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The Interaction of Race, Poverty, and CKD

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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 and 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

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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 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 $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$, while Bruce et al used the definition $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ 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 incidence 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; however, 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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