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Particulate Air Pollution and Clinical Cardiovascular Disease Risk Factors

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Abstract

BACKGROUND—Long-term exposure to ambient particulate matter (PM) air pollution is associated with increased cardiovascular disease (CVD); however, the impact of PM on clinical risk factors for CVD in healthy subjects is unclear. We examined the relationship of PM with levels of circulating lipids and blood pressure in the Third National Health and Nutrition Examination Survey (NHANES III), a large nationally-representative US survey.

METHODS—This study was based on 11,623 adult participants of NHANES III (1988–1994; median age 41.0). Serum lipids and blood pressure were measured during the NHANES III examination. Average exposure for 1988–1994 to particulate matter <10μm in aerodynamic diameter (PM_{10}) at the residences of participants was estimated based on measurements from U.S. Environmental Protection Agency monitors. Multivariate linear regression was used to estimate the associations of PM_{10} with lipids and blood pressure.

RESULTS—An interquartile range width (IQR_w) increase in PM_{10} exposure (11.1 $\mu g/m^3$) in the study population was associated with 2.42 percent greater serum triglycerides (95% confidence interval [CI]: 1.09–3.76); multivariate adjusted means of triglycerides according to increasing quartiles of PM_{10} were 137.6, 142.5, 142.6, and 148.9 mg/dL, respectively. An IQR_w increase in PM_{10} was associated with 1.43 percent greater total cholesterol (95% CI: 1.21–1.66). These relationships with triglycerides and total cholesterol did not differ by age or region. Associations of PM_{10} with blood pressure were modest.

CONCLUSIONS—Findings from this large diverse study indicate that greater long-term PM_{10} exposure is associated with elevated serum triglycerides and total cholesterol, potentially mediating air pollution-related effects on CVD.

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Keywords

air pollution; particulate matter; cardiovascular disease; triglycerides; cholesterol; diet; NHANES III

INTRODUCTION

Long-term exposure to ambient particulate matter (PM) air pollution is associated with increased cardiovascular disease (CVD) mortality[1–4], representing a serious public health problem estimated to result in >3,000,000 deaths worldwide in 2010[5]. Although PM is now recognized as causally associated with CVD mortality by the American Heart Association[6], there remains uncertainty regarding the impact of PM on common preclinical serologic and manometric indicators of CVD risk. Hypertension and hypercholesterolemia are known clinical CVD risk factors, and non-fasting triglycerides have been linked to increased risk of CVD even after controlling for HDL cholesterol levels[7,8]. However, there is very limited information on whether PM exposure affects these risk factors in humans. Prior studies based on small numbers of human subjects reported inconsistent results[9–13].

There is substantial evidence that the adverse CVD effects of PM air pollution are related to oxidative stress and a subsequent systemic inflammatory response[6,14]. Systemic inflammation has been linked to altered lipid metabolism[15] and high-density lipoprotein (HDL) cholesterol function[16], yet epidemiologic studies of air pollution and lipid levels have been limited and inconsistent[17]. Most previous studies of the PM-CVD association have focused on increases in incident or fatal CVD in older adults[1–4]. In this study, our objective was to examine the link between long-term PM exposure and clinical CVD risk factors in a relatively young and healthy population.

In a well-established, large U.S. survey with detailed data on blood markers, ambient air pollution, and diet and lifestyle factors, we examined whether long-term exposure to particulate matter <10μm in aerodynamic diameter (PM_{10}) is associated with recognized CVD risk factors including triglycerides, cholesterol, and blood pressure. Better understanding how PM contributes to CVD can help in the development of ambient air quality standards that adequately protect public health by mitigating susceptibility for future cardiovascular events.

METHODS

Study Population

The Third National Health and Nutrition Examination Survey (NHANES III) is a large survey, conducted between 1988 and 1994 by the U.S. Centers for Disease Control and Prevention (CDC) to examine the health and nutritional status of the non-institutionalized U.S. population two months of age or over[18]. The survey is unique in that it combines interviews and physical examinations for participants across the U.S. who were selected to be representative of the U.S. population with regard to age, race/ethnicity, and region. NHANES III used a stratified, multi-stage probability design, which has been described elsewhere[18]. Briefly, the survey sample was designed to be self-weighting within primary

sampling units for age, sex, and race/ethnicity. A sample of 89 primary sampling units (mostly individual counties; small adjacent counties were combined in a few cases) were selected with probability proportional to size, and were randomly divided into two phases; 44 locations were sampled during 1988–1991, and 45 locations were sampled during 1991–1994. Individuals were selected in each location to provide approximately self-weighting samples for age, sex, and race-ethnicity.

The NHANES III interview included detailed questions on demographic, socioeconomic, diet, and health-related conditions of study participants. The examination component consisted of medical and physiologic measurements and an extensive battery of laboratory tests. Of the 18,162 adult men and women who underwent the NHANES III interview and examination, participants with missing air pollution exposure estimates (n=5,266) and unavailable or unreliable CVD marker measurements (n=3,677) were excluded. The analytic data set included 11,623 participants (some participants were in more than one exclusion category). This study was approved by the NYU School of Medicine Institutional Review Boards and the CDC.

Air Pollution Exposure Assessment

We accessed air pollution exposure estimates for NHANES III participants, developed by Kravets and Parker at the Centers for Disease Control and Prevention (CDC)[19] and accessible through the CDC Research Data Center (http://www.cdc.gov/rdc/). PM_{10} was measured in 24-hour average sampling intervals, through the U.S. Environmental Protection Agency (EPA) Air Quality System (AQS) monitoring network. Monitor-specific annual average PM_{10} exposures were developed, based on 24-hour averages recorded during 1988–1994, with exclusion from monitors with <75% of the possible 24-hour observations. For monitoring sites with multiple monitors, the monitor with the highest number of 24-hour observations was used. PM_{10} exposures were estimated at the centroid of resident census blocks of NHANES III respondents, for census block groups where the centroid was within 20 miles of a measurement site, providing coverage for 73% of all NHANES III participants, and 71% of adult NHANES III participants who underwent both an interview and examination[19]. PM_{10} exposures were calculated by averaging all annual averages from monitors within 20 miles of the NHANES III respondent's block group centroid, weighted by the inverse of the squared distance between the centroid and the monitoring sites. Exposure estimates for years preceding the study were unavailable, but these average PM_{10} values for the years during which NHANES III was conducted are assumed to be representative of long-term PM_{10} exposures for the study participants.

Kravets and Parker previously showed that NHANES III participants for whom these long-term PM_{10} exposure estimates were available did not systematically differ by age, poverty status, or health status from the overall NHANES III population, but more were non-white, from the Northeastern or Western U.S., and from an urbanized county[19]. A comparison of those NHANES III participants included in our study (i.e. for whom PM_{10} exposure estimates, outcome, and covariate data were available) to those excluded from our study is presented in eTable 1.

Outcome Variables

NHANES III examination and laboratory procedures have been described in detail[20]. Blood samples were obtained by trained and certified phlebotomists, at the NHANES Mobile Examination Center, and the specimens were stored and analyzed according to detailed protocols. Serum levels (mg/dL) of non-fasting triglycerides, total cholesterol, and HDL cholesterol were measured with an Hitachi 704 Analyzer (Boehringer Mannheim Diagnostics, Indianapolis, IN). Serum low-density lipoprotein (LDL) cholesterol (mg/dL) was calculated based on the Friedewald equation[21] among a subset of fasted participants (n=5,038; 40%) in accordance with recommended protocol[22]. We excluded values >99.9% of population distribution (n=3 for >1,450 mg/dL triglycerides; n=1 for >600 mg/dL total cholesterol) to reduce the influence of these extreme values on our findings (however, results were robust to inclusion of these measurements; data not shown). Measurements of triglycerides, total cholesterol, LDL cholesterol, and HDL cholesterol were missing for 705 (6%), 708 (6%), 7,533 (65%), and 760 (7%) subjects in the analytic data set, respectively.

Multiple measurements (mm Hg) of systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken to account for variability, recorded using a mercury sphygmomanometer (W. A. Baum Co., Inc., Copiague, NY) to the nearest 2 mm Hg as recommended by national and international guidelines[23,24]. Up to three sets of measurements were taken during the physical examination by the NHANES III examining physician, and up to three sets were taken during the household interview by interview staff. The average of all available measurements was then calculated. Physicians and interview staff received initial blood pressure measurement training, quarterly recertification, and annual retraining. Pulse pressure was calculated as the difference between SBP and DBP. Mean levels of each lipid and blood pressure outcome variable are given within categories of major demographic characteristics in eTable 2.

Statistical Analysis

We evaluated associations of PM_{10} with clinical CVD risk factors (triglycerides, total cholesterol, LDL cholesterol, HDL cholesterol, SBP, DBP, and pulse pressure) using generalized linear regression, subsequent to log-transformation of outcome variables to improve linearity. Associations were expressed as percent elevation in clinical CVD risk factors per interquartile range width (IQR_w) increase in PM_{10} exposure (11.1 $\mu g/m^3$). Adjusted mean levels (least squares means) of clinical CVD risk factors were also reported, with respect to quartiles of PM_{10} (categorical analysis).

To account for the complex multistage probability sampling design of NHANES III, we included appropriate weights, strata, and clusters in all statistical models[18]. All models were controlled for age (continuous). Additional control, one variable at a time, for sex, race (white, black, or other/unknown), smoking status (current, former, or never), BMI (15–18.4, 18.5–24.9, 25–29.9, 30–34.9, or 35–60 kg/m^2, or unknown), and U.S. census region (Northeast, Midwest, South, or West) resulted in changes of >10% in risk estimates; these variables were included in multivariate risk models. Household poverty-income ratio was also included in fully-adjusted models to reduce risk of confounding by socioeconomic status. Further adjustment for educational attainment (<12 years, 12 years, >12 years,

unknown), marital status (currently married, not currently married, unknown), alcohol consumption (ever drinker, never drinker, unknown), smoking pack-years (continuous), and urbanization (based on the U.S. Department of Agriculture's Rural/Urban dichotomous coding for each participant's county of residence) did not change the risk estimates and were not included in multivariate risk models. We confirmed that regression assumptions (linearity of the relationships, and normality and homoscedasticity of the errors) were satisfied by residual plots. In sub-analyses, we defined specific hyperlipidemias (triglycerides ≥200 mg/dL, total cholesterol ≥240 mg/dL, LDL cholesterol ≥160 mg/dL, and low HDL as <40 mg/dL for males and <50 mg/dL for females) and hypertension (SBP ≥140 or DBP ≥90 mm Hg) based on recommendations from the American Heart Association[25], and we estimated odds ratios of hyperlipidemia or hypertension in relation to PM_{10} using multivariate logistic regression. Prevalence was >10% for all risk conditions, therefore odds ratios were not assumed to approximate risk ratios.

We performed several sensitivity analyses to determine whether exposure misclassification may have affected our findings. Analyses were repeated excluding participants who reported having lived in their city/town/area for less than one year (7% of subjects). We also repeated our analyses using inverse distance weighted averages of PM_{10} data from monitors within 10 miles (rather than 20 miles) of each participant's census block group centroid. Moreover, multilevel modeling was evaluated as an alternate approach to handling the potential lack of statistical independence among participants living in close proximity. In multilevel models, survey locations were treated as random effects, and models were adjusted for NHANES III sampling weights.

In secondary analyses, we evaluated whether the relationships found to be significant in the main analysis were consistent across major demographic categories by performing stratified analyses for age (above or below 50 years), sex, race-ethnicity (non-Hispanic white, non-Hispanic black, or Mexican-American), and U.S. census region (Northeast, Midwest, South, or West). To avoid collinearity, our stratified analysis for region excluded NHANES III weights, strata, and clusters.

All analyses were performed using SAS version 9.2 (SAS Institute, Cary, IN) and SAS-callable SUDAAN version 11.0.1 (Research Triangle Institute, Research Triangle Park, NC).

RESULTS

Among the 11,623 participants in our study, 72% were non-Hispanic white and 52% were female; median age was 39.0 (interquartile range (IQR) = 28, 61). Over 90% of study participants lived in their current city/area for at least one year. Individual estimated PM_{10} exposures ranged between 15.3 and 83.4 μg/m^3 (median = 29.4, IQR = 11.1 25.2–36.3 μg/m^3). Participants exposed to higher concentrations of PM_{10} were more likely to be younger, male, and less likely to be morbidly obese, compared to those exposed to lower PM_{10}; proportions of race/ethnicity and marital status categories also differed by quartile of PM_{10} exposure (Table 1). Participants exposed to higher concentrations of PM_{10} tended to live in urban areas.

Greater PM_{10} exposure was associated with greater levels of triglycerides. In an age-only adjusted model, an IQR increase in PM_{10} was associated with 2.02% greater levels of triglycerides (Table 2). Controlling for age, sex, race, BMI, smoking status, U.S. region, and poverty-income ratio in stepwise fashion did not diminish the effect size for the estimated association between PM_{10} and triglyceride levels (see Supplemental Material, eFigure 1). In a multivariate adjusted model, an IQR_w increase in PM_{10} was associated with 2.42% (95% confidence interval (CI) = 1.09–3.76) greater triglycerides; multivariate adjusted means of triglycerides according to increasing quartiles of PM_{10} were 137.6, 142.5, 142.6, and 148.9 mg/dL, respectively.

An IQR_w increase in PM_{10} was associated with 1.43% (95% CI = 1.21–1.66) greater levels of circulating total cholesterol in a multivariate model; adjusted means of total cholesterols according to increasing quartiles of PM_{10} were 198.7, 198.3, 200.6, and 203.5 mg/dL, respectively (Table 2). Likewise, long-term exposure to PM_{10} was associated with increased odds of hypercholesterolemia (odds ratio = 1.15 and 95% CI = 1.08–1.22; per IQR_w of PM_{10}; data not shown). An IQR increase in PM_{10} was also associated with 1.18% elevated LDL cholesterol (95% CI = 0.81–1.56; Table 2). HDL cholesterol was not associated with PM_{10}. For SBP, DBP, and PP, the linear regression effect estimates per IQR of PM_{10} were small, and no significant trend in least square means was found across quartiles of PM_{10} for these outcomes (Table 2).

Triglycerides, total cholesterol, and LDL cholesterol were similarly elevated per IQR_w of PM_{10} in the sensitivity analyses we performed. Our findings for triglycerides, total cholesterol, and LDL cholesterol were unchanged when we excluded participants with estimated PM_{10} exposures above 65 $\mu g/m^3$ (i.e. > 98^{th} percentile), or when we limited to participants who lived at least one year at current address (data not shown). Further, these lipids were similarly elevated per IQR_w of PM_{10} when using exposure estimates based on PM_{10} data from monitors within 10 miles (rather than 20 miles) of the participant's U.S. census block group centroid, or when using mixed effects modeling.

In secondary analyses, we evaluated whether the associations we found for PM_{10} with triglycerides and total cholesterol varied by demographic characteristics. We chose to focus on these two outcomes because they were significantly associated with PM_{10} in our main analyses; LDL cholesterol was excluded because it was strongly correlated with total cholesterol (r = 0.92) (eTable 3) and measured in less than half as many participants. The associations of PM_{10} with triglyceride and total cholesterol did not differ by age or census region (Table 3). Associations of PM_{10} with triglyceride and total cholesterol were stronger among males (p-values for interaction < 0.05), but were still present among females (Table 3). PM_{10} was associated with total cholesterol among all race-ethnicity categories.

DISCUSSION

In a large, nationwide survey population, we found that greater particulate air pollution was associated with greater levels of serum triglycerides and total cholesterol. These relationships did not differ by U.S. geographic region or age. Findings from this large diverse study indicate that greater long-term PM_{10} exposure is associated with elevated

serum triglycerides and total cholesterol, potentially mediating air pollution-related effects on CVD.

Previous studies of pathophysiological CVD effects of PM exposure have been largely based on animal studies[15,16,26] and small human studies[9,13,17], mostly with acute PM exposure[6]. This is the largest study to date of long-term PM exposure and CVD risk factors. Evidence to support an association between acute PM exposure and elevated triglycerides has been found among asthmatics[17], but our results provide new evidence for an association between chronic PM exposure and elevated triglycerides in a general population. The results of the present study are roughly similar per unit increase in PM_{10} to those of a recent Taiwanese study, which found about a 4% increase in total cholesterol associated per 10 $\mu g/m^3$ increase in annual average PM_{10}[3]. Our study provides valuable information to support PM's influence on these lipids, which has implications for prevention of CVD events.

Our findings suggest that the relationship of PM to clinical CVD risk factors is evident in early to middle adulthood. Previously, epidemiologic studies of air pollution and CVD risk factors have been limited to controlled or occupational exposures among small numbers of subjects[17,27,28]. The present findings of a positive association with PM is important because this is the first evidence that PM is associated with elevated levels of circulating lipids in both males and females, even in non-elder adults.

Inhaled PM has been shown to be strongly associated with inflammation and oxidative stress. Pulmonary inflammation induced by PM_{10} may trigger a systemic inflammatory response, and ultrafine PM constituents or co-pollutants may cross into the blood stream and directly cause systemic inflammation[6,14]. Induction of systemic inflammation can lead to altered lipid metabolism[16,26,29], reduced anti-inflammatory capacity of and cholesterol transport by HDL[16,29], and lipid oxidation[15,30,31], all of which can accelerate atherosclerosis. Furthermore, interleukin-6, an important inflammation mediator, has been linked to reduced lipoprotein lipase activity[32] and increased hepatic secretion of triglycerides[33]. These mechanisms may explain the association we found between long-term PM_{10} exposure and elevated circulating lipids.

There are several limitations of this study. First, this cross-sectional study precludes our ability to discern temporality of the associations of PM exposure with circulating triglycerides and total cholesterol, although it is unlikely that blood lipid levels influenced individual PM exposure. Further, our exposure assessment provided fairly accurate estimation of long-term exposure, given that several studies have found high correlations in PM levels over time within U.S. cities/regions[34,35].

Second, exposure estimation based on an inverse-distance weighted average of PM monitoring data is susceptible to exposure misclassification. However, a recent simulation study comparing exposure estimates based on nearby monitors to kriging yielded consistent health effect estimates in scenarios assuming greater spatial dependence, suggesting the robustness of both assessment methods[36]. Our PM_{10} exposure levels were similar to other studies based on NHANES III (using different exposure estimation)[37,38], as well as long-term average PM_{10} exposures estimated for the American Cancer Society Cohort for an

overlapping time period (1982–1998)[34]. Further, our findings were consistent when restricting our exposure assessment to data from monitors within 10 miles of participants' block group centroid (presumably greater spatial dependence than within 20 miles), therefore our findings are unlikely explained by misclassification.

Third, as with all air pollution health effect analyses, the possibility exists that PM_{10} is correlated with other air pollutants or traffic-related factors. Exposure estimates for other PM size fractions or specific PM constituents are not available for NHANES III participants, and we had insufficient power to include NO_2 exposure estimates in our analyses. Finally, although we carefully controlled for possible confounders, taking advantage of well measured demographic and lifestyle factors, findings could have been affected by unmeasured or residual confounding factors. Our results were robust to further adjustment for education, marital status, alcohol consumption, smoking pack-years, and urbanization.

Strengths of this study include representativeness in NHANES III of the overall non-institutionalized U.S. population with regard to age, poverty status, and health status, strengthening the generalizability of our findings. Our large sample size enabled us to examine CVD risk factors according to narrow PM categories across major demographic categories with ample statistical power. Finally, this study made use of validated estimates of long-term PM_{10} concentrations at participants' census block group.

The findings from this large and diverse study population suggest that greater PM_{10} air pollution is associated, in a dose-response fashion, with greater levels of circulating triglycerides and total cholesterol, which are major cardiovascular disease risk factors. Further, we have shown that these associations did not differ by age or region. If causal, our results have important potential implications for preclinical cardiovascular disease burden reduction through early air quality improvement interventions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Selected characteristics of the study population by quartile of annual average PM_{10} exposure in the Third National Health and Nutrition Examination Survey (NHANES III).

n=11,623	Quartile of PM_{10} exposure ($\mu g/m^3$)			
	15.3–25.2	25.2–29.4	29.4–36.3	36.3–83.4
Age – %				
17–29 years	27	25	27	28
30–44 years	36	31	36	37
45–64 years	21	27	27	22
65–90 years	16	17	11	13
Sex – %				
Female	52	54	53	49
Race – %				
Non-Hispanic white	80	69	70	68
non-Hispanic black	8	17	15	10
Mexican-American	5	5	3	10
Other	7	10	12	12
Smoking status – %				
Current	25	25	27	30
Former	26	24	24	26
Never	49	51	49	44
BMI (kg/m^2) – %				
Underweight (<18·5)	2	3	4	2
Normal weight (18·5–24·9)	42	45	46	46
Overweight (25·0–9·9)	33	32	29	32
Obesity (30·0–34·9)	14	15	14	14
Morbid obesity (> 35·0)	9	6	8	6
Unknown	<1	<1	<1	<1
Educational attainment,%				
< 12 years	22	26	23	24
12 years	33	31	30	31
13–14 years	16	16	19	18
15–17 years	28	27	28	26
Unknown	<1	<1	1	1
Alcohol,%				
Never drinker	11	11	13	11
Marital status, %				
Currently married	66	57	62	62
Socioeconomic status, %				
Below poverty threshold	12	15	11	13
Region, %				
Northeast	31	29	20	12

n=11,623	Quartile of PM_{10} exposure ($\mu g/m^3$)			
	15.3–25.2	25.2–29.4	29.4–36.3	36.3–83.4
Midwest	19	12	23	28
South	33	42	31	6
West	17	17	25	54
In urban/rural county, %				
Urban	42	73	69	77
Duration in city/area, %				
< 1 year (or unknown)	6	8	7	6
Triglycerides, mg/dL	135.3 ± 95.8	136.3 ± 96.9	134.9 ± 106.3	144.1 ± 118.3
Total cholesterol, mg/dL	201.0 ± 42.5	201.1 ± 41.2	200.0 ± 45.1	202.2 ± 43.8
LDL cholesterol, mg/dL	125.5 ± 38.8	125.1 ± 36.5	123.8 ± 37.3	128.2 ± 37.5
HDL cholesterol, mg/dL	50.7 ± 15.5	51.5 ± 14.8	50.9 ± 15.4	51.1 ± 15.3
Systolic blood pressure, mm Hg	120.7 ± 17.2	122.0 ± 18.4	120.3 ± 16.6	120.6 ± 16.4
Diastolic blood pressure, mm Hg	74.0 ± 10.0	72.9 ± 10.1	74.1 ± 10.2	73.2 ± 9.8
Pulse pressure, mm Hg	46.7 ± 14.9	49.1 ± 15.0	46.2 ± 13.5	47.4 ± 14.2
Portion of study population with high risk levels of outcome variables[a] – %				
Hypertriglyceridemia	16	15	14	18
Hypercholesterolemia	18	17	17	17
High LDL cholesterol	19	16	14	16
Low HDL cholesterol	38	33	39	39
Hypertension	17	17	15	15

Abbreviations: PM_{10} = particulate matter <10μm in aerodynamic diameter; BMI = body mass index; CRP = C-reactive protein; LDL = low density lipoprotein; HDL = high density lipoprotein.

[a]High risk levels of outcome variables were defined according to American Heart Association guidelines; hypertriglyceridemia was defined as triglycerides ≥ 200 mg/dL, hypercholesterolemia was defined as total cholesterol ≥ 240 mg/dL, high LDL cholesterol was defined as LDL cholesterol ≥ 160 mg/dL, low HDL cholesterol was defined as HDL cholesterol < 40 mg/dL (males) or < 50 mg/dL (females), and hypertension was defined as systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg.

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Table 2

Long-term exposure to PM_{10} and cardiovascular disease risk factors in the Third National Health and Nutrition Examination Survey (NHANES III)

		% difference per IQR_w of PM_{10} (95% CI)[a]	least squares mean per quartile of PM_{10} (95% CI)[b]				p for trend across PM_{10} quartiles
			15.3–25.3 μg/m^3	25.3–29.4 μg/m^3	29.4–36.3 μg/m^3	36.3–83.4 μg/m^3	
Triglycerides (mg/dL)	n		2,803	2,719	2,724	2,693	
	age adjusted[c]	2.02 (0.72, 3.35)	138.1 (132.5, 143.6)	137.5 (135.5, 139.6)	139.2 (136.3, 142.2)	148.1 (144.9, 151.4)	<0.001
	fully adjusted[cd]	2.42 (1.09, 3.76)	137.6 (132.3, 143.0)	142.5 (138.5, 146.5)	142.6 (140.8, 144.4)	148.9 (145.3, 152.4)	0.002
Total cholesterol (mg/dL)	n		2,804	2,719	2,725	2,694	
	age adjusted[c]	0.83 (0.51, 1.16)	203.4 (201.0, 205.8)	202.2 (200.9, 203.5)	203.7 (202.5, 204.8)	205.6 (203.5, 207.6)	0.074
	fully adjusted[cd]	1.43 (1.21, 1.66)	198.7 (196.4, 201.0)	198.3 (196.5, 200.0)	200.6 (199.1, 202.0)	203.5 (200.8, 206.1)	<0.001
Low density lipoprotein (LDL) cholesterol (mg/dL)	n		1,179	1,156	1,094	1,096	
	age adjusted[c]	0.52 (−0.18, 1.23)	127.4 (123.9, 130.9)	125.5 (124.1, 126.9)	126.2 (124.8, 127.6)	129.9 (128.1, 131.7)	0.276
	fully adjusted[cd]	1.18 (0.81, 1.56)	119.5 (113.5, 125.5)	118.8 (113.6, 123.9)	119.7 (113.9, 125.4)	124.8 (118.6, 130.9)	0.002
High density lipoprotein cholesterol (mg/dL)	n		2,791	2,711	2,708	2,679	
	age adjusted[c]	0.90 (0.53, 1.27)	50.7 (50.1, 51.4)	51.5 (50.9, 52.1)	51.0 (50.8, 51.2)	51.2 (50.9, 51.5)	0.339
	fully adjusted[cd]	0.18 (−0.32, 0.68)	50.3 (49.0, 51.7)	50.2 (48.6, 51.7)	49.7 (48.4, 51.1)	49.7 (48.0, 51.3)	0.021
Systolic blood pressure (mmHg)	n		2,826	2,831	2,828	2,868	
	age adjusted[c]	0.16 (−0.01, 0.34)	122.1 (121.6, 122.5)	122.5 (122.0, 123.1)	122.4 (122.1, 122.6)	122.5 (121.8, 123.1)	0.306
	fully adjusted[cd]	0.22 (0.03, 0.41)	125.0 (124.2, 125.8)	125.5 (124.4, 126.6)	125.4 (124.5, 126.3)	125.6 (125.0, 126.1)	0.163
Diastolic blood pressure (mmHg)	n		2,826	2,831	2,828	2,864	
	age adjusted[c]	−0.24 (−0.47,−0.02)	74.3 (74.1, 74.6)	73.0 (72.3, 73.8)	74.6 (74.1, 75.0)	73.7 (73.5, 73.8)	0.287

		% difference per IQR_w of PM_{10} (95% CI)[a]	least squares mean per quartile of PM_{10} (95% CI)[b]				p for trend across PM_{10} quartiles
			15.3–25.3 $\mu g/m^3$	25.3–29.4 $\mu g/m^3$	29.4–36.3 $\mu g/m^3$	36.3–83.4 $\mu g/m^3$	
Pulse pressure (mmHg)	fully adjusted[cd]	−0.18 (−0.31, −0.05)	77.2 (76.3, 78.1)	76.0 (75.4, 76.6)	77.3 (76.3, 78.3)	76.8 (76.0, 77.5)	0.717
	n		2,826	2,831	2,828	2,864	
	age adjusted[c]	0.77 (0.35, 1.20)	47.7 (47.3, 48.2)	49.5 (49.1, 49.9)	47.8 (47.4, 48.2)	48.8 (48.2, 49.4)	0.127
	fully adjusted[cd]	0.79 (0.14, 1.44)	47.8 (46.2, 49.4)	49.5 (48.1, 51.0)	48.1 (46.3, 49.9)	48.8 (47.7, 50.0)	0.249

Abbreviations: PM_{10} = particulate matter <10μm in aerodynamic diameter; IQR_W = interquartile range width.

[a] Generalized linear model regression was used to estimate the association between an IQR increase in PM_{10} as the independent variable and the percent difference in each clinical cardiovascular disease risk factor as the dependent variable; IQR of PM_{10} = 11.1 $\mu g/m^3$.

[b] Least squares means were estimated using generalized linear model regression for each clinical cardiovascular disease risk factor as the dependent variable, within each quartile of PM_{10} (independent variable), adjusted for NHANES III sampling weights, strata, and clusters, as well as age and other covariates as indicated in the second column.

[c] Model adjusted for age and NHANES III sampling weights, strata, and clusters.

[d] Model additionally adjusted for age, sex, race (white, black, other, or unknown), BMI (15–18.4, 18.5–24.9, 25–29.9, 30–34.9, 35–60 kg/m^2, or unknown), smoking status (current, former, never, or unknown), U.S. region (Northeast, Midwest, South, or West), and poverty-income ratio.

Table 3

Percent difference and 95% confidence interval (CI) for triglycerides and total cholesterol associated with an interquartile range increase in PM_{10}, stratified by demographic factorsin the Third National Health and Nutrition Examination Survey (NHANES III).

	n	triglycerides[1]	p for interaction[2]	n	total cholesterol[1]	p for interaction[2]
Age						
17–49 years	6,820	1.34 (0.03, 2.67)	0.23	6,821	1.09 (0.90, 1.29)	0.36
50–90 years	4,119	4.25 (2.49, 6.04)		4,121	1.51 (1.03, 1.99)	
U.S. Census Region[3]						
Northeast	1,847	0.83 (−4.31, 6.26)	0.81	1,847	1.28 (−0.73, 3.33)	0.57
Midwest	2,027	2.01 (−1.59, 5.76)		2,027	0.78 (−0.63, 2.21)	
South	3,697	−0.45 (−4.71, 3.99)		3,698	1.32 (−0.37, 3.05)	
West	3,368	1.58 (0.16, 3.02)		3,370	1.63 (1.04, 2.23)	
Sex						
Males	5,102	2.67 (1.36, 3.99)	0.04	5,104	2.07 (1.87, 2.27)	<0.01
Females	5,837	2.25 (0.73, 3.79)		5,838	0.81 (0.38, 1.25)	
Race-ethnicity						
Non-Hispanic white	4,065	2.44 (0.87,4.04)	0.17	4,066	1.49 (1.17, 1.80)	<0.01
Non-Hispanic black	3,175	−0.35 (−1.29, 0.60)		3,175	1.19 (0.83, 1.55)	
Mexican-American	3,108	0.65 (0.58, 0.71)		3,110	1.04 (1.02, 1.06)	

Abbreviations: PM_{10} = particulate matter <10μm in aerodynamic diameter.

[1]Generalized linear model regressionwas used to estimate the association between an interquartile range (IQR) of PM_{10} (11.1 $\mu g/m^3$) as the independent variable and the percent difference in lipid levels as the dependent variable, adjusting for NHANES III sampling weights, strata, and clusters, as well as age, sex, race (white, black, other, or unknown), body mass index (15–18.4, 18.5–24.9, 25–29.9, 30–34.9, 35–60 kg/m^2, or unknown), smoking status (current, former, never, or unknown), U.S. region (Northeast, Midwest, South, or West), and poverty-income ratio.

[2]P-value for interaction derived from F-test for the interaction term between PM_{10} and each demographic, health, or lifestyle factor.

[3]Stratified and interaction models for census region excluded NHANESIII sampling weights, clusters, and strata to avoid collinearity.