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Long-term exposure to ambient air pollution and renal function in African Americans: the Jackson Heart Study

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Abstract

Renal dysfunction is prevalent in the US among African Americans. Air pollution is associated with renal dysfunction in mostly white American populations, but not among African Americans. We evaluated cross-sectional associations between 1-year and 3-year fine particulate matter ($PM_{2.5}$) and ozone (O_3) concentrations and renal function among 5090 African American participants in the Jackson Heart Study. We used mixed-effect linear regression to estimate associations between 1-year and 3-year $PM_{2.5}$ and O_3 and estimated glomerular filtration rate (eGFR), urine albumin/creatinine ratio (UACR), serum creatinine, and serum cystatin C, adjusting for: sociodemographic factors, health behaviors, and medical history and accounting for clustering by census tract. At baseline, JHS participants had mean age 55.4 years, and 63.8% were female; mean 1-year and 3-year $PM_{2.5}$ concentrations were 12.2 and 12.4 μg/m^3, and mean 1-year and 3-year O_3 concentrations were 40.2 and 40.7 ppb, respectively. Approximately 6.5% of participants had reduced eGFR (<60 mL/min/1.73m^2) and 12.7% had elevated UACR (>30 μg/g), both indicating impaired renal function. Annual and 3-year O_3 concentrations were inversely associated with eGFR and positively associated with serum creatinine; annual and 3year $PM_{2.5}$

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Conflicts of interest

The authors declare that they have no conflicts of interest.

Disclaimers

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concentrations were inversely associated with UACR. We observed impaired renal function associated with increased O_3 but not $PM_{2.5}$ exposure among African Americans.

Introduction

Chronic kidney disease (CKD) is increasingly prevalent, affecting approximately 11% of men and 15% of women in the US (1). CKD is identified using indicators of poor renal function, including low glomerular filtration rate (GFR) and/or high urine albumin/creatinine ratio (UACR) (2). In addition to higher risk of morbidity and mortality linked to CKD itself, CKD is associated with increased risk of cardiovascular disease (CVD) and all-cause mortality (3–5).

Epidemiologic studies have linked long-term exposure to traffic-related air pollution, including coarse and fine particulate matter (PM_{10} and $PM_{2.5}$, respectively) and ozone (O_3) (6–10) with excess CVD morbidity and mortality. Kidneys are highly vascular organs and interact greatly with the cardiovascular system, including blood pressure regulation (11, 12). CKD and poor renal function have been identified as independent risk factors for development of CVD (arterial vascular disease and cardiomyopathy) (3) as well as being associated with major CVD risk factors such as diabetes (1, 13) and hypertension (1, 11), which are especially prevalent among African Americans (14–17). Given that kidney function is closely related to blood pressure control, vascular function, and risk of CVD, it is plausible that long-term exposure to ambient air pollution may be associated with renal function.

Prior studies of long-term exposure to traffic-related air pollution and renal function have had mixed results. In our previous study in a cohort of older, predominantly white stroke survivors in Boston, living near a major roadway was associated with lower estimated glomerular filtration rate (eGFR) (12). Similarly, Mehta et al. observed an inverse association between ambient $PM_{2.5}$ and eGFR, as well as annual decline in eGFR over time an eleven-year follow-up period among older, predominantly white men in the Boston area (18). Bowe et al. observed associations between $PM_{2.5}$ concentrations and incident CKD and reduced eGFR(19). However, among participants in the Multi-Ethnic Study of Atherosclerosis (MESA), O'Neill et al. did not find evidence of an association between PM_{10} concentrations and UACR (20). These prior studies included small numbers of African Americans. However, CKD is especially concerning for African Americans, who have been shown to have greater risk of incident CKD and higher prevalence of end-stage renal disease compared to whites (21). The very limited availability of data, particularly among African Americans, highlights the need for additional studies evaluating the association between long-term exposure to traffic pollution and measures of renal function especially among African Americans. This study addresses this gap in the literature by evaluating the association between $PM_{2.5}$ and O_3, two common indicators of traffic-related air pollution, and renal function in the Jackson Heart Study (JHS), the largest African American cohort of cardiovascular diseases.

Methods

The JHS is a longitudinal cohort study of 5301 non-institutionalized African-American men and women aged 21 years and older, recruited 2000–2004 residing in the tri-county Jackson, Mississippi Metropolitan Statistical Area (MSA) (22, 23). Although JHS consisted of three visits, we used cross-sectional data from the first visit only, due to loss to follow-up at visit 2 and lack of exposure data at visit 3. Upon enrollment, participants completed an in-person interview, detailing demographics, health history, lifestyle factors, and healthcare access. Shortly thereafter, participants were scheduled for a clinical exam and asked to bring in all medications that they used in the past two weeks, which were inventoried (23). Details of the home induction interview and baseline clinical exam are described elsewhere (22, 23). All participants provided written informed consent. All JHS protocols were reviewed and approved by the Institutional Review Boards at Jackson State University, Tougaloo College, and the University of Mississippi Medical Center. This analysis was approved by the Institutional Review Board at Indiana University.

Exposure assessment

Our primary exposures of interest were mean annual and 3-year $PM_{2.5}$ and O_3 concentrations prior to participants' visits at their zip codes of residence. Relevant exposure windows are unknown, but annual means are common measures of long-term air pollution exposure (24–26) (daily or monthly averages are typically used as measures of short-term exposure), and we additionally used 3-year means to explore the possibility of longer relevant exposure windows. We obtained geocoded address information from all JHS participants. We used publicly available data from the US Environmental Protection Agency's Community Multiscale Air Quality Model (CMAQ) in order to calculate 1-year and 3-year $PM_{2.5}$ and O_3 concentrations. CMAQ uses emissions data as well as hourly monitoring data to estimate pollutant concentrations and applies a spatiotemporal downscaler model (27) to estimate 1-year and 3-year mean $PM_{2.5}$ and O_3 concentrations at the zip code level. Briefly, downscaler models use linear regression with spatially-varying coefficients on CMAQ data, then model them as correlated spatial Gaussian process via the method of coregionalization (28, 29).

Indicators of renal function

We examined the following indicators for renal function at the baseline exam as outcomes of interest: eGFR, urine albumin/creatinine ratio (UACR), serum creatinine, and serum cystatin C. UACR is an indicator of albuminuria, or abnormally high concentrations of the protein albumin in urine (2, 20). Serum creatinine and cystatin C are both measures of kidney filtration functions, as kidneys contribute to the regulation of circulating levels of creatinine and cystatin C, although serum creatinine concentration may be affected by diet, muscle mass, or illness (2, 30). Both measures are used to estimate glomerular filtration rate, the rate at which kidneys filter blood. We measured eGFR, using the CKD-EPI creatinine/cystatin C equation:

$$eGFR = 135 * min(Scr/\kappa, 1)^{\alpha} * max(Scr/\kappa, 1)^{-0.601} * min(Scys/0.8, 1)^{-0.375} * max(Scys/0.8, 1)^{-0.711} * 0.995^{Age} * 0.969(if\ female) * 1.08$$

where Scr represents serum creatinine, κ is 0.7 for women and 0.9 for men, α is −0.329 for women and −0.411 for men, min and max denote the minimum or maximum of specified measurement or 1, and Scys denotes serum cystatin C (30). Urine albumin was measured using human albumin kit on a Dade Behring BN II nephelometer (Dade Behring, Newark, DE) (31). Serum and urine creatinine were measured using a multipoint enzymatic spectrophotometric assay on a Vitros 950 Ortho-Clinical Diagnostics analyzer (Raritan, NJ) (31).

At the baseline clinical exam, fasting blood samples were procured. Participants recruited from 2000–2002 were asked to collect a 24-hour urine sample for analysis, but only 586 out of 1914 participants agreed to provide one. To ease participant burden and increase compliance, spot urine samples were collected during baseline examinations beginning in 2002, although 24hour samples were still collected for those willing to provide one (31). After this change in protocol, 2576 out of 3176 participants provided urine samples. In our analyses, we used measurements from baseline spot urine samples if available (n = 2434); if spot urine samples were unavailable, we used measurements from baseline 24-hour urine samples (n = 726).

Covariates

We examined the variables described below as potential covariates. Sociodemographic variables included self-reported age (years) and sex (male or female). Education was categorized as the highest level of education completed (less than high school, high school/GED, college degree/certificate, or graduate/professional school). Medical insurance access was defined as any current medical insurance policy (public and/or private). Occupation was categorized based on Sims et al (32) and the distribution in our sample as management/professional, service, sales, or other. Household income level (low, lower-middle, upper-middle, and high) was determined based on self-reported income, family size, number of children <18 years old, and the US Census designated poverty level for the year of data collection (33). Neighborhood socioeconomic status (NSES) was determined at the census tract level, as described by Dubowitz et al (34); NSES was then converted to a z score, as described by Diez Roux et al (35). Anthropometrics, health behavior, and health history included measured body mass index (BMI, kg/m^2), calculated from measured weight (kg) divided by measured height (m) squared, self-reported smoking status (never, former, current) and alcohol consumption in the past 12 months (yes, no). Nutritional status and physical activity were categorized as poor, intermediate, or ideal according to Life's Simple 7 criteria (36, 37). Participants were classified as having hyperlipidemia if total cholesterol was ≥ 240 mg/dl or low-density lipoprotein cholesterol level was ≥ 160 mg/dl or they were taking lipid-lowering medications (38). We additionally examined the use of medications that may affect renal function: non-steroidal anti-inflammatory drugs (NSAIDs), diuretic medications, and statin medications, as recorded from self-report and medication inventory.

It is possible that diabetes and hypertension may be confounders or mediators in the association between traffic-related pollution and renal function. Participants were classified as having diabetes if they used any antidiabetic medications (self-report or medication inventory), measured hemoglobin A1c levels were ≥ 6.5%, or fasting glucose measurement was ≥ 126 mg/dl (39). Participants were classified as hypertensive if their supine blood pressure at the baseline clinical examination was ≥140/90 mmHg or they used blood pressure lowering medication; blood pressure medication use was defined as self-report of using medication for blood pressure or the following medications identified during medication inventory: beta blockers, calcium blockers, antihypertensives, or diuretics (40, 41).

Statistical analyses

We excluded participants whose address could not be geocoded at the street level due to inaccurate or incomplete address data. We used mixed-effect linear regression models to estimate the cross-sectional associations between 1-year and 3-year $PM_{2.5}$ and O_3 with estimated eGFR, UACR, serum creatinine, and cystatin C, individually accounting for clustering at the census tract level. Due to its extreme left skew, we used natural log-transformed UACR values in all models. We presented five statistical models: Model 1: unadjusted but accounting for clustering by census tract; Model 2: adjusted for all covariates that were associated with exposure and outcome at the $p = 0.05$ level by bivariate ANOVA or chi-square tests—age, sex, BMI, education level, NSES z-score, medical insurance, smoking status, physical activity, alcohol consumption, occupation, and hyperlipidemia, accounting for clustering by census tract; Model 3: adjusted for all covariates included in model 2 plus use non-steroidal anti-inflammatory drugs, diuretic medication, and statin medications, accounting for clustering by census tract; Model 4: adjusted for all covariates included in model 2 plus diabetes and hypertension, accounting for clustering by census tract; and Model 5: adjusted for all covariates included in model 2 plus the other pollutant (adjustment for O_3 in $PM_{2.5}$ models and for $PM_{2.5}$ in O3 models).

Sensitivity analyses

We conducted several sensitivity analyses. First, as many people were missing urine samples, we compared descriptive characteristics, renal function indicators derived from blood samples, and air pollution indicators between those missing and not missing urine samples. Second, we dichotomized indicators of renal function and used logistic regression to estimate associations with $PM_{2.5}$ and O_3. Specifically, we dichotomized UACR as ≤30 mg/g and >30 mg/g, and eGFR as <60 mL/min/1.73m^2 and ≥60 mL/min/1.73m^2, according to clinical cutpoints (2). We dichotomized serum creatinine and cystatin C at the median. Third, we modeled exposure using distance to major roadways as an indicator of air pollution exposure. We used residential distance to U.S. Census Feature Class Code A1 (primary highway with limited access) or A2 (primary road without limited access) roadways as a proxy for long-term exposure to traffic-related pollution (42–44). Fourth, we modeled exposure using distance to A1 roads only. We used ArcGIS (version 9.2; ESRI Inc., Redlands, CA) to geocode participants' addresses and calculate the Euclidian distance from each residence to the nearest major roadway. For all analyses, we categorized residential DTR using the following cutpoints: <150 m, 150–299 m, 300–999 m, and ≥1000 m, as well

as a log-transformed continuous measure (45–47). Last, we calculated the Pearson correlations between $PM_{2.5}$ and O_3 concentrations.

Code availability

Data were analyzed using SAS version 9.4 (SAS Institute, Cary, NC, USA). Analytic code is available upon request from the corresponding author.

Results

We included 5090 JHS participants, excluding 211 (4%) whose street address could not be geocoded to street level. Those with poor (<60 mL/min/1.73m^2) eGFR were, on average,older (67.9 years), less likely to be female (53.1%), had poorer SES indicators, but had better nutritional status (51.2% poor, 46.6% intermediate, 2.2% ideal), and were less likely to consume alcohol (24.3%) (Table 1). Those with poor eGFR lived in areas with higher mean 1-year and 3year $PM_{2.5}$ concentrations (12.3 μg/m^3 and 12.5 μg/m^3, respectively) but similar ozone concentrations compared to those with normal eGFR. Table 2 shows descriptive characteristics of indicators of renal function. Mean eGFR was normal at 92.9 mL/min/1.73m^2; The majority (93.4%) of participants had normal (≥90 mL/min/1.73m^2) eGFR and 6.5% had abnormal (<60 mL/min/1.73m^2) eGFR. Median UACR was normal at 6.0 mg/g and most (87.3%) of participants had normal (≤30 mg/g) UACR. Mean serum creatinine was approximately 1.0 mg/dL, and cystatin C was approximately 0.8 mg/L.

Adjusting for covariates in model 2, we observed inverse associations between UACR and 1-year (−0.09, 95% CI −0.2, −0.02) and 3-year $PM_{2.5}$ (−0.2, 95% CI −0.3, −0.06) concentrations (Table 3). After additional adjustment for NSAIDs, diuretic and statin medications, we observed inverse associations between eGFR and 1-year (−0.3, 95% CI −0.5, −0.01) and 3-year (−0.3, 95% CI −0.6, −0.04) ozone concentrations and positive associations between serum creatinine and 1year (0.005, 95% CI 0.0006, 0.01) and 3-year (0.005, 95% CI 0.0005, 0.01) ozone concentrations. Models 2, 3, 4, and 5 produced similar results, with varying levels of statistical significance.

In sensitivity analysis, compared to those who were not missing urine samples, those missing urine samples (n = 1930) were, on average, older, had poorer socioeconomic indicators (education, household income, neighborhood socioeconomic status, lower proportion in management or professional occupations), and were more likely to be former or current smokers and more likely to have diabetes (Table S1). Those missing urine samples had lower eGFR, higher serum creatinine, and higher cystatin C levels; they had higher 1-year and 3-year $PM_{2.5}$ concentrations and 1-year O_3 concentrations but lower 3-year O_3 concentrations compared to those who were not missing urine samples.

In our second sensitivity analysis, 1-year $PM_{2.5}$ concentration was inversely associated with high (> 0.9 mg/dL) serum creatinine (OR 0.86, 95% CI 0.75, 0.98) in model 2, with similar results for models 3 and 3 (Table S2). Third, those who lived 150–299 m from an A1 or A2 road had, on average, 3.2 mL/min/1.73m^2 (95% CI 1.2, 5.2 mL/min/1.72m^2) higher eGFR, 0.03 (95% CI −0.4, −0.1) lower log UACR (−0.03, 95% CI −0.4, −0.1), 0.06 mg/dL (95% CI −0.09, −0.03 mg/dL) lower serum creatinine concentration, and 0.05 mg/L (95% CI −0.08,

−0.03 mg/L) lower serum cystatin C compared to those living ≥1000 m from an A1 or A2 road (Table S3). Results were similar when examining distance to A1 roads only (Table S2). Annual and 3-year $PM_{2.5}$ were highly correlated (r = 0.86), as were annual and 3-year O_3 (r = 0.94). However, measures of $PM_{2.5}$ were only weakly correlated with annual O_3 concentration (r=0.12 for annual, 0.044 for 3year $PM_{2.5}$ concentration) and weakly inversely correlated with 3-year O_3 concentrations (r = 0.069 for annual, −0.033 for 3-year $PM_{2.5}$ concentration) (Table S5). We examined whether our sample size was adequate based on R-squared for full (0.31) and unadjusted (0.007) models with power of 90% of eGFR using SAS proc power; our sample size was adequate.

Discussion

This study examined the associations between 1-year and 3-year $PM_{2.5}$ and O_3 concentrations and renal function among African Americans. We observed inverse associations between 1-year and 3-year O_3 concentrations and eGFR and positive associations with serum creatinine, indicating poorer renal function with increased O_3 exposure. We observed inverse associations between 1-year and 3-year $PM_{2.5}$ concentrations and UACR, indicating better renal function with increased $PM_{2.5}$ exposure. We did not observe associations between either pollutant and serum cystatin C.

We observed inverse associations between 1-year and 3-year O_3 concentrations and eGFR. We are not aware of any other epidemiologic study that examined the effect of long-term ambient O_3 concentration on renal function. However, previous studies have shown inverse associations between other traffic-related pollutants and eGFR. Mehta et al. observed an inverse association between eGFR and annual $PM_{2.5}$ (−0.6, 95% CI −0.79, −0.40 mL/min/1.73m^2 per 2.1 μg/m^3 increase in $PM_{2.5}$) among older men living in the Boston, Massachusetts area (18). In our previous study, we observed that those who lived within 50 m from a major roadway had 3.9 mL/min/1.73 m^2 lower eGFR (95% CI 1.0, 6.7; p=0.007) compared to those living >1000 m from a major roadway in the Boston area (12). In our current study, in sensitivity analyses, we also observed that those who live nearest roads (<150 m) have lower (−1.4, 95% CI −4.7, 2.0 mL/min/1.73 m^2) eGFR, although this association was not statistically significant. The Jackson area is less densely populated compared to the Boston area and accordingly JHS did not have any participants living <50 m from a major roadway, making direct comparison to our previous study impossible. It is possible that these effects are strongest among those who live very near to major roads. Bowe et al. observed positive associations between annual $PM_{2.5}$ concentrations and incident eGFR <60 mL/min/1.73m^2 (HR 1.25, 95% CI 1.17, 1.34) in a national, predominately white older male cohort (19). Although we did not have follow-up data and could not estimate incidence, sensitivity analyses showed evidence of a possible positive association between 3-year (but not 1-year) $PM_{2.5}$ concentration and prevalent eGFR <60 mL/min/1.73m^2 that was not statistically significant (OR 1.14, 95% CI 0.91, 1.42). Notably, previous studies were conducted in very different populations compared to our present study: predominantly older, white men; the present study is in African American adults of all ages including young adults. Our present results add to the current literature, as this is the first such study conducted among an African American community in a mixed urban/rural

area in the American South, examining multiple indicators of renal function, and examining effects of O_3 exposure.

We observed inverse associations between 1-year and 3-year $PM_{2.5}$ concentrations and log UACR, indicating that those with higher exposure to $PM_{2.5}$ had better renal function. O'Neill et al. observed no associations between ambient particulate matter concentrations and UACR among a multi-ethnic population across 6 US centers (20). One important limitation of our study is that we were unable to calculate UACR for many (37.9%) of participants who were missing urine samples. Although 24-hour urine collection is standard for calculation of UACR, due to participant burden, we were unable to obtain this measure for most of our participants. In sensitivity analysis, those missing urine samples appeared to have generally poorer socioeconomic and health indicators, including serum-based measures of renal function, as well as higher $PM_{2.5}$ exposures. These participants would likely have had relatively high UACR, were we able to measure it. Those with better renal function may have been more likely to provide urine samples, potentially biasing our results. If all participants had provided a urine sample, it is possible that we would have observed null or even positive associations between $PM_{2.5}$ exposure and UACR; we believe our observed inverse associations may be due to bias.

Our results indicating poorer renal function with increased O_3 exposure supports previous research showing similar associations with other traffic-related pollutants (19) and research showing associations between overall and cardiovascular mortality with increased O_3 exposure (10, 12, 18). Different results when studying $PM_{2.5}$ exposure compared to O_3 exposure is not surprising given weak and even slightly inverse correlations between $PM_{2.5}$ and O_3 metrics. This study is representative of the mixed urban/rural African American population living near Jackson, Mississippi (48). However, this study may not be generalizable to African Americans living outside of the Jackson, Mississippi area.

In conclusion, we observed associations between O_3 exposure, an important air pollutant, and reduced renal function in an African American population. However, we did not observe such associations with $PM_{2.5}$. This study adds to the limited literature on the associations between air pollution and renal function among African Americans, although more studies are needed.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1.

Descriptive characteristics of participants in Jackson Heart Study by eGFR status

	eGFR <60 mL/min/1.73m^2 (n = 322)	eGFR ≥60 mL/min/1.73m^2 (n = 4604)	Total (N = 5090)
Characteristic	**Mean (SD) or %**	**Mean (SD) or %**	**Mean (SD) or %**
Age, years, mean (SD) *	67.9 (9.6)	54.5 (12.5)	55.4 (12.8)
Female *	171 (53.1)	2948 (64.0)	3245 (63.8)
BMI (kg/m^2), mean (SD)	32.0 (6.8)	31.7 (7.3)	31.7 (7.2)
Highest level of education completed *			
Less than high school	136 (42.6)	841 (18.3)	1023 (20.2)
High school/GED	103 (32.3)	1710 (37.3)	1866 (36.8)
College degree/certificate	40 (12.5)	1273 (27.7)	1352 (26.7)
Graduate/professional school	40 (12.5)	767 (16.7)	832 (16.4)
Household income status *			
Low	63 (23.7)	575 (14.7)	670 (15.5)
Lower-middle	84 (31.6)	930 (23.8)	1049 (24.3)
Upper-middle	67 (25.2)	1180 (30.1)	1283 (29.8)
High	52 (19.6)	1230 (31.4)	1310 (30.4)
Neighborhood SES z-score, mean (SD)[1] *	−1.42 (4.88)	−0.22 (5.01)	−0.29 (5.01)
Medical Insurance Access *	302 (94.4)	3952 (86.2)	4394 (86.7)
Smoking status *			
Never	197 (61.8)	3117 (68.3)	3436 (68.1)
Former	90 (28.2)	832 (18.2)	942 (18.7)
Current	32 (10.0)	616 (13.5)	667 (13.2)
Physical activity[2] *			
Poor	203 (63.2)	2224 (48.3)	2515 (49.5)
Intermediate	81 (25.2)	1471 (32.0)	1601 (31.5)
Ideal	37 (11.5)	907 (19.7)	970 (19.1)
Nutritional status *	165 (51.2)	2811 (61.1)	3082 (60.6)
Poor			
Intermediate	150 (46.6)	1752 (38.1)	1958 (38.5)
Ideal	7 (2.2)	41 (0.9)	50 (1.0)
Alcohol consumption, past 12 months *	78 (24.3)	2177 (47.5)	2318 (45.8)
Occupation *			

	eGFR <60 mL/min/1.73m^2 (n = 322)	eGFR ≥60 mL/min/1.73m^2 (n = 4604)	Total (N = 5090)
Characteristic	**Mean (SD) or %**	**Mean (SD) or %**	**Mean (SD) or %**
Management/professional	83 (25.9)	1674 (36.4)	1811 (35.6)
Service	98 (30.5)	1134 (24.6)	1277 (25.1)
Sales	40 (12.5)	820 (17.8)	890 (17.5)
Other	100 (31.2)	974 (21.2)	1108 (21.8)
Hypertension *	293 (91.0)	2669 (58.0)	3053 (60.0)
Diabetes *	145 (45.0)	928 (20.2)	1103 (21.9)
Hyperlipidemia *	136 (42.5)	1338 (29.1)	1505 (29.7)
$PM_{2.5}$ 1-year mean (μg/m^3) *	12.3 (0.6)	12.2 (0.6)	12.2 (0.6)
$PM_{2.5}$ 3-year mean (μg/m^3) *	12.5 (0.5)	12.4 (0.5)	12.4 (0.5)
O_3 1-year mean (ppb)	40.3 (3.0)	40.2 (2.7)	40.2 (2.7)
O_3 3-year mean (ppb)	40.6 (2.9)	40.7 (2.6)	40.7 (2.6)

Percent missing: BMI 0.2, Education level 0.3, Household income status 15.3, medical insurance access 0.4, smoking 0.9, physical activity 0.08, alcohol consumption 0.6, occupation 0.08, hypertension 0.08, diabetes 1.2, hyperlipidemia 0.3

* $p<0.05$

Table 2.

Markers of renal function of participants in Jackson Heart (N = 5090)

	Mean (SD) or n (%)
eGFR	
Mean (SD), mL/min/1.73m^2	92.9 (21.8)
Abnormal <60 mL/min/1.73m^2	322 (6.5)
Normal ≥60 mL/min/1.73m^2	4604 (93.4)
Urine Albumin/Creatinine ratio[1]	
Median (IQR), mg/g	6.0 (9.0)
Normal ≤30 mg/g	2759 (87.3)
Abnormal >30 mg/g	401 (12.7)
Serum Creatinine	
Mean (SD), mg/dL	1.00 (0.6)
Cystatin C	
Mean (SD), mg/L	0.76 (0.38)

Percent missing: eGFR 3.2, Urine Albumin/Creatinine Ratio 37.9, serum creatinine 1.7, serum cystatin C 2.8

[1] Calculated from spot urine samples of 2434 participants and 24-hour urine samples of 726 participants (37.9% missing urine)

Table 3.

Results from linear regression of $PM_{2.5}$ and O_3 and markers of renal function among Jackson Heart Study participants (N = 5090).

	Pollutant			
	1-year $PM_{2.5}$	**3-year $PM_{2.5}$**	**1-year O_3**	**3-year O_3**
	Beta (95% CI)	**Beta (95% CI)**	**Beta (95% CI)**	**Beta (95% CI)**
eGFR				
Model 1[a]	−3.0 (−4.5, −1.6)*	−2.9 (−5.8, −2.0)*	−0.1 (−0.3, 0.1)	0.2 (−0.08, 0.4)
Model 2[b]	0.7 (−0.2, 1.6)	0.8 (−0.4, 2.0) 0.3	−0.2 (−0.4, 0.01)	−0.2 (−0.4, −0.02)*
Model 3[c]	0.3 (−0.8, 1.3)	(−0.9, 1.6)	−0.3 (−0.5, −0.01)* −0.2	−0.3 (−0.6, −0.04)*
Model 4[d]	0.6 (−0.3, 1.5)	0.8 (−0.4, 1.9)	(−0.4, 0.03)	−0.2 (−0.4, −0.004)*
Model 5[e]	0.8 (−0.08, 1.7)	0.8 (−0.4, 2.0)	−0.2 (−0.4, −0.01)*	−0.2 (−0.4, −0.02)*
Log of urine albumin/creatinine ratio				
Model 1[a]	−0.0008 (−0.08, 0.08)	−0.007 (−0.1, 0.1)	0.005 (−0.02, 0.03)	−0.003 (−0.03, 0.02)
Model 2[b]	−0.09 (−0.2, −0.02)*	−0.2 (−0.3, −0.06)*	−0.009 (−0.03, 0.01)	−0.006 (−0.03, 0.02)
Model 3[c]	−0.09 (−0.2, 0.009)	−0.1 (−0.3, 0.004)	−0.0004 (−0.03, 0.03)	0.01 (−0.02, 0.04)
Model 4[d]	−0.1 (−0.2, −0.03)*	−0.2 (−0.3, −0.09)* −0.2	−0.008 (−0.03, 0.01)	−0.005 (−0.03, 0.02)
Model 5[e]	−0.09 (−0.2, −0.02)*	(−0.3, −0.06)*	0.004 (−0.02, 0.03)	0.0008 (−0.02, 0.03)
Serum Creatinine				
Model 1[a]	0.002 (−0.01, 0.02)	−0.01 (−0.03, 0.01)	0.003 (−0.002, 0.008)	0.002 (−0.003, 0.006)
Model 2[b]	−0.009 (−0.03, 0.007)	−0.02 (−0.04, 0.001)	0.004 (−0.0005, 0.008)	0.004 (−0.0001, 0.007)
Model 3[c]	0.009 (−0.02, 0.04)	0.008 (−0.02, 0.04)	0.005 (0.0006, 0.01)*	0.005 (0.0005, 0.01)*
Model 4[d]	−0.008 (−0.02, 0.008)	−0.02 (−0.04, 0.002)	0.004 (−0.0008, 0.008)	0.003 (−0.0004, 0.007)
Model 5[e]	−0.01 (−0.03, 0.004)	−0.02 (−0.04, 0002)	0.004 (−0.0002, 0.009)	0.004 (−0.0002, 0.007)
Cystatin C				
Model 1[a]	0.02 (0.005, 0.04)*	0.02 (0.005, 0.05)*	0.002 (−0.002, 0.005)	−0.001 (−0.004, 0.002)
Model 2[b]	−0.008 (−0.02, .005)	−0.01 (−0.03, 0.005)	0.002 (−0.002, 0.005)	0.002 (−0.001, 0.004)
Model 3[c]	0.004 (−0.01, 0.02)	0.009 (−0.01, 0.03)	0.002 (−0.0006, 0.005)	0.003 (−0.0007, 0.006)
Model 4[d]	−0.007 (−0.02, 0.006)	−0.01 (−0.03, 0.006)	0.001 (−0.002, 0.004)	0.001 (−0.002, 0.004)
Model 5[e]	−0.009 (−0.02, 0.004)	−0.01 (−0.03, 0.005)	0.002 (−0.001, 0.005)	0.002 (−0.001, 0.004)

[a]Model 1 unadjusted, accounting for clustering on census tract

[b] Model 2 adjusted for age, sex, BMI, education level, NSES z-score, medical insurance, smoking status, physical activity, alcohol consumption, occupation, and hyperlipidemia, accounting for clustering by census tract

[c] Model 3 adjusted for all covariates in model 2, plus use of non-steroidal anti-inflammatory drugs, diuretic medication, and statin medications, accounting for clustering by census tract

[d] Model 4 adjusted for all covariates in model 2, plus diabetes and hypertension, accounting for clustering by census tract

[e] Model 5 adjusted for all covariates in model 2, plus the other pollutant, O_3 in $PM_{2.5}$ models, and $PM_{2.5}$ in O_3 models, accounting for clustering by census tract

[*] $p<0.05$