Racial Differences in the Effectiveness of a Multifactorial Telehealth Intervention to Slow Diabetic Kidney Disease

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Background: African Americans are significantly more likely than non-African Americans to have diabetes, chronic kidney disease, and uncontrolled hypertension, increasing their risk for kidney function decline.

Objective: The objective of this study was to compare how African Americans and non-African Americans with diabetes responded to a multifactorial telehealth intervention designed to slow kidney function decline.

Research Design: Secondary analysis of a randomized trial. Primary care patients (N = 281, 56% African American) were allocated to either: (1) a multifactorial, pharmacist-delivered phone-based telehealth intervention focused on behavioral and medication management of diabetic kidney disease; or (2) an education control.

Measures: The primary study outcome was change in estimated glomerular filtration rate (eGFR). Linear mixed models were used to explore the moderating effect of race on the relationship between study arm and eGFR decline over time; the mean annual rate of eGFR decline was estimated by race and study arm.

Results: Findings demonstrated a differential intervention effect on kidney function over time by race ($P_{interaction} = 0.005$). Among African Americans, the intervention arm had significantly greater preservation of eGFR over time than the control arm (difference in the annual rate of eGFR decline = 1.5 mL/min/1.73 m^2; 95% confidence interval: 0.04, 3.02). For non-African Americans, the intervention arm had a faster decline in eGFR over time than the control arm (difference in the annual rate of eGFR decline = −1.7 mL/min/1.73 m^2; 95% confidence interval: −3.3, −0.02).

Conclusion: A multifactorial, pharmacist-delivered telehealth intervention for diabetic kidney disease may be more effective for slowing eGFR decline among African Americans than non-African Americans.

Key Words: diabetic kidney disease, racial disparities, telehealth intervention

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Type 2 diabetic kidney disease (DKD) is the leading cause of chronic kidney disease (CKD) worldwide.[1] The prevalence of diabetes and advanced DKD is particularly high among racial minorities.[1–3] Compared with non-African Americans, the high prevalence of DKD in African Americans is partially attributable to the increased prevalence of uncontrolled hypertension (41% vs. 28%), peripheral arterial disease (11.6% vs. 5.5%), and obesity (47% vs. 38%).[4–8] These disparities highlight the need for interventions focusing on modification of self-management behaviors important in DKD—such as medication adherence, diet, and exercise—among African Americans.

The *S*imultaneous Risk Factor Control Using *T*elehealth to sl*O*w *P*rogression of *D*iabetic *K*idney *D*isease Study (STOP-DKD, NCT01829256) examined the impact of pharmacist-delivered medication management and behavioral telehealth intervention on kidney function decline over 3 years of follow-up in adults with evidence for DKD and poorly controlled hypertension.[9] While the main results of this study showed no difference in attenuation of kidney function decline between intervention and control, this secondary analysis sought to determine whether there were differences in intervention response between African Americans and non-African Americans. The

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objective of this analysis was to understand how telehealth-based approaches might be employed to reduce racial disparities in DKD progression.

METHODS

STOP-DKD participants received usual care by their primary care physician and entered either: (1) a multifactorial, pharmacist-delivered phone-based telehealth intervention focused on behavioral and medication management; or (2) an education control, consisting of printed DKD management resources.[10] All intervention treatment recommendations were protocol-based and shared with the participant's primary care physician via the electronic health record for implementation.

All study procedures and protocols were approved by the Duke University Institutional Review Board. All subjects provided written informed consent.

Participant Eligibility

STOP-DKD participants were recruited from Duke-affiliated primary care clinics. Inclusion criteria included: (1) adult (age 18 y and older and 75 y and younger); (2) regular use of the Duke University Health System (≥ 2 primary care visits in 3 prior years); (3) diagnosis of type 2 diabetes [International Classification of Diseases, Ninth Revision (ICD-9) codes 250.×0, 250.×2]; (4) at least 2 serum creatinine values available in the 3 prior years, separated by at least 3 months; (5) preserved kidney function [estimated glomerular filtration rate (eGFR) ≥ 45 mL/min/1.73 m^2 on most recent creatinine estimated by calculating an eGFR using the 4-variable Modification of Diet in Renal Disease Study equation]; (6) evidence of diabetic nephropathy; (7) poorly controlled hypertension (2 blood pressure values of systolic blood pressure ≥ 140 mm Hg and/or diastolic blood pressure ≥ 90 mm Hg over 1 y); and (8) prescribed hypertension and diabetes medications.

Exclusion criteria included: lack of telephone access; lack of proficiency in English; residency in a nursing home/long-term care facility or receipt of home health care; impaired hearing/speech/vision; participation in another clinical trial (pharmaceutical or behavioral); plans to leave the area within 3 years; pancreatic insufficiency or diabetes secondary to pancreatitis; alcohol abuse (>14 alcoholic beverages/wk); diagnosis of non-DKD; active malignancy; life-threatening illness and probable death within 4 years; secondary hypertension; pregnancy and/or breastfeeding; long-term or chronic dialysis; dementia; and receipt of renal transplant.

Randomization

Two-arm randomization was stratified by eGFR (eGFR = 45–60 vs. >60 mL/min/1.73 m^2) and race (African American vs. non-African American).

Study Outcome

The primary outcome was estimated glomerular filtration rate (eGFR) at 36 months, calculated using the CKD-EPI creatinine equation.[11]

Additional Measures

Anthropometric measurements, biospecimen labs, self-reported demographics, and survey data were collected at baseline and yearly follow-up visits for 3 years.

For these analyses, the race was dichotomized into African American and non-African American (White/Caucasian, Asian, American Indian/Alaska, Native, or Other) due to a majority of participants self-reporting African American or White/Caucasian race.

Analyses

Demographics and baseline characteristics were summarized using descriptive statistics. Differences were assessed using 2-sample t tests for continuous variables and χ^2 tests for categorical variables. All analyses were conducted on an intention-to-treat basis.

A multivariable linear mixed model with a random intercept for participants was used to examine the intervention's differential effects on eGFR decline over time by race. Fixed effects included indicator variables for intervention and African American race, a continuous-time variable, and all 2-way and 3-way interaction terms between these variables. A common baseline across study arms within the race was not assumed due to small differences in eGFR between arms at baseline. The 3-way intervention by race by time interaction term was used to test the overall effect of race by arm over time. Appropriate contrasts of the estimated parameters were constructed and tested to estimate the annual rate of eGFR decline within each arm by race subgroups and to compare the rates of decline between arms within race subgroups. We also adjusted the primary model by baseline eGFR to determine if results were sensitive to baseline differences in eGFR between the race groups. We also assessed missingness in exploratory analyses and performed multiple imputation to determine if our results were sensitive to missing outcome data.

To corroborate findings from the primary analysis, we also constructed a linear mixed-effects model with a categorical time variable. With baseline visit as a reference, the 3 indicator functions corresponded to follow-up at 12, 24, and 36 months. Fixed effects included all 2-way and 3-way interaction terms between treatment arm, race, and visit indicator variables. Similarly, we estimated mean eGFR at each visit for each arm by race subgroups, and compared mean eGFR across arms within race subgroup over time.

Analyses were performed using SAS 9.4 (SAS Institute, Cary, NC) and R 3.4.4 (R Core Team, Vienna, Austria). Hypothesis tests were 2 sided. No adjustment for multiple testing was performed.

RESULTS

Participant Population

Among the 281 individuals in this study, 52% were male and mean (SD) age was 62 (8.8) years; 55.5% (n = 156) self-reported African American race, 41.3% (n = 116) non-Hispanic White, and 3.2% (n = 9) some other race (eg, Asian, American Indian/Alaska, Native, or Other). At baseline, mean (SD) eGFR was 81 (21.7) mL/min/1.73 m^2 overall, 85 (22.6)

TABLE 1. STOP-DKD Baseline Sample Characteristics, Overall, and Stratified by Race

Baseline Characteristics*	n (%) Overall (N = 281)	Non-AA (N = 125)	AA (N = 156)	*P*
Age [mean (SD)] (y)	62 (8.8)	64 (8.1)	61 (9.2)	< 0.001
Male	145 (51.6)	73 (58.4)	72 (46.2)	0.05
AA	156 (55.5)	0 (0)	156 (100)	
Education				0.69
High school or less	90 (32.0)	37 (29.6)	53 (34.0)	
Some college or technical school	94 (33.5)	44 (35.2)	50 (32.1)	
College graduate or more	95 (33.8)	44 (35.2)	51 (32.7)	
Retired or disabled (yes)	170 (60.5)	79 (63.2)	91 (58.3)	0.56
Household income				0.01
< $60,000	159 (56.6)	59 (47.2)	100 (64.1)	
> $60,000	112 (39.9)	63 (50.4)	49 (31.4)	
Health insurance (yes)	273 (97.2)	123 (98.4)	150 (96.2)	0.88
BMI [mean (SD)]	35.7 (7.9)	35.3 (7.9)	36.0 (7.9)	0.47
Medication Adherence Score† [mean (SD)]	1.7 (1.6)	1.5 (1.6)	1.9 (1.6)	0.05
Medication nonadherent† (MM > 2) (yes)	97 (34.5)	36 (28.8)	61 (39.1)	0.08
Depressive symptoms‡ (PHQ ≥ 2) (yes)	54 (19.2)	27 (21.6)	27 (17.3)	0.48
Cystatin C [mean (SD)]	1.1 (0.30)	1.2 (0.34)	1.0 (0.25)	< 0.001
Potassium [mean (SD)]	4.4 (0.47)	4.5 (0.46)	4.3 (0.44)	0.01
Systolic blood pressure [mean (SD)]	134 (19.5)	132 (18.3)	136 (20.2)	0.04
Diastolic blood pressure [mean (SD)]	76 (13.5)	72 (12.1)	80 (13.9)	< 0.001
Creatinine [mean (SD)]	1.0 (0.29)	1.0 (0.31)	1.0 (0.28)	0.8
ACR [mean (SD)]	186 (539.8)	191 (668.8)	182 (407.2)	0.9
Albuminuria (ACR ≥ 30)	135 (48.0)	63 (50.4)	72 (46.2)	0.67
eGFR by creatinine [mean (SD)]	81 (21.7)	75 (19.3)	85 (22.6)	< 0.001
CKD awareness§ (yes)	19 (6.8)	10 (8.0)	9 (5.8)	0.34
A1c [mean (SD)]	8.0 (1.8)	7.8 (1.6)	8.1 (2.0)	0.12
A1c categorized				0.06
5 ≤ A1c < 7	95 (33.8)	41 (32.8)	54 (34.6)	
7 ≤ A1c < 8.5	97 (34.5)	52 (41.6)	45 (28.9)	
8.5 ≤ A1c ≤ 15.1	88 (31.3)	32 (25.6)	56 (35.9)	
Out of control A1c‖ (yes)	126 (44.8)	54 (43.2)	72 (46.2)	0.67

*Two participants in the AA group were missing data for education level, employment status, health insurance status, medication adherence, depressive symptoms, and CKD awareness. For household income, 7 participants in the AA group were either missing data, refused response, or did not know and 3 participants in the non-AA group did not know. Seven participants in the AA subgroup and 3 participants in the non-AA group were missing data for albuminuria. One participant in the AA group was missing data for A1c. Those with missing data were included in the percentage calculations.

†Medication adhere was assessed using an 8-item measure of self-report adherence. Questions were reverse-scored such that higher scores indicated poorer medication adherence.[12] Medication nonadherence was defined by a total score > 2.

‡Depression was assessed using the PHQ-2, which evaluates the frequency of depressed mood and anhedonia over the previous 2 weeks.[13] Scores ranged from 0 to 6, with a score of ≥ 2 indicating depressive symptoms.

§CKD awareness was assessed by the question, "Have you ever been told by a doctor or other health professional that you had weak or failing kidneys? Do not include kidney stones, bladder infections, or incontinence." Responses were dichotomized as yes (CKD aware) or no (CKD unaware).[14]

‖Out of control HbA1c defined as ≥ 8%.

AA indicates African American; ACR, albumin creatinine ratio (mg/g); BMI, body mass index (kg/m^2); CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate (mL/min/1.73 m^2); MM, Morisky Medication Score; PHQ, Patient Health Questionnaire; STOP-DKD, *S*imultaneous Risk Factor Control Using *T*elehealth to sl*O*w *P*rogression of *D*iabetic *K*idney *D*isease Study.

among African Americans, and 75 (19.3) among non-African Americans.

Compared with non-African Americans, African Americans were younger (61 vs. 64 y), more likely to be female (54% vs. 42%), have an annual income <$60k (64% vs. 47%), less likely to be medication adherent (39% vs. 29%), and had slightly higher systolic (136 vs. 132 mm Hg) and diastolic (80 vs. 72 mm Hg) blood pressure at baseline. The racial subgroups were otherwise similar at baseline (Table 1).

Differences in Estimated Glomerular Filtration Rate Decline

At each timepoint, African Americans had higher eGFR compared with non-African Americans (Figs. 1A, B). In multivariable mixed models, African Americans receiving the intervention had a slower mean rate of annual decline in eGFR than control [−2.5 mL/min/1.73 m^2; 95%confidence interval (CI): −3.5, −1.4 vs. −4.0 mL/min/1.73 m^2; 95% CI: −5.1, −2.9], while non-African Americans receiving the intervention had faster decline than control (−3.4; 95% CI: −4.6, −2.3 vs. −1.8; 95% CI −2.9, −0.6) (Table 2). There was evidence for a differential intervention effect over time between the racial subgroups ($P_{interaction} = 0.005$). Results did not change substantively with baseline adjustment for eGFR or multiple imputation (results not shown).

Similar results were seen with time modeled categorically (Fig. 1C). The between-arm difference in eGFR decline from baseline between racial subgroups was significantly different at 36 months ($P = 0.006$), but not at 12 months ($P = 0.059$) or 24 months ($P = 0.11$) (Tables 3, 4). African Americans in the intervention had the highest predicted mean eGFR at 36 months of all race by intervention arms.

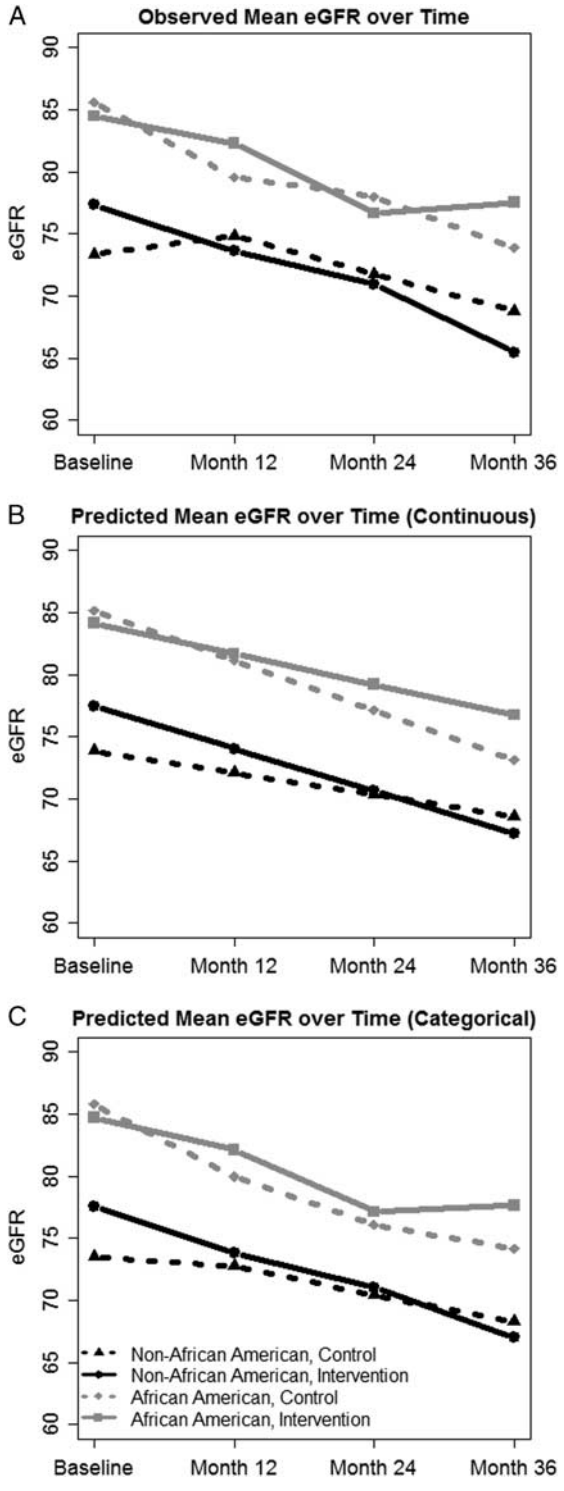

FIGURE 1. Observed and predicted mean eGFR over time. A, Observed mean eGFR over time. B, Primary analysis: model predicted eGFR over time (continuous). C, Secondary analysis: model predicted eGFR over time (categorical). eGFR indicates estimated glomerular filtration rate (mL/min/1.73 m^2). eGFR indicates estimated glomerular filtration rate.

TABLE 2. Predicted Annual Rate of eGFR Decline for Each Racial Subgroup

Race Category	Intervention Predicted Rate (95% CI)	Control Predicted Rate (95% CI)	Difference in Annual Rate for Intervention vs. Control (95% CI); *P*
AA	−2.5 (−3.5, −1.4)	−4.0 (−5.1, −2.9)	1.5 (0.04, 3.02); 0.044
Non-AA	−3.4 (−4.6, −2.3)	−1.8 (−2.9, −0.6)	−1.7 (−3.3, −0.02); 0.047

AA indicates African American; CI, confidence interval; eGFR, estimated glomerular filtration rate.

DISCUSSION

This secondary analysis examined racial differences in the effects of a multifactorial telehealth intervention focused on behavioral and medication management among adults with DKD and hypertension. Our analyses suggested the intervention was more effective among African Americans than non-African Americans. African Americans receiving the intervention experienced a lower rate of eGFR decline compared with control. The categorical time model showed this difference may take time to develop, suggesting that reductions in kidney function decline may accumulate over time. Even so, African Americans in the intervention experienced a clinically significant decline in eGFR of 2.5 mL/min/1.73 m^2 per year, suggesting a more intensive approach may be needed for the preservation of eGFR.

Our findings suggest that interventions targeting multiple modifiable behaviors, such as medication adherence, may attenuate some racial disparities in the burden of DKD. Despite the similar prevalence of overall DKD across races in the United States, advanced DKD and contributing conditions such as hypertension, cardiovascular disease, and obesity, disproportionately affect racial minorities.[4–8,15–19] Interventions like STOP-DKD, while perhaps not designed specifically to address racial disparities, may help bridge these disparities by targeting key factors involved in DKD progression, including uncontrolled hypertension, diabetes

TABLE 3. Predicted Mean eGFR and Estimated Differences in eGFR (95% CI) Between Study Arms Over Time

eGFR (mL/min/1.73 m^2)/ Subgroup/ Timepoint	Intervention Predicted Mean (95% CI)	Control Predicted Mean (95% CI)	Difference in eGFR for Intervention vs. Control (95% CI)
AA			
Baseline	84.5 (79.1, 89.3)	85.6 (80.9, 90.3)	−1.1 (−7.8, 5.6)
12 mo	81.9 (77.1, 86.7)	79.8 (74.9, 84.6)	2.1 (−4.7, 9.0)
24 mo	76.9 (72.0, 81.9)	75.9 (70.9, 80.8)	1.1 (−6.0, 8.1)
36 mo	77.4 (72.4, 82.4)	73.9 (68.9, 79.0)	3.5 (−3.7, 10.6)
Non-AA			
Baseline	77.3 (72.0, 82.7)	73.3 (68.1, 78.6)	4.0 (−3.5, 11.5)
12 mo	73.6 (68.1, 79.1)	72.6 (67.2, 78.0)	1.0 (−6.7, 8.7)
24 mo	70.8 (65.2, 76.4)	70.2 (64.7, 75.6)	0.6 (−7.2, 8.5)
36 mo	66.7 (61.1, 72.4)	68.0 (62.5, 73.6)	−1.3 (−9.2, 6.6)

AA indicates African American; CI, confidence interval; eGFR, estimated glomerular filtration rate.

TABLE 4. Predicted Differential Effect of STOP-DKD Compared With Control Over Time Among AAs Versus Non-AAs

Race Category	Change From Baseline (Difference in eGFR for Intervention vs. Control) (95% CI)	Difference in Change (AA−Non-AA) (95% CI); *P*
12 mo		
AA	3.2 (−1.1, 7.5)	6.2 (−0.2, 12.7); 0.059
Non-AA	−3.0 (−7.8, 1.8)	
24 mo		
AA	2.2 (−2.4, 6.7)	5.5 (−1.3, 12.3); 0.11
Non-AA	−3.4 (−8.4, 1.7)	
36 mo		
AA	4.6 (−0.2, 9.3)	9.9 (2.9, 16.9); 0.006
Non-AA	−5.3 (−10.5, −0.1)	

AA indicates African American; CI, confidence interval; eGFR, estimated glomerular filtration rate; STOP-DKD, *S*imultaneous Risk Factor Control Using *T*elehealth to sl*O*w *P*rogression of *D*iabetic *K*idney *D*isease Study.

self-management, health behaviors, and knowledge, communication skills, medication use, and side effects.

These results are consistent with prior reports on differential racial responses in blood pressure reduction in response to hypertension self-management and dietary interventions.[20–22] Our results, in conjunction with this body of literature, suggest lifestyle interventions are generally more efficacious among African Americans than non-African Americans.[20,23,24] However, it is not entirely clear which factors mediate these differences.

One possible explanation for the differential racial responses to lifestyle interventions may stem from disparities in social determinants of health. Socioeconomic factors, such as limited education, lack of health care access, and low income, are strong predictors for end-stage renal disease development.[25–29] Behavioral actions and cultural beliefs affect lifestyle factors such as diet, exercise, and medication adherence, as well as the patient perception of disease and social support.[30–34] Compared with non-African Americans, African Americans have less health insurance coverage and education, and are more likely to live in poverty, lack continuity in health care, and struggle with medication adherence.[25,35–37] These factors negatively impact access to health care, kidney transplants, and mortality in patients with end-stage renal disease.[38–42]

In our study, African Americans had lower adherence to medication use and lower household income at baseline. African Americans also trended toward having less awareness of their CKD status at baseline. STOP-DKD and similar multifactorial interventions directly address these barriers by improving access to care, engagement with health behaviors, and knowledge, and by providing added medication management support for hypertension and comorbid conditions. Addressing these health disparities may help explain the racial differences observed in this study and others.[20–22] Of note, there were no statistically significant differences in intervention adherence, as African Americans and non-African Americans completed a median of 32 and 33 (of 36) encounters with the pharmacist, respectively. These results further suggest that similar levels of engagement with multifactorial interventions like STOP-DKD may disproportionately benefit African Americans.

Of note, this analysis suggested that non-Africans receiving the intervention paradoxically fared worse than those receiving educational control. Given the borderline statistical significance of this finding, it is possible this within-subgroup difference may represent a type 1 error. Alternatively, there may be a threshold at which interventions like STOP-DKD become detrimental in populations with good access to care at baseline; it is possible that increased telehealth contact could potentially lead to fatigue, disengagement, and worse outcomes.[29] Additional research will be needed to confirm this finding and to understand how telehealth interventions should be tailored for specific populations.

This study has several limitations. STOP-DKD was not designed to detect differential subgroup responses to the intervention, limiting our ability to definitively conclude a differential racial effect. The presence of missing data in our analyses could also influence conclusions regarding baseline differences, although our sensitivity analysis with multiple imputation provides some reassurance against this possibility. In addition, it is unclear which multifactorial intervention components were most beneficial, and why these may have disproportionately affected African Americans compared with non-African Americans.

These analyses add to the growing literature suggesting differential racial responses to targeted interventions and may extend this literature to eGFR decline in individuals with DKD, a highly consequential outcome. Our findings highlight the need for patient-centered, multifactorial interventions capable of adequately addressing the needs of high-risk populations for kidney failure.

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