

# Perceived Discrimination and Longitudinal Change in Kidney Function Among Urban Adults

May A. Beydoun, PhD, MPH, Angedith Poggi-Burke, MPH, Alan B. Zonderman, PhD, Ola S. Rostant, PhD, Michele K. Evans, MD, and Deidra C. Crews, MD, ScM

## ABSTRACT

**Objective:** Perceived discrimination has been associated with psychosocial distress and adverse health outcomes. We examined associations of perceived discrimination measures with changes in kidney function in a prospective cohort study, the Healthy Aging in Neighborhoods of Diversity across the Life Span.

**Methods:** Our study included 1620 participants with preserved baseline kidney function (estimated glomerular filtration rate [eGFR]  $\geq 60$  mL/min/1.73 m<sup>2</sup>) (662 whites and 958 African Americans, aged 30–64 years). Self-reported perceived racial discrimination and perceived gender discrimination (PGD) and a general measure of experience of discrimination (EOD) (“medium versus low,” “high versus low”) were examined in relation to baseline, follow-up, and annual rate of change in eGFR using multiple mixed-effects regression ( $\gamma_{\text{base}}$ ,  $\gamma_{\text{rate}}$ ) and ordinary least square models ( $\gamma_{\text{follow}}$ ).

**Results:** Perceived gender discrimination “high versus low PGD” was associated with a lower baseline eGFR in all models ( $\gamma_{\text{base}} = -3.51$  (1.34),  $p = .009$  for total sample). Among white women, high EOD was associated with lower baseline eGFR, an effect that was strengthened in the full model ( $\gamma_{\text{base}} = -5.86$  [2.52],  $p = .020$ ). Overall, “high versus low” PGD was associated with lower follow-up eGFR ( $\gamma_{\text{follow}} = -3.03$  [1.45],  $p = .036$ ). Among African American women, both perceived racial discrimination and PGD were linked to lower follow-up kidney function, an effect that was attenuated with covariate adjustment, indicating mediation through health-related, psychosocial, and lifestyle factors. In contrast, EOD was not linked to follow-up eGFR in any of the sex by race groups.

**Conclusions:** Perceived racial and gender discrimination are associated with lower kidney function assessed by glomerular filtration rate and the strength of associations differ by sex and race groups. Perceived discrimination deserves further investigation as a psychosocial risk factors for kidney disease.

**Key words:** gender, kidney function, perceived discrimination, race, urban adults.

## INTRODUCTION

Chronic kidney disease (CKD) is a public health problem affecting 13% of US adults (1). Clinical factors, such as hypertension and diabetes, and genetic factors (2) do not fully explain CKD burden. Therefore, attention has been recently paid to other social, economic, and psychosocial factors, which may underlie kidney function decline (3,4). Among psychosocial factors, perceived discrimination (general experience of discrimination [EOD], race/ethnicity-related (perceived racial discrimination [PRD]), or perceived gender discrimination [PGD]) has been linked to adverse health outcomes, possibly through stress-related pathways, including hypertension, cardiovascular disease, poor general health status, and mental illness (5). Stress is a condition whereby environmental factors tax or exceed the adaptive capacity of individuals to a point where psychological and physiological responses may place them at risk for disease (6). Studies of stressors and their relation to pathophysiology have revealed alterations in blood pressure, heart rate, and vascular reactivity in response to acute stress (7–10).

These links suggest that adverse health outcomes are influenced by PRD (5,11–28) and in other instances by PGD (12,14,15,19–22,29). Nevertheless, in one earlier study, reporting no or low discrimination had an unexpected positive relationship with worse health outcomes, such as hypertension, specifically among AA women (12). Thus, the direction of the association between perceived discrimination is still debated, particularly within different sociodemographic strata, such as sex and race.

To our knowledge, there have been no empirical studies of the relation of perceived discrimination and kidney function. Therefore, we examined the associations of PRD, PGD, and EOD with

AA = African American, ACR = albumin:creatinine ratio, BMI = body mass index, CES-D = Center for Epidemiologic Studies-Depression, CKD = chronic kidney disease, EDS = elevated depressive symptoms, eGFR = estimated glomerular filtration rate, EOD = experience of discrimination, HANDLS = Health Aging in Neighborhoods of Diversity across the Life Span, OLS = ordinary least square, PGD = perceived gender discrimination, PRD = perceived racial discrimination

## SDC Supplemental Content

From the Laboratory of Epidemiology and Population Sciences (Beydoun, Poggi-Burke, Zonderman, Rostant, Evans), National Institute on Aging, National Institutes of Health; Division of Nephrology (Crews), Department of Medicine, Johns Hopkins University School of Medicine; and Welch Center for Prevention (Crews), Epidemiology and Clinical Research, Johns Hopkins Medical Institutions, Baltimore, Maryland.

Address correspondence and reprint requests to Deidra C. Crews, MD, ScM, Johns Hopkins University School of Medicine, 301 Mason F Lord Drive, Suite 2500, Baltimore, MD 21224. E-mail: dcrews1@jhmi.edu

Received for publication August 8, 2016; revision received March 6, 2017.

DOI: 10.1097/PSY.0000000000000478

Copyright © 2017 by the American Psychosomatic Society

longitudinal kidney function change in a biracial socioeconomically diverse sample from Baltimore City, Maryland, and tested differential associations by sex and race.

## METHODS

### Study Design

Initiated in 2004, the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study is an ongoing prospective cohort study focused on the cardiovascular and cognitive health of a socioeconomically diverse sample of AAs and whites (baseline age = 30–64 years), residing in Baltimore, Maryland. Race was self-reported in answer to the question: please look at this card and tell me which category best describes you. Are you: 1. white; 2. black/AA; 3. American Indian or Alaska Native; 4. Asian; 5. Native Hawaiian or other Pacific Islander; and 6. Some other race? Only those with self-described race of white or AA were eligible for the HANDLS study. Briefly, thirteen neighborhoods were selected using an area probability sampling methodology as detailed elsewhere (30). Phase 1 consisted of screening, recruitment, and household interviews, whereas phase 2 collected more extensive data in a mobile medical research vehicle. The present study uses baseline visit 1 (2004–2009) and the first follow-up visit 2 (2009–2013), with mean follow-up time of approximately 5 years.

All participants provided written informed consent, after accessing a protocol booklet in layman's terms and a video detailing all procedures and future recontacts. HANDLS study was ethically approved by the National Institute on Environmental Health Sciences, National Institutes of Health, and its institutional review board.

### Participants

Of 3720 total baseline HANDLS participants initially selected with complete phase 1 of visit 1 data (i.e., home visit), 2743 had complete data on estimated glomerular filtration rate (eGFR) at either visit measured on the medical research vehicle (phase 2, visit 1), whereas 1993 had complete eGFR data at both baseline and follow-up ( $n = 750$  at baseline only). We further excluded participants with missing data on PRD/PGD/EOD ( $n = 63$ ) or with baseline eGFR of less than 60 mL/min/1.73 m<sup>2</sup> ( $n = 84$ ). Of the remaining participants ( $n = 1846$ ) with preserved kidney function, those with missing data on any of the covariates entered into the model were excluded (i.e., complete case analysis;  $n = 68$  missing on hypertension or diabetes, an additional  $n = 125$  missing on smoking/drug use, an additional  $n = 35$  missing on elevated depressive symptoms (EDS) status at baseline, and an additional  $n = 2$  missing on education) yielding a final sample size of 1616. Compared with the HANDLS cohort that was not selected, our selected sample included a higher proportion who did not live in poverty and more females ( $p < .05$ ), although no age or race differences were detected. This sample selectivity was accounted for in the analysis through a two-stage Heckman selection model as discussed in the statistical analysis section.

### Perceived Racial Discrimination

Baseline PRD was measured using an adapted nine-item discrimination scale of the EOD questionnaire (12), and two global PRD items (31) (Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A393>), eliciting perceived discrimination because of race, ethnicity, or culture on a four-point Likert scale (“not at all” to “a lot”). The five PRD binary (yes/no) situations from the EOD were racial discrimination *at school*, *getting a job*, *at work*, *getting housing*, and *getting medical care*. The sum of the five situational items (range = 5–10), and that of the two global items (range = 2–8), were entered as two measured variables in a factor analysis with one common factor being extracted and predicted using the regression method. The predicted factor ( $z$ -score) was then grouped into the following categories: “low

PRD” (factor score  $< 0$ ), “medium PRD” (factor score = 0–1), and “high PRD” (factor score  $> 1$ ).

### Perceived Gender Discrimination

Similarly, PGD included one global item measured on a four-point Likert scale (“not at all” to “a lot”) and five binary “yes/no” items, namely, “Have you ever experienced discrimination, or has anyone stopped you from doing something, hassled you, or made you feel inferior because of your gender?” in the following five distinctive situations: *at school*, *getting a job*, *at work*, *at home*, or *when getting medical care?*, with a total score range of 5 to 10 (12,32). Using a similar approach, a factor analysis was conducted to combine the global measure with the situational measures that were also summed. The common factor was predicted and categorized as the following: “low PGD” ( $< 0$ ), “medium PGD” (0–1), and “high PGD” ( $> 1$ ). The correlation between the sum of global PGD items and the sum of situational PGD items was .49, whereas that of global versus situational PRD items was .54. The factor score for PGD was highly correlated with each sum of items ( $r = .87$  [factor score versus global],  $r = .86$  [factor score versus situational]). Those correlations were both .88 for PRD. Cronbach  $\alpha$  levels, assuming we are summing up items for each scale, were .79 and .67, for PRD and PGD, respectively. In addition to using the final factor score in the main analysis, summation of the items of PRD and PGD was also used as a continuous outcome in a small portion of the analysis.

### Experience of Discrimination

Perceived discrimination was also measured using the EOD (33,34). The nine-item EOD measures the everyday experiences of unfair treatment and is by far the most commonly used scale in previous studies. This measure asks respondents, “How often in your day-to-day life have the following things happened to you?” (e.g., “You are treated with less courtesy”; “You are treated with less respect”; “You get worse service at restaurants and stores”; “People act as if you are not smart”; “People act as if they are afraid of you”; “People act as if they think you are dishonest”) on a Likert response scale (1 = never, 2 = less than once a year, 3 = a few times a year, 4 = a few times a month, 5 = at least once a week, and 6 = almost every day). Items were reverse coded, so higher scores reflect more everyday discrimination (Cronbach  $\alpha = .84$  and item-total correlations ranging .54–.77).

A similar factor analytic approach was carried out whereby each of the nine items was entered as measured variables and one factor was extracted. This common factor was then predicted and categorized in a similar fashion as for PRD and PGD (“low EOD” [factor score  $< 0$ ], “medium EOD” [factor score = 0–1], and “high EOD” [factor score  $> 1$ ]). In addition to using the final factor score in the main analysis, summation of the items of EOD was also used as a continuous outcome in a small portion of the analysis.

### Kidney Function

Our primary outcomes were baseline, annual rate of change, and follow-up eGFR. Using participant fasting venous blood specimens, baseline serum creatinine was measured at the National Institute on Aging, Clinical Research Branch Core Laboratory, using a modified kinetic Jaffe method (CREA method, Dade Dimension X-Pand Clinical Chemistry System, Siemens Healthcare Diagnostics Inc, Newark, Del) for a small group of participants ( $n = 88$ ), whereas most participants ( $n = 1528$ ) had baseline serum creatinine analyzed at Quest Diagnostics, Inc, by isotope dilution mass spectrometry (Olympus America Inc, Melville, NY) and standardized to the reference laboratory, Cleveland Clinic. Although interassay coefficients of variation for this sample could not be calculated because of the use of only one or the other measurement of creatinine at baseline, only intra-assay coefficients of variation (mean/SD) could be estimated and those were .192 and .187 for the CREA and the isotope dilution mass spectrometry methods, respectively. All follow-up serum creatinine concentrations were measured using IDMS at Quest Diagnostics, Inc.

For participants having spot urine data, microalbumin concentration was measured at Quest Diagnostics, Inc, using an immunoturbidimetric assay

(Kamiya Biomedical Co, Seattle, Wash). Estimated GFR was calculated using the CKD Epidemiology Collaboration equation (35), truncating values at 150 mL/min/1.73 m<sup>2</sup> (36). Urine albumin:creatinine ratio (ACR) was estimated and included in a sensitivity analysis, because of its appreciable missingness from the selected sample (>10%).

### Covariates

Age, sex, race (white or AA), completed years of education, poverty status (household income <125% of 2004 Department of Health and Human Services guideline) (30), marital status, current cigarette smoking, illicit drug use, and self-rated health were self-reported at baseline. Baseline diabetes mellitus status combined fasting serum glucose concentration of 126 mg/dL or greater, self-reported diabetes, and/or prescription diabetes medication. Using two sitting blood pressure measurements, with brachial artery auscultation and an inflatable cuff (37), hypertension was defined as the average of two systolic or diastolic blood pressures of 140 mm Hg or greater or 90 mm Hg or greater, respectively, or self-reported hypertension, or antihypertensive medication prescription. Body mass index (BMI) was calculated as weight over height squared (kilogram per square meter). EDS were defined as 16 or higher score on the 20-item Center for Epidemiologic Studies-Depression scale (38,39).

### Statistical Analysis

Bivariate associations of PRD and PGD with each of the baseline covariates were tested using one-way analysis of variance from a bivariate ordinary least square (OLS) regression model for continuous variables and  $\chi^2$  tests of independence for categorical variables. Similarly, we compared means of baseline, follow-up, and annual rates of change in eGFR across PRD and PGD, stratifying by sex by race.

We used mixed-effects linear regression models to examine associations of baseline PRD and PGD (high versus low) with eGFR (baseline and annual rate of change), controlling for key confounders. To account for nonrandom participant selection by age, sex, race, and poverty status, in each mixed-effects regression model, we conducted a two-stage Heckman selection process, as described elsewhere (40,41). In the basic model, we estimated the alternative associations of PRD and PGD with baseline and annual rate of change in eGFR, adjusting slopes and intercepts for age, sex, and race (model 1). Moving forward, we adjusted for factors that were considered modifiable socioeconomic, life-style, and health-related factors. Although some can be considered potential confounders, others such as health-related factors are often the result of life-style and socioeconomic factors as well as psychosocial factors and thus may be mediating the effect of perceived discrimination on kidney function outcomes. Therefore, a stepwise adjustment was used to examine the potential omnibus effect of adding several groups of variables into the models in a cumulative manner. In model 2, we further adjusted model 1 for poverty status, education, and marital status (i.e., in addition to age, sex, and race); in model 3, we adjusted model 2 for current smoking and illicit drug use, self-rated health, BMI, and EDS; in model 4, we controlled model 3 further for diabetes and hypertensive status. We added interaction terms and stratified by sex and race, because AAs report greater PRD (42) and reactions to psychological stressors differ by gender (43). Predictive margins of eGFR from stratified mixed-effects regression models were selectively plotted across time to illustrate key findings. Finally, we conducted OLS regression models, evaluating PRD and PGD's independent associations with follow-up eGFR. Thus, two types of longitudinal analyses were conducted. Although the first method investigates whether discrimination has a potential effect on the rate of change in kidney function, the second method investigates the effect of baseline discrimination on the level of kidney function 5 years later. A type 1 error of .05 was considered in all analyses, which were conducted using Stata Version 13 (StataCorp, College Station, Tex). A sensitivity analysis is presented and discussed in Supplemental Digital Content 2 (<http://links.lww.com/PSYMED/A394>) whereby ACR was included in model 5, after excluding all participants with missing data on

ACR. In a second sensitivity analysis (data not reported), the method/laboratory used for creatinine measurement was added as an additional covariate in all models and results of the full models were compared. In a third sensitivity analysis (data not reported), the 1846 individuals with complete data on eGFR at both visits were selected, by including a category for missing (e.g., missing = "9"). Depressive symptoms were categorized as (0: <16, 1:  $\geq$ 16, 9: missing).

## RESULTS

### Baseline Study Characteristics by EOD Groups

Overall, participants' mean age was 48 years; 59% were AA; 41% were male. High PRD was reported by 13.7%, high PGD by 11.3%, and high EOD by 15.2%. Both PRD and PGD factor scores (see factor analysis in the methods section) had a positive and linear association with EOD tertiles. A larger proportion of AA men was found among participants with high EOD as opposed to low EOD (35.1% versus 21.6%). High EOD was also associated with a higher proportion below poverty, poor/fair self-rated health, current smoking, current illicit drug use, and EDS. Overall, there was only a marginally significant higher mean baseline eGFR in the "high EOD" as opposed to the "low EOD" group. No linear trend was detected between EOD and the prevalence rates of hypertension and diabetes, the distribution in educational level and marital status or in mean BMI (Table 1).

### Baseline, Follow-Up, and Annual Rate of Change in eGFR by PRD, PGD, and EOD Groups

Overall, the mean annual rate of change in eGFR was estimated at  $-1.10$  U/year, with an SD of 3.35 (range =  $-18.7$  to  $+17.1$ ). "High PRD" was associated with a faster rate of decline in eGFR among AA women as compared with "low PRD" (Fig. 1A). In contrast, PGD and EOD were not associated with the rate of change in eGFR in any of the sex by race groups (Figs. 1C, D).

When examining baseline and follow-up eGFR (overall M [SD] = 101.64 [19.11] and 101.32 [20.16], respectively), among AA women, high PRD (versus low PRD) and high PGD (versus low PGD) were both associated with lower follow-up eGFR (Figs. 2A, B). In contrast, "high EOD" was linked to higher eGFR among both AA women (baseline) and white men (baseline and follow-up), when compared with "low EOD" (Fig. 2C).

### Unadjusted Association Between PRD/PGD/EOD Summation Scores and Key Outcomes

Table S1 (Supplemental Digital Content 2, <http://links.lww.com/PSYMED/A394>) shows the unadjusted correlations between outcome measures and key exposures, overall and stratifying simultaneously by sex and race. Although most correlation coefficients were weak (<.3), statistical significance was observed for AA women, whereby the PRD summation score was inversely related to baseline, follow-up, and annual rate of change in eGFR. PGD among AA women was also inversely related to two of three outcomes, namely, baseline and follow-up eGFR. This is in stark contrast with the EOD summation score, which showed a positive association with baseline and follow-up eGFR, overall and among white men. Finally, the EOD summation score was also positively associated with baseline eGFR among AA women.

**TABLE 1.** Study Participant Baseline Characteristics, Overall and by EOD group, HANDLS Study

	Overall (n = 1616)	Low EOD (n = 934)	Medium EOD (n = 437)	High EOD (n = 245)	<i>p</i> Trend
%	100	57.8	27.0	15.2	
Sex by race					
White women	23.6	24.6	23.8	19.2	<.001
AA women	35.0	36.5	34.3	30.0	
White men	17.2	17.2	18.1	15.5	
AA men	24.1	21.6	23.8	35.1	
Baseline age, M (SE)	48.3 (0.2)	49.3 (0.3)	47.1 (0.4)	46.5 (0.5)	.001
Married, %	33.4	33.7	32.7	33.4	.94
Educational level, %					
<HS	6.5	6.4	6.4	6.9	.84
HS	58.0	57.8	60.0	60.8	
>HS	35.4	35.8	36.6	32.2	
Poverty status, %					.035
<125% PIR	38.0	36.3	37.8	45.3	
≥125% PIR	61.9	63.7	62.2	54.7	
Self-rated health, %					.001
Poor/fair	24.0	21.7	24.5	31.8	
Good	40.7	40.0	45.1	35.5	
Very good/excellent	35.4	38.4	30.4	32.7	
Current smoking status, %	45.2	42.7	44.2	56.3	.001
Current illicit drug use, %	17.3	14.3	18.8	25.7	<.001
BMI, M (SE)	30.1 (0.2)	29.9 (0.2)	30.7 (0.4)	29.6 (0.5)	.88
EDS, CES-D total score ≥16, %	40.1	32.2	49.0	54.3	<.001
Hypertension, % yes	44.7	45.6	44.2	42.4	.65
Diabetes, % yes	9.7	9.6	10.8	8.2	.54
Baseline eGFR, M (SE)	101.6 (0.5)	101.0 (0.6)	102.0 (0.9)	103.3 (1.3)	.09
PRD factor score, M (SE)	-0.003 (0.018)	-0.21 (0.02)	+0.18 (0.04)	+0.47 (0.06)	<.001
Low	59.4	71.2	47.4	35.9	<.001
Medium	26.8	22.8	33.9	29.4	
High	13.7	6.0	18.8	34.7	
PGD factor score, M (SE)	-0.004 (0.017)	-0.22 (0.02)	+0.21 (0.04)	+0.46 (0.05)	<.001
Low	58.8	80.7	35.1	10.3	<.001
Medium	30.0	17.4	55.9	33.2	
High	11.3	1.9	9.0	56.3	
EOD factor score, M (SE)	+0.007 (0.023)	-0.62 (0.013)	+0.42 (0.01)	+1.68 (0.03)	<.001

EOD = nine-item everyday of discrimination scale; M (SE) = mean (standard error); HS = high school; PIR = poverty income ratio; BMI = body mass index; CES-D = Center for Epidemiologic Studies-Depression; eGFR = estimated glomerular filtration rate; PGD = perceived gender discrimination.

Selected study participants had preserved kidney function. Values are percent or M (SE). *p* value for trend was based on a one-way analysis of variance when row variable is continuous and  $\chi^2$  when row variable is categorical.

### Net Associations Between PRD/PGD and EOD With Baseline and Annual Rate of Change in eGFR

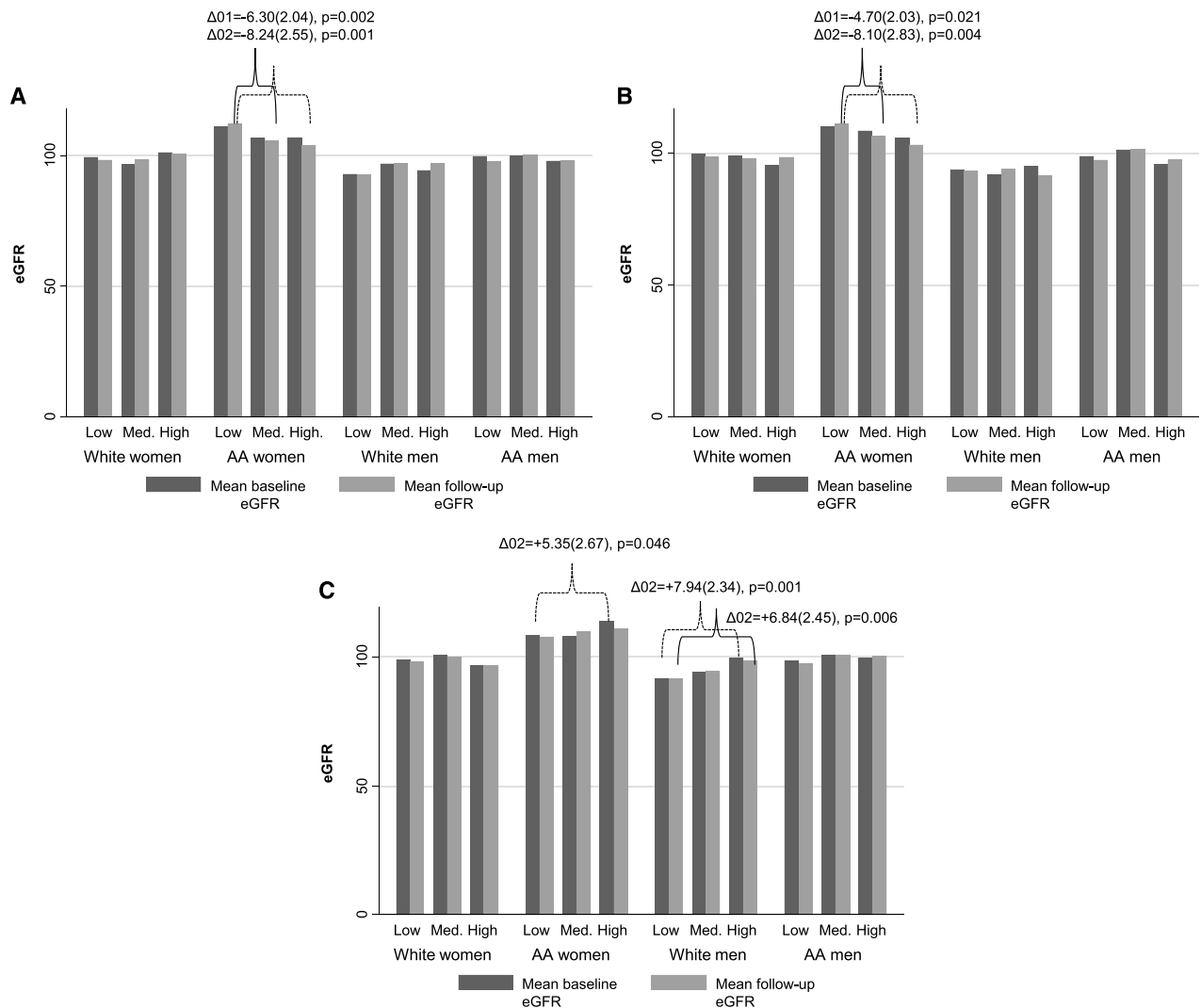
In mixed-effects regression models examining the net effect of PRD and PGD on eGFR (baseline and annual rate of change), in the total sample, “high versus low PGD” was associated with a lower baseline eGFR in all models (full model: PGD effect = -3.51 [1.34], *p* = .009), an effect restricted to whites (Table 2).

Other key findings emerged in the sex- and race-stratified mixed-effects regression models with EOD (Table 3). Specifically, among white women, high EOD was associated with lower baseline eGFR, an effect that was strengthened in the full model (full

model: EOD effect = -5.86 [2.52], *p* = .020). Among white men, high EOD was linked to a marginally significant faster decline in eGFR in model 1, which was fully attenuated by socioeconomic factors in model 2.

### PRD/PGD and EOD and Their Adjusted Associations With Follow-Up eGFR

Our sequential OLS models with alternative predictors PRD and PGD (Supplemental Digital Content 2, Table S2, <http://links.lww.com/PSYMED/A394>) indicated that overall, “high versus low” PGD was associated with lower follow-up eGFR (full model:



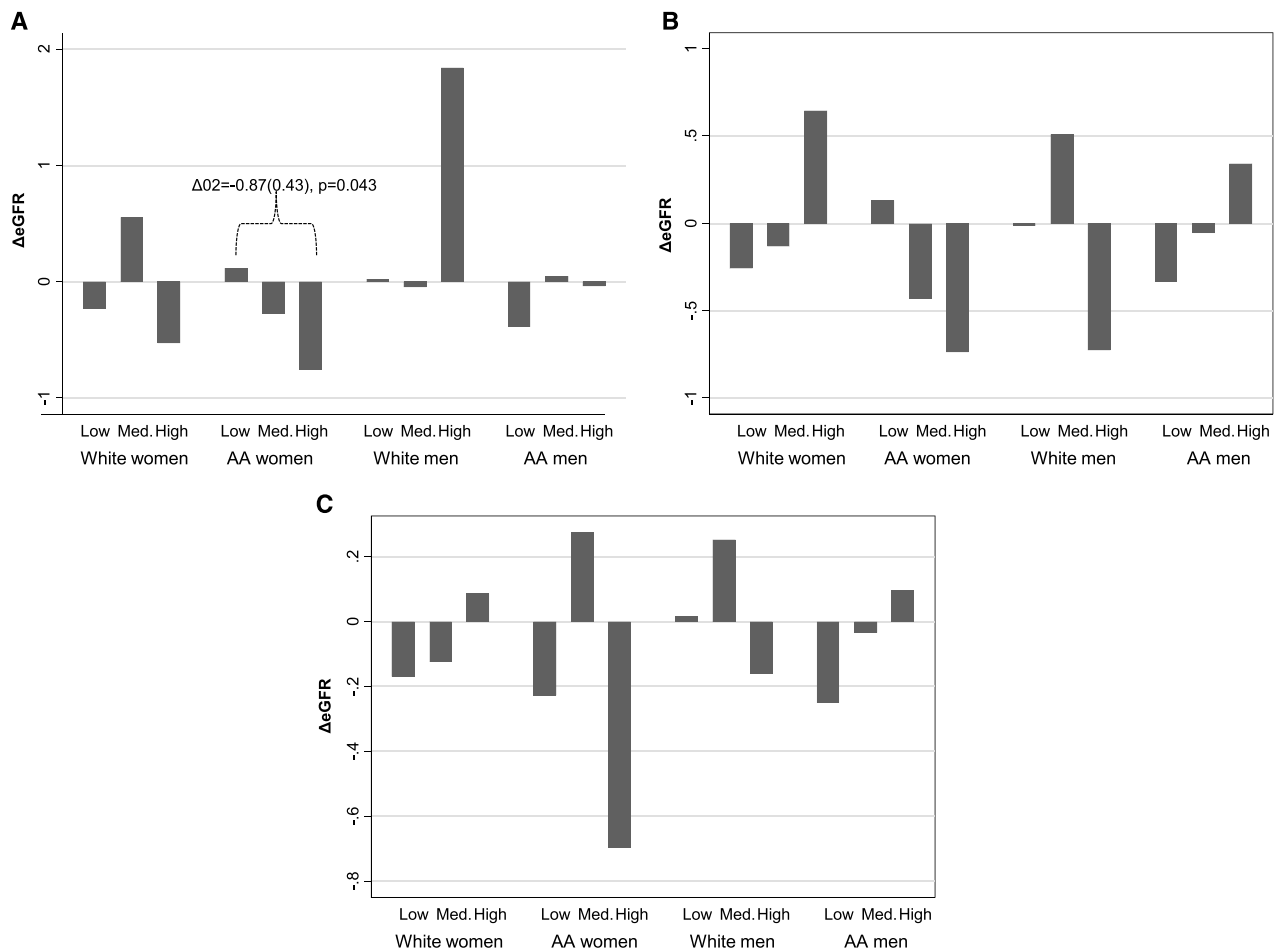
**FIGURE 1.** A, Annual rate of change in eGFR by PRD category. B, Annual rate of change in eGFR by PGD category. C, Annual rate of change in eGFR by EOD category.

PGD effect =  $-3.03 [1.45]$ ,  $p = .036$ ). “Medium versus low PRD” was specifically positively associated with eGFR, indicating better kidney function, among white men in model 3 (PRD effect =  $+4.33 [2.07]$ ,  $p = .037$ ), an effect attenuated with adjustment for hypertension and diabetes status (PRD effect =  $+4.09 [2.08]$ ,  $p = .050$ ). Among AA women, both PRD and PGD were linked to lower kidney function at follow-up, an effect that was attenuated systematically between models 2 and 4, indicating an effect of health-related (e.g., self-rated health, BMI, hypertension, and diabetes), psychosocial (depressive symptoms), and life-style factors (smoking and drug use). In contrast, EOD was not linked to follow-up eGFR in any of the sex by race groups (Supplemental Digital Content 2, Tables S3, <http://links.lww.com/PSYMED/A394>). A few marked changes were observed in the sensitivity analysis in the sample with complete ACR, mostly due to a reduced overall sample size ( $n = 1158$ ) (Supplemental Digital Content 2, Tables S4–S7, <http://links.lww.com/PSYMED/A394>). In a second sensitivity analysis (data not reported), the method/laboratory used for creatinine measurement was restricted to Quest Diagnostics, the

most commonly used laboratory at both waves, and the only one used in the follow-up wave ( $n = 1528$  of 1616). The results were not altered, as was the case for a third sensitivity analysis of 1846 individuals with complete baseline and follow-up visit eGFR (data not reported).

## DISCUSSION

Within a biracial urban sample of adults in Baltimore City, Maryland, high PRD was reported by 13.7%, high PGD by 11.3%, and high EOD by 15.2%. Associations between perceived discrimination and kidney function varied by race and sex groups. Among whites, high PGD was associated with a lower baseline eGFR. Among white women, high EOD was associated with lower baseline eGFR. Overall, high PGD was associated with lower follow-up eGFR. Notably, among AA women, both PRD and PGD were linked to lower kidney function at follow-up, an effect that appeared mediated by health-related, psychosocial, and life-style factors. In contrast, EOD was not linked to follow-up eGFR in any of the sex by race groups.



**FIGURE 2.** A, Baseline and follow-up mean eGFR by PRD category. B, Baseline and follow-up mean eGFR by PGD category. C, Baseline and follow-up mean eGFR by EOD category.

Our findings of variation of associations between perceived discrimination and kidney function change across sex and race groups are consistent with nuanced findings of several other studies, underscoring the complex effects of discrimination on health outcomes (17,20,22–24). For example, in a large sample of Asian American adults, perceived discrimination was associated with adverse health outcomes among both men and women, with the strongest association being with women's mental health. The threshold for an association of discrimination with adverse health outcomes was also lower among women as compared with men (16). Based on the Coronary Artery Risk Development in Young Adults study, the experience of one or two episodes of discrimination were only associated with higher levels of inflammation (as measured by C-reactive protein) among AA women. There were no such associations observed among men or white women (18).

The findings of our study could have implications for the well-established race and sex differences in kidney disease outcomes. For example, whites have equal or greater overall prevalence of reduced kidney function when compared with AAs (44); however, AAs experience faster declines in kidney function (45) and bear a greater burden of advanced and end-stage renal disease (46). Although few studies have examined the intersectionality of

race and sex in kidney disease, white women have been documented to have greater overall prevalence of reduced kidney function (47), as compared with women of other race/ethnic groups; however, AA men (48) have the highest incident rate for end-stage renal disease. Our study argues for closer examination of psychosocial stressors for their impact on these differences.

Biologically speaking, chronic psychosocial stress may induce changes in neuroendocrine, autonomic, and immune systems (49), and perceived discrimination has been linked with increased levels of oxidative stress (50), a pathway through which allostatic load (51) may be transduced into chronic diseases (52). Stress-induced allostatic load was hypothesized to cause an epigenetically induced proinflammatory state, leading to an increased risk for cardiovascular disease (53). Moreover, both racial and sex differences in coping with psychosocial stress, including discrimination, are important to consider, because they were detected in various non-CKD samples, and coping strategies have been noted to vary among men and women with CKD, with women showing a broader range of strategies that can buffer the effects of stress (54).

Perceived discrimination can lead to hopelessness and low self-efficacy (55), affecting the ability to self-manage one's health, perhaps differentially by sex and race (56). For instance, among

**TABLE 2.** Baseline and Annual Rate of Change in eGFR by Perceived Racial/Gender Discrimination (PRD, PGD), Overall and by Sex by Race: Mixed-Effects Linear Regression Models

Models	n	Model 1		Model 2		Model 3		Model 4	
		$\beta$ (SE)	<i>p</i>	$\beta$ (SE)	<i>p</i>	$\beta$ (SE)	<i>p</i>	$\beta$ (SE)	<i>p</i>
Overall	1616								
Model A: PRD									
PRD <sub>10</sub>		-1.72 (1.00)	.085	-1.63 (1.00)	.10	-1.59 (1.00)	.11	-1.60 (1.00)	.11
PRD <sub>10</sub> by time		+0.05 (0.19)	.80	+0.02 (0.19)	.91	+0.02 (0.20)	.91	+0.03 (0.19)	.88
PRD <sub>20</sub>		-1.37 (1.31)	.30	-1.11 (1.31)	.40	-1.35 (1.32)	.31	-1.31 (1.32)	.32
PRD <sub>20</sub> by time		-0.15 (0.25)	.55	-0.23 (0.25)	.37	-0.18 (0.25)	.49	-0.20 (0.25)	.42
Model B: PGD									
PGD <sub>10</sub>		-0.36 (0.92)	.70	-0.32 (0.92)	.72	-0.40 (0.92)	.66	-0.41 (0.92)	.66
PGD <sub>10</sub> by time		-0.12 (0.18)	.49	-0.14 (0.18)	.43	-0.12 (0.18)	.50	-0.14 (0.18)	.43
PGD <sub>20</sub>		-3.41 (1.33)	.010	-3.28 (1.37)	.014	-3.53 (1.34)	.009	-3.51 (1.34)	.009
PGD <sub>20</sub> by time		+0.13 (0.26)	.61	+0.09 (0.26)	.73	+0.14 (0.26)	.60	+0.11 (0.26)	.68
White women	381								
Model A: PRD									
PRD <sub>10</sub>		-2.81 (2.20)	.20	-3.25 (2.19)	.14	-3.35 (2.18)	.13	-3.55 (2.19)	.11
PRD <sub>10</sub> by time		+0.53 (0.46)	.25	+0.62 (0.46)	.17	+0.70 (0.45)	.12	+0.66 (0.45)	.14
PRD <sub>20</sub>		+0.83 (4.86)	.86	-0.37 (4.85)	.94	+0.23 (4.87)	.96	+0.33 (4.86)	.95
PRD <sub>20</sub> by time		+0.25 (1.00)	.81	+0.54 (0.99)	.58	+0.34 (0.98)	.73	+0.32 (0.98)	.74
Model B: PGD									
PGD <sub>10</sub>		+0.27 (1.73)	.88	-0.18 (1.75)	.92	-0.18 (1.75)	.92	-0.06 (1.76)	.97
PGD <sub>10</sub> by time		-0.02 (0.37)	.96	+0.01 (0.37)	.98	-0.01 (0.37)	.98	-0.10 (0.37)	.79
PGD <sub>20</sub>		-4.50 (2.55)	.078	-4.73 (2.55)	.064	-4.73 (2.55)	.065	-4.88 (2.57)	.058
PGD <sub>20</sub> by time		+0.99 (0.54)	.067	+1.02 (0.54)	.058	+1.02 (0.54)	.058	+0.93 (0.53)	.077
White men	278								
Model A: PRD									
PRD <sub>10</sub>		+3.57 (2.03)	.079	+3.82 (1.97)	.052	+3.37 (1.97)	.088	+3.28 (1.98)	.098
PRD <sub>10</sub> by time		+0.15 (0.40)	.72	+0.09 (0.40)	.81	+0.30 (0.40)	.47	+0.27 (0.40)	.50
PRD <sub>20</sub>		-1.84 (4.53)	.68	-2.25 (4.41)	.61	-3.46 (4.37)	.43	-3.66 (4.39)	.40
PRD <sub>20</sub> by time		-0.25 (0.89)	.78	-0.22 (0.88)	.80	+0.35 (0.89)	.70	+0.31 (0.88)	.73
Model B: PGD									
PGD <sub>10</sub>		-2.77 (1.92)	.15	-2.48 (1.87)	.19	-3.45 (1.84)	.062	-3.36 (1.85)	.069
PGD <sub>10</sub> by time		+0.55 (0.38)	.15	+0.54 (0.38)	.16	+0.63 (0.37)	.093	+0.63 (0.38)	.094
PGD <sub>20</sub>		-1.67 (5.36)	.76	-2.80 (5.20)	.59	-4.45 (5.22)	.39	-4.50 (5.22)	.39
PGD <sub>20</sub> by time		-0.54 (0.92)	.56	-0.46 (0.92)	.62	+0.05 (0.94)	.96	+0.05 (0.93)	.96
AA women	565								
Model A: PRD									
PRD <sub>10</sub>		-2.44 (1.77)	.16	-2.70 (1.79)	.13	-2.44 (1.81)	.18	-2.34 (1.81)	.20
PRD <sub>10</sub> by time		-0.33 (0.32)	.31	-0.33 (0.33)	.32	-0.33 (0.33)	.31	-0.33 (0.33)	.32
PRD <sub>20</sub>		-0.51 (2.23)	.82	-1.00 (2.27)	.66	-0.88 (2.31)	.70	-0.72 (2.32)	.76
PRD <sub>20</sub> by time		-0.73 (0.41)	.078	-0.62 (0.42)	.14	-0.57 (0.43)	.18	-0.57 (0.43)	.18
Model B: PGD									
PGD <sub>10</sub>		-0.70 (1.76)	.69	-1.08 (1.77)	.54	-0.93 (1.78)	.60	-0.92 (1.78)	.61
PGD <sub>10</sub> by time		-0.65 (0.33)	.044	-0.60 (0.33)	.066	-0.57 (0.33)	.081	-0.59 (0.32)	.074
PGD <sub>20</sub>		-1.25 (2.45)	.61	-1.81 (2.48)	.47	-1.67 (2.52)	.51	-1.59 (2.52)	.53
PGD <sub>20</sub> by time		-0.72 (0.45)	.11	-0.61 (0.45)	.18	-0.57 (0.46)	.21	-0.57 (0.46)	.21

*Continued on next page*

TABLE 2. (Continued)

Models	n	Model 1		Model 2		Model 3		Model 4	
		$\beta$ (SE)	<i>p</i>	$\beta$ (SE)	<i>p</i>	$\beta$ (SE)	<i>p</i>	$\beta$ (SE)	<i>p</i>
AA men	392								
Model A: PRD									
PRD <sub>10</sub>		-1.14 (2.00)	.57	-1.16 (2.01)	.57	-1.06 (1.98)	.59	-1.46 (1.98)	.46
PRD <sub>10</sub> by time		+0.52 (0.39)	.18	+0.40 (0.39)	.30	+0.40 (0.38)	.29	+0.48 (0.38)	0.21
PRD <sub>20</sub>		-1.20 (2.17)	.58	-0.78 (2.19)	.72	-1.31 (2.16)	.54	-1.22 (2.15)	.57
PRD <sub>20</sub> by time		+0.43 (0.42)	.30	+0.27 (0.42)	.72	+0.23 (0.41)	.57	+0.22 (0.41)	0.59
Model B: PGD									
PGD <sub>10</sub>		+1.82 (1.84)	.32	+2.09 (1.85)	.26	+1.86 (1.81)	.31	+1.79 (1.81)	0.32
PGD <sub>10</sub> by time		+0.34 (0.35)	.34	+0.24 (0.36)	.50	+0.24 (0.35)	.50	+0.25 (0.35)	0.47
PGD <sub>20</sub>		-3.03 (2.43)	.21	-2.90 (2.43)	.23	-3.90 (2.39)	.10	-3.84 (2.38)	.11
PGD <sub>20</sub> by time		+0.67 (0.47)	.15	+0.59 (0.46)	.20	+0.63 (0.46)	.17	+0.59 (0.45)	.11

SE = standard error; PRD = perceived racial discrimination; PGD = perceived gender discrimination.

Selected participants with preserved kidney function. PRD and PGD are coded as 2 = high, 1 = medium, and 0 = low and were entered separately in models A or B. For instance, PGD<sub>10</sub> refers to medium PGD contrasted with low PGD. Model 1: adjusted for inverse mills ratio, age, sex, and race; model 2: further adjusted for poverty status, marital status, and educational level; model 3: further adjusted for current smoking status and illicit drug use, BMI, self-rated health, and EDS; model 4: further adjusted for diabetes and hypertension.

hypertensive AAs, PRD was linked to lower medication adherence (57). Another study suggested that lifetime discrimination was associated with medical care delays and nonadherence, (58) a possible contributor to racial disparities in health, in general, and CKD progression in particular. Similarly, education-related discrimination was linked to poorer glycemic control among type 2 diabetes patients (59), whereas sex discrimination among women was linked to nonadherence to mammography services (29). Among CKD patients, lifetime discrimination was associated with lower odds of desiring a kidney transplant, suggesting that patients with significant previous exposure to discrimination do not want to risk new treatment situations, such as transplantation, because they have a lower expectation of successful outcomes (60). Using longitudinal data obtained from the Study of Women's Health Across the Nation ( $n = 2063$ ; mean age at baseline = 46.0), Upchurch et al. (28) found that race and SES's total effect on women's allostatic load was at least partially mediated by psychosocial factors such as perceived discrimination, perceived stress, and hostility. Another recent study exploring the association between perceived racism and ambulatory blood pressure among Hispanics reported that lower perceived racism was associated with ambulatory blood pressure nondipping, a cardiovascular risk factor, only among black Hispanics. This reveals a coping mechanism among this group that differs from white Hispanics (61). A third recent study examining heart rate variability across three racial groups (black, brown, and white) found a gradient (black > brown > whites) in heart rate variability that was clearly mediated by perceived discrimination (27). Examining sleep quality outcomes, another recent study reported that perceived discrimination mediated racial differences in most sleep quality measures, with nonwhites consistently showing poor sleep outcomes compared with whites (26). Finally, a study of multiple ethnic groups reported that perceived ethnic discrimination was positively associated with the metabolic syndrome and that ethnic differences in

metabolic syndrome were partially explained by this discrimination measure (25).

Our study had limitations, including residual confounding, specifically by time-dependent blood pressure, urinary albumin excretion, and apolipoprotein L1 risk variants among AAs. Third, perceived discrimination may have a different effect on kidney function decline from personally mediated or internalized forms of racism or sexism, which we did not examine. Fourth, kidney function decline was estimated only from two measures, whereas baseline ACR data were incomplete. Fifth, significant declines in eGFR were a relatively rare event, with 45 participants (2.8%) declining to an, for example, eGFR of less than 60 mL/min/1.73 m<sup>2</sup> at follow-up and 150 (9.3%) declining to an eGFR between 60 and 90 mL/min/1.73 m<sup>2</sup>, which precluded examining the association of perceived discrimination with the development of significantly reduced kidney function. Related to this limitation, our sample had limited kidney function decline in relation to the two main exposures, yielding our finding of potentially limited clinical significance. Moreover, no valid data were available on whether participants had received a diagnosis of or treatment for CKD. Finally, given the sampling methodology and the large percentage of missing data between initial screening, baseline, and follow-up examinations, our study findings are generalizable only to urban US adults. Thus, future studies should include geographically diverse samples, ideally with multiple eGFR and ACR assessments and longer follow-up.

The limitations of our study are balanced by its longitudinal design and the elucidation of a novel risk factor for kidney function decline. If validated in other studies, our findings emphasize the role of psychosocial stressors as potentially modifiable risk factors for adverse kidney outcomes. Further intervention studies addressing psychosocial stressors and CKD are likely warranted, and future studies should also examine potential biomarkers that may mediate the relationship between perceived discrimination and kidney function decline.



**TABLE 3.** Baseline and Annual Rate of Change in eGFR by Everyday Discrimination (EOD), Overall and by Sex by Race: Mixed-Effects Linear Regression Models

Models	n	Model 1		Model 2		Model 3		Model 4	
		$\beta$ (SE)	p	$\beta$ (SE)	p	$\beta$ (SE)	p	$\beta$ (SE)	p
Overall	1616								
EOD <sub>10</sub>		-0.57 (0.95)	.54	-0.55 (0.95)	.56	-0.55 (0.96)	.57	-0.57 (0.96)	.56
EOD <sub>10</sub> by time		+0.20 (0.19)	.30	+0.19 (0.19)	.75	+0.18 (1.20)	.99	+0.19 (0.19)	.31
EOD <sub>20</sub>		+0.35 (1.18)	.77 <sup>a</sup>	+0.38 (1.18)	.75 <sup>a</sup>	+0.02 (1.20)	.99 <sup>a</sup>	+0.03 (1.20)	.98 <sup>a</sup>
EOD <sub>20</sub> by time		-0.12 (0.23)	.61	-0.12 (0.23)	.61	-0.04 (0.23)	.86	-0.05 (0.23)	.84
White women	381								
EOD <sub>10</sub>		+0.19 (1.78)	.92	+0.12 (1.78)	.95	-0.07 (1.83)	.97	-0.05 (1.83)	.98
EOD <sub>10</sub> by time		-0.08 (0.38)	.84	-0.06 (0.38)	.87	+0.12 (0.39)	.76	+0.10 (0.39)	.80
EOD <sub>20</sub>		-4.28 (2.43)	.078	-4.81 (2.43)	.048	-5.55 (2.51)	.027	-5.86 (2.52)	.020
EOD <sub>20</sub> by time		+0.12 (0.52)	.82	+0.25 (0.52)	.64	+0.72 (0.53)	.18	+0.65 (0.53)	.22
White men	278								
EOD <sub>10</sub>		+0.91 (1.64)	.56	+0.99 (1.61)	.54	+0.01 (1.64)	1.00	-0.03 (1.64)	.99
EOD <sub>10</sub> by time		-0.28 (0.33)	.40	-0.31 (0.33)	.34	-0.24 (0.34)	1.00	-0.25 (0.33)	.46
EOD <sub>20</sub>		+4.98 (2.19)	.023	+3.49 (2.16)	.11	+2.83 (2.19)	.20	+2.77 (2.21)	.21
EOD <sub>20</sub> by time		-0.75 (0.44)	.088	-0.60 (0.44)	.17	-0.52 (0.45)	.25	-0.55 (0.45)	.22
AA women	565								
EOD <sub>10</sub>		-1.66 (1.83)	.37	-1.13 (1.84)	.54	-1.04 (1.86)	.58	-1.08 (1.86)	.56
EOD <sub>10</sub> by time		+0.40 (0.34)	.23	+0.35 (0.34)	.30	+0.40 (0.34)	.37	+0.40 (0.34)	.24
EOD <sub>20</sub>		+3.49 (2.39)	.14	+3.67 (2.39)	.13	+3.67 (2.43)	.13	+3.67 (2.43)	.13
EOD <sub>20</sub> by time		-0.58 (0.45)	.19	-0.53 (0.45)	.24	-0.41 (0.46)	.37	-0.41 (0.46)	.37
AA men	392								
EOD <sub>10</sub>		-0.03 (2.02)	.99	+0.00 (2.02)	1.00	+0.22 (2.02)	.91	+0.11 (2.01)	.96
EOD <sub>10</sub> by time		+0.33 (0.39)	.40	+0.29 (0.39)	.31	+0.09 (0.39)	.82	+0.12 (0.38)	.76
EOD <sub>20</sub>		-0.79 (2.16)	.72	-0.50 (2.16)	.82	-0.65 (2.12)	.76	-0.48 (2.12)	.82
EOD <sub>20</sub> by time		+0.42 (0.41)	.31	+0.42 (0.41)	.31	+0.32 (0.41)	.43	+0.27 (0.40)	.50

SE = standard error; EOD = experience of discrimination.

Selected participants with preserved kidney function. EOD is coded as 2 = high, 1 = medium, 0 = low. For instance, EOD<sub>10</sub> refers to medium EOD contrasted with low EOD. Model 1: adjusted for inverse mills ratio, age, sex, and race; model 2: further adjusted for poverty status, marital status, and educational level; model 3: further adjusted for current smoking status and illicit drug use, BMI, self-rated health, and EDS; model 4: further adjusted for diabetes and hypertension.

<sup>a</sup>In a separate model with sex by race (0 = white women versus each of the other categories), sex by race by time, sex by race by EOD<sub>k0</sub>, sex by race by EOD<sub>k0</sub> by time, (in addition to the other covariates in each model),  $p < .05$  for the null hypothesis that the term sex by race by EOD<sub>k0</sub> = 0.

In conclusion, in this sample of urban adults, PGD was associated with modestly lower kidney function among white women and AA men. Consistent findings were observed among AA women with respect to PRD and lower kidney function. Perceived discrimination, a psychosocial stressor, deserves further investigation for its potential contribution to kidney outcomes.

*Source of Funding and Conflicts of Interest:* This work was supported by the Intramural Research Program of the National Institute on Aging, National Institutes of Health. D.C.C. was supported by grant K23DK097184 from the National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. The authors report no conflicts of interest.

M.A.B. contributed in study concept, literature search and review, plan of analysis, data management, statistical analysis, and write-up and revision of the article. A.P.B. contributed in plan of analysis, data management, literature search and review, and write-up and revision of the article. A.B.Z. contributed in data

acquisition, plan of analysis, write-up of parts of the article, and revision of the article. O.S.R. contributed in plan of analysis, write-up of parts of the article, and revision of the article. M.K.E. contributed in data acquisition and revision of the article. D.C.C. contributed in plan of analysis, literature search and review, write-up of parts of the article, and revision of the article.

## REFERENCES

- Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, Van Lente F, Levey AS. Prevalence of chronic kidney disease in the United States. *JAMA* 2007;298:2038–47.
- Parsa A, Kao WH, Xie D, Astor BC, Li M, Hsu CY, Feldman HI, Parekh RS, Kusek JW, Greene TH, Fink JC, Anderson AH, Choi MJ, Wright JT Jr., Lash JP, Freedman BI, Ojo A, Winkler CA, Raj DS, Kopp JB, He J, Jensvold NG, Tao K, Lipkowitz MS, Appel LJ, AASK Study Investigators, CRIC Study Investigators. APOL1 risk variants, race, and progression of chronic kidney disease. *N Engl J Med* 2013;369:2183–96.
- Crews DC, Pfaff T, Powe NR. Socioeconomic factors and racial disparities in kidney disease outcomes. *Semin Nephrol* 2013;33:468–75.
- Bruce MA, Griffith DM, Thorpe RJ Jr. Stress and the kidney. *Adv Chronic Kidney Dis* 2015;22:46–53.

5. Williams DR, Neighbors HW, Jackson JS. Racial/ethnic discrimination and health: findings from community studies. *Am J Public Health* 2003; 93:200–8.
6. Cohen S, Kessler RC, Gordon LU. Personality Characteristics as Moderators of the Relationship Between Stress and Disorder. *Measuring Stress*, New York, NY: Oxford University Press; 1995.
7. Kovach JA, Nearing BD, Verrier RL. Angerlike behavioral state potentiates myocardial ischemia-induced T-wave alternans in canines. *J Am Coll Cardiol* 2001; 37:1719–25.
8. Lind L, Johansson K, Hall J. The effects of mental stress and the cold pressure test on flow-mediated vasodilation. *Blood Press* 2002;11:22–7.
9. Rosmond R. Role of stress in the pathogenesis of the metabolic syndrome. *Psychoneuroendocrinology* 2005;30:1–10.
10. Williams JE, Nieto FJ, Sanford CP, Couper DJ, Tyroler HA. The association between trait anger and incident stroke risk: the Atherosclerosis Risk in Communities (ARIC) Study. *Stroke* 2002;33:13–9.
11. Bratter JL, Gorman BK. Is discrimination an equal opportunity risk?: racial experiences, socioeconomic status, and health status among black and white adults. *J Health Soc Behav* 2011;52:365–82.
12. Krieger N. Racial and gender discrimination: risk factors for high blood pressure? *Soc Sci Med* 1990;30:1273–81.
13. Ro AE, Choi KH. Social status correlates of reporting gender discrimination and racial discrimination among racially diverse women. *Women Health* 2009; 49:1–15.
14. Shelton RC, Puleo E, Bennett GG, McNeill LH, Sorensen G, Emmons KM. The association between racial and gender discrimination and body mass index among residents living in lower-income housing. *Ethn Dis* 2009;19:251–7.
15. Borrell C, Muntaner C, Gil-González D, Artazcoz L, Rodríguez-Sanz M, Rohlfs I, Pérez K, García-Calvente M, Villegas R, Alvarez-Dardet C. Perceived discrimination and health by gender, social class, and country of birth in a Southern European country. *Prev Med* 2010;50:86–92.
16. Hahm HC, Ozonoff A, Gaumond J, Sue S. Perceived discrimination and health outcomes a gender comparison among Asian-Americans nationwide. *Womens Health Issues* 2010;20:350–8.
17. Brodish AB, Cogburn CD, Fuller-Rowell TE, Peck S, Malanchuk O, Eccles JS. Perceived racial discrimination as a predictor of health behaviors: the moderating role of gender. *Race Soc Probl* 2011;3:160–9.
18. Cunningham TJ, Seeman TE, Kawachi I, Gortmaker SL, Jacobs DR, Kiefe CI, Berkman LF. Racial/ethnic and gender differences in the association between self-reported experiences of racial/ethnic discrimination and inflammation in the CARDIA cohort of 4 US communities. *Soc Sci Med* 2012;75:922–31.
19. Perry BL, Harp KL, Oser CB. Racial and gender discrimination in the stress process: implications for African American women's health and well-being. *Sociol Perspect* 2013;56:25–48.
20. Kim IH, Noh S. Ethnic and gender differences in the association between discrimination and depressive symptoms among five immigrant groups. *J Immigr Minor Health* 2014;16:1167–75.
21. McDonald JA, Terry MB, Tehranifar P. Racial and gender discrimination, early life factors, and chronic physical health conditions in midlife. *Womens Health Issues* 2014;24:e53–9.
22. Otiniano Verissimo AD, Gee GC, Ford CL, Iguchi MY. Racial discrimination, gender discrimination, and substance abuse among Latina/os nationwide. *Cultur Divers Ethnic Minor Psychol* 2014;20:43–51.
23. Otiniano Verissimo AD, Grella CE, Amaro H, Gee GC. Discrimination and substance use disorders among Latinos: the role of gender, nativity, and ethnicity. *Am J Public Health* 2014;104:1421–8.
24. Brondolo E, Monge A, Agosta J, Tobin JN, Cassells A, Stanton C, Schwartz J. Perceived ethnic discrimination and cigarette smoking: examining the moderating effects of race/ethnicity and gender in a sample of Black and Latino urban adults. *J Behav Med* 2015;38:689–700.
25. Ikram UZ, Snijder MB, Agyemang C, Schene AH, Peters RJ, Stronks K, Kunst AE. Perceived ethnic discrimination and the metabolic syndrome in ethnic minority groups: the healthy life in an urban setting study. *Psychosom Med* 2017; 79:101–11.
26. Owens SL, Hunte HE, Sterkel A, Johnson DA, Johnson-Lawrence V. Association between discrimination and objective and subjective sleep measures in the midlife in the United States study adult sample. *Psychosom Med* 2017; 79:469–78.
27. Kemp AH, Koenig J, Thayer JF, Bittencourt MS, Pereira AC, Santos IS, Dantas EM, Mill JG, Chor D, Ribeiro AL, Benseñor IM, Lotufo PA. Race and resting-state heart rate variability in Brazilian civil servants and the mediating effects of discrimination: an ELSA-Brasil cohort study. *Psychosom Med* 2016; 78:950–8.
28. Upchurch DM, Stein J, Greendale GA, Chyu L, Tseng CH, Huang MH, Lewis TT, Kravitz HM, Seeman T. A longitudinal investigation of race, socioeconomic status, and psychosocial mediators of allostatic load in midlife women: findings from the study of women's health across the nation. *Psychosom Med* 2015; 77:402–12.
29. Dailey AB, Kasl SV, Jones BA. Does gender discrimination impact regular mammography screening? Findings from the race differences in screening mammography study. *J Womens Health (Larchmt)* 2008;17:195–206.
30. Evans MK, Lepkowski JM, Powe NR, LaVeist T, Kuczmarski MF, Zonderman AB. Healthy aging in neighborhoods of diversity across the life span (HANDLS): overcoming barriers to implementing a longitudinal, epidemiologic, urban study of health, race, and socioeconomic status. *Ethn Dis* 2010;20:267–75.
31. Krieger N, Smith K, Naishadham D, Hartman C, Barbeau EM. Experiences of discrimination: validity and reliability of a self-report measure for population health research on racism and health. *Soc Sci Med* 2005; 61:1576–96.
32. Williams DR. Measuring Discrimination Resource. Available at: [http://scholar.harvard.edu/files/davidrwilliams/files/measuring\\_discrimination\\_resource\\_feb\\_2012\\_0\\_0.pdf](http://scholar.harvard.edu/files/davidrwilliams/files/measuring_discrimination_resource_feb_2012_0_0.pdf). 2012. Accessed August 1, 2016.
33. Essed P. *Understanding Everyday Racism*. Newbury Park, CA: Sage; 1991.
34. Williams DR, Yan Yu, Jackson JS, Anderson NB. Racial differences in physical and mental health: socio-economic status, stress and discrimination. *J Health Psychol* 1997;2:335–51.
35. Levey AS, Stevens LA, Schmid CH, Zhang YL, Castro AF 3rd, Feldman HI, Kusek JW, Eggers P, Van Lente F, Greene T, Coresh J, CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration). A new equation to estimate glomerular filtration rate. *Ann Intern Med* 2009;150:604–12.
36. Peralta CA, Vittinghoff E, Bansal N, Jacobs D Jr., Muntner P, Kestenbaum B, Lewis C, Siscovick D, Kramer H, Shlipak M, Bibbins-Domingo K. 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. *Am J Kidney Dis [Comparative Study Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't]*. 2013;62:261–6.
37. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr., Jones DW, Materson BJ, Oparil S, Wright JT Jr., Roccella EJ, National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, National High Blood Pressure Education Program Coordinating Committee. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003;289:2560–72.
38. Radloff L. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Measur* 1977;1:385–401.
39. Beydoun MA, Fanelli Kuczmarski MT, Beydoun HA, Hibbeln JR, Evans MK, Zonderman AB.  $\omega$ -3 fatty acid intakes are inversely related to elevated depressive symptoms among United States women. *J Nutr* 2013;143:1743–52.
40. Beydoun MA, Beydoun HA, Kitner-Triolo MH, Kaufman JS, Evans MK, Zonderman AB. Thyroid hormones are associated with cognitive function: moderation by sex, race, and depressive symptoms. *J Clin Endocrinol Metab* 2013;98:3470–81.
41. Heckman JJ. Sample selection bias as a specification error. *Econometrica* 1979; 47:153–61.
42. Landrine H, Klonoff EA. *African American Acculturation: Deconstructing Race and Reviving Culture*. Thousand Oaks, CA: Sage; 1996.
43. Guidi J, Offidani E, Rafanelli C, Roncuzzi R, Sonino N, Fava GA. The assessment of allostatic overload in patients with congestive heart failure by clinimetric criteria. *Stress Health* 2014;32:63–9.
44. McClellan WM, Newsome BB, McClure LA, Howard G, Volkova N, Audhya P, Wamock DG. Poverty and racial disparities in kidney disease: the REGARDS study. *Am J Nephrol* 2010;32:38–46.
45. Peralta CA, Katz R, DeBoer I, Ix J, Sarnak M, Kramer H, Siscovick D, Shea S, Szklo M, Shlipak M. Racial and ethnic differences in kidney function decline among persons without chronic kidney disease. *J Am Soc Nephrol* 2011;22:1327–34.
46. Collins AJ, Foley RN, Chavers B, Gilbertson D, Herzog C, Johansen K, Kasiske B, Kutner N, Liu J, St Peter W, Guo H, Gustafson S, Heubner B, Lamb K, Li S, Li S, Peng Y, Qiu Y, Roberts T, Skeans M, Synder J, Solid C, Thompson B, Wang C, Weinhandl E, Zau D, Arko C, Chen SC, Daniels F, Ebben J, Frazier F, Hanzlik C, Johnson R, Sheets D, Wang X, Forrest B, Constantini E, Everson S, Eggers P, Agoda L. United States Renal Data System: USRDS. Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States. *Am J Kidney Dis* 2012;59:A7, e1–420.
47. Kramer H, Palmas W, Kestenbaum B, Cushman M, Allison M, Astor B, Shlipak M. Chronic kidney disease prevalence estimates among racial/ethnic groups: the Multi-Ethnic Study of Atherosclerosis. *Clin J Am Soc Nephrol* 2008; 3:1391–7.
48. Grams ME, Chow EK, Segev DL, Coresh J. Lifetime incidence of CKD stages 3–5 in the United States. *Am J Kidney Dis* 2013;62:245–52.
49. Tsigos C, Chrousos GP. Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. *J Psychosom Res* 2002;53:865–71.
50. Szanton SL, Rifkind JM, Mohanty JG, Miller ER 3rd, Thorpe RJ, Nagababu E, Epel ES, Zonderman AB, Evans MK. Racial discrimination is associated with a measure of red blood cell oxidative stress: a potential pathway for racial health disparities. *Int J Behav Med* 2012;19:489–95.
51. McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. *Ann N Y Acad Sci* 1998;840:33–44.
52. Kooman JP, Kotanko P, Schols AM, Shiels PG, Stenvinkel P. Chronic kidney disease and premature ageing. *Nat Rev Nephrol* 2014;10:732–42.
53. Saban KL, Mathews HL, DeVon HA, Janusek LW. Epigenetics and social context: implications for disparity in cardiovascular disease. *Aging Dis* 2014; 5:346–55.

54. Gemmell LA, Terhorst L, Jhamb M, Unruh M, Myaskovsky L, Kester L, Steel JL. Gender and racial differences in stress, coping, and health-related quality of life in chronic kidney disease. *J Pain Symptom Manage* 2016; 52:806–12.
55. Sanders-Phillips K, Settles-Reaves B, Walker D, Brownlow J. Social inequality and racial discrimination: risk factors for health disparities in children of color. *Pediatrics* 2009;124(Suppl 3):S176–86.
56. Ahmed AT, Mohammed SA, Williams DR. Racial discrimination & health: pathways & evidence. *Indian J Med Res* 2007;126:318–27.
57. Cuffee YL, Hargraves JL, Rosal M, Briesacher BA, Schoenthaler A, Person S, Hullett S, Allison J. Reported racial discrimination, trust in physicians, and medication adherence among inner-city African Americans with hypertension. *Am J Public Health* 2013;103:e55–62.
58. Casagrande SS, Gary TL, LaVeist TA, Gaskin DJ, Cooper LA. Perceived discrimination and adherence to medical care in a racially integrated community. *J Gen Intern Med* 2007;22:389–95.
59. Reynolds DB, Walker RJ, Campbell JA, Egede LE. Differential effect of race, education, gender, and language discrimination on glycemic control in adults with type 2 diabetes. *Diabetes Technol Ther* 2015;17:243–7.
60. Klassen AC, Hall AG, Saksvig B, Curbow B, Klassen DK. Relationship between patients' perceptions of disadvantage and discrimination and listing for kidney transplantation. *Am J Public Health* 2002;92:811–7.
61. Rodriguez CJ, Gwathmey TM, Jin Z, Schwartz J, Beech BM, Sacco RL, Di Tullio MR, Homma S. Perceived Discrimination and Nocturnal Blood Pressure Dipping Among Hispanics: The Influence of Social Support and Race. *Psychosom Med* 2016;78:841–50.