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The Importance of Potassium in Managing Hypertension

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Abstract Dietary potassium intake has been demonstrated to significantly lower blood pressure (BP) in a dose-responsive manner in both hypertensive and nonhypertensive patients in observational studies, clinical trials, and several meta-analyses. In hypertensive patients, the linear dose–response relationship is a 1.0 mm Hg reduction in systolic BP and a 0.52 mm Hg reduction in diastolic BP per 0.6 g per day increase in dietary potassium intake that is independent of baseline potassium deficiency. The average reduction in BP with 4.7 g (120 mmol) of dietary potassium per day is 8.0/4.1 mm Hg, depending race and on the relative intakes of other minerals such as sodium, magnesium, and calcium. If the dietary sodium chloride intake is high, there is a greater BP reduction with an increased intake of dietary potassium. Blacks have a greater decrease in BP than Caucasians with an equal potassium intake. Potassium-induced reduction in BP significantly lowers the incidence of stroke (cerebrovascular accident, CVA), coronary heart disease, myocardial infarction, and other cardiovascular events. However, potassium also reduces the risk of CVA independent of BP reductions. Increasing consumption of potassium to 4.7 g per day predicts lower event rates for future cardiovascular disease, with estimated decreases of 8% to 15% in CVA and 6% to 11% in myocardial infarction.

Keywords Arterial hypertension · Potassium · Sodium · Potassium/sodium ratio · Stroke · Coronary heart disease · Renal disease · Cardiovascular disease · Congestive heart failure · Dietary control of hypertension · Minerals and hypertension · Clinical trials of potassium and hypertension · Meta-analysis of potassium and hypertension · Nutritional medicine · Lifestyle modifications

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Introduction

Hypertension remains the leading cause of cardiovascular disease (CVD), affecting approximately 1 billion individuals worldwide [1]. More than 72 million Americans, or nearly 1 in 3 adults, are estimated to have hypertension but only 34% achieve blood pressure (BP) control [2–5]. Nearly 70 million more adults are at risk of developing prehypertension, BP between 120/80 mm Hg and 140/90 mm Hg. Over 90% of adults in the United States will probably develop hypertension, especially systolic elevations, by age 65 [3]. Hypertension is associated with an increased risk of morbidity and mortality from stroke (cerebrovascular accident, CVA), coronary heart disease (CHD), myocardial infarction, congestive heart failure, and end-stage renal disease. Poor BP control is even more of a challenge for patients with diabetes and chronic kidney disease, who have lower recommended BP goals [6]. Hypertension remains the most common reason for patient visits to physician's offices and is the primary reason for the use of prescription antihypertensive drugs, with an annual cost of almost $20 billion.

Diet in the Prevention and Treatment of Hypertension

Several epidemiologic studies [7–10] suggest that diet plays an important role in determining BP. Dietary therapies known to lower BP include reduced sodium intake, increased potassium and magnesium intake, a diet rich in

fruits and vegetables, various antioxidants, and other "designer foods" and nutritional supplements [11–14, 15•]. The landmark Dietary Approaches to Stop Hypertension (DASH) trials I and II [11–14] demonstrated that modifying diet significantly lowers BP in patients with stage 1 hypertension and high-normal BP. The DASH diet, which emphasizes fruits, vegetables, high fiber, and low-fat dairy products, also lowers BP in persons with isolated systolic hypertension [11–14]. The recent American Society of Hypertension (ASH) position paper on dietary approaches to lower blood pressure [16•], as well as the American Heart Association (AHA) recommendations for the prevention and management of hypertension [17], also recognize the role that various foods and nutrients play in lowering BP. Increased potassium intake and the DASH diet are also current recommendations of guidelines for lowering BP from the European Society of Hypertension (ESH) [18], the World Health Organization (WHO) and International Society of Hypertension (ISH) [19], and the British Hypertension Society [20].

The Relationship of Potassium to Hypertension, CVD, and CVA

The cardioprotective effects of dietary potassium have been hypothesized as the basis for low CVD rates in populations consuming primitive (Paleolithic) diets and in vegetarians in industrialized countries [21]. In isolated societies consuming diets high in fruits and vegetables, hypertension affects only 1% of the population, whereas in industrialized countries consuming diets high in processed foods, trans fats, saturated fats, refined carbohydrates, low fiber, large amounts of dietary sodium, and reduced dietary potassium and magnesium, about one third of the population have hypertension [22]. In Paleolithic diets, the daily intake ranges from 20 to 40 mmol/day for sodium and from 150 to 290 mmol/day for potassium. In comparison, the daily intake for populations of industrialized societies is 80 to 250 mmol/day for sodium and 30 to 70 mmol/day for potassium. In the modern Western diet, the potassium-to-sodium intake ratio on a molar basis is usually less than 0.4, whereas in primitive cultures, the intake ratio is greater than 3.0 and may be closer to 10.0 [7].

Low potassium intake in the United States is considered a major contributor to the prevalence of hypertension and CVD, with the percentage of attributable risk (PAR) for low potassium intake at 17% for hypertension (systolic BP [SBP] >140 mm Hg) [23]. In the United States, increased potassium intake alone would decrease the number of adults with known hypertension by 17% and would increase life expectancy by 5.1 years for over 12 million Americans [24]. Hypertension mortality was documented at 54,707 for 2008, and hypertension was mentioned as an underlying cause for mortality for about 300,000. Conservatively, an increase in potassium intake could save over half a million lives over the next decade.

In industrialized countries, potassium intake has been reported to be inversely related to BP [25–34] and to the incidence of CVA [27] and other cardiovascular diseases. An increase in dietary intake from approximately 60 to 80 mmol/day is inversely and significantly related to the incidence of CVA mortality in adult women [27]. A significant inverse relationship between potassium intake and the risk of CVA was also demonstrated in a study of 43,738 US men, aged 40 to 75 years, who were followed up for 8 years (P=0.007) [35]. Increasing potassium intake has a direct effect on preventing CVA, independent of its effect on BP, through a variety of mechanisms that are reviewed later [27, 35].

Evidence Showing a Link Between Potassium and Hypertension

A consistent body of evidence from observational studies [8, 27, 31, 36–41], clinical trials, and meta-analyses [23, 42–44] indicates that a high dietary intake of potassium is associated with lower BP. In three meta-analyses, increased potassium intake significantly lowered blood pressure in nonhypertensive and hypertensive individuals in a dose-dependent manner [23, 42, 43]. Although the results from observational trials are relatively consistent, data from individual clinical trials have been less consistent and compelling, perhaps because most trials have been of short duration and the type and dose of potassium used may have varied.

Epidemiologic Evidence

Epidemiologic studies demonstrate that BP is lower in populations consuming primitive (Paleolithic) diets and in vegetarians in industrialized countries, and this reduction in BP decreases CVD [7, 8, 41]. One population study in St. Lucia [45] suggested that an increase in dietary potassium from only 20 to 30 mmol/day (742–1173 mg/d) could result in a 2 to 3 mm Hg reduction of SBP in the population. This potassium intake is equivalent to an increase of three servings of either fruits or vegetables per day.

The Yanomami Indians in Brazil, who consume very little sodium and follow mostly a vegetarian diet, are also known for having low average BP, no hypertension, and no increase in BP with age [46]. As part of the INTERSALT study, this population was found to have very low urinary sodium excretion (0.9 mmol per 24 h), mean SBP of 94.5 mm Hg, and mean diastolic BP (DBP) of 61.4 mm Hg.

In addition, urinary sodium excretion showed a positive correlation with SBP and urinary potassium excretion, a negative correlation [8, 46]. In the presence of low sodium intake among the Yanomami, potassium, which was found to be related to BP in the overall INTERSALT study, was not consistently related to BP in this population. Furthermore, the Yanomami had the lowest urinary sodium excretion (0.2 mmol per 24 h) and had no increase of SBP with age [8]. The extent of BP reduction from potassium depends on concurrent levels of sodium chloride intake [16•]. Specifically, potassium lowers BP more in the presence of a high dietary intake of sodium chloride.

Observational Data

Although most observational studies have demonstrated an inverse relationship between potassium intake and BP [8, 27, 31, 36–41], not all studies are consistent [25, 47–50]. This difference may be due to the high degree of correlation (multicollinearity) among dietary factors, so that it is difficult to separate the effect of potassium from the effects of other nutrients found in potassium-rich foods [31]. Many of these studies included populations with diverse dietary patterns that were observed for over 4 years, providing insight into the long-term effects of habitually consuming a diet high or low in potassium.

The INTERSALT study [51] also provided evidence that potassium intake (as measured by 24-hour urinary potassium excretion) is an important determinant of population BP, independent of sodium. In INTERSALT, an inverse association between urinary excretion of potassium and BP levels was found across diverse populations. More specifically, an increase from the baseline potassium intake of 1173 to 1564 mg was associated with a reduction of SBP of approximately 2 to 3 mm Hg on a population level [51].

Although most intervention studies have focused on high levels of potassium intake, observational studies show that even increasing potassium by 750 to 1000 mg per day can lower BP by 2 to 3 mm Hg [8, 27, 31, 36–41]. This reduction translates into an important cardiovascular benefit in terms of reducing CVA and other CVD events.

Meta-Analyses and Dose Response

Several meta-analyses [23, 42–44] show a significant reduction in BP with potassium supplementation (Table 1). An earlier meta-analysis by Cappuccio and MacGregor [42] of 19 clinical trials examining the effect of potassium supplementation on BP found that oral potassium supplements significantly lowered SBP by 5.9 mm Hg and DBP by 3.4 mm Hg. The average amount of potassium given was 86 mmol/day (primarily as potassium chloride, KCl) with an average duration of 39 days. The magnitude of BP lowering was greater in patients with hypertension (8.2/4.5 mm Hg) and was more pronounced with longer duration of treatment ($P<0.05$ for SBP and $P<0.01$ for DBP).

A meta-analysis of 33 randomized controlled trials performed by Whelton et al. [43] also documented that potassium supplementation significantly lowered BP. This meta-analysis included 12 trials in normotensive individuals and 21 in hypertensive patients, with a duration ranging from 4 days to 3 years (median, 5 weeks). On average, a typical dose of 60 to 120 mmol/day (2.5–5.0 g/d) (median, 75 mmol/d) of supplemental potassium reduced SBP by 4.4 mm Hg and DBP by 2.5 mm Hg in hypertensives and by 1.8 mm Hg SBP and 1.0 mm Hg DBP in normotensives. Although most studies used KCl, some used diet and potassium citrate and bicarbonate as a source of potassium (60 mmol of potassium is equivalent to 4.5 g of KCl, 6 g of potassium bicarbonate, or 20 g of potassium citrate). The BP-lowering effect was more pronounced in blacks than in whites and was greater in those consuming a diet high in sodium chloride.

A meta-regression analysis by Geleijnse et al. [23] provided further evidence of increased BP sensitivity to potassium in hypertensives. In this analysis, an increased potassium intake (median, 44 mmol/d, or 1.7 g) resulted in a mean BP lowering of 2.4 mm Hg for SBP and 1.6 mm Hg for DBP. These estimates are somewhat conservative compared with the earlier meta-analyses by Whelton et al. [43] (3/2 mm Hg) and by Cappuccio and MacGregor [42] (6/3 mm Hg); the difference may be due to the exclusion of short-term trials (<2 weeks duration) from this study. As with the previous meta-analyses, the BP response was greater in hypertensives (3.5/2.5 mm Hg) than in normotensives (0.97/0.34 mm Hg), a difference that was of borderline statistical significance.

A more recent meta-analysis of five randomized controlled trials conducted by Dickinson et al. [44] showed that potassium supplementation resulted in large (but statistically nonsignificant) reductions in both SBP (3.9 mm Hg) and DBP (1.5 mm Hg). Overall reductions in BP were smaller when one trial in an African population with a very high baseline BP was excluded. Further sensitivity analysis limited to two high-quality trials also showed nonsignificant reductions in BP. Because of the small number of participants in these two trials, the short duration of follow-up (8–16 weeks), and substantial heterogeneity between trials, evidence concerning the effect of potassium supplementation on BP was found to be inconclusive [44].

All of these meta-analyses reveal the dose–response relationship between potassium intake and BP lowering. Significant BP lowering with doses of potassium in the

Table 1 Summary of meta-analyses of trials of the effect of potassium on blood pressure

Meta-analysis	Trials, *N*	Intervention[a]	Average duration	Mean BP lowering (SBP/DBP), *mm Hg*	95% CI (SBP; DBP), *mm Hg*
Cappuccio and McGregor [42]	19	100 mmol/d diet; 48–120 mmol/d KCl; 66 mmol/d K Glu + K Cit	39 day	5.9/3.4	−6.6 to -5.2;−4.0 to 2.8
Whelton et al. [43]	33	100–200 mmol/d diet; 60–120 mmol/d KCl; 120 mmol/d K Cit + K Bicarb	5 week	3.1/2	−1.9 to -4.3;−0.5 to -3.4
Geleijnse et al. [23]	27	44 mmol (1.7 g) (form not given)	>2 week	2.4/1.6	−3.8 to -1.0;−2.7 to -0.5
Dickinson et al. [44]	5	>100 mmol/d diet; 48–120 mmol/d KCl; 120 mmol/d K Cit + K Bicarb	≥8 week	11.2/5.0	−25.2 to 2.7;−12.5 to 2.4

[a] Forms of K + include potassium chloride (KCl), citrate (K Cit), gluconate (K Glu), and bicarbonate (K Bicarb). One milliequivalent (Meq) or millimole (mmol) of K + equals 39.09 mg (mg).

BP blood pressure; *CI* confidence interval; *DBP* diastolic blood pressure; *SBP* systolic blood pressure.

range of 1900 to 4700 mg/d (49–122 mmol/d) resulted in BP lowering of approximately 2 to 6 mm Hg for SBP and 2 to 4 mm Hg for DBP. The high variability between these results reflects the variability observed in different studies. In addition, the effect of potassium on BP is influenced by pretreatment BP level; age; race; gender; comorbid conditions; intake of sodium, magnesium, calcium or other ions; diet; exercise; weight; type of potassium used; concomitant medications; and duration of use. Figure 1 summarizes the findings of all meta-analyses to date on the effects of potassium on BP.

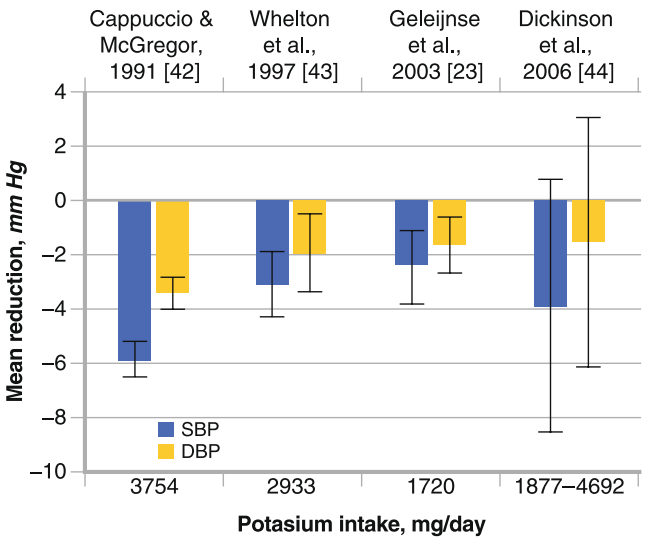

Fig. 1 Overview of meta-analyses of studies investigating the blood pressure–lowering effects of potassium. DBP, diastolic blood pressure; SBP, systolic blood pressure

The DASH Eating Plan

The Dietary Approaches to Stop Hypertension (DASH) study [11] was a controlled feeding study of 11 weeks' duration designed to assess the effects on BP of modifying whole diets. The DASH trial demonstrated that a diet rich in fruit and vegetables, fiber, and low-fat dairy products, with reduced saturated and total fat, can result in a clinically significant reduction in BP, compared with the typical American diet [14]. The reduction in BP began within 2 weeks of feeding and was maintained for the following 6 weeks. Among normotensive individuals, this diet reduced SBP by 3.5 mm Hg and DBP by 2.1 mm Hg. A subgroup analysis of the DASH trial also found that the BP-lowering effects were more pronounced in hypertensives (11.4 and 5.5 mm Hg) and in blacks [12]. Although potassium intake was increased by 1447 to 2776 mg per day through increased consumption of fruit and vegetables, reductions in BP cannot be attributed to potassium alone, as the diet was also rich in calcium, magnesium, and other nutrients.

The DASH diet is also effective as first-line antihypertensive therapy for the treatment of isolated systolic hypertension [11–14]. Svetkey et al. [3] further demonstrated that in patients with stage 1 hypertension, reduced sodium intake in combination with the DASH diet improved BP control. Among DASH-Sodium trial participants, sustained reductions in BP were observed over a 1-year period despite increased sodium intake [52]. The DASH diet is more effective than potassium, magnesium, and fiber supplements for lowering BP in obese, hypertensive patients, which suggests that the high consumption of fruits and vegetables in the DASH diet lowers BP and improves endothelial

function in the group by nutritional factors in addition to potassium, magnesium, and fiber, such as antioxidant and anti-inflammatory effects [53, 54].

Other Clinical Trial Evidence

Overall, conflicting results regarding the effects of potassium supplementation on BP have been reported in clinical studies [55–57]. More recent trials, however, have demonstrated results consistent with that of the meta-analysis by Whelton et al. [43] (Table 2). Gu et al. [58] found that moderate potassium supplementation (60 mmol KCl) taken for 12 weeks resulted in a substantial reduction in SBP, but not DBP, in a Chinese population. Similarly, Kawano et al. [59] documented that a 4-week potassium supplementation period (during which 64 mmol/d of potassium was given as slow-release KCl) resulted in small but significant reductions in office, home, and 24-hour BP in Japanese men and women. Braschi et al. [60] further examined the effect of low-dose potassium supplementation on BP and found that 24 mmol/day of slow-release KCl administered for 6 weeks significantly reduced mean arterial pressure and DBP in healthy volunteers.

Other Beneficial Effects of Potassium on Cardiovascular Disease

High potassium intake may have other beneficial effects independent of its effect on BP, such as reducing the risk of CVA and cardiac arrhythmias. For example, increasing dietary intake of potassium from approximately 60 to 80 mmol/day has been shown to be inversely and significantly related to the incidence of CVA mortality in women [27]. A similar pattern was demonstrated in US men: the multivariate relative risk of CVA of any type for men in the top fifth of potassium intake (median intake, 4.3 g/d) versus those in the bottom fifth (median intake, 2.4 g/d) was 0.62 [35].

Because increasing potassium intake lowers BP, it is difficult to distinguish between the effects of potassium on BP mediated by BP lowering alone from those mediated by a direct effect of potassium. A direct protective effect of potassium on CVA is suggested by studies in the rat model, in which a high potassium intake was associated with a large reduction in CVA mortality [61]. This was observed even when BP was precisely correlated with high and low potassium intakes. Epidemiologic studies further demonstrate that a high potassium intake is related to a lower risk of CVA, and some of this effect may be independent and additive to the effect of potassium on BP [62]. Many proposed mechanisms of potassium's effect on vascular function and health are independent of BP reduction and could be related to the decrease in CVA. (See the Mechanisms section below.)

Several observational studies have also found a link between potassium intake and risk reduction for CVA. In a 12-year prospective study by Khaw and Barret-Connor [27], an increase in potassium intake of 10 mmol/day among 859 men and women resulted in a 40% reduction in stroke mortality. This association was found to be independent of other dietary variables, independent of BP and CVD risk factors. In two additional studies with much larger cohorts, US health professional men (43,738 men) [35] and US nurses (85,764 women) [63], high potassium intake also resulted in a lower risk of CVA. Moreover, study in US professional men [35] demonstrated a dose–response relationship between potassium intake and risk of CVA, whereas the study of US nurses [63] showed a borderline significant association between the intake of potassium and CVA after adjusting for confounding variables.

For arrhythmias, hypokalemia results in prolonged repolarization, which underlies the pathologic mechanism of torsade de points, especially in patients with ischemic

Table 2 Other clinical trial evidence of the effect of potassium on blood pressure

Study	Subjects, *N*	Intervention	Average duration	Mean BP lowering (SBP/DBP), *mm Hg*	*P* value
Appel et al. [11]	459	DASH diet	8 week	3.5/2.1 Non-HTN; 11.4/5.5 HTN	<0.001/0.003; <0.001
Sacks et al. [13]	412	Intermediate-sodium (control diet vs DASH diet)	30 day	2.1/1.1 control; 1.3/0.6 DASH	<0.05; <0.001
		Low-sodium (control diet vs DASH diet)		4.6/2.4 control; 1.7/1.0 DASH	<0.001; <0.01
Gu et al. [58]	150	60 mmol/d KCl	12 week	5.0/0.63	<0.001/NS
Kawano et al. [59]	55	64 mmol/d KCl (slow-release)	4 week	2.7/1.4	<0.05
Braschi et al. [60]	59	24 mmol/d KCl (slow-release)	6 week	↓MAP 7.01; 7.60/6.46	<0.001/<0.001

BP blood pressure; *DASH* Dietary Approaches to Stop Hypertension; *DBP* diastolic blood pressure; *HTN* hypertensives; *KCl* potassium chloride; *MAP* mean arterial pressure; *non-HTN* nonhypertensives; *SBP* systolic blood pressure.

heart disease, heart failure, or left ventricular hypertrophy. Increasing serum potassium concentrations improves repolarization in patients with inherited or acquired long-QT syndromes [64]. The risk of arrhythmia is further increased in hypertensive patients taking non–potassium-sparing diuretics. In the Multiple Risk Factor Intervention Trial (MRFIT) [65], a 28% increase in ventricular arrhythmia was observed for every 1 mmol/day decrease in serum potassium among the 1,403 hypertensive men taking diuretics. MRFIT and other studies such as the Systolic Hypertension in the Elderly Program (SHEP) showed that low serum potassium induced by thiazide diuretic treatment resulted in minimal or no reduction in CV events, compared with patients who had normal serum potassium [65, 66].

Mechanisms by which Potassium Lowers BP

The homeostasis of sodium and potassium plays an important role in endothelium-dependent vasodilatation [67]. Sodium retention decreases the synthesis of nitric oxide, an arteriolar vasodilator elaborated by endothelial cells, and increases the plasma level of asymmetric dimethyl l-arginine, an endogenous inhibitor of nitric oxide production [67]. Sodium restriction induces the opposite effects.

A diet rich in potassium and increases in serum potassium (even within the physiologic range) cause endothelium-dependent vasodilatation by hyperpolarizing the endothelial cell through stimulation of the sodium pump and opening of potassium channels [68, 69]. Endothelial hyperpolarization is transmitted to the vascular smooth-muscle cells, resulting in decreased cytosolic calcium, which in turn promotes vasodilatation. In contrast, experimental potassium depletion inhibits endothelium-dependent vasodilatation [68].

In addition to increased vasodilatation, other proposed mechanisms by which potassium can influence BP include natriuresis, alterations in intracellular sodium and tonicity, modulation of baroreceptor sensitivity, reduced vasoconstrictive sensitivity to norepinephrine and angiotensin II, increased serum and urinary kallikrein, increased sodium/potassium ATPase activity and alteration in DNA synthesis and proliferation in vascular smooth muscle and sympathetic nervous system cells, improved insulin sensitivity, reduction in cardiac diastolic dysfunction, decrease in vascular neointimal formation, reduction in transforming growth factor (TGF)-β, and decreases in NADPH oxidase, oxidative stress, and inflammation [70–76].

Dietary Guidelines for Potassium

Several national and international guidelines have incorporated an increased dietary intake of potassium as part of their recommendations for the prevention and treatment of hypertension. Maintaining an adequate intake of dietary potassium (>90 mmol [3,500 mg] per day) has been recommended for the primary prevention of hypertension by the National High Blood Pressure Education Program Coordinating Committee (JNC 7) in 2003. The Institutes of Medicine have recommended a sodium intake below 65 mmol/day (3.8 g/d) and an increase in potassium to 120 mmol/day [77]. In 2006, the American Heart Association (AHA) issued new guidelines suggesting an increase in potassium intake to 120 mmol/day (4.7 g/d), which is the level provided in the DASH diet [17]. In 2010, the ASH recommended about 4.7 g/day of potassium [16•]. The most recent ESH guidelines also support increased potassium intake based on the DASH diet [18]. In addition, the 2003 World Health Organization (WHO)/ISH statement recommends a diet high in fruits and vegetables, reduction of dietary sodium intake, and increased dietary potassium intake to reduce the incidence of hypertension [19].

The primary precaution related to this amount of dietary potassium intake would pertain to patients with renal insufficiency or renal tubular acidosis, in which potassium and magnesium retention can be frequent problems. Once the glomerular filtration rate (GFR) is below about 20 mL per minute, the kidney has reduced secretory ability for potassium. In patients with diabetes and some other diseases, renal tubular acidosis may occur in the presence of only mild reductions in GFR and can increase serum potassium.

Not only the potassium intake is important in BP and CVD, but also the ratio of sodium to potassium in the diet. A higher sodium-to-potassium ratio in the diet or sodium-to-potassium ratio in the urinary excretion rate is associated with higher BP and increased risk of subsequent CVD, with an effect stronger than that of sodium or potassium alone [78•, 79]. A high ratio of sodium to potassium intake was positively associated with mortality from total CVA, ischemic CVA, CHD, and total CVD; risk increased from the highest to the lowest quartile in a multivariable hazard ratio analysis [78•, 79]. Increased dietary potassium resulted in a 50% reduction in the need for antihypertensive medications in one study [80]. A randomized clinical trial showed that a salt substitute containing 40% KCl significantly improved BP in patients with diabetes who had hypertension [81]. It should also be recognized that many patients may be magnesium-deficient, which complicates hypokalemia. In such cases, it is important to replace both potassium and magnesium to establish normal blood levels.

A comprehensive nonpharmacologic treatment program that includes increased dietary potassium, low dietary sodium, the DASH diet, exercise, weight control, and selected nutraceutical supplements may allow up to 62% of hypertensive patients to discontinue drug therapy within about 6 to 12 months [82•].

Conclusions

Americans consume double the sodium and about half of the potassium that is recommended by current guidelines. In fact, the average US dietary intake of potassium is 45 mEq/day, with a potassium-to-sodium ratio of less than 1:2—far less than the recommended intake of 650 mEq/day of potassium, with a potassium-to-sodium ratio of over 5:1. As epidemiologic studies in the Yanomami Indians suggest, if we were to achieve the correct potassium-to-sodium ratio through dietary means, there would be less hypertension and CVD in the population as a whole. A high intake of potassium with consumption of fruits and vegetables is important for the prevention of hypertension and major public health problems such as CHD and CVA. Small reductions in BP through increased dietary potassium intake on a population level would have a substantial impact in reducing CVD [82•]. Increasing potassium intake to 4.7 g/day would shift the population SBP distributions down by 1.7 to 3.2 mm Hg, similar to the predicted result of reducing sodium intake from 9 to 5 g/day [82•]. The estimated reduction in CVA mortality would be 8% to 15%, and the reduction in CHD risk would be 6% to 11% [82•].

Disclosure Conflicts of Interest: M Houston: Honoraria from Novartis, Daiichi Sankyo, Boehringer Ingelheim, Forest, Sankyo, Bristol-Myers Squibb; Speaker for Biotics, Institute for Integrative Medicine, American College for Advancement in Medicine, Novartis, Boehringer Ingelheim, Forest, Sankyo, Bristol-Myers Squibb; Consultant for Takeda.

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