Rates of Cardiovascular Disease and CKD Progression in Young Adults with CKD across Racial and Ethnic Groups

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Key Points
- The extent to which racial and ethnic disparities noted in older adult populations with CKD are present in young adulthood is unknown.
- Young adults with CKD who identify as Black or Hispanic have a higher burden of cardiovascular risk factors, some of which are modifiable.
- Rates of cardiovascular disease and CKD progression are higher in young adults who identify as Black or Hispanic.

Abstract

Background Significant racial and ethnic disparities in cardiovascular (CV) and kidney function outcomes in older adults with chronic kidney disease (CKD) have been reported. However, little is known about the extent to which these disparities exist in patients with CKD during the foundational period of young adulthood. The objective of this study was to determine risk factors and rates of CV disease and CKD progression in young adults with CKD across racial and ethnic groups.

Methods We studied all participants aged 21–40 years of age enrolled in the Chronic Renal Insufficiency Cohort (CRIC) Study (n=317). Baseline CV risk factors were described across racial and ethnic groups.

Results Outcomes included CV events or death (first incidence of heart failure, myocardial infarction, and stroke or death) and CKD progression (>50% decline in eGFR from baseline or end stage kidney disease [ESKD]). Incidence rate ratios (IRRs) were compared as a secondary analysis for participants identifying as Black or Hispanic with those identifying as White or another race and ethnicity. Adjusted models included age, sex, and per APOL1 high-risk allele. CV risk factors were higher in Black and Hispanic participants, including mean SBP, BMI, median UACr, and LDL. Black and Hispanic participants had higher incidence rates of HF (17.5 versus 5.1/1000 person-years), all-cause mortality (15.2 versus 7.1/1000 person-years), and CKD progression (125 versus 59/1000 person-years).

Conclusions In conclusion, we found a higher prevalence of CV risk factors, some modifiable, in young adults with CKD who identify as Black or Hispanic. Future strategies to ameliorate the racial and ethnic inequality in health outcomes earlier in life for patients with CKD should be prioritized.

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Introduction

In studies of older adults with CKD, the risks for cardiovascular (CV) disease and progression to ESKD are significantly higher for Black and Hispanic individuals compared with patients of other race and ethnicity (1–3). The reasons underlying these disparities are thought to be in part biologic from genetic variants such as APOL1, but also largely due to social determinants of health (4).

Health status in adulthood is centrally influenced by the experiences and conditions present at birth, during childhood, and through adolescence (5,6). Young adulthood (around 18–40 years of age) represents the summation of these early life circumstances and, as such, represents the foundation upon which individuals derive their health into adulthood. However, data on kidney and CV outcomes in young adults with CKD across race and ethnic groups remain limited.

Studies in young adults without CKD demonstrate racial and ethnic disparities in health care outcomes. In the CARDIA study, which enrolled young adults 18–30 years of age without CKD. Self-reported Black participants were more likely to have abnormal

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findings on echocardiogram, be diagnosed with heart failure (HF) before the age of 50, and experience an increased burden of HF over a lifetime compared with White participants (7–9). Moreover, the rate of GFR decline and CKD diagnoses was greater in Black participants compared with those who were White (1). More broadly, individuals who reported receiving higher rates of discrimination were more likely to have poor CV health, and many of the associations between race and ethnicity with CV disease (CVD) were attenuated by inclusion of socioeconomic indexes (7,10).

Study of clinical outcomes across racial and ethnic groups in young adults with CKD may identify an opportunity for interventions to reduce the disproportionate burden of adverse kidney and CV outcomes in this population before advancing to older age. Given the longevity of young-adult patients with CKD, any risk factors or disparities left undressed have the potential to compound over a lifetime. As such, the objective of our analysis was to describe the rates of, and risk factors for, CV events and CKD progression in young adults with CKD across racial/ethnic groups.

Materials and Methods

Study Population

We studied men and women aged 21–40 years with mild to moderate CKD enrolled in the Chronic Renal Insufficiency Cohort (CRIC) Study. In total, the CRIC Study enrolled 3939 participants ≥21 years of age between June 2003 and August 2008 at seven clinical centers across the United States. The CRIC Study enrolled patients with an eGFR of 20–70 ml/min per 1.73 m² using the Modification of Diet in Renal Disease study equation and excluded patients with New York Heart Association class III or IV HF. Additional details on study design, inclusion and exclusion criteria, and baseline characteristics of the participants have been detailed previously (11,12). We excluded all patients >40 years of age (N= 3622), leaving a study population of 317 young adults with CKD. All study participants provided written informed consent, and Institutional Review Boards at each of the participating sites approved the study protocol.

Primary Exposure

Race and ethnicity were based off participant self-identification. For regression models, participants were entered into two race and ethnicity categories based off previous work demonstrating worse outcomes for CKD patients of Black race or Hispanic ethnicity (2,3,13–16). Patients who self-identified as Black race or Hispanic ethnicity were combined into a higher-risk category, and participants who were White or of other race were combined into a lower-risk category. There was no assumption of common biologic determinants of health when combining participants who identified as either Black or Hispanic. Rather, these racial and ethnic groups were analyzed together to study broadly the magnitude of disparities starting in young adulthood within the confines of the sample size.

CV Outcomes

We utilized a composite outcome measure of CV events or death in our primary analysis, which included HF, myocardial infarction (MI), cerebrovascular events (CVA), and all-cause mortality. To identify the incidence of HF, MI, and CVA after the baseline visit, studied participants were queried every 6 months by alternating in-person and telephone visits. Relevant medical records were retrieved for review by at least two physicians to ascertain events of HF, MI, and CVA for all admissions (17–19). Death was identified through report from next of kin, review of hospital records, retrieval of death certificates or obituaries, and linkage with the Social Security Mortality Master File.

Kidney Function Outcomes

We utilized a composite CKD progression outcome. CKD progression was defined by the first incidence of ≥50% decline in eGFR from baseline or ESKD. Kidney function was assessed by centrally measured serum creatinine values at the baseline and annual research visits. eGFR was calculated using the CKD-EPI equation (20). Participants were defined as having a 50% decline in eGFR at the first visit with an eGFR that was <50% of the baseline value. ESKD, defined as receiving dialysis or kidney transplant, was determined by participant self-report/local clinical center ascertainment, or cross-linkage with the United States Renal Data System.

Covariates

All covariates used within the study analysis were collected at the baseline visit. Demographic and medical history variables were collected by participant interviews. Clinic blood pressure was measured by centrally trained staff using a standardized method in a quiet standardized setting per the CRIC protocol (21). This method has been described in greater detail previously (21). The mean of three seated resting blood pressure readings was used to define the systolic blood pressure (SBP) value for each visit.

Left ventricular hypertrophy (LVH) was defined by applying guideline-based definitions to echocardiographic measurements of left ventricular mass index (22). All echocardiograms were obtained using standardized practices per the CRIC protocol. Quality control and image interpretation were performed by the CRIC Central Echocardiography Laboratory at the University of Pennsylvania (23).

Urine albumin/creatinine ratios (UACr) reflect values from 24-hour urine collection. UACr values were missing for 24 participants. These data were missing completely at random. So, we performed multiple imputation when adding UACr as a covariable in adjusted models.

APOL1 G1, G2, and MYH9 haplotype-tagging single nucleotide polymorphisms were genotyped in Black participants. APOL1 genotyping in the CRIC cohort has been described in greater detail previously (13). Hispanic participants in CRIC did not have available APOL1 genotyping. So, for the purposes of this analysis, we assumed a 0% prevalence of two high-risk APOL1 alleles for this population (24). Studies have demonstrated that the true prevalence of two high-risk APOL1 alleles is around 0.5% in individuals of Hispanic ethnicity living in the United States (25).

Statistical Analyses

Participant characteristics were reported as mean and SD for normally distributed variables or as median and
interquartile range (IQR) for skewed variables. To assess the differences of CV risk factors across racial and ethnic groups, we utilized chi-squared and ANOVA to analyze categorical and continuous data, respectively.

For both the CV and CKD progression outcomes, a participant was assigned to have experienced that event at a certain time point with the first incidence of any of the component events. We evaluated CV events and CKD progression independently of one another, such that patients were not censored from one outcome if they met the criteria for the other. Participant follow-up in this study was censored at the time of death (for CKD progression only), withdrawal, lost to follow-up, or at the end of the follow-up period in 2018.

Incidence rates (IR) were calculated for CV events, HF, MI, stroke, death, and CKD progression for all participants and as stratified by participant race and ethnicity. All rates represent incidence per 1000 person-years.

In a secondary analysis, to compare differences in IR between participants identifying as Black or Hispanic participants with those who did not, we calculated unadjusted and adjusted incidence rate ratios (IRRs) and 95% confidence intervals (CI) for CV events and CKD progression using a Poisson generalized linear model with natural logarithm link function. Models were adjusted for biologic factors, including age, sex, and APOL (0, 1, or 2 high-risk alleles) status. These covariables were selected to control for imbalance between biologic risk factors between groups.

Kaplan–Meier estimates were used to generate survival curves estimating the probability of event-free survival for both CV events and CKD progression assessed independently of each other. The curves were stratified by participant racial/ethnic self-identity.

Statistical analyses were conducted using IBM SPSS Statistics for Windows v26 (IBM Corp., Armonk, NY) or R v3.6.2 (The R Foundation for Statistical Computing, Vienna, Austria).

Results
Baseline Characteristics

Young-adult study participants had a mean±SD age of 33±5 years. The baseline characteristics of young-adult CKD participants are shown in Table 1. Young adults had a mean±SD SBP of 120±20 mmHg. Before baseline, 3%, 4%, and 2% had a documented history of MI, CHF, and stroke, respectively. Mean eGFR was 51±15 ml/min per 1.73 m², and median 24-hour albumin/creatinine was 335 mg/g (IQR 37–1398 mg/g).

Prevalent CVD and CV Risk Factors among Young Adults with CKD

Overall, there were significant differences in sociodemographic risk factors, CV risk factors, and kidney function across racial and ethnic groups (Table 2). In terms of socioeconomic indexes, the proportion of participants with an income of more than $50,000/year differed by race and ethnicity (White/other: 50%; Black: 23%; Hispanic: 9%). Similarly, proportions with a maximum educational level of high school/diploma/General Educational Development/ or lower significantly varied, depending on race and ethnicity (P<0.001). Hispanic participants had the lowest mean eGFR and highest median UACr and LVH. SBP was highest in Hispanic participants (129±24 mm Hg) compared with Black (125±22 mm Hg) or White participants (113±14 mm Hg; Table 2). Body mass index was also not balanced across racial and ethnic groups (P<0.001). White participants had the lowest prevalence, or lowest-risk values, for all CV risk factors. Frequency of antihypertensive

| Table 1. Baseline characteristics of young-adult chronic kidney disease participants in the Chronic Renal Insufficiency Cohort Study |
|---------------------------------------------------------------|
| **Characteristics** | **Chronic Renal Insufficiency Cohort Age <40 Years (N=317)** |
| **Demographics** |  |
| Age, yr, mean±SD | 33±5 |
| Men, n (%) | 173 (55) |
| Race, n (%) |  |
| White, non-Hispanic | 123 (39) |
| Black | 118 (37) |
| Hispanic | 56 (18) |
| Other | 20 (6) |
| **Prevalent CV disease, n (%)** |  |
| History of MI or revascularization | 9 (3) |
| Congestive heart failure | 11 (4) |
| Stroke | 8 (2) |
| Atrial fibrillation | 23 (7) |
| Any history of CV disease or stroke | 20 (6) |
| **CV risk factors** |  |
| Current smoker, n (%) | 38 (12) |
| Smoked >100 cigarettes in lifetime, n (%) | 91 (29) |
| Diabetes, n (%) | 97 (31) |
| Triglycerides, mg/dl, median (IQR) | 119 (80–194) |
| HDL, mg/dl, mean±SD | 46±13 |
| LDL, mg/dl, mean±SD | 109±39 |
| **Body measures, mean±SD** |  |
| SBP, mm Hg | 120±20 |
| DBP, mm Hg | 77±13 |
| BMI, kg/m² | 30±8.4 |
| **Medications** |  |
| β-blocker, n (%) | 82 (26) |
| ACE-I or ARB, n (%) | 199 (63) |
| Calcium channel blocker, n (%) | 77 (24) |
| Antihypertensives, mean±SD | 1.7±1.4 |
| Statins, n (%) | 96 (30) |
| Diuretics, n (%) | 110 (35) |
| **Kidney function** |  |
| eGFR, ml/min per 1.73 m², mean±SD | 51±19 |
| 24-hour protein/creatinine ratio, g/g, median (IQR) | 0.51 (0.1–2.25) |
| 24-hour albumin/creatinine ratio, mg/g, median (IQR) | 335 (37–1398) |

CV, cardiovascular; MI, myocardial infarction; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; IQR, interquartile range.
therapies for all young adults and stratified by race and ethnicity are listed in Supplemental Table 1.

IR of CV Outcomes

The median follow-up time for CV events was 11.3 years, and CKD progression was 4.1 years. For all study participants across racial and ethnic categories, the IR were highest for HF (IR per 1000 person-years = 10.4 [IQR 7.1–14.8]) and death (IR per 1000 person-years = 10.9 [IQR 7.8–14.9]; Figure 1, Supplemental Table 2). In particular, Black and Hispanic participants had higher rates of HF, MI, all-cause death, and composite CV events compared with participants who identified as White or of other race and ethnicity.

Secondary Analysis: Association with Race and Ethnicity and CV Events

Figure 2 and Supplemental Table 3 present the IRR between participants identifying as Black or Hispanic and those who were White or of other race and ethnicity. Black and Hispanic participants with CKD had a significantly higher adjusted rate of CV events or death compared with participants who were White or of other race and ethnicity (adjusted IRR = 2.2; 95% CI 1.15 to 4.24). CV event-free survival is described via Kaplan–Meier curves in Figure 3A, showing a decreased rate of event-free survival in Black and Hispanic participants compared with participants who were White or of other race and ethnicity.

IR and IRR by Race and Ethnicity of CKD Progression

IR of CKD progression are described in Figure 1 and Supplemental Table 2. CKD progression had an IR of 85.9/1000 person-years when including all study participants. Participants identifying as White or of other race and ethnicity had lower IR than those identifying as Black or Hispanic. As seen in Figure 2 and Supplemental Table 3, Black and Hispanic participants had a significantly higher adjusted IIR when compared with participants who were White or of other race and ethnicity (IRR=2.17; 95% CI, 1.53 to 3.08). Kaplan–Meier curves, stratified by race and ethnicity, for survival free from CKD progression are presented in Figure 3B.

Discussion

This study of young adults with CKD demonstrates that those who identify as Black and Hispanic have greater burden of CV risk factors and socioeconomic disparities compared with participants who are White or of other race and ethnicity. Even when adjusting for biologic risk factors

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**Table 2. Description of cardiovascular risk factors across race and ethnicity in young-adult participants with chronic kidney disease <40 years of age**

| Baseline Variable | All Age <40 Years (N=317) | White or Other Race (N=413) | Black Race (N=118) | Hispanic Ethnicity (N=56) | P Value across Racial and Ethnic Groups |
|-------------------|---------------------------|-----------------------------|-------------------|-------------------------|---------------------------------------|
| **Social/demographic** |                           |                             |                   |                         |                                       |
| Education level of high school diploma/GED or less, n (%) | 86 (27) | 16 (11) | 39 (33) | 31 (55) | <0.001 |
| Income, n ($) |                           |                             |                   |                         |                                       |
| <$20,000/yr | 81 (26) | 19 (13) | 38 (32) | 24 (43) | <0.001 |
| $20,000–$50,000/yr | 83 (26) | 33 (23) | 35 (30) | 15 (27) | |
| $50,001–$100,000/yr | 72 (23) | 44 (31) | 23 (20) | 5 (9) | |
| >$100,000/yr | 31 (10) | 27 (19) | 4 (3) | 0 (0) | |
| Income not reported | 50 (16) | 20 (14) | 18 (15) | 12 (21) | |
| **Kidney function** |                           |                             |                   |                         |                                       |
| eGFR, ml/min per 1.73 m², mean±SD | 51±19 | 54±19 | 50±19 | 47±20 | 0.04 |
| UACr, mg/g; N=293, median (IQR) | 335 (37–1398) | 198 (21–945) | 301 (45–1259) | 1277 (153–3005) | 0.001 |
| 2 high-risk APOL1 alleles, n (%) | 45 (14.2) | 0 (0) | 45 (42.5) | N/A | <0.001 |
| **Measurements** |                           |                             |                   |                         |                                       |
| SBP, mm Hg, mean±SD | 120±20 | 113±14 | 125±22 | 129±24 | <0.001 |
| DBP, mm Hg, mean±SD | 77±13 | 74±12 | 79±15 | 79±13 | 0.001 |
| LVH, n (%) | 51 (24) | 18 (16) | 25 (32) | 8 (40) | 0.01 |
| Waist circumference, cm, mean±SD | 99±20 | 93±18 | 107±20 | 95±15 | <0.001 |
| BMI, kg/m², mean±SD | 30±8.4 | 27±6.8 | 35±9 | 29±7 | <0.001 |
| LDL, mg/dl, mean±SD | 109±39 | 104±35 | 112±42 | 119±40 | 0.03 |
| **Medical history** |                           |                             |                   |                         |                                       |
| Diabetes, n (%) | 97 (31) | 40 (28) | 34 (29) | 23 (41) | 0.17 |
| History of CVD, n (%) | 20 (6.3) | 7 (5) | 12 (10) | 1 (1.8) | 0.07 |

GED, general educational development; UACr, urine albumin/creatinine ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVH, left ventricular hypertrophy; LDL, low-density lipoprotein; IQR, interquartile range; CRIC, Chronic Renal Insufficiency Cohort.

**n=211 with non-missing data, per CRIC cohort. LVH calculated by body surface area indexed left ventricular mass index obtained from ECHO 1 year from baseline visit.**
such as age, sex, and APOL1, we found significant differences in the rates of CV events, death, and CKD progression among Black and Hispanic young adults with CKD compared with those who were White or of other race and ethnicity. Our analysis suggests that much of the inequitable distribution of risk across race and ethnicity for adverse outcomes may be related to nonbiologic risk factors. These data may help prioritize earlier interventions to improve

Figure 1. | Incidence rates per 1000 person-years for cardiovascular (CV) outcomes and CKD progression for Chronic Renal Insufficiency Cohort (CRIC) participants <40 years of age and stratified by racial/ethnic risk groups. Error bars represent 95% confidence intervals. Data labels describe mean incidence rates. HF, heart failure; MI, myocardial infarction; CVA, cerebrovascular accident; CV events, composite of HF, MI, CVA, and all-cause death; CKD progression, >50% decline in eGFR or ESKD.

Figure 2. | Incidence rate ratio (IRR) for CV events or death and CKD progression for CRIC participants identifying as Black and/or Hispanic compared with those identifying as White or of other race and ethnicity. CKD progression is defined by 50% decrease in eGFR or ESKD. Cardiovascular event defined by first incidence of MI, congestive heart failure, stroke, or any-cause death. Adjusted IRR includes adjustment for age, sex, and number of high-risk APOL1 alleles.
outcomes in young adults with CKD who self-identify as Black or Hispanic.

Our findings suggest that the burden of CVD and mortality in young adults of all races and ethnicities with CKD is substantial. Clinically evident CV events in children with CKD aged <18 years are rare, and it is intriguing what processes explain the increase in CV events to the relatively high rate seen in our analysis only 10–15 years later (26–29). This finding helps to illustrate the utility of studying young adults with CKD to inform understanding CVD as a lifetime process. Of all of the CV event types in this study, HF had the highest incidence. IR of HF in young adults as part of the general population is poorly described. Nonetheless, we observed that the IR in young adults with CKD (1% per year) is substantially higher compared with young adults without CKD (around 0.01%–0.025% per year) (30,31). HF is associated with significant morbidity and mortality in the CKD population and, from a larger societal perspective, is connected with significant health care utilization and economic burden (32–34). Our results identify young adults with CKD as a high-risk subpopulation that may benefit from intensified CV therapies.

Studies of CKD populations, in which young adults have been largely or fully excluded, have also found the presence of disparities relating to race and ethnicity. Black CRIC participants were found to have increased rates of LVH, whereas CRIC participants who identify as Hispanic were found to have faster progression of their CKD (2,35). Within the CRIC cohort, there have been inconsistent findings related to rates of CV and all-cause mortality as it relates to participant race or ethnicity (3,14,36). This is likely secondary to differences within the study methodologies, but also possibly a reflection of the challenge of using race and ethnicity as an isolated study variable.

There is a growing consensus that an individual’s race or ethnicity does not correspond to any differences in genetics or physiology, the singular exception in CKD research being findings related to APOL1 risk alleles (13,37,38). Our findings found that controlling for APOL1 risk alleles did little to attenuate the association between race and ethnicity and adverse outcomes, suggesting other factors are at play. This is similar to other research that demonstrates that high-risk APOL1 alleles only partially mediate the higher incidence of CV events and CKD progression in Black CKD patients (4). CV risk factors, which themselves may reflect social determinants of health, were most prevalent in Black and Hispanic individuals. Given the heterogeneity of social circumstances and experiences of discrimination, elucidating the underlying factors that could explain the mal-distribution of CV risk factors is challenging within the constraints of our modestly sized study population. Nonetheless, disparities in socioeconomic status closely mirrored those observed with CV risk factors, especially income, which has been shown in several contexts to stratify CV outcomes (10). We noted that even in early adulthood, the distribution of income was not similar across racial and ethnic identities, and adults with CKD who identify as Black or Hispanic may already be burdened with CV and socioeconomic risk factors starting in early adulthood.

Focusing on research in young adults is important for several reasons. From a broad perspective, improving our understanding of the epidemiology of CVD and CKD progression in young adults with CKD might inform age-appropriate treatment strategies, especially those of a preventative nature. From the perspective of identifying and reducing social disparities, young adults can serve as a bridge to connect relevant experiences and risk factors across the lifespan. Originating from the experiences of childhood, young adulthood might be considered the starting position from which future adult health derives. Our results suggest young adults with CKD are placed in a relatively poor starting position compared with the general population. Moreover, those identifying as Black or Hispanic find themselves starting far behind other similarly aged CKD patients. More research is needed to understand the association between social determinants of health (e.g., access to care) and modifiable CV risk factors (e.g., hypertension or hyperlipidemia) in young-adult patients with CKD, and how they relate to an individual’s self-identified race and ethnicity.

Pertinent strengths of this study include using leveraging data from the CRIC Study, a well characterized CKD cohort. CV outcome measures were identified using rigorous physician adjudication for the CV events and were adjudicated by two independent physician reviews. CKD progression was determined by annual research laboratory measurement to capture longitudinal decline in kidney
function. Furthermore, data on APOL risk alleles were available for Black participants. However, we recognize some weaknesses as well. Our sample size was modest, which reduced our power to investigate the association between race or ethnicity with CV events and CKD progression using multivariable regression models. We attempted to minimize confounding by using potentially relevant covariates; however, as with all observational studies, we could not eliminate the risk for unmeasured confounding. We identified age, sex, and APOL1 as biologic variables; however, there may be other unrecognized biologic variables. Our sample size and event rate further limited our ability to include a more comprehensive list of biologic variables in multivariable modeling. APOL1 was not determined in participants who self-identified as Hispanic. So, we had to assume 0% APOL1 prevalence for the purpose of the analyses. The genetic admixture in the Hispanic population likely means the true prevalence of APOL1 is >0%. However, on the basis of previous US data, the prevalence is likely quite low (around 0.1%-3% of Hispanic individuals with two high-risk alleles), and any unmeasured confounding due to APOL1 in Hispanic individuals is presumed to affect the overall trends of our results minimally (25,39). Performing APOL1 genotyping across racial and ethnic groups in young adults with CKD in future research would be important, especially to help move beyond arbitrary racial/ethnic designations in an increasingly diverse population when estimating medical risk. This was an observational study, and so causality cannot be determined. Our modest sample size also suggests that further research is required to understand the generalizability of our findings to other young-adult CKD populations. The study sample consisted of research volunteers closely followed in nephrology clinics, possibly limiting the generalizability of our results to the broader CKD population. This study was also not powered to study other racial/ethnic groups such as Asian or Native American CKD patients. Lastly, we would like to recognize the challenges working with the concepts of race and ethnicity, and that contemporary definitions tend toward oversimplification. Our hope is that this work is hypothesis generating and might lead to new strategies to improve equity and outcomes for all patients with CKD.

In conclusion, this observational study described racial and ethnic disparities in the burden of risk factors and relative risk for adverse CV and kidney outcomes. Rates of CVD, CKD progression, and death were notably higher for Black and Hispanic participants, even when considering biologic variables. As a result, the needs of young adults with CKD cannot be ignored, and doing so runs the chance of compounding CV risk and racial disparities over a lifetime. Our hope is that this study will inspire future work identifying CV risk factors of greatest significance and social factors underpinning the disparities experienced across racial and ethnic groups as a means to develop targeted, preventative therapies.

Disclosures

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Author Contributions

N. Bansal was responsible for funding acquisition and supervision; N. Bansal and A.J. Kula were responsible for conceptualization and data curation; N. Bansal, A.J. Kula, C.P. Limonte, and B.A. Young were responsible for the investigation; N. Bansal, C.P. Limonte, D.J. Prince, and B.A. Young were responsible for the methodology; A.J. Kula wrote the original draft of the manuscript; A.J. Kula and D.J. Prince were responsible for the formal analysis; D.J. Prince was responsible for visualization; and all authors reviewed and edited the manuscript and approved the final version of the manuscript.

Supplemental Material

This article contains supplemental material online at http://kidney360.asnjournals.org/lookup/suppl/doi:10.34067/KID.0006712021/-/DCSupplemental.

Supplemental Table 1. Description of antihypertensive and statin use across racial and ethnic groups at the baseline visit.

Supplemental Table 2. Incidence rates per 1000 person-years for cardiovascular outcomes and CKD progression for Chronic Renal Insufficiency Cohort participants <40 years of age and stratified by racial/ethnic risk groups.

Supplemental Table 3. Incidence rate ratios for death, cardiovascular (CV) events or death, and CKD progression for Chronic Renal Insufficiency Cohort participants identifying as Black and/or Hispanic compared with those who identified as White or other race and ethnicity.

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