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Authors

Moore, Linda
Bross, Rachelle
Cervantes, Mackenzie
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Dietary potassium intake, kidney function, and survival in a nationally representative cohort

Yoko Narasaki,[1,2] Amy S You,[1,2] Shaista Malik,[3] Linda W Moore,[4] Rachelle Bross,[5] Mackenzie K Cervantes,[1,5] Andrea Daza,[1,2] Csaba P Kovesdy,[6,7] Danh V Nguyen,[8] Kamyar Kalantar-Zadeh,[1,2] and Connie M Rhee[1,2]

[1]Harold Simmons Center for Chronic Disease Research and Epidemiology, Division of Nephrology, Hypertension, and Kidney Transplantation, University of California Irvine, Orange, CA, USA; [2]Tibor Rubin Veterans Affairs Long Beach Health Center, Long Beach, CA, USA; [3]Division of Cardiology, University of California Irvine, Orange, CA, USA; [4]Department of Surgery and Transplantation, Houston Methodist Hospital, Houston, TX, USA; [5]Lundquist Institute, Harbor-UCLA Medical Center, Torrance, CA, USA; [6]Division of Nephrology, University of Tennessee Health Science Center, Memphis, TN, USA; [7]Nephrology Section, Memphis Veterans Affairs Medical Center, Memphis, TN, USA; and [8]Division of General Internal Medicine, University of California Irvine, Orange, CA, USA

ABSTRACT

Background: In healthy adults, higher dietary potassium intake is recommended given that potassium-rich foods are major sources of micronutrients, antioxidants, and fiber. Yet among patients with advanced kidney dysfunction, guidelines recommend dietary potassium restriction given concerns about hyperkalemia leading to malignant arrhythmias and mortality.

Objectives: Given sparse data informing these recommendations, we examined associations of dietary potassium intake with mortality in a nationally representative cohort of adults from the NHANES.

Methods: We examined associations between daily dietary potassium intake scaled to energy intake (mg/1000 kcal), ascertained by 24-h dietary recall, and all-cause mortality among 37,893 continuous NHANES (1999–2014) participants stratified according to impaired and normal kidney function (estimated glomerular filtration rates <60 and ≥ 60 mL $\cdot$ min^{-1} $\cdot$ 1.73 m^{-2}, respectively) using multivariable Cox models. We also examined the impact of the interplay between dietary potassium, source of potassium intake (animal- compared with plant-based sources), and coexisting macronutrient and mineral consumption upon mortality.

Results: Among participants with impaired and normal kidney function, the lowest tertile of dietary potassium scaled to energy intake was associated with higher mortality (ref: highest tertile) [adjusted HR (aHR): 1.18; 95% CI: 1.02, 1.38 and aHR: 1.17; 95% CI: 1.06, 1.28, respectively]. Compared with high potassium intake from plant-dominant sources, participants with low potassium intake from animal-dominant sources had higher mortality irrespective of kidney function. Among participants with impaired kidney function, pairings of low potassium intake with high protein, low fiber, or high phosphorus consumption were each associated with higher death risk.

Conclusions: Lower dietary potassium scaled to energy intake was associated with higher mortality, irrespective of kidney function. There was also a synergistic relation of higher potassium intake, plant-based sources, and macronutrient/mineral consumption with survival. Further studies are needed to elucidate pathways linking potassium intake and coexisting dietary factors with survival in populations with and without chronic kidney disease. *Am J Clin Nutr* 2022;116:1123–1134.

Keywords: dietary potassium, chronic kidney disease, kidney function, mortality, plant-dominant

Introduction

Given that potassium is an essential nutrient needed for maintenance of normal cellular function (muscle contraction and nerve conduction), inadequate dietary potassium intake may heighten the risk of disease (1). In healthy adults, a relatively high potassium intake of 2600 mg/d for females and 3400 mg/d for males is recommended to prevent hypertension

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The authors report no conflicts of interest.

Supplemental Figure 1 and Supplemental Tables 1–8 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/ajcn/.

Address correspondence to CMR (e-mail: crhee1@uci.edu).

Abbreviations used: aHR, adjusted hazard ratio; AMPM, Automated Multiple-Pass Method; CKD, chronic kidney disease; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; NCHS, National Center for Health Statistics; NSAID, nonsteroidal anti-inflammatory drug; PURE, Prospective Urban Rural Epidemiology; RAAS, renin–angiotensin–aldosterone system.

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and cardiovascular disease (2). However, epidemiologic data have consistently shown that US adults consume less dietary potassium than the daily recommended intake, which has led to inadequate dietary potassium intake being a major public health concern (3).

Potassium is found in a wide variety of plant and animal foods, as well as in beverages. Fruits, vegetables, legumes, and nuts are rich in potassium, and are also a key source of micronutrients (antioxidant vitamins) and dietary fiber needed to process and absorb nutrients (fiber viscosity inhibits glucose and cholesterol absorption and traps bile acids in the intestine) (4). Population-based data indicate that, in addition to plant-based foods such as fruits/vegetables and grain-based dishes, animal-based foods including milk products and meats/poultry are the second and third highest contributors to dietary potassium intake in US adults (5). Notably, food processing reduces foods' potassium content, and a diet high in processed foods and low in fresh fruits and vegetables is often lacking in natural sources of potassium (6). It bears mention that although ultra-processed foods may contain potassium-rich additives, they have been associated with faster kidney function decline (7). Although a large body of evidence has shown that dietary factors influence clinical outcomes (8–10), there has been limited examination of the impact of *1*) source of dietary potassium intake and *2*) the interplay between potassium and other nutrient (fiber) consumption upon health and survival.

In contrast to healthy adults, dietary potassium restriction has been a mainstay in the management of patients with chronic kidney disease (CKD) (11). The kidneys, and to a lesser degree the colon, are responsible for regulating potassium homeostasis and its excretion in response to changes in dietary potassium intake (12), with a rise in the proportion of gastrointestinal excretion as kidney function declines (13). In patients with normal kidney function, potassium excretion rapidly increases after potassium consumption unless body stores are depleted (11). However, in CKD patients, there has been ongoing concern that high dietary potassium intake may lead to hyperkalemia and its life-threatening complications (malignant arrhythmia, sudden cardiac death) (12, 14), although evidence showing a direct relation between dietary and serum potassium remains unclear (15). Consequently, advanced CKD patients, including those receiving dialysis, are typically advised to limit dietary potassium intake despite the lack of evidence to support these recommendations.

Thus, to address these knowledge gaps, we aimed to examine the association of dietary potassium intake with all-cause mortality risk in a nationally representative cohort of US adults stratified by kidney function. In addition, we evaluated the impact of the interrelations of dietary potassium, sources of potassium intake, and other concurrent nutrient consumption upon survival in this population.

Methods

Source population

We conducted a retrospective cohort study using data from the National Health and Nutrition Examination Survey (NHANES), a survey conducted by the National Center for Health Statistics (NCHS) over the period of 1999–2014 to provide national estimates of the health and nutritional status of US children and adults. The survey used a complex, multistage, stratified, clustered sampling design in which children, elderly, Black, Mexican-American, and low-income White American participants were oversampled to enable precise estimation within these subgroups (16, 17). Study participants underwent standardized personal interviews followed by physical examination and laboratory tests in a mobile examination center (MEC) administered by highly trained medical personnel to collect information on socio-demographics, dietary intake, and health-related questions; medical, dental, and physiologic measurements; and laboratory data.

In the present study, participants were included if they *1*) were ≥18 y of age at study entry (i.e., date of dietary assessment via 24-h dietary recall) and had *2*) reliable 24-h dietary recall data, *3*) follow-up data on mortality status, *4*) serum creatinine data, and *5*) body weight and height data. Participants were excluded if they *1*) had missing data on mortality status (i.e., owing to lacking data needed for linkage to the National Death Index), *2*) were pregnant, or *3*) had an outlier estimated glomerular filtration rate (eGFR) value, defined as an eGFR >170 mL · min^{-1} · 1.73 m^{-2}, as calculated using the CKD Epidemiology Collaboration equation (**Supplemental Figure 1**) (18).

Exposure ascertainment

Our primary exposure of interest was daily dietary potassium intake using 24-h dietary recall. In primary analyses, we examined daily dietary potassium intake scaled to total energy intake (mg/1000 kcal), categorized as tertiles, using the Nutrient Density Model approach to account for differences in caloric intake (19, 20). To estimate the average relative causal effect of the association of dietary potassium intake with mortality risk, we also conducted sensitivity analyses in which we used the *1*) "Residual Model" approach, which indirectly adjusted for total energy intake by using a residual; and the *2*) "Standard Model" approach, which adjusted for total energy intake (19, 20).

Dietary potassium intake was ascertained by trained interviewers using 24-h dietary recall based on the USDA's Automated Multiple-Pass Method (AMPM), a 5-step multiple-pass process to enhance complete and accurate data collection. Reliable dietary recall data were defined as those in which *1*) the first 4 steps of the AMPM were completed and *2*) foods consumed for each reported meal were identified.

In secondary analyses, we sought to examine *1*) the interplay between dietary potassium and the source of potassium intake (animal- compared with plant-based sources), *2*) the interplay between dietary potassium and specific macronutrients (dietary fiber, protein intake), and *3*) the interplay between dietary potassium and specific minerals (dietary phosphorus, sodium intake) using 2-by-2 exposure groups, in which dietary potassium was categorized as low or high intake [lowest tertile and highest 2 tertiles (reference), respectively]. In analyses of potassium source, the daily proportion of dietary potassium intake from animal- and plant-based sources (%) was calculated as the total amount of dietary potassium intake from animal- and plant-based sources, respectively, divided by the sum of the total amount of dietary potassium intake from both sources; animal-dominant sources were defined as having >50% of dietary potassium intake from animal-based sources, whereas plant-dominant sources were defined as having >50% of dietary potassium intake from

plant-based sources. In analyses of macronutrients, dietary fiber scaled to total energy intake (g/1000 kcal) was categorized as low or high intake [lowest 2 tertiles and highest tertile (reference), respectively], whereas dietary protein scaled to total energy intake (g/1000 kcal) was parsed as low or high intake [lowest tertile and highest 2 tertiles (reference), respectively]. In analyses of minerals, dietary phosphorus scaled to total energy intake (mg/1000 kcal) was parsed as low or high intake [lowest tertile and highest 2 tertiles (reference), respectively], whereas dietary sodium scaled to total energy intake (g/1000 kcal) was parsed as low or high intake [<2.0 and ≥2.0 g/1000 kcal (reference), respectively] (21).

Outcome ascertainment

The primary outcome of interest was all-cause mortality. Mortality data were obtained through linkage of NHANES data to death certificate data from the National Death Index, which is a centralized database of death records compiled by the NCHS using identifying information such as Social Security number. At-risk time for the outcome of interest commenced the day after 24-h dietary recall data collection, and participants were censored for death or at the end of the follow-up period (31 December, 2014).

Sociodemographic, comorbidity, physical activity, and supplement use covariates

Information on socio-demographics, comorbidities, alcohol intake, and smoking status were self-reported by participants during the interview portion of the survey (22). Race/ethnicity was categorized as Hispanic (defined as Mexican-American and other Hispanic), non-Hispanic White, non-Hispanic Black, and other race/ethnicity (including multiracial participants). Family income status was defined by the poverty income ratio and was categorized as low family income (0.000–1.300), middle family income (1.301–3.500), and high family income (≥3.501).

Statistical methods

Baseline characteristics across exposure groups were compared as mean ± SD or median [IQR] values as dictated by data type. We first estimated the association between dietary potassium intake and all-cause mortality risk using Cox regression in 4 incremental models with the following covariates in the overall cohort, as well as in analyses stratified by the presence or absence of impaired kidney function (eGFR ≥60 and <60 mL · min^{-1} · 1.73 m^{-2}, respectively):

1) unadjusted analyses: no adjustment for covariates;
2) case-mix adjusted analyses: adjusted for age, sex, race/ethnicity, family income status, education status, and marital status;
3) expanded case-mix adjusted analyses: adjusted for case-mix covariates plus cardiovascular disease (CVD), cancer, diabetes, hypertension, smoking status, alcohol intake status, and BMI; and
4) expanded case-mix + laboratory adjusted analyses: adjusted for expanded case-mix covariates plus serum creatinine and serum potassium concentrations.

We a priori defined the expanded case-mix model as our primary model, which included core sociodemographic measures and other key confounders of the association between dietary potassium intake and survival. We designated the expanded case-mix + laboratory model as an exploratory model, which included confounders as well as potential causal pathway intermediates of the dietary potassium–mortality association. We also conducted sensitivity analyses in which we in addition adjusted for medications that may influence serum potassium concentrations (i.e., thereby potentially influencing the amount of dietary potassium intake), designated as the *1*) expanded case-mix + laboratory + renin–angiotensin–aldosterone system (RAAS) inhibitors + diuretics, *2*) expanded case-mix + laboratory + RAAS inhibitors + diuretics + potassium-sparing diuretics, and *3*) expanded case-mix + laboratory + RAAS inhibitors + diuretics + potassium-sparing diuretics + β blockers + nonsteroidal anti-inflammatory drugs (NSAIDs) models. There were no missing values for age, sex, race/ethnicity, marital status, smoking status, alcohol use, comorbidities, and BMI. The remaining covariates ascertained at baseline had <10% missing data, including education status (7%), family income status (8%), and serum potassium (<1%). For the aforementioned covariates, missing data were addressed using multiple imputation with 15 imputed data sets. Statistical analyses were conducted and figures were created using STATA version 13.1 (Stata Corp.) and SigmaPlot version 13 (Systat Software).

Results

Study population

The final study population consisted of 3172 and 34,721 adult participants with impaired and normal kidney function, respectively (eGFRs <60 and ≥60 mL · min^{-1} · 1.73 m^{-2}, respectively; Supplemental Figure 1). In participants with impaired kidney function, the median [IQR] daily dietary potassium scaled to total energy intake was 1406 mg/1000 kcal [1124–1738 mg/1000 kcal]. In participants with normal kidney function, the median [IQR] daily dietary potassium scaled to total energy intake was 1219 mg/1000 kcal [956–1550 mg/1000 kcal].

Table 1 shows baseline characteristics of the cohort stratified by *1*) dietary potassium scaled to total energy intake and *2*) kidney function. Among participants with normal and impaired kidney function, those with the lowest tertile of dietary potassium scaled to total energy intake tended to be younger and male; were more likely to be Black and were less likely to be non-Hispanic White; were more likely to have low family income; were less likely to have completed higher levels of education (i.e., college graduate or above); were less likely to have CVD and cancer; and had lower dietary energy and sodium intake compared with those with the highest tertile of intake. In participants with impaired kidney function, those with lower dietary potassium intake were more likely to have hypertension and lower eGFRs, whereas among participants with normal kidney function those with lower dietary potassium intake were less likely to have hypertension and were more likely to have higher eGFRs. Notably, in both participants with normal and impaired kidney function, mean serum potassium concentrations tended to be similar across all dietary potassium intake groups.

TABLE 1 Baseline characteristics of participants across tertiles of dietary potassium intake scaled to total energy intake (mg/1000 kcal), stratified by eGFR[1]

	eGFR <60 mL · min^{-1} · 1.73 m^{-2}				eGFR ≥60 mL · min^{-1} · 1.73 m^{-2}			
	Overall	Dietary potassium, mg/1000 kcal			Overall	Dietary potassium, mg/1000 kcal		
		Tertile 1 (0 to <1060)	Tertile 2 (1060 to <1437)	Tertile 3 (1437–26,588)		Tertile 1 (0 to <1060)	Tertile 2 (1060 to <1437)	Tertile 3 (1437–26,588)
Participants, *n*	3172	617	1050	1505	34,721	12,031	11,567	11,123
Age, y	73 ± 10	69 ± 12	73 ± 10	74 ± 10	45 ± 18	39 ± 16	46 ± 18	52 ± 18
Male, %	47	51	47	45	50	54	52	45
Race/ethnicity, %								
Hispanic	12	13	12	13	28	25	29	29
Non-Hispanic White	65	52	64	71	45	40	44	50
Non-Hispanic Black	19	31	20	12	21	29	19	13
Other/missing	4	3	4	4	7	6	7	8
Family income, %								
Low income	28	34	28	25	30	34	28	26
Middle income	40	38	40	42	34	34	34	33
High income	24	20	23	26	29	24	31	32
Education completion, %								
Less than 9th grade	17	21	16	17	11	8	11	14
9–11th Grade (includes 12th grade with no diploma)	17	21	18	15	14	16	14	13
High school graduate/GED or equivalent	25	24	27	25	21	23	21	20
Some college or AA degree	23	21	23	24	26	26	26	26
College graduate or above	17	13	17	19	20	14	21	25
Married/living with partner, %	52	52	52	52	56	48	58	62
Alcohol use (≥12 alcohol drinks/y), %	57	58	56	56	64	63	65	63
Smoking status (≥100 cigarettes in lifetime), %	51	54	53	48	43	43	43	43
Comorbidity status, %								
Cardiovascular disease	34	29	35	35	7	5	7	9
Cancer	22	20	22	23	7	5	7	10
Diabetes	28	28	26	29	9	6	9	12
Hypertension	72	75	73	69	29	23	29	34
BMI, kg/m^2	29.2 ± 6.4	30.0 ± 7.4	29.0 ± 6.3	28.9 ± 6.0	28.5 ± 6.7	28.7 ± 7.2	28.6 ± 6.6	28.2 ± 6.2
eGFR, mL · min^{-1} · 1.73 m^{-2}	46 ± 12	44 ± 15	47 ± 12	47 ± 12	101 ± 21	106 ± 20	100 ± 20	95 ± 20

(*Continued*)

TABLE 1 (*Continued*)

	eGFR <60 mL · min^{-1} · 1.73 m^{-2}				eGFR ≥60 mL · min^{-1} · 1.73 m^{-2}			
	Overall	Dietary potassium, mg/1000 kcal			Overall	Dietary potassium, mg/1000 kcal		
		Tertile 1 (0 to <1060)	Tertile 2 (1060 to <1437)	Tertile 3 (1437–26,588)		Tertile 1 (0 to <1060)	Tertile 2 (1060 to <1437)	Tertile 3 (1437–26,588)
Serum creatinine, mg/dL	1.3 [1.1–1.5]	1.4 [1.2–1.7]	1.3 [1.1–1.5]	1.3 [1.1–1.5]	0.8 [0.7–1.0]	0.8 [0.7–1.0]	0.8 [0.7–1.0]	0.8 [0.7–0.9]
Serum potassium, mmol/L	4.2 ± 0.5	4.2 ± 0.5	4.2 ± 0.5	4.2 ± 0.5	4.0 ± 0.3	4.0 ± 0.3	4.0 ± 0.3	4.0 ± 0.3
Dietary energy intake, kcal/d	1546 [1176–2039]	1777 [1281–2371]	1626 [1253–2161]	1425 [1087–1837]	1986 [1468–2678]	2279 [1674–3069]	2071 [1560–2715]	1673 [1253–2194]
Dietary sodium intake, mg/d	1615 [1305–1978]	1520 [1233–1818]	1613 [1333–1952]	1654 [1327–2063]	1565 [1267–1901]	1483 [1207–1776]	1582 [1296–1898]	1651 [1312–2069]
Medications, %								
RAAS inhibitors	25.2	24.6	25.3	25.4	9.1	6.2	9.5	11.8
Diuretics	12.0	10.5	12.3	12.4	3.1	2.2	3.2	4.0
Potassium-sparing diuretics	5.4	4.4	5.4	5.8	1.1	1.0	1.0	1.4
β Blockers	18.5	16.9	18.5	19.1	4.8	3.1	4.9	6.6
NSAIDs	4.3	4.5	4.4	4.1	4.1	4.0	4.2	4.7

[1]Values are means ± SDs or medians [IQRs] for continuous variables as dictated by data type and column percentages for categoric variables, unless otherwise indicated. Tertiles of dietary potassium scaled to total energy intake categorized as tertile 1 (<1060 mg/1000 kcal), tertile 2 (1060 to <1437 mg/1000 kcal), and tertile 3 (≥1437 mg/1000 kcal). AA, Associates of Arts; eGFR, estimated glomerular filtration rate; GED, General Education Development; NSAID, nonsteroidal anti-inflammatory drug; RAAS, renin–angiotensin–aldosterone system.

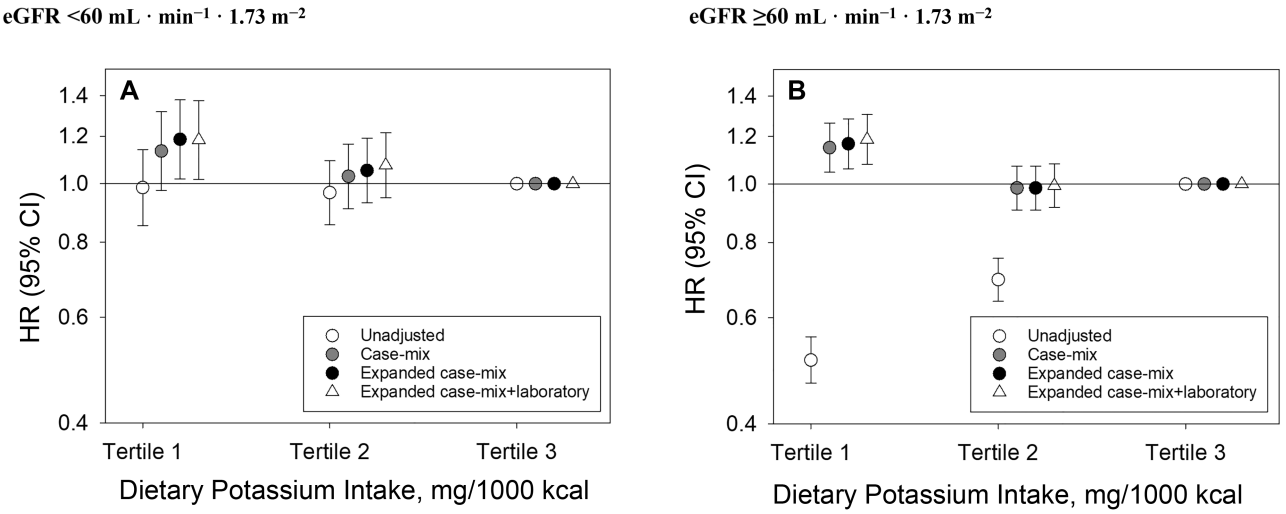

FIGURE 1 Association of dietary potassium intake scaled to total energy intake (mg/1000 kcal) with all-cause mortality risk, stratified by eGFR in participants with impaired kidney function (A; $n = 3172$) and normal kidney function (B; $n = 34{,}721$), estimated by Cox regression. Each plot shows HRs and their 95% CIs estimated using Cox regression models incrementally adjusted with the following covariates: *1*) unadjusted analyses; *2*) case-mix analyses adjusted for age, sex, race/ethnicity, family income status, educational status, and marital status; *3*) expanded case-mix analyses adjusted for case-mix covariates plus cardiovascular disease, cancer, diabetes, hypertension, smoking status, alcohol status, and BMI; and *4*) expanded case-mix + laboratory analyses adjusted for expanded case-mix covariates plus serum creatinine and serum potassium. Dietary potassium tertiles 1, 2, and 3 correspond to dietary potassium scaled to total energy intake categorized as <1060, 1060 to <1437, and ≥1437 mg · 1000 kcal^{-1} · d^{-1}, respectively, based on distributions in the overall cohort. eGFR, estimated glomerular filtration rate.

Dietary potassium intake and mortality risk

Participants with impaired kidney function contributed a total of 19,715 person-years of follow-up, during which 1332 death events were observed (crude mortality rate: 68 deaths/1000 person-years), whereas those with normal kidney function contributed a total of 285,140 person-years of follow-up, during which 3098 death events were observed (crude mortality rate: 11 deaths/1000 person-years). Median [IQR] follow-up times were 5.8 y [3.0–8.8 y] and 7.8 y [4.4–11.8 y] among participants with impaired and normal kidney function, respectively.

In primary analyses examining dietary potassium intake scaled to total energy intake and survival using the Nutrient Density Model approach, among participants with impaired and normal kidney function the lowest tertile of intake was associated with higher mortality risk in expanded case-mix analyses (ref: highest tertile): [adjusted HR (aHR): 1.18; 95% CI: 1.02, 1.38 and aHR: 1.17; 95% CI: 1.06, 1.28, respectively] (**Figure 1**, **Supplemental Table 1**). These associations were robust in models incrementally adjusted for expanded case-mix + laboratory covariates (aHR: 1.18; 95% CI: 1.02, 1.37 and aHR: 1.19; 95% CI: 1.08, 1.30, respectively). In sensitivity analyses that incrementally adjusted for medications that may affect serum potassium concentrations, associations between the lowest tertile of potassium intake and higher death risk persisted in the expanded case-mix + laboratory + RAAS inhibitors + diuretics, expanded case-mix + laboratory + RAAS inhibitors + diuretics + potassium-sparing diuretics, and expanded case-mix + laboratory + RAAS inhibitors + diuretics + potassium-sparing diuretics + β blockers + NSAIDs adjusted models among participants with impaired and normal kidney function (**Supplemental Table 2**).

In sensitivity analyses examining the relation between dietary potassium intake and mortality risk using the Residual Model approach, we observed a robust pattern of associations in which the lowest tertile of dietary potassium intake was associated with higher mortality risk among participants with impaired and normal kidney function in expanded case-mix analyses (ref: highest tertile) (aHR: 1.21; 95% CI: 1.04, 1.40 and aHR: 1.21; 95% CI: 1.10, 1.33, respectively) (**Supplemental Table 3**). In sensitivity analyses examining associations of dietary potassium intake with survival using the Standard Model approach, the lowest tertile of dietary potassium intake was also associated with higher mortality risk among participants with normal kidney function in expanded case-mix analyses (aHR: 1.25; 95% CI: 1.11, 1.40) (Supplemental Table 3). However, in participants with impaired kidney function, associations of the lowest tertile of dietary potassium intake with mortality were not significant (aHR: 1.09; 95% CI: 0.91, 1.31) in expanded case-mix analyses.

Interplay between dietary potassium intake and potassium source

In secondary analyses, we examined the impact of the interplay of dietary potassium intake and potassium source on survival by examining different pairings of dietary potassium intake scaled to total energy intake with potassium consumption from animal- or plant-dominant sources. Among participants with impaired kidney function, compared with high dietary potassium intake from plant-dominant sources, those who had low dietary potassium intake from animal-dominant sources had higher death risk in expanded case-mix analyses (aHR: 1.34; 95% CI: 1.09, 1.63) (**Figure 2**, **Supplemental Table 4**). Similarly, among participants with normal kidney function, compared with

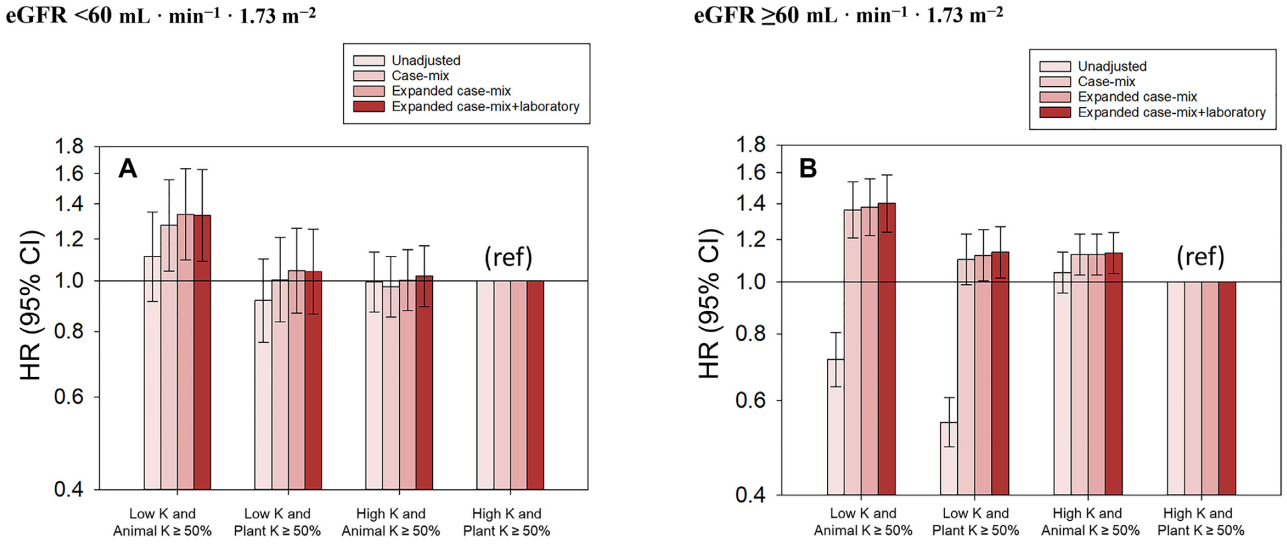

FIGURE 2 Association of pairings of dietary K intake scaled to total energy intake (mg/1000 kcal) and source of dietary K (animal- or plant-based) with all-cause mortality risk, stratified by eGFR in participants with impaired kidney function (A; $n = 3172$) and normal kidney function (B; $n = 34{,}721$), estimated by Cox regression. Each plot shows HRs and their 95% CIs estimated using Cox regression models incrementally adjusted with the following covariates: *1*) unadjusted analyses; *2*) case-mix analyses adjusted for age, sex, race/ethnicity, family income status, educational status, and marital status; *3*) expanded case-mix analyses adjusted for case-mix covariates plus cardiovascular disease, cancer, diabetes, hypertension, smoking status, alcohol status, and BMI; and *4*) expanded case-mix + laboratory analyses adjusted for expanded case-mix covariates plus serum creatinine and serum K. Low K intake was defined as the lowest tertile of dietary K intake, and high K intake was defined as the 2 highest tertiles of dietary K intake. Dietary K tertiles 1, 2, and 3 correspond to dietary K scaled to total energy intake categorized as <1060, 1060 to <1437, and ≥1437 mg · 1000 $kcal^{-1} \cdot d^{-1}$, respectively, based on distributions in the overall cohort. Dietary source was defined as animal-based sources (milk and milk products; meat, poultry, fish, and mixtures; and eggs) or plant-based sources (legumes, nuts, and seeds; grain products; fruits; and vegetables). The daily proportion of dietary K intake from animal- and plant-based sources (%) was calculated as the total amount of dietary K intake from animal- and plant-based sources, respectively, divided by the sum of the total amount of dietary K intake from both sources. Animal-dominant sources were defined as having >50% of dietary K intake from animal-based sources, whereas plant-dominant sources were defined as having >50% of dietary K intake from plant-based sources. eGFR, estimated glomerular filtration rate; K, potassium.

high dietary potassium intake from plant-dominant sources, those who had low dietary potassium intake from animal-dominant sources had higher mortality risk in expanded case-mix analyses (aHR: 1.38; 95% CI: 1.22, 1.56). Notably, among participants with normal kidney function, low dietary potassium intake from plant-dominant sources and high potassium intake from animal-dominant sources were each associated with worse survival, albeit with weaker point estimates for death risk (ref: high dietary potassium intake + plant-dominant sources) (aHR: 1.12; 95% CI: 1.00, 1.25 and aHR: 1.12; 95% CI: 1.03, 1.23, respectively). These associations were robust in models incrementally adjusted for expanded case-mix + laboratory covariates.

Interplay between dietary potassium and macronutrient intakes

We also examined the interplay of the consumption of dietary potassium and specific macronutrients by examining different pairings of potassium intake scaled to total energy intake with protein (separately) and fiber consumption. With respect to dietary potassium–protein interactions, among participants with impaired kidney function, compared with those with high potassium and high protein intake, those with low potassium and high protein intake had higher mortality risk in expanded case-mix analyses (aHR: 1.24; 95% CI: 1.02, 1.50), whereas associations of low potassium–low protein intake and high potassium–low protein intake with survival were not statistically significant (**Figure 3**, **Supplemental Table 5**). Among participants with normal kidney function, pairing of low potassium–low protein intake and low potassium–high protein intake were each associated with higher death risk (aHR: 1.20; 95% CI: 1.08, 1.34 and aHR: 1.19; 95% CI: 1.06, 1.34, respectively). These associations remained significant in models incrementally adjusted for expanded case-mix + laboratory covariates.

In analyses examining dietary potassium–fiber interactions, among participants with impaired and normal kidney function, compared with those with high dietary potassium–high fiber intake, those with low potassium–low fiber intake had higher mortality risk in expanded case-mix analyses (aHR: 1.20; 95% CI: 1.05, 1.37 and aHR: 1.09; 95% CI: 1.00, 1.20, respectively) (**Figure 4**, **Supplemental Table 6**). Notably, among participants with impaired kidney function, high potassium–low fiber intake was also associated with higher death risk in expanded case-mix analyses (aHR: 1.21; 95% CI: 1.04, 1.42). These associations were more potent in analyses adjusted for expanded case-mix + laboratory covariates.

Interplay between dietary potassium and mineral intakes

We also investigated the impact of pairings of dietary potassium and mineral intake on survival. In analyses examining dietary potassium–phosphorus interactions, among participants with impaired kidney function, compared with those with high

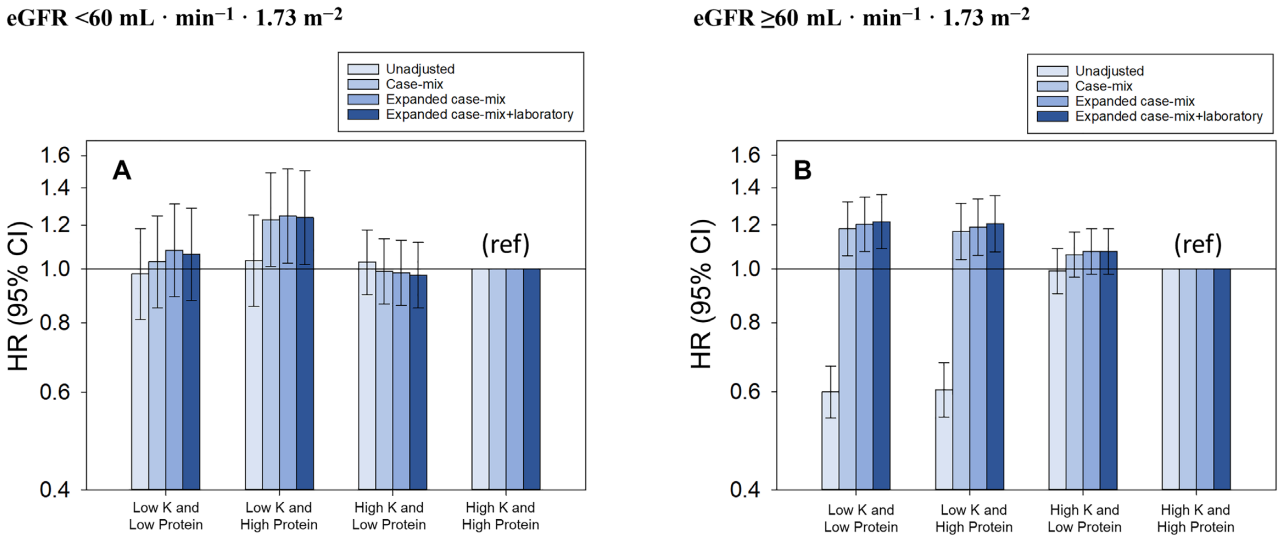

FIGURE 3 Association of pairings of dietary K intake scaled to total energy intake (mg/1000 kcal) and dietary protein intake scaled to total energy intake (g/1000 kcal) with all-cause mortality risk, stratified by eGFR in participants with impaired kidney function (A; $n = 3172$) and normal kidney function (B; $n = 34{,}721$), estimated by Cox regression. Each plot shows HRs and their 95% CIs estimated using Cox regression models incrementally adjusted with the following covariates: *1*) unadjusted analyses; *2*) case-mix analyses adjusted for age, sex, race/ethnicity, family income status, educational status, and marital status; *3*) expanded case-mix analyses adjusted for case-mix covariates plus cardiovascular disease, cancer, diabetes, hypertension, smoking status, alcohol status, and BMI; and *4*) expanded case-mix + laboratory analyses adjusted for expanded case-mix covariates plus serum creatinine and serum K. Low K intake WAS defined as the lowest tertile of dietary K intake, and high K intake WAS defined as the 2 highest tertiles of dietary K intake. Dietary K tertiles 1, 2, and 3 correspond to dietary K scaled to total energy intake categorized as <1060, 1060 to <1437, and ≥1437 mg · 1000 kcal^{-1} · d^{-1}, respectively, based on distributions in the overall cohort. Low protein intake was defined as the lowest tertile of dietary protein intake, and high protein intake was defined as the highest 2 tertiles of dietary protein intake. Dietary protein tertiles 1, 2, and 3 correspond to dietary protein scaled to total energy intake categorized as <33, 33 to <42, and ≥42 g · 1000 kcal^{-1} · d^{-1}, respectively. eGFR, estimated glomerular filtration rate; K, potassium.

potassium–high phosphorus intake, those who had a combination of low potassium–high phosphorus intake had higher death risk (aHR: 1.23; 95% CI: 1.00, 1.52), whereas low potassium–low phosphorus intake and high potassium–low phosphorus intake were not associated with worse survival (**Figure 5**, **Supplemental Table 7**). In contrast, among participants with normal kidney function, those who had a combination of low potassium–low phosphorus intake had higher mortality risk in expanded case-mix analyses (aHR: 1.21; 95% CI: 1.09, 1.35). Stronger point estimates were observed in models incrementally adjusted for expanded case-mix + laboratory covariates.

In analyses examining dietary potassium–sodium interactions, among participants with impaired kidney function, compared with high potassium–high sodium consumption, associations of low potassium–high sodium intake and low potassium–low sodium intake with survival were not significant in expanded case-mix analyses (aHR: 1.30; 95% CI: 0.92, 1.82 and aHR: 1.14; 95% CI: 0.95, 1.37, respectively) (**Figure 6**, **Supplemental Table 8**). Among participants with normal kidney function, compared with high potassium–high sodium intake, low potassium–high sodium and low potassium–low sodium intake were each associated with higher death risk (aHR: 1.35; 95% CI: 1.11, 1.65 and aHR: 1.21; 95% CI: 1.07, 1.36, respectively). These associations were amplified in models incrementally adjusted for expanded case-mix + laboratory covariates.

Discussion

In this large, nationally representative cohort of US adults who underwent rigorous protocolized 24-h dietary recall assessment, we observed that lower dietary potassium intake scaled to total energy intake was associated with higher mortality risk among participants with impaired and normal kidney function independently of socio-demographics, comorbidities, socioeconomic status, lifestyle factors, and BMI. Upon concomitantly considering the sources of dietary potassium, we found that participants with low potassium intake largely from animal-based foods had higher death risk irrespective of underlying kidney function. Furthermore, we also found that there was a synergistic relation between dietary potassium intake and specific macronutrients (dietary protein and fiber) and minerals (dietary phosphorus and sodium) upon survival among participants with impaired and normal kidney function. These associations were robust and/or further amplified in secondary and sensitivity analyses that utilized alternative approaches for accounting for total energy intake (i.e., Residual and Standard Model approaches), and in addition adjusted for kidney function, circulating potassium concentrations, and medications that influence serum potassium.

In the non-CKD population, there has been a large body of evidence demonstrating the beneficial effects of higher dietary potassium intake on blood pressure and cardiovascular health (23), whereas data examining the impact of dietary potassium intake on survival have been comparatively limited yet consistent. With respect to the latter outcome, in a study in 101,945 adults from the PURE (Prospective Urban Rural Epidemiology) cohort recruited from 17 countries who underwent 24-h urinary potassium collection as a surrogate for dietary potassium intake, lower potassium excretion was associated with higher risk of the composite endpoint of death and major cardiovascular events (24). In an extended follow-up study of the PURE cohort with

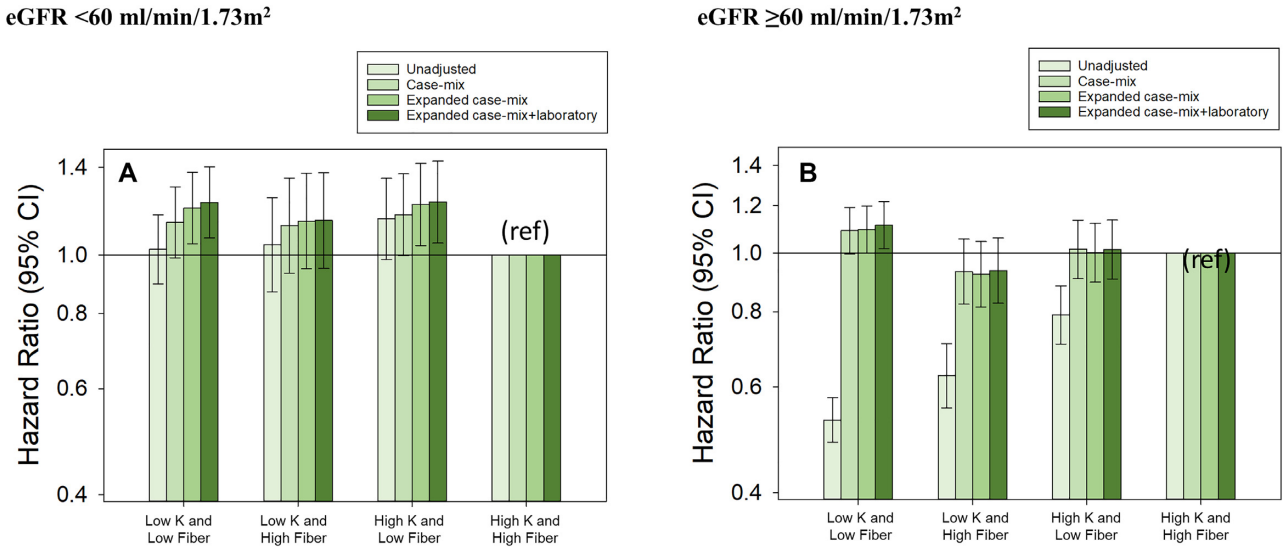

FIGURE 4 Association of pairings of dietary K intake scaled to total energy intake (mg/1000 kcal) and dietary fiber intake scaled to total energy intake (g/1000 kcal) with all-cause mortality risk, stratified by eGFR in participants with impaired kidney function (A; $n = 3172$) and normal kidney function (B; $n = 34{,}721$), estimated by Cox regression. Each plot shows HRs and their 95% CIs estimated using Cox regression models incrementally adjusted with the following covariates: *1*) unadjusted analyses; *2*) case-mix analyses adjusted for age, sex, race/ethnicity, family income status, educational status, and marital status; *3*) expanded case-mix analyses adjusted for case-mix covariates plus cardiovascular disease, cancer, diabetes, hypertension, smoking status, alcohol status, and BMI; and *4*) expanded case-mix + laboratory analyses adjusted for expanded case-mix covariates plus serum creatinine and serum K. Low K intake was defined as the lowest tertile of dietary K intake, and high K intake was defined as the 2 highest tertiles of dietary K intake. Dietary K tertiles 1, 2, and 3 correspond to dietary K scaled to total energy intake categorized as <1060, 1060 to <1437, and ≥1437 mg · 1000 $kcal^{-1}$ · d^{-1}, respectively, based on distributions in the overall cohort. Low fiber intake was defined as the 2 lowest tertiles of dietary fiber intake, and high fiber intake was defined as the highest tertile of dietary fiber intake. Dietary fiber tertiles 1, 2, and 3 correspond to dietary fiber scaled to total energy intake categorized as <5.6, 5.6 to <8.9, and ≥8.9 mg/1000 kcal/d, respectively. eGFR, estimated glomerular filtration rate; K, potassium.

103,570 participants from 18 countries (25), a similar inverse association of low urinary potassium excretion with higher risk of death and cardiovascular events was observed (25). Similarly, in an analysis of 90,137 older females from the Women's Health Initiative who underwent FFQ assessment, those with the highest quartile of dietary potassium had lower mortality risk than those with the lowest quartile of intake (26).

In the non-dialysis-dependent CKD population, there has been comparatively less data on the relation between dietary potassium intake and survival which have shown inconsistent findings. For example, in a post hoc analysis of 812 stage 3–5 CKD patients from the MDRD (Modification of Diet in Renal Disease) Study, there was an inverse association of higher urinary potassium excretion with decreased mortality risk yet no relation with CKD progression (27). In contrast, in a subsequent study in 3939 participants from the CRIC (Chronic Renal Insufficiency Cohort) study with eGFRs of 20–70 mL · min^{-1} · 1.73 m^{-2}, higher urinary potassium excretion was not associated with survival yet was linked with heightened risk of CKD progression (28). However, it bears mention that urinary potassium excretion may not fully reflect dietary potassium intake, particularly in CKD patients who have decreased urinary potassium excretion and increased gastrointestinal potassium secretion as GFR declines (29).

To our knowledge, this is the first study comparing the relation of dietary potassium intake measured by 24-h dietary recall with mortality risk among participants with impaired or normal kidney function using established eGFR thresholds for the presence or absence of CKD (eGFR < 60 and ≥60 mL · min^{-1} · 1.73 m^{-2}, respectively), and concurrently taking into consideration animal- compared with plant-based sources of dietary potassium. Our results showing that lower dietary potassium intake is associated with higher mortality risk in adults with normal kidney function support the USDA and US Department of Health and Human Services "Dietary Guidelines for Americans, 2020–2025" recommendations for higher dietary potassium intake in the general population. However, our findings demonstrating that lower dietary potassium intake is associated with worse survival in adults with impaired kidney function call into question long-standing paradigms and clinical practice guidelines advising dietary potassium restriction in CKD patients, which are based on sparse evidence and provide limited guidance with respect to dietary potassium source. When concomitantly examining the amount and source of dietary potassium intake, we also found that low potassium consumption paired with predominantly animal-based potassium sources was associated with worse survival as opposed to plant-based sources, irrespective of underlying kidney function. Although emerging data have shown the salutary benefits of plant-based foods, including lower net acid load and correction of metabolic acidosis; favorable effects on intestinal motility and microbiota composition; and a high content of fiber, antioxidants, and trace elements (1), further studies comparing animal- with plant-based dietary potassium sources are needed to inform clinical practice in those with and without CKD.

Another noteworthy finding of our study was the interrelation between dietary potassium intake and coexisting nutrient/mineral consumption upon survival, particularly in those with impaired

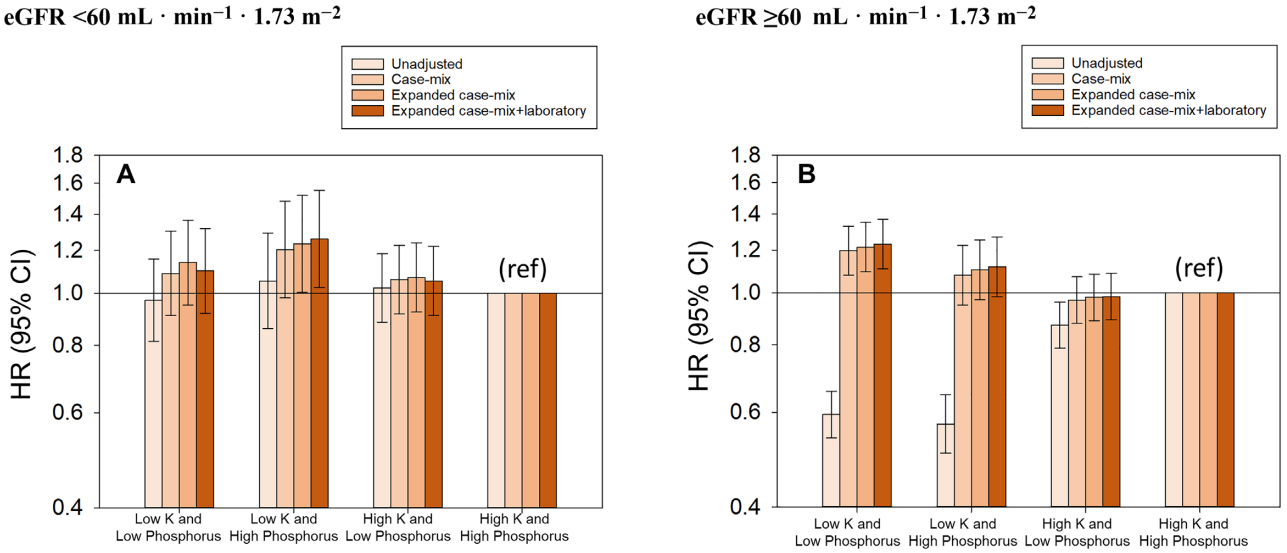

FIGURE 5 Association of pairings of dietary K intake scaled to total energy intake (mg/1000 kcal) and dietary phosphorus intake scaled to total energy intake (mg/1000 kcal) with all-cause mortality risk, stratified by eGFR in participants with impaired kidney function (A; $n = 3172$) and normal kidney function (B; $n = 34{,}721$), estimated by Cox regression. Each plot shows HRs and their 95% CIs estimated using Cox regression models incrementally adjusted with the following covariates: *1*) unadjusted analyses; *2*) case-mix analyses adjusted for age, sex, race/ethnicity, family income status, educational status, and marital status; *3*) expanded case-mix analyses adjusted for case-mix covariates plus cardiovascular disease, cancer, diabetes, hypertension, smoking status, alcohol status, and BMI; and *4*) expanded case-mix + laboratory analyses adjusted for expanded case-mix covariates plus serum creatinine and serum K. Low K intake was defined as the lowest tertile of dietary K intake, and high K intake was defined as the 2 highest tertiles of dietary K intake. Dietary K tertiles 1, 2, and 3 correspond to dietary K scaled to total energy intake categorized as <1060, 1060 to <1437, and ≥1437 mg · 1000 kcal^{-1} · d^{-1}, respectively, based on distributions in the overall cohort. Low phosphorus intake was defined as the lowest tertile of dietary phosphorus intake, and high phosphorus intake was defined as the highest 2 tertiles of dietary phosphorus intake. Dietary phosphorus intake tertiles 1, 2, and 3 correspond to dietary phosphorus intake scaled to total energy intake categorized as <544, 544 to <689, and ≥689 mg/1000 kcal, respectively. eGFR, estimated glomerular filtration rate; K, potassium.

kidney function. When low potassium intake was paired with high protein, low fiber, or high phosphorus consumption (considered to be the worst dietary pairings in non-dialysis-dependent CKD patients), we observed the strongest associations with mortality in participants with impaired kidney function when compared with other nutritional combinations. Whereas studies of renal nutrition have historically focused on single nutrients, these findings underscore the importance of using a holistic approach in the nutritional management of CKD patients that takes into account the additive/synergistic effects of food substances, may be more predictive of health outcomes, and may provide more meaningful and pragmatic guidance to patients in promoting sustainable dietary behaviors that promote overall health status.

The strengths of our study include its examination of a large nationally representative contemporary cohort of US adults; detailed availability of participant-level data on socio-demographics, dietary intake, nutritional status, health behaviors, kidney function, and laboratory results; and availability of rigorous 24-h recall data that allowed us to concomitantly take into consideration total energy intake and other dietary factors (i.e., macronutrients, minerals). However, several limitations of our study bear mention. First, dietary potassium intake was ascertained using a baseline 24-h dietary recall measurement collected at a single point in time, which may be prone to recall bias and may not be representative of long-term dietary potassium and/or habitual dietary intake, and which may have varying results according to the reference database used for analysis. However, it should be recognized that alternative metrics of dietary potassium estimation (24-h urinary potassium) are also prone to measurement bias and fluctuations over time and may not enable comprehensive examination of dietary intake (source of dietary potassium, dietary energy) as compared with 24-h dietary recall. Second, information regarding participants' access to dietitian-led dietary support was not collected within the NHANES study protocol; in addition, a sizable proportion of the study cohort with preserved kidney function may not have seen a dietitian by virtue of not having CKD. Third, given that the NHANES cohort does not have a high prevalence of patients with advanced CKD, our study's findings may not be generalizable to those with severe kidney dysfunction, particularly those requiring dialysis. Fourth, owing to data limitations, we were not able to ascertain the fasting status of participants' serum potassium assessments, nor their longitudinal serum potassium concentrations after food consumption, which is an area rendering corollary research studies (30). Finally, as with all observational studies, we cannot confirm a causal relation between dietary potassium and survival based on these findings.

In conclusion, our study has shown that, among participants with impaired and normal kidney function, lower dietary potassium consumption scaled to total energy intake was associated with higher death risk. Future studies are needed to elucidate the mechanistic pathways underlying the relation between higher dietary potassium intake and improved survival among those with and without CKD, as well as interventional studies examining

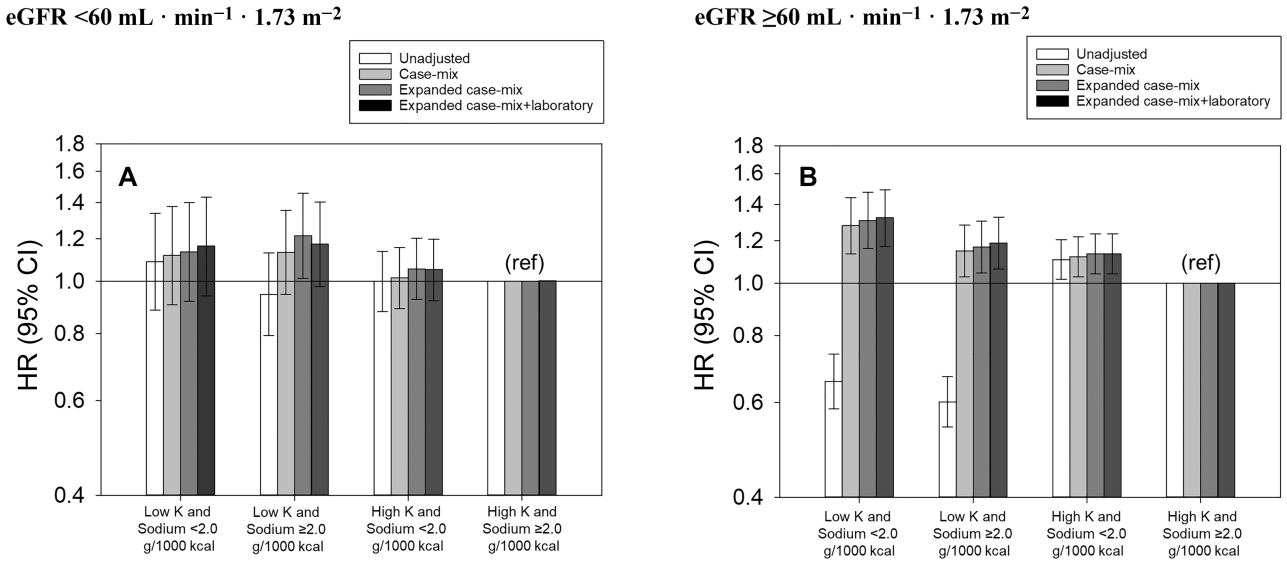

FIGURE 6 Association of pairings of dietary K intake scaled to total energy intake (mg/1000 kcal) and dietary sodium intake scaled to total energy intake (g/1000 kcal) with all-cause mortality risk, stratified by eGFR in participants with impaired kidney function (A; $n = 3172$) and normal kidney function (B; $n = 34{,}721$), estimated by Cox regression. Each plot shows HRs and their 95% CIs estimated using Cox regression models incrementally adjusted with the following covariates: *1*) unadjusted analyses; *2*) case-mix analyses adjusted for age, sex, race/ethnicity, family income status, educational status, and marital status; *3*) expanded case-mix analyses adjusted for case-mix covariates plus cardiovascular disease, cancer, diabetes, hypertension, smoking status, alcohol status, and BMI; and *4*) expanded case-mix + laboratory analyses adjusted for expanded case-mix covariates plus serum creatinine and serum K. Low K intake was defined as the lowest tertile of dietary K intake, and high K intake was defined as the 2 highest tertiles of dietary K intake. Dietary K tertiles 1, 2, and 3 correspond to dietary K scaled to total energy intake categorized as <1060, 1060 to <1437, and ≥ 1437 mg · 1000 kcal^{-1} · d^{-1}, respectively, based on distributions in the overall cohort. Low dietary sodium intake was defined as dietary sodium intake scaled to total energy intake <2.0 g · 1000 kcal^{-1} · d^{-1}, and high dietary sodium intake was defined as dietary sodium intake scaled to total energy intake ≥ 2.0 g · 1000 kcal^{-1} · d^{-1}. eGFR, estimated glomerular filtration rate; K, potassium.

the synergistic effects of potassium consumption, sources of its intake, and other coexisting dietary factors on health outcomes in these populations.

The authors' responsibilities were as follows—YN and CMR: designed the research (project conception, development of the overall research plan, and study oversight), conducted the research, and wrote the manuscript; YN: performed the statistical analyses; CMR: has primary responsibility for the final content; and YN, ASY, SM, LWM, RB, MKC, AD, CPK, DVN, KKZ, and CMR: read and approved the final manuscript.

Data availability

Data described in the article, code book, and analytic code will be made available upon request pending application and approval by the corresponding author.

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