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Trajectories of Kidney Function Decline in Young Black and White Adults With Preserved GFR: Results From the Coronary Artery Risk Development in Young Adults (CARDIA) Study

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Abstract

Background—Strong racial discrepancies in end-stage renal disease exist. Whether there are race differences in kidney function loss in younger, healthy persons is not well established.

Study Design—Longitudinal.

Setting & Participants—3348 Black and White adults with at least two measures of cystatin C-based estimated glomerular filtration rate ($eGFR_{cys}$) at scheduled Coronary Artery Risk Development in Young Adults (CARDIA) examinations (Years 10, 15, 20).

Predictor—Race.

Outcomes & Measurements—We used linear mixed models (LMM) to examine race differences in annualized rates of $eGFR_{cys}$ decline, adjusting for age, sex, lifetime exposure to systolic blood pressure above 120mmHg, diabetes, and albumin-creatinine ratio. We used Poisson regression to compare racial differences in rapid decline ($eGFR_{cys}$ decline >3% per year) by study period (10–15 years after baseline exam defining period 1 and >15–20 years after baseline exam defining period 2).

Results—Mean age was 35 ± 3.6 (SD) years, mean $eGFR_{cys}$ was 110 ± 20 ml/min/1.73m^2 for Blacks and 104 ± 17 ml/min/1.73m^2 for Whites at baseline. For both Blacks and Whites, $eGFR_{cys}$ decline was minimal at younger ages (<35 years) and $eGFR_{cys}$ loss accelerated at older ages. However, acceleration of $eGFR_{cys}$ decline occurred at earlier ages for Blacks than Whites. Blacks

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had somewhat faster annualized rates of decline compared with whites, but differences were attenuated after adjustment in period 1 (0.13 ml/min/1.73m^2 per year faster; p=0.2). In contrast, during period 2, Blacks had significantly faster annualized rates of decline, even after adjustment (0.32 ml/min/1.73m^2 per year faster; p=0.003). Prevalence of rapid decline was significantly higher among Blacks vs. Whites with prevalence rate ratios of 1.31 (95% CI, 1.04–1.63) for period 1 and 1.24 (95% CI, 1.09–1.41) for period 2. Differences were attenuated after full adjustment: adjusted prevalence rate ratios were 1.20 (95% CI, 0.95–1.49) for period 1 and 1.10 (95% CI, 0.96–1.26) for period 2.

Limitations—No measured GFR.

Conclusions—eGFR$_{cys}$ decline differs by race at early ages, with faster annualized rates of decline among blacks. Future studies are required to explain observed differences.

End stage renal disease (ESRD) affects Black Americans disproportionately, and reasons for the high rates of ESRD in Blacks remain unclear. Prior literature suggested that Blacks may experience faster progression from established chronic kidney disease (CKD), defined as an estimated glomerular filtration rate (eGFR) <60 ml/min/1.73m^2, to ESRD compared with Whites.(1, 2) Recent findings from the Coronary Artery Risk Development in Young Adults (CARDIA) Study showed that Blacks may have a higher risk of developing CKD.(3) Moreover, in a large sample of middle-aged adults with eGFR >60 ml/min/1.73m^2, Blacks had faster rates of kidney function decline compared with Whites, and these differences were not fully explained by traditional risk factors.(4)

While these findings suggest that race differences in kidney function decline may happen earlier in the disease course than previously thought, population trajectories of kidney function loss in young, healthy adults with preserved eGFR are not well established.(5) Moreover, racial differences in these trajectories have not been fully examined. In part, studies have been limited by the fact that repeated 24-hour urine collections over long periods are not feasible in large, contemporary epidemiological studies. In addition, serum creatinine, the current clinical standard, is a function of muscle mass which differs by race/ethnicity and may result in estimates of GFR biased by race. (6) Cystatin C is an alternative filtration marker that is not influenced by muscle mass, age or race, and, in some studies it has shown to be a more sensitive marker for GFR decline when the GFR is preserved.(7, 8) Cystatin C also has stronger and more linear associations with adverse outcomes, and improves CKD classification and risk stratification compared with creatinine.(9–11)

Therefore, we have designed this study to (1) evaluate trajectories of kidney function loss in a population of young, healthy Blacks and Whites with preserved kidney function; (2) investigate whether traditional kidney disease risk factors explain possible racial differences in early kidney function decline; and (3) identify racial differences and risk factors for rapid kidney function decline, defined as an eGFR loss of >3% ml/min/1.73m^2 per year. Understanding whether racial differences in the trajectories of kidney function loss are detectable early is paramount in developing adequate prevention strategies to reduce the overall burden and racial disparities of CKD.

METHODS

Participants

We included participants from the CARDIA Study. CARDIA is a prospective cohort study sponsored by the National Heart, Lung and Blood Institute designed to study early determinants of cardiovascular disease. Detailed methods for CARDIA have been published previously.(12) Briefly, CARDIA recruited 5,115 Black and White persons aged 18–30 years between 1985 and 1986 from four sites in the United States (Birmingham, AL,

Chicago, IL, Minneapolis, MN, and Oakland, CA). After the first examination, subsequent visits occurred at approximately 5-year intervals. Urinary albumin and creatinine were measured at years 10, 15 and 20 visits (corresponding to years 1995–1996, 2000–2001 and 2005–2006) as a part of the CARDIA protocol. Cystatin C was measured on stored sera from these exam visits as a part of an ancillary study sponsored by the National Institute of Diabetes and Digestive and Kidney Diseases. All appropriate institutional review boards approved this study.

Of 4,376 participants with a visit at year 10 or later, 4,311 had at least one cystatin C measurement. We excluded 29 persons with initial cystatin C–based eGFR ($eGFR_{cys}$) <60 ml/min/1.73m^2, and an additional 934 who did not have 2 sequential measures of cystatin C at years 10 and 15 or years 15 and 20. Finally, we excluded 14 persons with missing covariate data, for a total sample size of 3,334.

Measurement of Kidney Function

Kidney function was measured by cystatin C. Cystatin C was measured at exam years 10, 15 and 20 from frozen samples by nephelometer using the N Latex cystatin C kit (Dade Behring, now Siemens). All cystatin C measurements were performed simultaneously at the University of Minnesota. The coefficient of variation (CV) for cystatin C was 4.0%. The assay was calibrated for drift by re-assaying frozen serum samples from 48 participants using the Gentian Cystatin C Immunoassay and calibrated to international standards. Initial cystatin C assays were, on average, 12% lower prior to calibration. We estimated eGFR using cystatin C ($eGFR_{cys}$) using the equation $eGFR_{cys} = 76.7x \text{ cystatin } C^{-1.19}$ which was derived from a large, pooled cohort with measured GFR.(10) We specifically chose to use serum cystatin C (rather than creatinine) to estimate GFR in this population because it is not biased by age or race, our predictors of interest. In addition, measures were performed over the same time interval, in the same laboratory, from frozen samples in CARDIA, which improves our ability to interpret trajectories over time. Findings on race differences on incident CKD in CARDIA using serum creatinine have been previously published.(3)

Our main outcomes of interest included mean population trajectories of $eGFR_{cys}$, annualized rates of $eGFR_{cys}$ change (continuous) and rapid kidney function decline (dichotomous). We defined rapid kidney function decline as a loss of >3% ml/min/1.73m^2 per year. This definition was selected because it roughly corresponds to the highest decile of kidney function decline in the cohort, and prior work from our group has shown that declines in this range are associated with adverse cardiovascular outcomes.(13, 14)

Covariates

Age, gender and race were ascertained by self-report using standardized questionnaires. At each visit, blood pressure measurements were taken by centrally trained staff using sphygmomanometry and following guidelines. We estimated a lifetime exposure to systolic blood pressure (SBP) >120 mmHg as the area under the curve (AUC) of years exposed to SBP >120 mmHg (SBP-AUC).(15) Blood was collected at each study visit and stored at −70°C until measurements were performed. Both fasting glucose and a fasting lipid profile (triglycerides, total cholesterol, and high-density lipoprotein [HDL] cholesterol) were measured using a standard laboratory technique. Low-density lipoprotein (LDL) cholesterol was estimated using the Friedewald equation. The presence of diabetes was defined as fasting blood glucose ≥126 mg/dL or use of insulin and/or oral hypoglycemic agents. Urine albumin and creatinine were expressed as the albumin-creatinine ratio (ACR) in mg/g. Albuminuria was defined as ACR ≥30 mg/g.

Statistical Analyses

We first compared characteristics of Black and White participants at the beginning of our observations period (CARDIA exam Year 10) using *t*- and Chi-square tests as appropriate. Then, we estimated the trajectory of population mean $eGFR_{cys}$ between ages 25 and 55 years (which correspond to the age range of participants over the 10-year follow-up period of this study) using linear mixed models to account for repeated measures of $eGFR_{cys}$. To maximize the age span of these trajectories, each individual could contribute up to three data points to these trajectories. For example, each person could contribute a point corresponding to their age at the time of each $eGFR_{cys}$ measure. This approach allows for investigation of a larger age span, and has been previously used in large cohort studies.(16) In these models, age effects were captured using race-specific 5-knot restricted cubic splines. Individual departures from the trajectory of the population mean were modeled using random intercepts and cubic spline components. We included age at the first $eGFR_{cys}$ measurement as a covariate to rule out cohort effects. The race-specific averages were also adjusted for gender, SBP-AUC, diabetes and ACR. We present population mean $eGFR_{cys}$ graphically, by age, for Blacks and Whites separately, where a total of 9033 $eGFR_{cys}$ measurements over 28,495 person-years are included. We tested for race differences in the slopes of the population trajectories at ages 30 through 50.

Next, we used linear mixed models to estimate average annualized rates of decline in $eGFR_{cys}$ by race, with a knot at year 15 and controlling for age. Guided by the plotted trajectories, we tested for heterogeneity in rates of $eGFR_{cys}$ decline by study period ($p < 0.001$). Thus, we present all analyses stratified by period: the year 10–15 (herein period 1) and year 15–20 visits (herein period 2). To examine the effect of traditional risk factors and albuminuria in potential race differences in rates of kidney function decline, we constructed nested models. The first model adjusted for age and sex. Model 2 adjusted for diabetes, and for the cumulative exposure to systolic blood pressure of >120 mmHg over the CARDIA period (SBP-AUC). Finally, model 3 added ACR to the fully adjusted model. The ACR was handled as a time dependent covariate, but once a person developed albuminuria, that individual was considered as having albuminuria for the remainder of follow-up.

We conducted three sensitivity analyses. First, we truncated at $eGFR_{cys}$ of 150 ml/min/$1.73m^2$, as these may represent supraphysiologic values. Second, we used log-transformed $eGFR_{cys}$ rather than the absolute value; this approach has been suggested to reduce the influence of large changes at the high GFR values. Third, we assessed heterogeneity of the race differences by baseline $eGFR_{cys}$, stratified at >120, 90–120, and <90 ml/min/$1.73m^2$.

Finally, we used generalized estimating equation Poisson models to compare the proportions of Blacks and Whites with annualized declines in $eGFR_{cys}$ of at least 3% in the intervals between consecutive visits. We used Poisson rather than logistic models in order to obtain relative prevalence rather than odds ratios. All analyses were conducted using Stata Version 12.2 (StataCorp LP, College Station, TX:), using the “xtmixed” command for the linear mixed models and “xtpoisson” for the rapid loss analyses.

RESULTS

Cohort Characteristics

Among 3,348 CARDIA participants included in this study, mean age at baseline (CARDIA year 10) was 35 ± 3.6 (standard deviation) years, 288 (9%) had hypertension, and 225 (7%) had diabetes. Blacks were more likely to have diabetes, hypertension, and a higher prevalence of albuminuria. The $eGFR_{cys}$ at baseline was approximately 6 ml/min/$1.73m^2$ higher for Blacks compared with Whites (Table 1).

Trajectories of $eGFR_{cys}$ Decline by Race

In figure 1, we present the trajectories of adjusted mean $eGFR_{cys}$ as a function of age, for Blacks and Whites separately. Each point on the trajectories can be interpreted as the adjusted mean $eGFR_{cys}$ among Blacks and Whites at a particular age. We found that on average, Blacks had higher $eGFR_{cys}$ at the start of the follow-up period compared with Whites. For both Blacks and Whites, the change in $eGFR_{cys}$ was minimal at younger ages (<35 years), and $eGFR_{cys}$ loss accelerated at older ages. However, the point of inflection at which $eGFR_{cys}$ decline accelerates occurred, on average, almost a decade earlier for Blacks compared with Whites (Figure 1). We also tested whether there was a difference in the slope of decline by race. We found that on average, young Blacks had statistically significantly steeper rates of decline compared with Whites starting at age 35 years, but differences by race were attenuated at older ages. (Figure 1)

Race Differences in Annualized $eGFR_{cys}$ Decline

In period 1, in age- and sex-adjusted models, the magnitude of the mean change in $eGFR_{cys}$ was small for both Blacks and Whites. In models adjusted for demographic factors, Blacks had significantly faster rates of decline compared with Whites. However, race differences in annualized decline were significantly attenuated after adjustment for traditional risk factors and albuminuria. (Table 2)

During period 2, the magnitude of the observed decline in kidney function was larger for both Blacks and Whites. In contrast to period 1, Blacks had faster rates of kidney function decline, and these differences persisted after adjustment for diabetes, SBP-AUC and ACR (Table 2).

Race Differences in Rapid Kidney Function Decline

In period 1, 11% of Black participants had rapid decline in kidney function, defined as $eGFR_{cys}$ decline >3% per year, compared with 8% of Whites. In period 2, 26% of Blacks had rapid decline versus 21% of Whites. Among all persons with rapid decline (>3% per year), 90% had absolute rates of decline >3 ml/min/1.73m^2 per year, and the average rate of decline was 5.0 ± 2.4 ml/min.1.73m^2 per year. In age- and sex-adjusted models, Blacks had a 31% and 24% higher risk of rapid decline in periods 1 and 2, respectively. Adjustment for diabetes, SBP-AUC, and ACR attenuated the statistical significance of the observed differences (Table 3).

Sensitivity Analyses

First, we truncated at $eGFR_{cys}$ 150 ml/min/1.73m^2. Findings did not materially differ. In fully adjusted models, Blacks had a 0.10 (95% confidence interval [CI], −0.09 to 0.30) ml/min/1.73m^2 per year faster rate of decline compared with Whites in period 1, and this difference was 0.27 (95% CI, 0.08 to 0.47) ml/min/1.73m^2 faster in period 2. When we used log $eGFR_{cys}$, findings were also similar. In fully adjusted models, $eGFR_{cys}$ in Blacks declined 0.18% (95% CI, −0.04% to 0.41%) faster in period 1 and 0.24% (95% CI, 0.03% to 0.45%) in period 2, compared with Whites. We also found that the association of race and kidney function decline did not differ by baseline $eGFR_{cys}$ (P value for interaction >0.14 for both linear and rapid decline analyses).

DISCUSSION

In this large cohort of young Black and White persons with largely preserved kidney function, we found that, on average, decline in eGFR is minimal prior to age 35 years, and that rates of decline accelerate at older ages. However, we found important racial differences in these kidney function trajectories. Specifically, prior to age 35, $eGFR_{cys}$ decline was

fairly similar by race. After age 35, $eGFR_{cys}$ decline was significantly steeper for Blacks compared with Whites, and this decline started at earlier ages among Blacks. Moreover, we found that Blacks had faster rates of annualized $eGFR_{cys}$ decline, and that they also had higher relative prevalence of rapid kidney function decline. Accounting for diabetes, systolic blood pressure and albuminuria explained some, but not all of the observed differences.

Our observations of the population trajectories are in accordance with prior data using repeated 24 hour urine collections for creatinine clearance.(5) To our knowledge, this is the first contemporary report evaluating population trajectories in young, healthy adults with preserved kidney function. Our findings highlight the concept of “renal reserve” in which damage to the kidney may be accruing across the life-course, but the eGFR does not start to decline until a significant amount of damage is present.(12, 17) Therefore, evaluation of novel biomarkers that can identify persons with kidney injury while the eGFR is preserved is required in order to capture persons at risk.

Our results support a recent CARDIA report that Blacks had a higher incidence of CKD using eGFR by creatinine.(3) We also extend findings from another prior study in a middle-aged population(4) to show that these race differences in population trajectories are detectable early in life, and while the eGFR *is still* preserved. Our findings challenge the dogma that race differences in ESRD incidence are primarily attributable to progression of established CKD,(1, 2) and they offer hope that prevention strategies may be effective if implemented very early.

To that end, our findings that adjustment for the most established CKD risk factors explained some of the observed race differences in kidney function decline and rapid decline have several important implications. First, the attenuation by easily ascertainable factors such as systolic blood pressure and albuminuria offers guidance for future work to investigate strategies for identifying young adults at high risk for kidney function decline. Second, our findings suggest that additional exploration is required to understand whether primary prevention treatment strategies currently advocated for middle-aged adults (such as treatment of stage I hypertension) should be considered in young adulthood. Our study represents the largest contemporary investigation of population trajectories in young Blacks and Whites with preserved kidney function. Moreover, we were able to examine trajectories because we used an endogenous filtration marker (cystatin C) measured at the same laboratory, at the same time, from frozen samples. However, we must note several important limitations. We did not have direct measures of GFR. However, this is impractical in large epidemiological studies. We must also note that eGFR estimating equations have not been adequately validated in young persons with preserved GFR. We did not investigate individual trajectories. Rather, our analyses using repeated measures over time (up to 3 per individual) allow the trajectories to be interpretable as *typical* patterns for Blacks and Whites in CARDIA. Our sample size of persons above age 45 was small, limiting race comparisons of trajectories at older ages. Future studies should account for social, demographic and health care factors that may explain some of the observed differences.

In summary, we found that trajectories of kidney function loss follow a similar pattern in Blacks and Whites with minimal changes at younger ages and faster decline starting later in life. However, the $eGFR_{cys}$ decline for Blacks becomes steeper almost a decade earlier, and Blacks were at higher risk for $eGFR_{cys}$ decline and rapid decline. Adjustment for traditional CKD risk factors explained some but not all observed differences. Future studies are required to identify persons at high risk in order to design effective prevention strategies.

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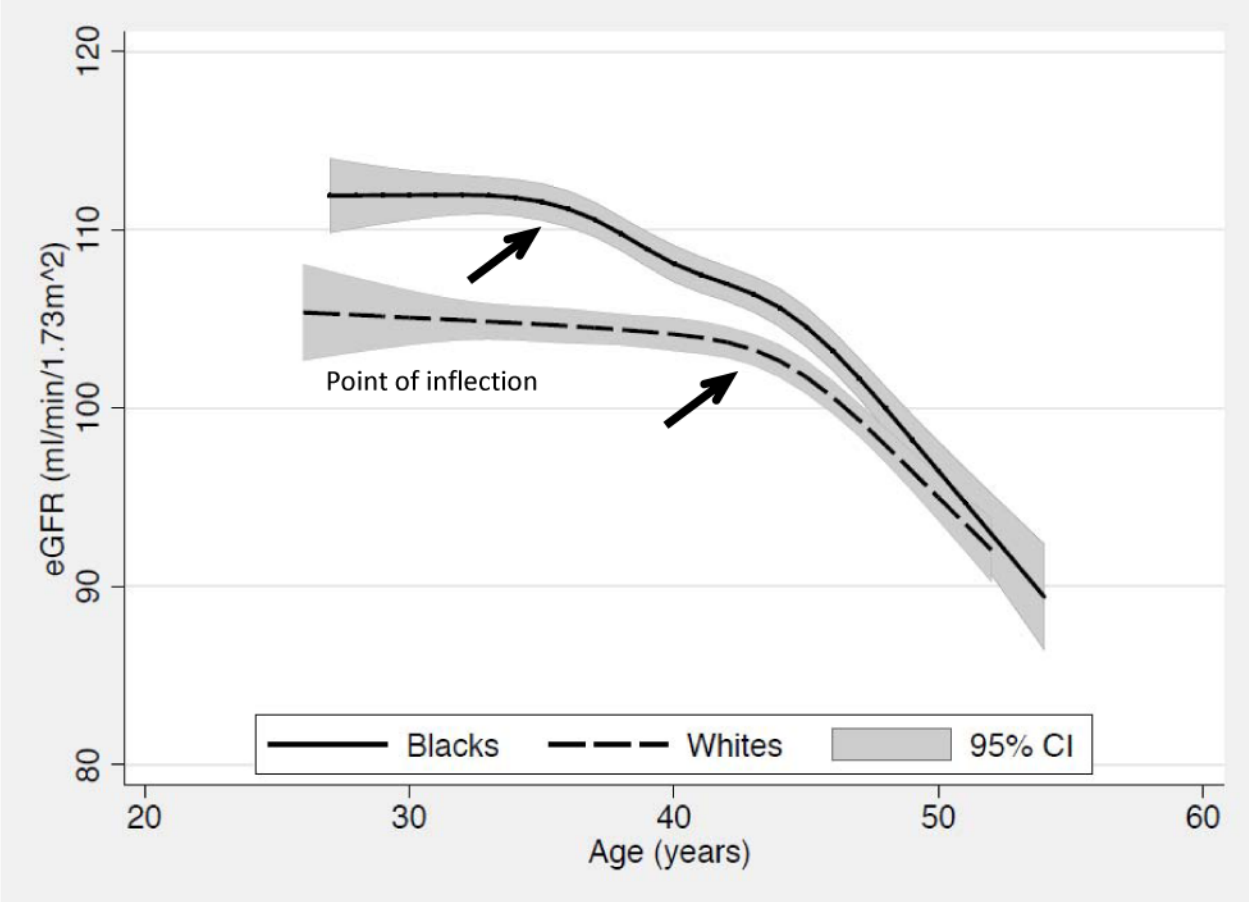

Age	P Value for Difference in Slope by Race
30	0.7
35	0.06
40	<0.001
45	0.09
50+	0.2

Figure 1.
Trajectories of mean $eGFR_{cys}$ among Blacks and Whites by Age. $eGFR_{cys}$, cystatin C–based estimated glomerular filtration rate.

Table 1

Characteristics of CARDIA Study Participants at Year 10 by Race

Characteristic	White	Black	P Value
No. of participants	1,816	1,518	
Age (y)	36 (3)	35 (4)	<.0001
Female sex	954 (53)	887 (59)	<0.001
Diabetes	94 (5)	131 (9)	<0.001
Hypertension	75 (4)	213 (14)	<0.001
Fasting Glucose (mg/dL)	87 (16)	89 (22)	0.002
LDL Cholesterol (mg/dL)	111 (31)	110 (34)	0.7
HDL Cholesterol (mg/dL)	50 (14)	52 (14)	<0.001
$eGFR_{cys}$ (mL/min/1.73 m^2)	104 (17)	110 (20)	<0.001
ACR (mg/g)	4.1 [2.9–6.2]	4.1 [2.8–7.5]	0.09
ACR ≥30 mg/g	48 (3)	99 (7)	<0.001
SBP (mm Hg)	108 (11)	114 (14)	<0.001
DBP (mm Hg)	71 (9)	75 (11)	<0.001

Note: Values for categorical variables are given as number (percentage); values for continuous variables, as mean ± standard deviation or median [interquartile range]. Conversion factor for units: glucose in mg/dL to mmol/L, ×0.05551; LDL and HDL cholesterol in mg/dL to mmol/L, ×0.02586.

CARDIA, Coronary Artery Risk Development in Young Adults; LDL, low-density lipoprotein; HDL, high-density lipoprotein; $eGFR_{cys}$, cystatin C–based estimated glomerular filtration rate; ACR, albumin-creatinine ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 2

Rate Differences in $eGFR_{cys}$ decline of Blacks Vs Whites in CARDIA Study by Period

	Age- and Sex-Adjusted $\Delta eGFR_{cys}$ (mL/min/ 1.73 m^2 per y)	β coefficients of models comparing Blacks to Whites*		
		Model 1: adj for Demographics	Model 2: Model 1 + adj for SBP-AUC & Diabetes	Model 3: Model 2 + adj for microalbuminuria
Period 1 (years 10–15)				
Whites	0.19 (0.05 to 0.33)	(referent group)	(referent group)	(referent group)
Blacks	−0.02 (−0.17 to 0.14)	0.21 (0.00 to 0.42)	0.14 (−0.07 to 0.35)	0.13 (−0.08 to 0.34)
Period 2 (years 15–20)				
Whites	−1.19 (−1.05 to −1.32)	(referent group)	(referent group)	(referent group)
Blacks	−1.58 (−1.42 to −1.74)	0.39 (0.18 to 0.60)	0.32 (0.11 to 0.53)	0.32 (0.11 to 0.54)

Note: values shown are in parentheses are 95% CIs.

*Estimates represent β coefficients of models comparing Blacks to Whites. These can be interpreted in ml/min/1.73 m^2 per year faster decline (for Blacks) compared to referent (Whites)

adj: adjusted; CI, confidence interval; CARDIA, Coronary Artery Risk Development in Young Adults; $eGFR_{cys}$, cystatin C–based estimated glomerular filtration rate; SBP-AUC= area under the curve of years exposed to systolic blood pressure >120 mm Hg

Table 3

Prevalence Rate Ratios for Rapid Decline in Blacks Vs Whites in CARDIA Study

	No. (%) with Rapid Decline	Prevalence Rate Ratios		
		Model 1: adj for Demographics	Model 2: Model 1 + adj for SBP-AUC & Diabetes	Model 3: Model 2 + adj for microalbuminuria
Period 1 (years 10–15)				
Whites	138 (8%)	1.00 (reference)	1.00 (reference)	1.00 (reference)
Blacks	148 (11%)	1.31 (1.04 – 1.63)	1.22 (0.97 – 1.53)	1.20 (0.95 – 1.49)
Period 2 (years 15–20)				
Whites	349 (21%)	1.00 (reference)	1.00 (reference)	1.00 (reference)
Blacks	335 (26%)	1.24 (1.09 – 1.41)	1.12 (0.97 – 1.28)	1.10 (0.96 – 1.26)

NOTE: Rapid decline is defined as >3% ml/min/1.73 m^2 per year.

adj: adjusted; CI, confidence interval; CARDIA, Coronary Artery Risk Development in Young Adults; SBP-AUC= area under the curve of years exposed to systolic blood pressure >120 mmHg