

ORIGINAL INVESTIGATIONS

Pathogenesis and Treatment of Kidney Disease

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

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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 aged 30-64 years residing within 12 neighborhoods selected for both socioeconomic and racial diversity in Baltimore City, MD.

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

Outcomes & Measurements: CKD defined as estimated glomerular filtration rate <60 mL/min/1.73 m². Logistic regression used to calculate ORs for relationship between poverty and CKD, stratified by race.

Results: Of 2,375 participants, 955 were white (347 low SES and 608 higher SES) and 1,420 were African American (713 low SES and 707 higher SES). 146 (6.2%) participants had CKD. Overall, race was not associated with CKD (OR, 1.05; 95% CI, 0.57-1.96); however, African Americans had a much greater odds of advanced CKD (estimated glomerular filtration rate <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, stratified by race, low SES was associated with CKD in African Americans (OR, 1.91; 95% CI, 1.54-2.38), but not whites (OR, 0.95; 95% CI, 0.58-1.55; *P* for interaction = 0.003).

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 whites, in an urban population of adults, and its role in the racial disparities seen in CKD is worthy of further investigation. *Am J Kidney Dis* 55:992-1000. © 2010 by the National Kidney Foundation, Inc. Published by Elsevier Inc. All rights reserved.

INDEX WORDS: Socioeconomic status; health disparities; epidemiology; renal disease.

Editorial, p. 977

An association between low socioeconomic status (SES) and chronic kidney disease (CKD) has been established both in the United States and worldwide.^{1,2} Low SES also has been associated with important precursors and risk factors for CKD, including micro- and macroalbuminuria,³ diabetes,⁴ and hypertension,⁵ and with

an increased risk of progressive CKD in specific populations, including older adults⁶ and white men.⁷ There also is extensive literature documenting a relationship between low SES and end-stage renal disease (ESRD) in multiple clinical populations.⁸⁻¹¹

Racial and ethnic disparities are profound in CKD.^{12,13} Although African American race is not associated with an increased prevalence of

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less severe stages of CKD,¹⁴ African Americans have up to a 4 times greater risk of ESRD compared with whites.¹⁵ Additionally, the annual incidence of ESRD in African Americans is greater than 1,000 per million persons in several cities, including Baltimore, MD.¹⁵ This disparity is believed to be caused in part by 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 in African Americans,¹⁶ and some other studies have drawn similar conclusions.^{17,18} Furthermore, a study examining racial disparities in diabetes-related kidney function decreases found that SES accounted for 52% of this disparity.¹⁹ In addition, in a retrospective analysis of individuals who had initiated dialysis therapy in the Southeastern United States, it recently 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 is 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 aged 30-64 years at enrollment drawn from 12 neighborhoods, each of which is composed of contiguous US census tracts in Baltimore City that reflect socioeconomic and racial diversity. Participants were sampled representatively using a factorial cross of 4 factors (age, sex, race, and SES) with approximately equal numbers of participants per "cell." Individuals who identi-

fied with neither African American nor white race were excluded from the study. Household enrollment was from August 2004 to November 2008. Response rates in eligible individuals varied by neighborhood and were 42.9%-79.6%, similar to those of other population-based studies of African Americans.²³ 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 measurements. These participants were slightly older (48.3 vs 46.7 years), more likely to be women (49% vs 43%), more likely to be of low SES (45% vs 41%), and less likely to be employed (55% vs 59%) than those without creatinine data ($P < 0.05$ for all), but both groups were of similar race.

Measurements

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 or higher SES based on whether a participant reported an annual household income $<125\%$ or $>125\%$ of the 2004 Department of Health and Human Services poverty guideline.²⁴ This cutoff value for low SES was selected by a panel of experts and has been used in initiatives such as the National School Lunch Program.²⁵ 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 cutoff?" Race was self-reported (African American or white) during the initial household survey. Individuals identifying themselves as multiethnic 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, and occupational and educational history, also were 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 use was assessed on the mobile research vehicle. Fasting venous blood 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 using 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 size.²⁶ Hypertension was defined as an average of seated and standing systolic blood pressure ≥ 140 mm Hg, an average of seated and standing diastolic blood pressure ≥ 90 mm Hg,²⁶ a history of blood pressure medication use, and/or a self-report of hypertension. Diabetes mellitus was defined as fasting plasma glucose concentration ≥ 126 mg/dL (≥ 7.0 mmol/L)²⁷ or self-report of diabetes. Cardiovascular disease was defined as self-reported history of congestive heart failure, enlarged heart, angina, myocardial infarction, coronary artery disease, transient

ischemic attack, and/or stroke. Anthropometric measurements were performed, including height and weight, and were used to calculate body mass index to determine the presence of obesity (defined as body mass index ≥ 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 6 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 6 months or longer than 6 months ago.

The dependent variable was CKD, which was determined using single laboratory measurements of serum creatinine (n = 2,375) and urine albumin (n = 1,472). Serum creatinine was measured for 236 participants at the NIA Clinical Research Branch Core Laboratory using a modified kinetic Jaffé method (CREA method, Dimension Xpand Clinical Chemistry System; Siemens Healthcare Diagnostics Inc, www.medical.siemens.com) and was measured for the rest of participants (n = 2,139) at Quest Diagnostics Inc using 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 immunoturbidimetric assay (Kamiya Biomedical Co, www.kamiyabiomedical.com).

CKD was defined primarily as the presence of estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m² (using the 4-variable IDMS-traceable Modification of Diet in Renal Disease [MDRD] Study equation²⁸). In a sensitivity analysis, CKD was defined as eGFR < 60 mL/min/1.73 m² or urine albumin excretion ≥ 30 mg/g creatinine. CKD stages were defined using National Kidney Foundation guidelines.²⁹

Statistical Analysis

Participant characteristics stratified by SES and race were compared using Fisher exact tests for categorical variables and *t* tests for continuous variables. Descriptive statistics and Fisher 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. Analyses stratified by race were performed and an interaction between race and SES was considered in overall regression models.

Potential confounders were chosen based on variables associated with CKD in the 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 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 level) 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 eGFR < 60 mL/min/1.73 m² or albuminuria as noted by including

available measurements 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 used in a prior study of CKD and SES.² Third, annual household income was categorized into 24 strata (in \$5,000 increments) and examined for its relationship to CKD in participants who reported their income specifically (n = 1,812), beyond just stating whether they were 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 participants who had laboratory measurements performed at Quest Diagnostics Inc underwent IDMS-traceable serum creatinine measurements, 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 modeling.³⁰ A 2-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, be employed, or have completed a high school education compared with those with higher SES. Also, those with low SES were more likely to report tobacco, cocaine, and/or heroin use than higher SES individuals. Serum albumin levels were lower in low-SES participants, and the low-SES group was more likely to have reported an emergency department visit in the preceding year and the absence of a regular health care provider compared with higher SES participants. These 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 in low-SES whites than higher SES whites, but in African Americans, there was no difference by SES. Diabetes was more prevalent in 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

Table 1. Participant Characteristics by SES and Race

Characteristic	No. of Participants	SES			African Americans			Whites		
		Low	Higher	P	Low SES	Higher SES	P	Low SES	Higher SES	P
Total	2,375	1,060	1,315	—	713 (67.3)	707 (53.8)	—	347 (32.7)	608 (46.2)	—
Demographics										
Age (y)	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
Men	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. of people in household	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)	1,029 (80.9)	<0.001	475 (66.8)	573 (83.4)	<0.001	199 (60.7)	456 (82.3)	<0.001
Years of education	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
Comorbid conditions										
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 (mm Hg)	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 measurements										
Fasting plasma glucose (mg/dL)	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)	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)	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
Serum albumin (g/dL)	2,374	4.20 ± 0.37	4.30 ± 0.32	<0.001	4.17 ± 0.39	4.26 ± 0.31	<0.001	4.27 ± 0.31	4.34 ± 0.32	0.004
Health care use										
Regular health care provider	2,311	573 (55.2)	912 (71.7)	<0.001	390 (54.9)	494 (70.2)	<0.001	183 (55.8)	418 (73.6)	<0.001
Emergency department visit in past year	1,879	382 (46.5)	383 (36.2)	<0.001	274 (47.2)	236 (39.6)	0.02	108 (44.8)	147 (31.9)	0.001
Hospitalization in past year	1,643	60 (8.7)	70 (7.3)	0.3	35 (7.2)	32 (6.1)	0.5	25 (12.2)	38 (8.9)	0.2

Note: Values expressed as mean ± standard deviation or number (percentage). SES is defined as low or higher SES based on whether a participant reported annual household income <125% or >125% of the 2004 US Department of Health and Human Services poverty guideline.²⁴ Conversion factors for units: glucose in mg/dL to mmol/L, ×0.05551; cholesterol in mg/dL to mmol/L, ×0.02586; uric acid in mg/dL to μmol/L, ×59.48; albumin in g/dL to g/L, ×10.

Abbreviations: SBP, systolic blood pressure; SES, socioeconomic status.

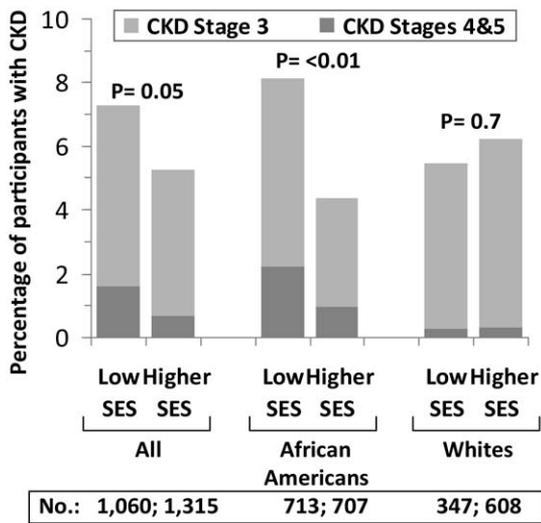


Figure 1. Prevalence of chronic kidney disease (CKD) by socioeconomic status (SES) and race. SES is defined as low or higher SES based on whether a participant reported annual household income <125% or >125% of the 2004 Department of Health and Human Services poverty guideline.²⁴ CKD stages were defined by National Kidney Foundation guidelines (stage 3, estimated glomerular filtration rate [eGFR] ≤30 to <60 mL/min/1.73 m²; stage 4, eGFR ≤15 to <30 mL/min/1.73 m²; and stage 5, eGFR <15 mL/min/1.73 m²). Conversion factor for eGFR in mL/min/1.73 m² to mL/s/1.73 m², ×0.01667.

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 with whites in univariate analysis, 1.05; 95% confidence interval [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 in those of low SES (7.3%) as opposed to higher SES (5.3%), although this reached only borderline statistical significance (Fig 1). However, with racial stratification, low SES was associated with a greater prevalence of CKD in African Americans, but not whites.

Logistic regression models of the relationship between low SES and CKD showed 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, 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 whites (OR, 0.95; 95% CI, 0.58-1.55; P interaction = 0.003). Further adjustment for tobacco and illicit drug use yielded similar results; however, the OR was slightly attenuated for African Americans.

Table 2. Logistic Regression Models of Low SES and CKD (eGFR <60 mL/min/m²) Overall and Within Racial Groups

Model	Variables Included	No. of Participants	OR (95% CI)			P for Interaction (poverty × race)
			All	African Americans	Whites	
1	Low SES	2,375	1.41 (0.99-2.01)	1.93 (1.33-2.80)	0.87 (0.47-1.60)	0.05
2	+ age	2,375	1.52 (1.09-2.12)	2.15 (1.58-2.94)	0.89 (0.48-1.64)	0.02
3	+ demographics, ^a education, health insurance status	2,310	1.61 (1.17-2.23)	2.26 (1.69-3.04)	0.91 (0.49-1.69)	0.01
4	+ comorbid illnesses ^b	1,941	1.59 (1.27-1.99)	1.91 (1.54-2.38)	0.95 (0.58-1.55)	0.003
5	Tobacco and drug use ^c	1,816	1.42 (1.11-1.82)	1.78 (1.38-2.30)	0.83 (0.49-1.40)	0.006

Note: SES is defined as low or higher SES based on whether a participant reported annual household income <125% or >125% of the 2004 Department of Health and Human Services poverty guideline.²⁴ Factor for conversion of eGFR in mL/min/1.73 m² to mL/s/1.73 m², ×0.01667.

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

^aDemographics include race, sex, and marital status.

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

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

Sensitivity Analyses

CKD defined as 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 results similar to our primary definition, with a CKD OR for those with low SES versus 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 also were noted in a similar model, with low SES having a strong association with CKD in African Americans (OR, 1.88; 95% CI, 1.61-2.19), but not whites (OR, 0.82; 95% CI, 0.48-1.38), with *P* for interaction < 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 comorbid conditions. Although there was a trend toward a greater relationship between poverty and CKD in African Americans, the relationship between low SES and CKD did not statistically significantly vary by race (*P* for interaction = 0.3).

Income status categorized into 24 strata for the 1,812 participants who reported these data showed that the mean income category generally was higher for low-SES whites than for low-SES African Americans (12.6 vs 10.9, corresponding to annual incomes of \$11,000-\$12,000 and \$9,000-\$10,000, respectively; *P* = 0.001). Additionally, no statistically significant association between income category and CKD overall was observed, however, stratification by race showed such a relationship in African Americans, but not whites, although the *P* for 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 in African Americans (*P* interaction < 0.001). Years of education were associated negatively with CKD in African Americans, but not whites, although *P* interaction was not significant at 0.4.

Analyses restricted to only 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 for at least 1 covariate

(55% were African American). Comparing African Americans with missing data with whites, 64% versus 27% were of low SES. Multiple imputation of missing covariates yielded results similar to our primary analysis, with a 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 in African Americans (OR, 1.75; 95% CI, 1.38-2.22) and not whites (OR, 0.88; 95% CI, 0.56-1.39), with *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 in African Americans, but not whites. In African Americans, low SES was associated independently with a nearly 2-fold greater risk of CKD compared with higher SES. In addition, we found that poverty had no statistically significant relationship with CKD in whites; however, there was a trend toward a negative association. Our major finding was robust to adjustment for several risk factors for CKD and to the use of stricter eGFR cutoff values. We found that in general, further adjustment for potential confounders strengthened the association between poverty and CKD; however, this was caused in part by negative confounding (eg, both age and health insurance were associated positively with CKD, but negatively with poverty).

Few studies have reported on the relationship between SES and pre-ESRD CKD, and of these, variable associations with race have been reported. Shoham et al² found in the Atherosclerosis Risk in Communities (ARIC) Study that working class membership (a measure of low SES) in the life course was associated more strongly with CKD in African Americans than whites, even independent of hypertension and diabetes. However, in an earlier report from the same study population, Merkin et al⁷ noted that living in a low-SES area was associated with progressive CKD in only white men. In studies examining SES, race, and CKD in participants with specific diseases, significant contributions of SES to racial disparities in CKD have been observed in persons with diabetic¹⁹ and hypertensive³¹ kidney disease.

There are several reasons that 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 included 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, 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 in both African Americans and whites.

The major implication of our study is that poverty may impact on African Americans differently than on whites in the development of CKD. Poverty may exert its differential effect on African Americans through several mechanisms. Plausible biological mechanisms include the increased prevalence of low birth weight observed in African Americans, a condition associated with poverty. Low birth weight is a risk factor for ESRD and is believed to be a contributor to the racial disparities seen in ESRD.³² Also, the gene encoding non-muscle myosin heavy chain type II isoform A (*MYH9*) has been associated with nondiabetic ESRD in African Americans, but not whites.³³ Poverty and its consequences (ie, toxic environmental exposures, such as heavy metals) may have a role in the probable gene-environment interactions that lead to ESRD in these individuals. It also has been reported that living in low-SES neighborhoods is associated more strongly with greater cumulative biological risk profiles (defined using 9 indicators of increased risk) in African Americans than whites.³⁴ Many of these indicators, such as blood pressure and waist-to-hip ratio, also are associated with an increased prevalence of CKD.

Poverty also may differentially impact on health beliefs and behaviors in African Americans compared with 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 in African Americans, but not 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 on 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 Americans.³⁶ Because 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 participants who completed laboratory assessments necessary for this analysis differed from noncompleters on a number of potentially relevant covariates, including age, sex, 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. However, prior longitudinal studies have supported the notion that poverty often may precede the development of progressive CKD and ESRD.^{6,7,20} There also were some limitations to the definition of poverty (low SES) used in our study. We were restricted primarily to a self-report of being 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 on our findings. Finally, because our study was conducted in an urban setting, our findings may not be generalizable to nonurban populations.

The potential role of poverty in the greater burden of advanced kidney disease seen in 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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The Interaction of Race, Poverty, and CKD

Related Articles, p. 992 and p 1001

Poverty is highly prevalent in the United States, disproportionately affects minorities, and is inextricably linked to poor health outcomes including cardiovascular disease,¹ incidence of type 2 diabetes,² obesity,³ hypertension,⁴ and death.^{5,6} In 2008, nearly 40 million Americans lived at or below the poverty level. This reflected an increase for whites and most racial and ethnic minorities, but not for African Americans, although African Americans continue to suffer the highest poverty level in the United States at 24.7%.⁷ Poverty or low socioeconomic status (SES) incorporates 3 major determinants of health: health care access, environmental exposure, and health behavior,⁸ all of which contribute to health care disparities directly and indirectly through a multitude of mechanisms, including decreased access to preventive and ongoing medical care, lack of healthy food choices, higher rates of hypertension and diabetes, and poorer social and physical environments.⁸⁻¹¹ Although neighborhood and individual poverty have been associated with an increased risk of end-stage renal disease¹²⁻¹⁵ (ESRD, defined as kidney failure treated by dialysis or transplant), data for the association of poverty with earlier stages of chronic kidney disease (CKD) have been variable.

In this issue of the *American Journal of Kidney Diseases*, 2 separate studies evaluate poverty and the prevalence of earlier stages of CKD.^{16,17} The first evaluates the association of poverty and race with the prevalence of CKD among urban African Americans and whites who took part in the National Institutes of Health (NIH)-sponsored population-based Healthy Aging in Neighborhoods of Diversity across the Lifespan (HANDLS) study, while the other evaluates the association of poverty and the prevalence of CKD among a more rural, southern African American population followed as part of the NIH-sponsored Jackson Heart Study.¹⁸ Crews et al defined CKD by estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m²; Bruce et al defined CKD by eGFR <60 mL/min/1.73 m² or the presence of microalbuminuria (defined as a urine albumin-creatinine ratio >30 mg/g). Crews et al found that African Americans were not more likely to

have CKD in general, but were 5 times more likely than whites (odds ratio [OR], 5.04; 95% confidence interval [CI], 1.21-21.01) to have advanced stages of CKD at baseline, defined as stage 4 or 5 CKD (eGFR <30 mL/min/1.73 m²). They also ascertained that those living in poverty (defined as <125% of the national poverty level) were 59% more likely to be diagnosed with CKD than those above this level; however, when stratified by race, low SES was only significantly 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), confirming an interaction or effect modification between poverty and African American status on the prevalence of CKD. Similarly, Bruce et al found affluent African Americans in the Jackson Heart Study had a 41% lower prevalence of CKD than their less affluent counterparts. Prevalent CKD differed slightly by sex and was lower for married versus not married, but did not differ by educational level. Together, these papers begin to dissect the complicated relationship between poverty, race, and CKD.

Previous studies have evaluated the association between poverty and CKD with inconsistent definitions of CKD and results. Tarver-Carr et al found that sociodemographic factors such as poverty, education, and marital status contributed to 23.7% of the 2.7-fold excess risk of CKD (defined as incident dialysis or death related to kidney disease) for African Americans compared to whites in the second National Health and Nutrition Examination Survey (NHANES II). Simultaneous adjustment for sociodemographic, clinical, and lifestyle variables decreased risk by 44%; however, not all increased risk was accounted for in their model (54%).¹⁹ Martin et al evaluated the odds of prevalent albuminuria by

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poverty level (defined as <200% of the federal poverty level) in the NHANES III population and found that African Americans had higher odds of both micro- and macroalbuminuria if they lived in poverty.²⁰ Evaluating the association of neighborhood poverty status and progression of CKD in the Atherosclerosis Risk in Communities (ARIC) Study, Merkin and colleagues found that only white men in the lowest SES quintile had a higher risk of CKD progression compared to white men in the highest SES quintile, while African American men and women and white women did not have an increased risk of progressive CKD by SES quintile.²¹ Using data from the same study but evaluating the lifetime exposure to poverty, Shoham et al found being from the “working class” (defined by responses to a 5-item questionnaire) was more strongly associated with prevalent CKD (defined as eGFR <45 mL/min/1.73 m² or an annual hospital discharge diagnosis of CKD) for African Americans (OR, 1.9) than for whites (OR, 1.4) after adjusting for prevalent diabetes and hypertension.²² In addition, restricting analyses to only those with diabetes in the ARIC study, Krop et al found that African Americans had a 3-fold greater risk of progression of CKD (based on increase of serum creatinine) and that 7% of the 82% in excess risk of progression was explained by SES, while 30% of the risk was due to modifiable risk factors such as physical activity, cigarette smoking, serum glucose, and systolic blood pressure, arguably factors that are related to and increased among those living in poverty.²³ Finally, evaluating individual and neighborhood SES and association with progressive CKD in the Cardiovascular Health Study, Merkin and colleagues found that those living in the lowest SES quartile had a 50% greater risk (HR, 1.5; 95% CI, 1.0-1.5) of progressive CKD compared with the highest quartile after adjustment for age, sex, and individual-level SES.²⁴

The articles by Crews et al and Bruce et al suggest the possibility of a race-poverty interaction which may be implicated in the paradox of CKD progression, whereby African Americans exhibit a greater proportion of earlier stages of CKD than whites in general populations, but a higher incidence of ESRD.²⁵ This suggests that African Americans may have a faster rate of CKD progression to ESRD compared to whites.²⁵ Interestingly, although many of the participants

in the Baltimore population (Crews et al) had access to health insurance, there was a high prevalence of CKD. The increased prevalence of CKD may be accentuated by a culture of poverty, which would include such aspects as lower educational level, higher levels of obesity, greater levels of hypertension and diabetes, decreased exercise, and more limited access to healthy foods, all of which may act as CKD accelerators.

The strengths of the 2 articles are that both are NIH-sponsored, observational longitudinal cohort studies, which include standardized methods for obtaining variables of interest such as blood pressure, body mass index, and laboratory outcome measures including albuminuria and serum creatinine. Both studies used calibrated creatinine measurements and used eGFR as a measure of the outcome of interest. In addition, self-report of SES factors such as race/ethnicity, insurance status, income, and education strengthen both studies, while the use of census tract geocoding to determine neighborhood poverty by Crews et al strengthens their study.

There are several limitations that must be kept in mind while reading these studies. First, Crews et al defined CKD as eGFR <60 mL/min/1.73 m², while Bruce et al used the definition eGFR <60 mL/min/1.73 m² or the presence of albuminuria (but did not adjust for differences in microalbuminuria by sex). Second, both included different measures of poverty, which makes comparisons difficult. Efforts to develop standard measures of poverty and individual wealth have resulted in several measures in the literature, none of which were used in the 2 articles. Some standard measures include <200% of the federal poverty level, a poverty ratio,²⁶ 5% to 20% gradations below the federal poverty level,⁵ the life course of socioeconomic status,²² and a combined or created measure of several variables that evaluate SES and wealth.¹⁴ Crews et al used <125% of the poverty level as their measure of SES, while Bruce et al used a variable that started at the poverty level. Finally, because both studies were cross-sectional by design, both fail to establish a causal link of poverty with CKD.

Given the association of poverty with CKD, evaluation of potential mechanisms can be approached by use of the biopsychosocial model,²⁷ which incorporates individual, neighborhood, and biologic factors that potentially affect CKD inci-

dence and progression. Individual factors include such issues as income and wealth (resources, purchasing power, and safety),²⁸⁻³⁰ access to health care (insurance coverage, preventive health, access to general healthcare),³¹ education level, diet, exercise, poor prenatal care, obesity, and increased diagnoses of risk factors for CKD such as cardiovascular disease, diabetes, and hypertension.^{1-4,32,33} Neighborhood or environmental factors include access to physical activity facilities, neighborhood safety, availability of healthy food, environmental exposures to heavy metals or other toxins such as pollutants, residential or neighborhood racial segregation^{29,30} and attendant poor educational opportunities, lifetime exposure to poverty (working class),^{22,34} urban versus rural living conditions,^{35,36} transplant, and mortality.³⁷ Finally, genetic or biologic predisposition, such as the presence of the myosin *MYH9* gene mutation in nondiabetic patients with focal segmental glomerulosclerosis, may be associated with accelerated CKD progression in the setting of poverty.^{38,39}

In conclusion, poverty is widespread in our society, is difficult to treat, and is associated with poor outcomes, including CKD. By some estimates, health disparities contributed over \$1 trillion in direct and indirect expenses from 2003-2006, at least some of which could be blamed on poverty.⁴⁰ The “War on Poverty” first proposed by President Lyndon B. Johnson in 1964, which led to the Economic Opportunity Act of 1964 and the formation of the Head Start and Job Corps programs, resulted in decreased poverty rates from a high of 19% in 1964 to 13.2% in 2008. The current administration has a somewhat different perspective on the problem of poverty. In the words of President Barack Obama in his first State of the Union address: “In the 21st century, the best anti-poverty program around is a world-class education.”⁴¹ Education may be a key for increasing CKD awareness in the general public and particularly among those at risk such as minorities, the uninsured, and those who live in poverty. As a society, we are willing to pay for the initiation of dialysis, an entitlement program established by Republican President Richard Nixon, but we are unwilling to pay for its prevention. Determining an association between CKD and poverty should not lure us into “blaming the victims” for their increased risk of CKD; how-

ever, it should force us to search for better solutions to decrease CKD progression for all 26 million Americans with CKD.⁴² The culture of poverty and CKD must be broken by increased community outreach, better education, improved economic opportunity, and access to preventive health care for those most at risk.

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