Original Contribution

Do Psychosocial Stress and Social Disadvantage Modify the Association Between Air Pollution and Blood Pressure?

The Multi-Ethnic Study of Atherosclerosis

Margaret T. Hicken, Sara D. Adar, Ana V. Diez Roux, Marie S. O'Neill, Sheryl Magzamen, Amy H. Auchincloss, and Joel D. Kaufman

Correspondence to Dr. Margaret T. Hicken, Department of Epidemiology, School of Public Health, University of Michigan, 3634 SPH Tower, 1416 Washington Heights, Ann Arbor, MI 48109 (e-mail: mhicken@umich.edu).

Researchers have theorized that social and psychosocial factors increase vulnerability to the deleterious health effects of environmental hazards. We used baseline examination data (2000–2002) from the Multi-Ethnic Study of Atherosclerosis. Participants were 45–84 years of age and free of clinical cardiovascular disease at enrollment ($n = 6814$). The modifying role of social and psychosocial factors on the association between exposure to air pollution comprising particulate matter less than 2.5 µm in aerodynamic diameter ($PM_{2.5}$) and blood pressure measures were examined using linear regression models. There was no evidence of synergistic effects of higher $PM_{2.5}$ and adverse social/psychosocial factors on blood pressure. In contrast, there was weak evidence of stronger associations of $PM_{2.5}$ with blood pressure in higher socioeconomic status groups. For example, those in the 10th percentile of the income distribution (i.e., low income) showed no association between $PM_{2.5}$ and diastolic blood pressure (b = −0.41 mmHg; 95% confidence interval: −1.40, 0.61), whereas those in the 90th percentile of the income distribution (i.e., high income) showed a 1.52-mmHg increase in diastolic blood pressure for each 10-µg/m^3 increase in $PM_{2.5}$ (95% confidence interval: 0.22, 2.83). Our results are not consistent with the hypothesis that there are stronger associations between $PM_{2.5}$ exposures and blood pressure in persons of lower socioeconomic status or those with greater psychosocial adversity.

air pollution; blood pressure; population groups; social environment; social medicine; social psychology

A large body of work has assessed the health impacts of social factors and environmental hazards, although this work has generally appeared in separate disciplinary literatures. Consensus is growing on the need to investigate their joint effects, as they are often spatially correlated, may operate through common biological mechanisms, and may act synergistically to affect health (1–6). Indeed, the United States Air Pollution Prevention and Control Act (also known as the Clean Air Act) requires that the National Ambient Air Quality Standards protect populations that may be particularly vulnerable to the health effects of air pollution (7). It is therefore a public

health imperative to understand the factors that increase vulnerability to the health effects of air pollution.

There is some evidence that social and psychosocial factors may be important modifiers of the associations between air pollution and health. Socially and economically disadvantaged groups, such as blacks and those of low socioeconomic status (SES), exhibit stronger associations between air pollution and health than do whites or persons in higher SES groups, respectively (8–11), although this finding has not been consistent across all studies (12–14). Evidence has also suggested that psychosocial stress increases the harmful effects

of air pollution on asthma (4, 15, 16) and other respiratory diseases (17).

Although several studies have focused on respiratory or mortality outcomes, a growing body of work has documented positive associations between particulate matter less than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) and cardiovascular disease (CVD) (5, 18–20) and blood pressure (21, 22). A parallel body of work has documented associations of social disadvantage and psychosocial factors with CVD (23–25), including hypertension (26–29). The impact of air pollution on cardiovascular outcomes may be modified by social disadvantage or psychosocial factors. Limited evidence has suggested that social disadvantage enhances the unhealthy associations between $PM_{2.5}$ and health (9, 30, 31), although results have not always been consistent (32), and the research on the interactive associations between air pollution and social or psychosocial factors and blood pressure is scant.

We used data from the Multi-Ethnic Study of Atherosclerosis (MESA) and the MESA and Air Pollution (MESA Air) Study to investigate the extent to which short-term exposures to $PM_{2.5}$ and social and psychosocial exposures may act together to affect blood pressure. This work builds on findings of a cross-sectional association between $PM_{2.5}$ and baseline blood pressure in MESA (21) by exploring the modifying role of social and psychosocial factors. Specifically, we examined 1) whether SES or race/ethnicity modified the association between $PM_{2.5}$ and blood pressure, and 2) whether psychosocial factors previously linked to changes in blood pressure modified the association between $PM_{2.5}$ and blood pressure. We hypothesized that associations of $PM_{2.5}$ with blood pressure would be stronger in socially disadvantaged groups and in persons with greater levels of psychosocial adversity.

MATERIALS AND METHODS

Study population and data

Data came from the baseline examination of MESA, a longitudinal study in 6 sites (Baltimore, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles County, California; Northern Manhattan, New York; and St. Paul, Minnesota). Details of the MESA cohort have been published elsewhere (33). Briefly, 6,814 adults of white or black race or Hispanic or Chinese ethnicity who were between the ages of 45–85 years and free of clinical CVD were recruited for the study in 2000–2002 through various population-based approaches. Although MESA is currently a longitudinal study, the present study builds on prior work using baseline data only (21).

On the basis of prior work (21), systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP), and mean arterial pressure (MAP) were the outcomes examined. Blood pressure was measured using the Critikon Dinamap Pro 100 (Critikon, Tampa, Florida). The average of the second and third of 3 seated blood pressure readings taken 2 minutes apart were used in the analyses. PP was calculated as SBP minus DBP. MAP was calculated as follows: $(((2 \times DBP) + SBP)/3)$.

To focus analyses on temporal rather than spatial variation in exposure, daily $PM_{2.5}$ estimates were calculated for each

MESA site using the average of all United States Environmental Protection Agency Air Quality System monitoring stations within each MESA site with complete time series during the 2000–2002 study period. Data from all such monitors within the boundaries of the following areas were included in the site-wide averages: Baltimore County, Maryland; Cook County, Illinois; Los Angeles County, California; Bronx County, New York; Ramsey and Hennepin Counties, Minnesota; and Forsyth County, North Carolina. Each participant was assigned the $PM_{2.5}$ concentration for that site for the month prior to their examination visit. On the basis previous work that showed that the most consistent associations of $PM_{2.5}$ with blood pressure were observed for the average exposure for the month prior to the examination (21), we used a prior 1-month average $PM_{2.5}$ as the key measure, with other averaging periods examined in sensitivity analyses.

Social disadvantage was measured using race/ethnicity and SES information that were collected via questionnaire. Race/ethnicity was categorized as: non-Hispanic white, Chinese, non-Hispanic black, and Hispanic. Educational level was examined as a continuous variable representing the number of years of education. Family income for the previous 12 months was reported at each examination and divided into 13 categories. Income was operationalized as continuous by using the midpoint of the reported category. Median household income in the census tract of the respondent was collected from the 2000 Census.

The psychosocial factors examined included chronic stress, depressive symptoms, trait anger, trait anxiety, and lack of emotional support. The measure of chronic stress was a chronic burden of stress scale collected via questionnaire (34). Chronic stress was measured as the sum of the number of times the participant affirmed the current presence of a moderately or very stressful ongoing problem in any of 5 domains (own health, close person's health, job, finances, or relationship) that lasted more than 6 months. The range of the scale was 0 to 5. Depressive symptoms were assessed via responses to 20 questions from the Center for Epidemiologic Studies Depression Scale (35) about the frequency of depressive feelings and behaviors over the previous week. Responses were on a Likert-like scale ranging from 1 (<1 day) to 4 (5–7 days). The range of the scale was 20 to 80. Trait anger and trait anxiety scores were determined as the sum of the responses to 10 questions each from the Spielberger Trait Anger Expression Inventory and Spielberger Trait Anxiety Inventory about the current frequency with which the participant had anger-related or anxiety-related feelings and behaviors, respectively (36). The responses on were on a Likert-like scale ranging from 1 (almost never) to 4 (almost always), with the range of the scale 10 to 40. Emotional support was measured as the sum of the responses to 6 questions from the Emotional Social Support Index about the frequency of the respondent's perceived availability of current emotional support. Responses were on Likert-like scale ranging from 1 (all of the time) to 5 (none of the time). The items were reverse-coded so that greater values represented lack of emotional support, which is a more adverse psychosocial situation,. The range of the scale was 6 to 30.

Additional covariates included sociodemographic and health-related measures. Data on sex, age (in years), current alcohol use (yes or no), current diabetes status (yes or no, using American
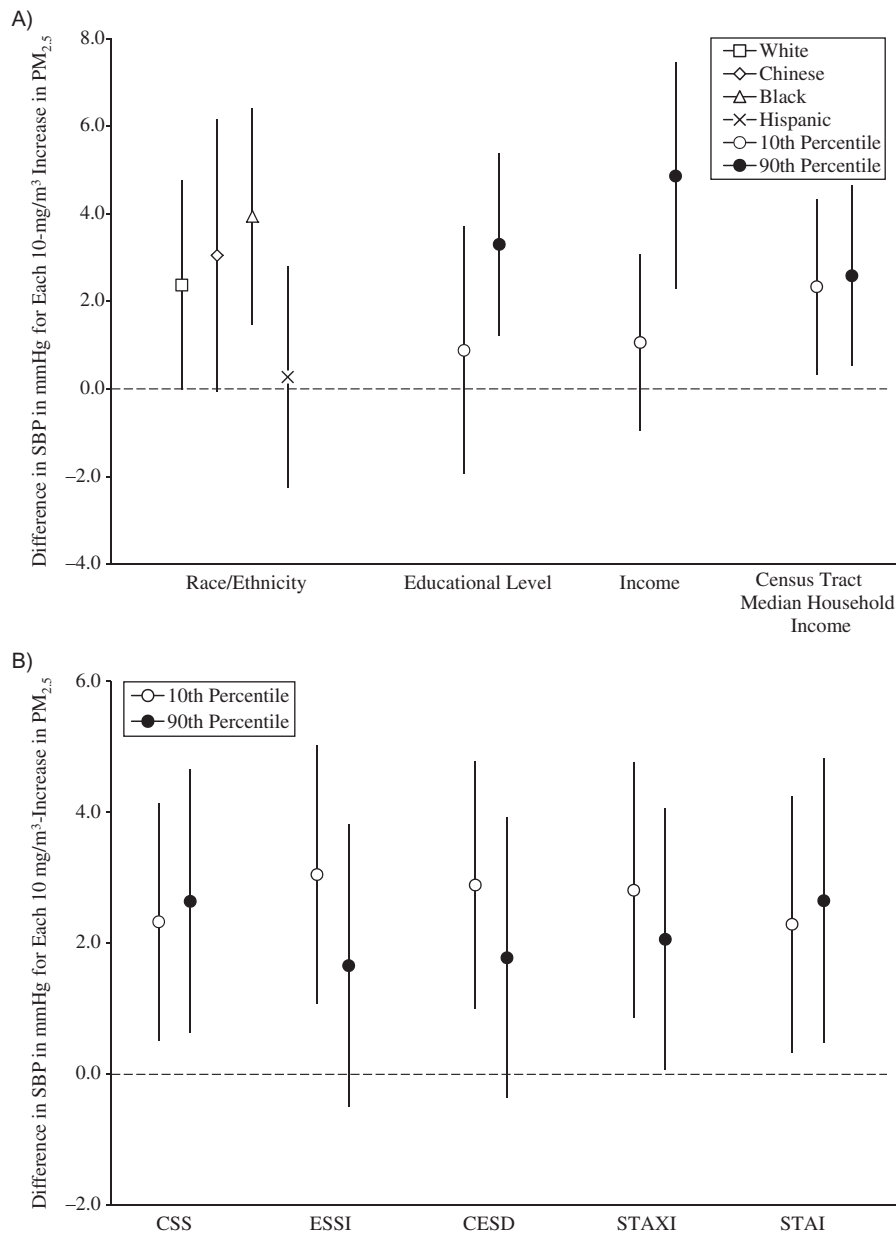
Figure 1. Associations between concentrations of particulate matter less than 2.5μm in aerodynamic diameter (PM₂.₅) and systolic blood pressure (SBP), by social disadvantage and psychosocial level or category, Multi-Ethnic Study of Atherosclerosis, 2000–2002. A) Associations by social disadvantage level or category. B) Associations by psychosocial level. All socioeconomic measures were operationalized so that higher values represented a greater socioeconomic resource. All psychosocial measures were operationalized so that higher values represent greater adversity. The scale of the psychosocial measures is per 1 unit. Models were adjusted for age, sex, season, site, season-by-site interaction, antihypertensive medication use, apparent temperature, and all other socioeconomic and psychosocial variables in the figure. Results are the difference in SBP per 10-μg/m³ increase in PM₂.₅ exposure averaged over the month prior to the examination. Income and census tract median household income values are per $10,000. CESD, Center for Epidemiologic Studies Depression Scale; CSS, Chronic Stress Scale; ESSI, Emotional Social Support Index (reverse-coded); STAI, Spielberger Trait Anxiety Inventory; STAXI, Spielberger Trait Anger Expression Inventory. Bars, 95% confidence intervals.

Diabetes Association 2003 criteria (37)), and antihypertensive medication use were collected via questionnaire. Body mass index was calculated from measured height and weight. A categorical variable to capture the combined role of smoking and exposure to second-hand smoke (ETS) was created from reports

of smoking and ETS. The categories were current smoker, former smoker with less than 1 hour/week of ETS, former smoker with 1 or more hours/week of ETS, (d) never smoker with less than 1 hour/week of ETS, and never smoker with 1 or more hours/week of ETS. Apparent temperature for the month prior to the
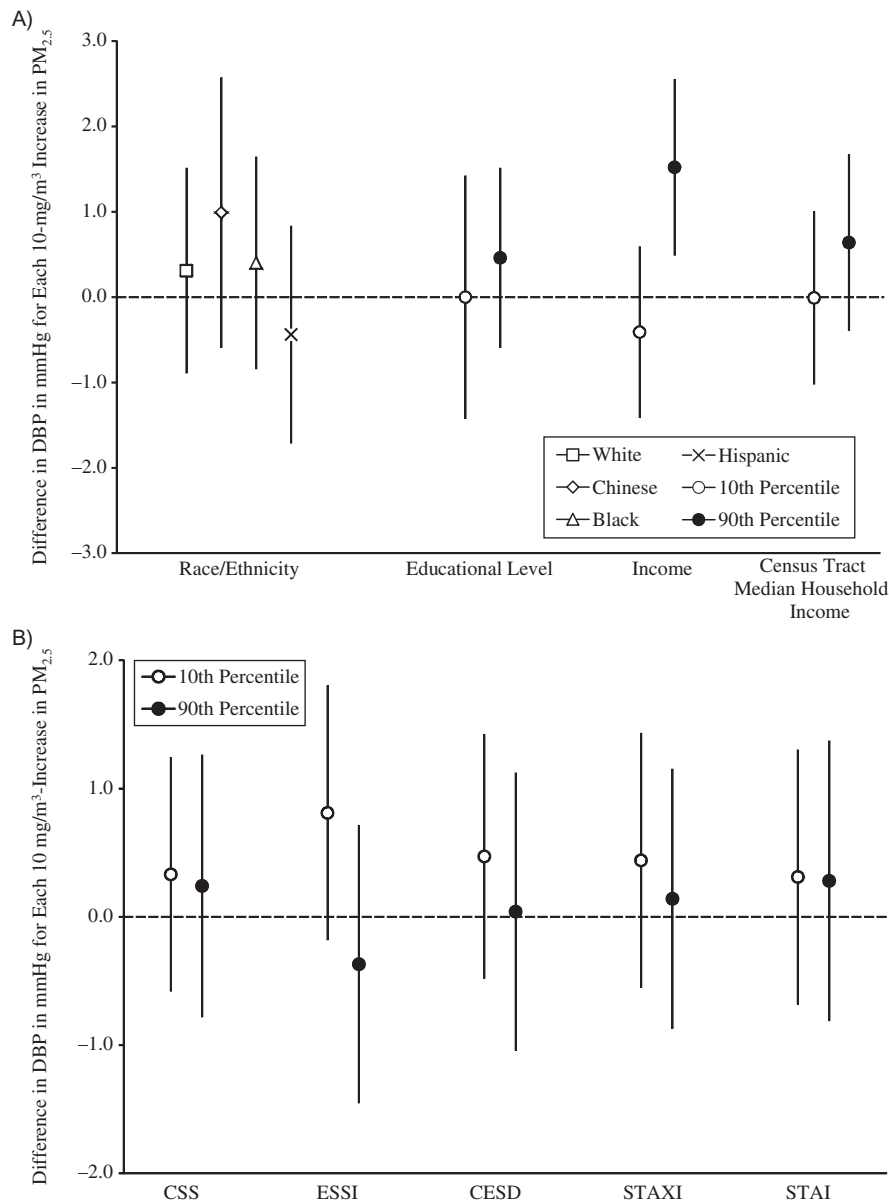
Figure 2.   Associations between concentrations of particulate matter less than 2.5μm in aerodynamic diameter (PM$_{2.5}$) and diastolic blood pressure (DBP), by social disadvantage and psychosocial level or category, Multi-Ethnic Study of Atherosclerosis, 2000–2002. A) Associations by social disadvantage level or category. B) Associations by psychosocial level. All socioeconomic measures were operationalized so that higher values represented a greater socioeconomic resource. All psychosocial measures were operationalized so that higher values represent greater adversity. The scale of the psychosocial measures is per 1 unit. Models were adjusted for age, sex, season, site, season-by-site interaction, anti-hypertensive medication use, apparent temperature, and all other socioeconomic and psychosocial variables in the figure. Results are the difference in DBP per 10-μg/m^3 increase in PM$_{2.5}$ exposure averaged over the month prior to the examination. Income and census tract median household income values are per $10,000. CESD, Center for Epidemiologic Studies Depression Scale; CSS, Chronic Stress Scale; ESSI, Emotional Social Support Index (reverse-coded); STAI, Spielberger Trait Anxiety Inventory; STAXI, Spielberger Trait Anger Expression Inventory. Bars, 95% confidence intervals.

examination was calculated using relative humidity, temperature, and an equation from the National Oceanic and Atmospheric Administration (38). Because apparent temperature is related to blood pressure in a nonlinear fashion, splines were created with a knot at 11.4°C based on exploratory analyses. The month of each examination was noted, and variables to

capture season (January–March, April–June, July–September, and October–December) were created as was done in a previous study (21).

The number of participants at the baseline examination was 6,814. No information on 1-month average PM$_{2.5}$ concentration was available for 811 persons, and information on blood
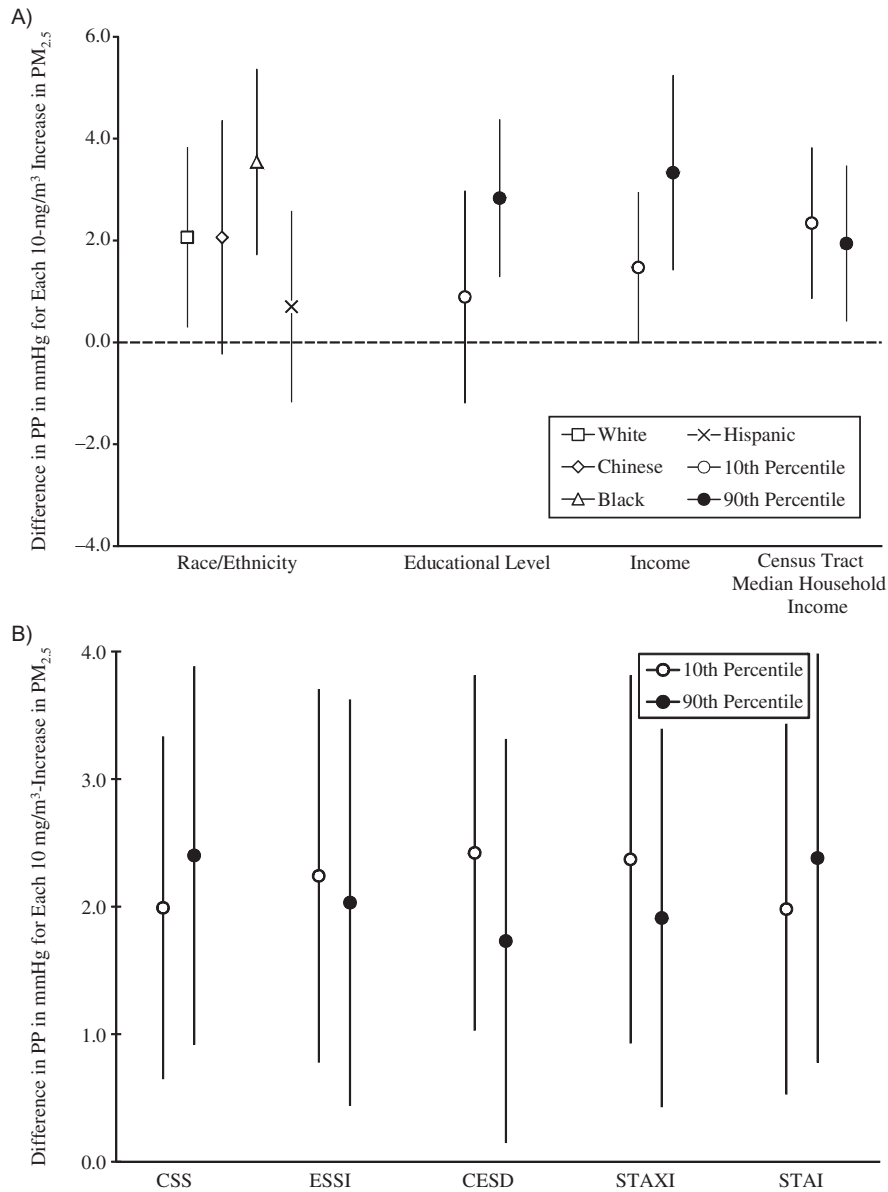
Figure 3. Associations between concentrations of particulate matter less than 2.5µm in aerodynamic diameter ($PM_{2.5}$) and pulse pressure (PP), by social disadvantage and psychosocial level or category, Multi-Ethnic Study of Atherosclerosis, 2000–2002. A) Associations by social disadvantage level or category. B) Associations by psychosocial level. All socioeconomic measures were operationalized so that higher values represented a greater socioeconomic resource. All psychosocial measures were operationalized so that higher values represent greater adversity. The scale of the psychosocial measures is per 1 unit. Models were adjusted for age, sex, season, site, season-by-site interaction, antihypertensive medication use, apparent temperature, and all other socioeconomic and psychosocial variables in the figure. Results are the difference in PP per 10-µg/m^3 increase in $PM_{2.5}$ exposure averaged over the month prior to the examination. Income and census tract median household income values are per $10,000. CESD, Center for Epidemiologic Studies Depression Scale; CSS, Chronic Stress Scale; ESSI, Emotional Social Support Index (reverse-coded); STAI, Spielberger Trait Anxiety Inventory; STAXI, Spielberger Trait Anger Expression Inventory. Bars, 95% confidence intervals.

pressure was lacking from an additional 3 persons. The following key information was missing for an additional 539 persons: annual income ($n = 210$), smoking/ETS ($n = 149$), census tract median household income ($n = 72$), educational level ($n = 19$), diabetes status ($n = 17$), and alcohol use ($n = 16$). This yielded a final analytic sample size of 5,570 persons. The demographic characteristics of this subset were similar to those of the entire MESA cohort (results not shown).

Statistical analysis

First, descriptive analyses of the sample were conducted, and the distributions of potential modifying factors and covariates were summarized across quartiles of $PM_{2.5}$ exposure.

Then, to examine the independent associations between blood pressure and each of the focal independent variables ($PM_{2.5}$ exposure and social and psychosocial factors), linear regression
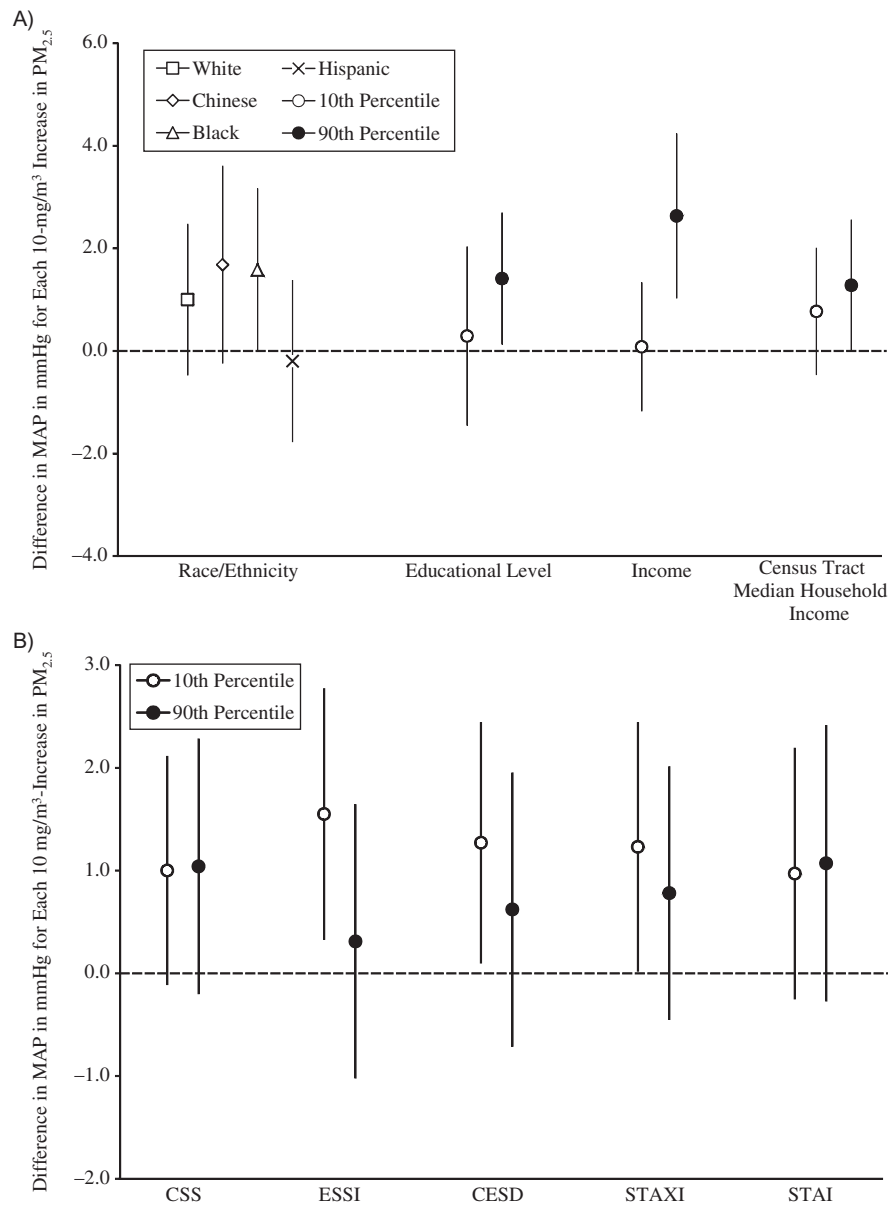
Figure 4. Associations between concentrations of particulate matter less than 2.5μm in aerodynamic diameter (PM$_{2.5}$) and mean arterial pressure (MAP), by social disadvantage and psychosocial level or category, Multi-Ethnic Study of Atherosclerosis, 2000–2002. A) Associations by social disadvantage level or category. B) Associations by psychosocial level. All socioeconomic measures were operationalized so that higher values represented a greater socioeconomic resource. All psychosocial measures were operationalized so that higher values represent greater adversity. The scale of the psychosocial measures is per 1 unit. Models were adjusted for age, sex, season, site, season-by-site interaction, antihypertensive medication use, apparent temperature, and all other socioeconomic and psychosocial variables in the figure. Results are the difference in MAP per 10-μg/m^3 increase in PM$_{2.5}$ exposure averaged over the month prior to the examination. Income and census tract median household income values are per $10,000. CESD, Center for Epidemiologic Studies Depression Scale; CSS, Chronic Stress Scale; ESSI, Emotional Social Support Index; STAI, Spielberger Trait Anxiety Inventory; STAXI, Spielberger Trait Anger Expression Inventory. Bars, 95% confidence intervals.

models were estimated, with a separate model for each blood pressure outcome. Model 1 included the social disadvantage measures: race/ethnicity, educational level, income, and census tract median household income. Model 2 further included the 5 psychosocial measures. Model 3 included further adjustment for additional risk factors for hypertension: body mass index,

smoking/ETS, diabetes, and current alcohol use. Model 3 was investigated in order to determine whether any observed associations were confounded and/or mediated by hypertension risk factors that have been linked to social disadvantage or psychosocial factors. All models included adjustment for age, sex, apparent temperature, season, antihypertensive medication use,

Table 1. Sociodemographic Characteristics, Psychosocial Factors, and Blood Pressure Outcomes by Levels of Exposure to Particulate Matter Less Than 2.5 μm in Aerodynamic Diameter Averaged Over the Month Before the Examination, The Multi-Ethnic Study of Atherosclerosis, 2000–2002

Variable	Quartile of PM$_{2.5}$ Exposure[a]								P for trend[b]
	1		2		3		4		
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	
Sociodemographic characteristics									
Age, years	62.5 (10.6)		61.9 (9.9)		61.5 (10.1)		61.1 (9.8)		<0.001
Female sex		52		55		54		50	<0.05
Race/ethnicity									
White		50		39		39		29	<0.001
Chinese		3		7		16		25	
Black		22		30		26		25	
Hispanic		25		23		18		21	
Educational level, years	13.2 (3.9)		13.0 (3.9)		13.5 (3.9)		13.1 (4.4)		
Income/$10,000	4.8 (3.3)		4.7 (3.2)		5.3 (3.5)		5.1 (3.7)		<0.001
Census tract median household income/ $10,000	4.4 (1.2)		4.2 (2.1)		4.6 (2.2)		4.5 (2.4)		<0.001
Site									
Forsyth County, North Carolina		21		15		15		11	<0.001
New York, New York		14		29		13		5	
Baltimore, Maryland		10		24		14		10	
St. Paul, Minnesota		44		13		6		0	
Chicago, Illinois		11		12		26		26	
Los Angeles County, California		1		7		27		49	
Psychosocial characteristics									
Chronic stressors	0.9 (1.1)		0.9 (1.1)		0.9 (1.1)		0.8 (1.1)		<0.05
Lack of emotional support	12.1 (5.3)		11.8 (5.3)		11.5 (5.1)		11.5 (5.2)		<0.05
Depressive symptoms	7.8 (7.3)		7.8 (7.6)		7.0 (6.9)		6.9 (7.8)		<0.001
Trait anger	14.7 (3.6)		14.8 (3.7)		14.7 (3.4)		14.8 (3.6)		
Trait anxiety	16.0 (4.4)		15.9 (4.5)		15.7 (4.3)		15.7 (4.6)		<0.05

Table continues

MESA site, and the interaction between season and MESA site. This interaction was included because of the seasonal characteristics that may confound the association between PM$_{2.5}$ and blood pressure differ by site.

Because social disadvantage and the psychosocial measures were included in the same models, correlations among these variables were computed and variance inflation factors examined for each of these factors as in model 2, which was the base model for all interactions presented in the figures (described below). High multicollinearity was defined as an average variance inflation factor greater than or equal to 10 (39). The average variance inflation factor was 4.49, with the individual variance inflation factors of the PM$_{2.5}$, social, and psychosocial measures ranging from 1.28 (trait anger) to 2.87 (PM$_{2.5}$).

Finally, to examine the presence of additive interactions between PM$_{2.5}$ and each of the social disadvantage and psychosocial factors, the interaction between PM$_{2.5}$ and each of these factors was added to model 2. In order to maximize power and utilize all available data, interactions were tested using continuous predictors. Graphical displays show predicted associations of PM$_{2.5}$ with blood pressure for each category of race/ethnicity and at the 10th (i.e., low) and 90th (i.e., high) percentiles of continuous SES and psychosocial measure distribution (Figures 1–4).

Several sets of sensitivity analyses were performed. First, the impact of different approaches to account for use of antihypertensive medications was examined. These included 1) excluding those who reported antihypertensive medication use (*n* = 2536) and 2) adding 10 mmHg and 3) 15 mmHg to the SBPs of those who reported medication use. This latter approach has previously been shown to be as valid as more complex imputation approaches (40), and it may produce less bias than adjusting for medication use, which is a consequence of the outcome (41).

Second, other acute PM$_{2.5}$ exposures, including 1-, 3-, and 7-day and 2-month averages, were examined. Finally, because PM$_{2.5}$ exposure may vary within a site, all analyses were performed using predicted individual-level PM$_{2.5}$ exposures at each participant's residence that accounted for both time and

Table 1.   Continued

Variable	Quartile of PM$_{2.5}$ Exposure[a]								P for trend[b]
	1		2		3		4		
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	
Hypertension risk factors and other confounders									
Smoking/ETS status									
Never smoker/ETS <1 hour/week		26		31		34		37	<0.001
Never smoker/ETS ≥1 hour/week		20		18		20		16	
Past smoker/ETS <1 hour/week		18		20		19		22	
Past smoker/ ETS ≥1 hour/week		20		17		16		13	
Current smoker		14		13		12		12	
Current alcohol use		61		57		56		52	<0.001
Body mass index[c]	28.8 (5.3)		28.7 (5.6)		27.8 (5.3)		27.5 (5.3)		<0.001
Diabetes		25		27		23		26	
Antihypertensive medication use		34		41		33		35	<0.001
1-month average apparent temperature, °C	10.1 (8.3)		8.0 (8.5)		12.1 (9.8)		12.9 (10.8)		<0.001
Blood pressure measures, mm Hg									
Systolic blood pressure	126.0 (21.2)		126.6 (20.7)		124.5 (20.6)		126.3 (21.4)		
Diastolic blood pressure	71.5 (10.2)		72.2 (10.1)		71.4 (10.1)		72.1 (10.3)		
Pulse pressure	54.5 (17.1)		54.3 (16.6)		53.1 (16.2)		54.2 (17.3)		
Mean arterial pressure	89.6 (12.4)		90.3 (12.2)		89.1 (12.3)		90.1 (12.5)		

Abbreviations: ETS, exposure to second hand smoke; PM$_{2.5}$, particulate matter less than 2.5 μm in aerodynamic diameter; SD, standard deviation.

[a] Averaged over the month before the examination. Quartile 1, <13.2 μg/m^3; quartile 2, 13.2–15.69 μg/m^3; quartile 3, 15.7–19.19 μg/m^3; and quartile 4, ≥19.2 μg/m^3.

[b] P values (2-sided) by χ^2 for trend for categorical variables and by regression for continuous variables.

[c] Weight (kg)/height (m)^2.

space (42, 43). These PM$_{2.5}$ exposures were predicted from a combination of measurements from the Environmental Protection Agency Air Quality System and MESA Air monitoring stations and spatial covariates, such as proximity to roadways and local land use. These measures were available only for the 1-month and 2-month averaged time periods.

Institutional review board approval was granted at each study site, and written informed consent was obtained from all participants. All statistical tests for significance were 2-sided. Analyses were conducted in STATA MP, version 11.0 (StataCorp LP, College Station, Texas).

RESULTS

The distributions of covariates and outcomes across quartiles of PM$_{2.5}$ exposure for the month prior to the visit are shown in Table 1. In general, these characteristics were similar across levels of air pollution, although air pollution and some covariates varied substantially among sites (data not shown), as has been documented in other work based on these data (32, 44). There were modest positive associations between SES and air pollution and modest inverse associations between psychosocial adversity and air pollution. There were no associations between any of the blood pressure outcomes and air pollution.

After adjusting for age, sex, season, site, season-by-site interaction, antihypertensive medication use, apparent temperature, and the SES measures, higher PM$_{2.5}$ exposure during the month before the examination was associated with higher SBP (mean difference per 10-μg/m^3 increase in PM$_{2.5}$ = 2.49 mmHg; 95% confidence interval: 0.82, 4.15; SBP model 1 in Table 2). This association did not change after adjustment for psychosocial factors (SBP model 2) and was slightly attenuated after further adjustment for health-related factors (SBP model 3). PM$_{2.5}$ was not associated with DBP (Table 2, DBP models 1–3). PM$_{2.5}$ was positively associated with PP (mean difference per 10-μg/m^3 increase in PM$_{2.5}$ = 2.17 mmHg; 95% confidence interval: 0.94, 3.40; Table 3, PP Model 1). The association did not change after adjustment for psychosocial measures (PP model 2) and was slightly attenuated after adjustment for the health-related factors (PP model 3). There was also a positive association between PM$_{2.5}$ and MAP (mean difference per 10-μg/m^3 increase in PM$_{2.5}$ = 1.04 mmHg; 95% confidence interval: 0.01, 2.07; MAP model 1 in Table 3). The association did not change with the addition of psychosocial (MAP model 2) or health-related (MAP model 3) factors. (Differences between these results and previously published results (21) are due to adjustment for the season-by-site interaction).

Socially disadvantaged groups generally had higher SBP, PP, and MAP, but not necessarily DBP, compared with more

Table 2. Mean Difference in Systolic and Diastolic Blood Pressure Associated With Exposure to Particulate Matter Less Than 2.5 μm in Diameter in the Previous Month and Covariates in Sequentially Adjusted Models, The Multi-Ethnic Study of Atherosclerosis, 2000–2002[a]

Variable	Systolic Blood Pressure, mm Hg						Diastolic Blood Pressure, mm Hg					
	Model 1[b]		Model 2		Model 3		Model 1		Model 2		Model 3	
	b	95% CI	b	95% CI	b	95% CI	b	95% CI	b	95% CI	b	95% CI
1-month average PM$_{2.5}$ /10 μg/m^3	2.49**	0.82,4.15	2.45**	0.78,4.11	2.39**	0.74,4.03	0.32	−0.52, 1.16	0.30	−0.54, 1.14	0.31	−0.53, 1.15
Social disadvantage												
Race/ethnicity												
White		Referent		Referent		Referent		Referent		Referent		Referent
Chinese	1.60	−0.36, 3.56	1.11	−1.18, 3.39	2.87**	0.56, 5.19	2.08***	1.09, 3.06	2.04***	1.04, 3.04	2.47***	1.43, 3.52
Black	6.47***	5.06, 7.88	9.51***	7.88, 11.14	6.81***	5.20, 8.43	3.97***	3.26, 4.67	3.91***	3.20, 4.63	3.80***	3.07, 4.53
Hispanic	4.40***	2.72, 6.09	4.31***	2.36, 6.27	2.33***	0.42, 4.24	1.26**	0.42, 2.11	1.21**	0.36, 2.06	1.10*	0.24, 1.96
Educational level (per year)[c]	−0.38***	−0.54, −0.22	−0.39***	−0.55, −0.23	−0.36***	−0.52, −0.20	−0.14***	−0.22, 0.06	−0.14***	−0.22, 0.06	−0.15***	−0.23, 0.07
Income (per $10,000)[c]	−0.23*	−0.42, −0.03	−0.27**	−0.47, −0.08	−0.27**	−0.47, −0.08	0.01	−0.09, 0.10	−0.02	−0.11, −0.08	−0.03	−0.13, 0.07
Census tract median household income/ $10,000[c]	0.00	−0.28, 0.29	0.00	−0.28, 0.29	0.02	−0.26, 0.30	0.04	−0.11, 0.18	0.04	−0.11, 0.18	0.03	−0.11, 0.17
Psychosocial adversity[d]												
Chronic stressors			0.39	−0.13, 0.90	0.15	−0.37, 0.66			0.04	−0.23, 0.30	0.00	−0.27, 0.26
Lack of emotional support			−0.09	−0.20, 0.01	−0.08	−0.19, 0.03			−0.03	−0.08, 0.03	−0.02	−0.08, 0.03
Depressive symptoms			−0.03	−0.13, 0.06	−0.05	−0.14, 0.04			0.00	−0.05, 0.04	0.00	−0.05, 0.04
Trait anger			0.10	−0.05, 0.26	0.06	−0.09, 0.21			0.05	−0.03, 0.13	0.04	−0.04, 0.12
Trait anxiety			−0.08	−0.23, 0.07	−0.03	−0.18, 0.12			−0.05	−0.13, 0.03	−0.04	−0.12, 0.04

Abbreviations: CI, confidence interval; PM$_{2.5}$, particulate matter less than 2.5μm in aerodynamic diameter.

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ by χ^2 for trend for categorical variables and by regression for continuous variables.

[a] Results from main effect models ($n = 5570$).

[b] Model 1 was adjusted for age, sex, season, site, season-by-site interaction, antihypertensive medication use, apparent temperature, race/ethnicity, educational level, income, and census tract median household income. Model 2 was adjusted for the factors in model 1 as well as ongoing burdens, depressive symptoms, trait anger, trait anxiety, and lack of emotional support. Model 3 was adjusted for the factors in model 2 as well as current alcohol use, smoking/exposure to tobacco smoke, body mass index, and diabetes.

[c] All socioeconomic measures were operationalized so that higher values represented a greater socioeconomic resource. All psychosocial measures were operationalized so that higher values represent greater adversity.

[d] The scales of the psychosocial measures are per 1 unit.

Table 3. Mean Difference in Pulse Pressure and Mean Arterial Pressure Associated With Exposure to Particulate Matter Less Than 2.5 μm in Diameter in the Previous Month and Covariates in Sequentially Adjusted Models, The Multi-Ethnic Study of Atherosclerosis, 2000–2002[a]

Variable	Pulse Pressure, mm Hg						Mean Arterial Pressure, mm Hg					
	Model 1[b]		Model 2		Model 3		Model 1		Model 2		Model 3	
	b	95% CI	b	95% CI	b	95% CI	b	95% CI	b	95% CI	b	95% CI
1-month average PM$_{2.5}$ /10 μg/m^3	2.17***	0.94, 3.40	2.15***	0.92, 3.38	2.08***	0.87, 3.29	1.04*	0.01, 2.07	1.01	0.00, 2.04	1.00	0.00, 2.03
Social disadvantage												
Race/ethnicity												
White		Referent		Referent		Referent		Referent		Referent		Referent
Chinese	−0.48	−1.93, 0.96	−0.40	−1.87, 1.06	0.63	−0.88, 2.14	1.92**	0.71, 3.13	1.91**	0.68, 3.13	2.68***	1.41, 3.96
Black	2.50***	1.46, 3.54	2.45***	1.40, 3.50	1.32*	0.26, 2.38	4.80***	3.93, 5.67	4.73***	3.85, 5.61	4.24***	3.35, 5.13
Hispanic	3.14***	1.90, 4.38	3.14***	1.89, 4.39	2.27***	1.02, 3.51	2.31***	1.27, 3.35	2.26***	1.21, 3.30	1.86***	0.80, 2.91
Educational level (per year)[c]	−0.24***	−0.36, −0.13	−0.25***	−0.37, −0.13	−0.22***	−0.33, −0.10	−0.22***	−0.32, −0.12	−0.22***	−0.32, −0.13	−0.22***	−0.31, −0.13
Income (per $10,000)[c]	−0.23***	−0.37, −0.09	−0.26***	−0.40, −0.11	−0.24***	−0.39, −0.10	−0.07	-0.19, −0.05	−0.10	−0.22, 0.02	−0.11	−0.23, 0.01
Census tract median household income/ $10,000[c]	−0.03	−0.24, 0.17	−0.03	−0.24, 0.17	−0.02	−0.22, 0.19	0.03	−0.15, 0.20	0.03	−0.15, 0.20	0.03	−0.15, 0.20
Psychosocial adversity[d]												
Chronic stressors			0.35	−0.03, 0.73	0.15	−0.23, 0.53			0.15	−0.17, 0.47	0.05	−0.27, 0.37
Lack of emotional support			−0.07	−0.15, 0.01	−0.06	−0.14, 0.02			−0.05	−0.12, 0.02	−0.04	−0.11, 0.02
Depressive symptoms			−0.03	−0.10, 0.04	−0.05	−0.11, 0.02			−0.01	−0.07, 0.05	−0.02	−0.08, 0.04
Trait anger			0.05	−0.06, 0.17	0.02	−0.09, 0.14			0.07	−0.03, 0.16	0.05	−0.05, 0.14
Trait anxiety			−0.03	−0.14, 0.08	0.01	−0.10, 0.12			−0.06	−0.16, 0.03	−0.04	−0.13, 0.06

Abbreviations: CI, confidence interval; PM$_{2.5}$, particulate matter less than 2.5μm in aerodynamic diameter.

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ by χ^2 for trend for categorical variables and by regression for continuous variables.

[a] Results from main effect models ($n = 5570$).

[b] Model 1 was adjusted for age, sex, season, site, season-by-site interaction, antihypertensive medication use, apparent temperature, race/ethnicity, educational level, income, and census tract median household income. Model 2 was adjusted for the factors in model 1 as well as ongoing burdens, depressive symptoms, trait anger, trait anxiety, and lack of emotional support. Model 3 was adjusted for the factors in model 2 as well as current alcohol use, smoking/exposure to tobacco smoke, body mass index, and diabetes.

[c] All socioeconomic measures were operationalized so that higher values represented a greater socioeconomic resource. All psychosocial measures were operationalized so that higher values represent greater adversity.

[d] The scales of the psychosocial measures are per 1 unit.

socially advantaged groups. For example, higher SBP was found for blacks compared with whites, and higher SES was associated with lower SBP. Psychosocial adversity was not associated with any of the blood pressure outcomes.

Figures 1–4 show the associations of $PM_{2.5}$ with SBP, DBP, PP, and MAP, respectively, for different levels of social disadvantage (Figures 1A, 2A, 3A, and 4A) and psychosocial factors (Figures 1B, 2B, 3B, and 4B) as predicted from regression models that included an additive interaction between each social/psychosocial measure and 1-month prior $PM_{2.5}$ exposure, setting other covariates to their mean. In general, the patterns of results were similar across blood pressure outcomes. There was no evidence of a modifying role for race/ethnicity, educational level, or Census tract median household income in the association between $PM_{2.5}$ and blood pressure. However, there was evidence of a modifying role for income in the associations between $PM_{2.5}$ and SBP, DBP, and MAP. In general, greater levels of income were associated with stronger relationships between $PM_{2.5}$ and blood pressure, and although the differences were small, the interactions reached statistical significance at the $P < 0.05$ level (for SBP and income, interaction term = 0.37, 95% confidence interval: 0.06, 0.68; for DBP and income, interaction term = 0.19, 95% confidence interval: 0.03, 0.34; for PP and income, interaction term = 0.18, 95% confidence interval: −0.04, 0.41; and for MAP and income, interaction term = 0.25, 95% confidence interval: 0.06, 0.44). There was no evidence of a modifying role for any of the psychosocial measures.

Results from sensitivity analyses using individual-level $PM_{2.5}$ exposures that accounted for space yielded similar results. $PM_{2.5}$ was not associated with blood pressure outcomes when using the 1-, 3-, or 7-day averaged exposures, as was found in previous work (21). Results using 2-month average exposures were similar to the associations found when using the 1-month averaged exposure period. Results when using alternative adjustments for antihypertensive medication use yielded results similar to those presented.

DISCUSSION

We examined the associations between exposure to $PM_{2.5}$ and blood pressure by levels of social disadvantage and psychosocial factors in a large, population-based cohort of adults without a history of CVD. These associations did not differ significantly by most measures of social disadvantage and or psychosocial adversity. Contrary to the study hypotheses, higher levels of income were associated with stronger associations between $PM_{2.5}$ and blood pressure.

The pathways through which $PM_{2.5}$ exposure could affect blood pressure have been hypothesized to include oxidative stress and inflammatory processes, as well as alterations in cardiovascular reactivity and autonomic function (5, 45). Both of these pathways may also be affected by social and psychosocial factors (25, 46, 47), which suggests that both types of exposures may act synergistically. Moreover, some of the behavioral correlates of social disadvantage (e.g., sedentary lifestyles, unhealthy diets) could also enhance the adverse cardiovascular effects of environmental exposures. However, few studies have had information on $PM_{2.5}$ exposures together with the social and psychosocial factors necessary to investigate their interactive effects.

Although few studies have evaluated the modifying role of social disadvantage on the association between $PM_{2.5}$ and blood pressure, researchers have examined other cardiovascular outcomes, with mixed results. For example, contrary to our results, Ostro et al. (9) reported that, compared with persons of higher SES, those of lower SES showed a stronger association between exposure to $PM_{2.5}$ and cardiovascular mortality. However, others reported that the association between $PM_{2.5}$ and self-reported hypertension was stronger in whites than in nonwhites (30) and that the association between $PM_{2.5}$ and aortic calcification was stronger in persons with higher incomes compared with those with lower incomes (31). Finally, others reported no racial/ethnic differences in the association between $PM_{2.5}$ and microvasculature narrowing (32).

Only a few studies have examined heterogeneity in the air pollution–health association by level of psychosocial factors, and those studies only examined respiratory outcomes (4, 15). In all of the studies, persons who reported greater levels of stressors or strain showed a stronger association between air pollution and asthma risk (15, 48) or clinical asthma symptoms (16) than did those who reported lower levels of stressors or strain. To our knowledge, there have been no studies on the modifying role of psychosocial adversity on the association between air pollution and cardiovascular outcomes.

A number of methodological factors may have contributed to the absence of synergies between $PM_{2.5}$ exposures and social disadvantage or psychosocial factors. First, the MESA sample excluded persons with clinical CVD symptoms at enrollment. Because of this, participants may be healthier than the general United States population and perhaps less vulnerable to the influence of social and psychosocial factors (as main effects or as modifiers of the effects of other exposures such as air pollution).

In addition, single measures of social disadvantage or psychosocial conditions may not adequately reflect the causal factors that increase vulnerability to the hypertensive effects of $PM_{2.5}$. Researchers have argued that multiple social and psychosocial factors operate cumulatively to increase vulnerability to the health effects of environmental hazards (1, 6, 49, 50). In the future, researchers should examine measures of social disadvantage or psychosocial stress that capture the multiple and co-occurring factors that could increase vulnerability to the health effects of environmental hazards, such as residential segregation, composite individual-level social factors (e.g., social participation, unfair treatment, exposure to violence), or composite neighborhood-level social factors (e.g., crime, unemployment, green spaces).

Undoubtedly, the psychosocial factors were measured with error. Furthermore, the measures themselves may have been limited in their validity and reliability. They may not have captured the long-term exposure to psychosocial stress that is likely more relevant as an effect modifier of the hypertensive effects of air pollution compared with a more contemporaneous measure. These factors could have severely limited the ability to detect interactions.

An unexpected finding was that the associations of $PM_{2.5}$ exposures with blood pressure were actually stronger among persons with higher incomes. CVD prevalence is higher in

persons of lower SES compared with those of higher SES (51, 52). Persons who were of low SES and still free from CVD (a requirement for inclusion in MESA) may have other factors that buffer them against the $PM_{2.5}$–blood pressure association. Adjustment for standard hypertension risk factors did not alter the interaction patterns, and it is difficult to pinpoint any specific omitted factors correlated with SES exposures that explain the interaction. Environmental exposures may have a weaker effect in the presence of blood pressure that is already elevated due to other important risk factors, as may be the case in persons with low SES. This may make it difficult to detect the added association of another risk factor, such as $PM_{2.5}$ exposure, with its relatively weak associations with blood pressure. Although some of these unexpected interactions were statistically significant, chance remains a possible explanation for the results.

Other health outcomes that are more strongly linked to inflammation and oxidative stress may be more appropriate in the study of the synergistic effects between $PM_{2.5}$ and social disadvantage/psychosocial factors. Environmental researchers have theorized that inflammation and oxidative stress are major pathways that link $PM_{2.5}$ and cardiovascular health (18). Researchers have also linked stress and cardiovascular health through these pathways (25).

The lack of associations between shorter-term $PM_{2.5}$ exposures and blood pressure were consistent with results from prior work (21). The authors of previous work had posited that accumulated exposures may have stronger impacts on health or that smoothing across the wide shorter-term fluctuations in exposures may reduce error, allowing for the detection of any $PM_{2.5}$–blood pressure associations (21). Overall, the results of the present study do not support the hypothesis that social disadvantage or psychosocial adversity confers increased vulnerability to the hypertensive effects of $PM_{2.5}$. Unexpectedly, higher SES appeared to be associated with a slightly stronger relationship between $PM_{2.5}$ and blood pressure than did lower SES. This is the first examination of the possible modifying role of race/ethnicity, socioeconomic factors, and psychosocial factors on the association between $PM_{2.5}$ and blood pressure. Further investigation using improved social disadvantage and psychosocial measures in population-representative samples is necessary to replicate these findings.

ACKNOWLEDGMENTS

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