Title

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Journal

Journal of Renal Nutrition, 32(6)

ISSN

1051-2276

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Publication Date

2022-11-01

DOI

10.1053/j.jrn.2022.01.013

REVIEW ARTICLE

Taking the Kale out of Hyperkalemia: Plant Foods and Serum Potassium in Patients With Kidney Disease

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Traditionally, diets for kidney disease were low in potassium. This recommendation was based on outdated research and often wrong assumptions that do not reflect current evidence. In fact, studies conducted over the past decades show patients with CKD, including kidney failure, do not benefit from the restriction of plant foods relative to control. Generally, dietary potassium does not correlate with serum potassium, and we posit that this is due to the effects of fiber on colonic potassium absorption, the alkalinizing effect of fruits and vegetables on metabolic acidosis, and the bioavailability of dietary potassium in plant foods. Also, consumption of plant foods may provide pleiotropic benefits to patients with CKD. Emerging dietary recommendations for kidney health should be devoid of dietary potassium restrictions from plant foods so that patient-centered kidney recipes can be encouraged and promoted.

Keywords: potassium; renal insufficiency; diet; vegan; metabolic acidosis; nutrition policy

Introduction

HYPERKALEMIA IS A common electrolyte abnormality often defined by a serum potassium (sK) > 5 mEq/L, but definitions vary and can include cutoffs as high as 5.5 mEq/L. As the most abundant cation in the body, potassium plays a vital role in many systems via modulation of the electric potential of the cell and cell membrane. Although the real incidence and prevalence of hyperkalemia is unknown, it is estimated to be 2-3% in the general population and 1-10% of hospitalized patients.[1-5] This increases drastically in patients with chronic kidney disease (CKD) and kidney failure with estimates ranging from 12-54%.[2]

Although it does not frequently appear in patients with normal kidney function, hyperkalemia is common in those with kidney disease and has several causes. Low glomerular filtration rate (GFR) is the main predictor for hyperkalemia, while other common risk factors include CKD, diabetes mellitus (DM), metabolic acidosis, heart failure, as well as the use of renin-angiotensin-aldosterone system inhibitors.[4-7]

While hyperkalemia is associated with fatigue and weakness, it is of more immediate concern due to its potential for causing fatal arrhythmias.[4-6,8,9] In a meta-analysis of 151,153 all-cause deaths across 26 cohorts, elevated sK > 5.5 mEq/L was associated with a 22% increased risk of mortality compared to baseline.[8] These findings were consistent in the meta-analysis' individual cohorts, as well as for cardiovascular and kidney failure mortality.[8] Plant foods, which contain potassium, are often thought to raise sK and are frequently restricted in patients with kidney disease.[10]

The guidelines created by Kidney Disease Improving Global Outcomes (KDIGO) in 2012 made no recommendations about broader dietary patterns in management of CKD regarding potassium-rich foods, instead targeting specific changes like promoting the avoidance of salt substitutes.[11] Indeed, the 2020 KDIGO Diabetes in CKD Guideline makes similar recommendations instructing clinicians to control high potassium levels by "[moderating] potassium intake, with specific counseling to avoid potassium-containing salt substitute or food products containing the salt substitute;" further, the guidelines recommend that "in advanced CKD, potassium may need to be restricted, and people may be advised to eat lower potassium fruits and vegetables, and to limit nuts."[12] Other guidelines in North America, including provincial and national Canadian bodies made similar claims, while the National Kidney Foundation's (NKF) guidelines recommend restricting specific potassium-rich foods in management

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Financial Disclosure: Dr. Joshi has received consulting fees from Vifor Pharma, Otsuka, and Insyght Interactive.

Support: This research did not receive any funding from agencies in the public, commercial, or not-for-profit sectors.

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1051-2276/$36.00

https://doi.org/10.1053/j.jrn.2022.01.013

Table 1. Incidence of Hyperkalemia in Patients With Non-dialysis CKD After Plant Food Interventions

Study Name	Dietary Pattern	Participants	Type	Duration	Hyperkalemia	CKD Stage
Goraya et al. (2012)[16]	2 to 4 cups fruits & vegetables daily	199	Controlled trial	30 days	No	I/II
Goraya et al. (2014)[17]	2 to 4 cups fruits & vegetables daily	108	RCT	3 years	No	III
Tyson et al. (2016)[18]	DASH diet	10	Controlled trial	2 weeks	No	III
Moorthi et al. (2014)[19]	70% plant protein	13	Controlled trial	4 weeks	Yes*	III/IV
Barsotti et al. (1996)[20]	Vegetarian diet	37	Controlled trial	3 months	No†	III-V
Goraya et al. (2013)[21]	2 to 4 cups fruits & vegetables daily	76	RCT	1 year	No	IV

CKD, chronic kidney disease; DASH, Dietary Approaches to Stop Hypertension; RCT, randomized controlled trial.
*1 patient with pre-existing type IV RTA.
†14 months of data on potassium not reported.

of hyperkalemia based on research from over 30 years ago.[10,13] However, the recently released NKF Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines mention the lack of evidence on the subject, noting that "there is a scarcity of studies on … how modifying diet can influence sK levels in patients with CKD."[14] Also, guidance from KDIGO discusses the paucity of evidence and widespread opinion-based guidelines on low-potassium diets for preventing hyperkalemia in patients with CKD and kidney failure, reporting that although there are associations between dietary potassium and sK, these associations are weak.[15] We seek to show through a review of the literature that the opinion-based concerns of potassium-rich plant-based dietary patterns and sK do not match the evidence, and that these broad dietary changes may have more benefits than harms for many patients with CKD and kidney failure.

Hyperkalemia in Plant-Based Diets in CKD

There have been numerous studies, including controlled trials, of plant foods for patients with nondialysis CKD since1996. Table 1 summarizes these data, and shows that generally, patients did not exhibit greater sK despite eating patterns with increased dietary potassium and across stages I-V of CKD.[16-21] The earliest trial, conducted in Italy, showed that patients adhering to a "special vegan diet" (a plant-based diet focused on the now outdated complementary protein theory for obtaining essential amino acids) did not have significant increases in sK, and had better palatability than the conventional, low-protein diet.[20,22] Goraya et al. have shown in several controlled trials that an intervention of approximately 2 to 4 cups of fruits and vegetables did not significantly change subjects' sK.[16,17,21] This study included fruits such as apples and oranges and vegetables such as tomatoes and carrots, many of which have been traditionally discouraged for patients with CKD.[10,11,13,15-17,21] It is important to note, however, that in the trials by Goraya et al., patients with sK $>$ 4.6 mEq/L were excluded citing the fact that participation of individuals with sK $<$ 4.6 mEq/L carried low-risk of developing sK $>$ 5.0 mEq/L.[17] In a study by Tyson et al., researchers similarly did not observe incident hyperkalemia in patients following the Dietary Approaches to Stop Hypertension (DASH) diet, although this pattern included low-fat dairy, lean meats, fish, poultry, in addition to whole plant foods.[18] Moorthi et al. took a different approach with their intervention, giving patients a diet containing 70% plant protein; in this trial, they had two incidences of sK at 5.8 mEq/L in the same subject with a pre-existing type IV renal tubular acidosis, which can cause hyperkalemia.[19] The hyperkalemia resolved by replacing "raw edamame," which has one of the highest potassium densities, with fried tofu, which has less potassium than the raw edamame.[19] Aside from this individual, there was no significant change in sK among 12 other participants after two and 4 weeks from baseline.[19]

Hyperkalemia in Plant-Based Diets in Kidney Failure

As hyperkalemia accounts for 24% of emergency dialysis cases and is prevalent in 19% of patients on hemodialysis (HD), there is an additional concern for preventing elevated sK in patients with kidney failure.[23-25] Plant foods are often restricted or omitted from the diets of those with kidney failure owing to their elevated potassium concentration; however, there is little to no evidence for the development of hyperkalemia after the consumption of plant foods in those on HD.[24,26]

As shown in Table 2, there are several observational and prospective studies demonstrating that patients with kidney failure on HD do not exhibit hyperkalemia with increased dietary potassium from plant foods.[7,24,27-30] In the 2011 cross-sectional study by Wu et al., 19 vegetarian participants with HD did not have significantly different sK than the nonvegetarian HD cohort.[27] In a 2019 study of 8,078 participants with kidney failure on HD, the participants were grouped into tertiles based on low, medium, and high fruit and vegetable consumption; however, there was not a significant difference in predialysis sK between the cohorts.[28] Last, a prospective study by González-Ortiz et al. showed a similar outcome when tracking the dietary patterns of 150 patients undergoing HD over 1 year.[29] This study grouped patients into tertiles based on their adherence to a "healthy plant-based diet" (HPD) and collected sK values at each

Table 2. Incidence of Hyperkalemia in Patients With Kidney Failure on Hemodialysis With Increasing Plant Food

Study Name	Dietary Pattern	Participants	Type	Duration	Hyperkalemia	CKD Stage
Wu et al. (2011)[27]	Vegetarian diet	19	Cross-sectional	6 months	No	HD
Saglimbene et al. (2019)[28]	Median 8 servings of fruits & vegetables per week	8078	Prospective observational	2.7 years	No	HD
González Ortiz et al. (2021)[29]	Plant-based diet score	150	Prospective	1 year	No	HD

CKD, chronic kidney disease; HD, hemodialysis.

Table 3. Incidence of Hyperkalemia in Patients With Kidney Failure on Hemodialysis With Increasing Potassium Intake

Study Name	Dietary Pattern	Participants	Type	Duration	Hyperkalemia	CKD Stage
Ramos et al. (2020)[7]	Increasing potassium intake	117	Cross-sectional	—	No	HD
St-Jules et al. (2016)[24]	Increasing potassium intake	140	Cross-sectional	16 weeks	No*	HD
Bernier-Jean et al. (2021)[31]	Increasing potassium intake	8043	Prospective	4 years	No	HD
Noori et al. (2010)[30]	Increasing potassium intake	224	Prospective	5 years	Yes†	HD

CKD, chronic kidney disease; HD, hemodialysis.
*No correlation between serum and dietary potassium.
†Weakly correlated with r = 0.14.

patient visit; researchers found no statistically significant relationship between low, medium, and high HPD adherence to sK or to hyperkalemia.[29]

Similar findings have been shown for dietary potassium (whether from plants or not) and sK in patients on dialysis as shown in Table 3. Ramos et al. performed a cross-sectional study of 117 patients on dialysis and found no association between sK and dietary potassium.[7] St-Jules et al. investigated patients in the cross-sectional BalanceWise study and found that there were no significant relationships between dietary potassium intake or potassium density with sK across 140 participants on HD, which remained nonsignificant when accounting for age, gender, race, and body mass.[24] Furthermore, a recent study by Bernier-Jean et al. came to similar conclusions finding no significant association between potassium intake and sK levels in a group of 8043 participants.[31] Noori et al. did find a weak correlation with dietary potassium and sK in a prospective study spanning 5 years, including 224 patients on dialysis.[30] Although this study showed an association with dietary potassium and sK, the association was small (r = 0.14). As dietary potassium increased from 500 mg/day to 4,500 mg/day (a 9-fold increase), sK was only about 0.4 mEq/L higher.[30] The authors also note that residual renal function (RRF) may affect the predialysis potassium level for patients on HD, especially if the RRF is large. However, RRF tends to be minimal in this patient population. As such, renal excretion of potassium in kidney failure is negligible, and extrarenal potassium disposal plays a bigger role in protecting against hyperkalemia for most patients on dialysis.[30,32,33]

Unlike for CKD, there is a paucity of randomized control trials in patients on HD regarding dietary interventions with plant foods and sK.[26] Thus, providers should remain vigilant about dietary management in patients on HD. Still, the evidence that does exist indicates that patients across multiple studies are not at increased risk for hyperkalemia when eating plant-based diets.[24,27–29]

Reconciling the Evidence

There is a lack of documented hyperkalemia with plant-based foods in studies of patients with CKD/kidney failure. Recent research indicates that dietary potassium is not strongly associated with sK or hyperkalemia in patients with nondialysis dependent CKD and on HD, accounting for ~2% of sK variation.[7] This contrasts with prior research that showed increased sK after nondietary potassium supplementation via mineral salt, Slow-K, potassium chloride elixir, etc. in controlled settings of healthy individuals and those with CKD, although these supplements had potassium in levels above typical dietary recommendations.[7,34–37] Moreover, although fruit, bean, and overall fiber consumption are associated with increased dietary potassium intake, a dietary pattern high in these foods has not been associated with hyperkalemia.[7] This suggests that dietary potassium has different effects in the body compared to nondietary potassium.

There are likely multiple factors that mitigate the impact of dietary potassium in plant foods on sK, including fiber, the alkalinizing effect of fruits and vegetables, and the bioavailability of potassium in plant foods.[7,10,21]

The Gut Implications of Potassium Homeostasis

Dietary fiber has been shown to provide a variety of positive cardiovascular and gastrointestinal health benefits and is implicated in increased bowel movement frequency and bulk as consumption increases.[38–41] Constipation, a symptom often associated with lack of fiber, is common in patients with kidney disease; in patients on HD, the

Table 4. Rectal Potassium Excretion in Kidney Failure

Study Name	Group	Dietary Potassium (mEq/day)	Rectal Potassium (mEq/day)	% Excreted	% Difference vs Control
Kopple & Coburn (1973)[51]	Low Protein (20 g/day)	32.4	10.1	31.2%	—
	High Protein (40 g/day)	41.1	11.8	28.7%	—
Blumenkrantz et al. (1982)[52]	Low Protein (0.98 g/kg body wt/day)	64.0	16.9	26.4%	—
	High Protein (1.44 g/kg body wt/day)	84.1	20.4	24.3%	—
Martin et al. (1986)[46]	Control	—	2.9*	—	—
	Chronic Renal Failure	—	7.5*	—	160%

*Converted from μEq/min to mEq/day.

incidence of constipation varies according to the definition of constipation, but it was noted to be as high as 71.7% in a 2013 cross-sectional study using the Roma III criteria.[42] Aside from these outcomes, fiber itself is implicated in increased fecal potassium, with one trial showing that consumption 3.5 g of psyllium fiber twice a day for 11 week led to a 32% increase in excretion; however, this study had a sample size of only one subject.[43] In a study with 6 participants, for every 1 g of additional fiber in the diet from whole grains, there was an increase in fecal potassium output of approximately 25 mg.[44] Also, increased fiber is correlated with reduced fecal transit time; as faster intestinal transit reduces potassium absorption, regular bowel movements due to fiber may inhibit increased sK.[45] Thus, consumption of plant foods, although high in potassium, may not lead to hyperkalemia because of the increased elimination of potassium via increased fecal bulk and bowel movement frequency due to their fiber content.

It is theorized that colonic secretion of potassium in patients with kidney failure increases compared to those with normal kidney function.[46,47] Research by Hayes et al. in the 1960s indicated that patients on dialysis could have a three-fold rate of fecal excretion of potassium at 37% of K balance versus 12% in controls.[24,48-50] Table 4 highlight studies since Hayes et al. that show elevated rectal excretion of potassium in kidney failure. In a study done in 1973, patients with CKD stage 5 grouped in lower and higher protein intakes had a fecal potassium excretion of 10.1 and 11.8 mEq/day with a dietary potassium intake of 32.4 and 41.1 mEq/day, yielding fecal potassium excretion to dietary potassium rations of 31.2 and 28.7%, respectively.[51] A 1982 study grouped patients on continuous ambulatory peritoneal dialysis (CAPD) similarly; the lower and higher protein intakes had a fecal potassium excretion of 16.9 mEq/day and 20.4 mEq/day with a dietary potassium intake of 64 mEq/day and 84.1 mEq/day, yielding fecal potassium excretion to dietary potassium rations of 26.4% and 24.3%, respectively.[52] Although neither of these studies included a control group, the 1973 and 1982 studies cited control values from Dempsey et al. remarking that "the mean daily fecal potassium of 10.9 mEq/day was significantly greater than the value of 7.2 mEq/day reported in nonuremic patients eating diets higher in potassium content" and "fecal potassium excretion was 19 mEq/day or 26% of dietary intake, a percentage greater than the reported value of 15% in individuals with normal renal function."[51-53] Regardless, it is likely that the fecal potassium excretion of dietary potassium ratios in the 1982 study are lower than those of the 1973 study as patients on CAPD lose a significant amount of potassium in their dialysate.[54] Another study in 1986 found similar results, showing that "the fraction of dietary K excreted in feces was increased in subjects with a low filtration rate."[46,48] While the measured rectal potassium values are lower in this 1986 study than the other studies found on Table 4, the control group yielded an average fecal potassium excretion of 2.9 mEq/day whereas the chronic renal failure group yielded an average fecal potassium excretion of 7.5 mEq/day, a 160% increase in potassium excretion.[46]

Other studies reviewed similar phenomena instead of focusing on rectal potassium flux; these studies are outlined in Table 5. Sandle et al. published two studies in 1986 and 1987 tracking rectal potassium secretion of patients with uremia.[55,56] The 1986 study compared the rectal potassium excretion measured as rectal potassium flux between control patients and patients with renal insufficiency, yielding potassium excretion of 1.5 μmol/cm^{2}h and 3.2 μmol/cm^{2}h, respectively; this is an increase in rectal potassium secretion of 167% of the study group versus the control.[55] The 1987 study gathered similar data across four groups: control, patients on CAPD, and patients on HD before and after dialysis.[56] The control, CAPD, HD: Predialysis, and HD: Postdialysis groups had potassium excretion rates of 1.2 μmol/cm^{2}h, 2.4 μmol/cm^{2}h, 3.7 μmol/cm^{2}h, and 2.4 μmol/cm^{2}h, respectively; this is an increase in rectal potassium secretion of 100% for the CAPD group, 208% for the HD: Predialysis group, and 100% for the HD: Postdialysis group when compared to the control.[56] A more recent study in 2005 further supports these findings, as participants with kidney failure, half of whom were on HD, had rectal potassium secretion nearly of 2.21 μmol/cm^{2}h compared to the control group's 0.87 μmol/cm^{2}h, an increase of 154%.[47] The authors posited that colonic potassium excretion appears to be an active process mediated by an

Table 5. Rectal Potassium Flux in Kidney Failure

Study Name	Group	Dietary Potassium (mEq/day)	Serum Potassium (mmol/L)	Rectal potassium flux (μmol/cm^{2}h)	% Flux Difference vs Control
Sandle et al. (1986)[55]	Control	—	4.1	1.5	—
	Renal insufficiency —		5.3	3.2	167%
Sandle et al. (1987)[56]	Control	80-100	4.1	1.2	—
	CAPD	70-80	4.3	2.4	100%
	HD: Predialysis	60-70	5.3	3.7	208%
	HD: Postdialysis	60-70	3.5	2.4	100%
Mathialahan et al. (2005)[47]	Control	—	4.6	0.87	—
	ESRD	—	4.7	2.21	154%

CAPD, chronic ambulatory peritoneal dialysis; HD, hemodialysis; ESRD, end-stage renal disease.

upregulation of high-conductance (BK) apical potassium channels in surface colonic epithelial cells.[47]

Nevertheless, these values are higher than the 10-12% seen in those without advanced kidney disease, and may play a role in maintaining sK balance in patients with diminished kidney function.[24,48,49] A case study confirms the necessary role that colonic potassium secretion plays in those with kidney disease; following a 56-year-old patient with kidney failure on HD, the report tracked sK values before an ileocecal diversion and temporary ileostomy, while the diversion was in place, and after the stoma reversal.[57] While the patient had elevated sK between 4.9 and 6.1mEq/L before the procedure, her postoperative predialysis sK was higher with measurements between 6.1 and 8.3 mEq/L.[57] Following bowel continuity restoration after the stoma was reversed, the patient's sK returned to previous values without changes to diet, medication, or dialysis.[57] By measuring fecal potassium while the intervention was in place and after, researchers found that the changes to her sK were strongly correlated to the ability to excrete potassium via the bowels, with values before the reversal of 23.4 and 23.1 mEq/L versus 49.6 and 60 mEq/L after its removal.[57] They posited that the worsened hyperkalemia in this patient was caused by reduced colonic potassium secretion owing to the ileostomy.[57] These data support constipation management as a method for hyperkalemia management in those with CKD.[58]

Metabolic Acidosis and Alkalinizing Foods

Researchers have previously identified metabolic acidosis as a prominent risk factor for hyperkalemia in patients with kidney disease.[7] As recommended by KDOQI guidelines, patients with CKD who have metabolic acidosis should be given alkali, often taken in the form of pharmacologic sodium citrate or sodium bicarbonate, to correct the issue.[7,14] As discussed previously, Goraya et al. have shown that fruit and vegetable intake acts similarly to pharmacologic alkali, reducing urinary acid output and increasing serum bicarbonate.[16,17,21,59,60] Unlike pharmacologic alkali, usually in the form of sodium bicarbonate or sodium citrate, fruit and vegetable intake does not contribute to hypervolemia and/or hypertension from added sodium and does not contribute to pill burden.[17] Because amelioration of metabolic acidosis can lower the sK levels in patients with kidney disease, fruits and vegetables, along with other plant foods, may temper the potential rise in sK from these foods' potassium content through this mechanism and others.

Potassium Bioavailability

Although plant foods contain potassium in relatively high amounts, the potassium in plant foods may be less bioavailable, which may be another reason why the ingestion of plant foods do not exhibit a reflexive rise in sK in patients with kidney disease.[10,61] Potassium within the cells of plants may be difficult to digest and may explain the reduced bioavailability of potassium from plants when they are consumed in an unprocessed form.[10,61-63] In a 10-day crossover feeding trial of 11 volunteers, participants were fed a "high bioaccessible diet" and a "low bioaccessible diet" of potassium-containing foods; bioavailability of potassium in the "high bioaccessible diet" (including animal foods and fruit juices) was 96% whereas the "low bioaccessible diet" (consisting of minimally processed fresh fruits and vegetables) had a potassium bioavailability of 77%.[63] However, only 60% of the potassium in the "low bioaccessible diet" was derived from minimally processed fresh fruits and vegetables. The remaining 40% of potassium came from foods that would have led the potassium to be "almost completely absorbed." As such, the authors ultimately "concluded that the bioaccessibility of potassium in unprocessed plant foods could have been little more than 60%."

Three other studies on the bioavailability of potassium in DASH diets on participants with and without CKD have shown similar results in regards to bioavailability.[18,64,65] In a 1997 trial, patients within the control (Standard American Diet) group had a potassium bioavailability of 87%. However, those in the fruit and vegetable diet and DASH diet groups were found to have potassium bioavailabilities of 67%, and 66%, respectively.[64] The follow-up study from

the DASH Collaborative Research Group in 2001 organized the participants differently, but had similar results: the control (Standard American Diet) group had bioavailability of potassium at 94% while the three DASH diet groups had bioavailabilities of 62%, 68%, and 68%.[65] Until 2016, there had not been research conducted on DASH diets in CKD when Tyson et al. found similar results between the CKD and non-CKD cohorts, both exhibiting a bioavailability of 61%.[18] However, there is evidence that foods included in the 10 day crossover feeding trial may have greater bioavailability; a randomized controlled trial on bioavailability of potassium in potatoes found a bioavailability of greater than 94%.[66] While the research is not in complete alignment, generally, unprocessed plant foods may have an absorption rate around 65% whereas animal foods and K additives, supplements, and mineral salts have potassium bioavailabilities of 70 to 100%.[10,63,67] Future randomized controlled trials on patients with CKD should be conducted to further clarify this area.

As potassium exchangers increase potassium excretion in the stool, they are indicated for the treatment of chronic hyperkalemia.[68] Newer potassium exchangers, like patiromer and sodium zirconium cyclosilicate, allow patients with hyperkalemia or risk factors for hyperkalemia to adopt "a more liberal and healthy diet" with a focus on plant foods.[69] Although it is not necessary for all patients consuming plant-based diets to be on a potassium exchanger, they may be used judiciously in those who need it while balancing their risks and costs.

Salutatory Effects of Consuming Potassium-Rich Plant Foods

Adherence to low potassium "traditional renal diet" in patients with CKD and kidney failure may carry an opportunity cost as plant-centered diets are associated with several health benefits, including improvements to blood pressure (BP), bone health, and decreased risk for coronary artery disease and cardiovascular disease.[70,71] The "traditional renal diet" also conflicts with the American Heart Association's dietary recommendations for maintaining cardiac health, creating confusion for patients and healthcare providers alike.[70]

Fortunately, the evidence shows that plant-food consumption is associated with a reduced progression of CKD and kidney failure, with both prospective and cross-sectional studies showing a reduction in risk of CKD, lowered risk of estimated GFR decline, lowered urine albumin-to-creatinine ratio, and a lowered prevalence ratio for the association of CKD with kidney function impairment.[72-77]

Plant foods are associated with a reduction in mortality in both CKD and kidney failure. In a 2017 meta-analysis by Kelly et al. of seven studies following 15,285 subjects with CKD, healthy dietary patterns high in plant foods and low in animal and processed foods were associated with a 27% lower risk of mortality.[78] Another study by Saglimbene et al. found that patients with kidney failure who consumed at least four servings of fruit and vegetables had lower all-cause mortality and noncardiovascular death over a median follow-up period of 2.7 years.[28,78]

Also, numerous studies show the pleiotropic benefits of plant foods in kidney disease. As plant foods have lower phosphorus bioavailability than animal foods, plant-based diets may help better control hyperphosphatemia in those with CKD.[71] As discussed prior, diets with higher plant food content are better at mitigating metabolic acidosis than those without, or diets with supplementary bicarbonate.[21,60,79] Goraya and Wesson's 2013 interventional study on metabolic acidosis and plant foods for those with CKD also found significant reductions in weight and BP in the group given fruits and vegetables but not in those given bicarbonate.[21,79] Last, diets rich in plant foods are implicated in the reduction of comorbidities for those with kidney disease, including the prevalence of diabetes mellitus and the incidence of cardiovascular events.[79]

However, there is one study to suggest otherwise. A 2010 longitudinal study on dietary potassium and mortality in 224 patients on HD showed that increased dietary potassium was associated with increased mortality.[30] However, the authors expressed the need for randomized control trials to confirm the findings due to various confounding factors, such as the inaccuracy of Food Frequency Questionnaire data in assessing dietary potassium; additionally, the ten most common sources of dietary potassium in the study came from beef, chicken, Mexican food, hamburgers, legumes, fresh fruits, real fruit juices, fried potatoes, cheeseburgers, and canned fruits, most of which are not recommended in any healthy, plant-rich dietary pattern.[30]

Thus, the traditional renal diet may be preventing patients from receiving the benefits of plant foods and actively inhibiting the improvement of their associated comorbidities.

Balancing Potassium Restriction Versus Omitting Healthy Foods

While additional concern has been given and is necessary for those with kidney disease, there is evidence that dietary sources of potassium, which affect CKD and kidney failure patients (and have been restricted previously), may be dangerous for those without CKD; a case review by te Dorsthorst et al. in 2018 showed numerous hyperkalemic patients with and without CKD following consumption of potassium-rich foods.[80] However, these sources were, in most instances, mildly to heavily processed plant foods like dried fruit, juices, and sauces.[80]

While cases such as those evaluated in te Dorsthorst's review show that hyperkalemia is possible even in those without CKD, they also show that it is typically processed

plant foods and potassium-rich salt supplements that raise sK to dangerous levels.[80]

Conclusion

Hyperkalemia is a common electrolyte abnormality in those with kidney disease. Because of the dangers associated with this condition, previous consensus and guidelines have cautioned the addition of potassium-rich plant foods in the diets of those with CKD and kidney failure.[11,14] Based on the evidence from numerous studies of patients with CKD and kidney failure, we believe that the correlation between dietary potassium and sK is small to nonexistent, and that whole, unprocessed plant foods should not be restricted to the extent that they have been in the "renal diet."[16,17,28] These foods likely possess factors that mitigate a potential rise in sK like their high fiber content, alkalinizing potential, and reduced bioavailability of potassium.[10,21,48] Last, there are many well documented benefits of plant-based diets and the pleiotropic benefits of plant foods that reduce mortality, symptoms, and comorbidities in CKD and kidney failure patients.[28,70,78] Indeed, there are dietary interventions such as the patient-centered plant-dominant low-protein (PLADO) diet and other plant-centered diets that may improve CKD outcomes and prevent or delay renal replacement therapy.[79,81-84] We suggest a reconsideration of the previous low potassium guideline that restricts some fruits and vegetables since evidence for this is weak. The current evidence of the benefits of a healthy dietary pattern that centers on plant foods should be cautiously encouraged.

Practical Application

Traditionally, patients were counseled to avoid consuming potassium-rich plant foods to prevent hyperkalemia. Current research does not reflect a correlation between dietary and serum potassium. Plant-based foods do not appear to increase serum potassium, and these foods have added health benefits, which include reduced mortality and symptoms. The lack of hyperkalemia from plant-based foods may be due to their high fiber content, alkalinizing potential, and reduced bioavailability of potassium. Patients with kidney disease should be educated on the benefits of a healthy diet pattern that includes fruits, vegetables, and other plant foods.

Acknowledgments

The authors would like to thank Dr. Michelle McMacken for connecting them and for her invaluable guidance and mentorship.

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