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ORIGINAL RESEARCH ARTICLE

Particulate Matter Air Pollution Exposure and Heart Disease Mortality Risks by Race and Ethnicity in the United States

1997 to 2009 National Health Interview Survey With Mortality Follow-Up Through 2011

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BACKGROUND: Most US studies of mortality and air pollution have been conducted on largely non-Hispanic white study populations. However, many health and mortality outcomes differ by race and ethnicity, and non-Hispanic white persons experience lower air pollution exposure than those who are non-Hispanic black or Hispanic. This study examines whether associations between air pollution and heart disease mortality differ by race/ethnicity.

METHODS: We used data from the 1997 to 2009 National Health Interview Survey linked to mortality records through December 2011 and annual estimates of fine particulate matter ($PM_{2.5}$) by census tract. Proportional hazards models were used to estimate hazard ratios and 95% confidence intervals between $PM_{2.5}$ (per 10 μg/m^3) and heart disease mortality using the full sample and the sample adults, which have information on additional health variables. Interaction terms were used to examine differences in the $PM_{2.5}$-mortality association by race/ethnicity.

RESULTS: Overall, 65 936 of the full sample died during follow-up, and 22 152 died from heart disease. After adjustment for several factors, we found a significant positive association between $PM_{2.5}$ and heart disease mortality (hazard ratio, 1.16; 95% confidence interval, 1.08–1.25). This association was similar in sample adults with adjustment for smoking and body mass index (hazard ratio, 1.18; 95% confidence interval, 1.06–1.31). Interaction terms for non-Hispanic black and Hispanic groups compared with the non-Hispanic white group were not statistically significant.

CONCLUSIONS: Using a nationally representative sample, the association between $PM_{2.5}$ and heart disease mortality was elevated and similar to previous estimates. Associations for non-Hispanic black and Hispanic adults were not statistically significantly different from those for non-Hispanic white adults.

Key Words: air pollution ■ heart disease ■ Hispanic origin ■ linked mortality files ■ mortality ■ National Health Interview Survey ■ race

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Clinical Perspective

What Is New?

- This study describes the association between air pollution and heart disease mortality using nationally representative population health data in the United States, with a focus on whether the association differs by race and ethnicity.
- The association between air pollution and health disease mortality in the national sample was elevated and similar to estimates found in prior studies.
- After controlling for sociodemographic and geographic factors, associations between air pollution and heart disease mortality for non-Hispanic black and Hispanic adults were not statistically significantly different from that for non-Hispanic white adults.

What Are the Clinical Implications?

- Many studies of air pollution and mortality in the United States have used subnational cohorts of largely non-Hispanic white adults. This study supports the application of findings from these studies for adults of other races and ethnicities in the United States.

Published studies of chronic effects of long-term particulate matter air pollution exposure and mortality outcomes have long been used to inform air quality standards set by the US Environmental Protection Agency (EPA). Two of the original studies were the Six Cities Study[1] and the American Cancer Society study,[2] with further analyses of these cohorts[3–9] using longer follow-up time. Numerous studies have subsequently been, and continue to be, published in the United States and elsewhere.[10] Additional cohorts recently used to assess long-term air pollution and mortality in the United States include the Health Professional's Study,[11] the Nurses' Health Study,[12–14] the Women's Health Initiative,[15] and the California Teachers Study.[16,17] The American Heart Association has concluded that the evidence is consistent with a causal relationship between fine particulate matter ($PM_{2.5}$) exposure and cardiovascular morbidity and mortality.[18]

Most US studies have been limited to predominantly white study populations; in studies using more heterogeneous cohorts, sample sizes have been insufficient to examine separate associations by race and ethnicity or to examine whether associations differ by race and ethnicity. However, disadvantaged populations are overrepresented in areas with poorer air quality[19–21] and closer proximity to roads, a source of particulate matter air pollution.[22]

Associations between $PM_{2.5}$ and mortality could differ among race and ethnicity groups for a variety of reasons, including differences in comorbidities and other risk factors. In the United States, non-Hispanic white adults are less likely to be obese[23] or have diabetes mellitus[24] than non-Hispanic black and Hispanic adults. Non-Hispanic black adults are more likely to have hypertension than either non-Hispanic white or Hispanic adults.[25] More generally, non-Hispanic white adults are more likely to report excellent or very good health compared with Hispanic and non-Hispanic black adults.[26] Among these 3 groups, non-Hispanic black adults have the highest and Hispanic adults have the lowest death rates.[26,27]

Associations between air pollutant concentrations and mortality for the non-Hispanic black and Hispanic populations in the United States were recently examined in the Medicare population by Di and colleagues.[28] The Di study of >60 million Medicare enrollees reported that air pollution effects were more pronounced among self-identified racial minorities and those with Medicaid eligibility, where Medicaid eligibility served as a proxy for low income.

The objective of this analysis is to investigate the association between air pollution and heart disease mortality in the United States among adults ≥25 years of age, with attention to whether the association differs by race and ethnicity. Differences in exposure along with differing vulnerabilities among these groups related to underlying health and factors related to socioeconomic status may affect associations between air pollution and mortality.[29]

Toward this objective, we used the 1997 to 2009 National Health Interview Survey (NHIS) linked to the National Death Index (NDI) through 2011. Modeled and monitored air quality information obtained from the National Environmental Public Health Tracking Program, Centers for Disease Control and Prevention, for 2004 was merged by census tract. The NHIS is a nationally representative survey and includes a large number of non-Hispanic black and Hispanic adults. In addition, the NHIS includes information on underlying health status, health behaviors, and socioeconomic status.

METHODS

Data Access

The restricted use–linked NHIS-NDI files described previously[30] are available to researchers through the National Center for Health Statistics Research Data Center.[31] The air quality data described previously[32] are available from the National Center for Environmental Health and can be combined with the NHIS-NDI files in the Research Data Center. One author (N.K.) had full access to the data and performed the primary data analysis.

Data

The NHIS is a large nationally representative survey of the civilian noninstitutionalized US population. Briefly, the NHIS

is a cross-sectional household interview survey that has been conducted continuously throughout the year since the 1950s. Although the design of the survey changes over time, in general, the first stage of its multistage probability design consists of a sample of primary sampling units (PSUs) drawn from a set of geographically defined PSUs. PSUs are counties or groups of counties or a metropolitan statistical area. Approximately every decade, the NHIS design changes, including changes in PSUs and oversampled subgroups. The probabilities of selection, along with adjustments for nonresponse and poststratification, are reflected in the sample weights. Additional information is available elsewhere.[33,34]

NHIS survey years 1985 to 2009 have been linked with mortality follow-up through December 31, 2011. Mortality information is based on the results from a probabilistic match between the NHIS and NDI death certificate records. The NHIS-NDI files are described elsewhere.[30]

Restricted-use NHIS data files with geographic information, including county of residence and other census units (eg, tracts), allowed the merging of the air quality indicators described later.

Data collection and informed consent procedures for the NHIS and linkage of the NHIS to the NDI are approved by the National Center for Health Statistics Ethics Review Board. The health data used in this article are deidentified survey data linked to mortality status, with exposures aggregated to the census tract level. Research with deidentified survey data are not subject to the federal regulations on the protection of human research subjects, 45 CFR 46.101(b)(4).

This study is limited to the 1997 to 2009 NHIS. Although the sample sizes varied during these years, generally ≈35 000 to 40 000 households (yielding 80 000–100 000 respondents) were sampled annually. NHIS household response rates during this time period ranged from a high of 92% in 1997 to a low of 82% in 2009. Of those in the survey, some had insufficient data for linkage and were not included. The 1995 to 2005 sample design included 358 PSUs, and the 2006 to 2015 design included 428 PSUs. During the years used for this study, non-Hispanic black and Hispanic populations were among the subgroups that were oversampled.

Air Quality Data

The Centers for Disease Control and Prevention have been collaborating with the US EPA on the development of air quality metrics for public health surveillance.[35,36] For this analysis, measurements of daily 24-hour average $PM_{2.5}$ concentrations ($\mu g/m^3$) were obtained from the US EPA's Data Mart[37] for monitors designated as Federal Reference Methods in the Air Quality System (AQS). A daily census tract-level dataset was created by estimating the average concentration among all monitors within the tract for each monitored day. In addition, we obtained daily estimates of $PM_{2.5}$ that were generated using a Bayesian space-time downscaler fusion model developed by the US EPA and its partners.[38] The downscaler modeling approach combines output from the Community Multiscale Air Quality model with measurements from the AQS, yielding air quality predictions at specific point locations. The theory, development, and initial evaluation of the downscaler have been published.[38–41] Daily predictions of 24-hour average $PM_{2.5}$ concentrations ($\mu g/m^3$) were generated at the 2010 US census tract centroid locations over the entire conterminous United States using the US EPA's downscaler software.[38] In addition, daily county-level estimates of $PM_{2.5}$ were generated from both downscaler and AQS data products using a population-weighted approach, where tract populations were used to weight daily tract-level $PM_{2.5}$.

Using the restricted-use NHIS files with geographic information, our analysis relied primarily on census tract concentrations. County-specific exposure concentrations were used in some sensitivity analyses. For the primary analysis, we used annual average $PM_{2.5}$ estimates from the downscaler model, averaged over the calendar year 2004, the approximate midpoint of the study period, and calculated by census tract. For comparison, we evaluated several alternative $PM_{2.5}$ concentration estimates, including 2004 monitored estimates (AQS county level) and tract-level downscaler estimates for other years (2001, 2008). These air quality indicators were linked to NHIS respondents at the census tract level.

We used a single year (2004) for the primary analysis to simplify the exposure assignment for multiple reasons. First, survey respondents entered the study and, if they died, exited the study at different times. Although average exposures based on all follow-up time could have been calculated, this approach would have required appropriate control of exposure assignment for the survivors to avoiding biasing comparisons. In addition, these exposures would not capture time-use patterns, residential mobility, or exposure occurring before survey participation. Second, for survey years before 2000, $PM_{2.5}$ measurements are less consistently available, making the aforementioned method untenable for earlier survey years. Third, we found high correlations in exposure concentrations across years, suggesting that relative exposures across locations may be similar at the national level.

Mortality Outcomes

Our primary outcome was heart disease mortality. Because the *International Classification of Diseases* codes changed from the *International Classification of Diseases, Ninth Revision*, to the *International Classification of Diseases, Tenth Revision*, during the years of these data, we used the composite codes available on the NHIS-NDI file to define heart disease mortality (variable U-113, codes 056–064 and 067–071, in the online documentation[42]). In secondary analyses, we examined all-cause mortality (excluding unintentional injury U-113 codes 114–123), ischemic heart disease mortality (U-113 codes 059–063), and cerebrovascular disease mortality (U-113 code 070).

Covariates

The NHIS includes information on numerous health-related indicators; however, some indicators differ by survey year, both in the actual questions and the allowed responses. Because the span of survey years in this analysis used different survey instruments, an effort was made to create a set of comparable covariates related to mortality.

Race and Hispanic origin are collected in the NHIS using 2 separate questions. Although the allowed survey responses have changed over time, particularly in the collection of multiple-race information, responses were categorized for this study as

non-Hispanic white, non-Hispanic black, Hispanic, and all other races and ethnicities (which includes multiple races). Although the other category is too heterogeneous for meaningful inference, respondents who reported other races and ethnicities are included in the total, and estimates are shown for completeness.

Age at interview (single years) and sex (male or female) were easily combined across survey years. Education at the time of interview was coded into the following categories: less than high school education, high school diploma or General Equivalency Diploma, and more than high school diploma or General Equivalency Diploma. Marital status at the time of the interview was coded as married, widowed, never married, or divorced/separated.

The percentage of poverty level was based on family income and family size and composition using US Census Bureau poverty thresholds. Because reported family income on the NHIS is often missing, we used the multiply imputed income files to obtain poverty status.[43]

During the survey data years used for this study, only 1 adult in a family was asked for detailed health information on the sample adult questionnaire. As a result, fewer participants have information for body mass index (BMI). BMI is equal to weight (in kg) divided by height (in cm) squared. BMI was categorized as <18.4, 18.4–25, 25–30, and >30) and smoking status (never, former, current) at the time of interview. The impact of these variables on the associations is assessed in separate models, and the smaller subset is referred to as the sample adult subset.

Three county-level characteristics were included: median household income for 2000, urbanization, and climate region of the country. Urbanization was coded using the 2006 Urban Rural Classification Scheme for Counties.[44] Using this scheme, counties were coded into 6 groups: large central metropolitan counties, large fringe metropolitan counties, medium metropolitan counties, small metropolitan counties, micropolitan counties, and noncore counties (nonmetropolitan counties that are not in a micropolitan statistical area). Climate regions are aggregations of states based on homogenous long-term climatology. A description of these regions is available from the National Center for Environmental Information.[45]

Study Population

Using the 1997 to 2009 NHIS-linked mortality files, this study was limited to adults ≥25 years of age at interview, eligible for the NDI linkage, who were living in 1 of the 48 contiguous states. Of these adults, 0.4% were missing information on marital status, 1.0% were missing information on education, and 1.0% were missing information on health status. After deleting records missing data for ≥1 of those variables, 657 238 adults were available for the full-sample analysis. Smoking status and BMI were only asked of 1 adult per household. Among this group, 320 512 had complete data for the sample adult subset analyses.

Analysis

Statistical Methods

Discrete-time proportional hazards models were used to describe the relationship between mortality outcomes and air pollution indicators. For these models, the date of the interview was considered the beginning time and the date of death for decedents was considered the end time. In the heart disease analyses, decedents with other causes of death were censored at the date of death. For survivors, death was censored at December 31, 2011, the last day of mortality follow-up. All models were stratified by sex and age at interview, which was grouped into 10-year intervals (25–34, 35–44, 45–54, 55–64, 65–74, 75–84, ≥85) to allow for sufficient sample sizes for assessing interactions and certain cause-specific analyses. Sensitivity analyses examined using age in 5-year intervals (with top coding at 85 years of age). Additional sensitivity analyses assessed using age as the time scale in the survival models, with exact date of birth at the beginning time rather than the interview date.[46,47]

Hazard ratios (HRs) are expressed per 10 U of $PM_{2.5}$ concentration, where $PM_{2.5}$ concentration is modeled linearly as a continuous variable. The relationship between $PM_{2.5}$ concentration and mortality has been shown to be close to linear for daily mortality,[48] and this simplifying assumption facilitates comparisons by race/ethnicity in this study and with prior studies that report HR on a linear scale. Sensitivity analyses examined quartiles of $PM_{2.5}$ rather than $PM_{2.5}$ as a continuous variable.

A series of adjusted models were fit for each mortality outcome. First, we included poverty status, education, and marital status as covariates. We fit additional models controlling for geographic (region and urbanicity) and demographic (median household income within census tract) contextual variables. Because health-related factors may act as either confounders or intervening variables, separate models were fit with health status, cigarette smoking, and BMI. Models including smoking and BMI were fit to the sample adult subset.

To address our study question, models included interaction terms for race/ethnicity and air pollution.

SUDAAN software was used for the complex survey design of the NHIS. We used survey weights recalibrated in SUDAAN to control for the differential ineligibility for the NDI match and, as needed, for the unavailable exposure information (AQS models).[49]

RESULTS

The description of the study population is shown in Table 1. Demographic and geographic factors, such as age group, marital status, education, and poverty status, all differed by race and ethnicity groups. For example, ≈10% of the non-Hispanic white adults were ≥75 years of age at the time of the interview compared with ≈6% of the non-Hispanic black and <4% of the Hispanic adults. Fewer than half of non-Hispanic white adults (≈20%) lived in the most urban counties at the interview compared with >40% of non-Hispanic black (43%) and ≈50% of Hispanic (52%) adults. Patterns were similar using the sample adult subset, although this sample adult subset had relatively fewer males and fewer who were married at the time of the interview than the full sample (data not shown).

The distribution of $PM_{2.5}$ by quartile differed by race/ethnicity (P<0.01). About 37% of non-Hispanic black and one third of Hispanic adults lived in areas with $PM_{2.5}$ concentrations in the highest quartile in 2004,

Table 1. Characteristics of Study Participants

	Distribution, %*				
	Race and Ethnicity				All Other Races and Ethnicities
	Total	Non-Hispanic White	Hispanic	Non-Hispanic Black	
	n=657 238	n=422 450	n=121 109	n=84 880	n=28 799
Characteristic					
Sex					
Male	47.8	48.0	50.1	43.9	47.2
Female	52.2	52.0	49.9	56.1	52.8
Age at interview, y					
25–35	21.3	18.6	32.5	24.8	28.8
36–45	23.5	22.3	28.0	25.6	26.2
46–55	21.6	22.0	18.5	22.0	21.2
56–65	15.1	16.2	10.6	14.0	12.8
66–75	10.1	11.0	6.5	8.3	7.1
>75	8.5	9.9	3.8	5.7	4.0
Marital status					
Married	64.8	67.5	65.4	43.6	71.1
Widowed	7.4	7.9	4.4	8.4	4.8
Divorced/separated	13.7	13.1	13.6	20.4	8.5
Never married	14.1	11.6	16.7	27.7	15.6
Education					
< 12 y or no high school	16.5	12.0	43.4	21.1	13.3
High school or GED	30.0	31.0	24.7	32.3	20.3
More than high school	53.5	57.0	31.9	46.6	66.4
Family income (percentage of poverty threshold)					
<100%	10	6.7	21.3	19.8	11.3
100% to 199%	17.4	14.9	28.6	23.0	16.9
200% to 299%	17.1	16.9	18.2	18.1	15.6
300% to 399%	14.5	15.2	11.6	13.0	13.1
400% to 499%	11.3	12.2	7.4	8.9	10.6
≥500%	29.8	34.0	13.0	17.2	32.5
Urbanization[44]					
Large central metropolitan	27.5	20.4	51.7	42.9	46.9
Large fringe metropolitan	24.6	26.0	17.4	21.5	26.2
Medium metropolitan	20.7	22.1	17.6	17.9	13.8
Small metropolitan	9.9	11.3	5.7	6.9	5.2
Micropolitan (nonmetropolitan)	10.7	12.4	5.5	6.7	5.1
Noncore	6.7	7.9	2.3	4.1	2.9
Climate region[45]					
Central	17.4	19.8	5.8	16.1	9.0
East North Central	8.9	10.6	2.1	5.8	5.9
Northeast	21.8	22.6	15.5	23.1	21.0
South	13	11.4	21.8	16.4	9.7
Southeast	17.9	16.9	14.5	30.7	11.5
Southwest	4.8	4.7	8.9	1.1	4.8
West	10.6	7.4	28.7	5.8	31.3

(*Continued*)

Table 1. Continued

	Race and Ethnicity				All Other Races and Ethnicities
	Total	Non-Hispanic White	Hispanic	Non-Hispanic Black	
	n=657 238	n=422 450	n=121 109	n=84 880	n=28 799
Northwest	3.9	4.5	2.4	0.8	5.7
West North Central	1.7	2.2	0.3	0.3	1.1
Quartiles† of $PM_{2.5}$ concentrations (μg/m³)					
<10.1	25.2	26.1	34.7	10.5	23.4
≥10.1 but <11.8	25.0	26.9	15.8	22.1	24.6
≥11.8 but <13.6	25.0	25.8	16.3	30.2	21.8
≥13.6	24.8	21.3	33.3	37.2	30.2

GED indicates General Equivalency Degree.
*Percentages may not sum to 100% because of rounding.
†Quartile values are also shown in Table 2.
Source: 1997 to 2009 National Health Interview Survey with mortality follow-up through December 30, 2011.[30]

>13.6 μg/m³. In contrast, only 21% of non-Hispanic white adults lived in areas with $PM_{2.5}$ concentrations in the highest quartile (Table 1).

Overall, 65 936 of the full analytic sample died during follow-up (9112 non-Hispanic black, 47 654 non-Hispanic white, and 7472 Hispanic). Of these, 22 152 died from heart disease (3249 non-Hispanic black, 16 171 non-Hispanic white, and 2237 Hispanic). In the sample adult subset, 37 683 died during follow-up (5398 non-Hispanic black, 28 165 non-Hispanic white, and 3415 Hispanic); 13 000 died from heart disease.

Percentiles of $PM_{2.5}$ levels calculated for 2004, 2001, and 2008 by census tract and for 2004 using only AQS values were calculated using the weighted values for the sample and are shown in Table 2. Weighted correlations for survey participants between years for the tract-level estimates were 0.96 for years 2001 and 2004 and 0.91 for years 2004 and 2008.

Using the whole sample, the HR for the unadjusted association between heart disease mortality and 2004 $PM_{2.5}$ was 1.19 (95% confidence interval [CI], 1.12–1.27) (Table 3). In the fully adjusted model using the whole sample (after controlling for demographic, health, contextual factors, and survey year), the estimated HR decreased to 1.16 (95% CI, 1.08–1.25). This estimate was similar using the sample adult subset and adjusting for BMI and smoking (HR, 1.18; 95% CI, 1.06–1.31).

For all-cause mortality, the unadjusted HR (1.08; 95% CI, 1.04–1.13) decreased to 1.03 (95% CI, 0.99–

Table 2. Distribution of $PM_{2.5}$ μ/m³ Annual Concentration (Weighted)

	10th Percentile	25th Percentile	Median	75th Percentile	90th Percentile
2004 DS whole sample	8.7	10.1	11.8	13.6	14.7
2008 DS whole sample	8.0	9.7	11.1	12.3	13.1
2001 DS whole sample	9.2	10.9	13.1	15.2	16.8
2004 DS AQS subsample*	8.8	10.6	12.3	14.0	15.1

AQS indicates Air Quality System; DS, downscaler; and $PM_{2.5}$, fine particulate matter.
*The AQS subsample has 458 920 adults.

Table 3. Hazard Ratios and 95% Confidence Intervals for Association Between $PM_{2.5}$ (per 10 μg/m³) and Mortality

	Full Sample			Sample Adult Subset		
	HR	95% CI		HR	95% CI	
Heart disease						
Unadjusted	1.19	1.12	1.27			
Plus demographic factors*	1.16	1.10	1.24			
Plus reported health status	1.13	1.06	1.20			
Plus contextual factors†	1.17	1.08	1.26			
Plus survey year	1.16	1.08	1.25	1.16	1.05	1.29
Plus BMI/smoking				1.18	1.06	1.31
All cause‡						
Unadjusted	1.08	1.04	1.13			
Plus demographic factors*	1.06	1.02	1.10			
Plus reported health status	1.03	0.99	1.07			
Plus contextual factors†	1.04	0.99	1.09			
Plus survey year	1.03	0.99	1.08	1.05	0.99	1.12
Plus BMI/smoking				1.08	1.01	1.16

BMI indicates body mass index; CI, confidence interval; HR, hazard ratio; and $PM_{2.5}$, fine particulate matter.
*Demographic factors include sex, family income as percentage of poverty threshold, marital status, education, and race/ethnicity.
†Contextual factors include county-level income, region of the county, and urbanization.
‡All-cause mortality excludes unintentional injuries.

1.08) in the fully adjusted model. Controlling for BMI and smoking status using the sample adult subset increased the HR for all-cause mortality slightly (HR, 1.08; 95% CI, 1.01–1.16).

HRs for heart disease and all-cause mortality by race and ethnicity from models with interaction terms for race/ethnicity and $PM_{2.5}$ are shown in Table 4. Although HRs were not identical by race and ethnicity, *P* values for the interaction terms (data not shown) were not statistically significant in these models for the non-Hispanic black and Hispanic groups, indicating insufficient evidence that associations for these groups differ from the association for the non-Hispanic white group. In the models for heart disease, *P* values for the other races and ethnicities group indicated that the relationship for that group differed from that for the non-Hispanic white group.

Using the full sample, the HR for the association between $PM_{2.5}$ and ischemic heart disease mortality in the fully adjusted model was 1.13 (95% CI, 1.01–1.26), and the HR for the association between cerebrovascular heart disease mortality and $PM_{2.5}$ was 1.15 (95% CI, 0.97–1.37) (data not shown). In fully adjusted models with interaction terms for race/ethnicity and $PM_{2.5}$, interaction terms were not significant (data not shown).

Sensitivity Analysis

In sensitivity analyses, HR estimates for the models using age as the time scale were close to those presented using time since interview (data not shown) and did not lead to different conclusions. HR estimates using 5-year strata in place of 10-year strata were similar. For example, in the full sample with full adjustment, the HR for heart disease death was 1.18 (95% CI, 1.10–1.26), and in the sample adults subset with full adjustment, including adjustment for BMI and smoking, the HR for heart disease death was 1.21 (95% CI, 1.09–1.34); interactions by race and ethnicity were not significant.

Sensitivity analyses for the $PM_{2.5}$ measure were robust to other years of $PM_{2.5}$ estimates and to using the modeled versus monitored air data among the 458 920 respondents in locations with AQS county-level data (Table 5). In the full sample, with full adjustment, using the quartiles of $PM_{2.5}$ shown in Tables 1 and 2 rather than the continuous variable led to the following HR for heart disease death for the second, third, and fourth quartiles, respectively, compared to the first: 1.04 (0.98–3.04), 1.07 (1.02–3.05), and 1.09 (1.14–3.11).

Finally, HR estimates from full models fit to the full sample for all-cause mortality, without excluding unintentional injuries, were nearly identical (data not shown).

DISCUSSION

Our findings suggest a positive relationship between $PM_{2.5}$ and heart disease mortality using a large, nationally representative sample of US adults. Overall, the risk of heart disease mortality increases ≈16% for each 10-U increase in $PM_{2.5}$ concentration. Estimates were generally robust to covariate adjustment, including adjustment for demographic factors, reported health status, urbanization, and region of the county. Further adjustment for BMI and smoking status at baseline had minimal impact on the HR estimate.

Consistent with other studies that have documented disparities in exposures,[19–21] we showed that the distribution of $PM_{2.5}$ by quartile differed by race/ethnicity (Table 1). More than one-third of Hispanic adults live in areas

Table 4. Hazard Ratio and 95% Confidence Interval for Association Between $PM_{2.5}$ (per 10 μg/m³) and Mortality by Race and Ethnicity

	Full Sample*			Sample Adult Subset*			Sample Adult Subset†		
	HR	95% CI		HR	95% CI		HR	95% CI	
Heart disease									
Non-Hispanic white	1.21	1.11	1.32	1.19	1.06	1.33	1.20	1.07	1.35
Hispanic	1.07	0.91	1.25	1.17	0.92	1.48	1.23	0.97	1.56
Non-Hispanic black	1.08	0.89	1.32	1.06	0.80	1.40	1.03	0.78	1.36
All other races and ethnicities	0.81	0.57	1.15	0.96	0.58	1.58	0.98	0.59	1.61
All cause‡									
Non-Hispanic white	1.05	1.00	1.11	1.06	0.98	1.14	1.09	1.01	1.17
Hispanic	0.97	0.88	1.06	1.01	0.89	1.15	1.09	0.95	1.24
Non-Hispanic black	1.11	0.97	1.28	1.18	0.99	1.41	1.14	0.94	1.37
All other races and ethnicities	0.89	0.70	1.13	0.89	0.63	1.26	0.93	0.66	1.30

BMI indicates body mass index; CI, confidence interval; HR, hazard ratio; and $PM_{2.5}$, fine particulate matter.

*Model includes interaction term for $PM_{2.5}$ and race/ethnicity. Adjusted for sex, family income as percentage of poverty threshold, marital status, education, county-level income, region of the county, urbanization, and survey year.

†Also adjusted for BMI and smoking.

‡All-cause mortality excludes unintentional injuries.

Table 5. Hazard Ratios and 95% Confidence Intervals for Association Between $PM_{2.5}$ (per 10 U) and Mortality Using Alternative Exposure Measures

	All		
	HR	95% CI	
Heart disease			
Adjusted model* using 2001 $PM_{2.5}$, full sample	1.15	1.06	1.25
Adjusted model using 2008 $PM_{2.5}$, full sample	1.15	1.01	1.32
Adjusted model using 2004 AQS data, full sample in places with AQS data†	1.17	1.06	1.28
All cause‡			
Adjusted model using 2001 $PM_{2.5}$, full sample	1.02	0.98	1.06
Adjusted model using 2008 $PM_{2.5}$, full sample	1.02	0.96	1.08
Adjusted model using 2004 AQS data, full sample in places with AQS data†	1.00	1.00	1.01

AQS indicates Air Quality System; CI, confidence interval; HR, hazard ratio; and $PM_{2.5}$, fine particulate matter.

*Adjusted for sex, family income as percentage of poverty threshold, marital status, education, reported health status, race/ethnicity, county-level income, region of the county, urbanization, and survey year.

†AQS sample size is 458 920.

‡All-cause mortality excludes unintentional injuries.

in the lowest quartile of $PM_{2.5}$ levels, whereas only ≈10% of non-Hispanic black adults are in these lower exposure areas. However, both of these groups are overrepresented in the top quartile of exposure. These differences may be related to patterns of urbanization and region.

We found no statistically significant evidence that associations between $PM_{2.5}$ and heart disease mortality differ for Hispanic and non-Hispanic black adults compared with non-Hispanic white adults. In other words, the interaction terms for $PM_{2.5}$ with Hispanic and non-Hispanic blacks in the models were not statistically significant at the 0.05 level. Although not statistically significant, some HR estimates between non-Hispanic black and non-Hispanic white adults differed by >10%. Although this study included >120 000 Hispanic and ≈85 000 non-Hispanic black adults, the survey design only sampled from ≈350 to 430 locations during each sample design. In addition to increasing the design effect, the clustering limits the exposure variation and geographic residential differences among groups. Although we controlled for many factors that could affect differences in the mortality-$PM_{2.5}$ relationships, other factors not examined here may affect race and ethnicity differences in these associations. We did not examine effect modification by including interaction terms for factors other than race/ethnicity, such as other environmental factors (eg, temperature and ozone), socioeconomic status, occupation, BMI, or diagnosed diabetes mellitus, which, if present, could affect our inferences. Additionally, factors may be related to the mortality linkage that affects race/ethnicity-specific comparisons; linkages for Hispanics, for example, may be incomplete because of missing linkage information.[50,51]

Unlike the study by Di and colleagues[28] that reported stronger relationships for the risk of death and $PM_{2.5}$ for Hispanic and non-Hispanic black Medicare beneficiaries, we found no statistically significant differences in the association between all-cause mortality and $PM_{2.5}$ among these groups. In addition to the much smaller size of our study, we examined adults ≥25 years of age, not just Medicare beneficiaries.

The associations between $PM_{2.5}$ and mortality reported in our study are similar to those reported by earlier studies. A review by Hoek and colleagues[10] reported a pooled increased risk of 11% for cardiovascular mortality, albeit with heterogeneity among the studies. The same study reported a pooled risk 6% per 10-U increase in $PM_{2.5}$ for overall mortality, which is slightly higher than the 3% we report for overall mortality for the fully adjusted model using the full sample, although the CI includes 1.0. The pooled risk of 6% is slightly lower than the 8% we report for the fully adjusted model, with additional adjustment for BMI and smoking, using the sample adult subset.

We relied on a single year of pollution concentration estimates assigned at the census tract level for our main inferences. However, consistent with a recent analysis of the Nurse's Health Study,[12] our results were robust when limited to participants close to an air monitor; they were also robust using estimates from other years. The high correlations of tract-level $PM_{2.5}$ across years suggest that relative exposures among locations were similar over time.

In summary, we find no differential association between particulate pollution and heart disease mortality for Hispanic and non-Hispanic black adults compared with non-Hispanic white adults in the United States using a large, nationally representative sample. Overall associations (and those for non-Hispanic white adults) are comparable to those reported in other studies. These findings provide additional support for the application of findings from studies using largely non-Hispanic white cohorts to policies targeted to the general US population.

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