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Ambient air pollution as a mediator in the pathway linking race/ethnicity to blood pressure elevation: The multi-ethnic study of atherosclerosis (MESA)

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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. The role of air pollution exposure to racial/ethnic differences in hypertension has not been explored.

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

Methods: We studied 6,463 White, Black, Hispanic and Chinese adults enrolled 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, 0.6) 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 O_3 for SBP among women and men and for DBP among men. In contrast, racial/ethnic disparities were attenuated due to exposure to NO_X.

Conclusion: Racial disparities in blood pressure were reduced after accounting for $PM_{2.5}$ and ozone while increased after accounting for NO_X.

1. Introduction

Hypertension is a highly prevalent condition relevant to many cardiovascular events in the US(Kochanek et al., 2016; Vital Signs, 2012). Racial/ethnic differences in hypertension prevalence and risk, have been shown in various studies (Redmond et al., 2011; Kramer et al., 2004; Rasmussen-Torvik et al., 2016; Fei et al., 2017) even after controlling for clinical risk factors of hypertension. The Chicago Area Sleep Study (CASS, 494 adults aged 35–64 years) (Rasmussen-Torvik et al., 2016), a cross-sectional study of 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%). In the Multi-Ethnic Study of Atherosclerosis (MESA), after multivariable adjustment, African American and Chinese race/ethnicity were significantly associated with hypertension compared to Whites (Kramer et al., 2004). 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 (Rasmussen-Torvik et al., 2016; Basu et al., 2014; Hicken et al., 2014a; Bosworth et al., 2006; Suglia et al., 2013; Kato, 2012; Thorpe et al., 2014; Bell et al., 2010).

Previous studies have accorded importance to the role of fine particulate matter (particles < 2.5 μm in aerodynamic diameter [$PM_{2.5}$])

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(Brook and Rajagopalan, 2009; Brook et al., 2002, 2011a; Auchincloss et al., 2008; Coogan et al., 2012; Liang et al., 2014; Fuks et al., 2011; Urch et al., 2005; Johnson and Parker, 2009; Chan et al., 2015) and nitrogen oxides (NO_X) (Brook et al., 2011a; Coogan et al., 2012, 2017; Chan et al., 2015) in the development of hypertension and blood pressure elevation, and there is a paucity of evidence that exposure to ozone (O_3) (Coogan et al., 2017; Urch et al., 2005; Brook et al., 2002) also elevates blood pressure. In a 2014 meta-analysis including 22 studies, exposure to $PM_{2.5}$, especially long-term exposure (1-year cumulative exposure), was positively associated with systolic (SBP) and diastolic blood pressure (DBP) levels (Combined mean difference per 10 μg/m^3 increase in $PM_{2.5}$: 1.393 mmHg [95% CI: 0.874, 1.912] for SBP and 0.895 mmHg [95% CI: 0.49, 1.299] for DBP) (Liang et al., 2014). 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/m^3 increase); this association differed by race/ethnicity and was statistically significant in White but not Black or Hispanic adults (Johnson and Parker, 2009). In a cohort of Black women in the Black Women's Health Study (BWHS), long-term exposures to O_3, $PM_{2.5}$ and NOx have been associated with increased hypertension incidence (Coogan et al., 2012, 2016, 2017).

Exposure to air pollution has been shown to differ by race/ethnicity (Su et al., 2011; Morello-Frosch and Lopez, 2006; Jones et al., 2014). 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 (Chakraborty and Zandbergen, 2007). 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 (Jones et al., 2014). Despite this evidence, there is an absence of published studies that have examined the mediating effect of air pollution on the relationship between race/ethnicity and hypertension outcomes. 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.

2. Methods

2.1. 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 White, Black, Hispanic and Chinese participants aged 45–84 years from 6 major metropolitan areas—Los Angeles, CA, St. Paul, MN, Chicago, IL, New York, NY, Baltimore, MD and Winston-Salem, NC (Bild et al., 2002). 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. Institutional review board approval was granted at each study site, and all participants provided written informed consent. From the 6,814 MESA participants, the following exclusion process was applied: firstly, 258 participants were excluded for missing information on air pollution exposures; secondly, 3 participants without measures of systolic and diastolic blood pressure at baseline were excluded; thirdly, 5 participants were excluded for being enrolled from sites with few participants of the same race/ethnicity; and finally, 85 participants with missing data on potential covariates were excluded, leaving 6,463 participants for this analysis.

2.2. 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 (Kramer et al., 2004; Jones et al., 2014; Adar et al., 2010; Mujahid et al., 2011).

2.3. Ambient air pollution concentrations

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 of $PM_{2.5}$, NO_X and O_3 from national monitoring data from US Environmental Protection Agency (EPA) Air Quality System (AQS) supplemented with data collected specifically for the MESA Air study were integrated into hierarchical spatiotemporal models to predict annual average exposure at each participant residence. Details of the MESA Air monitoring campaign and adopted spatiotemporal models were described elsewhere (Kaufman et al., 2012; KellerCasey Olives Kimet al., 2015; Wang et al., 2015; Cohen et al., 2009). 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, O_3 data were collected in warm seasons between April and September. In Los Angeles, O_3 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 concentrations were right-skewed and log-transformed in our analyses.

2.4. Blood pressure levels and hypertension

Resting systolic and diastolic blood pressure was measured in the right arm after 5 min 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. Hypertension was defined as SBP ≥140 mmHg or DBP ≥90 mmHg, according to JNC-VII (Chobanian et al., 2003), or the use of any antihypertensive medication.

2.5. 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 (Ainsworth et al., 1999) 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). (Ogunmoroti et al., 2017; Lloyd-Jones et al., 2010).

2.6. Statistical analysis

Racial/ethnic differences in blood pressure outcomes have been shown to differ for men and women (Krieger, 1990; Agyemang et al., 2008), 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/ethnicity 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. To improve estimation of mediating effects, we adjusted models for sociodemographic and cardiovascular risk factors shown to be associated with blood pressure, air pollution exposure and race/ethnicity. 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, education, household income, antihypertensive medication use, statin use, diabetes, alcohol intake, smoking status and physical activity; 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 is 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. To evaluate the potential influence of study site, models were further adjusted for study sites. Additionally, stratification by hypertension 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. All statistical analyses were performed using STATA version 14.0 (StataCorp. 2015. Stata St, 2015).

3. Results

3.1. Participant characteristics

Characteristics of study participants at baseline are shown in Table 1. Among the 6,463 participants, 38.9% were White, 27.7% were Black, 21.9% were Hispanic, and 11.4% were Chinese-American. 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 ($P = 0.005$). Educational attainment, family income and physical activity were higher among Whites and lower among Hispanics ($P < 0.001$). BMI was highest among Hispanics and Blacks, lowest among Chinese ($P < 0.001$). Smoking was highest among Blacks and lowest among Chinese ($P < 0.001$). Alcohol intake was highest among Whites and lowest among Chinese ($P < 0.001$). Diabetes was highest among Blacks and Hispanics and lowest among Whites ($P < 0.001$). Statin use was lower among Hispanics and Chinese ($P = 0.002$). Antihypertensive medication use was highest among Blacks and lowest among Chinese ($P < 0.001$).

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 O_3 was higher among Whites, intermediate among Blacks and lower among Hispanics and Chinese.

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

After adjustment for demographics and cardiovascular risk factors, the mean SBP for women was 5.8 (95% CI: 4.0, 7.6) mmHg higher in Blacks, 1.2 (95% CI: -0.8, 3.3) mmHg higher in Hispanics and 1.2 (95% CI: -1.3, 3.8) mmHg higher in Chinese women compared to that in White women. There was no statistically significant mediation by $PM_{2.5}$ concentrations in the association of race/ethnicity on SBP levels among women (Table 2, Model 1). In contrast, after adjustment for NO_X and O_3 concentrations, differences in SBP for Black, Hispanic and Chinese women compared to White women increased indicating that these exposures may obscure racial/ethnic disparities in SBP among women (Indirect associations [95% CI] for NO_X concentrations were −0.6 [-1.0, −0.3], −1.3 [-2.0, −0.6] and −1.2 [-1.9, −0.6] mmHg for Black, Hispanic and Chinese women, respectively compared to White women and −0.6 [-0.9, −0.3], −1.7 [-2.5, −0.9] and −1.4 [-2.0, −0.7] mmHg, respectively for O_3 concentrations, Table 2, Model 1). These findings were similar both in women with and without hypertension. After further adjusting for study site, the mean SBP for women was 5.2 (95% CI: 3.4, 7.1) mmHg higher in Blacks, 3.9 (95% CI: 1.6, 6.1) mmHg higher in Hispanics and 2.9 (95% CI: 0.1, 5.8) mmHg higher in Chinese compared to that in White women (Table 2, Model 2). Indirect associations (overall and stratified by hypertension status) were attenuated and no longer statistically significant (Table 2, Model 2), suggesting no mediating effects of air pollution exposure after accounting for study site, except for some evidence of mediation by O_3 concentrations for Chinese women compared to White women (indirect association: 0.2 [95% CI: 0.0, 0.4] mmHg, (Table 2, Model 2). Among men, the mean SBP was 4.0 (95% CI: 2.3, 5.7) mmHg higher in Blacks, 0.3 (95% CI: -2.2, 1.6) mmHg lower in Hispanics, and 1.9 (95% CI: -0.6, 4.3) mmHg higher in Chinese compared to White men (Table 3, Model 1). 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, 0.6) 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. Similar to our findings for women, indirect associations for NO_X and O_3 concentrations for SBP among men were significantly negative and demonstrate that racial/ethnic differences in NO_X and O_3 exposures may obscure racial/ethnic disparities in SBP for Black, Hispanic and Chinese men compared to their White counterparts (Table 3, Model 1). Among men, the significant findings for NO_X were observed only among men without hypertension (Table 3, Model 1).When further adjusting for study site, compared to White men, the mean SBP was 3.5 (95% CI: 1.5, 5.3) mmHg higher in Black men, 1.4 (95% CI: -0.7, 3.5) mmHg higher in Hispanic men, and 1.6 (95% CI: -1.1, 4.3) mmHg higher in Chinese men (Table 3, Model 2). After adjustment for study site, there was no longer evidence of mediation by air pollution concentrations, either among men with or without hypertension (Table 3, Model 2).

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
Air pollution concentrations								
PM2.5 concentration, μg/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)
NOX 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.

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

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 (Tables 4 and 5, Model 1). With further adjustment for air pollution exposures, the association estimators only slightly changed, as indicated by the indirect associations of small magnitude (Tables 4 and 5, Model 1). Racial/ethnic disparities in DBP mediated by $PM_{2.5}$ were marginally significant for both women and men (Tables 4 and 5, Model 1). For NO_X, indirect associations were insignificantly positive among women and insignificantly negative among men. Indirect associations for mediation by O_3 were statistically significant for men and marginally significant for women (Tables 4 and 5, Model 1). After adjustment for study site (Tables 4 and 5, Model 2), association estimates were attenuated in the same way as for SBP levels. None of the indirect associations was significant after controlling for study site, no matter whether stratification by hypertension status was applied.

4. Discussion

This study assessed the potential role of air pollution exposure to explain racial/ethnic disparities in blood pressure levels. Our analysis of White, Black, Hispanic and Chinese adults from 6 US cities shows the potential role of $PM_{2.5}$ concentrations on racial/ethnic disparities in SBP among men, especially on the disparity between Chinese and Whites, as well as the role of O_3 on racial/ethnic disparities in SBP and

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

	N	Total association	$PM_{2.5}$ Concentrations		NO_x Concentrations		Ozone Concentrations	
			Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Overall (N = 3,414)								
Model 1								
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	**5.8(4.0, 7.6)**	**5.6(3.8, 7.4)**	**0.2(0.0, 0.5)**	**6.5(4.6, 8.3)**	**−0.6(-1.0, -0.3)**	**6.4(4.6, 8.2)**	**−0.6(-0.9, -0.3)**
Hispanic	734	1.2 (-0.8, 3.3)	1.0 (-1.1, 3.0)	0.3 (-0.1, 0.6)	**2.5(0.4, 4.7)**	**−1.3(-2.0, -0.6)**	**2.9(0.7, 5.2)**	**−1.7(-2.5, -0.9)**
Chinese	378	1.2 (-1.3, 3.8)	0.6 (-2.1, 3.3)	0.7 (-0.1, 1.5)	2.5 (-0.1, 5.1)	**−1.2(-1.9, -0.6)**	**2.6(0.0, 5.3)**	**−1.4(-2.0, -0.7)**
Model 2								
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	**5.2(3.4, 7.1)**	**5.2(3.3, 7.1)**	0.0 (-0.1, 0.2)	**5.2(3.4, 7.1)**	0.0 (-0.04, 0.04)	**5.3(3.4, 7.1)**	−0.01 (-0.1, 0.1)
Hispanic	734	**3.9(1.6, 6.1)**	**3.8(1.6, 6.1)**	0.0 (-0.03, 0.1)	**3.9(1.6, 6.1)**	0.0 (-0.1, 0.1)	**3.7(1.5, 6.0)**	0.1 (-0.01, 0.3)
Chinese	378	**2.9(0.1, 5.8)**	**2.9(0.1, 5.7)**	0.0 (-0.04, 0.1)	**2.9(0.1, 5.8)**	0.0 (-0.1, 0.1)	2.7 (-0.1, 5.6)	**0.2(0.0, 0.4)**
With hypertension (N = 1,688)								
Model 1								
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	**5.8(3.3, 8.2)**	**5.2(2.7, 7.7)**	**0.6(0.2, 1.0)**	**6.2(3.7, 8.7)**	−0.4 (-1.0, 0.1)	**6.3(3.8, 8.8)**	**−0.5(-0.9, -0.1)**
Hispanic	342	**3.6(0.6, 6.6)**	2.7 (-0.4, 5.7)	**0.9(0.2, 1.6)**	**4.6(1.4, 7.8)**	−1.0 (-2.1, 0.1)	**5.3(2.1, 8.5)**	**−1.7(-2.9, -0.4)**
Chinese	153	**4.2(0.4, 8.0)**	2.2 (-1.8, 6.3)	**1.9(0.6, 3.3)**	**5.1(1.1, 9.0)**	−0.9 (-1.9, 0.1)	**5.4(1.5, 9.3)**	**−1.3(-2.2, -0.3)**
Model 2								
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	**5.4(2.8, 8.0)**	**5.4(2.8, 8.0)**	0.0 (-0.1, 0.1)	**5.3(2.8, 7.9)**	0.0 (-0.1, 0.2)	**5.4(2.8, 7.9)**	0.0 (-0.1, 0.1)
Hispanic	342	**5.4(2.0, 8.8)**	**5.4(2.0, 8.8)**	0.0 (-0.1, 0.1)	**5.4(2.0, 8.7)**	0.0 (-0.1, 0.2)	**5.3(1.9, 8.7)**	0.1 (-0.1, 0.3)
Chinese	153	**4.2(-0.1, 8.5)**	**4.2(-0.1, 8.7)**	0.0 (-0.1, 0.1)	4.3 (-0.1, 8.6)	0.0 (-0.2, 0.1)	4.1 (-0.2, 8.5)	0.1 (-0.1, 0.3)
Without hypertension (N = 1,726)								
Model 1								
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)**	0.0 (-0.2, 0.2)	**3.8(2.2, 5.5)**	**−0.4(-0.7, -0.1)**	**3.8(2.2, 5.4)**	**−0.3(-0.6, -0.1)**
Hispanic	392	−1.1 (-2.8, 0.8)	−1.0 (-2.8, 0.7)	0.0 (-0.2, 0.2)	−0.4 (-2.3, 1.4)	**−0.6(-1.2, -0.1)**	−0.3 (-2.1, 1.6)	**−0.8(-1.4, -0.2)**
Chinese	225	0.2 (-2.0, 2.4)	0.4 (-1.9, 2.7)	0.1 (-0.6, 0.7)	0.8 (-1.4, 3.1)	**−0.6(-1.2, -0.1)**	0.9 (-1.4, 3.1)	**−0.7(-1.2, -0.1)**
Model 2								
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	**3.1(1.4, 4.8)**	**3.1(1.4, 4.8)**	0.0 (-0.1, 0.3)	**3.1(1.4, 4.8)**	0.0 (0.0, 0.0)	**3.2(1.5, 4.9)**	−0.0 (-0.1, 0.0)
Hispanic	392	0.6 (-1.3, 2.5)	0.6 (-1.3, 2.5)	0.0 (-0.1, 0.1)	0.6 (-1.3, 2.5)	0.0 (-0.1, 0.1)	0.5 (-1.4, 2.4)	0.1 (-0.1, 0.2)
Chinese	225	1.6 (-0.8, 4.0)	1.6 (-0.9, 4.0)	0.0 (-0.1, 0.2)	1.6 (-0.8, 4.0)	0.0 (-0.1, 0.1)	1.5 (-1.0, 3.9)	0.1 (-0.1, 0.4)

Bold indicate statistically significant findings at $p < 0.05$.
Model 1 adjusted for age, education, income, smoking status, alcohol intake, BMI, waist circumference, physical activity, diabetes, antihypertensive medication use and statin use.
Model 2 further adjusted for study site.
*The total association represents the mean difference in SBP by race/ethnicity after adjustment for demographics and cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (i.e., the mean difference in SBP by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in SBP that is not due to the potential explanations ($PM_{2.5}$, NO_x, ozone in separate analyses).

DBP among both men and women. The differences in blood pressure levels due to racial/ethnic differences in air pollution among minority populations compared to Whites in this study may provide insight to the potential explanations for racial/ethnic disparities in cardiovascular morbidity and mortality in the US. 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.

Previous studies have mainly considered race/ethnicity as an effect modifier in associations between air pollution and health outcomes. In MESA, race/ethnicity did not modify associations between $PM_{2.5}$ and blood pressure (Adar et al., 2018), although the association between air pollution and left-ventricular mass index, which has been associated with hypertension, was shown to be stronger among Black participants (Hicken et al., 2016). Studies have additionally considered whether psychosocial factors, that have been associated with race/ethnicity, may modify the association between air pollution exposure and blood pressure outcomes. In MESA, there was no evidence of synergistic effects of higher PM2.5 and adverse social/psychosocial factors on blood pressure, however there was some evidence of a stronger associations of PM2.5 with blood pressure in higher SES groups (Hicken et al., 2013). In the Detroit Healthy Environments Partnership (HEP) survey, while race/ethnicity did not modify the association between PM2.5 and blood pressure, the association between PM2.5 and SBP was stronger for those who reported high levels of stress (Hicken et al., 2014b). In a 2018 review examining whether associations between air pollution exposure and hypertensive disorders of pregnancy were modified by social determinants of health including race/ethnicity, there was limited evidence suggesting racial differences in associations between particulate matter and adverse maternal health effects (Koman et al., 2018). The presence of effect modification in the association between air pollution and blood pressure levels by race/ethnicity may reflect differences due to other factors such as gene-environment interactions or nutrition-environment interactions. Our approach focuses on mediation rather than effect modification, which is conceptually different, and requires that there is no interaction between the exposure and the mediator, as well as confounding, or at least to relax these assumptions (Corraini et al., 2017). Assessing if air pollution exposure mediates the association between race/ethnicity and air pollution builds on documented findings that some racial/ethnic minority populations are disproportionately exposed to higher levels of air pollution compared to Whites and allows us to estimate the potential impact for racial/ethnic minority populations if air pollution exposure was reduced to levels experienced by White participants. Such findings provide support for strategies to reduce racial/ethnic disparities in the US as air pollution levels can be monitored and controlled.

The significant association between long-term $PM_{2.5}$ exposure and baseline blood pressure observed in our study are consistent with results of previous studies (Auchincloss et al., 2008; Coogan et al., 2012;

Table 3
Mean difference in systolic BP (mm Hg) at baseline for Black, Hispanic and Chinese men compared with White men stratified by hypertension status, 2000–2002.

	N	Total association	$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
			Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Overall (N = 3,049)								
Model 1								
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	**4.0(2.3, 5.7)**	**3.7(2.0, 5.4)**	**0.3(0.1, 0.6)**	4.4 (2.7, 6.2)	**−0.4(-0.8, -0.1)**	**4.6(2.9, 6.3)**	**−0.6(-0.9, -0.3)**
Hispanic	681	−0.3 (-2.2, 1.6)	−0.6 (-2.5, 1.3)	**0.3(0.1, 0.6)**	0.4 (-1.5, 2.4)	**−0.7(-1.3, -0.1)**	1.1 (-0.9, 3.1)	**−1.4(-2.1, -0.7)**
Chinese	359	1.9 (-0.6, 4.3)	0.8 (-1.8, 3.4)	**1.0(0.2, 1.8)**	2.6 (0.1, 5.1)	**−0.7(-1.3, -0.1)**	**3.2(0.7, 5.7)**	**−1.3(-2.0, -0.7)**
Model 2								
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	**3.5(1.7, 5.3)**	**3.5(1.7, 5.3)**	0.05 (-0.1, 0.2)	**3.5(1.7, 5.3)**	0.0 (-0.04, 0.1)	**3.5(1.8, 5.3)**	0.0 (0.0, 0.0)
Hispanic	681	1.4 (-0.7, 3.5)	1.4 (-0.7, 3.5)	0.02 (-0.05, 0.1)	1.4 (-0.7, 3.5)	0.0 (-0.1, 0.1)	1.4 (-0.7, 3.5)	0.0 (-0.1, 0.1)
Chinese	359	1.6 (-1.1, 4.3)	1.5 (-1.2, 4.3)	0.1 (-0.1, 0.2)	1.5 (-1.2, 4.3)	0.0 (-0.2, 0.1)	1.6 (-1.1, 4.3)	0.0 (-0.2, 0.2)
With hypertension (N = 1,449)								
Model 1								
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)**	**4.3(1.9, 6.6)**	−0.5 (-1.0, 0.1)	**4.5(2.1, 6.8)**	**−0.6(-1.1, -0.2)**
Hispanic	285	1.8 (-1.2, 4.7)	1.0 (-2.0, 4.0)	**0.8(0.1, 1.4)**	2.7 (-0.4, 5.8)	−0.9 (-2.0, 0.1)	**3.5(0.4, 6.7)**	**−1.8(-2.9, -0.6)**
Chinese	146	0.9 (-2.9, 4.8)	−0.7 (-4.9, 3.4)	**1.7(0.4, 3.0)**	1.8 (-2.1, 5.7)	−0.9 (-1.9, 0.1)	2.5 (-1.3, 6.4)	**−1.6(-2.6, -0.5)**
Model 2								
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	**3.1(0.6, 5.5)**	**3.0(0.5, 5.4)**	0.0 (-0.1, 0.2)	**3.0(0.6, 5.4)**	0.1 (-0.1, 0.2)	**3.0(0.5, 5.4)**	0.0 (-0.1, 0.1)
Hispanic	285	**3.5(0.3, 6.7)**	**3.5(0.6, 5.5)**	0.0 (-0.1, 0.1)	**3.4(0.2, 6.6)**	0.1 (-0.2, 0.4)	**3.5(0.5, 6.9)**	0.0 (-0.2, 0.3)
Chinese	146	0.8 (-3.5, 5.0)	0.7 (-3.5, 5.0)	0.0 (-0.1, 0.2)	0.8 (-3.4, 5.1)	0.0 (-0.1, 0.1)	0.7 (-3.3, 5.2)	0.0 (-0.2, 0.3)
Without hypertension (N = 1,600)								
Model 1								
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	**1.8(0.2, 3.4)**	**1.9(0.3, 3.5)**	−0.1 (-0.4, 0.1)	**2.2(0.5, 3.8)**	**−0.4(-0.7, -0.1)**	**2.2(0.6, 3.8)**	**−0.5(-0.7, -0.2)**
Hispanic	396	0.1 (-1.5, 1.8)	0.1 (-1.5, 1.8)	−0.1 (-0.2, 0.1)	0.6 (-1.1, 2.3)	**−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)	**2.2(0.0, 4.3)**	−0.3 (-1.0, 0.3)	**2.5(0.4, 4.6)**	**−0.6(-1.1, -0.1)**	**2.9(0.8, 5.0)**	**−0.9(-1.5, -0.4)**
Model 2								
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	**1.8(0.1, 3.5)**	1.7 (-0.01, 3.4)	0.1 (-0.1, 0.3)	1.8 (0.1, 3.5)	0.0 (-0.02, 0.03)	**1.8(0.1, 3.5)**	0.0 (-0.1, 0.1)
Hispanic	396	1.2 (-0.6, 3.0)	1.1 (-0.7, 2.9)	0.1 (-0.1, 0.2)	1.2 (-0.6, 3.0)	0.0 (-0.03, 0.03)	1.2 (-0.6, 3.0)	0.0 (-0.1, 0.04)
Chinese	213	**2.8(0.5, 5.0)**	**2.6(0.3, 4.9)**	0.1 (-0.1, 0.4)	**2.7(0.5, 5.0)**	0.0 (-0.1, 0.2)	**2.9(0.6, 5.1)**	−0.1 (-0.3, 0.1)

Bold indicate statistically significant findings at $p < 0.05$.
Model 1 adjusted for age, education, income, smoking status, alcohol intake, BMI, waist circumference, physical activity, diabetes, antihypertensive medication use and statin use.
Model 2 further adjusted for study site.
*The total association represents the mean difference in SBP by race/ethnicity after adjustment for demographics and cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (i.e., the mean difference in SBP by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in SBP that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

Liang et al., 2014; Johnson and Parker, 2009; Chan et al., 2015). We also observed a positive association between long-term O_3 exposure and blood pressure, which was also demonstrated in the BWHS study (Coogan et al., 2017). After adjustment for study site, all observed indirect associations were attenuated and no longer statistically significant. Air pollution concentrations were significantly different across 6 study sites. Compared with Winston Salem ($PM_{2.5}$ 16.3 μg/m^3, NO_X 23.0 ppb, O_3 27.9 ppb), mean difference (95% CI) for $PM_{2.5}$ was 0.1 (0.01, 0.2) ug/m^3 higher in New York, −0.4 (−0.5, −0.3) ug/m^3 lower in Baltimore, −3.3 (−3.4, −3.2) ug/m^3 lower in St. Paul, 0.2 (−01, 0.3) ug/m^3 higher in Chicago and 5.3 (5.2, 5.4) ug/m^3 higher in Los Angeles; NO_X was 59.3 (58.2, 60.5) ppb higher in New York, 19.0 (17.9, 20.2) ppb higher in Baltimore, 1.5 (0.3, 2.6) ppb higher in St. Paul, 20.8 (19.7, 22.0) ppb higher in Chicago and 58.1 (57.0, 59.3) ppb higher in Los Angeles; O_3 was −13.8 (−14.0, −13.7) ppb lower in New York, −7.3 (−7.4, −7.1) ppb lower in Baltimore, −4.3 (−4.5, −4.2) ppb lower in St. Paul, −7.2 (−7.3, −7.0) lower in Chicago and −12.3 (−12.4, −12.1) ppb lower in Los Angeles. 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.

We observed that hypertension status may modify the mediation of air pollution exposure, especially $PM_{2.5}$ concentrations, on racial/ethnic disparities in blood pressure. This finding is consistent with previous studies that have reported that the cardiovascular effects of traffic-related exposures may be greater in adults with hypertension. In MESA, the association of exposure to $PM_{2.5}$ with progression in coronary calcification was greater in individuals with hypertension (Kaufman et al., 2016). Additionally, in a study of 6,450 participants of the second survey of the Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA 2), the adverse effect of transportation noise on blood pressure levels was larger among participants that reported physician-diagnosed hypertension (Dratva et al., 2012).

The epidemiologic evidence for an association of air pollution exposure with incidence of hypertension is mixed. Among women who participated in the BWHS in Los Angeles, the incidence rate ratio (IRR) for hypertension for a 10 μg/m^3 increase in $PM_{2.5}$ was 1.48 (95% CI: 0.95, 2.31), and for a 12.4 ppb (interquartile range increase) in NO_X was 1.14 (95% CI: 1.03, 1.25) (Coogan et al., 2012), however the BWHS study included only Black women. In contrast, in a study of 57,053 Danish residents, long-term NOx exposure was not associated with incident hypertension during follow-up (Sørensen et al., 2012). We additionally explored the role of air pollution exposure on the development of incident hypertension at Exam 2 (2002–2004) among 3,089 adults without hypertension at baseline and found no evidence of a mediating effect of ambient air pollution exposure on racial/ethnic disparities in incident hypertension (data not shown).

This study has several strengths. MESA is a unique, multi-ethnic

Table 4
Mean difference in diastolic BP (mm Hg) at baseline for Black, Hispanic and Chinese women compared with White women stratified by hypertension status, 2000–2002.

	N	Total association	$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
			Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Overall (N = 3,414)								
Model 1								
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)**	0.05 (-0.1, 0.2)	**5.1(4.2, 6.0)**	0.03 (-0.1, 0.2)	**5.1(4.2, 6.0)**	0.02 (-0.1, 0.1)
Hispanic	734	0.7 (-0.3, 1.7)	0.6 (-0.4, 1.6)	0.1 (-0.1, 0.2)	0.6 (-0.5, 1.7)	0.1 (-0.3, 0.4)	0.6 (-0.5, 1.7)	0.07 (-0.3, 0.4)
Chinese	378	**1.8(0.5, 3.1)**	**1.7(0.3, 3.0)**	0.2 (-0.2, 0.6)	**1.8(0.4, 3.1)**	0.1 (-0.3, 0.4)	**1.8(0.4, 3.1)**	0.1 (-0.2, 0.4)
Model 2								
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	**4.5(3.6, 5.4)**	**4.5(3.6, 5.4)**	0.0 (-0.1, 0.0)	**4.5(3.6, 5.4)**	0.0 (0.0, 0.0)	**4.5(3.6, 5.4)**	0.0 (0.0, 0.0)
Hispanic	734	**1.1(-0.1, 2.2)**	**1.1(-0.1, 2.2)**	0.0 (-0.1, 0.0)	**1.0(-0.1, 2.2)**	0.0 (0.0, 0.1)	**1.0(-0.1, 2.2)**	0.1 (0.0, 0.1)
Chinese	378	**2.4(1.0, 3.8)**	**2.4(1.0, 3.8)**	0.0 (-0.1, 0.0)	**2.4(1.0, 3.9)**	0.0 (-0.1, 0.0)	**2.3(1.0, 3.8)**	0.1 (0.0, 0.2)
With hypertension (N = 1,688)								
Model 1								
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	**5.0(3.8, 6.2)**	**4.9(3.6, 6.1)**	0.1 (-0.1, 0.3)	**4.9(3.6, 6.1)**	0.2 (-0.1, 0.4)	**4.9(3.7, 6.1)**	0.1 (-0.1, 0.3)
Hispanic	342	1.2 (-0.2, 2.7)	1.0 (-0.5, 2.4)	0.2 (-0.1, 0.5)	0.9 (-0.6, 2.4)	0.4 (-0.2, 0.9)	0.9 (-0.6, 2.5)	0.3 (-0.3, 0.9)
Chinese	153	**3.7(1.8, 5.6)**	**3.2(1.2, 5.3)**	0.5 (-0.2, 1.1)	**3.4(1.4, 5.4)**	0.3 (-0.2, 0.8)	**3.5(1.5, 5.4)**	0.2 (-0.2, 0.6)
Model 2								
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	**4.4(3.2, 5.7)**	**4.4(3.2, 5.7)**	0.0 (-0.1, 0.03)	**4.4(3.1, 5.7)**	0.0 (-0.03, 0.1)	**4.4(3.2, 5.7)**	0.0 (-0.03, 0.1)
Hispanic	342	1.0 (-0.6, 2.6)	1.0 (-0.6, 2.6)	0.0 (-0.1, 0.1)	1.0 (-0.6, 2.6)	0.0 (-0.04, 0.1)	1.0 (-0.6, 2.6)	0.0 (-0.04, 0.1)
Chinese	153	**3.8(1.7, 6.0)**	**3.8(1.6, 6.0)**	0.0 (-0.1, 0.1)	**3.9(1.7, 6.0)**	0.0 (-0.1, 0.1)	**3.8(1.7, 5.9)**	0.0 (-0.1, 0.1)
Without hypertension (N = 1,726)								
Model 1								
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	**4.4(3.4, 5.4)**	**4.4(3.4, 5.4)**	0.0 (-0.1, 0.2)	**4.3(3.3, 5.4)**	0.1 (-0.1, 0.3)	**4.3(3.3, 5.3)**	0.1 (-0.1, 0.3)
Hispanic	392	0.2 (-1.0, 1.4)	0.2 (-1.0, 1.4)	0.0 (-0.1, 0.1)	0.03 (-1.2, 1.3)	0.1 (-0.2, 0.5)	−0.1 (-1.3, 1.2)	0.2 (-0.2, 0.6)
Chinese	225	1.0 (-0.5, 2.4)	0.9 (-0.6, 2.4)	0.1 (-0.4, 0.5)	0.8 (-0.7, 2.3)	0.1 (-0.2, 0.5)	0.8 (-0.7, 2.3)	0.2 (-0.1, 0.5)
Model 2								
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	**3.9(2.8, 5.0)**	**3.9(2.8, 5.0)**	0.0 (-0.1, 0.1)	**3.9(2.8, 5.0)**	0.0 (0.0, 0.0)	**3.9(2.8, 5.0)**	0.0 (-0.1, 0.03)
Hispanic	392	0.4 (-0.9, 1.7)	0.4 (-0.9, 1.7)	0.0 (-0.1, 0.1)	0.4 (-0.9, 1.7)	0.0 (-0.1, 0.1)	0.4 (-0.9, 1.7)	0.0 (-0.05, 0.1)
Chinese	225	1.3 (-0.3, 2.9)	1.3 (-0.3, 2.9)	0.0 (-0.1, 0.1)	1.3 (-0.3, 2.9)	0.0 (-0.04, 0.1)	1.2 (-0.4, 2.8)	0.1 (-0.1, 0.2)

Bold indicate statistically significant findings at $p < 0.05$.
Model 1 adjusted for age, education, income, smoking status, alcohol intake, BMI, waist circumference, physical activity, diabetes, antihypertensive medication use and statin use.
Model 2 further adjusted for study site.
*The total association represents the mean difference in SBP by race/ethnicity after adjustment for demographics and cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (i.e., the mean difference in SBP by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in SBP that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

population-based study with high-quality standardized protocol including 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. This cohort allowed us to quantitatively assess the extent of ethnic disparities in blood pressure and hypertension that could be attributable to air pollution exposure. The detailed information on demographic characteristics and cardiovascular disease risk factors allowed for comprehensive adjustment for important confounders in the mediation analysis.

However, there are some limitations. MESA participants of the four race/ethnicities were unbalanced across the six cities due to study enrollment procedures, and variations of air pollution predictions may differ across different locations (KellerCasey Olives Kimet al., 2015). Due to limited sample sizes and lack of statistical power we are unable to report site-stratified estimates of the associations. 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 (Coogan et al., 2012). Compared to other racial/ethnic groups, Chinese American participants in our analyses had fewer years living in the US (median [IQR] for years lived in the US was 19 [11–27] years for Chinese-American participants compared to 31 [20–41] years for Hispanic participants, 33 [24–52] years for Black participants, and 46 [35–61] years for White participants). Air pollution exposures prior to enrollment or before leaving for the US is unknown, therefore the mediating effects observed for Chinese participants compared to White participants in our study could be influenced by their previous air pollution exposures, which could reflect levels restricted in the US.

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) (Imai et al., 2010a, 2010b), 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 cross-sectional analysis, ambient air pollution was associated with baseline blood pressure after adjustment for race/ethnicity and other risk factors. Total and HDL cholesterol, which have been shown to be associated with blood pressure elevation, could potentially be mediators in the relationship between air pollution exposure and blood pressure; sensitivity analyses further adjusting for

Table 5
Mean difference in diastolic BP (mm Hg) at baseline for Black, Hispanic and Chinese men compared with White men stratified by hypertension status, 2000–2002.

	N	Total association	$PM_{2.5}$ Concentrations		NO_X Concentrations		Ozone Concentrations	
			Direct association	Indirect association	Direct association	Indirect association	Direct association	Indirect association
Overall (N = 3,049)								
Model 1								
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	**3.0(2.1, 3.9)**	**2.9(2.0, 3.8)**	0.1 (-0.1, 0.2)	**3.1(2.2, 4.0)**	−0.1 (-0.3, 0.04)	**3.2(2.3, 4.0)**	**−0.2(-0.3, -0.03)**
Hispanic	681	0.6 (-0.4, 1.6)	0.6 (-0.3, 1.6)	0.1 (-0.1, 0.2)	0.9 (-0.1, 1.9)	−0.2 (-0.5, 0.1)	**1.1(0.1, 2.2)**	**−0.4(-0.8, -0.1)**
Chinese	359	**2.4(1.2, 3.6)**	**2.2(0.9, 3.5)**	0.1 (-0.3, 0.6)	**2.6(1.4, 3.9)**	−0.2 (-0.5, 0.1)	**2.8(1.5, 4.1)**	**−0.4(-0.7, -0.1)**
Model 2								
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	**2.7(1.8, 3.6)**	**2.6(1.7, 3.6)**	0.1 (0.0, 0.1)	**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	681	**1.1(0.0, 2.2)**	**1.0(0.0, 2.1)**	0.0 (0.0, 0.1)	**1.1(0.0, 2.2)**	0.0 (-0.1, 0.0)	**1.1(0.0, 2.2)**	0.0 (-0.1, 0.0)
Chinese	359	**2.6(1.2, 3.9)**	**2.5(1.1, 3.9)**	0.1 (0.0, 0.2)	**2.5(1.2, 3.9)**	0.0 (-0.1, 0.1)	**2.6(1.2, 4.0)**	0.0 (-0.1, 0.0)
With hypertension (N = 1,449)								
Model 1								
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	**2.9(1.8, 4.1)**	**2.8(1.6 4.0)**	0.1 (-0.05, 0.3)	**3.0(1.8, 4.2)**	−0.04 (-0.3, 0.2)	**3.0(1.8, 4.2)**	−0.1 (-0.3, 0.1)
Hispanic	285	**1.9(0.4, 3.4)**	**1.7(0.1, 3.2)**	0.2 (-0.1, 0.5)	**2.0(0.4, 3.6)**	−0.1 (-0.6, 0.4)	**2.1(0.5, 3.7)**	−0.2 (-0.8, 0.4)
Chinese	146	1.3 (-0.6, 3.2)	0.9 (-1.2, 2.9)	0.5 (-0.2, 1.1)	1.4 (-0.5, 3.4)	−0.1 (-0.6, 0.4)	1.5 (-0.4, 3.5)	−0.2 (-0.7, 0.3)
Model 2								
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	**2.5(1.3, 3.8)**	**2.5(1.2, 3.7)**	0.1 (-0.04, 0.2)	**2.6(1.3, 3.8)**	0.0 (-0.1, 0.1)	**2.5(1.2, 3.7)**	0.0 (-0.03, 0.1)
Hispanic	285	**2.1(0.4, 3.7)**	**2.1(0.4, 3.8)**	0.0 (-0.1, 0.0)	**2.1(0.4, 3.7)**	0.0 (-0.1, 0.1)	**2.1(0.4, 3.8)**	0.0 (-0.1, 0.1)
Chinese	146	1.2 (-0.9, 3.3)	1.1 (-0.9, 3.2)	0.0 (-0.1. 0.1)	1.2 (-0.9, 3.3)	0.0 (0.0, 0.0)	1.1 (-0.9, 3.2)	0.0 (-0.1, 0.2)
Without hypertension (N = 1,600)								
Model 1								
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	**1.9(0.9, 2,9)**	**2.1(1.0, 3.1)**	−0.2 (-0.3, 0.0)	**2.1(1.0, 3.1)**	−0.2 (-0.4, 0.01)	**2.1(1.1, 3.2)**	**−0.2(-0.4, -0.03)**
Hispanic	396	0.5 (-0.6, 1.6)	0.6 (-0.5, 1.7)	−0.1 (-0.2, 0.0)	0.8 (-0.4, 1.9)	−0.3 (-0.5, 0.01)	0.9 (-0.2, 2.1)	**−0.4(-0.8, -0.1)**
Chinese	213	**2.8(1.5, 4.1)**	**3.2(1.9, 4.6)**	**−0.4(-0.9, -0.01)**	**3.1(1.7, 4.4)**	−0.3 (-0.6, 0.02)	**3.2(1.9, 4.5)**	**−0.4(-0.8, -0.1)**
Model 2								
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	**1.9(0.8, 3.0)**	**1.9(0.8, 2.9)**	0.1 (-0.1, 0.2)	**1.9(0.8, 3.0)**	0.0 (0.0, 0.0)	**1.9(0.8, 3.0)**	0.0 (-0.04, 0.1)
Hispanic	396	0.7 (-0.4, 1.9)	0.7 (-0.5, 1.9)	0.0 (-0.05, 0.1)	0.7 (-0.4, 1.9)	0.0 (0.0, 0.0)	0.7 (-0.4, 1.9)	0.0 (-0.1, 0.03)
Chinese	213	**3.6(2.2, 5.1)**	**3.5(2.1, 5.0)**	0.1 (-0.1, 0.2)	**3.6(2.2, 5.1)**	0.0 (-0.1, 0.1)	**3.7(2.3, 5.2)**	−0.1 (-0.2, 0.04)

Bold indicate statistically significant findings at $p < 0.05$.
Model 1 adjusted for age, education, income, smoking status, alcohol intake, BMI, waist circumference, physical activity, diabetes, antihypertensive medication use and statin use.
Model 2 further adjusted for study site.
*The total association represents the mean difference in SBP by race/ethnicity after adjustment for demographics and cardiovascular risk factors. For each pollutant, the total association is decomposed into the indirect association (i.e., the mean difference in SBP by race/ethnicity that is due to the difference in air pollution exposure by race/ethnicity) and the direct association of race/ethnicity in SBP that is not due to the potential explanations ($PM_{2.5}$, NO_X, ozone in separate analyses).

HDL and total cholesterol did not change our overall conclusions in this study (data not shown). Findings of long-term ambient air pollution concentrations with incident hypertension have been mixed, with a study in MESA finding no statistically significant associations (Adar et al., 2018), while BWHS studies (Coogan et al., 2012, 2017) found increased hypertension incidence. Additionally, 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. Lastly, 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 some studies, short-term air pollution exposures were shown to be associated with hypertension or elevated BP (Auchincloss et al., 2008; Brook and Kousha, 2015; Szyszkowicz et al., 2012; Brook et al., 2011b; Hoffmann et al., 2012).

5. Conclusions

In this study, racial/ethnic disparities in blood pressure were reduced after accounting for $PM_{2.5}$ and O_3 concentrations while increased after accounting for NO_X concentrations. These findings were no longer statistically significant after adjustment for study site, except for some evidence of mediation by O_3 concentrations for Chinese women compared to White women. Our findings suggest that controlling ambient $PM_{2.5}$ and O_3 could help reduce racial/ethnic disparities in blood pressure, especially among adults with hypertension. This study supports additional strategies for reducing such disparities through pollution management. Epidemiological studies evaluating the role of environmental exposures, such as ambient air pollution to racial/ethnic health disparities are needed.

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Declaration of competing interest

None declared.

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