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Ambient air pollution and racial/ethnic differences in carotid intima-media thickness in the Multi-Ethnic Study of Atherosclerosis (MESA)

Miranda R Jones¹, Ana V Diez-Roux^{2,3}, Marie S O'Neill², Eliseo Guallar¹, A Richey Sharrett¹, Wendy Post⁴, Joel D Kaufman⁵, and Ana Navas-Acien¹

¹Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland, USA

²University of Michigan School of Public Health, Ann Arbor, Michigan, USA

³Drexel University School of Public Health, Philadelphia, Pennsylvania, USA

⁴Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

⁵University of Washington School of Public Health, Seattle, Washington, USA

Abstract

Background—In the USA, ethnic disparities in atherosclerosis persist after accounting for known risk factors. Ambient air pollution is associated with increased levels of atherosclerosis and differs in the USA by race/ethnicity. We estimated the influence of ambient air pollution exposure to ethnic differences in common carotid intima-media thickness (IMT).

Methods—We cross-sectionally studied 6347 Caucasian-American, African-American, Hispanic and Chinese adults across 6 US cities in 2000–2002. Annual ambient air pollution concentrations (fine particulate matter [PM_{2.5}] and oxides of nitrogen [NO_x]) were estimated at each participant's residence. IMT was assessed by ultrasound.

Results—The mean IMT was 19.4 and 37.6 μ m smaller for Hispanic women and men, 53.6 and 7.1 μ m smaller for Chinese women and men, and 23.4 and 38.7 μ m higher for African-American women and men compared with Caucasian-American women and men. After adjustment for PM_{2.5}, the differences in IMT remained similar for Hispanic and African-American participants but was even more negative for Chinese participants (mean IMT difference of –58.4 μ m for women and –15.7 μ m for men) compared with Caucasian-American participants. The IMT

Correspondence to Dr Miranda R Jones, Department of Epidemiology, Johns Hopkins Bloomberg, School of Public Health, 615 N, Wolfe St, Office E6133, Baltimore, MD 21205, USA; mjone132@jhu.edu.

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Data sharing statement The Multi-Ethnic Study of Atherosclerosis (MESA) has hundreds of investigators, many active scientific working groups and dozens of ongoing analytic projects. We are always looking for outside investigators interested in using the data to answer their research questions. To help interested parties navigate the data and topics and find collaborations, we encourage you to contact the Coordinating Center or a MESA investigator. Here is a link to the website for more details: <http://www.mesa-nhlbi.org/>.

Contributors MRJ and AN-A conceptualised the study and its design. JDK and WP acquired data and ensured quality control. MRJ performed the statistical analysis and drafted the manuscript. All the coauthors made substantial contributions to the interpretation of the analyses and critically revised the manuscript.

difference in Chinese participants compared with Caucasian-American participants related to their higher PM_{2.5} exposures was 4.8 μ m (95% CI 0.2 to 10.8) for women and 8.6 μ m (95% CI 3.4 to 15.3) for men. NO_x was not related to ethnic differences in IMT.

Conclusions—The smaller carotid IMT levels in Chinese participants were even smaller after accounting for higher PM_{2.5} concentrations in Chinese participants compared with Caucasian-American participants. Air pollution was not related to IMT differences in African-American and Hispanic participants compared with Caucasian-American participants.

INTRODUCTION

Despite national declines in cardiovascular disease mortality in the past decades, many subgroups defined by race/ethnicity show striking disparities in clinical and subclinical cardiovascular disease even after adjustment for clinical risk factors.^{1–7} This has led to the exploration of other potential explanations for these disparities.^{8–15} Exposure to air pollution is markedly different by race/ethnicity^{10,16–18} and studies have consistently shown increased risk for cardiovascular morbidity and mortality associated with exposure to ambient air pollution, including exposure to fine particulate matter (particles <2.5 μ m in aerodynamic diameter [PM_{2.5}]) and nitrogen oxides (sum of nitric oxide, nitrogen dioxide [NO₂], nitrous acid and nitric acid, [NO_x]).^{19–24} Potential mechanisms for the relationship between air pollution exposure and increased cardiovascular disease risk include the development and progression of atherosclerosis and/or the triggering of cardiovascular events in persons with subclinical disease.^{25–29} Indeed, increased exposure to fine particulate matter and roadway traffic has been associated with 1–10% larger carotid intima-media thickness (IMT).^{30–34}

The role of air pollution exposure in racial/ethnic differences in atherosclerosis has not been explored. Prior studies of air pollution with disparities in other health outcomes have been limited to ecological exposure assessment, lack of adjustment for relevant risk factors and self-reported study outcomes, especially for cardiovascular disease.^{35–38} The objective of this study was to estimate the influence of exposure to PM_{2.5} and NO_x, estimated at the household level, to racial/ethnic differences in carotid IMT. Given the higher air pollution exposure among non-Caucasian-American individuals (especially Chinese Americans) compared with Caucasian-American individuals,¹⁸ and the positive association between air pollution and IMT,^{30–34} we hypothesised that accounting for exposure to ambient air pollution would result in smaller IMT levels for African-American, Hispanic and Chinese participants compared with Caucasian-American participants. Among African-American participants, who have somewhat higher air pollution exposure and higher IMT levels compared with Caucasian-American participants^{31,8} this would be reflected as a decrease of the higher IMT for African-American participants compared with Caucasian-American participants. For Hispanic and Chinese participants who have greater air pollution exposure but smaller IMT levels compared with Caucasian-American participants^{31,8} accounting for air pollution exposure would result in an even smaller IMT for Hispanic and Chinese individuals compared with Caucasian-American individuals.

METHODS

Study population

The Multi-Ethnic Study of Atherosclerosis (MESA) enrolled 6814 Caucasian-American, African-American, Hispanic and Chinese participants aged 45–84 years who were free of cardiovascular disease from Forsyth County (Winston-Salem), North Carolina; New York, New York; Baltimore, Maryland; St Paul, Minnesota; Chicago, Illinois and Los Angeles, California between 2000 and 2002 (baseline examination). Additional details are provided elsewhere.³⁹ In summary, Caucasian-American participants were recruited from all six study sites; African-American participants were recruited from all sites except St Paul; Hispanic participants were only recruited in Los Angeles and St Paul; and Chinese participants were only recruited in Chicago and Los Angeles. From the 6814 participants, we excluded 258 participants with missing data on air pollution exposure, 5 participants who were enrolled from sites with few participants of the same race/ethnicity (2 Chinese Americans in New York and 3 Hispanics in Winston-Salem), 87 participants with missing information on common carotid IMT at baseline and 117 participants missing other relevant covariate data, leaving a total of 6347 participants (3356 women and 2991 men) for this analysis.

Demographics

Self-reported race/ethnicity was categorised as non-Hispanic Caucasian-American, non-Hispanic African-American, Hispanic and Chinese. Self-reported educational attainment and annual family income at baseline were used as primary measures of participant socioeconomic status. Participant education was measured as the highest level completed and categorised as less than high school, high school, some college/technical school, and college/graduate degree. Annual family income was collected in 13 categories. For this analysis, we categorised annual family income as unknown, less than \$24 999, \$25 000–\$49 999, \$50 000–\$74 999, and \$75 000 and greater.

Ambient air pollution exposures

Concentrations of PM_{2.5} and NO_x at individual residence locations were predicted by MESA Air for the MESA cohort using area-specific hierarchical spatiotemporal models^{40–42} that employ spatially varying long-term average concentrations and seasonal and long-term trends, as well as spatially correlated but temporally independent residuals. The MESA Air exposure models are built using monitoring data from the US Environmental Protection Agency (EPA) Air Quality System, supplemented with data collected specifically for the MESA Air study. MESA Air includes data from 27 fixed site monitors situated in MESA communities which collected over 100 consecutive 2-week integrated air samples over the course of the study, monitors placed at a subset of nearly 700 participant homes, and for NO_x monitoring, over 100 samples in each MESA region during each of three seasons during simultaneous deployment ('snapshot' campaigns). This sampling and modelling approach is described in more detail elsewhere.^{43,44} Each model also included geographic variables such as roadway density, land use, and outputs from dispersion models, among others. To characterise household-level ambient air pollution exposure, we used likelihood-based annual average concentrations of PM_{2.5} and NO_x for the year 2000 that were estimated for each participant based on the location(s) lived in during that year.

Intima-media thickness

Images of the right and left common and internal carotid arteries were obtained from supine participants using high-resolution B-mode ultrasound (Logiq 700, 13 MHz; GE Medical Systems) during the MESA baseline examination (July 2000–August 2002). Videotaped images collected were digitised and analysed centrally at Tufts Medical Center (Boston, Massachusetts) masked to participant characteristics and clinical information. IMT for the common carotid was assessed as the mean of all available maximum wall thicknesses across the near and far walls of the left and right sides. Intraclass correlation coefficients for intrareader and inter-reader reproducibility of the common carotid IMT were 0.98 and 0.87, respectively.⁴⁵

Other variables

Cardiovascular disease risk factors at the MESA baseline examination included cigarette smoking, body mass index (BMI), blood pressure levels, diabetes, cholesterol and medication use (statins and antihypertensives). Cigarette smoking was categorised as current, former or never, and lifetime pack-years of smoking were estimated. BMI was calculated by dividing measured weight in kilograms by measured height in metres squared. Blood pressure was measured using a Dinamap PRO 100 automated oscillometric device after 5 min of rest. Three systolic and diastolic blood pressures were measured on the same day in the sitting position and the average of the second and third readings was used in this analysis. A central laboratory measured total and high-density lipoprotein (HDL) cholesterol and glucose levels from blood samples obtained after a 12 h fast. 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).

Statistical analysis

Racial/ethnic differences in subclinical atherosclerosis have been shown to vary by gender; therefore, analyses were conducted separately for women and men. Descriptive statistics were stratified by participant race/ethnicity (Caucasian-American, Chinese, African-American and Hispanic) and gender.

To evaluate the association between race/ethnicity and IMT ('total association'), we compared the differences in baseline common carotid IMT in African-American, Hispanic and Chinese participants with Caucasian-American participants using linear regression models. All our models were adjusted for age (continuous), education (categorical) and annual family income (categorical), smoking status (categorical), pack-years of smoking (continuous), BMI (continuous), diabetes (categorical), systolic blood pressure (continuous), total cholesterol (continuous), HDL cholesterol (continuous), antihypertensive medication use (dichotomous) and statin use (dichotomous). To control for the influence of factors that may vary geographically and could be related to cardiovascular disease, all models were also adjusted for metropolitan area.

The total association between race/ethnicity and carotid IMT, however, does not account for racial/ethnic differences in air pollution exposure that may influence differences in carotid

IMT (Figure 1). We therefore evaluated the association between race/ethnicity and carotid IMT after accounting for differences in air pollution by adding measures of log-transformed PM_{2.5} or NO_x concentrations to the models ('direct association'), in separate models for each air pollutant. We modelled PM_{2.5} and NO_x as log-transformed because both variables are right skewed and log-transformed variables are less susceptible to influential points. Analyses with PM_{2.5} and NO_x in the original scale resulted in similar results (data not shown).

We also estimated the mean difference in carotid IMT in African-American, Hispanic and Chinese participants compared with Caucasian-American participants that is related to race/ethnic differences in air pollution exposures that are in turn associated with carotid IMT ('indirect association', figure 1, see online supplementary material, methods). The sum of the direct and indirect associations equals the total association between race/ethnicity and IMT. Since higher air pollution exposures are associated with higher carotid IMT in MESA,³⁴³² and African-American, Hispanic and Chinese participants have higher air pollution levels than Caucasian-American participants,¹⁸ we hypothesised that the indirect association would be positive for all race/ethnic groups compared with Caucasian-American participants. We estimated 95% CIs for the indirect association by bootstrapping with 2000 resamplings using the mediation package (V.4.3.1) in R.^{46–50}

As a comparison, we also evaluated the indirect association of race/ethnicity on IMT through differences in systolic blood pressure, a known risk factor for atherosclerosis that explains differences in cardiovascular mortality by race/ethnicity in the US population.⁵¹ Since systolic blood pressure levels are also higher in African-American, Hispanic and Chinese participants compared with Caucasian-American participants,⁵²⁵³ and blood pressure levels are also positively associated with carotid IMT,^{54–57} we also hypothesised that the indirect association would be positive for all three race/ethnic groups compared with Caucasian-American participants. All statistical analyses were performed using R software (V.3.0.1).⁵⁸ All statistical tests were two-sided and CIs were set at 95%.

RESULTS

Participant characteristics

Among the study participants, 39% were Caucasian-American, 27% were African-American, 22% were Hispanic and 12% were Chinese. Forty-seven per cent of participants were male and the mean age was 62 years, with no differences by race/ethnicity. IMT was highest for African-American women and men and lowest for Chinese women and men compared with other races/ethnicities (table 1).

PM_{2.5} concentrations were higher in Chinese participants (geometric mean 19.3 µg/m³ for men and women), followed by Hispanic (17.2 µg/m³ for women and 16.8 µg/m³ for men) and African-American (16.5 µg/m³ for men and women) participants, and lowest in Caucasian-American participants (15.7 µg/m³ for men and 15.6 µg/m³ for women) (table 1). NO_x concentrations followed similar patterns (geometric means 59.0, 56.8, 42.5 and 33.0 ppb for Chinese, Hispanic, African-American and Caucasian-American men, and 60.3, 61.0,

43.9 and 34.4 ppb for Chinese, Hispanic, African-American and Caucasian-American women).

Results among women

After adjustment for demographics, cardiovascular disease risk factors and metropolitan area, the mean common carotid IMT was larger for African-American women compared with Caucasian-American women (difference: 23.4 μm , 95% CI 7.5 to 39.3) and smaller for Hispanic and Chinese women compared with Caucasian-American women (difference (95% CI) -19.4 μm (-42.8 to 3.9) and -53.6 μm (-87.9 to -19.3) for Hispanic and Chinese women, respectively) (table 2, 'total association'). After adjustment for differences in $\text{PM}_{2.5}$ concentrations, IMT differences remained similar for African-American and Hispanic participants but became even smaller for Chinese women compared with Caucasian-American women (mean difference -58.4 μm) ('direct association'). The difference (95% CI) in IMT levels by race/ethnicity that was related to their higher $\text{PM}_{2.5}$ exposures compared with Caucasian-American women ('indirect associations') were around 0 for African-American and Hispanic women and 4.8 μm (95%CI 0.2 to 10.8) for Chinese women. For NO_x , we found little evidence in support of an influence on racial/ethnic differences in IMT (table 2).

Results among men

After adjustment for demographics, cardiovascular disease risk factors and metropolitan area, the mean common carotid IMT was significantly larger for African-American men compared with Caucasian-American men (difference: 38.7 μm , 95% CI 19.6 to 57.7) and significantly smaller for Hispanic and Chinese men compared with Caucasian-American men (difference (95% CI) -37.6 μm (-65.0 to -10.3) and -7.1 μm (-41.2 to 27.0) for Hispanic and Chinese men, respectively) (table 2). Adjusting for $\text{PM}_{2.5}$ concentrations intensified the inverse association for Chinese men compared with Caucasian-American men, with the mean IMT changing from -7.1 to -15.7 μm smaller. The difference in IMT levels in Chinese men compared with Caucasian-American men related to their higher $\text{PM}_{2.5}$ exposures ('indirect association') was 8.6 (95% CI 3.4 to 15.3) μm . Exposure to $\text{PM}_{2.5}$ did not influence differences in IMT for African-American and Hispanic men compared with Caucasian-American men, although the indirect association estimates were positive. Differences in NO_x exposure among African-American, Hispanic and Chinese participants were not associated with differences in common carotid IMT compared with Caucasian-American participants (table 2).

Comparison to systolic blood pressure

We compared our findings of the influence of air pollution exposure to racial differences in IMT with the influence of systolic blood pressure, a known cardiovascular risk factor that has been related to race/ethnic disparities in cardiovascular disease.⁵¹ Systolic blood pressure was highest among African-American women and men compared with other races/ethnicities (table 1). Among women, systolic blood pressure was lowest in Caucasian-American women. Among men, systolic blood pressure was lowest in Caucasian-American and Chinese men. After adjustment for sociodemographic and cardiovascular risk factors, accounting for differences in systolic blood pressure attenuated the positive association on

carotid IMT for African-American women and intensified the inverse association for Hispanic women and Chinese women compared with Caucasian-American women (indirect associations (95% CI) 7.1 (3.9 to 10.5) μm , 7.0 (2.0 to 7.7) μm and 4.4 (0.0 to 9.5) μm for African-American, Hispanic and Chinese women, respectively) (table 3). Systolic blood pressure also similarly influenced differences in carotid IMT for African-American, Hispanic and Chinese men compared with Caucasian-American men, with positive indirect associations for all race/ethnic groups, although the indirect association for Hispanic men was not statistically significant (indirect associations (95% CI) 3.5 (1.0 to 6.9) μm , 2.3 (–1.6 to 7.1) μm and 8.3 (1.2 to 16.7) μm , respectively).

DISCUSSION

We found that the association of Chinese ethnicity with smaller carotid IMT compared with Caucasian-American became stronger after accounting for the higher $\text{PM}_{2.5}$ exposures among Chinese men and women compared with their Caucasian-American counterparts. The influence of $\text{PM}_{2.5}$ concentrations on IMT differences for Chinese women and men were consistent with our initial hypothesis and similar in magnitude to the influence of systolic blood pressure, an established risk factor for atherosclerosis. $\text{PM}_{2.5}$ exposures, however, did not influence IMT differences for African-American and Hispanic participants compared with Caucasian-American participants. The lack of influence of $\text{PM}_{2.5}$ for African-American and Hispanic participants in explaining differences in carotid IMT levels could be due to the relatively small differences in $\text{PM}_{2.5}$ exposure levels for African-American and Hispanic participants compared with Caucasian-American participants in MESA. Differences in IMT by race/ethnicity were not influenced by increased exposure to NO_x .

No previous study has examined the role of air pollution exposure to ethnic differences in atherosclerosis and few studies have examined the role of air pollution exposure to racial/ethnic health disparities,^{35–38,59–61} with most studies focusing on respiratory outcomes^{36,37,59–61} and only one study including cardiovascular outcomes (hospital admissions for cardiovascular events).³⁶ Most of these studies were ecological and examined racial/ethnic composition and patterns of air pollution exposure with regional-level health outcomes without the ability to account for individual risk factors or to evaluate associations separately for men and women.^{35–38} A study conducted in California examined racial/ethnic disparities in cardiovascular and respiratory hospital admissions and asthma emergency room visit rates resulting from exposure to ozone and $\text{PM}_{2.5}$ concentrations in excess of federal standards.³⁶ This study used zip code-level daily average air pollution exposures and hospital event rates from state databases to calculate excess attributable risk over 2005–2007 for Caucasian-American, African-American, Hispanic and Asian/Pacific Islander residents by zip code and found that for cardiovascular admissions African-American residents experienced significantly higher excess attributable risk compared with Caucasian-American residents, and Hispanic and Asian/Pacific Islander residents experienced significantly lower excess attributable risk compared with Caucasian-American residents; this study, however, did not evaluate air pollution exposure as a mediator of the association between race/ethnicity and hospital event rates.

The findings for Chinese participants in MESA could potentially have implications for cardiovascular disease prevention in this community. Chinese Americans are more likely to live in urban settings in the USA, particularly in central city areas.^{62,63} In MESA, Chinese participants were exposed to the highest levels of air pollution of all race/ethnic groups evaluated after adjustment for metropolitan area.¹⁸ Chinese participants in MESA had less atherosclerosis than Caucasian-American participants, and after adjustment for PM_{2.5} they had even less. It is well established that Chinese Americans have less cardiovascular disease risk than other US race/ethnic groups.^{64–66} The reasons have not been fully evaluated, although they could be related to differences in diet, physical activity, genetics or social support. Analysing the reasons for less atherosclerosis in Chinese participants compared with Caucasian-American participants in MESA is beyond the scope of this study, as our objective was to evaluate the role of air pollution exposures in race/ethnic differences in carotid IMT. Despite their smaller IMT, Chinese participants are being exposed to higher concentrations of air pollution, and this exposure is having an impact on their cardiovascular health. Reducing exposure to PM_{2.5} among Chinese individuals to levels similar to those experienced by Caucasian-American individuals could have similar impacts in this population as reducing systolic blood pressure levels.

The interpretation of further improvement in atherosclerosis after adjustment for PM_{2.5} concentrations in Chinese participants compared with Caucasian-American participants could be related to either confounding or mediation. Studies comparing changes in cardiovascular disease in Asian populations migrating to the USA have focused on Japanese American Communities. Coronary heart disease incidence and mortality increased relatively rapidly and markedly among Japanese men who migrated from Japan to Hawaii and California.^{67,68} Those studies were conducted in the 1970s and 1980s and did not consider the potential role of air pollution. In China, where exposure to air pollution has skyrocketed in recent years, quasi-experimental analyses have shown important declines in life expectancy, at least partly due to increases in cardiovascular disease mortality.^{69,70} If our findings are related to mediation, we could hypothesise that reducing air pollution exposure in communities where Chinese Americans live could further improve their risk of cardiovascular disease. We compared our findings of the influence of air pollution exposure to racial/ethnic differences in IMT with the influence of systolic blood pressure, an established risk factor for atherosclerosis that has previously been shown to explain differences in cardiovascular mortality by race/ethnicity.⁵¹ A full investigation of the influence of systolic blood pressure on racial/ethnic differences in atherosclerosis, independent of air pollution exposure, was not the goal of this manuscript and is beyond its scope.

This study has several strengths including individual-level objective measures of air pollution exposure, the evaluation of racial/ethnic differences in a multiethnic cohort, and the high-quality standardised protocol. Our study is also strengthened in the assessment of the influence of air pollution exposure to racial/ethnic differences in atherosclerosis by using statistical analyses that formally test for the significance of this mediation. The estimation of the indirect association assumes no feedback (reverse causation) between the independent variable, dependent variable and mediator, and the effects of these variables in the mediation model are not confounded by unmeasured variables.^{47,48} It is very difficult to assess if these

assumptions were met in this epidemiological study. Although race/ethnicity cannot be caused by either air pollution exposure or IMT, different races/ethnicities might choose to live in areas that are more or less polluted due to economic and social factors that are difficult to adjust for. We assessed air pollution exposure using concentrations of PM_{2.5} and NO_x accounting for metropolitan area in order to examine the impact of exposure to ambient air pollution specifically and not just differences that may arise due to place of residence. Metropolitan area, however, is a major determinant of air pollution, and adjustment for it could induce overadjustment. Although unmeasured confounding is possible, in our study we were able to account for important confounding risk factors including geographic location which can serve as a surrogate for other environmental and medical care variables that are difficult to adjust for. We estimated the influence of air pollution exposure to racial/ethnic differences in IMT after adjusting for cardiovascular risk factors. However, some of these risk factors could potentially be mediators in the relationship between air pollution exposure and IMT. Sensitivity analyses without adjustment for diabetes, blood pressure and cholesterol (potential mediators of the association between race/ethnicity and cardiovascular disease that could also be mediators of the association between air pollution and atherosclerosis) did not change our overall conclusions in this study (data not shown).

Some limitations should be taken into account. MESA participants were enrolled at each study site with the intent of having specific distributions across strata defined by race/ethnicity gender and age group and not by random sample of the population; this sampling strategy may have resulted in populations that are different from the general population for each race/ethnicity, especially in terms of representing the typical air pollution exposure levels that are characteristic of each race/ethnic group. Few studies have examined racial/ethnic differences in air pollution exposure comparing Chinese individuals to Caucasian-American individuals,¹⁸ and it is unknown if the differences in exposure levels observed by participants in MESA cities are representative of Chinese American participants across the US population. In addition, MESA participants were required to be free of clinical cardiovascular disease at baseline, which may have resulted in healthier individuals than in the general population, and this could have been differential by race/ethnicity as prevalence rates of cardiovascular disease are different across races/ethnicities. Potentially, this could result in smaller differences comparing cardiovascular outcomes by race/ethnicity and limit our ability to detect the relatively small impacts expected due to air pollution exposures. Additionally, we cannot discard the possibility that cardiovascular disease disparities related to differential air pollution exposure may be related to the triggering of cardiovascular events rather than the changes in long-term subclinical atherosclerosis examined in this study. In our study, mediation and confounding could be possible interpretations for an influence of PM_{2.5} concentrations comparing carotid IMT levels in Chinese versus Caucasian-American participants.

CONCLUSIONS

In this study, consistent with our initial hypothesis, adjustment for the higher exposure to PM_{2.5} concentrations among Chinese participants intensified their smaller carotid IMT levels compared with Caucasian-American participants. This finding was independent of

socioeconomic status. In a sensitivity analysis without adjustment for education and income in the statistical model, the estimates of the indirect association were attenuated but remained statistically significant for PM_{2.5} for Chinese men but not Chinese women (although the direction of the association was the same). An analysis further adjusting our current models for neighbourhood socioeconomic status, assessed using the census tract median family income, resulted in similar estimates of the indirect association, although the indirect association remained statistically significant for the influence of PM_{2.5} only in Chinese men while in Chinese women it was only borderline significant.

Reducing ambient air pollution exposure among Chinese individuals may further advance the cardiovascular health in this population group. For African-American and Hispanic participants, differences in PM_{2.5} concentrations were small and did not influence differences in carotid IMT levels compared with Caucasian-American participants. Additional studies in African-American and Hispanic populations exposed to levels of air pollution that are higher than in MESA are probably needed to confirm or discard the role of air pollution in explaining racial disparities in cardiovascular disease. The direction and magnitude of the indirect association estimate among Chinese compared with Caucasian-American participants was similar for blood pressure and PM_{2.5} levels. Overall, this study highlights the complexities and importance of evaluating the role of environmental exposures, such as ambient air pollution to racial/ ethnic differences in cardiovascular disease outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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What is already known on this subject

Epidemiological studies have shown increased risk for atherosclerosis associated with exposure to ambient air pollution. Risk for atherosclerosis and exposure to air pollution differ by race/ethnicity in the USA. Previous studies, however, have not evaluated the contribution of air pollution exposure to ethnic differences in atherosclerosis.

What this study adds

Using data on individual cardiovascular risk factors and household-level air pollution exposure, our study found that the association of Chinese ethnicity with smaller carotid intima-media thickness (IMT) compared with Caucasian- American became stronger after accounting for the higher PM_{2.5} (particles <2.5 µm in aerodynamic diameter) exposures among Chinese men and women. The influence of PM_{2.5} concentrations on IMT differences for Chinese participants was similar in magnitude to the influence of systolic blood pressure, an established risk factor for atherosclerosis.

Figure 1.

The total association (A) and the decomposition of the total association of race/ethnicity on intima-media thickness (IMT) into direct and indirect associations (B). The total association of race/ethnicity on IMT is decomposed into the direct association of race/ethnicity on IMT that is not due to air pollution exposure and the indirect association of race/ethnicity on IMT that is related to the difference in air pollution exposure by race/ethnicity. For simplicity, sociodemographic and cardiovascular risk factors are not shown in the figure and are considered to be fully accounted for in the analysis.

Table 1

Participant characteristics by race/ethnicity and stratified by gender, 2000–2002

	Women				Men			
	Caucasian-American	African-American	Hispanic	Chinese	Caucasian-American	African-American	Hispanic	Chinese
Number of participants	1295	965	721	375	1188	780	663	360
Age, year	62.6 (10.3)	62.2 (10.0)	61.6 (10.4)	62.3 (10.4)	62.6 (10.1)	62.3 (10.1)	61.4 (10.3)	62.5 (10.3)
Education								
<High school	5.8	11.2	48.0	31.5	4.1	12.4	40.3	16.9
High school	21.1	19.2	22.3	21.1	12.5	19.1	18.4	11.1
Some college	31.7	35.1	22.5	21.3	23.8	34.1	28.2	19.7
College degree	41.4	34.5	7.2	26.1	59.6	34.4	13.1	52.2
Income								
<\$24 999	20.5	31.9	53.4	53.6	10.5	21.9	40.9	41.9
\$25 000–\$49 999	29.0	32.8	30.9	21.6	23.0	26.3	34.2	23.1
\$50 000–\$74 999	18.5	16.5	8.7	10.7	21.9	21.2	13.1	13.1
\$75 000	29.4	11.9	4.2	13.3	42.3	21.8	10.4	21.9
Unknown	2.5	6.8	2.8	0.8	2.3	8.8	1.4	0.0
Smoking								
Never	49.2	52.7	68.9	95.7	40.1	37.1	40.0	53.1
Former	39.2	31.3	21.2	2.4	49.1	43.3	43.7	37.2
Current	11.6	16.0	9.8	1.9	10.9	19.6	16.3	9.7
Pack-years of smoking*	23.3 (22.6)	20.0 (21.6)	11.8 (17.7)	17.8 (24.9)	31.3 (39.3)	23.3 (21.7)	20.2 (22.5)	20.4 (23.0)
Body mass index, kg/m ²	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 (4.3)	24.1 (3.1)
Systolic BP, mm Hg	122.9 (22.0)	132.8 (22.9)	127.5 (23.5)	125.0 (23.2)	124.2 (18.6)	130.2 (19.5)	126.0 (20.2)	124.0 (19.3)
Diastolic BP, mm Hg	66.9 (9.6)	72.5 (10.3)	68.3 (9.5)	69.3 (10.5)	73.9 (9.1)	77.1 (9.6)	74.9 (9.6)	75.0 (9.3)
Diabetes								
Normal	86.4	72.2	70.2	74.9	78.6	63.3	62.7	64.2
Impaired	8.9	12.0	13.0	12.8	13.8	16.9	18.3	21.1
Diabetes	4.7	15.8	16.8	12.3	7.6	19.7	19.0	14.7
Total cholesterol, mg/dL	202.4 (34.6)	196.5 (36.6)	202.3 (38.1)	195.0 (31.9)	189.0 (34.7)	181.8 (34.1)	193.6 (37.5)	189.4 (31.4)
HDL cholesterol, mg/dL	58.7 (15.7)	57.1 (15.8)	52.5 (13.8)	53.4 (13.2)	45.3 (12.2)	46.7 (12.5)	42.7 (10.2)	45.7 (11.0)

	Women				Men			
	Caucasian-American	African-American	Hispanic	Chinese	Caucasian-American	African-American	Hispanic	Chinese
Medication use, % yes								
Statin use	14.9	16.8	13.0	15.2	18.2	14.4	11.9	11.9
Antihypertensive	26.5	50.6	32.9	30.9	26.2	44.1	26.8	24.4
Common CIMT, μm	842 (183)	884 (187)	838 (177)	811 (182)	892 (209)	936 (198)	880 (202)	844 (166)
PM _{2.5} concentration, $\mu\text{g}/\text{m}^3$	15.7	16.5	17.2	19.3	15.6	16.5	16.8	19.3
NO _x concentration, ppb	34.4	43.9	61.0	60.3	33.0	42.5	56.8	59.0

Values represent percentages for categorical variables and means (SD) for continuous variables except PM_{2.5} and NO_x for which geometric means are reported.

* Pack years of smoking for former and current smokers only.

BP, blood pressure; CIMT, carotid intima-media thickness; HDL, high-density lipoprotein; PM_{2.5}, particles <2.5 μm in aerodynamic diameter.

Table 2

Mean difference (95% CI) in common carotid IMT (μm) for African-American, Hispanic and Chinese participants compared with Caucasian-American participants, 2000–2002*

	N	Total association	PM _{2.5} concentrations		NO _x concentrations		
			Direct association	Indirect association	Direct association	Indirect association	
Women							
Caucasian-American	1295	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	
African-American	965	23.4 (7.5 to 39.3)	23.2 (7.2 to 39.2)	0.2 (−1.0 to 1.7)	23.6 (7.6 to 39.5)	−0.2 (−1.1 to 0.4)	
Hispanic	721	−19.4 (−42.8 to 3.9)	−19.3 (−42.6 to 4.0)	−0.1 (−1.3 to 0.9)	−19.7 (−43.2 to 3.7)	0.3 (−2.1 to 2.9)	
Chinese	375	−53.6 (−87.9 to −19.3)	−58.4 (−93.1 to −23.8)	4.8 (0.2 to 10.8)	−54.4 (−88.7 to −20.1)	0.8 (−1.4 to 4.2)	
Men							
Caucasian-American	1188	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	0.0 (ref)	
African-American	780	38.7 (19.6 to 57.7)	38.3 (19.2 to 57.4)	0.4 (−0.8 to 1.7)	39.5 (20.4 to 58.6)	−0.8 (−2.4 to 0.3)	
Hispanic	663	−37.6 (−65.0 to −10.3)	−37.8 (−65.2 to −10.4)	0.2 (−0.9 to 1.7)	−37.1 (−64.5 to −9.6)	−0.6 (−3.3 to 1.5)	
Chinese	360	−7.1 (−41.2 to 27.0)	−15.7 (−50.1 to 18.7)	8.6 (3.4 to 15.3)	−7.4 (−41.4 to 26.6)	0.2 (−3.3 to 3.8)	

Adjusted for age, education, income, smoking status, pack-years of smoking, body mass index, diabetes, systolic blood pressure, total cholesterol, HDL cholesterol, antihypertensive medication use, statin use and metropolitan area.

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

HDL, high-density lipoprotein; IMT, intima-media thickness; PM_{2.5}, particles <2.5 μm in aerodynamic diameter.

Table 3

Contribution of systolic blood pressure to mean difference in common carotid intima-media thickness (IMT) (μm) for African-American, Hispanic and Chinese participants compared with Caucasian-American participants after adjusting for demographics and cardiovascular disease risk factors, 2000–2002*

	Total association	Direct association	Indirect association
Women			
Caucasian- American	0.0 (ref)	0.0 (ref)	0.0 (ref)
African- American	30.5 (14.4 to 46.6)	23.4 (7.5 to 39.3)	7.1 (3.9 to 10.5)
Hispanic	−12.4 (−36.2 to 11.4)	−19.4 (−42.8 to 3.9)	7.0 (2.1 to 13.1)
Chinese	−45.3 (−80.0 to −10.6)	−53.6 (−87.9 to −19.3)	8.3 (1.7 to 16.0)
Men			
Caucasian- American	0.0 (ref)	0.0 (ref)	0.0 (ref)
African- American	42.1 (23.0 to 61.3)	38.7 (19.6 to 57.7)	3.5 (1.0 to 6.9)
Hispanic	−35.4 (−63.0 to −7.7)	−37.6 (−65.0 to −10.3)	2.3 (−1.6 to 7.1)
Chinese	1.2 (−33.5 to 35.8)	−7.1 (−41.2 to 27.0)	8.3 (1.2 to 16.7)

95% CIs shown in parentheses.

* The total association represents the mean difference in IMT by race/ethnicity after adjustment for cardiovascular risk factors. The total association is decomposed into the indirect association (ie, the mean difference in IMT by race/ethnicity that is due to the difference in systolic blood pressure by race/ethnicity) and the direct association of race/