Should we eat more potassium to better control blood pressure in hypertension?

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ABSTRACT

Changes in lifestyle and nutrition are recommended as the first-step approach to the management of hypertension by all national and international guidelines. Today, when considering nutritional factors in hypertension, almost all the attention is focused on the reduction of salt intake to improve blood pressure (BP) control. Changes in potassium intake are only briefly evoked in guidelines. Few physicians actually think about proposing to eat more foods that are high in potassium (fruits, vegetables, nuts) to better control BP. Yet, during the last 40 years, increasing evidence has accumulated demonstrating that increasing potassium intake, either with food products or with supplements, is associated with significant reductions of both systolic and diastolic BP. The hypotensive effect of potassium is particularly marked in patients with hypertension and in subjects with a very high sodium intake, suggesting that potassium counterbalances the effects of sodium. In addition, several meta-analyses have now confirmed that high potassium intake reduces the risk of stroke by ~25%. Finally, increasing potassium in the diet may perhaps be beneficial for some renal patients, as post hoc analyses have suggested that a high potassium intake may retard the decline of renal function in patients with early chronic kidney disease (CKD) stages. However, high potassium intake may be risky and sometimes even dangerous in hypertensive patients with CKD stages 3–5, specifically diabetics. In this context, however, as the level of evidence remains low, more prospective clinical studies are needed. The goal of this review is to discuss the actual evidence that supports the recommendation to eat more potassium in order to better control BP in essential hypertension and to review the restrictions in CKD patients with hypertension.

Keywords: chronic kidney disease, hyperkalaemia, meta-analysis, sodium, sodium:potassium ratio

INTRODUCTION

Lifestyle behaviours and nutrition are two important determinants of blood pressure (BP) and the risk of developing hypertension. All hypertension guidelines recommend initiating the management of patients with hypertension or pre-hypertension by non-pharmacological approaches such as increasing physical activity and, when necessary, reducing body weight, decreasing alcohol consumption and stopping tobacco smoking [1–3]. As far as nutritional factors are concerned, recommendations are mainly focused on the reduction of sodium intake. Indeed, epidemiological surveys and clinical studies have demonstrated well-documented associations between salt intake and BP, the increase in BP with age, the risk of developing hypertension and the risk of developing high BP-induced cardiovascular and renal complications [4–7]. Moreover, there is strong evidence that reducing salt intake is associated with a reduction in BP in hypertensive patients and the lower target of salt intake to be reached remains a topic of debate because of an apparent increase in mortality at very low levels of salt intake [4, 8–11].

Many other dietary factors may be considered in the recommendations for healthy nutrition in patients with hypertension, such as potassium, calcium, proteins and magnesium. The results of the Dietary Approaches to Stop Hypertension (DASH) trial have clearly established that a diet rich in fruits, vegetables, low-fat dairy products with reduced total and saturated fat, cholesterol and sugar-sweetened products significantly lowers BP in participants without hypertension as well as in patients with pre-hypertension or Stage 1 hypertension [10]. The benefits of the non-salt components of the diet were actually additive to those of reducing salt intake. Therefore, application of the DASH diet, which contains 4.7 g of potassium, should be part of the recommended lifestyle modifications for all hypertensive patients, as mentioned in the Eighth Joint National Committee update on lifestyle changes in hypertension [3].
The favourable impact of potassium on BP is not a new story. In the 1950s, Meneely et al. [12, 13] discussed the toxicity of sodium and its role in hypertension and the protective effect of adding potassium to the diet. Later, in the 1980s, Tobian et al. [14] published several important experimental studies in various models of hypertensive rats demonstrating the protective role of potassium against hypertension-induced cerebral lesions and stroke mortality and the development of renal arteriolar lesions independent of any change in BP [15]. One important observation made by Tobian and his team is that increasing potassium in the diet prevented an increase in BP in spontaneously hypertensive rats almost exclusively on a high-sodium diet [16]. In humans, well-designed studies showing that potassium lowers BP were also conducted in the early 1980s [17, 18]. In 1987, Khaw and Barrett-Conner [19] examined stroke mortality in a population-based study according to potassium intake and reported for the first time a significant 40% reduction in the risk of stroke-associated mortality for each 10 mmol increase in daily potassium intake.

The purpose of the present article is to review the actual evidence supporting the recommendation to increase potassium not only to lower BP in patients with essential hypertension but also to reduce the risk of cardiovascular and renal complications in patients with mild to moderate chronic kidney disease (CKD).

**WHAT IS THE EVIDENCE LINKING POTASSIUM INTAKE AND BP?**

Multiple observational and interventional studies have investigated the impact of potassium on BP and the risk of developing hypertension and the ability of potassium supplementation to reduce BP in hypertensive patients. In recent years, several large surveys have been published adding further evidence supporting greater potassium intake to lower BP. Yet, many questions remain unanswered, limiting our ability to propose firm recommendations with a high level of confidence. These studies have been compiled and analysed in several meta-analyses that will be discussed below.

**Observational population studies**

Almost all early epidemiological studies have reported an inverse correlation between potassium intake and BP and the prevalence of hypertension [24]. The first large population survey to demonstrate an association between potassium intake, measured indirectly through 24-h urinary potassium excretion, and BP was the International Study of Salt (INTERSALT study), which reported a weak but significant negative association between potassium excretion and both systolic and diastolic BP [25]. In the more recently published PURE survey, in which more than 100 000 subjects from 18 countries around the world were studied, potassium excretion was inversely associated with systolic BP, and the association was steeper in subjects with hypertension and in those with increased age [6]. However, one has to mention that in this study, potassium intake was extrapolated from a single morning sample of urine, a method that has been criticized for its reliability to reflect the daily intake of sodium and potassium [26]. In a subanalysis of the Prevention of Renal and Vascular Endstage Disease (PREVEND) study excluding hypertensive patients, Kieneker et al. [27] reported an increased risk of developing hypertension in subjects on a low potassium diet (men: <68 mmol/24 h; women: <58 mmol/24 h) based on a 24-h urine collection (Figure 1). In a cohort of 233 children 5–17 years of age, the systolic BP slope over time was lower when potassium intake was higher and also when the sodium:potassium (Na:K) ratio was higher [28], suggesting that the relative sodium and potassium content of food may play a role in the early phase of development of hypertension.

Beyond BP studies, several studies have examined the association between potassium intake and the risk of cardiovascular...
events and mortality. These studies were part of the meta-analysis of D’Elia et al. [29], who included 11 studies providing 15 cohort samples with 247,510 male and female participants and a follow-up of 5–19 years. Potassium intake was assessed with different methods in the various studies, but the overall result was that higher potassium intake was associated with a 21% lower risk of stroke and also a trend towards a lower risk of CHD and total CVD that attained statistical significance only after the exclusion of a single cohort. The reduced risk of stroke [19, 30] in subjects on a high-potassium diet appears to be particularly relevant and was confirmed in the review of evidence by Hunt and Cappuccio [31], who found a 24% reduction in the risk of stroke on a high-potassium diet in the Third National Health and Nutrition Examination Survey (NHANES III), high potassium intake was associated with a 20% lower mortality risk [32], and in the Japan Collaborative Cohort Study for Evaluation of Cancer Risk, in which 58,730 subjects without CVD at baseline were enrolled, potassium intake was inversely associated with mortality from CHD and total CVD [33]. In this latter study, the multivariable hazard ratio (HR) for the highest versus the lowest quintiles of potassium intake was 0.65 for CHD and 0.73 for CVD. Interestingly, these associations were more evident for women than for men.

One of the critical methodological aspects of all epidemiological studies is the accuracy of the assessment of daily sodium or potassium intake. Indeed, although 24-h urine collection is still considered the gold standard, this method has been criticized for both electrolytes since only 90% of sodium and ~80% of potassium are excreted in urine. Moreover, how many urine collections are needed to correctly assess urinary electrolyte excretion as a surrogate marker of daily intake remains unknown [34]. Of note, a formula based on a single spot urine has also been shown to have limited reliability for sodium [26], and the situation may be even worse for potassium, for which there is a circadian rhythm of excretion.

**Is the Na:K ratio more interesting than urinary sodium or potassium excretion separately?**

Rather than considering urinary sodium or potassium excretion separately, several authors have suggested use of the Na:K ratio to evaluate the impact of salt and potassium intake on BP and cardiovascular complications. The advantages of the ratio are it is not influenced by the quality of the urine collection and it may be a better indicator of the high-sodium, low-potassium diet characteristic of the actual pattern of nutrition. Studies have indeed shown that the Na:K ratio is a better marker than either sodium or potassium alone in relation to BP and also in the evaluation of BP outcomes and incident hypertension [35].

In a population of 584 men and 718 women, 30–79 years of age, in Southern California, Khaw and Barrett-Connor [36] reported a strong association between the Na:K ratio and both systolic and diastolic BP. An important age effect was found in this survey, particularly in men, suggesting an increasing sensitivity to the dietary Na:K ratio with age. In INTERSALT, the estimated reduction in BP was larger for the Na:K ratio compared with sodium and potassium analysed separately. Similarly, in the Swiss survey, urinary potassium excretion was weakly associated with BP and the risk of hypertension whereas the Na:K ratio was significantly associated with both factors [22]. In the last NHANES 2005–2010 survey, the mean Na:K ratio was significantly associated with BP and the prevalence of hypertension [37]. In obese patients with normal BP at baseline, the Na:K ratio significantly predicted systolic BP after controlling for age, sex, body mass index and the use of hypertensive medications [38].

Similarly, in both the NHANES and the Japanese survey discussed above [32, 33], the HRs for all-cause mortality, CVDs and ischaemic heart disease were significantly greater with the Na:K ratio than with urinary sodium or potassium excretion taken separately. In the Trial of Hypertension Prevention (THOP) trial, which investigated the impact of a reduction in salt intake in adults 30–54 years of age with pre-hypertension, the Na:K ratio was strongly associated with an increased risk of subsequent CVDs [39].

Thus the Na:K ratio appears to be a better indicator of a high-sodium, low-potassium diet being associated with higher BP and a greater risk of developing hypertension as well as cardiovascular complications, including cardiovascular mortality. The WHO proposes a target Na:K ratio <1, but a reasonable ratio could be 1.0–1.2, corresponding to a diet containing 100 mmol sodium/day and 90–100 mmol potassium/day. In NHANES, the ratio was 1.41 and in Switzerland the average ratio was 2.4 in women and 2.6 in men, thus way above the desirable target. Considering the actual data, wider use of the Na:K ratio in clinical practice should be recommended to easily assess the imbalance between sodium and potassium intake as proposed recently by Iwahori et al. [40].

**FIGURE 1: Association between 24-h urinary potassium excretion and risk of developing hypertension in the PREVEND study (from Kieneker et al. [27] with permission).**

[Image: potassium-intake-and-blood-pressure.png]
Interventional studies

As mentioned earlier, the first randomized controlled studies assessing the effect of potassium supplementation on BP were conducted in the early 1980s. Potassium chloride supplements were given to healthy normotensive subjects [17] and to patients with mild to moderate hypertension [18]. In both studies, the administration of potassium (40 mmol/day in normotensive subjects and 60 mmol/day in hypertensive patients for 4 weeks) was associated with lower BP or with a significant, though modest, reduction in BP when compared with placebo. Since then, many additional studies have been performed and they have been included in at least three meta-analyses [41–43].

In the first meta-analysis, published in 1991, Cappuccio and MacGregor [41] reviewed 19 clinical studies with a total of 586 participants (412 of whom had essential hypertension). The administration of oral potassium supplements was associated with a significant decrease in systolic BP of 5.9 mmHg (−6.6 to −5.2 mmHg) and diastolic BP of 3.4 mmHg (−4.0 to −2.8 mmHg). The BP response was greater in patients with a higher BP at baseline (−8.2 mmHg for systolic and −4.5 mmHg for diastolic BP). In 2001, the pivotal DASH trial was published, which clearly demonstrated the benefits on BP of lowering salt intake and also of increasing potassium in the diet, and this in normotensive as well as patients with early stages of hypertension [10]. In 2003, Geleijnse et al. [42] published another meta-regression analysis in which randomized trials evaluating the impact of a sodium reduction or potassium supplementation on BP were collected. Twenty-seven trials on potassium supplementation were identified with a minimum duration of 2 weeks. As observed in the meta-analysis of Cappuccio and MacGregor, increasing urinary potassium excretion by a mean of 44 mmol/day was associated with a significant reduction in both systolic and diastolic BP (−2.4/−1.5 mmHg) and the effect was greater in hypertensive patients (−3.5/−2.5 mmHg). These figures were obtained after adjustments for trial design, duration, age, gender, initial BP, initial urinary sodium and potassium excretion as well as changes in urinary sodium and potassium excretion during the intervention. Last, in 2013, Aburto et al. [43] performed the largest Cochrane-type meta-analysis in which all randomized controlled trials (n = 22) and cohort studies (n = 11) reporting the effects of potassium intake on BP, renal function, blood lipids, catecholamine concentrations, all-cause mortality, CVD, stroke and CHD were included. This analysis concluded that there is now high-quality evidence demonstrating that increasing potassium intake lowers BP in people with essential hypertension and has no adverse effect on lipids, catecholamines or renal function in adults. In addition, high potassium intake was again associated with a 24% lower risk of stroke. In this meta-analysis, the decrease in systolic BP reached 7.6 mmHg when the achieved urinary potassium excretion ranged between 90 and 120 mmol/day. The impact of a high potassium intake on BP in various groups of subjects is presented in Figure 2 and the impact on cardiovascular events is shown in Figure 3. Of note, potassium supplementation had no impact on BP in children and the effects on CHD, CVDs and mortality were not significant.

Yet, regarding the impact of a high potassium intake on cardiovascular events, one interesting study is the one published by Chang et al. [44], in elderly men in retirement homes. In this study, kitchens were randomized into two groups to use either potassium-enriched salt or regular salt. Patients with CKD (creatinine ≥3.5 mg/dL) were excluded. In the next 31 months, among the 1981 subjects enrolled in the study, the incidence of CVD-linked deaths was 13.1 per 1000 persons in the experimental group and 20.5 per 1000 persons in the control group, reflecting a 59% reduction in CVD mortality.

Finally, as reported initially by Tobian et al. [15], a high potassium intake is particularly effective in blunting the hypertensive effect of sodium in salt-sensitive animals (Dahl salt-sensitive rats, deoxycorticosterone acetate [DOCA]-salt hypertension models, etc.) and also in salt-sensitive humans such as elderly adults, overweight and obese patients and hypertensive patients of African American origin when they are on a high-sodium diet [45–47]. Indeed, a study has shown that potassium supplementation in patients on a low-sodium diet has almost no effect [48].

Thus there are now sufficient data and evidence to promote a higher potassium intake, at least in patients with an elevated BP, and lowering sodium intake together with an increase in potassium in the regular diet, which may be an excellent first step in the management of patients with mild to moderate hypertension and also in obese and elderly patients without renal impairment.

What kind of potassium supplement should be given?

One of the practical questions is in what form should potassium supplement be prescribed in order to be effective? Most clinical studies have used potassium chloride. However, in fruits and vegetables, the anion accompanying potassium is not chloride. To answer this question, He et al. [49] performed the first randomized crossover study in a small group of hypertensive patients comparing the effects on BP of potassium chloride and potassium citrate given for 1 week. The BP-lowering effect was comparable with the two forms of potassium supplementation. In contrast, a recent double-blind, placebo-controlled study compared the effects of potassium magnesium citrate (KMgCit), potassium chloride (KCl) and potassium citrate (KCit) on 24-h ambulatory BP in hypertensive and prehypertensive subjects, using a randomized crossover design. The goal of this study was to clarify which of the three components of K, Mg and citrate is important in lowering BP [50]. Interestingly, a significant reduction of BP was found with KCl but not with the two other preparations of potassium, indicating that potassium is the key element and that KCl and KMgCit or KCit supplementation have differential effects on BP. Thus these data differ from the initial observation reporting no difference between potassium chloride and potassium citrate.

HOW DOES POTASSIUM LOWER BP?

The pathogenesis of essential hypertension is complex and results from an interplay between many factors, among which the sodium and potassium balance plays a definite role in maintaining blood volume, hydro-electrolyte balance and cell

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Although most of the attention has been focused on sodium, there is good evidence that potassium balance is crucial for BP regulation and that potassium modulates the effects of sodium on BP. The main renal mechanism that has been evoked to explain the BP-lowering effect of a high potassium intake is the development of a negative sodium balance due to pronounced natriuresis explained in part by a downregulation of the sodium chloride cotransporter (NCC) [53]. In humans, both increasing potassium in the diet and infusing KCl results in an increase in urinary sodium excretion and a reduction in BP in hypertensive patients [24]. The natriuretic effect of a high potassium intake is particularly surprising, as it occurs despite elevated plasma aldosterone levels, which are expected to promote sodium retention rather than give rise to sodium loss. High potassium intake has been shown to suppress NCC and low potassium intake has been reported to increase NCC activity [54, 55]. Terker et al. [56] recently demonstrated that extracellular potassium regulates NCC function directly, independent of any hormonal regulation. According to their experiments, plasma potassium affects urinary potassium excretion by altering sodium delivery to the connecting tubule. A low potassium intake hyperpolarizes plasma membrane via a reduction in plasma potassium and causes a reduction of intracellular chloride in the distal convoluted tubule (DCT). The latter has been reported to activate NCC via the With No-lysine Kinase (WNK) pathway, inducing a temporary increase in BP in mice. These experiments suggest that serum potassium modulates NCC function in a hormone-independent manner through changes in intracellular chloride concentrations, which in turn modulate the function of WNK, which regulates several renal transporters including NCC.

The opposite effect is supposed to be determined by high potassium intake. Figure 4 illustrates how changes in potassium intake may affect BP through an adrenorenal pathway involving NCC. Yet, still other renal tubular mechanisms may contribute to explain the effect of potassium intake on BP, and some of them are being studied, such as tissue kallikrein and kinase pathways (SGK1, WNK/SPAK) [53]. Of note, there is also a gastrointestinal regulation of potassium excretion that increases urinary potassium excretion after meals independent of changes in serum potassium [57].

Several non-renal mechanisms for the hypotensive effects of potassium have also been proposed, including reduced renal renin release, decreased vascular smooth muscle cell proliferation, reduced vascular smooth muscle cell migration, decreased free radical formation, reduced low-density lipoprotein cholesterol oxidation, decreased platelet aggregation, improvement of endothelium-dependent vasodilatation and reduction of oxidative stress [58]. Experimentally, we have demonstrated that an increase in potassium in DOCA-salt uninephrectomized mice reverses cardiac and renal hypertrophy [59] and restores vasorelaxation of resistance arterioles independent of BP [60]. All these observations support the role of potassium in lowering BP, but also in preventing the cardiac and cerebral complications of hypertension.

**WHAT ABOUT A HIGH POTASSIUM DIET IN CKD PATIENTS?**

In the nephrology community, recommending potassium supplements is a very sensitive issue unless patients are treated for diseases inducing marked hypokalaemia. The main reason is of course the risk of hyperkalaemia and its potentially lethal
cardiac consequences. However, the incidence of hyperkalemia depends on the CKD stage. It is relatively infrequent in hypertensive patients in Stages 1–3a unless patients receive aldosterone antagonists, very high doses or combinations of blockers of the renin–angiotensin system (RAS) [61] or suffer from concomitant diseases such as severe congestive heart failure or diabetes or consume a very large amount of potassium (>5 g/day) [62]. Table 1 shows a list of drugs that increase serum potassium and may be of concern in patients on a high-potassium diet. Yet, one has to acknowledge that in patients with essential hypertension and normal renal function the risk is very low. In a retrospective analysis of a national cohort comprising 2 103 422 records from 245 808 veterans with at least one hospitalization and at least one inpatient or outpatient serum potassium record during the year 2005, hyperkalemia (potassium > 5.5 mmol/L) was found in 3.2% of the records [63]. In a small group of CKD patients (glomerular filtration rate [GFR] < 60 mL/min/1.73 m²), serum potassium was found to increase by ~ 0.3 mmol/L upon introduction of an RAS blocker [64]. In a recently published survey of 69 426 new users of angiotensin-converting enzyme (ACE) inhibitor/angiotensin II receptor blocker (ARB) therapy in the Stockholm Creatinine
Kidney injury when renal function becomes unstable.

Serum potassium and mortality is U-shaped, with an increased serum potassium levels [62, 67, 68]. In one study, the mortality rate was actually lower in CKD patients with a serum potassium level between 4.0 and 5.5 mmol/L than in those with serum potassium < 4.0 mmol/L [67]. In addition, hypokalaemia was associated with a faster decline in renal function in both white and black males with CKD [68]. This actually suggests that potassium restriction should perhaps not be recommended to CKD patients before Stages 4 and 5.

Interestingly, in a recent post hoc analysis of the ONTARGET (ONgoing Telmisartan Alone and in combination with Ramipril Global Endpoint Trial) and TRANSCEND (Telmisartan Randomised AssessmeNt Study in ACE iNtolerant subjects with cardiovascular Disease) studies, which enrolled 28 879 patients with high cardiovascular risk, there was no significant association between the estimated sodium intake and any renal outcomes defined either as a decrease in eGFR > 30 or 40% or the need for dialysis or progression of proteinuria [69]. However, in this same post hoc analysis, after multiple adjustments, the highest potassium excretion was associated with a 26% risk reduction for renal disease progression (decrease in eGFR > 30% and the need for chronic dialysis) and a 24% risk reduction for the progression of proteinuria. But these patients also had a 16% higher risk of developing hyperkalaemia. Of note, BP was comparable in the various potassium excretion subgroups. In patients with type 2 diabetes and normal renal function (eGFR > 60 mL/min/1.73 m²), high urinary potassium excretion was also associated with a slower decline of renal function and a lower incidence of cardiovascular complications [70]. These observations suggest that an elevated potassium intake may have a greater beneficial impact on renal disease progression than lowering sodium intake in a population with a high cardiovascular risk or in diabetic patients with relatively preserved renal function (Stages 1–3). In contrast, He et al. [71] recently published an analysis of the Chronic Renal Insufficiency Cohort Study in which the associations of urinary sodium and potassium excretion with CKD progression and all-cause mortality were assessed. This analysis concerned 3939 patients with an eGFR of 20–70 mL/min/1.73 m² (mean ~ 45 mL/min/1.73 m²). In this cohort, both elevated urinary sodium and urinary potassium excretion (> 65 mmol/day) were associated with an increased HR for CKD progression (HR 1.59) but not for mortality (HR 0.98). Interestingly, systolic BP was significantly lower in the quartile of patients with potassium excretion > 67 mmol/L when compared with the lowest quartile (126.1 versus 129.9 mmHg; P < 0.001) despite a much higher urinary sodium excretion (197 versus 123 mmol/day, quartile 4 versus quartile 1).

The results of these apparently contradictory studies indicate that as of today, the impact of potassium on BP and the course of renal diseases is still insufficiently documented. In hypertensive patients with a GFR > 60 mL/min/1.73 m², a potassium-enriched diet can be recommended without any fear. However, it is rather obvious that potassium supplements should not be given to patients with advanced CKD (eGFR < 45 mL/min/1.73 m²) because of the risk of hyperkalaemia and perhaps the risk of accelerating the disease. But, in earlier CKD stages (stages 1–3a), whether potassium supplements are damaging or beneficial because they lower BP and reduce cardiovascular complications remains to be demonstrated, as observations suggesting a benefit rather than a risk are increasing. The only way...
to resolve this question will be to perform a randomized controlled prospective trial testing the administration of potassium supplements versus placebo in patients with CKD Stages 1–3. A study protocol has been written, but so far financial support for this trial has not been granted. Today, one study is ready to be started in patients with CKD Stages 3b and 4 in The Netherlands supported by the Dutch Kidney Foundation and entitled Potassium Supplementation in CKD (ClinicalTrials.gov, NCT03253172). Data are expected in a few years but at this level the definite answer to the title of this article is yes, one should eat more potassium to lower BP and prevent cardiovascular events.

CONCLUSIONS

Primitive humans consumed a diet very rich in potassium and poor in sodium. Today this pattern is completely reversed. Food manufacturing is probably responsible for both the increased sodium and reduced potassium content of food products. There is now sufficient scientific evidence to support an increase in potassium intake to reach a urinary potassium excretion between 90 and 120 mmol/day in patients with essential hypertension and preserved renal function (eGFR > 60 mL/min/1.73 m²) in order to help lower their BP. There is also rather good evidence that a high-potassium diet decreases the incidence of stroke and CVDs, although for these latter there is no level A evidence from trials at the moment. Regarding patients with impaired renal function, there is a definite need for new randomized prospective trials in all CKD stages in order to determine the potential benefits and risks of increasing potassium in the diet.

In clinical practice, these conclusions could be translated as a change in the conventional lifestyle recommendations given to patients with hypertension or cardiovascular or renal disease. Indeed, physicians could give a more positive recommendation for better nutrition, encouraging the consumption of more healthy products with a high potassium content rather than the semiperal message 'Don’t eat salt!'. As illustrated in Figure 5, the recommendations on salt could thus be tailored to the urinary potassium excretion or to the Na:K ratio in urine, recommending primarily an increase in potassium intake with fruits, vegetables and nuts in patients with a moderate excess of salt intake or a combined reduction of sodium and increase in potassium intake in those with excessive salt consumption. In any case, the definite answer to the title of this article is yes, one should eat more potassium to lower BP and prevent cardiovascular events.

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**CONFLICT OF INTEREST STATEMENT**

None declared.


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