Impact of Kidney Function on Effects of the Dietary Approaches to Stop Hypertension (Dash) Diet

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Abstract

Objectives: Although the Dietary Approaches to Stop Hypertension (DASH) diet lowers blood pressure in adults with hypertension, how kidney function impacts this effect is not known. We evaluated whether Estimated Glomerular Filtration Rate (eGFR) modifies the effect of the DASH diet on blood pressure, markers of mineral metabolism, and markers of kidney function.

Methods: Secondary analysis of the DASH-Sodium trial, a multicenter, randomized, controlled human feeding study that evaluated the blood pressure lowering effect of the DASH diet at three levels of sodium intake. Data from 92 participants with pre-hypertension or stage 1 hypertension during the 3450 mg /day sodium diet assignment contributed to this analysis. Stored frozen plasma and urine specimens were used to measure kidney related laboratory outcomes.

Results: Effects of the DASH diet on blood pressure, phosphorus, intact parathyroid hormone, creatinine, and albuminuria were not modified by baseline eGFR (mean 84.5 ± 18.0 ml/min/1.73 m², range 44.1 to 138.6 ml/min/1.73 m²) or the presence of chronic kidney disease (N=13%).

Conclusions: The impact of the DASH diet on blood pressure, markers of mineral metabolism, and markers of kidney function does not appear to be modified by eGFR in this small subset of DASH-Sodium trial participants with relatively preserved kidney function. Whether greater reduction in eGFR modifies the effects of DASH on kidney related measures is yet to be determined. A larger study in individuals with more advanced kidney disease is needed to establish the efficacy and safety of the DASH diet in this patient population.

Keywords: Kidney disease; Diet; Blood pressure; Hypertension

Introduction

Although often underemphasized in clinical practice, a mainstay of therapy for all individuals with hypertension is lifestyle modification [1]. One such modification that effectively lowers BP in adults is the Dietary Approaches to Stop Hypertension (DASH) diet [2,3]. Benefits of the DASH diet have been established in various patient populations; however, its efficacy in individuals with reduced kidney function is not well studied. In fact, the presence of kidney disease was a criterion for participant exclusion from major DASH studies [4,5]. Because 69% of US adults with Chronic Kidney Disease (CKD) are estimated to have uncontrolled hypertension, identifying and employing multiple strategies to lower blood pressure may help improve hypertension control rates [6]. Assessing whether kidney function modifies both the BP lowering effect and safety profile of the DASH diet is an appropriate first step in determining whether this intervention is a viable treatment option for patients with comorbid CKD and hypertension.

The DASH diet is rich in fruits, vegetables, and low-fat dairy products, and low in fat, sweets, and added sugars. It emphasizes healthy sources of protein, such as, lean meats, nuts, seeds, and legumes. The resultant nutritional profile is high in potassium, calcium, phosphorus, and protein. Because high intake of these nutrients may cause significant metabolic derangements and increased morbidity in some individuals with reduced kidney function, current kidney disease outcome quality initiative guidelines do not recommend routine adoption of the DASH diet by patients with moderate disease (estimated glomerular filtration rate [eGFR] <60 ml/min/1.73 m²) [7]. However, mounting data suggests that a diet high in fruits and vegetables can be safely consumed by patients with early and even advanced CKD [8,9]. It is also more widely recognized that the dietary sources of phosphorus (inorganic vs. organic) and protein (animal vs. plant derived) influence the bioavailability and potential health effects of these nutrients, with organic phosphorus and plant derived protein being less harmful [10-13]. Hence, it seems appropriate to reassess the validity of traditional dietary restrictions that are often recommended for patients with CKD. The above findings support the relevancy of taking a closer look at the DASH diet, a diet high in fruits and vegetables, organic phosphorus, and plant derived protein, to determine whether it may be appropriate at reduced levels of kidney function for hypertension management.

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In this secondary analysis of the DASH-Sodium trial, which includes participants with preserved or mildly reduced kidney function, we evaluated whether the BP lowering effect of the DASH diet is modified by eGFR. We also evaluated whether the DASH diet negatively impacted kidney-related laboratory measures as eGFR declined.

Methods

The DASH-Sodium trial was a multi-center, randomized, controlled human feeding study that evaluated the effects of the DASH diet at three levels of sodium intake on BP in adults with pre-hypertension or untreated stage 1 hypertension. Detailed descriptions of trial methods and main results have been previously published [3,4]. An Institutional Review Board at each participating study site approved the research protocol. All participants provided written informed consent and a Data and Safety Monitoring Board provided trial oversight.

Study population

DASH-Sodium participants were ≥22 years old with mean Systolic BP (SBP) of 120-159 mmHg and mean Diastolic BP (DBP) of 80-95 mmHg. Exclusion criteria were use of antihypertensive medications or nutritional supplements, history of cardiovascular disease, type 1 or poorly controlled type 2 diabetes mellitus, poorly controlled hyperlipidemia, Body Mass Index (BMI) >40 kg/m², consumption of >14 alcoholic beverages per week, pregnancy or breast-feeding, and kidney disease. Kidney disease was defined as either a serum creatinine >1.2 mg/dl for women and >1.5 mg/dl for men (unless eGFR was ≥60 ml/min/1.73 m²), or if albuminuria was present at any eGFR. Participants were classified as having albuminuria if baseline urine albumin-to-creatinine ratio (UACR) was ≥17 mg/g in men or ≥25 mg/g in women [16]. Based on the original DASH trial [2], participants were classified into the following standard stages: Stage 1=eGFR ≥ 90 ml/min/1.73 m²; Stage 2=eGFR of 30-59 ml/min/1.73 m² or if albuminuria was present at any eGFR; Stage 3=eGFR of 30-59 ml/min/1.73 m² with albuminuria, and Stage 4=eGFR <30 ml/min/1.73 m².

Trial design

After a 2 week run-in period, participants were randomized to receive either the DASH diet or a control diet in parallel group design. The composition of each diet is shown in Table 1. Assigned diets were administered for three consecutive 30-day feeding periods each at 3 levels of sodium in random crossover fashion. The compositions of each diet were as follows:

- Low sodium (1150 mg NaCl)
- Intermediate sodium (2300 mg NaCl)
- High sodium (3450 mg NaCl)

The composition of each diet is shown in Table 1. Assigned diets were administered for three consecutive 30-day feeding periods each at levels of sodium in random crossover fashion.

Blood pressure

Detailed description of the BP measurement protocol has been previously published [3]. BP measurements obtained during the screening and run-in phases were averaged to determine baseline BP. Those obtained during the first week of the high sodium intervention period were averaged to determine pre-intervention BP used in our analysis. Measurements obtained during five of the last nine days of the high sodium intervention period were averaged to determine post-intervention BP.

Laboratory data

Fasting plasma specimens and 24-hour urine collections were obtained during screening and the last week of each intervention period and subsequently frozen at -70°C. We retrieved and thawed available plasma specimens to measure phosphorus (colorimetric method), intact parathyroid hormone (iPTH; electrochemiluminescence immunoassay), and creatinine (kinetic Jaffe method). Urine specimens were thawed to measure albumin (immunoturbidimetric method) and creatinine concentrations (LabCorp, Burlington, NC). Frozen specimens had no prior freeze-thaw cycles. We were unable to measure plasma potassium and calcium concentrations because blood was collected into vacutainers containing Ethylenediaminetetraacetic Acid (EDTA), an anticoagulant that contains potassium salts and chelates calcium, resulting in inaccurate potassium and calcium measurements.

Estimated GFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration equation [15]. Participants were classified as having albuminuria if baseline urine albumin-to-creatinine ratio (UACR) was ≥17 mg/g in men or ≥25 mg/g in women [16]. Based on the original DASH trial and sample collected during the screening period, participants were classified as having CKD if eGFR was ≤60 ml/min/1.73 m² or if albuminuria was present at any eGFR. Participants with CKD were classified into the following standard stages:

- Stage 1=eGFR ≥ 90 ml/min/1.73 m² with albuminuria
- Stage 2=eGFR of 60-89 ml/min/1.73 m² with albuminuria, and Stage 3=eGFR of 30-59 ml/min/1.73 m².

Outcomes

Primary outcomes were change in SBP and DBP during the 30 day high sodium intervention period, comparing DASH to control by level of kidney function. Secondary outcomes were changes in phosphorus and iPTH (measures of mineral metabolism) and changes in creatinine, eGFR, and UACR (measures kidney function) from the screening period to the end of the high sodium intervention period.
Statistical analysis

Standard descriptive statistics were used to determine baseline demographics, physical measures, and laboratory data. Means, standard deviations, and ranges were computed for continuous variables and frequency counts with percentages were computed for categorical variables. Between group differences in continuous variables were compared using student’s t-test and categorical variables using chi-square test. Difference in between group changes in outcome variables was compared using general linear models. The relationship between eGFR and change in outcome variables were determined using multiple linear regressions. All models were adjusted for age, race, site, and intervention period, baseline eGFR, and each respective baseline variable. Additionally, SBP and DBP were adjusted for race, gender, age and BMI. Regression models included an assessment for interaction between diet and baseline eGFR. Interactions between diet and the following variables were also assessed: evenly distributed eGFR quartiles (Q1: ≥98, Q2: 81.7-98, Q3: 72-81.7, Q4: <72), CKD stages (1-3), and CKD status (yes, no). Goodness-of-fit of the models were assessed. These were exploratory analysis and no adjustment is made for multiple testing. A significance level of 0.05 was used for all tests. Statistical analysis was performed using SAS 9.3 (SAS Institute, Cary, NC).

Results

Participant characteristics

There were a total of 412 participants enrolled in the DASH-Sodium trial. After exclusion of participants with missing baseline plasma or baseline urine samples, 92 (22%) individuals were included in our analysis. Compared to those who were included, our subset of participants were younger (46.0 ± 10.2 vs. 48.8 ± 9.7 years; p=0.02) and had a higher mean BMI (30.4 ± 5.5 vs. 28.8 ± 4.6 kg/m²; p<0.01). All other demographic and baseline physical measurements were similar. Table 2 shows demographic and baseline physical and laboratory measurements for participants included in our analysis, overall and by diet assignment. A majority were black (65%) and female (63%). Forty-four (48%) participants received the control diet and 48 (52%) participants received the DASH diet. Mean eGFR was 84.5 ± 18.0 ml/min/1.73 m² with a wide range from 44.1 to 138.6 ml/min/1.73 m² and 70% had a baseline eGFR >75 ml/min/1.73 m². A total of 12 (13%) participants had CKD (eGFR <60 ml/min/1.73 m² or presence of albuminuria irrespective of eGFR). Compared to those with normal kidney function, participants with CKD had a higher mean baseline plasma creatinine concentration (1.3 ± 0.3 vs 1.0 ± 0.2 mg/dl; p<0.001), lower eGFR (64.8 ± 20.2 vs 87.4 ± 15.7 ml/min/1.73 m²; p<0.001), greater albuminuria (128.1 ± 250.8 vs. 2.8 ± 4.1 mg/g; p<0.001), and higher mean SBP (138.8 ± 11.2 vs. 132.8 ± 9.3 mmHg; p=0.05) at study entry. Participants with CKD also tended to be older than those with normal kidney function but this was not statistically significant (50.3 ± 10.7 vs 45.4 ± 10.0 years; p=0.12).

Blood pressure

After a 4-week feeding intervention, there was a significant

<table>
<thead>
<tr>
<th>Total (N=92)</th>
<th>Control (N=44)</th>
<th>DASH (N=48)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y (SD)</td>
<td>46.0 (10.2)</td>
<td>45.8 (11.1)</td>
<td>46.2 (9.4)</td>
</tr>
<tr>
<td>Men, N (%)</td>
<td>34 (37.0)</td>
<td>15 (34.1)</td>
<td>19 (39.6)</td>
</tr>
<tr>
<td>Black, N (%)</td>
<td>60 (65.2)</td>
<td>31 (70.5)</td>
<td>29 (60.4)</td>
</tr>
<tr>
<td>Household income</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>≤60,000/year, N (%)</td>
<td>67 (74.4)</td>
<td>35 (81.4)</td>
<td>32 (68.1)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Some college or less, N (%)</td>
<td>49 (53.9)</td>
<td>23 (53.5)</td>
<td>26 (54.2)</td>
</tr>
<tr>
<td>Alcohol, N (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤7 drinks/week</td>
<td>91 (99.0)</td>
<td>44 (100)</td>
<td>47 (97.9)</td>
</tr>
<tr>
<td>SBP, mmHg, mean (SD)</td>
<td>133.6 (8.1)</td>
<td>134.5 (10.4)</td>
<td>132.8 (9.1)</td>
</tr>
<tr>
<td>DBP, mmHg, mean (SD)</td>
<td>84.6 (4.6)</td>
<td>85.0 (3.5)</td>
<td>84.3 (5.5)</td>
</tr>
<tr>
<td>Hypertension* (yes)</td>
<td>34 (37.0)</td>
<td>15 (34.1)</td>
<td>19 (39.6)</td>
</tr>
<tr>
<td>Weight, baseline, kg, mean (SD)</td>
<td>87.6 (16.8)</td>
<td>89.9 (18.2)</td>
<td>85.5 (15.2)</td>
</tr>
<tr>
<td>BMI, kg/m², mean (SD)</td>
<td>30.4 (5.5)</td>
<td>31.2 (6.0)</td>
<td>29.7 (5.0)</td>
</tr>
<tr>
<td>Phosphorus, mg/dl, mean (SD)</td>
<td>3.2 (0.5)</td>
<td>3.1 (0.5)</td>
<td>3.2 (0.5)</td>
</tr>
<tr>
<td>Intact PTH, pg/ml, mean (SD)</td>
<td>45.7 (17.8)</td>
<td>47.2 (16.9)</td>
<td>44.2 (18.7)</td>
</tr>
<tr>
<td>Creatinine, mg/dl, mean (SD)</td>
<td>1.0 (0.2)</td>
<td>1.0 (0.2)</td>
<td>1.04 (0.2)</td>
</tr>
<tr>
<td>eGFR ml/min/1.73m², mean (SD)</td>
<td>84.5 (18.0)</td>
<td>86.8 (18.9)</td>
<td>82.4 (16.9)</td>
</tr>
<tr>
<td>eGFR range:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;90, N (%)</td>
<td>31 (34)</td>
<td>15 (34)</td>
<td>16 (33)</td>
</tr>
<tr>
<td>75-89, N (%)</td>
<td>33 (36)</td>
<td>17 (39)</td>
<td>16 (33)</td>
</tr>
<tr>
<td>60-74, N (%)</td>
<td>20 (22)</td>
<td>9 (20)</td>
<td>11 (23)</td>
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<tr>
<td>&lt;60, N (%)</td>
<td>8 (9)</td>
<td>3 (9)</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Albuminuria† (yes), N (%)</td>
<td>7 (8)</td>
<td>5 (11)</td>
<td>2 (4)</td>
</tr>
<tr>
<td>CKD‡ (yes), N (%)</td>
<td>12 (13.0)</td>
<td>6 (13.6)</td>
<td>6 (12.5)</td>
</tr>
<tr>
<td>CKD Stage:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1, eGFR ≥ 90, N (%)</td>
<td>1 (1.1)</td>
<td>0 (0)</td>
<td>1 (2.1)</td>
</tr>
<tr>
<td>Stage 2, eGFR 60-89, N (%)</td>
<td>3 (3.3)</td>
<td>3 (6.8)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Stage 3, eGFR &lt;60, N (%)</td>
<td>8 (8.7)</td>
<td>3 (6.8)</td>
<td>5 (10.4)</td>
</tr>
</tbody>
</table>

*Hypertension defined as SBP ≥140 mmHg or DBP ≥90 mmHg. †Albuminuria defined as urine albumin-to-creatinine ratio ≥17 mg/g for men and ≥25 mg/g for women. ‡CKD defined as eGFR less than 60 ml/min/1.73 m² or presence of albuminuria at any eGFR. SBP, systolic blood pressure; DBP, diastolic blood pressure; PTH, parathyroid hormone; CKD, chronic kidney disease; BMI, body mass index; eGFR, estimated glomerular filtration rate

Table 2: Demographics and baseline measures for 92 participants of the DASH-Sodium trial overall and by diet assignment.
reduction in SBP for the DASH group (-2.6 ± 9.7 mmHg; 95% CI: -5.1, -0.1 mmHg) but not the control group (0.9 ± 9.6 mmHg; 95% CI: -1.8, 3.5) in both the unadjusted (data not shown) and adjusted models (Figure 1). Between group difference for change in SBP was significant (-3.5 ± 1.7 (SE) mmHg, p=0.05). DBP did not change for either the DASH or control group in unadjusted and adjusted models. Baseline eGFR was not a significant covariate for change in SBP or DBP.

**Mineral metabolism**

Plasma phosphorus did not change after the 4-week feeding intervention within or between the two diet groups (Figure 2). In the unadjusted model, there was no significant change in mean plasma iPTH for either diet group. However, after adjusting for site, intervention period, and baseline iPTH, there was a significant reduction in mean plasma iPTH in the DASH group (-5.7 pg/ml; 95% CI: -10.2, -1.2 pg/ml) but not the control group (-2.2 pg/ml; 95% CI: -6.7, 2.2 pg/ml). Between group difference for change in iPTH did not reach statistical significance (-3.4 pg/ml, p=0.27). Baseline eGFR was not a significant covariate for change in plasma phosphorus or iPTH.

**Kidney function**

Plasma creatinine and eGFR did not change significantly in either the DASH or control groups after the 4-week feeding intervention in both unadjusted and adjusted models (Figure 3). Between groups differences for change in these variables were also not significant. There were also no significant changes in UACR for either the DASH or control groups in the unadjusted models. However, UACR increased in the DASH group (11.3 mg/g; 95% CI: 3.9, 18.8 mg/g), but not the control group (5.7 mg/g; CI: -2.0, 13.4 mg/g) after adjusting for site, intervention period, and baseline UACR. Between group difference for change in UACR was not significant (5.6 mg/g, p=0.27). Baseline eGFR was not a significant covariate for change in creatinine or UACR; however, it was significantly related with post intervention eGFR after adjustment (beta estimate -0.30, SE 0.15, p=0.045).

**Impact of eGFR on the relationship between diet and change in outcome variables**

Adjusted multiple linear regression models were performed to determine if the relationship between diet and change in each outcome variable was altered by kidney function. Separate models were performed to evaluate kidney function by baseline eGFR, eGFR...
Discussion

In this secondary analysis of the DASH-Sodium trial, we were able to evaluate the impact of the DASH diet on BP, markers of mineral metabolism, and kidney function as well as how these relationships may be influenced by level of kidney function. After a 4-week feeding intervention in adults with pre-hypertension and untreated stage 1 hypertension, we confirmed previous findings from the DASH-Sodium trial and other feeding studies that the DASH diet lowers SBP, even in a subpopulation with mildly decreased kidney function [2,3,17]. Although we did not observe an association between the DASH diet and change in DBP, phosphorus, creatinine, or eGFR, we did observe an associated reduction in iPTH and an associated increase in UACR and change in DBP, phosphorus, creatinine, or eGFR, we did observe no significant interactions between eGFR and the DASH diet (p-values >0.05).

Table 3:

<table>
<thead>
<tr>
<th></th>
<th>Beta Estimate (SE)</th>
<th>95% CI for Beta</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>∆ SBP (mmHg)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>eGFR</td>
<td>-0.01 (0.07)</td>
<td>-0.15, 0.13</td>
</tr>
<tr>
<td>DASH</td>
<td>-2.07 (0.63)</td>
<td>-19.22, 15.08</td>
</tr>
<tr>
<td>eGFR×DASH</td>
<td>0.02 (0.10)</td>
<td>-0.18, 0.22</td>
</tr>
<tr>
<td><strong>∆ DBP (mmHg)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>eGFR</td>
<td>0.03 (0.05)</td>
<td>-0.07, 0.13</td>
</tr>
<tr>
<td>DASH</td>
<td>-3.81 (5.76)</td>
<td>-15.26, 7.64</td>
</tr>
<tr>
<td>eGFR×DASH</td>
<td>0.03 (0.07)</td>
<td>-0.11, 0.17</td>
</tr>
<tr>
<td><strong>∆ phosphorus (mg/dl)</strong></td>
<td></td>
<td></td>
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<tr>
<td>eGFR</td>
<td>0.005 (0.005)</td>
<td>-0.005, 0.015</td>
</tr>
<tr>
<td>DASH</td>
<td>0.57 (0.52)</td>
<td>-0.46, 1.60</td>
</tr>
<tr>
<td>eGFR×DASH</td>
<td>0.01 (0.01)</td>
<td>-0.01, 0.03</td>
</tr>
<tr>
<td><strong>∆ iPTH (pg/ml)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>eGFR</td>
<td>-0.13 (0.15)</td>
<td>-0.43, 0.17</td>
</tr>
<tr>
<td>DASH</td>
<td>12.66 (16.06)</td>
<td>-19.31, 44.63</td>
</tr>
<tr>
<td>eGFR×DASH</td>
<td>-0.19 (0.19)</td>
<td>-0.57, 0.19</td>
</tr>
<tr>
<td><strong>∆ creatinine (mg/dl)</strong></td>
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<td></td>
</tr>
<tr>
<td>eGFR</td>
<td>0.004 (0.010)</td>
<td>-0.016, 0.024</td>
</tr>
<tr>
<td>DASH</td>
<td>0.22 (0.43)</td>
<td>-0.64, 1.08</td>
</tr>
<tr>
<td>eGFR×DASH</td>
<td>0.002 (0.005)</td>
<td>-0.008, 0.012</td>
</tr>
<tr>
<td><strong>∆ eGFR (ml/min/1.73 m²)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>eGFR</td>
<td>-0.30 (0.15)</td>
<td>-0.60, 0.00</td>
</tr>
<tr>
<td>DASH</td>
<td>2.44 (15.83)</td>
<td>-29.03, 33.91</td>
</tr>
<tr>
<td>eGFR×DASH</td>
<td>0.08 (0.18)</td>
<td>-0.28, 0.44</td>
</tr>
<tr>
<td><strong>∆ UACR (mg/g)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>eGFR</td>
<td>0.35 (0.25)</td>
<td>-0.15, 0.85</td>
</tr>
<tr>
<td>DASH</td>
<td>-6.50 (28.69)</td>
<td>-63.72, 50.72</td>
</tr>
<tr>
<td>eGFR×DASH</td>
<td>0.14 (0.33)</td>
<td>-0.52, 0.80</td>
</tr>
</tbody>
</table>

All models were adjusted for site, intervention period, baseline eGFR, and each respective baseline measure. SBP and DBP were additionally adjusted for race, gender, age and body mass index. SBP, systolic blood pressure; DBP, diastolic blood pressure; PTH, parathyroid hormone; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; UACR, urine albumin-to-creatinine ratio.

Table 3: Results for linear regression models showed no significant interaction between estimated glomerular filtration rate and the DASH diet.
lowering effect of the DASH diet would outweigh the potential risks of such a modest increase in urine albumin excretion.

We acknowledge that our study has some limitations. Inclusion of a small subset (22%) of participants who were enrolled in the DASH-Sodium trial potentially introduces bias from unbalanced comparison groups. Compared to participants who were excluded, our subset was younger and had a higher BMI. However, in a subgroup analysis of the DASH-Sodium trial the effect of the DASH diet did not vary by age (>45 years) or BMI (≥30 kg/m²) suggesting that this difference may not necessarily influence the expected impact of DASH on BP in our study [24]. Another study limitation is our inability to directly measure key electrolytes, such as, potassium and calcium, due to plasma samples containing EDTA. Assessing the impact of the DASH diet on these electrolytes in patients with CKD is essential to determine whether this intervention is a safe treatment option for hypertensive patients with CKD. Perhaps most critical, our study is limited by the range of eGFR included and classifying participants as having CKD based on a single plasma and urine sample. Only 9% had eGFR less than 60 ml/min/1.73 m², none less than 44 ml/min/1.73 m², and only 13% were classified as having CKD. A larger study in patients with lower kidney function is clearly needed to definitively establish the efficacy and safety of the DASH diet in this population, albeit with caution due to risk of hyperkalemia.

Conclusion

In this small subset of DASH-Sodium participants, our findings suggest that kidney function, as measured by eGFR, does not modify the effect of the DASH diet on blood pressure or markers of mineral metabolism and kidney function. If confirmed in a larger population of patients with less preserved kidney function, the DASH diet may be a valuable, nonpharmacologic strategy for BP control in individuals with CKD.

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