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Poverty, Race, and CKD in a Racially and Socioeconomically Diverse Urban Population

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Abstract

Background—Low socioeconomic status (SES) and African American race are both independently associated with end-stage renal disease and progressive chronic kidney disease (CKD), however, despite their frequent co-occurrence, the effect of low SES independent of race has not been well-studied in CKD.

Study Design—Cross-sectional study.

Setting & Participants—2,375 community-dwelling adults age 30–64 years residing within 12 neighborhoods selected for both socioeconomic and racial diversity in Baltimore City, Maryland.

Predictors—Low SES [self-reported household income <125% of 2004 Department of Health and Human Services guideline], higher SES (≥125% of guideline); white and African American race.

Outcomes & Measurements—CKD defined as estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m². Logistic regression used to calculate odds ratios (OR) for relationship between poverty and CKD, stratified by race.

Results—Of 2,375 participants; 955 were white (347 low SES and 608 higher SES); 1,420 were African American (713 low SES and 707 higher SES). A total of 146 (6.2%) participants had CKD. Overall, race was not associated with CKD [OR, 1.05; 95% confidence interval (CI), 0.57–1.96]; however, African Americans had a much greater odds of advanced CKD (eGFR <30 mL/min/1.73 m²). Low SES was independently associated with 59% greater odds of CKD after adjustment for demographics, insurance status and comorbid disease (OR, 1.59; 95% CI, 1.27–1.99). However, when stratified by race, low SES was associated with CKD in African Americans (OR, 1.91; 95% CI, 1.54–2.38), but not in whites (OR, 0.95; 95% CI, 0.58–1.55; P for interaction, 0.003).

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N section: Because the Editor-in-Chief recused himself from consideration of this manuscript, the Deputy Editor (Daniel E. Weiner, MD, MS) served as Acting Editor-in-Chief. Details of the journal's procedures for potential editor conflicts are given in the Editorial Policies section of the AJKD website.

Limitations—Cross-sectional design; findings may not be generalizable to non-urban populations.

Conclusions—Low SES has a profound relationship with CKD in African Americans but not in whites in an urban population of adults, and its role in the racial disparities seen in CKD is worthy of further investigation.

Keywords

Socioeconomic status; health disparities; epidemiology; renal disease

INTRODUCTION

An association between low socioeconomic status (SES) and chronic kidney disease (CKD) has been established both in the U.S. and worldwide^{1, 2}. Low SES has also been associated with important precursors and risk factors for CKD, including micro- and macroalbuminuria³, diabetes⁴ and hypertension⁵, and with an increased risk of progressive CKD in specific populations, including older adults⁶ and white men⁷. There is also extensive literature documenting a relationship between low SES and ESRD in multiple clinical populations⁸⁻¹¹.

Racial and ethnic disparities are profound in CKD^{12, 13}. While African American race is not associated with an increased prevalence of less severe stages of CKD¹⁴, African Americans have up to four times greater risk of ESRD compared to whites¹⁵. Additionally, the annual incidence of ESRD among African Americans exceeds 1000 per million persons in several cities, including Baltimore¹⁵. This disparity is believed to be due in part to socioeconomic factors. However, studies of this topic have yielded inconsistent findings. One population-based study found that socioeconomic factors accounted for 11% of the excess risk of kidney disease seen among African Americans¹⁶; and some other studies have drawn similar conclusions^{17, 18}. Furthermore, a study examining racial disparities in diabetes-related kidney function decline found that SES accounted for 52% of this disparity¹⁹. And recently, in a retrospective analysis of individuals who had initiated dialysis in the southeastern U.S., it was shown that increasing neighborhood-level poverty is associated with greater racial disparity in ESRD incidence.²⁰ However, other researchers have reported insignificant contributions of SES to the racial disparities seen in CKD^{8, 11, 21, 22}. The inconsistency may arise, in part, from studies that are not specifically designed to disentangle the effects of SES and race.

The contribution of SES in explaining racial disparities in CKD thus remains unclear. Therefore, the objectives of our study were to determine whether the prevalence of CKD differs by individual-level SES or race in an urban population of African American and white adults sampled across a wide range of socioeconomic circumstances, and whether the relationship between SES and CKD prevalence varies by race.

METHODS

Study Design and Population

We examined cross-sectional data from the National Institute on Aging (NIA) Healthy Aging in Neighborhoods of Diversity Across the Lifespan (HANDLS) Study. HANDLS is a population-based cohort study of the influences and interaction of race and SES on the development of cardiovascular and cerebrovascular health disparities among minority and lower SES subgroups. Participants are community-dwelling African Americans and whites age 30-64 years at enrollment, drawn from 12 neighborhoods, each of which is composed of contiguous U.S. census tracts in Baltimore City that reflect socioeconomic and racial diversity. Participants were sampled representatively using a factorial cross of four factors (age, sex, race

and SES) with approximately equal numbers of participants per “cell”. Individuals who identified with neither African American nor white race were excluded from the study. Household enrollment was from August 2004 to November 2008. Response rates among eligible individuals varied by neighborhood, and were between 42.9 and 79.6%, similar to that of other population-based studies of African Americans²³. Each participant provided informed consent. The MedStar Research Institute Institutional Review Board approved the study protocol.

The total HANDLS study population is 3,720, and to date, 70% of participants have completed the initial examination. For the purposes of this study, we limited our sample to the 2,375 participants with serum creatinine measures. These participants were slightly older (48.3 years versus 46.7 years), more likely to be female (49% versus 43%), more likely to be of low SES (45% versus 41%), and less likely to be employed (55% versus 59%) than those without creatinine data ($P < 0.05$ for all); but both groups were of similar race.

Measurements

The independent variables of interest were SES and race. Poverty was chosen as the measure of SES in this cohort to allow ease of selection of a representative sample. SES was defined as low SES or higher SES based on whether a participant reported annual household income below or above 125% of the 2004 Department of Health and Human Services poverty guideline²⁴. This cut-point for low SES was selected by a panel of experts, and has been used in initiatives such as the National School Lunch Program²⁵. Low SES status was determined at the doorstep during household enrollment based on several screening questions including “how many people are in your household?” and “is your family income above or below this cut-off?” Race was self-reported (African American or white) during the initial household survey. Individuals identifying themselves as multi-ethnic were included in the racial group with which they most strongly identified. Additional demographic data including, age, sex, marital status, number of household members, health insurance status, occupational and educational history were also assessed during an initial household survey.

A mobile research vehicle was the site of health care provider ascertained medical history, substance use history and physical examination. Additionally, health care utilization was assessed on the mobile research vehicle. Fasting venous blood specimen and spot urine samples were collected on the mobile research vehicle and analyzed at the NIA Clinical Research Branch Core Laboratory and Quest Diagnostics, Inc. (www.questdiagnostics.com).

The presence of relevant comorbid diseases was ascertained via medical history, physical examination and laboratory assessment. Each participant underwent sitting and standing blood pressure measurements on each arm using the brachial artery auscultation method with an inflatable cuff of appropriate size²⁶. Hypertension was defined as an average of seated and standing systolic blood pressure ≥ 140 mmHg, an average of seated and standing diastolic blood pressure ≥ 90 mmHg²⁶, a history of blood pressure medication use, and/or a self-report of hypertension. Diabetes mellitus was defined as a fasting plasma glucose concentration of ≥ 126 mg/dl (7.0 mmol/l)²⁷, or self-report of diabetes. Cardiovascular disease was defined as a self-reported history of congestive heart failure, enlarged heart, angina, myocardial infarction, coronary artery disease, transient ischemic attack and/or stroke. Anthropometric measures were performed, including height and weight, and were used to calculate body mass index (BMI) to determine the presence of obesity (defined as a BMI ≥ 30 kg/m²). Tobacco use was defined as a report of at least 100 cigarettes smoked in the participant's lifetime. Excess alcohol use was defined as a report of either the participant or a family member thinking in the past six months that the participant drank too much alcohol. History of regular use of cocaine or heroin was defined as a report of using these drugs regularly in the past six months or greater than six months ago.

The dependent variable was CKD, and was determined using single laboratory measures of serum creatinine (n=2,375) and urine albumin (n=1,472) concentrations. Serum creatinine was measured for 236 participants at the NIA Clinical Research Branch Core Laboratory using a modified kinetic Jaffe method (CREA method, Dimension Xpand Clinical Chemistry System, Siemens Healthcare Diagnostics Inc., www.medical.siemens.com); and was measured for the remainder of participants (n=2,139) at Quest Diagnostics, Inc. by isotope dilution mass spectrometry (IDMS) (Olympus America Inc., www.olympusamerica.com) and standardized to the reference laboratory at the Cleveland Clinic. Urine albumin concentration was measured at Quest Diagnostics, Inc. using an immunoturbimetric assay (Kamiya Biomedical Co., www.kamiyabiomedical.com).

CKD was primarily defined as the presence of estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m² [by 4-variable IDMS traceable Modification of Diet in Renal Disease (MDRD) Study equation²⁸]. In a sensitivity analysis, CKD was defined as an eGFR <60 mL/min/1.73 m² or urine albumin ≥ 30 mg/g creatinine. CKD Stages were defined by National Kidney Foundation guidelines²⁹.

Statistical Analysis

Participant characteristics stratified by SES and race were compared using Fisher's exact tests for categorical variables and *t* tests for continuous variables. Descriptive statistics and Fisher's exact tests were used to compare the unadjusted prevalence of CKD by SES and race. Multivariable logistic regression was performed to determine the presence, direction, magnitude, and independence of the association between low SES and prevalent CKD. Stratified analyses by race were performed and an interaction between race and SES was considered in overall regression models.

Potential confounders were chosen based on variables that have been shown to be associated with CKD in published literature and by examining the relationships between each variable and SES and between the variable and CKD (*P*<0.20 for each association). Based on these criteria, confounders included in the multivariable models were age, race, sex, marital status, high school education, tobacco use, illicit drug use, hypertension, diabetes, cardiovascular disease, and obesity. Variables that were collinear with CKD (such as serum uric acid) were excluded from the models. Model-wise deletion was used to handle missing data in the models.

Several sensitivity analyses were performed to test our findings. First, CKD was redefined as the presence of an eGFR <60 mL/min/1.73 m² or albuminuria as noted above, by including available measures of urine albumin:creatinine ratio (n=1,472). Second, the relationship between CKD and poverty was re-examined using eGFR <45 mL/min/1.73 m² as the definition of CKD as was used in a prior study of CKD and SES². Third, annual household income was categorized into 24 strata (in \$5,000 increments) and examined for its relationship with CKD among those participants who reported their income specifically (n=1,812), beyond just stating whether or not they fell above or below the poverty threshold for their family size. Fourth, we examined educational and employment status as alternative measures of SES to our dichotomized poverty index. Fifth, given that only those participants who had laboratory measures performed at Quest Diagnostics, Inc. underwent IDMS-traceable serum creatinine measures, analyses restricted to only these participants were performed. Finally, given that a number of covariates were not complete for all participants, multiple imputation was performed to examine the impact of missing data on our primary analysis.

In all analyses, the possibility of confounding by neighborhood was controlled with fixed-effects modeling³⁰. A two-sided *P* < 0.05 was used as the level of significance for all tests. Statistical analyses were performed using Stata, version 10 (StataCorp, www.stata.com).

RESULTS

Participant Characteristics by SES and Race

Of 2,375 participants, 955 were white and 1,420 were African American (Table 1). Low SES was present for 347 (36%) whites and 713 (50%) African Americans. Participants living in poverty (low SES) were less likely to be insured, employed or have completed a high school education when compared to those with higher SES. Also, those with low SES were more likely to report tobacco, cocaine and/or heroin use than the higher SES individuals. Serum albumin was lower among the low SES participants, and the low SES group was more likely to have reported an emergency room visit in the preceding year and the absence of a regular health care provider, when compared to the higher SES participants. The aforementioned findings were present and statistically significant across both racial groups.

A number of important differences in comorbid disease status between African American and white participants were present (Table 1). Hypertension was more prevalent among low SES whites than higher SES whites, but among African Americans there was no difference by SES. Diabetes was more prevalent among higher SES African Americans than low SES African Americans (although not statistically significant), but the converse was true for whites, with low SES whites having the greatest burden of diabetes. Similar findings were noted for obesity.

CKD Prevalence by SES and Race

A total of 146 (6.2%) participants had CKD (eGFR <60 mL/min/1.73 m²), including 89 (6.3%) African American and 57 (6.0%) white participants. In univariate analysis, race was not significantly associated with CKD overall [Odds Ratio (OR) for CKD comparing African Americans to whites in univariate analysis = 1.05, 95% CI, 0.57-1.96] however, when stages of CKD were examined, African Americans were much more likely than whites to have advanced CKD, defined as CKD stages 4 and 5, or eGFR <30 mL/min/1.73 m², (OR, 5.04; 95% CI, 1.21-21.01). Univariate analysis of CKD prevalence by SES suggested a 27% greater prevalence of CKD among those of low SES (7.3%) as opposed to higher SES (5.3%), although this only reached borderline statistical significance (Figure 1). However, with racial stratification, low SES was associated with a greater prevalence of CKD among African Americans, but not among whites.

Logistic regression models of the relationship between low SES and CKD revealed that low SES was nearly statistically significantly associated with CKD in the univariate model (Table 2), however, in multivariable models low SES was independently associated with CKD (OR = 1.59, 95% CI, 1.27-1.99 after adjustment for demographics, education, insurance status and comorbid disease). Notably, when stratified by race in multivariable analyses, low SES was associated with CKD in African Americans (OR= 1.91, 95% CI, 1.54-2.38), but not in whites (OR= 0.95, 95% CI, .58-1.55); *P* interaction= 0.003. Further adjustment for tobacco and illicit drug use yielded similar results, however the OR was slightly attenuated for African Americans.

Sensitivity Analyses

CKD defined as an eGFR <60 mL/min/1.73 m² or the presence of albuminuria classified 175 (7.4%) participants as having CKD. This definition of CKD yielded similar results to our primary definition, with a CKD OR for those with low SES versus with higher SES of 1.51 (95% CI, 1.16-1.97), after adjustment for demographics, education, insurance status and comorbid disease. Differential findings by race were also noted in a similar model, with low SES having a strong association with CKD among African Americans (OR=1.88, 95% CI, 1.61-2.19) but not among whites (OR=0.82, 95% CI, 0.48-1.38), with a *P* interaction of <0.001. CKD defined as eGFR <45 mL/min/1.73 m² classified 51 (2.1%) participants as having CKD.

This definition also showed large effect sizes; OR for CKD by poverty status= 2.14, 95% CI, 1.52-2.99 after adjustment for demographics, education, insurance and comorbidities. While there was a trend towards a greater relationship between poverty and CKD among African Americans, the relationship between low SES and CKD did not statistically significantly vary by race (*P* interaction 0.3).

Income status categorized into 24 strata for the 1,812 participants who reported this data revealed that the mean income category was generally higher for low-SES whites than for low-SES African Americans (12.6 versus 10.9; corresponding to annual incomes of \$11,000 to \$12,000 and \$9,000 to \$10,000, respectively; *P* 0.001). Additionally, no statistically significant association between income category and CKD overall was observed, however, stratification by race revealed such a relationship among African Americans, but not among whites; although the *P* interaction did not reach statistical significance (data not shown).

Lack of employment was associated with CKD in African Americans and whites, but with a stronger association among African Americans (*P* interaction <0.001). Years of education were negatively associated with CKD in African Americans, but not in whites, although the *P* interaction was not significant at 0.4.

Analyses restricted to only those participants who underwent IDMS-traceable serum creatinine measurements yielded similar inferences to the total cohort (data not shown). A total of 558 participants were missing data on at least 1 covariate (55% were African American). Comparing African Americans with missing data to whites, 64% versus 27% were of low SES. Multiple imputation of missing covariates yielded similar results to our primary analysis, with an CKD OR for low SES versus higher SES of 1.45 (95% CI 1.12-1.86), in our final model. Additionally, this effect was seen only among African Americans (OR 1.75, 95% CI 1.38-2.22) and not among whites (OR .88, 95% CI .56-1.39), with a *P* for interaction 0.006.

Discussion

In a racially and socioeconomically diverse urban population of adults, we observed that individual-level poverty (low SES) was associated with prevalent CKD among African Americans, but not among whites. Among African Americans, low SES was independently associated with a nearly 2-fold greater risk of CKD when compared to higher SES. Further, we found that poverty had no statistically significant relationship with CKD among whites; however there was a trend towards a negative association. Our major finding was robust to adjustment for several risk factors for CKD and to the use of stricter eGFR cutoffs. We found that, in general, further adjustment for potential confounders strengthened the association between poverty and CKD; however this was in part due to negative confounding (for example, both age and health insurance were positively associated with CKD but were negatively associated with poverty).

Few studies have reported on the relationship between SES and pre-ESRD CKD, and among these, variable associations with race have been reported. Shoham et al. found in the Atherosclerosis Risk in Communities (ARIC) Study that working class membership in the life-course was more strongly associated with CKD among African Americans than among whites, even independent of hypertension and diabetes². However, in an earlier report from the same study population, Merkin et al. noted that living in a low SES area was associated with progressive CKD only among white men⁷. In studies examining SES, race and CKD in participants with specific diseases, significant contributions of SES to racial disparities in CKD have been observed among persons with diabetic¹⁹ and hypertensive³¹ kidney disease.

There are several reasons why the results of our study may differ in some cases from previous reports in the literature. First, although more balanced than many studies of racial and SES

differences, our study did include more African Americans than whites, and in general, whites in our study were of higher income than African Americans. This may have contributed to the differential findings by race that we observed. Additionally, the sole use of the poverty threshold as our measure of SES may not have been appropriate for whites in our study. We did, for example, find that lack of employment was associated with CKD among both African Americans and whites.

The major implication of our study is that poverty may impact African Americans differently than whites in the development of CKD. Poverty may exert its differential effect on African Americans via several mechanisms. Plausible biological mechanisms include the increased prevalence of low birth weight observed among African Americans, a condition associated with poverty. Low birth weight is a risk factor for ESRD, and is thought to be a contributor to the racial disparities seen in ESRD³². Also, the gene encoding non-muscle myosin heavy chain type II isoform A (*MYH9*) has been associated with non-diabetic ESRD in African Americans, but not in whites³³. Poverty and its consequences (i.e. toxic environmental exposures such as heavy metals) may play a role in the probable gene-environment interactions that lead to ESRD in these individuals. It has also been reported that living in low socioeconomic status neighborhoods is more strongly associated with greater cumulative biological risk profiles (defined using nine indicators of increased risk) among African Americans than among whites.³⁴ Many of these indicators, such as blood pressure and waist-to-hip ratio, are also associated with increased prevalence of CKD.

Poverty may also differentially impact health beliefs and behaviors among African Americans as compared to whites, which could lead to increased risk of CKD and its progression. A recent report from the Americans' Changing Lives Survey, for example, found a positive association between number of unhealthy behaviors and number of chronic conditions among African Americans but not among Whites. They postulated that these behaviors may serve as a coping mechanism for those living in chronically stressful environments.³⁵ Also, life stressors commonly encountered in poverty, such as unemployment and discrimination, may impact African Americans differently than whites. Notably, a positive association between blood pressure and acceptance of unfair treatment has been shown in a population of working-class African Americans³⁶. As hypertension is an important risk factor for CKD, the stress of discrimination may serve as a mediator of the relationship between poverty and CKD in African Americans.

Our study has certain limitations. As an observational study, the possibility of selection bias is of concern, as is participant drop-out and failure to complete study measures. We noted that those participants who completed laboratory assessments necessary for this analysis differed from the non-completers on a number of potentially relevant covariates, including age, gender and employment status. An additional limitation is that the cross-sectional analyses performed do not allow for determination of causality. Therefore, although very improbable, reverse causality (CKD causing poverty) is a possibility. Prior longitudinal studies, however, have supported the notion that poverty may often precede the development of progressive CKD and ESRD^{6, 7, 20}. There were also some limitations to the definition of poverty (low SES) used in our study. We were primarily restricted to a self-report of falling above or below poverty level as reported during the initial household survey. Only 76% of participants included in our analysis gave detailed information regarding their actual household income, and we were lacking other important measures of SES, such as inherited wealth and life-course SES which may have impacted our findings. Finally, because our study was conducted in an urban setting, our findings may not be generalizable to non-urban populations.

The potential role of poverty in the greater burden of advanced kidney disease seen among African Americans is worthy of further investigation. Future studies should focus on specific

factors related to poverty that may account for the strong differential influence it appears to have on African Americans in the development of kidney disease.

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Figure1.

Prevalence of CKD (chronic kidney disease) by Socioeconomic Status (SES) and Race. SES is defined as low SES or higher SES based on whether a participant reported annual household income below or above 125% of the 2004 Department of Health and Human Services poverty guideline.²⁴ CKD Stages were defined by National Kidney Foundation guidelines (Stage 3: $30 \leq \text{eGFR} < 60 \text{ mL/min/1.73 m}^2$, Stage 4: $15 \leq \text{eGFR} < 30 \text{ mL/min/1.73 m}^2$, Stage 5: $\text{eGFR} < 15 \text{ mL/min/1.73 m}^2$). Conversion factor for CKD in mL/min/1.73 m^2 to mL/s/1.73 m^2 , $\times 0.01667$.

Table 1

Participant Characteristics by Socioeconomic Status* and Race

Characteristic	No.	SES		P	African Americans		P	Whites		P
		Low N (%)	Higher N (%)		Low SES N (%)	Higher SES N (%)		Low SES N (%)	Higher SES N (%)	
Total	2,375	1,060	1,315	---	713 (67.3)	707 (53.8)	---	347 (32.7)	608 (46.2)	---
Demographics										
Age, years [mean(SD)]	2,375	47.7 (9.0)	48.8 (9.2)	0.004	47.6 (8.9)	49.1 (9.1)	0.001	47.9 (9.1)	48.3 (9.3)	0.5
Male	2,375	434 (40.9)	599 (45.6)	0.03	302 (42.4)	312 (44.1)	0.5	132 (38.0)	287 (47.2)	0.007
Ever Married	2,311	620 (59.7)	921 (72.4)	<0.001	389 (54.7)	486 (69.0)	<0.001	231 (70.4)	435 (76.6)	0.05
No. People in Household [mean(SD)]	1,892	3.9 (3.8)	3.4 (1.6)	<0.001	3.9 (4.5)	3.7 (1.7)	0.2	3.7 (1.9)	3.0 (1.4)	<0.001
Insured	2,311	594 (57.2)	964 (75.8)	<0.001	410 (57.7)	514 (73.0)	<0.001	184 (56.1)	450 (79.2)	<0.001
Currently employed	2,311	397 (38.2)	871 (68.5)	<0.001	277 (39.0)	467 (66.3)	<0.001	120 (36.6)	404 (71.1)	<0.001
Education ≥ high school	2,311	674 (64.9)	1029 (80.9)	<0.001	475 (66.8)	573 (83.4)	<0.001	199 (60.7)	456 (82.3)	<0.001
Years of education [mean(SD)]	2,311	11.7 (4.6)	13.2 (3.2)	<0.001	11.7 (4.0)	12.9 (2.7)	<0.001	11.6 (5.7)	13.5 (3.7)	<0.001
Health Behaviors										
Tobacco use	2,106	682 (74.5)	758 (63.7)	<0.001	444 (74.3)	419 (64.4)	<0.001	238 (74.8)	339 (62.9)	<0.001
Excess alcohol use	2,096	48 (5.3)	42 (3.5)	0.06	35 (5.9)	24 (3.7)	0.08	13 (4.1)	18 (3.4)	0.6
Ever used cocaine regularly	2,103	248 (27.3)	230 (19.3)	<0.001	175 (29.7)	139 (21.3)	0.001	73 (22.8)	91 (16.9)	0.04
Ever used heroin regularly	2,005	167 (19.2)	127 (11.2)	<0.001	130 (23.4)	94 (14.8)	<0.001	37 (11.8)	33 (6.6)	0.01
Comorbidities										
Hypertension	2,301	492 (48.3)	544 (42.4)	0.01	346 (51.0)	329 (48.3)	0.4	146 (43.1)	215 (35.7)	0.03
SBP [mmHg, mean SD]]	2,258	121 (19)	120 (17)	0.09	122 (19)	122 (17)	0.8	119 (19)	118 (17)	0.3
Diabetes	2,375	179 (16.9)	217 (16.5)	0.8	107 (15.0)	133 (18.8)	0.06	72 (20.8)	84 (13.8)	0.006
Cardiovascular disease	2,026	183 (20.7)	179 (15.7)	0.004	126 (22.1)	105 (16.7)	0.02	57 (18.2)	74 (14.4)	0.2
Obesity	2,296	422 (41.1)	565 (44.5)	0.1	260 (37.8)	326 (48.1)	<0.001	162 (47.8)	239 (40.4)	0.03
Laboratory Measures										
Fasting plasma glucose [mg/dL, mean (SD)]	2,374	105 (44)	106 (45)	0.5	103 (44)	106 (45)	0.3	109 (45)	107 (46)	0.5
Total serum cholesterol [mg/dL, mean(SD)]	2,372	184 (42)	189(42)	0.008	183 (42)	186 (42)	0.2	187 (42)	193 (43)	0.05
Serum uric acid [mg/dL, mean(SD)]	2,371	5.4 (1.6)	5.5 (1.6)	0.2	5.4 (1.7)	5.6 (1.7)	0.02	5.4 (1.5)	5.3 (1.5)	0.7

Characteristic	No.	SES		African Americans		Whites	
		Low N (%)	Higher N (%)	Low SES N (%)	Higher SES N (%)	Low SES N (%)	Higher SES N (%)
Serum albumin [g/dL, mean(SD)]	2,374	4.20 (0.37)	4.30 (0.32)	4.17 (0.39)	4.26 (0.31)	4.27 (0.31)	4.34 (0.32)
Health Care Utilization							
Regular health care provider	2,311	573(55.2)	912 (71.7)	390 (54.9)	494 (70.2)	183 (55.8)	418 (73.6)
Emergency room visit in past year	1,879	382 (46.5)	383 (36.2)	274 (47.2)	236 (39.6)	108 (44.8)	147 (31.9)
Hospitalization in past year	1,643	60 (8.7)	70 (7.3)	35 (7.2)	32 (6.1)	25 (12.2)	38 (8.9)
				0.3	0.5		0.2

Socioeconomic status (SES) is defined as low SES or higher SES based on whether a participant reported annual household income below or above 125% of the 2004 US Department of Health and Human Services poverty guideline²⁴

Conversion factors for units: glucose in mg/dL to mmol/L, $\times 0.05551$; cholesterol in mg/dL to $\mu\text{mol/L}$, $\times 0.02586$; uric acid in mg/dL to $\mu\text{mol/L}$, $\times 59.48$; albumin in g/dL to g/L, $\times 10$.

Abbreviation : SBP, systolic blood pressure.

Table 2

Logistic Regression Models of Low SES and CKD (eGFR <60 mL/min/m²), Overall and Within Racial Groups

Model	Variables Included	No.	OR (95% CI)			P for interaction (poverty × race)
			All	African Americans	Whites	
1	low SES	2375	1.41 (.99-2.01)	1.93 (1.33-2.80)	.87 (0.47-1.60)	0.05
2	+ age	2375	1.52 (1.09-2.12)	2.15 (1.58-2.94)	.89 (0.48-1.64)	0.02
3	+ demographics ^a , education, health insurance status	2310	1.61 (1.17-2.23)	2.26 (1.69-3.04)	.91 (0.49-1.69)	0.01
4	+ comorbid illnesses ^b	1941	1.59 (1.27-1.99)	1.91 (1.54-2.38)	.95 (0.58-1.55)	0.003
5	tobacco and drug use ^c	1816	1.42 (1.11-1.82)	1.78 (1.38-2.30)	.83 (0.49-1.40)	0.006

Socioeconomic Status (SES) is defined as low SES or higher SES based on whether a participant reported annual household income below or above 125% of the 2004 Department of Health and Human Services poverty guideline ²⁴

Abbreviations: CI, confidence interval; OR, odds ratio; SES, socioeconomic status; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

Factor for conversion of eGFR in mL/min/1.73 m² to mL/s/1.73 m², ×0.01667.

^aDemographics include: race, sex and marital status

^bComorbid illnesses include: hypertension, diabetes, cardiovascular disease, and obesity

^cDrug use is defined as a previous history of regular heroin and/or cocaine use.