



The Feasibility of Using the Dietary Approaches to Stop Hypertension (DASH) Diet in People with Chronic Kidney Disease and Hypertension

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Abstract

Hypertension control is fundamental in the treatment of Chronic Kidney Disease (CKD). The Dietary Approaches to Stop Hypertension (DASH) diet is recommended as part of the management of hypertension in the general population, but there are concerns about its safety for people with CKD.

We conducted a feasibility study to investigate the safety and acceptability of the DASH diet in free living, non-diabetic, hypertensive patients with stage 3 CKD.

Thirty-two participants completed the intervention (22 male, mean age was 69.9 years, range 43-87). Mean eGFR was 48.2 ± 8 ml/min/1.73m² (range 31-59).

Subjects self-selected the DASH diet for 5 weeks.

Serum urea, creatinine, potassium, sodium, bicarbonate, calcium and phosphate, estimated GFR, urinary sodium, potassium and urea, body weight, height and sitting blood pressure were measured. Total body water and extracellular fluid were estimated by multifrequency bioimpedance.

Serum sodium decreased significantly (139.7 ± 2.6 to 138.9 ± 2.6 mEq/L, $p=0.03$). Urine sodium was significantly reduced ($p<0.02$). Total fat consumption decreased ($p<0.001$) contributing to a reduction in overall energy intake ($p<0.001$). Protein and carbohydrate intake did not change. Sodium intake decreased (2784.9 mg to 1583.4 mg per day, $p<0.001$). Mean weight (81.4 ± 16.1 kg to 79 ± 5.4 kg, $p<0.001$) and extracellular fluid volume decreased (ECF) (18.3 ± 3.2 L to 17.9 ± 3.2 L, $p=0.002$), as did systolic and diastolic blood pressure (by 9.5 mmHg ($p<0.009$) and 5.3 mmHg ($p<0.006$) respectively).

Introduction

Current estimates suggest that 13% of the UK and US adult population are living with Chronic Kidney Disease (CKD) [1,2]. The incidence of CKD is thought to be rising with an aging population and as the prevalence of hypertension, obesity and diabetes increase. The majority of people with CKD have hypertension and hypertension is a risk factor for both renal disease progression and cardiovascular disease, the main cause of death in this population group. In 2009–10 the cost of CKD to the National Health Service (NHS) in England was estimated to be around £1.44 - £1.45 billion (1.3% of NHS spending) [3]. Over 50% of this figure was associated with the costs of renal replacement therapy [3]. Strategies aimed at the treatment of hypertension and the prevention of CKD progression are therefore crucial. The National Institute for Health and Care Excellence (NICE) CKD clinical guidelines [3], recommend such strategies. However lifestyle and dietary modifications are often overlooked as a potential adjunct or alternative to medical therapy.

The Dietary Approaches to Stop Hypertension (DASH) diet describes a pattern of eating designed to decrease blood pressure by increasing the intake of nutrients that lower blood pressure and decreasing the intake of nutrients that raise blood pressure. Target nutrients include potassium, calcium, magnesium, fibre and protein [4]. The original DASH diet did not focus on lowering sodium intake [5]. However a subsequent controlled feeding study combining the DASH diet with high, intermediate and low sodium intakes demonstrated the independent and synergistic effect of reducing sodium intake on blood pressure control [6]. Subjects with renal insufficiency were excluded from these original studies.

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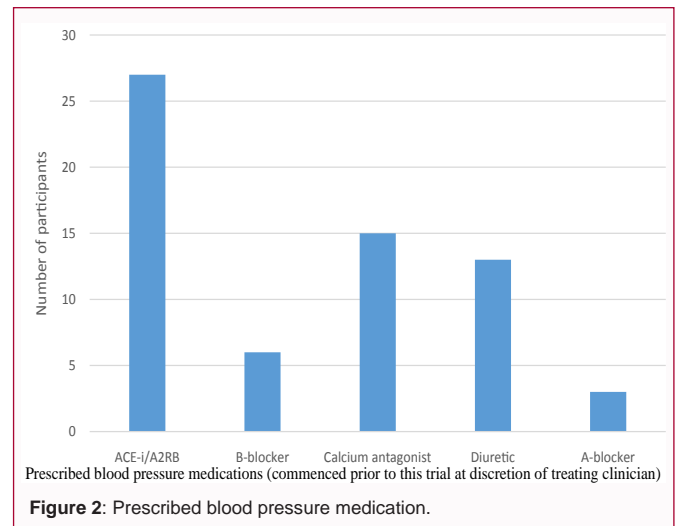
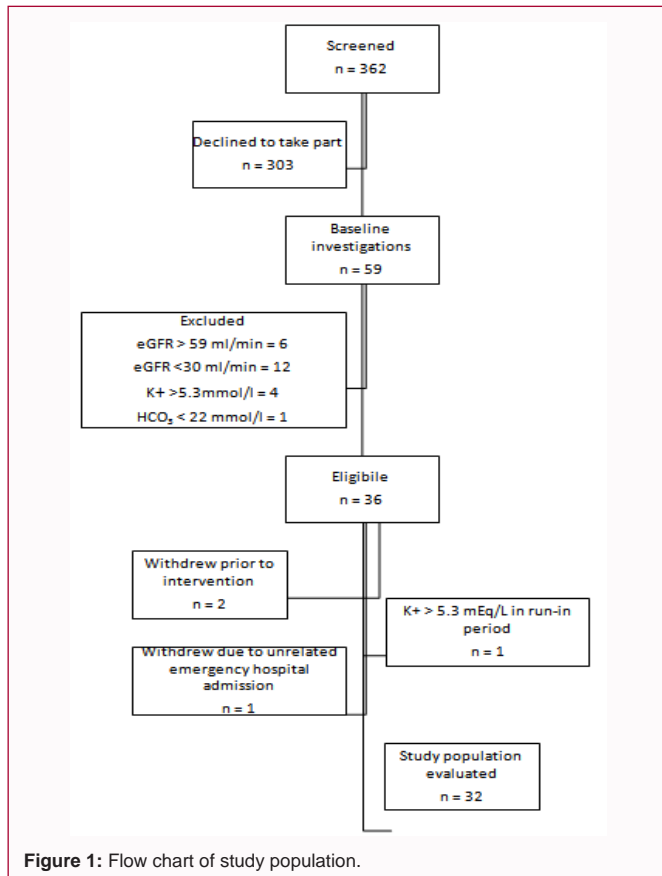
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ACE-i/A2RB	B-blocker	Calcium antagonist	Diuretic	A-blocker
ramipril	atenolol	nifedipine	bendroflumethiazide	doxazosin
lisinopril	carvedilol	amlodipine	indapamide	
enalapril	bisoprolol	lercanidipine	furosemide	
perindopril		diltiazem		
losartan				
valsartan				
irbesartan				
candesartan				

The National Service Framework (NSF) for Renal Services [7], promotes the use of the NICE clinical guidelines on management of hypertension in primary care, which recommends ‘a healthy diet’; reduced sodium intake, regular exercise, and reduced alcohol and caffeine intake if excessive [8]. The equivalent American guidance added the DASH diet to the previously recommended lifestyle modifications as initial treatment for hypertension in the general population [9]. The intermediate sodium DASH diet is advocated as a population based blood pressure management strategy due to predicted difficulties in achieving a low sodium intake in free living individuals.

The National Kidney Foundation – Kidney Disease Outcomes Quality Initiative (NKF-KDOQI) guidelines from 2004 specifically states that the DASH diet should not be routinely recommended to patients with a GFR <60 ml/min/1.73 m² as the protein content (18% protein; approximately 1.4 g/kg/day), and potassium and phosphorus content is higher than is usually recommended in stage 3-4 CKD [3]. The 2008 Scottish Intercollegiate Guidelines Network (SIGN) [11] cited the DASH diet as being of potential benefit to the hypertensive CKD population, but its use is not promoted for the same reasons [12].

A recent pilot controlled feeding study in the USA [13] investigated use of a reduced –sodium DASH diet in 11 participants with stage 3 CKD for 2 weeks, with 1 participant withdrawing at the end of the first week. The authors observed a transient significant increase in serum potassium after week 1 (+0.28 mg ± 0.4 mg/dL) and a significant decrease in serum bicarbonate at week 2 (-2.5 mg ± 3.0 mg/dL), but no clinically relevant changes in serum electrolytes in the short term, 2 week use of the DASH diet.

We are not aware of any previous studies investigating the safety or efficacy of the DASH diet in free living people with CKD.

Materials and Methods

Study design

This is a single centre feasibility study investigating the safety and acceptability of using the DASH diet in free living subjects with stage 3 CKD and hypertension. Data were collected prospectively from June 2012 to June 2014. It is an independent study with no conflict of interest. This study was performed in accordance with the Declaration of Helsinki and was approved by the National Research Ethics Service (NRES) Committee - Yorkshire and The Humber (NRES approval No. 11/YH/0195) and all participants provided written informed consent. The trial was registered on the UK Clinical Research Network Study Portfolio prior to the enrolment of subjects (CLRN No. 10871). The trial was conducted, and all data generated, documented and recorded in compliance with Good Clinical Practice (GCP) and applicable regulatory requirements. Safety reporting was carried out according to the Medicines for Human Use (Clinical Trials) Regulations 2004.

The study was sponsored by York Teaching Hospital NHS Foundation Trust and was funded by the British Renal Society.

Inclusion/exclusion criteria

Participants were recruited from CKD and hypertension primary care registers, or through secondary care nephrology clinics at York Teaching Hospital NHS Foundation Trust. Participants were 18 years of age or older with stage 3 CKD (estimated GFR 30-59 ml/min/1.73m²) and a diagnosis of hypertension. Exclusion criteria were history of cardiac arrhythmia, current hyperkalaemia (serum

Table 1: Changes in serum biochemical markers.

	Baseline	Week 5	p
Urea (mg/dL)	51.2 ± 13.9	53 ± 14.5	0.52
Creatinine (mg/dL)	1.39 ± 0.3	1.4 ± 0.35	0.84
eGFR (ml/min)	48.2 ± 8.0	48.2 ± 9.4	0.46
Sodium (mEq/L)	139.7 ± 2.6	138.9 ± 2.6	0.03
Potassium (mEq/L)	4.51 ± 0.46	4.53 ± 0.47	0.44
Bicarbonate (mEq/L)	27.3 ± 2.7	27.1 ± 2.2	0.38
Calcium (mg/dL)	9.2 ± 0.28	9.6 ± 0.32	0.17
Phosphate (mg/dL)	3.19 ± 0.62	3.35 ± 0.62	0.13

Table 2: Changes in urine biochemistry.

Urine	Baseline	Week 5	p
Volume (ml)	2067.7 ± 613.7	2275 ± 640.9	0.13
Sodium (mEq)	102.1 ± 41.1	89.1 ± 37.1	0.02
Potassium (mEq)	77.2 ± 22.0	84.6 ± 21.7	0.17
Urea (g/24 hours)	20.8 ± 5.6	22.1 ± 6.6	0.70

potassium >5.3 mEq/l), serum phosphate above reference range (>4.3 mg/dl), a current prescription of spironolactone or amiloride, uncontrolled metabolic acidosis (venous bicarbonate <22 mEq/l), a diagnosis of diabetes, pregnant or lactating women or people unable to give informed consent.

Intervention diet

The diet used was based on the intermediate sodium DASH diet [6], adapted to suit British portion sizes and food choices [14]. The number of daily servings of each food group in this diet depends on predicted energy requirements and fit into four predicted energy levels. Energy requirements were assessed using the Schofield equation [15], and a pre intervention 5 day food diary reflecting the subject’s usual diet. Practical information on how to achieve the recommended servings of food groups was provided. All participants were given food label advice in line with current UK guidelines to assist in choosing lower fat, sugar and salt options. No added salt was advised and information on high salt foods was provided.

An initial 1 week run in period was used to gradually build up to DASH diet portions, to avoid any potential gastrointestinal side effects from increasing fibre intake. This was followed by 4 weeks on the full DASH diet, resulting in a 5 week intervention period in total.

Study visits and measurements

An initial non-fasting blood test for urea, creatinine, estimated GFR (MDRD formula), potassium, sodium, bicarbonate, calcium and phosphate was taken to confirm eligibility and subjects completed a pre intervention 5-day food diary [16]. This initial eligibility blood test was used as baseline data. At baseline and study completion (end of week 5) body weight, height, non-fasting blood test results and medication history were recorded. At baseline and study completion sitting blood pressure was taken in the non-dominant arm after at least 5 minutes of rest, using a validated automated blood pressure device and according to British Hypertension Society [17] and NICE hypertension guidelines 2011 [18].

Total body water and extracellular fluid were estimated by multifrequency bioimpedance. A 24 hour urine collection for volume, sodium, potassium and urea levels was arranged. The intervention diet was started immediately after the baseline measurements were

Table 3: Changes in nutrient intake.

	Baseline	Week 5	p
Energy intake (kcal)	2059.4 ± 543.1	1734 ± 438.4	<0.001
Protein intake (g)	83.0 ± 22.9	82.7 ± 20.6	0.50
Total Fat intake (g)	78.7 ± 27.8	51.3 ± 17.0	<0.001
Saturated (g)	27.0 ± 10.3	14.7 ± 5.6	<0.001
Monounsaturated (g)	25.1 ± 9.8	16.9 ± 6.7	<0.001
Polyunsaturated (g)	14.3 ± 6.3	11.7 ± 5.7	0.004
Cholesterol (g)	258.8 ± 70.7	239.6 ± 63.2	0.03
Fibre (g)	12.5 ± 5.4	16.2 ± 5.8	0.01
Sodium intake (mg)	2784.9 ± 1037.5	1583.4 ± 540.0	<0.001
Potassium intake (mg)	3712.4 ± 907.6	3952.0 ± 785.9	0.17
Calcium intake (mg)	994.5 ± 389.4	901.1 ± 238.9	0.06
Phosphate intake (mg)	1528.8 ± 497.8	1552.1 ± 405.1	0.97
Magnesium intake (mg)	350.1 ± 103.4	397.2 ± 112.0	0.007

Table 4: Changes in outcome measures.

	Baseline	Week 5	p
Weight (kg)	81.4 ± 6.1	79.0 ± 15.4	<0.001
BMI (kg/m ²)	28.3 ± 5.0	27.4 ± 4.7	<0.001
Total body water (L)	40.7 ± 7.7	40.3 ± 7.8	0.28
ECF (L)	18.3 ± 3.2	17.9 ± 3.2	0.002
ICF (L)	22.2 ± 4.7	22.4 ± 4.9	0.9
ECF/TBW	0.45 ± 0.03	0.45 ± 0.03	0.09
Systolic blood pressure (mmHg)	142.8 ± 25.2	133.3 ± 17.4	0.009
Diastolic blood pressure (mmHg)	77.3 ± 12.7	72.0 ± 10.7	0.006

recorded.

At interim study visits, at the end of week 1 and week 3, a non-fasting blood test for urea, creatinine, estimated GFR, potassium, sodium, bicarbonate, calcium and phosphate was taken. Sitting blood pressure was measured and the study dietitian discussed progress or any difficulties with the diet. Between study visits participants were contacted by the research team via the telephone to discuss progress with the diet.

A 5 day food diary was completed within the final week of the intervention diet. At the final study visit participants were also given a questionnaire designed to assess acceptability of the study diet.

Safety considerations

Study blood tests were reviewed by the qualified physician responsible for the study and documented on the Case Record Form within 24 hours.

The protocol stipulated that if a blood sample showed a serum potassium of greater than 5.3 mEq/l then the patient would be asked to return to their normal diet. If serum potassium exceeded 6.5 mEq/l they would also be asked to attend for a repeat sample and an ECG would be performed.

All participants and their GPs were provided with safety information advising against the addition of medication known to raise serum potassium during the study period, unless clinically indicated. This included any new prescription of amiloride (the potassium sparing diuretic that is available in the United Kingdom), spironolactone, trimethoprim, ACE inhibitors or angiotensin-2

Table 5: Acceptability questionnaire results.

Question	Scale	Median (range)
How difficult or easy was it to follow the diet?	Very difficult 1 2 3 4 5 6 7 Very easy	6 (2-7)
How much did you enjoy the diet?	Enjoyed very much 1 2 3 4 5 6 7 Did not enjoy at all	2 (1-7)
Did the diet cost you more or less than your usual food costs?	Cost a lot less than usual 1 2 3 4 5 6 7 Cost a lot less than usual 4 = Cost the same as usual	4 (2-7)
How willing would you be to continue with this diet?	Not willing at all 1 2 3 4 5 6 7 Extremely willing	6 (1-7)

blockers.

Participants were also told to avoid the use of potassium based salt substitutes, to remind their GP they are part of the trial if new medications were being prescribed and to avoid starting any new herbal medicines or health food supplements during the trial period. All participants were given a card with working hours and out of hours study contact details, allowing 24 hour access to the on call renal physician if symptoms of hyperkalaemia developed.

Dietary assessment

5 day food intake diaries [16], were analysed and assessed prior to commencing the intervention diet. This information was used to determine the appropriate intervention diet. A further 5 day food diary was used to assess nutritional intake within the final week of the intervention diet. Food diaries were analysed using Microdiet[®] version 2 analysis package (Downtree Systems Ltd, Chapel-en-le-Frith, Derbyshire, UK). Energy (kcal), protein, carbohydrate, total fat, saturated fat, polyunsaturated fat, monounsaturated fat, sodium, potassium, phosphorus, calcium, magnesium and fibre intake were assessed.

Outcome measures

The primary outcome measures were changes in serum and urine biochemistry and changes in sodium, potassium, phosphorus and protein consumption over the 5 week diet intervention period. Secondary end points were changes in blood pressure and ECF volume, changes in intake of other macro- and micro nutrients, and acceptability of the diet.

Statistical analyses

Data were analysed by comparing baseline and week 5 results using Wilcoxon Signed Ranks test for paired data. Data from the acceptability questionnaire was analysed using the median of the Likert 7 point ordinal scale.

Results and Discussion

A flowchart of the study population is shown (Figure 1). Three hundred and sixty two potential participants were identified, from which 34 participants commenced the intervention diet. One participant was withdrawn due to a serum potassium of 5.5 mEq/L at the end of the initial run in week. A second was withdrawn for an unrelated emergency hospital admission. A study population of 32 participants (22 male) completed the 5 week intervention diet. All were white Caucasian (reflecting local demographics). Mean age was 69.9 years (43-87) and mean eGFR 48.2 ± 8 ml/min/1.73m² (31-59). All but one participant were prescribed antihypertensive medication (mean number of prescribed drugs=2.1, range 0-5, see Figure 2) and 27 were taking an ACE-inhibitor or angiotensin-2 receptor blocker as recommended by NICE guidance [19]. There were no medication changes during the study.

Serum sodium decreased significantly (139.7 ± 2.6 to 138.9 ± 2.6 mEq/L, $p=0.03$), but remained within the reference range. No other

significant changes in serum biochemical markers were observed (Table 1). There was a significant reduction in urine sodium ($p<0.02$) and no change in other urine biochemistry (Table 2).

There was a significant reduction in intake of total fat ($p<0.001$) leading to a reduction in overall energy intake ($p<0.001$) (Table 3). Protein and carbohydrate intake did not change. A protein intake of 19% of total energy was consumed equating to 1.1 g/kg/day. Sodium intake decreased from 2784.9 mg to 1583.4 mg per day ($p<0.001$) and magnesium and fibre intake increased ($p < 0.007$ and $p < 0.01$ respectively). There was a non-significant increase in potassium intake (3712.4 mg to 3952 mg per day) and no change in phosphate or calcium intake.

Mean weight decreased from 81.4 ± 16.1 kg to 79 ± 15.4 kg ($p<0.001$) and this was associated with a reduction in ECF volume from 18.3 ± 3.2 L to 17.9 ± 3.2 L ($p<0.002$) (Table 4). Mean systolic and diastolic BP both decreased by 9.5 mmHg ($p<0.009$) and 5.3 mmHg ($p<0.006$) respectively.

The results of the acceptability questionnaire (Table 5) demonstrated that the diet was easy to follow, enjoyable, cost approximately the same as participants' usual food expenditure and the majority of participants would have been willing to continue with the diet.

Safety outcomes and adverse events

Two participants reported adverse events that may have been study related. One complained of headache, light headedness and a fall in blood pressure. A second reported hand and leg cramp. Both episodes were of short duration and resolved spontaneously. One participant had a serum potassium of 6.5 mEq/l on their final visit (week 5). A repeat blood test 4 days later was in normal range without consequence or intervention as the participant's usual diet reflected the DASH diet principles.

Discussion

The original Dietary Approaches to Stop Hypertension (DASH) trial was a randomised, controlled, multi centre feeding study of 459 American adults that aimed to identify a practical and acceptable diet that reduced blood pressure [4]. The three diets, a control diet reflecting typical American food choices, a diet similar to the control diet but high in fruits and vegetables and a combination diet all had the same sodium content. The combination diet had the greatest effect, with the largest reductions in blood pressure seen in hypertensive subjects, and became known as the DASH diet [5]. This dietary pattern encourages the consumption of fruits, vegetables, low fat dairy produce, whole grain foods and beans, pulses and nuts, with less animal protein, saturated fats and refined sugar. A follow up study which randomised subjects on the DASH diet to high (150 mmol), intermediate (100 mmol) or low (50 mmol) sodium levels demonstrated a synergistic effect of reducing sodium intake on blood pressure reduction [6]. A sodium intake lower than 100 mol per day in combination with the DASH diet is therefore recommended for

Table 6: DASH diet target nutrient composition compared to calculated achieved nutrient intakes.

	DASH Nutrient target*	DASH achieved nutrient intake†	UK DASH style diet #	Present free living study
Energy (Kcal)	2100	2609 ± 561	2173 ± 154	1737 ± 432
Protein (% TE)	18	18 ± 0.4	21 ± 1	19 ± 2.9
Carbohydrate (% TE)	55	58 ± 0.3	52 ± 1	55 ± 5.1
Total fat (% TE)	27	27 ± 0.4	24 ± 1	27 ± 4.9
Sodium (mg)	3000 (130mmol)	3406 ± 589	1850 ± 190	1583.4 ± 540.0
Potassium (mg)	4700 (120mmol)	5163 ± 668	5140 ± 370	3952.0 ± 785.9
Calcium (mg)	1240	1446 ± 236	1050 ± 100	901.1 ± 238.9
Phosphorus (mg)‡	n/a	1729 ± 302	1950 ± 160	1552.1 ± 405.1
Magnesium (mg)	500	535 ± 81	510 ± 40	397.2 ± 112.0
Fibre (NSP) (g)	31	35 ± 6	37 ± 4	16.2 ± 5.8

* target nutrients based on the planned 2100kcal diet [5].

† Mean± standard deviation nutrient intake achieved by DASH participants [5].

Mean± standard deviation nutrient intake achieved using the UK based DASH style diet [6].

‡ not a target nutrient

greatest effect on blood pressure reduction.

These original studies used controlled feeding conditions, but the DASH diet has subsequently been widely studied in free living individuals, demonstrating its acceptability and efficacy in the American population [20], and emphasising greater blood pressure reductions achieved by combined dietary and lifestyle changes [21].

The DASH diet has also been adapted to reflect British food preferences and portion sizes for the free living UK population [14]. The target and achieved nutrient intakes for the original DASH trial, the UK modified DASH diet and for the present study are shown in Table 6.

In this free living study, the potassium, phosphorus, magnesium and fibre intake achieved were lower than that demonstrated in the original DASH trials or the UK-modified DASH diet. In our free living study whilst participants achieved a protein intake of 19% of total energy this equated to only 1.1 g/kg/day. Participants of the present study also consumed a lower sodium intake when compared to previous trials. The diet was well tolerated by participants. One subject had an elevated serum potassium, but this did not appear to be related to the dietary intervention and resolved spontaneously. Two subjects reported symptoms consistent with either hypotension or volume depletion. Again these symptoms resolved without alterations to prescribed antihypertensive medication.

Concerns have been raised recently with regards the U-shaped relationship between mortality and dietary sodium intake [22]. This relationship has been made in observational cross-sectional data. A postulated mechanism of harm is activation of the renin-angiotensin-aldosterone system by low sodium intake. The majority of patients in the present study were appropriately prescribed drugs that inhibit angiotensin.

The study has a number of limitations and strengths. Of the initial 362 potential participants identified, 303 declined to take part. Further exclusions, due to a failure to meet potassium, bicarbonate or eGFR inclusion criteria following baseline investigations, resulted in an evaluated study population of 32 participants. While small, this observed reduction from original participant identification to final sample size is comparable to that in a recently published dietary study, examining dietary sodium reduction in CKD [23]. In the 2017 study by Saran et al. [23], an initial eligibility screening of

622 patients generated a randomized sample of 58 participants. The study population and length of follow up in our study both compare favourably with a sample size of 11 participants and a two week follow up period in the feeding study by Tyson et al. [13], which also investigated the potential use of the DASH diet in CKD.

Whilst many of the previous DASH trials were conducted as feeding studies, in which all food is provided to support adherence to the prescribed dietary intake, a strength of this study is the use of the DASH diet in free living subjects with CKD.

Since the study aimed to identify the feasibility of the DASH diet in CKD, as well as safety, it was important to assess the sustainability of this dietary intervention by using a 'real life' model, whilst acknowledging that this may result in varying adherence to the diet.

Despite its small sample size and not achieving the DASH target mineral intake, within the self-selected method, over a 5 week follow up period, this study demonstrated a significant reduction in extracellular fluid volume and systolic and diastolic blood pressure. The magnitude of blood pressure reduction would translate into a significant reduction in risk of progression of renal failure and in mortality. A limitation of the study was that blood pressure measurement relied on office readings. Ambulatory blood pressure monitoring would be preferable in a larger study.

Conclusions

The DASH diet appears to be safe in non-diabetic subjects with stage 3 CKD even when prescribed antihypertensive medication that blocks the angiotensin-aldosterone system. Additional monitoring of serum potassium may be required. Significant and symptomatic falls in blood pressure may occur, which may be a desirable outcome, but requires active monitoring.

A larger population based study, including patients with diabetes, is warranted.

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