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Excess Risk of Chronic Kidney Disease among African-American *versus* White Subjects in the United States: A Population-Based Study of Potential Explanatory Factors

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Abstract. African Americans experience higher rates of chronic kidney disease (CKD) than do whites. It was hypothesized that racial differences in modifiable factors would account for much of the excess risk of CKD. A cohort study of 9082 African-American and white adults of age 30 to 74 yr, who participated in the Second National Health and Nutrition Examination Survey in 1976 to 1980 and were monitored for vital status through 1992 in the Second National Health and Nutrition Examination Survey Mortality Study, was conducted. Incident CKD was defined as treated CKD cases (ascertained by linkage to the Medicare Registry) and deaths related to kidney disease. The incidence of all-cause CKD was 2.7 times higher among African Americans, compared with whites. Adjustment for sociodemographic factors decreased the relative risk (RR) to 2.49, explaining 12% of the excess risk of CKD among African Americans. Further adjustment for lifestyle factors explained 24% of the excess risk, whereas adjustment for clinical factors alone explained 32%. Simultaneous adjustment for sociodemographic, lifestyle, and clinical factors attenuated the RR to 1.95 (95% confidence interval, 1.05 to 3.63), explaining 44% of the excess risk. Although the excess risk of CKD among African Americans was much greater among middle-age adults (30 to 59 yr of age; RR = 4.23, statistically significant) than among older adults (60 to 74 yr of age; RR = 1.27), indicating an interaction between race and age, the same patterns of explanatory factors were observed for the two age groups. Nearly one-half of the excess risk of CKD among African-American adults can be explained on the basis of potentially modifiable risk factors; however, much of the excess risk remains unexplained.

Since the late 1970s, the incidence of end-stage renal disease (ESRD) has increased at a fourfold higher rate among African-American individuals, compared with white individuals (1). Suggested explanations for this racial disparity include lower socioeconomic status among African Americans (2–6), higher prevalence and greater severity of diabetes mellitus and hypertension among African Americans (7–11), and increased inherited susceptibility of African Americans to kidney damage (12–14). In the long term, identification of susceptibility genes might lead to the development of more preventive measures for African-American and white subjects; however, it is important to identify risk factors for African Americans that can be modified with current approaches. One set of potentially modifiable factors is represented by socioeconomic status, which is an indirect marker of suboptimal health behaviors, inadequate access to health care, and possible adverse environmental exposures (15–18). Another set of potentially modifiable factors includes complex traits such as BP and glycemic status, which are modifiable with behavior modification and pharmacotherapy.

Previous studies that sought to explain the African-American/white differences in chronic kidney disease (CKD) rates demonstrated several limitations. Some studies used case-control study designs, which limit causal inferences (4), or relied on ecologic exposure data for information that was not available at the individual level (3,7,19–21). Other studies either used study populations that were not typical of the United States (3,11,22–24) or relied on intermediate markers of kidney disease as outcomes (6). With these considerations in mind, we conducted a population-based, prospective study to determine how much of the excess risk of CKD among African Ameri-

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cans could be explained on the basis of racial disparities in potentially modifiable risk factors.

Materials and Methods

Study Design and Sample

This study was a national, nonconcurrent, population-based, cohort study, in which we analyzed how baseline risk factors in the Second National Health and Nutrition Examination Survey (NHANES II) were related to CKD in 12 to16 yr of passive follow-up monitoring. NHANES II was a cross-sectional, multistage, probability survey of noninstitutionalized American individuals (age, 6 mo to 74 yr) that was conducted by the National Center for Health Statistics from February 1976 to February 1980 (25,26). Details of the plan, complex sampling design, responses, and data collection procedures were previously described (25). The vital status of examined adults of age 30 to 74 yr was passively determined by matching to the National Death Index and the Social Security Administration Death Master Files, using a previously described algorithm (27). Participants were also linked (on the basis of their full name, race, and date of birth) to the Medicare Registry, to determine whether they had received renal replacement therapy (*i.e.*, dialysis or kidney transplantation).

This analysis included African-American and white subjects who were 30 to 74 yr of age at the time of the baseline evaluation. Of these 9087 individuals, five who were receiving renal replacement therapy (*i.e.*, listed in the Medicare Registry) at the time of the baseline examination were excluded, as were those with missing baseline data on family income ($n = 369$) or other key variables ($n = 155$). A similar percentage of those excluded developed CKD, compared with those included in the analysis (2.2% *versus* 1.3%, $P = 0.16$). After exclusions, 894 African-American and 7664 white adults remained for analysis.

Data Collection

The race of the participants in NHANES II was determined by the interviewers and was classified as "white," "African American," or "other races" (25). Questions regarding participant age, gender, marital status, education, and household income were also asked during the baseline interviews. To judge the resources available to families as a function of their total income, all household incomes (assessed using income categories, to increase the response likelihood) were expressed relative to the federal poverty thresholds for equally sized households. A poverty income ratio of <1.00 is below the federal poverty level ("poor"), a poverty income ratio of 1.00 to 1.99 is at or near the poverty level ("near poor"), and a poverty income ratio of ≥ 2.00 is 200% above the federal poverty level ("not poor") (28).

Participants were also asked two questions regarding their level of physical activity in leisure and nonleisure activities, *i.e.*, (*1*) whether they were getting "much," "moderate," or "little" exercise for recreation and (*2*) whether in their usual day, aside from recreation, they were "very active," "moderately active," or "quite inactive." Individuals reporting much exercise in recreation and/or a very physically active day were classified as being "very active." Those reporting little recreational exercise and a "quite inactive" daily routine were categorized as being "inactive." All other combinations were considered to be "moderately active." The frequency of beer, wine, and liquor consumption was obtained from a food frequency questionnaire and was categorized as never, seldom (<1 time/wk), weekly (1 to 6 times/wk), or daily (≥ 1 time/d). Smoking history at baseline was used to classify participants as never, former, or current smokers.

The body mass index was calculated from the measured height (in meters) and weight (in kilograms) and was categorized as not overweight (<25 kg/m^2), overweight (25 to 29.9 kg/m^2), or obese (≥ 30 kg/m^2) (29). BP was measured twice in the right arm, by using a standard mercury sphygmomanometer, with the subject seated. The mean of the two readings was used for analysis. Persons were considered hypertensive if they demonstrated a systolic BP of ≥ 140 mmHg or a diastolic BP of ≥ 90 mmHg or if they reported being told by a doctor that they had hypertension. Persons who responded affirmatively to the question, "Has a doctor ever told you that you had diabetes?" were classified as having diabetes mellitus. A history of cardiovascular disease (CVD) was defined on the basis of a self-report of previous heart failure, heart attack, or stroke.

Total serum cholesterol levels were measured according to the protocol described for the Lipid Research Clinics Program (30). Serum creatinine determinations were performed by using the Jaffe reaction, and GFR were estimated by using the Cockcroft-Gault creatinine clearance equation (31). Because the nonresponse rate for serum creatinine levels was 28%, these measurements were considered only in subsidiary analyses (25).

Ascertainment of Outcomes

Incident all-cause CKD was defined as either treatment for ESRD or death related to kidney disease. Treated ESRD cases from February 1976 through December 31, 1992, were ascertained from the Medicare ESRD Registry, by matching participants in NHANES II on the basis of their full name, birth date, gender, and race. Those who were matched to the registry perfectly or nearly perfectly with respect to all of the matching factors were classified as definite matches ($n = 33$), whereas those with slight differences with respect to any of the matching factors were classified as probable matches ($n = 4$). All those who did not match in multiple fields or who did not match with respect to race alone were classified as possible or poor matches ($n = 46$). In our analyses, only definite and probable matches were counted as treated ESRD events.

Kidney-related deaths were identified through 1992 in the NHANES II Mortality Study. Deaths were ascertained by using the National Death Index and the Social Security Administration Death Master Files. Decedents who were not listed in the Medicare ESRD Registry but for whom any of the following kidney-related *International Classification of Diseases* (9th Revision) codes designated the underlying or contributing cause of death were also counted as CKD events: 250.4 (diabetes mellitus with nephropathy), 274.1 (gouty nephropathy), 275.4 (nephrocalcinosis), 403 (hypertensive renal disease), 404 (hypertensive heart and renal disease), 580 to 589 (nephritis, nephrotic syndrome, or nephrosis), or 593.9 (renal disease not otherwise specified). This broad definition was used to include individuals who received Medicare-funded renal replacement therapy, those who might have received it before being enrolled in Medicare, and those who might have chosen not to initiate therapy.

Analyses

All statistical analyses accounted for the complex survey design, providing nationally representative estimates. By using weighted Poisson models in Stata 6.0 (32) and the age distribution of the entire NHANES II cohort, directly age-adjusted CKD incidence rates were calculated per 100,000 person-yr of risk. Cumulative lifetime CKD incidence rates were estimated by using a weighted life-table method, and the cumulative risks of the two race groups were compared by using log-rank tests. Baseline variables were grouped as (*1*) sociodemographic factors, including poverty status, educational attainment, and marital status; (*2*), lifestyle factors, including smoking status, physical activity, alcohol use, and body mass index; and (*3*) clinical

factors, including diabetes mellitus, hypertension, cardiovascular diseases, systolic BP, and serum cholesterol levels. To assess relative differences in CKD incidence rates for African Americans, compared with whites, we conducted a series of five Cox proportional-hazards model analyses with Sudaan 7.5 (33), adjusting for (*1*) age (continuously) and gender; (*2*) age, gender, and sociodemographic factors; (*3*) age, gender, and lifestyle factors; (*4*) age, gender, and clinical factors; and (*5*) all of the aforementioned variables simultaneously. The percentage of excess risk of CKD for African Americans that was explained by a set of risk factors was calculated by using the formula

$$\% \text{ excess risk} = \frac{RR_1 - RR_2}{RR_1 - 1}$$

where RR_1 is the relative risk (RR) of CKD for African Americans *versus* whites adjusted only for age and gender and RR_2 is the RR adjusted for age, gender, and the set of risk factors (34,35).

To test the robustness of the associations, three subsidiary analyses were performed. We first excluded individuals who developed CKD within the first 5 yr of follow-up monitoring, to determine whether the same associations between race and CKD were present. Next, we analyzed the subpopulation with serum creatinine measurements, adjusting for GFR. Finally, we limited the outcome to treated ESRD only. Because analyses performed with treated ESRD as the outcome yielded mostly similar results (data not shown), only analyses with the combined end point of CKD (defined as either treated ESRD or death related to kidney disease) are presented.

Results

Baseline Characteristics of the Cohort

Sociodemographic and clinical characteristics of the 8558 adults who were included in the analysis are presented in Table 1. Compared with white subjects, African-American adults were more likely to have less education, live below the federal poverty level, and be unmarried. They were also more likely to be current cigarette smokers, to be more obese, to be physically inactive, and to drink less alcohol than their white counterparts. In addition, African-American adults demonstrated higher prevalences of diabetes mellitus and hypertension than did white subjects, as well as higher mean systolic BP values and GFR.

Table 1. Baseline characteristics of 8558 adults of age 30 to 74 yr, in NHANES II, according to race[a]

Characteristic	African American (n = 894)	White (n = 7664)
Age (yr)[b]	48.2 ± 0.5	49.3 ± 0.3
Male (%)	45.1	47.4
Sociodemographic		
education (%)[c]		
1 to 8th grade	31.1	17.3
9 to 12th grade	52.4	51.9
college or more	16.5	30.8
poverty status (%)[c]		
poor	26.9	8.3
near poor	33.2	22.4
not poor	39.9	69.4
marital status (%)[c]		
never married	7.4	4.6
no longer married	34.0	15.9
currently married	58.6	79.5
Lifestyle		
smoking status (%)[d]		
never smoked	40.8	37.9
former smoker	19.2	26.6
current smoker	40.0	35.4
physical activity (%)[d]		
inactive	13.0	9.9
moderately active	56.2	62.6
highly active	30.7	27.6
alcohol use (%)[d]		
0	43.8	36.4
<1/wk	16.6	20.8
1 to 6/wk	28.4	27.9
≥1/d	11.2	14.9
obesity (%)[c]		
thin to normal (<25 kg/m^2)	36.9	49.2
overweight (25 to 29.9 kg/m^2)	36.1	35.2
obese (≥30 kg/m^2)	27.0	15.6
Clinical		
Hypertension (%)[c]	59.5	47.0
Diabetes mellitus (%)[c]	7.0	4.0
History of CVD (%)[c]	6.5	6.1
Systolic BP (mmHg)[b,c]	133.0 ± 0.9	129.0 ± 0.6
Serum cholesterol level (mg/dl)[b]	219.9 ± 3.1	223.4 ± 1.1
GFR (ml/min)[b,c,e]	90.8 ± 1.7	87.8 ± 0.8

[a] NHANES II, Second National Health and Nutrition Examination Survey; CVD, cardiovascular disease.
[b] Mean ± SEM.
[c] $P < 0.01$; all comparisons compare African Americans with whites.
[d] $P < 0.05$.
[e] Serum creatinine measurements were available for 6005 adults. GFR was calculated by using the Cockcroft-Gault equation.

Incident CKD

With a median follow-up period of 14 yr, we identified 172 cases of CKD. Of these, 37 patients entered the Medicare ESRD Registry and 135 died without receiving Medicare-funded renal replacement therapy. The age-adjusted incidences of all-cause CKD and treated ESRD were 2.7- and 8.9-fold higher, respectively, among African-American adults, compared with white adults (Table 2). Moreover, the age-adjusted incidence of kidney disease attributable to diabetes mellitus or hypertension was almost 12 times higher among African-American adults, compared with whites. Figure 1 presents the cumulative lifetime incidences of CKD for African Americans *versus* whites. African Americans demonstrated a higher cumulative risk of CKD for every age after 45 yr, compared with whites. By age 80 yr, African Americans who had not died as a result of other causes demonstrated a 9.4% cumulative risk of developing CKD, compared with 3.8% among whites ($P < 0.001$).

Explanatory Factors for Excess Risk

To determine how much of the excess risk of CKD among African-American adults was related to potentially modifiable factors, a series of multivariate proportional-hazard analyses were performed. The base model, adjusted for age and gender only, revealed that African Americans were more than two times more likely than whites to develop CKD [RR = 2.69; 95% confidence interval (CI), 1.55 to 4.51]. Further adjustment for sociodemographic variables reduced the RR to 2.49, corresponding to an 11.8% reduction in the excess risk of African

Table 2. Incident CKD among African-American and white adults of age 30 to 74 yr, in NHANES II[a]

Outcome	African Americans (n = 894)		Whites (n = 7664)	
	No. of Events	Incidence Rates (per 100,000 person-yr)[b]	No. of Events	Incidence Rates (per 100,000 person-yr)[b]
Treated ESRD[c]	12	97	25	11
Diabetic or hypertensive CKD[d]	13	83	28	7
All-cause CKD[e]	33	222	139	84

[a] CKD, chronic kidney disease, defined as receipt of renal replacement therapy or death with kidney disease; ESRD, end-stage renal disease.
[b] Weighted and age-adjusted using the direct method, with the NHANES II African-American and white populations combined as the standard.
[c] Entry into the Medicare ESRD registry.
[d] Entry into the Medicare ESRD registry with renal disease attributed to hypertension or diabetes mellitus or death resulting from hypertensive or diabetic renal disease.
[e] Entry into the Medicare ESRD registry or kidney-related death.

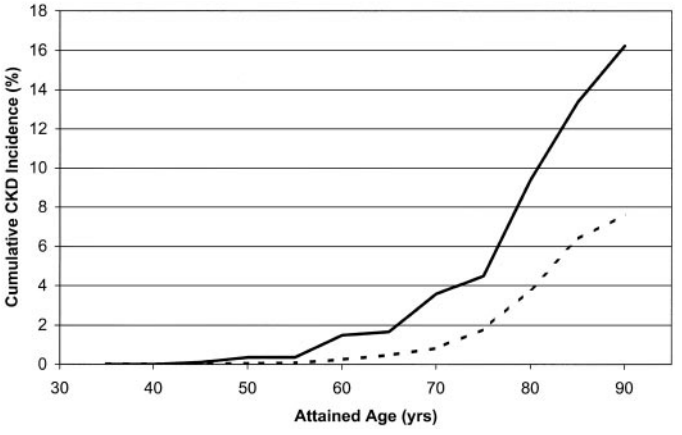

Figure 1. Cumulative incidence of chronic kidney disease (CKD), according to race and attained age, in the Second National Health and Nutrition Examination Survey (NHANES II), 1976 to 1992. Results are weighted to the general United States population. Solid line, African Americans; dashed line, whites. The cumulative incidence of CKD among African Americans was significantly higher than that among whites (log-rank test, $P < 0.001$).

Table 3. Excess risk of CKD among African Americans *versus* whites in relation to potentially modifiable risk factors[a]

Adjusted for	RR for African Americans (*versus* Whites)	Excess Risk Explained (%)[b]
Age and gender only	2.69 (1.50 to 4.82)	
Sociodemographic factors[c]	2.49 (1.33 to 4.67)	11.8
Lifestyle factors[d]	2.29 (1.31 to 4.01)	23.7
Clinical factors[e]	2.15 (1.18 to 3.92)	32.0
All risk groups[f]	1.95 (1.05 to 3.63)	43.8

[a] RR, relative risk. Values in parentheses are 95% confidence intervals. All models were adjusted for age and gender.
[b] Calculated using the formula $(RR_{age,\ gender} - RR_{age,\ gender,\ factor})/(RR_{age,\ gender} - 1)$.
[c] Sociodemographic factors include poverty status, education, and marital status.
[d] Lifestyle factors include smoking status, body mass index, alcohol use, and physical activity.
[e] Clinical factors include diabetes mellitus, hypertension, systolic BP, cardiovascular disease history, and serum cholesterol levels.
[f] Adjusted for lifestyle, clinical, and sociodemographic variables.

Americans developing CKD (Table 3). The addition of lifestyle variables to the base model reduced the excess risk by 23.7%, whereas the addition of clinical factors to the base model substantially reduced it by 32.0% (Table 3). In fact, simply adjusting for diabetes mellitus and hypertension in the base model reduced the excess risk by 35.3% (data not shown). Simultaneously controlling for all variables yielded a 43.8% reduction in the excess risk and attenuated the RR of CKD among African Americans; however, the excess risk remained statistically significant (RR = 1.95; 95% CI, 1.05 to 3.63).

Effect Modification by Age

Testing for possible interactions between race and other variables revealed age to be a significant modifier of the association between race and CKD ($P = 0.04$). African-American adults of age 30 to 59 yr at baseline demonstrated a 5.4-fold higher risk of CKD, compared with whites of similar ages, whereas those of age 60 to 74 yr at baseline exhibited a RR of 1.61 (95% CI, 1.04 to 2.49), after adjustment for age and gender (Figure 2). Compared with the overall population, adjustment for lifestyle or clinical factors explained similar proportions of the excess risk among African-American adults of age 30 to 59 yr or 60 to 74 yr. In contrast, sociodemographic factors attenuated the risk among African Americans of age 60 to 74 yr by 37.7% but did not account for any excess risk of CKD among African Americans of age 30 to 59 yr. Adjustment for lifestyle and clinical factors alone explained similar percentages of the excess risk of CKD among African Americans of age 30 to 59 yr and those of age 60 to 74 yr (40.9% *versus* 50.8%). Simultaneous adjustment for sociodemographic, lifestyle, and clinical factors decreased the RR among African

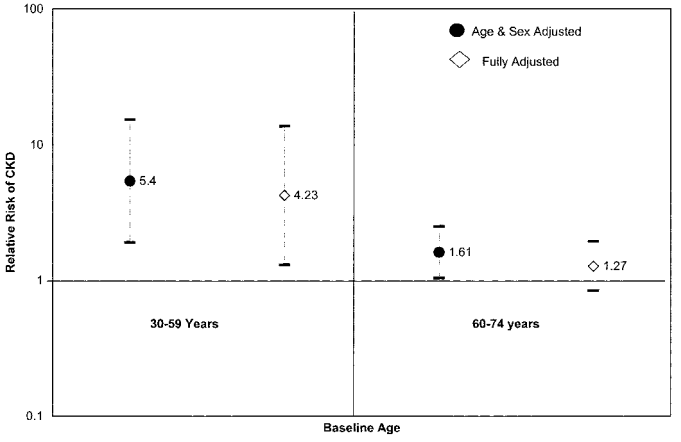

Figure 2. Adjusted relative hazard of CKD for African-American *versus* white adults in NHANES II, stratified according to age at baseline. All comparisons are African Americans *versus* whites. The fully adjusted model included age, gender, diabetes mellitus, hypertension, history of cardiovascular disease, serum cholesterol levels, smoking, poverty status, education, marital status, obesity, and alcohol use.

Americans in both age groups (Figure 2), with the racial disparity in CKD risk remaining statistically significant for the younger age group (RR = 4.23; 95% CI, 1.30 to 13.74).

Subsidiary Analyses

Further analyses of the entire cohort were performed by excluding individuals with <5 yr of follow-up monitoring, to assess the effects of early events on the observed associations between race and CKD. In these analyses, an almost identical attenuation of the excess risk among African Americans was observed after adjustment for all risk factors (39%). Finally, analyses limited to those without missing serum creatinine and urinalysis measurements, with adjustment for GFR, yielded similar patterns of percentages of excess risk explained by given sets of modifiable factors (as listed in Table 3), with the estimated risks explained being slightly higher (*i.e.*, 35% sociodemographic, 12% lifestyle, 35% clinical, and 51% all factors).

Discussion

Our results confirm that African-American adults in the United States develop CKD at rates far exceeding those for white adults, particularly during middle age. Much of the racial disparity in CKD in the United States is explained by potentially modifiable factors such as diabetes mellitus and hypertension. However, most of the excess risk of CKD experiences by African Americans remains unexplained by traditionally measured risk factors. Strengths of this study that lend weight to these conclusions include a nationally representative, population-based cohort, with comprehensive, individual-level, exposure measurements obtained before the development of CKD, and virtually complete outcome ascertainment.

Results of this study are consistent with previous findings of a higher risk of CKD among African Americans. Since 1977, when Easterling first documented a 3.8-fold higher risk of CKD among African Americans *versus* whites, using the Michigan ESRD registry, there have been many studies focusing on African-American/white differences in CKD (2–11,19,23). All of those studies have documented an excess risk for African Americans, compared with whites, with estimates of the association ranging from 1.9 to 7.4. Most of those studies have been limited by reliance on data from the United States Renal Data System or other ESRD registries, which lack information on potential explanatory factors (5,7,9,10,19,36–39).

Four prospective studies used geographically aggregated measures of exposure as indirect estimates of individual-level measures (2,3,8,11). For example, Whittle *et al.* (8) used prevalence estimates of explanatory factors (such as hypertension and diabetes mellitus) obtained from a regional survey to examine the racial differences in CKD risk. They observed that adjustment for age, prevalence and severity of hypertension, diabetes mellitus, and level of education in the population reduced the risk of hypertensive ESRD in African-American areas, but the racial association remained strong and statistically significant.

Although they had individual measurements of chronic medical conditions and behaviors at baseline, Brancati *et al.* (11) and Klag *et al.* (3) lacked individual measurements of income for Multiple Risk Factor Intervention Trial screenees, which hindered accurate quantification of the effects of socioeconomic factors on the individual risk of kidney disease. Nevertheless, those groups both concluded that higher BP, lower income, and higher prevalence of diabetes mellitus and hypertension among African-American adults explained some of the racial differences in CKD risk, with a significant amount remaining unexplained.

Like Rostand *et al.* (36) and Lopes *et al.* (20,37,38), we observed that racial differences in CKD risk were modified by age, with middle-age adults (30 to 59 yr) exhibiting a greater racial disparity than older adults (60 to 74 yr). The reasons for this age interaction are unclear; however, previous authors have speculated regarding accelerated kidney damage attributable to poorer control of BP, lower potassium intake, sustained higher levels of psychologic stress, and underdeveloped kidneys (38).

In our study, the large residual excess risk observed could possibly be explained on the basis of suboptimal measurement of exposures in NHANES II. In particular, factors such as BP were measured on only one occasion, which could result in an underestimation of the strength of the attenuation between race and CKD. Additionally, better characterization of potentially modifiable risk factors such as diabetes mellitus (*e.g.*, using hemoglobin A-1c levels) might have yielded greater reductions in the excess risk. By grossly assessing socioeconomic status (using education and poverty levels), we might have inadequately adjusted for the local environment or access to and quality of health care. It is possible that these factors in combination could account for all of the residual excess risk among African Americans.

Another potential explanation for the unexplained excess risk among African Americans might be unmeasured environmental, behavioral, sociocultural, or developmental factors that were beyond the scope of NHANES II data. Literature reports suggest that undernutrition in fetal life imparts a higher risk of CKD in adulthood (40,41). Because African Americans exhibit much higher rates of low birth weights, compared with whites (42), and low birth weights are associated with kidney underdevelopment, the low birth weight theory has been advanced to help explain the racial differences in CKD rates (13). Additionally, African Americans are more likely to be exposed to occupational and environmental toxins such as lead (43), to experience viral infections (44), and to have less access to preventive medical care, as well as being referred to treatment for CKD late in the course of their disease (45). Enhanced susceptibility of African-American kidneys to injury resulting from hypertension (46,47) and racial differences in renal vascular hemodynamics (48,49) have also been cited as explanations for the racial disparity in CKD risk.

In addition to previously mentioned limitations related to the assessment of exposures, other limitations deserve comment. First, because NHANES II was restricted to the noninstitutionalized population, the absolute risks of kidney disease among whites and especially African Americans were likely underestimated (15,50,51). Second, the NHANES II Mortality Study determined vital status only for adults of age 30 to 74 yr at baseline, limiting inferences to the middle-age United States population. Third, race was determined not by self-report but by interviewer assessment, leading to potential misclassification of African Americans, Hispanics, and Native Americans as whites. A number of studies have demonstrated discrepancies between the interviewer-observed race and the self-reported race, with approximately 6% of persons self-identifying as African American being classified as white (52,53). Fourth, renal function was not assessed at baseline times for all participants. Therefore, we could not completely establish whether renal insufficiency was already present in some of the subjects who later developed CKD.

Finally, the use of passive follow-up techniques without social security numbers to determine vital status and treatment for ESRD might have led to some misclassification of outcomes. Previous studies that evaluated the effectiveness of the National Death Index and the Social Security Administration Death Master Files suggested that underascertainment of vital status occurs more commonly for African Americans than for whites (27,54,55), whereas other work proposed that there is no racial difference in ascertainment (56). Additionally, whites are more likely to have multiple causes of death coded on their death certificates (57), which could increase the ascertainment of CKD events for whites while underestimating the number for African Americans. Because the majority of our events were deaths related to kidney disease, this differential ascertainment could significantly affect our results. In addition to deaths, only individuals treated through the Medicare program were included in our analyses. Therefore, possible racial differences in enrollment in Medicare *versus* private insurance could affect our findings. We might have underestimated the true racial disparity in CKD by relying on these methods. Lastly, our use of CKD attributable to any cause as the primary outcome instead of cause-specific CKD reduced the possibility of misclassification bias, which is known to be influenced by race (58,59).

In light of the exorbitant costs, to individuals and to society, of treatment for CKD, the identification of modifiable risk factors for CKD is an important public health priority, as indicated in Healthy People 2010. Our results suggest that interventions aimed at reducing the racial disparity in CKD risk should focus on primary prevention and improved treatment of diabetes mellitus and hypertension, lifestyle modification, and elimination of health disparities attributable to socioeconomic status. Our data further suggest that the benefits of these improvements might be greatest for middle-age adults. Further prospective studies are needed to identify novel environmental, developmental, and genetic causes of the large African-American/white disparity in the incidence of CKD.

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