ambient air pollution

The geometric mean of $PM_{2.5}$ was higher among Chinese participants compared with other races/ethnicities (Table 1). The geometric mean of NOx was higher among Hispanics and Chinese, intermediate among Blacks, and lower among Whites. On the contrary, the geometric mean of ozone was higher among Whites, intermediate among Blacks and lower among Hispanics and Chinese. Such relationships were also demonstrated with estimators given by the first equations within the GSEM models, where ambient $PM_{2.5}$ and NOx exposures were positively associated with Black, Hispanic and Chinese races ($p<0.001$), while ozone was negatively associated with Black, Hispanic and Chinese races ($p<0.001$).

Ambient air pollution exposure and racial/ethnic disparities in blood pressure levels

After adjustment for demographics and hypertension risk factors, the mean systolic blood pressure (SBP) for women was 5.9 (95% CI: 4.1, 7.6) mmHg higher in Blacks, 1.3 (95% CI: -0.8, 3.5) mmHg higher in Hispanics and 1.5 (95% CI: -1.0, 4.0) mmHg higher in Chinese compared to that in Whites (Table 2). Among men, the mean systolic blood pressure was 4.1 (95% CI: 2.3, 5.8) mmHg higher in Blacks, 0.1 (95% CI: -1.9, 1.8) mmHg lower in Hispanics, and 2.3 (95% CI: -0.1, 4.6) mmHg

higher in Chinese compared to White men (Table 2). Among men, the difference in SBP levels by race/ethnicity that was related to higher $PM_{2.5}$ concentrations compared with White men (indirect association) was 0.3 (95% CI: 0.1, 0.5) mmHg for Black men, 0.3 (95% CI: 0.1, 0.6) mmHg for Hispanic men and 1.0 (95% CI: 0.2, 1.8) mmHg for Chinese men. Indirect associations for $PM_{2.5}$ among women were similar but only marginally significant (Table 2). When considering potential mediation due to ozone exposure in the association between race/ethnicity and SBP, the indirect associations were significantly negative among both women and men (Table 2), reflecting a positive mediation effect of ozone on racial/ethnic disparities in SBP. In this study, White participants had the highest ambient ozone exposure levels compared to the other race/ethnicities. On the contrary, in the case of NOx, the indirect association was -0.6 (95% CI: -1.0, -0.3) mmHg for Black women, -1.3 (95% CI: -2.0, -0.6) for Hispanic women and -1.3 (95% CI: -2.0, -0.6) for Chinese women compared to White women with similar associations for men (Table 2).

Similar disparities were found in the relationship between race/ethnicity and diastolic blood pressure (DBP) levels. Among both women and men, Black and Chinese participants had significantly higher DBP levels ($p<0.001$ and $p<0.05$ for Black and Chinese participants, respectively) and Hispanics had similar DBP levels ($p>0.05$), compared with Whites. Black-White differences were higher among women, while Chinese-White differences were higher among men (Table 3). With further adjustment for air pollution exposures, the association estimators slightly changed, as indicated by indirect associations (Table 3). Racial/ethnic disparities in DBP mediated by $PM_{2.5}$ were marginally significant for both women and men (Table 3). For NO_X, indirect associations tended to be positive among women and negative among men, but the estimates were all not statistically significant. Indirect associations for mediation by ozone were statistically significant for men and marginally significant for women.

In the models stratified by hypertension status at baseline, the indirect associations for air pollution concentrations on racial/ethnic disparities in blood pressure level were shown to be higher

among participants with hypertension compared to those without hypertension (Table 5, Table 6). Especially for $PM_{2.5}$, indirect associations were significantly positive among women and men with hypertension, while positive but not statistically significant among women without hypertension and negative among men without hypertension.

Ambient air pollution exposure racial/ethnic disparities in incident hypertension

Among the 3,089 participants without hypertension at baseline,422 participants (209 men and 213 women) developed hypertension by Exam 2. Compared to White women, the odds ratio for incident hypertension was 2.1 (95% CI: 1.4, 3.2) for Black women, 1.6 (95% CI:1.0, 2.6) for Hispanic women and 1.5 (95% CI: 0.8, 2.7) for Chinese women (Table 4). Among men, the odds ratio for incident hypertension was 1.5 (95% CI: 0.9, 2.2) in Blacks, 0.8 (95% CI: 0.5, 1.3) in Hispanics and 1.2 (95% CI: 0.7, 2.0) in Chinese (Table 4). Racial/ethnic disparities were barely mediated by $PM_{2.5}$, indicated by indirect associations around 1 (OR=1, P>0.05) (Table 4) among both women and men. The potential mediating effects of NOx and ozone exposure on racial/ethnic differences in incident hypertension were marginally significant among Black, Hispanic and Chinese men compared to White men with no mediation observed among women (Table 4).

Sensitivity analysis: Ambient air pollution exposure and racial/ethnic disparities in blood pressure levels and incident hypertension, further adjustment for study site

In models further adjusting for study site, associations were attenuated. Indirect mean differences in systolic and diastolic blood pressure were approaching 0 (P>0.05, Supplementary Tables 1, 2), and indirect odds ratio of incident hypertension were diminished to 1 (P>0.05, Supplementary Table 3). In other words, indirect associations were pulled toward the null, suggesting no mediating effects of air pollution exposure after accounting for site.

DISCUSSION

Given that MESA is a unique multi-ethnic population-based study with detailed individual-level information about race/ethnicity, blood pressure, cardiovascular risk factors, and household-level estimates of participant air pollution exposure, which was provided by the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air) ancillary study, it has the strength to contribute to our topic and hypotheses substantially by helping us quantitatively assess the extent of ethnic disparities in blood pressure and hypertension attributable to air pollution exposure, and then conceive additional strategies for reducing such disparities through pollution management.

Our main analyses provided statistical evidence of the mediation effect of $PM_{2.5}$ on racial/ethnic disparities in SBP among men, especially on the disparity between Chinese and Whites, as well as the mediating effects of ozone on racial/ethnic disparities in SBP and DBP among both men and women and on racial disparities in incident hypertension among men. We also observed that hypertension status may modify the mediation of air pollution exposure on racial/ethnic disparities in blood pressure. Mediating effects of air pollutant concentrations, especially PM2.5 concentrations on blood pressure were more significant for hypertensive population. On the contrary, racial/ethnic disparities increased after adjusting for ambient NOx exposure, and NOx had an adverse association with blood pressure. To our knowledge this is the first study to examine potential mediation of long-term ambient air pollution exposure to racial/ethnic disparities in hypertension outcomes. Racial/ethnic disparities in blood pressure and hypertension in this study were consistent with those from previous studies[3-6] as was the general pattern of the racial/ethnic differences in air pollution exposure.[24-27] The significant association between long-term $PM_{2.5}$ exposure and baseline blood pressure observed in our models was consistent with results of previous MESA, BWHS and NHIS studies[16,17,22] , although the NHIS study examined race/ethnicity as an effect measure modifier for this association and $PM_{2.5}$ was not necessarily a mediator between race/ethnicity and high blood pressure.[22] We also observed a positive association between long-term ozone exposure and blood

pressure, which was evident in the BWHS study.[18] The inverse association between NOx and blood pressure was also seen in a Danish population-based study of adults 50-64 years of age.[39] In the BWHS study, NO_2 was shown to be negatively associated with hypertension incidence, while NOx was positively associated with hypertension incidence.[17,18] A previous MESA study also showed that the presence of high NO_2 modifies the association between $PM_{2.5}$ and BP.[16] Therefore, a possible explanation for our NOx results is that the proportion of NO_2 concentration within the NOx concentration would influence the effects of NOx exposure on blood pressure.

In sensitivity analyses, the observed indirect associations were attenuated and no longer statistically significant after adjustment for study site. Since differences in climate features such as altitude, temperature and humidity and other environmental characteristics such as traffic, industry and human activities would account for differences in ambient air pollution levels across the six cities, adjustment for study sites could result in over-adjustment for ambient air pollution exposure.

This study has several strengths. MESA is a multiethnic cohort with high-quality standardized protocol. The household-level air pollution estimates and detailed information on demographic characteristics and cardiovascular disease risk factors allowed for comprehensive adjustment for confounders in the mediation analysis. However, there are some limitations. MESA participants of the four races/ethnicities were unbalanced across the six cities due to different enrollment procedures, and variations of air pollution predictions were different across different locations. For instances, Hispanic participants were not enrolled in Chicago and Winston-Salem, and in Winston-Salem, only one AQS monitoring location for NO_2 and NOx met inclusion criteria.[30] As a result, the predicted NO_X concentrations may less likely reflect the true exposure for participants in this site. Due to limited sample sizes and lack of statistical power we are unable to report site-stratified estimates of the associations. But in general, effects differed across cities. For example, the inverse association between NOx and blood pressure after adjustment for risk factors was only observed in Chicago and Winston Salem, as opposed to the other cities. As we used pooled data across study sites, random

effects by sites were not taken into account, which could be another reason why the effects of NOx on blood pressure in MESA were different from studies restricted to only one city.[17]

As for assumptions underlying the mediating analyses (i.e., no reverse causation between the independent variable, dependent variable and mediator, and the effects of these variables in the mediation analysis are not confounded by unmeasured variables)[40,41], since race/ethnicity is a fixed attribute that comes prior to environmental factors and hypertension, and ambient air pollution exposures are not likely to be influenced by one's blood pressure, temporality would not be a concern for the potential mediating effects of ambient air pollution in the pathway linking race/ethnicity to blood pressure and hypertension. Although unmeasured confounding is possible, in our GSEM models we were able to account for several important confounders in the exposure-mediator and exposure-outcome associations. In this study, ambient air pollution was found to be associated with baseline blood pressure after adjustment for race/ethnicity and other risk factors, however no significant associations between long-term ambient air pollution concentrations and incident hypertension were observed, neither by our analyses nor previous hypertension-related analyses of $PM_{2.5}$ and NO_2 in MESA[42], which was not consistent with the BWHS study results[17,18], and may have impacted our ability to assess the mediating effects of ambient air pollution in this setting. Besides, MESA participants were free of cardiovascular disease at enrollment, which may have resulted in a healthier population than other study populations, and thereby had smaller racial/ethnic differences in BP and hypertension and limited potential to detect the relatively small contributions expected due to air pollution. Additionally, we cannot discard the possibility that the racial/ethnic disparities in hypertension outcomes related to air pollution exposure may be related to recent air pollution exposures rather than the long-term exposures examined in this study, since in MESA and several other studies, short-term air pollution exposures were shown to be associated with hypertension or elevated BP[16,43-46] .

Conclusions

Racial disparities in blood pressure was reduced after accounting for $PM_{2.5}$ and ozone while increased after accounting for NO_X. But we failed to find mediating effects of air pollution on the pathway linking race/ethnicity to incident hypertension. Racial/ethnic disparities in incident hypertension among older populations may not be simply reduced by reducing ambient air pollution. But as implied, controlling ambient $PM_{2.5}$ and ozone could help reduce racial/ethnic disparities in blood pressure, especially among adults with hypertension.

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Table 1. Participant characteristics at baseline by gender and race/ethnicity, 2000–2002

	Women				Men			
	White	Black	Hispanic	Chinese	White	Black	Hispanic	Chinese
Number of participants	1310	992	734	378	1208	801	681	359
Age, year	62.5(10.3)	62.2(10.0)	61.5(10.4)	62.2(10.3)	62.7(10.1)	62.3(10.1)	61.3(10.3)	62.6(10.3)
Education, %								
<High school	5.7	11.5	47.7	31.1	4.1	12.6	40.1	16.9
High school	21.2	18.9	22.6	20.8	12.6	19.5	18.7	10.8
Some college	32.3	35.3	22.6	21.6	24.3	34.3	28.3	19.4
≥College degree	40.9	34.4	7.1	26.4	59.0	33.6	12.9	52.8
Income, %								
<$24 999	20.7	32.7	53.5	53.0	10.7	22.2	41.1	41.9
$25 000–$49 999	28.9	32.7	30.9	21.6	23.3	26.2	34.4	23.1
$50 000–$74 999	18.7	16.0	8.7	10.8	21.5	21.2	12.7	13.1
≥$75 000	29.2	11.8	4.1	13.7	42.1	21.7	10.4	21.9
Unknown	2.4	6.9	2.7	0.8	2.3	8.6	1.3	0.0
Body mass index, kg/m2	27.5(5.8)	31.3(6.5)	30.0(5.6)	23.9(3.5)	28.0(4.1)	28.7(4.7)	28.7(4.2)	24.1(3.1)
Physical activity								
Poor	18.6	23.8	33.7	27.7	15.7	24.1	27.1	21.7
Intermediate	20.2	19.2	18.9	21.4	15.1	15.9	13.3	18.6
Ideal	61.3	57.1	47.4	50.9	69.2	60.1	59.6	59.7
Smoking, %								
Never	48.6	51.9	68.0	95.8	39.5	36.7	39.4	53.1
Former	39.7	32.3	21.7	2.4	49.3	43.3	44.5	36.9
Current	11.8	15.8	10.4	1.9	11.2	20.0	16.1	10.0
Waist circumference	95.1(16.2)	101.6(16.1)	100.3(14.5)	86.3(10.7)	101.1(11.5)	100.8(12.6)	100.8(11.1)	87.8(9.0)
Alcohol intake, %								
Never	13.1	24.7	44.0	73.1	5.6	9.1	7.3	33.6
Former	18.4	31.6	19.4	6.9	19.0	35.1	34.1	21.1
Current	68.6	43.8	36.7	20.1	75.3	55.8	58.6	45.3

Diabetes, %								
Normal	86.5	71.9	70.2	74.7	78.9	62.9	63.3	64.2
Impaired	9.0	12.2	13.2	13.2	13.8	17.2	18.0	21.1
Diabetes	4.5	15.9	16.6	12.1	7.3	19.9	18.7	14.7
Total cholesterol, mg/dL	202.4(34.5)	196.2(36.4)	202.2(38.0)	195.2(31.9)	189.0(34.7)	181.6(34.2)	193.5(37.3)	189.6(31.3)
HDL cholesterol, mg/dL	58.9(15.8)	57.1(15.7)	52.6(13.9)	53.4(13.2)	45.2(12.2)	46.6(12.5)	42.7(10.1)	45.7(11.0)
Medication use, % yes								
Statin use	15.0	17.0	12.8	15.3	18.3	14.4	11.9	11.9
Antihypertensive	32.4	52.9	34.5	29.3	33.9	47.2	30.9	28.3
Systolic BP, mm Hg	122.9(22.0)	132.9(22.9)	127.4(23.5)	125.0(23.2)	124.1(18.5)	130.2(19.6)	125.9(20.1)	124.0(19.3)
Diastolic BP, mm Hg	66.9(9.6)	72.5(10.3)	68.3(9.6)	69.3(10.4)	73.8(9.0)	77.0(9.6)	75.0(9.5)	74.9(9.3)
Hypertension, % yes	43.2	60.8	46.6	40.3	44.4	59.9	42.0	40.8
$PM_{2.5}$ concentration, $\mu g/m^3$	15.7(15.6, 15.8)	16.5(16.4, 16.6)	17.1(16.9, 17.4)	19.3(19.0, 19.6)	15.6(15.5, 15.7)	16.5(16.4, 16.6)	16.8(16.5, 17.0)	19.3(19.0, 19.6)
NO_X concentration, ppb	34.4(33.6, 35.3)	44.1(42.7, 45.5)	60.7(58.2, 63.4)	60.0(57.7, 62.4)	32.9(32.0, 33.8)	42.8(41.2, 44.4)	56.4(53.9, 59.4)	58.9(56.5, 61.3)
Ozone concentration, ppb	21.7(21.4, 21.9)	20.0(19.7, 20.3)	16.5(16.3, 16.8)	17.2(17.0, 17.5)	22.0(21.7, 22.2)	20.2(19.8, 20.5)	17.0(16.7, 17.3)	17.0(16.8, 17.3)

Values represent percentages for categorical variables and means (SD) for continuous variables except for $PM_{2.5}$, NO_X and ozone for which geometric means (95% CI) are reported.

BP, blood pressure; HDL, high-density lipoprotein

Table 2. Mean difference in systolic BP (mm Hg) at baseline for Black, Hispanic and Chinese participants compared with White participants stratified by gender, 2000–2002

			$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
	N	Total association*	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Women								
White	1310	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	992	5.9(4.1, 7.6)	5.6(3.9, 7.4)	0.2(-0.02, 0.5)	6.5(4.7, 8.3)	-0.6(-1.0, -0.3)	6.4(4.6, 8.2)	-0.6(-0,9, -0.3)
Hispanic	734	1.3(-0.8, 3.5)	1.0(-1.1, 3.0)	0.3(-0.02, 0.6)	2.6(0.4, 4.8)	-1.3(-2.0, -0.6)	3.0(0.8, 5.2)	-1.8(-2.6, -0.9)
Chinese	378	1.6(-1.0, 4.1)	0.8(-1.9, 3.4)	0.7(-0.1, 1.5)	2.8(0.2, 5.4)	-1.3(-2.0, -0.6)	3.0(0.4, 5.6)	-1.4(-2.1, -0.8)
Men								
White	1208	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	801	4.1(2.3, 5.8)	3.5(1.7, 5.2)	0.3(0.1, 0.5)	4.4(2.7, 6.2)	-0.4(-0.7, -0.1)	4.6(2.9, 6.3)	-0.6(-0.8, -0.3)
Hispanic	681	-0.1(-1.9, 1.8)	-0.5(-2.4, 1.4)	0.3(0.1, 0.6)	0.6(-1.3, 2.5)	-0.7(-1.3, -0.2)	1.4(-0.6, 3.3)	-1.5(-2.2, -0.8)
Chinese	359	2.3(-0.1, 4.6)	1.1(-1.4, 3.6)	1.0(0.2, 1.8)	3.0(0.6, 5.4)	-0.7(-1.3, -0.2)	3.7(1.3, 6.1)	-1.4(-2.0, -0.7)

Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol, antihypertensive medication use and statin use.

*The total association represents the mean difference in SBP and DBP by race/ethnicity after adjustment for demographics and cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (ie, the mean difference in blood pressure by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in blood pressure that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

$PM_{2.5}$, particles <2.5 μm in aerodynamic diameter.

Table 3. Mean difference in diastolic BP (mm Hg) at baseline for Black, Hispanic and Chinese participants compared with White participants, stratified by gender, 2000–2002

			$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
	N	Total association*	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Women								
White	1310	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	992	5.1(4.3, 6.0)	5.1(4.2, 6.0)	0.07(-0.05, 0.2)	5.1(4.2, 6.0)	0.05(-0.1, 0.2)	5.1(4.2, 6.0)	0.04(-0.1, 1.2)
Hispanic	734	0.7(-0.3, 1.7)	0.6(-0.4, 1.6)	0.1(-0.1, 0.2)	0.7(-0.4, 1.8)	0.1(-0.2, 0.4)	0.7(-0.4, 1.8)	0.1(-0.3, 0.5)
Chinese	378	2.0(0.7, 3.3)	1.9(0.5, 3.2)	0.2(-0.2, 0.6)	2.0(0.7, 3.3)	0.1(-0.2, 0.4)	2.0(0.7, 3.3)	0.1(-0.2, 0.4)
Men								
White	1208	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	801	2.9(2.1, 3.8)	2.9(2.0, 3.7)	0.1(-0.1, 0.2)	3.1(2.2, 4.0)	-0.1(-0.3, 0.03)	3.2(2.3, 4.0)	-0.2(-0.3, -0.1)
Hispanic	681	0.6(-0.4, 1.6)	0.6(-0.4, 1.5)	0.1(-0.1, 0.2)	0.8(-0.2, 1.9)	-0.2(-0.5, 0.1)	1.1(0.1, 2.1)	-0.5(-0.8, -0.1)
Chinese	359	2.5(1.3, 3.7)	2.3(1.1, 3.6)	0.2(-0.2, 0.6)	2.7(1.5, 4.0)	-0.2(-0.5, 0.1)	3.0(1.7, 4.2)	-0.5(-0.8, -0.1)

Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol, antihypertensive medication use and statin use.

*The total association represents the mean difference in SBP and DBP by race/ethnicity after adjustment for demographics and cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (ie, the mean difference in blood pressure by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in blood pressure that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

Table 4. Odds ratio of incident hypertension at Exam 2 for Black, Hispanic and Chinese participants compared with White participants, stratified by gender

			$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
	N	Total association*	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Women								
White	710	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Black	335	2.22(1.31, 3.13)	2.19(1.45, 3.29)	1.02(0.96, 1.07)	2.22(1.46, 3.38)	1.00(0.93, 1.07)	2.24(1.48, 3.40)	0.99(0.93, 1.05)
Hispanic	356	1.53(0.82, 2.23)	1.50(0.94, 2.39)	1.02(0.96, 1.07)	1.53(0.96, 2.47)	1.00(0.87, 1.13)	1.57(0.97, 2.55)	0.97(0.83, 1.12)
Chinese	208	1.54(0.63, 2.45)	1.47(0.80, 2.71)	1.05(0.88, 1.21)	1.55(0.85, 2.85)	1.00(0.87, 1.13)	1.58(0.87, 2.88)	0.98(0.85, 1.10)
Men								
White	639	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Black	293	1.59(0.90, 2.28)	1.64(1.06, 2.54)	0.97(0.91, 1.03)	1.70(1.10, 2.63)	0.94(0.86, 1.02)	1.68(1.08, 2.59)	0.95(0.87, 1.02)
Hispanic	352	0.82(0.40, 1.23)	0.83(0.50, 1.38)	0.98(0.93, 1.03)	0.89(0.54,1.49)	0.91(0.79, 1.03)	0.91(0.54, 1.53)	0.89(0.74, 1.04)
Chinese	196	1.40(0.60, 2.19)	1.52(0.85, 2.72)	0.92(0.76, 1.08)	1.55(0.87, 2.76)	0.90(0.78, 1.03)	1.55(0.87, 2.79)	0.90(0.76, 1.04)

Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol and statin use.

*The total association represents the odds ratio of incident hypertension by race/ethnicity after adjustment for demographics and cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association and the direct association between race/ethnicity and incident hypertension that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

Table 5. Mean difference in systolic BP (mm Hg) at baseline for Black, Hispanic and Chinese participants compared with White participants, stratified by hypertension status and gender

			$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
	N	Total association	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
With hypertension (N=3,137)								
Women								
White	562	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	631	5.9(3.4, 8.4)	5.3(2.8, 7.8)	0.6(0.2, 1.0)	6.3(3.8, 8.9)	-0.5(-1.0, 0.1)	6.4(3.9, 8.9)	-0.5(-1.0, -0.1)
Hispanic	342	3.7(0.6, 6.7)	2.7(-0.3, 5.8)	1.0(0.2, 1.7)	4.7(1.5, 7.8)	-1.0(-2.1, 0.1)	5.4(2.1, 8.6)	-1.7(-3.0, -0.5)
Chinese	153	4.2(0.4, 8.1)	2.3(-1.8, 6.4)	1.9(0.6, 3.3)	5.2(1.2, 9.1)	-1.0(-2.0, 0.1)	5.5(1.6, 9.5)	-1.3(-2.3, -0.3)
Men								
White	537	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	481	3.8(1.4, 6.2)	3.4(1.0, 5.8)	0.4(0.1, 0.8)	4.2(1.9, 6.6)	-0.5(-1.0, 0.1)	4.4(2.1, 6.8)	-0.6(-1.1, -0.2)
Hispanic	285	1.9(-1.1, 4.8)	1.1(-2.0, 4.1)	0.8(0.2, 1.5)	2.8(-0.2, 5.9)	-1.0(-2.0, 0.1)	3.7(0.6, 6.9)	-1.9(-3.1, -0.7)
Chinese	146	1.1(-2.8, 4.9)	-1.0(-4.8, 3.5)	1.7(0.4, 3.0)	2.0(-1.9, 5.9)	-1.0(-2.0, 0.1)	2.8(-1.1, 6.6)	-1.7(-2.8, -0.6)
Without hypertension (N=3,326)								
Women								
White	748	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	361	3.5(1.9, 5.1)	3.5(1.9, 5.1)	0.02(-0.2, 0.2)	3.9(2.3, 5.6)	-0.4(-0.7, -0.1)	3.9(2.3, 5.5)	-0.3(-0.6, -0.1)
Hispanic	392	-1.0(-2.8, 0.8)	-1.0(1.9, 5.1)	0.02(-0.2, 0.2)	-0.3(-2.2, 1.5)	-0.7(-1.2, -0.1)	-0.2(-2.0, 1.7)	-0.8(-1.5, -0.2)
Chinese	225	0.5(-1.8, 2.7)	0.4(-1.9, 2.7)	0.1(-0.5, 0.7)	1.2(-1.1, 3.4)	-0.7(-1.2, -0.1)	1.2(-1.1, 3.5)	-0.7(-1.3, -0.2)
Men								
White	671	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	320	1.7(0.1, 3.3)	1.8(0.2, 3.4)	-0.1(-0.3, 0.1)	2.1(0.5, 3.7)	-0.4(-0.7 tp -0.1)	2.2(0.6, 3.8)	-0.5(-0.7, -0.2)
Hispanic	396	0.1(-1.5, 1.8)	0.2(-1.5, 1.8)	-0.1(-0.2, 0.1)	0.7(-1.0, 2.4)	-0.6(-1.0, -0.1)	1.1(-0.7, 2.8)	-1.0(-1.5, -0.4)
Chinese	213	1.9(-0.1, 4.0)	2.3(0.1, 4.4)	-0.3(-1.0, 0.3)	2.6(0.5, 4.7)	-0.6(-1.1, -0.2)	2.9(0.8, 5.0)	-1.0(-1.5, 0.4)

Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol, antihypertensive medication use and statin use.

Table 6. Mean difference in diastolic BP (mm Hg) at baseline for Black, Hispanic and Chinese participants compared with White participants, stratified by hypertension status and gender

			$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
	N	Total association	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
				With Hypertension (N=3,137)				
Women								
White	562	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	631	5.1 (3.9, 6.3)	5.0(3.7, 6.2)	0.1(-0.1, 0.3)	5.0(3.7, 6.2)	0.2(-0.1, 0.4)	5.0(3.8, 6.2)	0.1(-0.1, 0.3)
Hispanic	342	1.2 (-0.2, 2.6)	1.0(-0.5, 2.4)	0.2(-0.1, 0.5)	0.8(-0.7, 2.3)	0.4(-0.2, 0.9)	0.9(-0.6, 2.4)	0.3(-0.3, 0.9)
Chinese	153	3.5 (1.6, 5.5)	3.1(1.0, 5.1)	0.5(-0.2, 1.1)	3.2(1.2, 5.2)	0.3(-0.1, 0.8)	3.3(1.3, 5.3)	0.2(-0.2, 0.7)
Men								
White	537	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	481	3.0 (1.8, 4.2)	2.9(1.7, 4.1)	0.1(-0.04, 0.3)	3.1(1.8, 4.3)	-0.04(-0.3, 0.2)	3.1(1.9, 4.3)	-0.1(-0.3, 0.1)
Hispanic	285	1.9 (0.4, 3.4)	1.7(0.2, 3.2)	0.2(-0.1, 0.5)	2.0(0.4, 3.6)	-0.1(-0.6, 0.4)	2.2(0.6, 3.8)	-0.2(-0.8, 0.3)
Chinese	146	1.4 (-0.5, 3.3)	0.9(-1.1, 2.9)	0.5(-0.1, 1.2)	1.5(-0.5, 3.4)	-0.1(-0.6, 0.4)	1.6(-0.3, 3.5)	-0.2(-0.7, 0.3)
				Without Hypertension (N=3,326)				
Women								
White	748	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	361	4.5(3.4, 5.5)	4.4(3.4, 5.5)	0.02(-0.1, 0.2)	4.4(3.3, 5.4)	0.1(-0.1, 0.3)	4.4(3.3, 5.4)	0.1(-0.1, 0.2)
Hispanic	392	0.3(-1.0, 1.5)	0.2(-1.0, 1.4)	0.02(-0.1, 0.2)	0.1(-1.1, 1.3)	0.1(-0.2, 0.5)	0.03(-1.2, 1.3)	0.2(-0.2, 0.6)
Chinese	225	1.2(-0.2, 2.7)	1.2(-0.3, 2.7)	0.1(-0.3, 0.5)	1.1(-0.4, 2.6)	0.1(-0.2, 0.5)	1.0(-0.5, 2.5)	0.2(-0.1, 0.5)
Men								
White	671	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	320	2.0(1.0, 3.0)	2.2(1.1, 3.2)	-0.2(-0.3, 0.0)	2.2(1.1, 3.2)	-0.2(-0.4, 0.01)	2.2(1.2, 3.2)	-0.2(-0.4, -0.03)
Hispanic	396	0.5(-0.6, 1.5)	0.6(-0.5, 1.6)	-0.1(-0.2, 0.02)	0.7(-0.4, 1.8)	-0.3(-0.5, 0.01)	0.9(-0.2, 2.0)	-0.4(-0.8, -0.1)
Chinese	213	2.8(1.5, 4.1)	3.2(1.9, 4.6)	-0.4(-0.8, -0.01	3.1(1.8, 4.4)	-0.3(-0.6, 0.02)	3.2(1.9, 4.6)	-0.4(-0.8, -0.1)

Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol, antihypertensive medication use and statin use.

SUPPLEMENTARY MATERIAL

Supplementary Table 1. Sensitivity analysis: Mean difference in systolic BP (mm Hg) at Exam 1 for Black, Hispanic and Chinese participants compared with White participants, further adjusted for study sites

			$PM_{2.5}$ concentrations		NO_X concentrations		Ozone concentrations	
	N	Total association*	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Women								
White	1327	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	1020	5.4(3.6, 7.3)	5.4(3.5, 7.3)	0.0(-0.1, 0.1)	5.4(3.5, 7.3)	0.0(-0.1, 0.1)	5.4(3.6, 7.3)	-0.03(-0.1, 0.05)
Hispanic	739	4.2(2.0, 6.5)	4.2(2.0, 6.5)	0.0(-0.1, 0.1)	4.2(1.9, 6.5)	0.0(0.0, 0.0)	4.1(1.8, 6.4)	0.1(-0.01, 0.3)
Chinese	380	3.3(0.5, 6.2)	3.3(0.5, 6.2)	0.0(-0.1, 0.1)	3.4(0.5, 6.2)	0.0(-0.1, 0.1)	3.1(0.3, 6.0)	0.2(0.0, 0.4)
Men								
White	1225	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	813	3.4(1.6, 5.2)	3.3(1.5, 5.1	0.1(-0.1, 0.3)	3.4(1.6, 5.2)	0.0(-0.03, 0.05)	3.4(1.6, 5.2)	0.0(0.0, 0.0)
Hispanic	684	1.8(-0.3, 3.9)	1.8(-0.3, 3.8)	0.1(-0.1, 0.2)	1.8(-0.3, 3.8)	0.0(-0.1, 0.1)	1.8(-0.2, 3.9)	0.0(-0.1, 0.1)
Chinese	365	2.2(-0.5, 4.8)	2.1(-0.6, 4.8)	0.0(-0.05, 0.1)	2.2(-0.5, 4.9)	0.0(-0.2, 0.1)	2.2(-0.5, 4.9)	-0.1(-0.3, 0.1)

Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol, antihypertensive medication use, statin use and study site.

*The total association represents the mean difference in SBP and DBP by race/ethnicity after adjustment for cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (ie, the mean difference in blood pressure by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in blood pressure that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

Supplementary Table 2. Sensitivity analysis: Mean difference in diastolic BP (mm Hg) at Exam 1 for Black, Hispanic and Chinese participants compared with White participants, further adjusted for study site

			$PM_{2.5}$ concentrations		NO_X concentrations		Ozone concentrations	
	N	Total association*	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Women								
White	1327	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	1020	4.6(3.7, 5.5)	4.6(3.7, 5.6)	0.0(-0.1, 0.0)	4.6(3.7, 5.5)	0.0(0.0, 0.0)	4.6(3.7, 5.5)	0.0(0.0, 0.0)
Hispanic	739	1.2(0.0, 2.3)	1.2(0.1, 2.3)	0.0(-0.1, 0.0)	1.2(0.0, 2.3)	0.0(0.0, 0..1)	1.1(0.0, 2.2)	0.1(0.0, 0.1)
Chinese	380	2.7(1.2, 4.1)	2.7(1.2, 4.1)	0.0(-0.1, 0.0)	2.7(1.2, 4.1)	0.0(-0.1, 0.0)	2.6(1.1, 4.0)	0.1(0.0, 0.2)
Men								
White	1225	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)
Black	813	2.7(1.8, 3.6)	2.6(1.7, 3.6)	0.1(0.0, 0.2)	2.7(1.8, 3.6)	0.0(0.0, 0.0)	2.7(1.8, 3.6)	0.0(0.0, 0.0)
Hispanic	684	1.2(0.1, 2.2)	1.1(0.0, 2.2)	0.0(0.0, 0.1)	1.2(0.1, 2.2)	0.0(-0.1, 0.0)	1.2(0.1, 2.3)	0.0(-0.1, 0.0)
Chinese	365	2.8(1.5, 4.2)	2.8(1.4, 4.1)	0.1(0.0, 0.2)	2.8(1.5, 4.2)	0.0(-0.1, 0..1)	2.9(1.5, 4.2)	-0.1(-0.1, 0.0)

*Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol, antihypertensive medication use, statin use and study site.

The total association represents the mean difference in SBP and DBP by race/ethnicity after adjustment for cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (ie, the mean difference in blood pressure by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in blood pressure that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

Supplementary Table 3. Sensitivity analysis: Odds ratio of incident hypertension by Exam 2 for Black, Hispanic and Chinese participants compared with White participants, further adjusted for study site

			$PM_{2.5}$ concentrations		NO_X concentrations		Ozone concentrations	
	N	Total association*	Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Women								
White	710	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Black	335	2.06(1.19, 2.92)	2.03(1.33, 3.08)	1.02(0.96, 1.07)	2.05(1.35, 3.12)	1.00(1.00, 1.00)	2.05(1.35, 3.11)	1.00(0.99, 1.02)
Hispanic	356	1.51(0.72, 2.30)	1.50(0.89, 2.53)	1.00(0.98, 1.03)	1.51(0.89, 2.55)	1.00(0.96, 1.03)	1.51(0.89, 2.56)	0.99(0.96, 1.02)
Chinese	208	1.29(0.46, 2.12)	1.28(0.67, 2.43)	1.01(0.97, 1.05)	1.28(0.67, 2.43)	1.00(0.98, 1.03)	1.29(0.68, 2.46)	0.99(0.93, 1.05)
Men								
White	639	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)	1.0 (ref)
Black	293	1.51(0.84, 2.19)	1.55(0.99, 2.43)	0.98(0.93, 1.03)	1.52(0.97, 2.37)	1.00(0.99, 1.01)	1.52(0.97, 2.37)	1.00(1.00, 1.00)
Hispanic	352	0.73(0.34, 1.13)	0.75(0.44, 1.28)	0.99(0.96, 1.02)	0.74(0.43, 1.27)	1.00(0.99, 1.01)	0.74(0.43, 1.27)	1.00(0.98, 1.02)
Chinese	196	1.61(0.61, 2.62)	1.65(0.89, 3.09)	0.98(0.92, 1.03)	1.62(0.87, 3.02)	1.00(0.96, 1.04)	1.62(0.87, 3.01)	1.00(0.93, 1.07)

Adjusted for age, education, income, smoking status, alcohol intakes, body mass index, waist circumference, physical activity, diabetes, total cholesterol, HDL cholesterol, statin use and study site.

*The total association represents the odds ratio of incident hypertension by race/ethnicity after adjustment for cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association and the direct association between race/ethnicity and incident hypertension that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

Curriculum Vitae

Lanxin Song was born in 1993 in China.

Education

- BLOOMBERG SCHOOL OF PUBLIC HEALTH, JOHNS HOPKINS UNIVERSITY — 2016 - 2018

  Degree: Master of Science in Epidemiology.
  Track: General Epidemiology and Methodology.
  Relevant coursework: Epidemiologic Methods, Statistical Methods in Public Health, Analysis of Longitudinal Data, Multilevel Statistical Models in Public Health, Qualitative Research Theory and Methods, Qualitative Data Analysis, Stata Programming, Spatial Analysis.
- SCHOOL OF PUBLIC HEALTH, SUN YAT-SEN UNIVERSITY — 2011-2016

  Degree: Bachelor Degree of Medicine.
  Major: Preventive Medicine.
  Relevant coursework: Calculus, Chemistry, Courses of Basic Medical and Clinical Medical Sciences, Epidemiology, Environmental Health and Occupational Health Sciences, Biostatistics, Nutrition and Food Sciences.

Research Experience

- Since June, 2017, as a master student in Johns Hopkins, I've been working with Dr. Miranda Jones on my thesis, of which the topic is to assess the mediation effect of air pollution on racial/ethnic disparities in the development of hypertension. We participated in the MESA study, which is a long-term ongoing large cohort study with 6814 participants enrolled in 6 cities across the U.S. What I completed include establishment of research questions, hypotheses and methods, proposal crafting, data cleaning and analysis, and writing the thesis.

- Since August, 2014, I had worked as a data collector, analyst, and coordinator in an epidemiological project on investigation and knowledge promotion of Dengue Fever. This project was conducted by my college advisor Associate Professor Zhang Jinxin, under the cooperation with Health Education Center of Guangzhou. Besides interviewing subjects in hospitals and communities, data collecting, managing and analyzing, I also conducted a Delphi investigation and had accomplished it by the end of March, 2015.

Work Experience

Teaching Assistant
Fundamentals of Epidemiology — Sept. - Dec. 2017.
Primary Duties: Participate in faculty meetings; Lead discussion sections as a section instructor; Hold office hours.
Research Assistant
B'FRIENDS Program, Baltimore City Health Department. — Oct. 2017 - Present
Primary Duties: Assess data-sets for inclusion in falls database and manage data requests from community partners; Assist with data analyses for falls-related CRISP data-sets; Assist with writing quarterly report on falls in Baltimore City; Implement contracts for data matching.

Research Assistant
Bloomberg School of Public Health. — Feb. 2018 - Present
Primary Duties: Working on systematic reviews in collaboration with Johns Hopkins and faculty from the University of Washington.

Internship

- 3-month public health internship in Guangzhou Municipal Provincial Centre for Disease Prevention and Control — Oct. - Dec. 2015
- 3-month clinical clerkship in Guangzhou Twelfth Municipal People's Hospital and Guangzhou infectious disease hospital — Jul. - Sept. 2015
- 8-week clinical probation in The Third Affiliated Hospital, Sun Yat-sen University — Sept. - Nov. 2014.
- 3-week public health internship in China National Health Development Research Center — Jul. - Aug. 2014.
- 4-week preventive medicine probation in Guangdong Provincial Centre for Disease Prevention and Control — Aug. - Sept. 2013.
- 4-week preventive medicine probation in Qinghai Provincial Institute for Endemic Disease Control and Research — Jul. - Aug. 2013. Participated in an epidemiological investigation over a plague outbreak.

Volunteer

- Association "Allshare" and NGO "JIA" — 2011- 2015

  Helped secluded cured leprosy patients in several aspects of their lives in isolated "leprosy villages".
- Tour of Qinghai Lake Road Bicycle Racing — Jul. 2013

  Served as an emergency aid assistant in the medical team along the entire tour.

Skills

- Advanced Knowledge in Epidemiology, Biostatistics, Basic Medical and Clinical Medical Sciences. Capability of writing, numeracy, data savvy, management and analysis, collaboration, active communication and being attentive to details.
- Computer competency: Proficiency with statistical packages (Stata, SAS, R, SPSS), Spatial Analysis tools (ArcGIS), Microsoft Office (Excel, Word, PowerPoint) and data management tools (SQL).
- Experiment skills in Basic Medical Science disciplines. e.g. Biochemistry tests such as Western Blot.
- Fundamental clinical skills and technologies. e.g. First aids, Surgical suturing, Lumbar puncture, Bone marrow aspiration.
- Hygienic investigation and assay skills: Familiarity with sampling, assay and analysis methods in Environmental, Occupational and Nutrition Health Sciences.

- Language: Chinese (native); English (proficient in writing and speaking); Japanese (proficient in writing and speaking).