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Particulate matter air pollution and racial differences in cardiovascular disease risk

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Abstract

Objective—We aimed to assess racial differences in air pollution exposures to ambient fine particulate ($PM_{2.5}$) and black carbon (BC) and their association with cardiovascular disease (CVD) risk factors, arterial endothelial function, incident CVD events and all-cause mortality.

Approach and Results—Data from the Heart Strategies Concentrating on Risk Evaluation (HeartSCORE) study were used to estimate one-year average air pollution exposure to $PM_{2.5}$ and BC using land-use regression models. Correlates of $PM_{2.5}$ and BC were assessed using linear regression models. Associations with outcomes were determined using Cox proportional hazards models, adjusting for traditional CVD risk factors. Data were available on 1,717 participants (66% female, 45% Blacks, 59±8 years). Blacks had significantly higher exposure to $PM_{2.5}$ (mean 16.1±0.75 vs. 15.7±0.73μg/m^3, p=0.001) and BC (1.19±0.11 vs. 1.16±0.13abs, p=0.001) compared to Whites. Exposure to $PM_{2.5}$, but not BC, was independently associated with higher blood glucose and worse arterial endothelial function. $PM_{2.5}$ was associated with a higher risk of incident CVD events and all-cause mortality combined over median follow-up of 8.3 years. Blacks had 1.45 (95% CI:1.00,2.09) higher risk of combined CVD events and all-cause mortality than Whites in models adjusted for relevant covariates. This association was modestly attenuated with adjustment for $PM_{2.5}$.

Conclusion—$PM_{2.5}$ exposure was associated with elevated blood glucose, worse endothelial function, and incident CVD events and all-cause mortality. Blacks had a higher rate of incident CVD events and all-cause mortality than Whites that was only partly explained by higher exposure to $PM_{2.5}$.

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Introduction

Racial differences in mortality and cardiovascular disease (CVD) morbidity pose challenges for health care in the United States and worldwide.[1–4] Understanding the role of environmental pollution in race-related differences in CVD risk factors and clinical CVD outcome may elucidate a pathophysiologic mechanism for such differences and guide preventive strategies. Epidemiological studies have shown that chronic exposure to airborne fine particulate matter (particles with median aerodynamic diameter <2.5 μm [$PM_{2.5}$]) is associated with increased CVD risk and mortality.[5–8] Although the pathophysiological underpinnings of these associations are not fully understood, potential mechanisms identified in animal and human studies include increased oxidative stress and inflammation leading to endothelial dysfunction, atherosclerotic plaque progression and thrombosis.[5, 9–11] Studies of black carbon (BC), a component of ultrafine particulate matter used as a tracer for diesel related emissions, have yielded less consistent results[12, 13]

Epidemiological data also indicate that racial/ethnic minorities are more likely to reside in areas close to environmental pollution sources, including point sources and heavy roadway traffic areas.[14–17] However, racial differences in the exposure to environmental air pollution and their role in disparities in CVD risk and mortality have not been fully elucidated. Therefore, we assessed racial differences in urban air pollution exposures to $PM_{2.5}$ and BC and their association with CVD risk factors and incident CVD events and all-cause mortality in the Heart Strategies Concentrating on Risk Evaluation (HeartSCORE) study, a prospective community-based cohort in Western Pennsylvania that is prospectively examining racial differences in CVD risk and outcomes since 2003.

Participants and Methods

Study population

HeartSCORE is an ongoing community-based prospective cohort study of 2000 participants with approximately equal representation of Blacks (44%) and Whites (56%) assessing racial and socioeconomic disparities in cardiovascular risk. The methods of HeartSCORE have been described previously.[18, 19] Eligibility criteria included age 45 to 75 years at study entry, residence in the greater Pittsburgh, PA, metropolitan area, ability to undergo baseline and annual follow-up visits, and absence of known co-morbidities expected to limit life expectancy to less than 5 years. The Institutional Review Board at the University of Pittsburgh approved the study protocol and all study participants provided written informed consent. The present study included 1717 participants who had available data on air pollution exposures to ambient fine particulate ($PM_{2.5}$) and black carbon (BC). Data are available from authors upon request, for the purposes of replicating the study.

Exposure determination

We estimated chronic exposures to urban $PM_{2.5}$ and BC for the year prior to each individual's baseline clinical date, using adapted versions of previously published land use regression (LUR) methods, incorporating AERMOD dispersion models to better account for the influence of local point sources, as reported in the National Emissions Inventory (NEI)

(https://www.epa.gov/air-emissions-inventories/2014-national-emissions-inventory-nei-data)[20, 21] Hybrid LUR models (including AERMOD dispersion model terms) were derived from a spatial monitoring campaign including 37 sampling sites distributed across metropolitan Pittsburgh during summer (June 5 to July 26, 2012) and winter (January 8 to March 10, 2013). Geographic information system (GIS)-based covariates were calculated to capture variability in a range of pollution source indicators (e.g., traffic density, industrial emissions, population).[20] Hybrid LUR models were developed as mixed models adjusted for repeated measures at each site by season, predicting spatial variation in $PM_{2.5}$ and BC as a function of the GIS-based source density indicators. We geocoded participant addresses using a composite address locator in ArcGIS to generate point features of residential locations. We used the LUR models to estimate the mean concentrations of $PM_{2.5}$ and BC for the 300 m surrounding each participant's residential address, for the 12 months prior to the month of each participant's baseline clinical date, using daily regulatory data from a centrally-located U.S. EPA Air Quality System (AQS) monitor.[20, 21]

Covariates and dependent variables

Demographic and medical histories were collected at the baseline visit (2001–2004). Race was self-reported. Participants completed demographic questionnaire including information on marital/co-habiting status, education, and income. Highest education level was categorized as less than high school, beyond high school, and beyond Bachelor's degree. Annual income was collected in categories < $10K, $20–40K, $40–80K and > $80K. Physical measurements included measurement of vital signs and body fat distribution. Hypertension was defined as blood pressure >140/90 or use of anti-hypertensive medications. Body mass index was calculated as weight/height^2 (kg/m^2). Laboratory assessments of cholesterol levels were performed on venous blood drawn in the fasting state using the commercially available vertical auto profile technique (VAP, Atherotech, Birmingham, AL). Fasting blood glucose was measured using the glucose oxidase method. Measurement of high-sensitivity C-reactive protein (hsCRP) was performed using an immunoturbidimetric assay on the Roche P Modular system (Roche Diagnostics, Indianapolis, IN), using reagents and calibrators from DiaSorin (Stillwater, MN). Serum interleukin-6 (IL-6) concentrations were measured using commercially available ELISA assay kits (R&D Systems, Minneapolis, MN).

Endothelial function was measured using an Endo-PAT2000 device (Itamar Medical, Caesarea, Israel) adapted from the protocol used by the Framingham Heart Study as previously reported.[18, 19, 22] In brief, digital pulse amplitude was measured using the PAT device placed on the tip of each index finger. Baseline PAT signal was measured for 5 minutes on both fingers. Arterial flow was then interrupted on one finger by applying occlusive arm pressure. After 5 minutes the cuff-pressure was abruptly deflated and PAT signal was measured on both fingers for the subsequent 5 minutes. The data were recorded electronically and analyzed using a computerized, automated algorithm. Pulse amplitude response to hyperemia was calculated from the hyperemic fingertip as the ratio of the post-occlusion pulse amplitude to the baseline-pulse amplitude. The result was divided by the corresponding ratio in the control hand to give the PAT ratio (also known as reactive

hyperemia index [RHI]). The Framingham reactive hyperemia index (fRHI) was calculated as the natural log-transformation of the RHI.[19, 22]

Clinical outcomes (all-cause mortality and incident CVD events)

Participants were assessed for incident hospitalization and CVD events by semi-annual questionnaires and during annual follow-up study visits. Incident CVD events were pre-defined as non-fatal myocardial infarction, acute coronary syndrome, stroke, coronary revascularization, or cardiac death. The primary outcome of interest was a combination of CVD events and all-cause mortality. Medical records were obtained for each reported hospitalization. A research nurse and study physician adjudicated incident events independently. Cause of death as cardiac or non-cardiac was ascertained by review of the death certificate obtained from the Commonwealth of Pennsylvania.

Statistical methods

Baseline variables are presented by tertiles of $PM_{2.5}$ and BC. We also presented baseline variables by race in complementary analyses. Continuous variables are expressed as means (SD) and categorical variables are expressed as proportions. Associations of $PM_{2.5}$ and BC with CVD risk factors were assessed using linear regression models, adjusted for age, sex, smoking status, and race. Further adjustment was made for income and education status to assess the impact of socioeconomic status. Potential effect modification of the associations of $PM_{2.5}$ or BC with CVD risk factors by race or sex was investigated by fitting interactions terms between race or sex and the pollutants.

The associations of $PM_{2.5}$ and BC with incident CVD outcome and all-cause mortality were examined using multivariable-adjusted Cox proportional hazards models. The assumptions of the proportionality of hazards were evaluated using Schoenfeld residuals. Follow-up time was determined by calculating the duration (in years) from the date of initial visit to the date of event, date of last follow-up or the date of censoring, which was on August 7,2014. Adjustment was made for income and education status, as in the linear regression models.

We performed a mediation analyses to assess the potential role of air pollution in explaining the association between Black race and clinical outcomes by adding $PM_{2.5}$ or BC to Cox proportional hazards models relating race and CVD outcomes, in a model adjusted for CVD risk factors, namely, age, sex, smoking, systolic blood pressure, diabetes, body mas index, total cholesterol, and HDL-cholesterol. The analyses were conducted using the methods described by Ananth and VanderWeele, based on the estimated direct and indirect effects estimated for Black race, as computed on the risk difference scale.[23] Given the high correlation between race an socioeconomic status, we did not include markers of socioeconomic status in the model used for mediation analyses. All analyses were performed with Stata software (Stata Corp., version 11, Texas, USA). A p-value <0.05 was considered statistically significant. Study data are available from the authors upon request for the purposes of replicating the study.

Results

Baseline characteristics and bivariate correlations for $PM_{2.5}$ and BC

The analyses involved 1717 participants (66% female, 45% Blacks, 59±8 years) with available information on $PM_{2.5}$ and BC. Baseline characteristics of participants are presented by tertiles of $PM_{2.5}$ and BC in Table 1 and Supplemental Table I, respectively. The median estimated $PM_{2.5}$ exposure was 15.7 μg/m^3 (range: 14.3–19.1; inter-quartile range: 15.3–16.4). The median estimate of BC concentration was 1.16 abs (range: 0.93–1.92; inter-quartile range: 1.09–1.24). The mean (SD) estimates of $PM_{2.5}$ and BC concentrations were 15.7±0.77 μg/m^3 and 1.17±0.12 abs, respectively. Blacks had, on average, higher exposures to $PM_{2.5}$ and BC than did Whites. Mean $PM_{2.5}$ among Blacks was 16.1 (SD = 0.75) μg/m^3 vs. 15.7 (0.73) μg/m^3 among White. Mean BC exposure among Blacks was 1.19 (SD = 0.11) abs vs. 1.16 (0.13) abs among Whites (Figure 1) The baseline characteristics of the participants by race is shown in Supplemental Table II.

In univariate models, $PM_{2.5}$ was correlated with a broad spectrum of factors. For example, $PM_{2.5}$ exposures decreased with increasing income. There was also linear increase in mean blood glucose, BMI, and IL-6 concentrations and decrease in arterial endothelial function measured by fRHI across tertiles of $PM_{2.5}$. (Table 1) There were similar but weaker patterns of associations for BC. (Supplemental Table I)

Multivariable correlates of $PM_{2.5}$ and BC

The Black-White participant difference in exposure to $PM_{2.5}$ and BC remained statistically significant after adjustment for age, sex, smoking status, income, and education. Furthermore, higher $PM_{2.5}$ exposures were associated with higher systolic blood pressure, body mass index, blood glucose and IL-6, lower fRHI (i.e., worse endothelial function) in age- and sex-adjusted models. (Table 2) The associations of $PM_{2.5}$ with glucose and fRHI remained statistically significant after further adjusting for smoking, race, income and education. Each 1.5-μg/m^3 higher concentration of $PM_{2.5}$ was associated with a 3.7-mg/dl (95% CI: 1.0 – 6.4) increase in blood glucose levels and a 0.06-unit (95% CI: 0.00 – 0.11) decrease in fRHI in the fully-adjusted model (Table 2). The associations between $PM_{2.5}$ and CVD risk factors did not vary significantly by sex or race (p-value for interaction >0.05 for all). There were similar patterns but statistically nonsignificant associations observed for BC. (Supplemental Table III)

Environmental pollutants, race, and incident CVD and mortality outcome

Over a median follow-up period of 8.3 years (12,888 person-years of follow-up), 140 incident events (70 deaths and 70 nonfatal CVD events) were observed. Each 1.5-μg/m^3 higher concentration of $PM_{2.5}$ was associated with 1.39 (95% CI 0.96 – 1.83) increase in hazard ratio of combined all-cause mortality and CVD events, after adjusting for age and sex. The association was similar after further adjustment for race and CVD risk factors. Black carbon was not significantly associated with events. (Figure 2)

Blacks had 1.45 (95% CI, 1.00,2.09) higher risk of combined incident CVD events and all-cause mortality than Whites in models adjusted for traditional CVD risk factors. This

association was modestly attenuated to 1.34 [0.91, 1.96] with adjustment for $PM_{2.5}$ (Table 3) Mediation analyses showed that 24% of the association between race and combined clinical outcome is mediated by exposure to $PM_{2.5}$. The association between race and clinical outcome was no longer significant with adjustment for income and education. (Table 3)

Discussion

We found that Blacks had significantly higher exposures to air pollutants ($PM_{2.5}$, BC) and an increased risk of the combined endpoint of CVD events and death in a community-based cohort of adults in Western Pennsylvania. Particulate matter air pollution measured by $PM_{2.5}$ was also independently associated with increased risk of combined CVD events and all-cause mortality as well as with elevated blood glucose and worse endothelial function after accounting for potential confounders, including race. The increased risk of clinical events in Blacks was partly mediated by exposure to $PM_{2.5}$.There was no significant association of BC with clinical outcomes.

This study contributes towards a better understanding of the mechanism of racial differences in CVD events and mortality. Our findings suggest that higher exposures to $PM_{2.5}$ may contribute to the racial differences in CVD outcomes observed in the Heart SCORE cohort, and is consistent with previous studies reporting associations between airborne fine particulate matter and CVD.[5–8, 24] Of note, the association of Black race with higher risk of combined CVD events and all-cause mortality in our study was attenuated with adjustment for $PM_{2.5}$. Indeed, mediation analyses showed that approximately 24% of the association observed between race and CVD events and all-cause mortality may be explained by exposure to $PM_{2.5}$. However, this association was no longer statistically significant in models adjusting for markers of socioeconomic status (i.e., income and education), suggesting that socioeconomic status, race and exposure to environmental pollutants, have complex and interdependent relationships with CVD events and mortality. Given the high correlation between race an socioeconomic status, we did not include markers of socioeconomic status in the model used for mediation analyses.

Our findings also suggest potential mechanisms for the associations of $PM_{2.5}$ with CVD and mortality, which may include hyperglycemia and endothelial dysfunction. These findings complement prior epidemiological and basic science studies of the mechanistic pathways that relate environmental pollution and CVD.[5, 25–29] By contrast, the association of $PM_{2.5}$ with IL-6, body mass index and blood pressure was attenuated and no longer significant after adjusting for race, income and education in the present study, although prior studies have indicated significant associations, in particular with inflammatory variables.[9, 30, 31] The attenuation of the association observed in this study may be due to the intricate relationships that likely exist between race, socioeconomic status, exposure to environmental pollutants and inflammatory mileu, including confounding, effect modification and/ or effect mediation.

We observed a similar pattern of associations between BC and the various CVD outcomes as was observed for $PM_{2.5}$, although effects were not statistically significant. Reported data supporting associations between BC and CVD outcomes are limited. BC is often interpreted

as marker for diesel-related emissions, and observed associations with CVD events have been inconsistent, particularly in individuals without pre-existing atherosclerotic disease.[12, 13] The current findings suggest that other sources or components of air pollution such as $PM_{2.5}$ may be more important in the association of air pollution with CVD.

The present study has a number of strengths that merit consideration. First, we studied a racially diverse, community-based cohort of individuals not selected based on preexisting disease, such as diabetes or CVD. Hence, the findings are applicable to understanding associations between air pollution exposures, race and CVD among broad populations. Second, we were able to estimate residence-specific exposures for each participant for the year prior to clinical assessment using a spatial model for air pollution concentrations derived from a large number of concentration measures collected across the region. Third, the stability of the population in Western Pennsylvania was associated with a long residence of this cohort in their current homes, which provided a reliable and complete measure of pollution exposure over time.

Our study has a number of limitations. First, it is a single-center study and the range of air pollution concentrations across the study participants is somewhat smaller than that observed in multi-center studies such as MESA.[8, 32] The smaller range of exposures may limit our ability to detect how differences in pollution affect risk. Second, we did not have information on duration of residence of participants in each location prior to entry into the study; hence, there may be misclassification of long-term exposure status depending on how long participants lived in a certain location. Third, the significant correlation between race, socioeconomic status and exposure to air pollutants makes identifying the individual effects of these variables challenging in mutually adjusted, multivariable models in this medium-sized study. Race may be more reflective of the social construct of ethnicity rather than underlying biological differences, and hence has more likelihood of being confounded by social factors, such as education and income.

Of note, we did not assess indoor sources of $PM_{2.5}$ in the present study. Indoor air pollution is a serious concern. However, an important portion of indoor pollution is derived from outdoors, and these are importantly correlated.[33] Residence-based outdoor pollution exposure estimates, which we used in this study, are repeatedly shown to significantly predict a wide range of health outcomes in studies worldwide.[5–8] These exposure estimates do not represent the entirety of each individual's pollution exposure, but rather reflect the persistent contrast in exposures across urban cohorts.

Regarding measurement of dependent variables, we used single measurement of the CVD risk factor correlates of the environmental pollutants presented in this study. Single measurement of exposure or outcome (compared to repeat measurement) is more likely to lead to random misclassification. Such non-differential misclassification is not likely to cause a systematic bias; instead it weakens any observed association between exposure and outcome (regression dilution). Therefore, any observed association would be considered valid, although, it may be weaker than the actual underlying relationship. Prior studies of environmental exposures have estimated air pollution over long periods of time (chronic exposures), even where a given CVD risk factor is measured at only one or a few points in

time.[34, 35] This is because pollution is a minor burden that accumulates daily over many years and many years of exposure can often precede any apparent physiologic alteration

In conclusion, we found significant racial differences in exposures to urban air pollutants and outcomes in a community-based cohort in Western Pennsylvania. Exposures to $PM_{2.5}$ were associated with elevated blood glucose, worse endothelial function, and incident CVD events and all-cause mortality. Compared to Whites, Blacks had higher rate of CVD events and all-cause mortality that was partly explained by higher exposure to $PM_{2.5.}$ Further larger-sized, multicenter studies can help to better understand the role and mechanisms of environmental pollution exposures in racial differences in cardiovascular risk and outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

BC	Black carbon
BMI	Body mass index
CI	Confidence Interval
CVD	Cardiovascular disease
fRHI	Framingham reactive hyperemia index
HeartSCORE	Heart Strategies Concentrating on Risk Evaluation
HR	Hazard ratio
IL-6	Interleukin-6
$PM_{2.5}$	Particles with median aerodynamic diameter < 2.5 μm
SD	standard deviation

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Highlights

- We found Black individuals had significantly higher exposure to ambient fine particulate ($PM_{2.5}$) compared to Whites.
- Exposure to $PM_{2.5}$, was independently associated with elevated blood glucose and worse endothelial function.
- $PM_{2.5}$ was associated with a higher risk of incident CVD events and all-cause mortality combined
- Black participants, compared to Whites, had higher risk of combined incident CVD events and all-cause mortality, which was in part explained by higher concentration of $PM_{2.5}$ in Blacks.

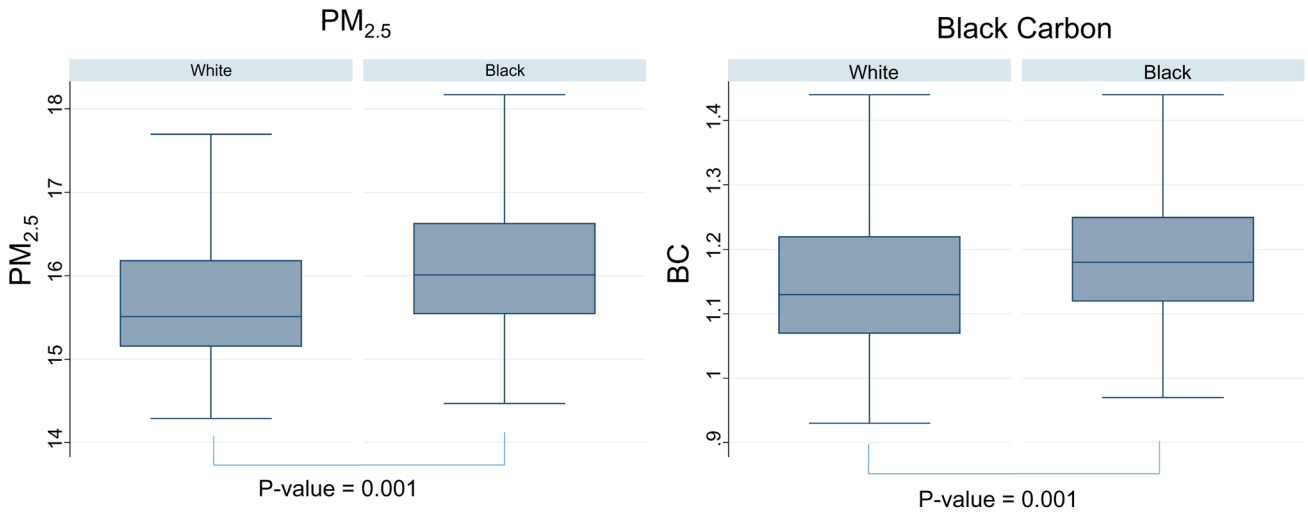

Figure 1.

Distribution of environmental pollutants by race

*Association was significant after adjusting for age, sex, smoking, income and education

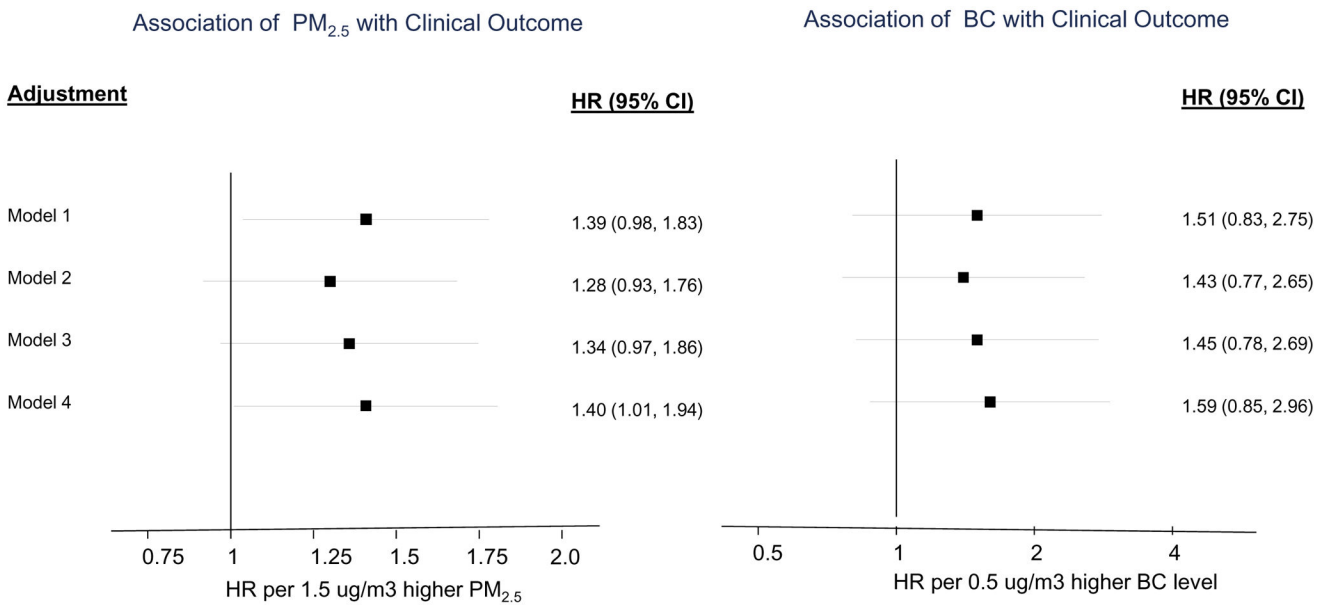

Figure 2.

Association of environmental pollutants with clinical outcomes

Model 1 - Age + sex

Model 2 – Model 1 + smoking + race

Model 3 - Model 3 + SBP + diabetes + BMI

Model 4 – Model 3 + TC + HDL-c + TG

Table 1

Baseline characteristics of participants by thirds $PM_{2.5}$

Variable	Overall summary statistics		Summary statistics by thirds of $PM_{2.5}$						p-value
	No of subjects	Mean (SD) or %	Bottom Third		Middle Third		Top Third		
			n	Mean (SD) or %	n	Mean (SD) or %	n	Mean (SD) or %	
$PM_{2.5}$ (ug/m3)	1717	15.7(0.77)	578	15.1 (0.27)	567	15.8 (0.21)	572	16.8 (0.49)	
Age (years)	1717	59 (8)	578	59 (8)	567	59 (8)	572	59 (8)	0.84
Female	1717	1129 (66%)	578	359 (62%)	567	386 (68%)	572	384 (67%)	0.07
Race - Black	1717	773 (45%)	578	150 (26%)	567	294 (52%)	572	329 (58%)	<0.0001
Race - White	1717	902 (53%)	578	411 (71%)	567	261 (46%)	572	230 (40%)	
Smoker	1713	192 (11%)	578	55 (10%)	566	67 (12%)	569	70 (12%)	0.007
Diabetes	1710	177 (10%)	575	55 (10%)	566	57 (10%)	569	65 (11%)	0.32
HTN	1715	757 (44%)	578	215 (37%)	566	261 (46%)	571	281 (49%)	<0.0001
Systolic BP	1715	137 (20)	577	136 (18)	567	137 (20)	571	138 (21)	0.05
Diastolic BP	1714	81 (10)	576	81 (10)	567	81 (10)	571	82 (10)	0.016
Glucose (mg/dl)	1711	99 (26)	576	97 (22)	565	99 (25)	570	102 (31)	<0.0001
BMI (Kg/M^2)	1701	30 (6)	571	30 (6)	562	30 (6)	568	31 (7)	<0.0001
WHR	1584	0.89 (0.09)	546	0.89 (0.08)	518	0.89 (0.09)	520	0.89 (0.09)	0.67
fRHI	1232	0.74 (0.46)	429	0.78 (0.46)	425	0.76 (0.48)	378	0.68 (0.43)	0.0013
Log-hscrp (log-mg/l)	1611	0.37 (1.24)	547	0.29 (1.17)	532	0.35 (1.28)	532	0.46 (1.25)	0.016
Log-il6 ((log-pg/ml)	1585	0.53 (0.75)	538	0.42 (0.76)	527	0.54 (0.75)	520	0.63 (0.72)	<0.0001
TC (mg/dl)	1705	213 (42)	573	217 (42)	563	213 (42)	569	209 (43)	0.005
HDL-c (mg/dL)	1705	58 (15)	573	56 (15)	563	59 (15)	569	58 (15)	1.6
Log-TG (mg/dL)	1704	4.67 (0.49)	573	4.76 (0.51)	562	4.63 (0.48)	569	4.62 (0.48)	<0.0001
Income < $10K	1554	93 (6.0)	527	20 (3.8)	506	30 (5.9)	521	43 (8.3)	<0.0001
Income - $10K–20K	1554	201 (12.9)	527	36 (6.8)	506	70 (13.8)	521	95 (18.2)	
Income - $20K–40K	1554	451 (29.0)	527	145 (27.5)	506	161 (31.8)	521	145 (27.8)	
Income - $40K–80K	1554	515 (33.1)	527	184 (34.1)	506	165 (32.1)	521	166 (31.9)	
Income > $80K	1554	294 (18.9)	527	142 (26.9)	506	80 (15.8)	521	72 (13.8)	

Variable	Overall summary statistics		Summary statistics by thirds of $PM_{2.5}$						p-value
	No of subjects	Mean (SD) or %	Bottom Third		Middle Third		Top Third		
			n	Mean (SD) or %	n	Mean (SD) or %	n	Mean (SD) or %	
Education < HS	1713	42 (2.5)	578	11 (1.9)	567	15 (2.7)	568	16 (2.8)	0.009
Education- HS+	1713	844 (49.3)	578	259 (44.8)	567	295 (52.0)	568	290 (51.1)	
Education- Bachelor+	1713	827 (48.3)	578	308 (53.3)	567	257 (45.3)	568	262 (46.1)	

$PM_{2.5}$ – particulate matter with median aerodynamic diameter < 2.5 um, HTN- hypertension, BP – blood pressure, BMI – body mass index, WHR – waist-hip ratio, fRHI - Framingham reactive hyperemia index, Hx – history, BP – blood pressure, hsCRP –high sensitivity C-reactive protein, IL6 – interleukin-6, TC – total cholesterol, HDL-c – high-density lipoprotein cholesterol, TG – triglycerides.

Table 2

Association of environmental exposure to $PM_{2.5}$ (per 1.5 ug/m^3 higher concentration) with continuous variables

Outcome	Adjustment	N	Beta (95% CI)	p-value
SBP	Unadjusted	1710	1.73(−0.07,3.54)	0.06
	Age & sex	1710	1.86(0.10,3.62)	0.04
	Above + smoking	1707	1.82(0.06,3.59)	0.04
	Above + race	1665	−0.50(−2.33,1.32)	0.59
	Above + income	1505	−1.10(−3.01,0.81)	0.26
	Above + education	1505	−0.98(−2.89,0.93)	0.32
Glucose	Unadjusted	1706	4.79(2.38,7.20)	<0.001
	Age & sex	1706	4.96(2.56,7.37)	<0.001
	Above + smoking	1702	5.01(2.60,7.43)	<0.001
	Above + race	1660	3.72(1.14,6.29)	<0.001
	Above + income	1499	3.63(0.92,6.35)	0.01
	Above + education	1499	3.71(0.99,6.42)	0.01
BMI	Unadjusted	1696	1.08(0.53,1.63)	<0.001
	Age & sex	1696	1.06(0.51,1.61)	<0.001
	Above + smoking	1694	1.10(0.55,1.65)	<0.001
	Above + race	1652	0.16(−0.41,0.72)	0.59
	Above + income	1495	0.16(−0.44,0.76)	0.60
	Above + education	1495	0.19(−0.42,0.79)	0.54
fRHI	Unadjusted	1229	−0.09(−0.14,−0.03)	<0.001
	Age & sex	1229	−0.09(−0.14,−0.04)	<0.001
	Above + smoking	1228	−0.09(−0.14,−0.04)	<0.001
	Above + race	1196	−0.05(−0.10,0.00)	0.06
	Above + income	1076	−0.06(−0.11,−0.00)	0.05
	Above + education	1076	−0.06(−0.11,−0.00)	0.05
Log-hsCRP	Unadjusted	1608	0.14(0.02,0.26)	0.02
	Age & sex	1608	0.12(0.01,0.24)	0.04
	Above + smoking	1605	0.11(−0.01,0.22)	0.07
	Above + race	1566	−0.03(−0.15,0.09)	0.61
	Above + income	1413	−0.04(−0.17,0.08)	0.51
	Above + education	1413	−0.04(−0.17,0.09)	0.55
Log-IL6	Unadjusted	1582	0.18(0.11,0.25)	<0.001
	Age & sex	1582	0.18(0.11,0.25)	<0.001
	Above + smoking	1578	0.17(0.10,0.24)	<0.001
	Above + race	1538	0.06(−0.01,0.14)	0.09
	Above + income	1386	0.05(−0.02,0.13)	0.18

Outcome	Adjustment	N	Beta (95% CI)	p-value
	Above + education	1386	0.06(−0.02,0.13)	0.15

$PM_{2.5}$ – particulate matter with median aerodynamic diameter < 2.5 um, SBP – systolic blood pressure, BMI – body mass index, fRHI - Framingham reactive hyperemia index, hsCRP –high sensitivity CRP, IL6 – interleukin-6

Table 3

Effect of adjusting for $PM_{2.5}$ or BC on the association between race and combined CVD events and all-cause mortality outcomes

Adjustment	N	Cases	HR 95% (CI)	P-value	HR 95% (CI)	P-value
			Adjusted for model on the left		Further adjusted for $PM_{2.5}$	
Model 1	1616	139	1.78(1.27,2.49)	0.00	1.69(1.19,2.40)	0.00
Model 2	1596	139	1.42(0.99,2.03)	0.06	1.32(0.91,1.92)	0.14
Model 3	1586	136	1.45(1.00,2.09)	0.05	1.34(0.91,1.96)	0.14
Model 4	1437	124	1.29(0.86,1.93)	0.22	1.23(0.81,1.87)	0.33
			Adjusted for model on the left		Further adjusted for BC	
Model 1	1616	139	1.78(1.27,2.49)	0.00	1.75(1.25,2.46)	0.00
Model 2	1596	139	1.42(0.99,2.03)	0.06	1.39(0.97,1.99)	0.08
Model 3	1586	136	1.45(1.00,2.09)	0.05	1.41(0.97,2.04)	0.07
Model 4	1437	124	1.29(0.86,1.93)	0.22	1.26(0.84,1.90)	0.27

Model 1: Age & sex

Model 2: Age, sex, smoking, SBP, diabetes, BMI

Model 3: Age, sex, smoking, SBP, diabetes, BMI, TC & HDL-c

Model 4: Age, sex, smoking, SBP, diabetes, BMI, TC, HDL-c, income & education