

Published in final edited form as:

Kidney Int. 2019 June; 95(6): 1433–1442. doi:10.1016/j.kint.2018.12.027.

Poor accordance to a DASH dietary pattern is associated with higher risk of ESRD among adults with moderate chronic kidney disease and hypertension

Tanushree Banerjee¹, Deidra C. Crews², Delphine S. Tuot³, Meda E. Pavkov⁴, Nilka Rios Burrows⁴, Austin G. Stack⁵, Rajiv Saran^{6,7}, Jennifer Bragg-Gresham⁶, Neil R. Powe^{1,8}, and Centers for Disease Control and Prevention Chronic Kidney Disease Surveillance Team⁹

¹Division of General Internal Medicine, Department of Medicine, University of California, San Francisco, California, USA; ²Division of Nephrology, Department of Medicine, Johns Hopkins University School of Medicine and Welch Center for Prevention, Epidemiology and Clinical Research, Johns Hopkins Medical Institutions, Baltimore, Maryland, USA; ³Division of Nephrology, Department of Medicine, University of California, San Francisco, California, USA; ⁴Division of Diabetes Translation, Centers of Disease and Control and Prevention, Atlanta, Georgia, USA; ⁵Department of Nephrology and Internal Medicine, University Hospital Limerick, Limerick, Ireland; ⁶Kidney Epidemiology & Cost Center, University of Michigan, Ann Arbor, Michigan, USA; ⁷Division of Nephrology, Department of Medicine and Kidney Epidemiology & Cost Center, University of Michigan, Ann Arbor, Michigan, USA; ⁸Department of Medicine, Zuckerberg San Francisco General Hospital, San Francisco, California, USA

Abstract

The Dietary Approaches to Stop Hypertension (DASH) diet lowers blood pressure, an important risk factor for chronic kidney disease (CKD) and end-stage renal disease (ESRD). However, it is unclear whether adherence to a DASH diet confers protection against future ESRD, especially among those with pre-existing CKD and hypertension. We examined whether a DASH diet is associated with lower risk of ESRD among 1,110 adults aged 20 years with hypertension and CKD (estimated glomerular filtration rate, eGFR 30–59 ml/min/1.73 m²) enrolled in the National Health and Nutrition Examination Survey (1988–1994). Baseline DASH diet accordance score was assessed using a 24-hour dietary recall questionnaire. ESRD was ascertained by linkage to the U.S. Renal Data System registry. We used the Fine-Gray competing risks method to estimate the relative hazard (RH) for ESRD after adjusting for sociodemographics, clinical and nutritional factors, eGFR, and albuminuria. Over a median follow-up of 7.8 years, 18.4% of subjects developed ESRD. Compared to the highest quintile of DASH diet accordance, there was a greater risk of ESRD among subjects in quintiles 1 (RH[1.7; 95% CI 1.1–2.7) and 2 (RH 2.2; 95% CI 1.1–

⁹ Members of the Centers for Disease Control and Prevention Chronic Kidney Disease Surveillance Team are listed in the Appendix. **Correspondence:** T. Banerjee, Priscilla Chan and Mark Zuckerberg San Francisco General Hospital, 1001 Potrero Avenue, Bldg 10, Ward 13, 1311N, San Francisco, California 94110, USA. tanushree.banerjee@ucsf.edu. DISCLOSURE

All the authors declared no competing interests.

Supplementary material is linked to the online version of the paper at www.kidney-international.org.

4.1). Significant interactions were observed with diabetes status and race/ethnicity, with the strongest association between DASH diet adherence and ESRD risk observed in individuals with diabetes and in non-Hispanic blacks. Low accordance to a DASH diet is associated with greater risk of ESRD in adults with moderate CKD and hypertension, particularly in non-Hispanic blacks and persons with diabetes.

Keywords

CKD; diet; diabetes; ESRD

The constituents of one's diet may confer important health benefits, especially among those with chronic medical conditions. For patients with chronic kidney disease (CKD), estimated to affect more than 20 million Americans, dietary approaches to prevent or slow the progression of CKD are of major interest and potential public health importance. Studies have suggested that unhealthy dietary patterns may contribute to kidney injury and disease progression. These consequences may in turn lead to further metabolic derangements and further deterioration in kidney function that may amplify the risks of cardiovascular diseases, disability, and mortality. 5–8

The Dietary Approaches to Stop Hypertension (DASH) diet, a dietary pattern rich in fruits and vegetables, has been proven to be effective in reducing blood pressure (BP), which is a primary risk factor for heart disease and stroke. Randomized controlled clinical trials have demonstrated the clinical efficacy of the DASH diet in lowering BP in high-risk individuals. ^{9,10} Although hypertension is a major risk factor for CKD, few studies have examined the association between a DASH dietary pattern and the risk of CKD progression. A study by Lin et al. 11 found that adherence to a DASH-type dietary pattern was associated with lower odds of estimated glomerular filtration rate (eGFR) decline among older white women, suggesting a potential benefit. Furthermore, observations by Goraya et al. 12,13 provide additional support for the hypothesis that diets with high fruit and vegetable components have favorable effects in patients with early and advanced CKD. However, current CKD guidelines do not recommend routine adoption of the DASH diet by patients with moderate kidney disease, ¹⁴ likely because the nutritional profile provides higher quantities of potassium, calcium, phosphorus, and protein than is recommended for patients with CKD stages 3 to 4. One might speculate that perhaps adults with hypertension and moderate impairment of kidney function could safely benefit from diets that improve BP without adverse consequences. Therefore, we undertook this study to examine the association of a DASH dietary pattern on risk of end-stage renal disease (ESRD) in adults with moderate CKD and hypertension.

RESULTS

Baseline characteristics

Among adults aged 20 years with moderate CKD (stage 3, defined as eGFR between 30 and 59 ml/min per 1.73 m² calculated using the Modification of Diet in Renal Disease [MDRD] study equation¹⁵) and hypertension, who did not have missing data on dietary

intake and were not pregnant, the mean age was 70.2 years. The median DASH diet accordance score, calculated using the method by Mellen *et al.*, 16 was 3.5 of a possible 9 (25th–75th percentile, 2.0–4.0). Adults in the higher quintiles of DASH diet accordance were more likely to be older, women, and have a higher body mass index (BMI) than those in the lower DASH quintiles (all P < 0.05) (Table 1).

DASH diet adherence and risk of ESRD

Among adults with moderate CKD and hypertension, 18.4% developed ESRD over 14.7 years (median follow-up, 7.8 years; interquartile range, 4.7–12.4). The percent of ESRD events by DASH diet accordance was 24.5% (quintile1), 26.2% (quintile 2), 18.7% (quintile 3), 16.5% (quintile4), and 15.9% (quintile 5) (Table 1). The unadjusted analysis showed a higher risk of ESRD in people with lower accordance scores compared with those with the greatest accordance score (quintile 5): relative hazard (RH), 2.6 (95% confidence interval, 1.9–3.8) for quintile 1; 2.1 (95% CI, 1.3–3.3) for quintile 2; 1.7 (95% CI, 1.1–2.4) for quintile 3, and 1.2 (95% CI, 0.7-1.8) for quintile 4 (Figure 1). Results were similar after adjustment for demographics (age, gender, race/ethnicity), socioeconomic position (SEP), diabetes, systolic BP, serum potassium, total caloric intake, BMI, eGFR, and albumin-tocreatinine ratio (ACR): RH 1.7 (95% CI, 1.1-2.7) for quintile 1, 2.2 (95% CI, 1.1-4.1) for quintile 2, 1.15 (95% CI, 0.6–1.8) for quintile 3, and 1.1 (95% CI, 0.7–1.7) for quintile 4 (P for trend 0.04; Figure 1). Further adjustment for the use of angiotensin-converting enzyme/ angiotensin-receptor blocker medication did not change the estimates for the association between DASH accordance score and ESRD (results not shown). The association between the individual nutrients of the DASH diet and risk of ESRD are presented in Supplementary Table S1.

Mediation analysis

On performing analyses, to examine mediating factors for the risk, the estimates were attenuated and the statistical significance was lost in the quintiles with respect to the highest quintile when dietary potassium and magnesium intake were added to the full model. In case of dietary potassium intake with respect to the highest quintile, RHs for the subsequent quintiles were 0.7 (95% CI, 0.4–1.11) for quintile 1; 0.6 (95% CI, 0.3–1.1) for quintile 2;0.4 (95% CI, 0.2–0.7) for quintile 3; and 0.2 (95% CI, 0.1–0.4) for quintile 4. In case of dietary magnesium intake RHs for the subsequent quintiles were 0.7 (95% CI, 0.4–1.2) for quintile 1,0.9 (95% CI, 0.5–1.8) for quintile 2, 0.3 (95% CI, 0.1–0.6) for quintile 3, and 0.2 (95% CI, 0.1–0.4) for quintile 4. On adding dietary acid load (DAL), estimated by using a model by Remer and Manz, 17 and dietary protein to the full model a partial mediation was noted: RH 1.3 (95% CI, 0.8–2.0) for quintile 1,1.4 (95% CI, 0.7–2.7) for quintile 2; 0.8 (95% CI, 0.5–1.5) for quintile 3, and 0.7 (95% CI, 0.4–1.2) for quintile 4 for DAL and 1.2 (95% CI, 0.8–1.9) for quintile 1, 1.3 (95% CI, 0.6–2.5) for quintile 2, 0.9 (95% CI, 0.5–1.7) for quintile 3, and 1.1 (95% CI,0.7–1.8) for quintile 4 in case of dietary protein (Supplementary Table S2).

There was effect modification by diabetes status for the relation between DASH score quintile and ESRD (*P* interaction < 0.001). Effect modification by race/ethnicity for the relation between DASH score quintile and ESRD was particularly evident when non-

Hispanic blacks (NHBs) and non-Hispanic whites (NHWs) were compared (P interaction = 0.02).

Subgroup analyses

For NHBs, DASH accordance score in quintile 1 was significantly associated with greater risk of ESRD compared with quintile 5 (RH 1.7 [95% CI, 1.1–2.8]). Among NHWs we did not observe a significant association between DASH diet accordance score and risk of ESRD after adjustment for the confounders, although a significant trend was noted (*P* trend = 0.01; Figure 2a). Analysis stratified by diabetes status showed a higher risk of ESRD with lower accordance among adults with diabetes (RH 3.5 [95% CI, 1.7–6.5] for quintile 1 and 3.1 [95% CI, 1.3–5.3] for quintile 2) compared with their counterparts in quintile 5 (Figure 2b). Among those without diabetes, risk of ESRD among adults with lower accordance was generally not significant.

Sensitivity analyses

Adjustment for smoking and physical activity.—The relationship between DASH accordance score and risk of ESRD appeared to be stronger on adjusting the model further for smoking and physical activity. Compared with quintile 5, the RH for the quintiles were 1.76 (95% CI, 1.10–2.81) for quintile 1, 2.73 (95% CI, 1.34–5.57) for quintile 2, 1.79 (95% CI, 0.94–3.42) for quintile 3, and 1.90 (95% CI, 0.79–3.47) for quintile 4.

GFR estimated using CKD Epidemiology Collaboration equation.—Of 977 adults aged 20 years with moderate CKD and hypertension in the National Health and Nutrition Examination Survey (NHANES) III for whom baseline CKD was defined using the CKD Epidemiology Collaboration equation, ¹⁸ 18.8% developed ESRD. The results from the fully adjusted model showed similar risk of ESRD associated with the quintiles of DASH diet accordance score (compared with quintile 5; RH 2.2 [95% CI, 1.5–3.2] for quintile 1, 1.5 [95% CI, 1.0–2.1] for quintile 2, 1.6 [95% CI, 0.9–3.0] for quintile 3, and 0.99 [95% CI, 0.5–1.6] for quintile 4; *P* for trend = 0.002) to that estimated using the MDRD equation to calculate baseline CKD status.

No CKD and no hypertension population.—When we examined the association of the DASH diet accordance score with incident ESRD in adults without CKD and without hypertension at baseline (n = 9202) we found1.1% of adults developed ESRD. Baseline characteristics are presented in Supplementary Table S3. In the multivariable model adjusted for demographics, SEP, diabetes, systolic BP, serum potassium, total caloric intake, BMI, eGFR, and ACR, the RH for incident ESRD among adults who had a lower DASH diet accordance score was 2.32 (95% CI, 0.60–5.97) for quintile 1, 0.56 (95% CI, 0.20–1.56) for quintile 2, 1.66 (95% CI, 0.40–4.87) for quintile 3, and 0.66 (95% CI, 0.35–1.69) for quintile 4 (*P* for trend = 0.15) when compared with those with a higher accordance.

DASH diet adherence and mortality.—In competing-risk analyses, results were no longer significant if death was included as the end point in lieu of treatment as a competing risk. The number of deaths observed in this cohort were 626. The RH for death in adults with the lowest accordance was 1.07 (95% CI, 0.84–1.35) for quintile 1, 0.89 (95% CI,0.65–

1.23) for quintile 2, 0.87 (95% CI, 0.67-1.12) for quintile 3, and 0.80 (95% CI, 0.62-1.04) for quintile 4 (P for trend = 0.03) when compared with adults with CKD having the greatest accordance.

DISCUSSION

In this nationally representative study we demonstrate for the first time a relationship between low DASH dietary accordance rates and higher risks of ESRD among individuals with pre-existing moderate CKD and hypertension. These findings suggest that adults with moderate CKD and hypertension may derive clinical benefit from a DASH diet in reducing the risk of CKD progression. In our study the mean age of the study population was somewhat high, which reflects the average age of people with CKD measured by reduced eGFR in the United States. Our observations not only extend the findings from cross-sectional associations that have suggested a link between the DASH diet and lower odds of CKD¹⁹ but also suggest that the adoption of a DASH diet may be beneficial in reducing future ESRD risk.

A striking observation is the magnitude of the RH associated with low accordance to a DASH diet and risk of ESRD. Adults with moderate CKD as defined by the MDRD had an almost 2-fold higher risk of developing ESRD compared with those who had the greatest accordance score, even after controlling for covariates. The individual components of the DASH accordance score may also have a considerable impact on the association of the DASH accordance score with risk of ESRD. Our exploratory analysis supports dietary K and Mg as strong mediators of the association between DASH accordance score and risk of ESRD. Fruits and vegetables are rich in potassium and bicarbonate that may confer kidney protective effects. 12,13 The increased level of dietary magnesium in the DASH accordance score may lead to lower production of inflammatory and proatherogenic cytokines in endothelial cells, a possible pathway to restore the kidney function. 20,21 Our findings also suggest that DAL and dietary protein intake might be partial mediators for the association between accordance to a DASH diet and risk of ESRD. The absolute RHs were slightly attenuated when both DAL and dietary protein intake were added to the model.

Although recent literature supports diet as an important CKD-related death and progression risk factor, few dietary studies in adults with CKD have investigated the association between accordance to a DASH-like dietary pattern and progression of CKD, due to concerns about its use in patients with CKD stages 3 to 4 (eGFR 15–59 ml/min). ¹⁴ In contrast to the potential harm of following a DASH diet among patients with more severe CKD, the findings from this study suggest a possible benefit associated with adhering to a DASH diet in adults with moderate (stage 3) CKD and hypertension.

Race/ethnicity-stratified analyses showed that the benefit of adhering to a DASH-like diet is more evident in NHBs than NHWs. This may be due to the few ESRD events for NHWs. This disparity may also be related to socioeconomic barriers and cultural factors in eating habits. Education, income, physical environment, language barriers, disability, immigrant status, and residence (urban vs. rural) have all been shown to play roles in diet-related disparities. ^{22–25} For example, lack of access to grocery stores with fresh produce may result

in lower fruit and vegetable consumption, and the inability to communicate in English or leave one's home due to a disability can interfere with ability to comprehend and execute complex health regimens and better disease self-management, thereby leading to consumption of less healthy foods. We speculate that the racial/ethnic differences in the association of dietary patterns and risk of ESRD may be due to genetic differences between NHBs and NHWs.^{26,27} Because of an upregulated intrarenal renin-angiotensinal dosterone system in blacks. ²⁶ hypertensive blacks are more likely to have a greater increase in BP in response to dietary sodium than whites. Low accordance to a DASH diet, being low in baseinducing food such as in dietary K and Mg and high in acid-inducing food such as in dietary protein, may result in metabolic acidosis, particularly in patients with CKD. This may stimulate aldosterone secretion that has adverse hemodynamic effects and induces renal fibrosis. 28 Additionally, the high-risk apolipoprotein L1 alleles associated with CKD progression are noted in African Americans, and these alleles may modify the relationship between lower accordance to a DASH diet and CKD progression.²⁷ Moreover, the DASH diet is based on a defined set of food choices (rather than nutrients) that reflects a healthy dietary choice, and the accordance score obscures the food constituents. This may explain differences in the subgroups where there may be differing cuisine patterns. We believe further studies are needed to explore the association of a DASH-type dietary pattern with risk of ESRD in NHBs and NHWs.

In the stratified model we found higher levels of DASH diet accordance may lower the risk of CKD progression to ESRD among adults with diabetes. The mechanism whereby the DASH diet could reduce the risk of ESRD in adults with moderate CKD, hypertension, and diabetes is unclear. Prior research has shown an association between high DAL and CKD progression.² In industrialized society humans eat diets that are largely hydrogen ion (H⁺) inducing, and continued ingestion of dietary H+ might increase H+ retention that in turn may activate the renin-angiotensin system. As noted by Wesson et al., 29 in subjects with moderately reduced GFR, H⁺ retention increases plasma levels of endothelin and aldosterone and increases their urine excretion. Consequently, H+ retention with increased kidney endothelin and aldosterone production might contribute to progressive GFR decline in subjects with moderately reduced GFR.²⁹ Because the DASH diet is rich in fruits and vegetables, it is likely to be high in dietary K and Mg and low in in Na, which is reflective of lower DAL than a typical Western diet and hence the lower risk of ESRD that we noted in our participants. Second, a diet low in base-inducing K and Mg will be higher in acid precursors, characterized by lower adherence to the DASH diet, and may produce a state of high endogenous acid production over time that may result in insulin resistance and metabolic syndrome.³⁰ Insulin resistance syndrome is accompanied by elevated uric acid, endothelial dysfunction, and CKD progression. 31-33 Because foods and nutrients are not consumed in isolation, our data emphasize the importance of an overall healthy diet high in dietary K and Mg and low in dietary protein and DAL that may lead to potential strategies for slowing or preventing the risk of CKD progression among adults with diabetes. Furthermore, the manipulation of macronutrients, including fat and carbohydrates, in DASH diet may show additional benefits in slowing risk of CKD progression.

We found that DASH diet accordance was not associated with mortality when it was included as the endpoint in the competing risk model in those with moderate CKD and

hypertension. Our findings are in contrast to recent studies that have examined the association of dietary patterns and mortality in individuals with CKD. A meta-analysis of cohort studies examining healthy dietary patterns and risk of mortality and ESRD in CKD found healthy dietary patterns were associated with lower mortality in people with kidney disease.³⁴ Another study examining adherence to a Mediterranean dietary pattern with risk of mortality showed greater adherence to this diet independently predicted survival in patients with CKD.³⁵ We suspect the difference in our findings with other recent studies may have been due to heterogeneity in how CKD was defined, and the meta-analysis of the cohort studies involved people with CKD defined as an eGFR < 60 to 70 ml/min per 1.73 m² or albuminuria. We speculate that the difference in the results could be due to dietary differences accounted for by individual nutrients. A DASH-type dietary pattern is generally high in fruits and vegetables, whereas in the Mediterranean diet, apart from the alkaliinducing fruits and vegetables, the other important components are the high fiber intake, high polyunsaturated fatty acid, and low saturated fatty acid. High polyunsaturated fatty acid intake, especially of marine origin, has been considered as renal protective, ^{36,37} whereas high saturated fatty acid intake may deteriorate kidney function.³⁸

We additionally investigated the association of DASH diet and risk of ESRD in adults with no CKD and no hypertension. We noted that the total caloric intake in this group was considerably higher compared with that of the CKD group. This was likely due to the lower mean age of the participants in the non-CKD group than that in the CKD group that resulted in a higher caloric intake. Interestingly, our results did not show a risk of ESRD with lower accordance to DASH dietary pattern. This is possibly because our study was underpowered to detect these smaller effect sizes. Further studies with larger sample sizes are required to investigate the association of DASH diet with risk of ESRD in persons without CKD.

Our study has a number of limitations. First, the observational design precludes conclusions of causation. Second, our study lacked follow-up data on laboratory values including measures of kidney function. Thus, there is a possibility of misclassification of CKD risk factors, such as diabetes, hypertension, eGFR, and ACR status that are defined from measurements at a single time point. Third, DASH diet accordance scores were determined using a dietary recall questionnaire at baseline only rather than updated data on dietary intake and may not represent long-term habitual intake relevant to ESRD. Incorporating changes in diet over time would likely attenuate rather than strengthen observed associations.³⁹ Moreover, a 24-hour dietary recall is limited in capturing a participant's usual dietary intake compared with the other dietary assessment methods such as food frequency questionnaire and diet records. This is likely the reason we observe the mean total caloric intake in the overall CKD group and across strata of the DASH accordance score to be low. Fourth, our results may have been influenced by unmeasured confounders (e.g., genetic differences). Fifth, the baseline data analyzed were from 3 decades ago, before the publication of the DASH trial. Since then there have been many secular changes in food processing and dietary patterns that may impact our study's generalizability to current individuals with CKD. Sixth, NHANES does not differentiate between type 1 and type 2 diabetes. However, limiting our analysis to adults would suggest that most have type 2 diabetes. These limitations, however, are counterbalanced by several major strengths. The large sample size from a representative sample of the U.S. population gives statistical power

and makes the results nationally applicable. The extended duration of follow-up allowed us to examine moderately long-term effects and provided enough ESRD cases to conduct stratified analyses. Moreover, unlike previous studies, we had outcome data on the occurrence of ESRD, a major endpoint for the study of CKD progression.

In summary, our findings extend evidence on the possible benefits of high fruit and vegetable diets, suggesting that accordance to a DASH dietary pattern may reduce the risk of progression to ESRD among adults with moderate CKD and hypertension, especially among NHBs and those with diabetes. Clinical trials of sufficient size and diversity may enable researchers to evaluate the impact of dietary modification on reducing the risk of CKD progression and long-term ESRD risk.

METHODS

Study population and baseline data

NHANES III is a national probability survey of U.S. noninstitutionalized civilians conducted between 1988 and 1994 by the National Center for Health Statistics. The National Center for Health Statistics created an NHANES III linked ESRD file and Mortality file that contains information on participants diagnosed with ESRD and mortality follow-up data from the time of NHANES III participation. The ESRD data used was from the Combined ESRD Patient Profile and Death Notification (form 2746) File. This information was based on the results from a probabilistic match between NHANES III participants and U.S. Renal Data System administrative records and National Death Index death certificate records, the details of which are provided elsewhere. We conducted a study of NHANES III participants with moderate CKD and hypertension linked to the ESRD and mortality files. In this study we included participants 20 years of age, eGFR between 30 and 59 ml/min, who had dietary information, and were not pregnant (n = 1441). We further limited our sample size to participants who had hypertension (n = 1171), and excluded 61 participants who had missing data on dietary intake (n = 1110).

Sociodemographic and clinical measurements

Medical history and demographic data were collected through a standardized questionnaire conducted at the participant's home followed by a medical examination and laboratory testing that occurred in a mobile examination center. ⁴² Sociodemographic factors were also assessed during the interview. Racial/ethnic categories were self-reported by participants and assigned as NHW, NHB, Mexican American, or other race/ethnicities. Self-reported information on SEP (i.e., education, income, and sex) were also included. Self-reported income was assessed using the poverty income ratio, which is a ratio of household income to the U.S. household poverty level. ⁴²

Diabetes (other than during pregnancy) was defined by self-report of the condition or measured hemoglobin A_{1C} 6.5%. ⁴³ Hypertension was defined by self-report of health care provider diagnosis, a measured average systolic BP 140 mm Hg or average diastolic BP 90 mm Hg, or reported use of antihypertensive medications. ⁴⁴ BMI was calculated as weight in kilograms divided by the measured height in meters squared.

Dietary score

Diet history was obtained using a 24-hour recall administered by a trained interviewer in the mobile examination center. To assess accordance with the DASH dietary pattern, we generated a DASH score based on work by Mellen *et al.*¹⁶ The 9 target nutrients, namely total fat, saturated fat, protein, fiber, cholesterol, sodium, calcium, magnesium, and potassium, identified for the DASH score were indexed to total energy intake, and the DASH score was generated by the sum of all nutrient targets met. Individuals who met a specific DASH target for a nutrient received a score of 1, whereas those who achieved the intermediate target for a nutrient received a score of 0.5 (Table 2). DAL was estimated by net endogenous acid production (NEAP), which results predominantly from the amount of net acid (acid minus base) produced by the metabolic system every day.⁴⁵

The following algorithm developed by Remer and Manz 17 was used to calculate DAL. This equation estimates the potential renal acid load from average intestinal absorption rates of ingested protein and additional minerals: potential renal acid load (mEq/d) = $0.49 \times$ protein (g/d) + $0.037 \times$ phosphorus (mg/d) – $0.021 \times$ potassium (mg/d) – $0.026 \times$ magnesium (mg/d) – $0.013 \times$ calcium (mg/d). This method of calculation was experimentally validated in healthy adults and children under controlled conditions. 46,47

Measurement and classification of albuminuria and kidney function

Serum creatinine measurements obtained using a kinetic rate Jaffé method in NHANES III were recalibrated to standardized creatinine measurements obtained at the Cleveland Clinic Research Laboratory (Cleveland, OH) as standard creatinine $0.184 + 0.9603 \times$ measured serum creatinine. Random spot urine samples were obtained and frozen. Urine albumin was measured using a solid-phase fluorescence immunoassay, and urine creatinine was measured using the modified Jaffé kinetic method in the same laboratory. eGFR was calculated according to the isotope dilution mass spectrometry traceable 4-variable MDRD study equation for calibrated creatinine. Habuminuria, which was calculated as ACR, was expressed as milligrams of albumin per gram of creatinine using American Diabetes Association categories: normal (<30 mg/g creatinine) and albuminuria (30 mg/g creatinine).

Outcomes

The development of ESRD was defined as entry into the U.S. Renal Data System Registry from the time of the survey through December 31, 2006. Mortality was ascertained through the National Death Index⁴¹ from the time of the survey through December 31, 2006.

Statistical analyses

We investigated nonlinear associations between the DASH Accordance Score and risk of ESRD by incorporating restricted cubic splines model. Because of non-linearity, we presented our results as quintiles of DASH Accordance Score to study the dose–response relation.

Patient characteristics were compared at baseline across quintiles of scores reflecting the accordance patterns with a DASH diet using the χ^2 tests for categorical and 1-way ANOVA

for continuous variables. The Kruskal-Wallis test was used for continuous variables if the normality assumption of the residuals was not met. The P value computed tests the difference of the variables across the categories of DASH diet accordance score.

To explore the relationship between adherence to DASH diet and hazard of future ESRD events, we used multivariable Cox regression to model risk. Covariates hypothesized to contribute to CKD progression were included in the adjusted models if they were associated with progression of CKD in univariate analyses (P < 0.20). The model was adjusted for demographic characteristics, SEP, diabetes, systolic BP, serum potassium, total caloric intake, BMI, eGFR, and ACR. SEP was defined as education less than high school or poverty income ratio 2. Potential effect modification between DASH diet accordance and diabetes and race/ethnicity was examined using interaction terms in the adjusted models. To account for competing risks of death and ESRD, we used the Fine-Gray method model competing events. 50

We also conducted 3 sets of sensitivity analyses to examine the robustness of our assertions. First, we reanalyzed associations of DASH diet accordance with outcomes using the CKD Epidemiology Collaboration equation 18 versus the MDRD 15 equation for determination of eGFR in analyses. Second, we performed another analysis in the non-CKD population without hypertension to explore the analysis of DASH diet accordance score with risk for ESRD (n = 13,976). Third, in the moderate CKD group with hypertension, we ran additional analyses for mortality as the primary endpoint and ESRD as a competing risk to determine whether DASH diet accordance was also a risk factor for mortality.

All analyses included the total NHANES III Mobile Examination Center–examined sample final weight to account for the complex sample design following the analytical guidelines for NHANES III data. ⁵¹ For variance estimates we used Fay's balanced repeated replication procedure, an approach for estimation of SEs for multistage samples that consist of many sampling units. ⁵² Results were considered significant if P < 0.05. All analyses were performed using SAS 9.2 (SAS Institute, Cary, NC).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGMENTS

We thank the participants and staff of the NHANES.

The Establishing a Surveillance System for Chronic Kidney Disease project is supported under a cooperative agreement from the Centers for Disease Control and Prevention through grant number NU58DP003836-05-01. The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

APPENDIX

List of Centers for Disease Control and Prevention Chronic Kidney Disease Surveillance Team

University of California, San Francisco: Neil Powe (Principal Investigator), Tanushree Banerjee, Delphine Tuot, Chi-yuan Hsu, Charles McCulloch, Deidra Crews, Raymond Hsu, Vanessa Grubbs, Kirsten Bibbins-Domingo, Michael Shlipak, Carmen Peralta, Anna Rubinsky, and Josef Coresh

University of Michigan: Rajiv Saran (Principal Investigator), Vahakn Shahinian, Brenda Gillespie, Hal Morgenstern, Michael Heung, William Herman, William McClellan, Jennifer Bragg-Gresham, Diane Steffick, Anca Tilea, Maggie Yin, Ian Robinson, Kara Zivin, Vivian Kurtz, and April Wyncott

The Centers for Disease Control and Prevention: Nilka Ríos Burrows (Technical Advisor), Mark Eberhardt, Linda Geiss, Juanita Mondesire, Bernice Moore, Priti Patel, Meda Pavkov, Deborah Rolka, Sharon Saydah, Sundar Shrestha, and Larry Waller.

REFERENCES

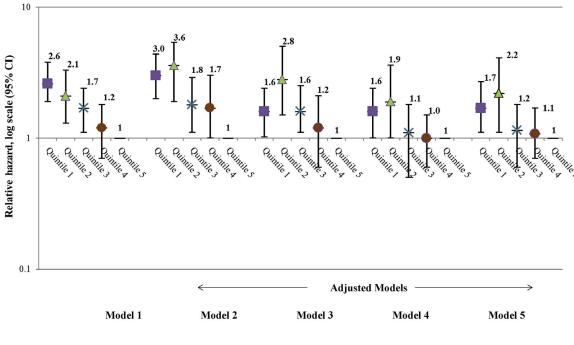
- Centers for Disease Control and Prevention. National Chronic Kidney Disease Fact Sheet: General Information and National Estimates on Chronic Kidney Disease in the United States, 2014 Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2014
- 2. Banerjee T, Crews D, Wesson D, et al. High dietary acid load predicts end stage renal disease among chronic kidney disease adults. J Am Soc Nephrol. 2015;26:1693–1700. [PubMed: 25677388]
- 3. Scialla JJ, Appel LJ, Astor BC, et al. Net endogenous acid production is associated with a faster decline in GFR in African Americans. Kidney Int. 2012;82:106–112. [PubMed: 22475819]
- 4. Kanda E, Ai M, Kuriyama R, et al. Dietary acid intake and kidney disease progression in the elderly. Am J Nephrol. 2014;39:145–152. [PubMed: 24513976]
- 5. Khan SS, Kazmi WH, Abichandani R, et al. Health care utilization among patients with chronic kidney disease. Kidney Int. 2002;62:229–236. [PubMed: 12081582]
- Go AS, Chertow GM, Fan D, et al. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. N Engl J Med. 2004;351: 1296–1305. [PubMed: 15385656]
- Fried LF, Shlipak MG, Crump C, et al. Renal insufficiency as a predictor of cardiovascular outcomes and mortality in elderly individuals. J Am Coll Cardiol. 2003;41:1364–1372. [PubMed: 12706933]
- 8. Frassetto LA, Nash E, Morris RC Jr, et al. Comparative effects of potassium chloride and bicarbonate on thiazide-induced reduction in urinary calcium excretion. Kidney Int. 2000;58:748–752. [PubMed: 10916098]
- 9. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. N Engl J Med. 1997;336:1117–1124. [PubMed: 9099655]
- Writing group of the Premier Collaborative Research Group. Effects of comprehensive lifestyle
 modification on blood pressure control. Main results of the Premier Clinical trial. JAMA.
 2003;289:2083–2093. [PubMed: 12709466]
- 11. Lin J, Fung TT, Hu FB, et al. Association of dietary patterns with albuminuria and kidney function decline in older white women: a subgroup analysis from the Nurses' Health Study. Am J Kidney Dis. 2011;57:245–254. [PubMed: 21251540]

12. Goraya N, Simoni J, Jo C, et al. Dietary acid reduction with fruits and vegetables or bicarbonate attenuates kidney injury in patients with a moderately reduced glomerular filtration rate due to hypertensive nephropathy. Kidney Int. 2012;81:86–93. [PubMed: 21881553]

- Goraya N, Simoni J, Jo CH, et al. A comparison of treating metabolic acidosis in CKD stage 4 hypertensive kidney disease with fruits and vegetables or sodium bicarbonate. Clin J Am Soc Nephrol. 2013;8:371–381. [PubMed: 23393104]
- Kidney Disease Outcomes Quality Initiative. K/DOQI clinical practice guidelines on hypertension and antihypertensive agents in chronic kidney disease. Am J Kidney Dis. 2004;43:S1–S290. [PubMed: 15114537]
- Levey AS, Coresh J, Greene T, et al. Chronic Kidney Disease Epidemiology Collaboration.
 Expressing the Modification of Diet in Renal Disease Study equation for estimating glomerular filtration rate with standardized serum creatinine values. Clin Chem. 2007;53:766–772. [PubMed: 17332152]
- Mellen PB, Gao SK, Vitolins MZ, et al. Deteriorating dietary habits among adults with hypertension: DASH dietary accordance, NHANES 1988–1994 and 1999–2004. Arch Intern Med. 2008;168:308–314. [PubMed: 18268173]
- 17. Remer T, Manz F. Potential renal acid load of foods and its influence on urine pH. J Am Diet Assoc. 1995;95:791–797. [PubMed: 7797810]
- 18. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med. 2009;150:604–612. [PubMed: 19414839]
- 19. Lee HS, Lee KB, Hyun YY, et al. DASH dietary pattern and chronic kidney disease in elderly Korean adults. Eur J Clin Nutr. 2017;71:755–761. [PubMed: 27966569]
- Ferre S, Baldoli E, Leidi M, Maier JA. Magnesium deficiency promotes a pro-atherogenic phenotype in cultured human endothelial cells via activation of NFkB. Biochim Biophys Acta. 2010;1802:952–958. [PubMed: 20600865]
- Tin A, Grams ME, Maruthur NM, et al. Results from the atherosclerosis risk in communities study suggest that low serum magnesium is associated with incident kidney disease. Kidney Int. 2015;87:820–827. [PubMed: 25272232]
- 22. Ross CE, Wu CL. The links between education and health. Am Sociol Rev. 1995;60:719–745.
- 23. Maitra S Can patient self-management explain the health gradient? Goldman and Smith's "Can patient self-management help explain the SES health gradient?" (2002) revisited. Soc Sci Med. 2010;70:802–812. [PubMed: 20117869]
- 24. Cutler DM, Lleras-Muney A. Understanding differences in health behaviors by education. J Health Econ. 2010;29:1–28. [PubMed: 19963292]
- 25. Berkman ND, Sheridan SL, Donahue KE, et al. Low health literacy and health outcomes: an updated systematic review. Ann Intern Med. 2011;155:97–107. [PubMed: 21768583]
- 26. Martins D, Agodoa L, Norris KC. Hypertensive chronic kidney disease in African Americans: strategies for improving care. Cleve Clin J Med. 2012;79:726–734. [PubMed: 23027732]
- Parsa A, Kao LWH, Xie D, et al. APOL1 risk variants, race, and progression of chronic kidney disease. N Engl J Med. 2013;369:2183–2196. [PubMed: 24206458]
- 28. Ponda MP, Hostetter TH. Aldosterone antagonism in chronic kidney disease. Clin J Am Soc Nephrol. 2006;1:668–677. [PubMed: 17699271]
- 29. Wesson DE, Simoni J, Broglio K, et al. Acid retention accompanies reduced GFR in humans and increases plasma levels of endothelin and aldosterone. Am J Physiol. 2011;300:F830–F837.
- 30. Souto G, Donapetry C, Calvino J, et al. Metabolic acidosis-induced insulin resistance and cardiovascular risk. Metab Syndr Relat Disord. 2011;9:247–253. [PubMed: 21352078]
- 31. Hess B Prophylaxis of uric acid and cystine stones. Urol Res. 1990;18:S41–S44. [PubMed: 2291249]
- 32. Sowers JR. Metabolic risk factors and renal disease. Kidney Int. 2007;71: 719–720. [PubMed: 17429418]
- 33. Caravaca F, Cerezo I, Macias R, et al. Insulin resistance in chronic kidney disease: its clinical characteristics and prognostic significance. Nefrologia. 2010;30:661–668. [PubMed: 21113216]

34. Kelly JT, Palmer SC, Wai SN, et al. Healthy dietary patterns and risk of mortality and ESRD in CKD: a meta-analysis of cohort studies. CJASN. 2017;12:272–279. [PubMed: 27932391]

- 35. Huang X, Jimenez-Moleon JJ, Lindholm B, et al. Mediterranean diet, kidney function, and mortality in men with CKD. CJASN. 2013;8:1548–1555. [PubMed: 23744002]
- 36. Lauretani F, Semba RD, Bandinelli S, et al. Plasma polyunsaturated fatty acids and the decline of renal function. Clin Chem. 2008;54:475–481. [PubMed: 18202159]
- 37. Gopinath B, Harris DC, Flood VM, et al. Consumption of long-chain n-3 PUFA, a-linolenic acid and fish is associated with the prevalence of chronic kidney disease. Br J Nutr. 2011;105:1361–1368. [PubMed: 21255476]
- 38. Lin J, Hu FB, Curhan GC. Associations of diet with albuminuria and kidney function decline. Clin J Am Soc Nephrol. 2010;5:836–843. [PubMed: 20299364]
- 39. Dehghan M, Mente A, Teo KK, et al. Relationship between healthy diet and risk of cardiovascular disease among patients on drug therapies for secondary prevention: a prospective cohort study of 31,546 high-risk individuals from 40 countries. Circulation. 2012;126:2705–2712. [PubMed: 23212996]
- 40. Golden C, Driscoll AK, Simon AE, et al. Linkage of NCHS population health surveys to administrative records from Social Security Administration and Centers for Medicare and Medicaid Services. National Center for Health Statistics. Vital Health Stat 1. 2015;58:1–53.
- 41. National Center for Health Statistics. The Third National Health and Nutrition Examination Survey (NHANES III) Linked Mortality File: Matching Methodology. Hyattsville, MD: National Center for Health Statistics; 2005.
- 42. Anonymous. Plan and operation of the Third National Health and Nutrition Examination survey, 1988–94. Series 1: programs and collection procedures. Vital Health Stat. 1994;32:1–407.
- 43. International Expert Committee. International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes. Diabetes Care. 2009;32:1327–1334. [PubMed: 19502545]
- 44. Chobanian AV, Bakris GL, Black HR, et al. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood Institute National High Blood Pressure Education Program Coordinating Committee. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003;42:1206–1252. [PubMed: 14656957]
- 45. Pizzorno J, Frassetto LA, Katzinger J. Diet-induced acidosis: is it real and clinically relevant? Br J Nutr. 2010;103:1185–1194. [PubMed: 20003625]
- 46. Remer T, Manz F. Estimation of the renal net acid excretion by adults consuming diets containing variable amounts of protein. Am J Clin Nutr. 1994;59:1356–1361. [PubMed: 8198060]
- 47. Remer T, Dimitriou T, Manz F. Dietary potential renal acid load and renal net acid excretion in healthy, free-living children and adolescents. Am J Clin Nutr. 2003;77:1255–1260. [PubMed: 12716680]
- 48. Coresh J, Selvin E, Stevens LA, et al. Prevalence of chronic kidney disease in the United States. JAMA. 2007;298:2038–2047. [PubMed: 17986697]
- 49. Molitch ME, DeFronzo RA, Franz MJ, et al. American Diabetes Association: nephropathy in diabetes. Diabetes Care. 2004;27(Suppl1):S79–S83. [PubMed: 14693934]
- 50. Fine JP, Gray RJ. A proportional hazards model for the subdistribution of a competing risk. J Am Stat Assoc. 1999;94:496–509.
- 51. National Center for Health Statistics. Analytical and Reporting Guidelines: The Third National Health and Nutrition Examination Survey, 1988–1994. Hyattsville, MD: National Center for Health Statistics; 1996.
- 52. Fay RE. Theory and application of replicate weighting for variance calculations In: Proceedings of the Survey Research Methods Section. American Statistical Association; 1989:212–217.



Model 1- Unadjusted

Model 2- Model 1+ adjusted for socio-demographics

Model 3- Model 2+ adjusted for diabetes, systolic BP, serum K

Model 4- Model 3+ adjusted for total caloric intake, body mass index

Model 5- Model 4+ adjusted for eGFR, ACR

Figure 1 |. Longitudinal association of baseline Dietary Approaches to Stop Hypertension diet accordance score quintile with progression to end-stage renal disease among U.S. adults aged 20 and over with moderate chronic kidney disease and hypertension, unadjusted and adjusted for covariates.

Median follow-up, 7.8 years (25th–75th percentiles, 4.7–12.4). (CI, confidence interval.)

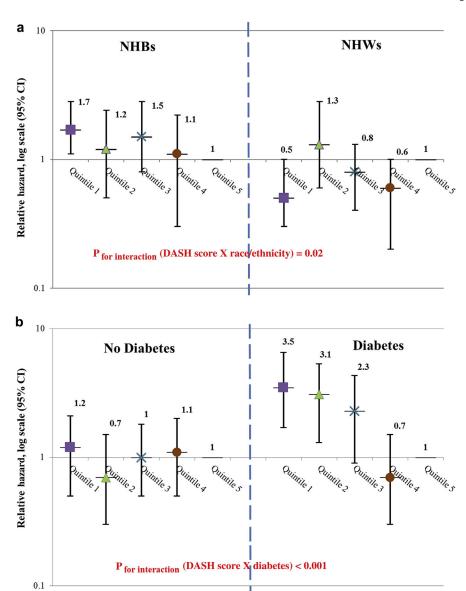


Figure 2 |.

(a) Association of Dietary Approaches to Stop Hypertension (DASH) diet adherence score with end-stage renal disease among U.S. adults aged 20 and over with moderate chronic kidney disease and hypertension, stratified by race/ethnicity. (b) Association of DASH diet adherence score with end-stage renal disease among U.S. adults aged 20 and over with moderate chronic kidney disease and hypertension, stratified by diabetes status. NHB, non-Hispanic black; NHW, non-Hispanic white; CI, confidence interval.

Author Manuscript

Table 1

Characteristics of U.S. adults aged 20 and over with moderate CKD and hypertension by DASH diet accordance score (n = 1110): NHANES III and ESRD linked data

			ď	DASH diet accordance score quintile	score quintile		
Characteristics	Total	1 (n = 321) Score 0 to $<$ 2.5	2 (n = 256) Score 2.5 to <3.5	3 (n = 127) Score 3.5 to <4.0	4 (n = 209) Score 4.0 to <5.0	5 (n = 197) Score 5.0 to 9.0	P^a
Sociodemographic factors							
Age, yr, mean \pm SD	70.2 ± 12.9	66.5 ± 15.5	65.9 ± 14.1	71.9 ± 14.1	71.7 ± 10.0	73.3 ± 10.9	0.045
Male, %	44.5	52	35	50	39	39	0.04
Race/ethnicity, %							0.02
Non-Hispanic white	62.5	55	29	59	56	61	
Non-Hispanic black	23.4	36	20	33	24	18	
Mexican American	11.2		6	1	18	15	
Others	2.8		2		3	5	
Poverty income ratio, 2, %	56.5	51.9	67.2	47	59	52.5	0.28
Education level, %							0.01
<high school<="" td=""><td>52.4</td><td>56.9</td><td>43.9</td><td>45.7</td><td>63.</td><td>49</td><td></td></high>	52.4	56.9	43.9	45.7	63.	49	
Some college	36.4	36.5	48.6	25.2	24.5	40.3	
>College	11.2	9.9	7.5	29.1	12.5	10.7	
Socioeconomic position (poverty income ratio # 2 or education less than high school), %	71.1	71.1	72.5	68.8	70.9	70.2	0.91
Body mass index, kg/m ² , mean \pm SD	28.2 ± 6.1	26.5 ± 4.9	26.9 ± 5.1	28.1 ± 4.7	29.6 ± 7.9	28.7 ± 6.0	0.04
Diabetes, yes, %	27.9	27	39	19	34	31	0.01
Glycated hemoglobin, %	6.4 ± 0.05	6.1 ± 0.1	7.0 ± 0.1	5.9 ± 0.1	6.3 ± 0.1	6.4 ± 0.1	<0.0001
Average systolic blood pressure, mm Hg, mean \pm SD	152.6 ± 0.8	154.1 ± 1.4	149.8 ± 1.8	151.7 ± 2.9	155.1 ± 1.8	151.5 ± 1.6	0.18
Use of angiotensin-converting enzyme/angiotensin-receptor blocker, %	11.7	17.6	7.0	22.8	9.4	6.8	0.04
Serum potassium, mmol/l, mean ± SD	4.1 ± 0.5	4.0 ± 0.5	4.1 ± 0.4	4.2 ± 0.4	4.2 ± 0.5	4.1 ± 0.4	0.15
Total caloric intake, kcal/d, mean \pm SD	1576.2 ± 674.8	1701.8 ± 734.6	1683 ± 755.4	1463.9 ± 532.7	1501.7 ± 575.9	1203.5 ± 455.8	0.002
Physical activity, %							0.72
Moderate	95.2	96	93.2	9.66	93.4	93.8	
Intense	4.8	4	8.9	0.4	9.9	6.2	
Smoking, %							0.05

_
$\overline{}$
_
_
0
$\overline{}$
$\overline{}$
<
\boldsymbol{a}
_
_
\subseteq
S
C
-
$\overline{\mathbf{C}}$
$\stackrel{\sim}{}$

Author Manuscript

Author Manuscript

			ď	DASH diet accordance score quintile	score quintile		
Characteristics	Total	1 (n = 321) Score 0 to $<$ 2.5	2 (n = 256) Score 2.5 to <3.5	3 (n = 127) Score 3.5 to <4.0	4 (n = 209) Score 4.0 to <5.0	5 (n = 197) Score 5.0 to 9.0	P^a
Current	18.3	22	31.4	16.8	10.2	6.3	
Past	38.6	37.4	25.9	49.2	47.4	53.4	
Never	43.1	40.6	42.7	33.3	42.4	40.3	
Dietary constituents, mean \pm SD							
Fiber, g	9.9 ± 0.2	6.7 ± 0.1	8.6 ± 0.2	9.6 ± 0.3	11.5 ± 0.4	16 ± 0.4	<0.0001
Magnesium, mg	163.6 ± 1.7	116.9 ± 1.3	160.8 ± 2.5	150.5 ± 3.9	178.8 ± 2.9	238.5 ± 3.9	<0.0001
Calcium, mg	421.8 ± 7.1	300.8 ± 7.1	455.1 ± 13.4	352.8 ± 16.6	431.1 ± 18.9	611.8 ± 18.4	<0.0001
Potassium, mg	1622 ± 16.1	1227.7 ± 15.1	1602.2 ± 25.7	1512.2 ± 36.4	1749.7 ± 31.5	2249.6 ± 35.4	<0.0001
Sodium, mg	1676 ± 20.4	1809.9 ± 26	1747 ± 33.3	1484.8 ± 71.4	1551 ± 71.1	1597.9 ± 48.1	<0.0001
Cholesterol, mg	157.3 ± 4.3	208.1 ± 7.3	177.6 ± 11.8	118.6 ± 8.8	123.9 ± 6.8	101.9 ± 7.3	<0.0001
Protein, g	42.4 ± 0.4	40.8 ± 0.7	43.1 ± 0.9	36.2 ± 1.2	45.3 ± 1.4	45.8 ± 1	<0.0001
Total fat, g	36.1 ± 0.3	43.1 ± 0.4	37.8 ± 0.4	34.5 ± 0.7	32.2 ± 0.6	27 ± 0.7	<0.0001
Saturated fat, g	11.8 ± 0.1	13.9 ± 0.2	12.9 ± 0.2	10.3 ± 0.3	10.9 ± 0.3	8.4 ± 0.2	<0.0001
Dietary acid load, mEq/d, mean \pm SD	5.5 ± 0.2	7.3 ± 0.4	5.2 ± 0.3	7.3 ± 0.6	5.2 ± 0.4	2.2 ± 0.3	<0.0001
Kidney markers							
eGFR, ml/min per 1.73 m ² , mean \pm SD	48.4 ± 7.9	47.7 ± 8.3	50.7 ± 6.5	48.5 ± 6.9	46.6 ± 8.4	53.1 ± 6.0	0.32
Urinary albumin-to-creatinine ratio, mg/g, median (25th-75th percentile)	25.6 (8.3–201.0)	90.2 (10.8–291.7)	19.5 (5.2–84.3)	29.7 (14.8–126.2)	71.7 (11.9–167.5)	8.4 (3.3–88.5)	0.30
ESRD events, %	18.4	24.5	26.2	18.7	16.5	15.9	0.01

CKD, chronic kidney disease; DASH, Dietary Approaches to Stop Hypertension; NHANES, National Health and Nutrition Examination Survey; ESRD, end-stage renal disease; eGFR, estimated glomerular filtration rate;—, suppressed data, small n < 5.

 $^{\it 2}{\rm P}$ value tests the difference of the variable across the categories of DASH diet accordance.

Table 2 |

DASH diet accordance nutrient intake targets

Nutrient	DASH target	DASH intermediate target
Saturated fat, % energy	6	11
Total fat, % energy	27	32
Protein, % energy	18	16.5
Cholesterol, mg/1000 kcal	71.4	107.1
Fiber, g/1000 kcal	14.8	9.5
Magnesium, mg/1000 kcal	238	158
Calcium, mg/1000 kcal	590	402
Potassium, mg/1000 kcal	2238	1534
Sodium, mg/1000 kcal	1143	1286

Individuals meeting the DASH target for a nutrient received a score of 1, whereas those who achieved the intermediate target for a nutrient received a score of 0.5 for that nutrient, for a total possible score of 9.16 DASH, Dietary Approaches to Stop Hypertension.