## **ORIGINAL ARTICLE**



# **Potassium and fber: a controversial couple in the nutritional management of children with chronic kidney disease**

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## **Abstract**

**Background** Fruit and vegetable intake is commonly discouraged in children with chronic kidney disease (CKD) to avoid hyperkalemia. However, direct evidence in support of this widespread practice is lacking. Furthermore, the resultant restricted fber exposure may deprive CKD patients from potential health benefts associated with the latter. Therefore, we investigated associations between dietary potassium intake, fber intake, and serum potassium levels in pediatric CKD.

**Methods** This study is a longitudinal analysis of a 2-year, prospective, multi-institutional study, following children with CKD at 3-month intervals. At each visit, dietary potassium and fber intake were assessed, using 24-h recalls and 3-day food records. On the same occasion, serum potassium concentrations were determined. Associations between dietary potassium intake, dietary fber intake, and serum potassium concentrations were determined using linear mixed models.

**Results** Fifty-two CKD patients (7 transplant recipients, none on dialysis) aged 9 [4;14] years with an estimated glomerular filtration rate (eGFR) of 49 [25;68] mL/min/1.73 m<sup>2</sup> were included. For every g/day decrease in dietary potassium intake, the estimated mean daily fber intake was 5.1 g lower (95% confdence interval (CI), 4.3–5.9 g/day; *p*<0.001). Neither dietary potassium intake ( $p=0.40$ ) nor dietary fiber intake ( $p=0.43$ ) was associated with circulating potassium in a model adjusted for time point, eGFR, treatment with a renin–angiotensin–aldosterone system blocker, serum bicarbonate concentration, and body surface area.

**Conclusions** Dietary potassium and fber intake are closely related but were not associated with circulating potassium levels in pediatric CKD.

**Keywords** Chronic kidney disease · Pediatric · Diet · Fiber intake · Potassium intake · Serum potassium

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# **Introduction**

Strict dietary potassium restriction is recommended by many nephrology teams in children with chronic kidney disease (CKD) with or out of assumed risk of hyperkalemia [\[1\]](#page-6-0). Nevertheless, robust data underpinning the risk of hyperkalemia of the desired ranges are lacking, and the psycho-social impact of such dietary restrictions on the child and the family is seldom highlighted [[2](#page-6-1), [3](#page-6-2)]. In addition, there are no data regarding the extent of dietary potassium restriction needed to prevent or treat hyperkalemia [[1](#page-6-0)]. While randomized trials are lacking, several observational studies report no or poor associations between dietary potassium intake estimations and serum potassium levels or rates of hyperkalemia in adults with CKD [\[4](#page-7-0)–[9\]](#page-7-1). In fact, there is only limited, low-quality evidence supporting the claim that a low-potassium diet

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actually reduces serum potassium levels [\[4\]](#page-7-0). To date, the relationship between dietary potassium intake and serum potassium levels in pediatric CKD is unexplored.

In addition, an important undesirable side-efect of limiting potassium intake through avoidance of fruit and vegetable consumption is the lower fber exposure. In adult CKD patients, higher fber intake has been linked to a positive efect on uremia-associated gut dysbiosis, improved metabolic acidosis, better lipid profles, lower cardiovascular disease risk, lower risk of infammation, and reduced mortality [\[10–](#page-7-2)[12\]](#page-7-3). In parallel, the study of plant-based dietary patterns in adult CKD gained popularity, since a growing body of evidence indicates potential benefts in cardiovascular and mortality outcomes. Plant-based dietary patterns characteristically consist of fruits, vegetables, legumes, whole grains, nuts and seeds, thus being rich in both potassium and fber [[11](#page-7-4), [13](#page-7-5)–[16](#page-7-6)]. It remains unclear whether these benefts are attributable to potassium intake per se, a higher associated intake of vitamins, anti-oxidants and fber, or an overall healthier lifestyle [\[11\]](#page-7-4).

The aim of this study was to investigate associations between dietary potassium or fber intake and serum potassium levels across diferent stages of pediatric CKD and to examine whether potassium and fber intake correlate.

## **Methods**

## **Study design and population**

This study analyzes observational, longitudinal data from the 2-year, prospective, multicenter UToPaed study (NCT02624466), running from September 2015 to March 2019. Patients were children aged<18 years, with a confrmed CKD diagnosis stage 1–5 (defned using the Kidney Disease Improving Global Outcomes (KDIGO) guidelines), including kidney transplant recipients. Estimated glomerular fltration rate (eGFR), calculated by the updated Schwartz equation [\[17\]](#page-7-7), was used to defne CKD strata. Children undergoing dialysis were excluded. Assessments were made during a stable disease status; children with active infection or infammatory disease and malignancies were excluded. The 52 eligible patients who had available concurrent serum potassium measurements and assessments of dietary potassium and fber intakes in the present cohort were recruited from the Departments of Pediatric Nephrology of Ghent University Hospital and Antwerp University Hospital. The study fow chart is depicted in Fig. [1](#page-1-0). The study protocol was approved by the Ethics Committee of both centers (B670201524922). Written informed consent or assent was obtained from all legal guardians or participants above the age of 12, respectively.

#### <span id="page-1-0"></span>**Fig. 1** Study fow chart



#### **Study variables and biochemical assessments**

Data of interest for this analysis, collected at baseline and prospectively every trimestral visit, included demographics, cause of CKD, history of kidney transplant, anthropometric data for nutritional status assessment (standard deviation scores (SDS) for height, weight and body mass index (BMI)), medications, laboratory data, and diet histories (as detailed below).

Blood samples were collected from all patients as part of the routine clinical examination. Serum creatinine (photometric method), bicarbonate (photometric method), and potassium (indirect potentiometric method) concentrations were analyzed by the respective Clinical Laboratories of the University hospitals of Ghent and Antwerp.

## **Dietary assessment**

Detailed diet histories were obtained using a 3-day food record or a 24-h dietary recall at an aimed 50:50 ratio across the total follow-up period. For the 3-day food record, patients and/or their caregivers were asked to complete a printed, structured diary that was subsequently verifed by the dietician in a face-to-face interview. Twenty-four-hour recalls comprised a detailed recollection of everything the patient consumed (foods, beverages, sauces, condiments, dietary supplements) the day prior to the consultation and were completed directly by the dietician. If for some reason patients forgot to fll out or bring the 3-day food record for the visit, a 24-h recall was carried out instead so that dietary data could be matched to laboratory data. Standardized food models, a color photo atlas with choice between varying portion sizes and their corresponding weight of diferent food groups (Portiegroottes boek, Valetudo Consulting, third edition, March 2014), and a manual for the conversion of household measures to weight equivalents and standardized quantifcation of food items were used to increase accuracy of serving size estimations [\[18](#page-7-8)]. Non-standard mixed meals and recipes were broken down into their constituents. Subsequently, food records were entered into Evry-Diëtist 6.7.7.0 (Evry BV, Alphen aan den Rijn, The Netherlands), according to the Belgian Branded Food Products Database (Nubel, 5<sup>th</sup>) Edition). Alternatively, unknown food items were searched in the Dutch nutrient database (Nevo,  $4<sup>th</sup>$  Edition) or in the online database of trade names (Internubel). In case nutritional information was unavailable, manufacturers' labels or online sites of the branded foods were used. Total energy, potassium, and fber intake for every child was computed as the sum of all food items. All participants in our study received standard dietary counselling by a renal dietician. Dietary potassium restrictions were only implemented when clinically meaningful, repetitive hyperkalemia occurred.

Estimated daily potassium intake was expressed in mg/day and standardized to mg per kg body weight to compare to age-dependent National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines to prevent and treat hyperkalemia. Fiber, expressed in g/day, was corrected for body surface area (BSA), calculated by the Haycock formula (BSA =  $0.024265 \times \left( \text{length}^{0.3964} \right) \times \left( \text{weight}^{0.5378} \right) \text{m}^2$ ). Finally, the percentage dietary reference intake (%DRI) was calculated, expressing total dietary fber intake as a percentage of the age-dependent DRI for fber, according to the Belgian nutrition recommendation for healthy children [\[19](#page-7-9)].

## **Statistical analyses**

Normality of distributions was assessed by the Shapiro–Wilk test. Continuous variables are expressed as mean $\pm$ standard deviation (SD) or median [25th;75th percentile], as appropriate. Categorical variables are reported as frequencies and proportions. To compare potassium intake between the CKD stages, a Kruskal–Wallis test with post hoc Dunn's analysis and Bonferroni correction was used to account for multiple testing. Fiber intake was compared between the CKD stages by a one-way ANOVA test with post hoc Tukey comparison. In order to examine the correlation between dietary fber intake and potassium intake, a simple linear mixed model for daily fber intake was ftted with a random intercept for patient and with daily potassium intake as the only explanatory variable in the fxed efects part of the model. The proportional reduction in within-subject variance and betweensubject variance (compared to a null model) was computed as analogue to  $R^2$  for multilevel data. Linear mixed models for serum potassium concentration were ftted with a random intercept for patient, to take into account the repeated measurements within patients over time. The fxed efects part of a frst model contained the main efects of dietary intake (g/ day) and time point (categorical 0, 3, 6, 9, 12, 15, 18, 21, and 24 months). A second model was further adjusted for eGFR  ${\rm (ml/min/1.73 \ m^2)}$ , treatment with a renin–angiotensin–aldosterone system (RAAS) blocker (yes/no), metabolic acidosis represented by serum bicarbonate concentration (mEq/L), and body surface area  $(m<sup>2</sup>)$  as a proxy for age. All hypothesis testings were performed at the two-sided 5% signifcance level. All statistical analyses were performed using SPSS 26.0 (IBM, New York, USA), while the graphics were made in R version 3.6.1. Statistical analyses were conducted by the Biostatistics Unit of Ghent University.

# **Results**

Data from 52 children with CKD 1–5, of which 7 kidney transplant recipients, were analyzed. This accounted for a total of 279 repeated patient visits with a mean of 5 visits

(range 1–9) per patient and a median follow-up time of 19 [14;23] months. Baseline demographic, clinical, laboratory and dietary characteristics by eGFR category are presented in Table [1.](#page-3-0) Our cohort were mainly boys with a median age of 9 [4;14] years and an eGFR of 49 [25;68] mL/  $min/1.73$  m<sup>2</sup>. Metabolic acidosis, defined as serum bicarbonate<22 mEq/L, was present in 23/51 (45%) patients. Of the 20 patients treated with RAAS inhibitors, only one was on an angiotensin receptor blocker. One patient took a loop diuretic (furosemide) in addition to an angiotensinconverting enzyme inhibitor, while none of the patients were on potassium sparing diuretics. Potassium and fber intakes were estimated by 3-day food records/24-h recalls in a 40/60 ratio in comparison to the intended 50/50 ratio.

As summarized in Table [1,](#page-3-0) the median dietary potassium intake was 62 [44;81] mg/kg/day. In children aged 1–5 years, the median potassium intake of 76 [54;135] falls within the potassium-restricted diet range of 40–120 mg/kg/day

provided by the KDOQI guidelines as a starting point for infants and young children (Supplementary Table 1). The vague KDOQI guideline does not allow a similar comparison for older children. The %DRI for fber varies between CKD stages ( $p = 0.007$ ), with a lower fiber intake in patients with CKD stages  $4-5$  versus stages  $1-2$  ( $p=0.005$ ).

A simple linear mixed model for daily fber intake with daily potassium intake as the only explanatory variable revealed that for a daily potassium intake decrease of 1 g, the estimated mean daily fber intake decreased by 5.1 g (95% confdence interval (CI), 4.3 to 5.9 g/day; *p*<0.001). The within-subject variance in daily fiber intake could be explained for 28% by the daily potassium intake. The between-subject variance in daily fiber intake could be explained for 65% by the daily potassium intake (Fig. [2\)](#page-4-0).

Linear mixed model analysis revealed no association between dietary potassium intake (g/day) and serum potassium concentration in a model adjusted for time point

<span id="page-3-0"></span>**Table 1** Baseline demographic, clinical, laboratory, and dietary characteristics across diferent stages of CKD



Data are expressed as mean $\pm$ standard deviation (SD), number (percentage), or median (25th–75th percentile) as appropriate. *SDS*, standard deviation score; *BMI*, body mass index; *CAKUT*, congenital abnormalities of the kidney and urinary tract; *RAAS*, renin–angiotensin–aldosterone system; *eGFR*, estimated glomerular fltration rate; *BSA*, body surface area, *DRI*, dietary reference intake. \**n*=51; missing data from one patient

 $(p=0.34)$ , nor in a model adjusted for time point, eGFR, treatment with RAAS-inhibitor therapy, serum bicarbonate concentration, and body surface area  $(p=0.40)$ . Neither could an association be found between dietary fber intake



<span id="page-4-0"></span>Fig. 2 Scatter plot of the correlation between dietary fiber and potassium intake

<span id="page-4-1"></span>**Table 2** Linear mixed model analysis of serum potassium concentration

(g/day) and serum potassium concentration, using the same two models (Table [2\)](#page-4-1).

## **Discussion**

This study is the frst to investigate the link between dietary potassium and fber intake and serum potassium levels across diferent stages of CKD in a pediatric population. The key fndings are (i) dietary potassium intake correlates with dietary fiber intake and (ii) there is no association between the estimated amount of dietary potassium consumed nor dietary fber intake and circulating potassium in these patients.

First, we observed that every g/day decrease in dietary potassium intake was associated with an approximate fvefold decrease in dietary fber. The present association between dietary potassium intake and fber intake gives strength to our hypothesis that excessive restrictions in high-potassium foods, which are part of the intensifcation of nutritional counselling as CKD advances play an important role in limiting fber intake. Fiber sources are exclusively plant-based and often have a high potassium content.



Model 1: adjusted for time point

Model 2: adjusted for time point, dietary fber or dietary potassium, eGFR, RAAS blocker use, serum bicarbonate, and BSA

The estimate can be interpreted as the estimated mean change in serum potassium concentration for each unit increase in the explanatory variable

*eGFR*, estimated glomerular fltration rate; *RAAS*, renin–angiotensin–aldosterone system; *BSA*, body surface area (as a proxy for age)

As such, the consumption of vegetables and fruits is often a target of potassium restriction. However, there is a large spectrum of potassium content per unit of fber in plant foods [[20](#page-7-10), [21](#page-7-11)]. Also, there are numerous non-plant based dietary potassium sources, containing little to no fiber at all (e.g., foods of animal-origin, fast foods, beverages, sugar and sweets). Intriguingly, the top 3 contributors of potassium in adults on hemodialysis (HD) were meat products (beef, chicken, and 'Mexican food') [[5](#page-7-12)]. Vegetables do not even appear in the reported top 3 of major dietary potassium sources in childhood CKD, namely milk, fruit (of which the contributory proportion diminished as age advanced), and fast foods [[22\]](#page-7-13). Similarly, we observed an overall low fruit and vegetable intake in our cohort, in which fruits and vegetables had a median contribution of 34% [14;50] to the calculated potassium intake (data not shown). Of note, potato was the single largest contributor in the fruit/vegetable group and after the exclusion of potato and banana, the contribution further dropped to 16% [12;33]. It is often unrecognized that meat products contain nearly as much or more potassium than many fruits and vegetables, especially when potassium additives are used in enhanced meats or processed foods, resulting in a 2- to threefold increase in potassium content [\[6](#page-7-14), [23](#page-7-15)]. In addition, potassium in plant foods (50–60%) is absorbed to a lesser extent, compared to animal sources  $(80\%)$  and additives  $(100\%)$   $[24-26]$  $[24-26]$ .

Second, we found no association between dietary potassium intake and serum potassium, which is in line with observational studies in the adult CKD and HD population and a recent meta-analysis [[5–](#page-7-12)[7,](#page-7-18) [9,](#page-7-1) [27](#page-7-19)[–29](#page-7-20)]. The scarce potassium balance studies in adults with CKD showing an elevation of serum potassium after receiving potassium supplements are not clinically relevant, as the reported doses of potassium supplements exceed those of a normal diet [[4,](#page-7-0) [30](#page-7-21)[–32\]](#page-7-22). Potassium is not ingested in isolation, but as part of a meal, in which other nutrients infuence potassium distribution and excretion [[6\]](#page-7-14). Plant sources have the advantage that they promote intracellular potassium deposition because of their alkaline and insulin-stimulating properties [\[6,](#page-7-14) [24](#page-7-16)[–26](#page-7-17), [33](#page-7-23)]. Moreover, the accompanying fiber content in plant-based foods has been described to have a protective efect on serum potassium as it improves constipation, hereby facilitating fecal potassium excretion [\[4](#page-7-0)]. In addition, it has been reported that several non-dietary factors (e.g., use of RAAS inhibitors, catabolism, metabolic acidosis) might be more important determinants of serum potassium and hyperkalemia, further questioning the impact of diet on serum potassium [\[4](#page-7-0), [6,](#page-7-14) [11,](#page-7-4) [27\]](#page-7-19). Notwithstanding our endeavour to approximate potassium intake to the best of our ability, exact quantifcation of dietary potassium exposure is impossible (unless the child is exclusively on formula feeding) in this as in any other study  $[21]$  $[21]$ . Hidden sources (salt substitutes, additives in enhanced and processed foods which are not mentioned on food labels or changing manufacturers' recipes), lack of bioavailability data, all potentially underestimate the actual dietary potassium exposure. On the other hand, unreported cooking methods might underestimate potassium losses during food preparation and result in an overestimation [[4,](#page-7-0) [13,](#page-7-5) [21\]](#page-7-11). The assessment of dietary potassium alone cannot explain the lack of association between dietary potassium and serum potassium, since adults with CKD and on HD report potassium and fber intakes below those of non-CKD controls, as well as in children with CKD [[6,](#page-7-14) [9](#page-7-1), [34](#page-7-24), [35](#page-7-25)]. In addition, fber intake was not associated with serum potassium either. Nonetheless, opinion-based dietary potassium restriction is still widely recommended in CKD patients with or at risk of hyperkalemia [[1,](#page-6-0) [4,](#page-7-0) [36](#page-7-26)]. Data and formal recommendations for potassium and fber requirements in children with CKD are lacking [[1](#page-6-0), [21](#page-7-11)]. However, in a recently published clinical practice guideline, the Pediatric Renal Nutrition Taskforce advises not to modify dietary potassium intake, unless the child exhibits dyskalemia [[21\]](#page-7-11).

Emerging evidence in adults with CKD points to a relationship between higher consumption of plant foods, a delay in CKD progression and lower cardiovascular mortality [\[15](#page-7-27), [16,](#page-7-6) [37](#page-8-0)]. Three seminal experimental trials showed that in adult patients with CKD and metabolic acidosis, increased fruit and vegetable consumption was associated with improved cardiovascular risk factors, without any repercussions on serum potassium [\[38](#page-8-1)–[40\]](#page-8-2). Subsequently, whole-diet approaches and plant-based dietary patterns have gained popularity [[41](#page-8-3)], since the traditional attempt to synchronize multiple single nutrient restrictions (phosphorous, salt, potassium) in CKD patients usually results in ambiguous and complicated nutritional messages and compromises overall diet quality [\[20,](#page-7-10) [42\]](#page-8-4). In adults with CKD, dietary interventions have been reported as burdensome, confusing, and constraining, mostly resulting in poor compliance. Moreover, overzealous dietary restrictions and lifestyle modifcations may negatively impact on quality of life (QoL) [[2,](#page-6-1) [42](#page-8-4)[–44\]](#page-8-5). With the adherence and QoL of our patients in mind, we plead for a whole-diet rather than a traditional single-nutrient strategy, in the absence of established hyperkalemia. Dietary advice should be a concerted efort, marrying the expertise of the nephrologist to that of the dietician and requires a tailored approach as needs will likely difer per CKD stage, patient preferences, food literacy, socio-economic factors etc. A possible approach to safeguard fber intake would be to map all non-dietary causes facilitating hyperkalemia, preferentially targeting foods of low nutritional quality with a high potassium content, avoidance of potassium additives, next reviewing, and educating on cooking procedures to lower potassium content (e.g., boiling and shredding) and prioritizing plant foods with a low potassium to fiber ratio  $[20, 21]$  $[20, 21]$  $[20, 21]$  $[20, 21]$  $[20, 21]$ .

This study has some limitations. Inherent reporting inaccuracies of food records and dietary recalls cannot be ruled out. However, dietary recalls allow us to (partially) account for preparation methods and have a lesser recall bias compared to food frequency questionnaires [[4](#page-7-0)]. Ideally, dietary assessment should be complemented by multiple 24-h urinary potassium excretion profles, (since using a monospot is a poor surrogate due to the circadian aldosterone secretion), but this is burdensome and impractical, especially in non-potty-trained children. We report data from a single country and therefore geographic, racial, and cultural diferences in eating pattern should be kept in mind. Causality assessment of these observational fndings requires interventional trials. We believe that it is necessary to approach and analyze non-dialysis and dialysis populations separately in light of the great diferences in potassium handling, comorbidities, frailty, and possible differing effects of dietary patterns  $[4, 11, 13]$  $[4, 11, 13]$  $[4, 11, 13]$  $[4, 11, 13]$  $[4, 11, 13]$  $[4, 11, 13]$ . Therefore, our observations need to be confrmed in children with CKD requiring HD.

The major strength of the present study includes its originality, as it is the frst to demonstrate the absence of an association between dietary potassium or fber intake and circulating potassium in pediatric patients at diferent stages of (non-dialysis) CKD. Repeated measurements of dietary intakes as well as serum potassium levels over a 2-year period allowed us to account for possible intrapatient variability and seasonal variation in dietary intake. In light of the scarcely available pediatric data, our fndings are useful from an epidemiological point of view and provide insight into the controversial potassium management in pediatric CKD. Our observations fuel the discussion whether pre-emptive (in the absence of clinical signs or lab fndings) restriction of fruits and vegetables is truly efective in preventing hyperkalemia [[6](#page-7-14)]. These fndings should raise awareness among clinicians and make them critically refect whether the widely implemented potassium restriction is fully justifed, since it may deprive patients of other potential health benefts of plant-based dietary patterns. They further underscore the need for and importance of interventional studies examining the efect of plant-based diets on relevant outcomes in addition to its safety and efficacy in pediatric CKD, especially in advanced stages.

In conclusion, dietary potassium and fber intake are closely related but were not associated with serum potassium levels in pediatric non-dialysis CKD. Excessive dietary potassium restriction may deprive patients of other potential benefcial efects of plant-based dietary patterns and deserves reconsideration. Therefore, if downward adjustments of potassium intake are deemed necessary, the focus should shift from mere potassium content to potassium sources to safeguard fiber intake.

**Supplementary Information** The online version contains supplementary material available at<https://doi.org/10.1007/s00467-021-05365-5>.

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**Author contribution** Conceptualization, A.E.A., E.S., S.E., A.R., J.V.W., G.G. and W.V.B.; methodology, A.E.A., E.S., G.G., S.E. and A.R.; formal analysis, A.E.A., K.D., A.F., C.V.M. and E.S.; investigation, A.E.A., A.F., C.V.M., E.S., K.V.H.; resources, A.R., J.V.W., K.V.H., G.G. and S.E.; data curation, E.S., A.F., C.V.M., A.E.A.; writing—original draft preparation, A.E.A.; writing—review and editing, A.E.A., K.D., E.S., S.E., A.R., J.V.W., G.G. and W.V.B.; visualization, A.E.A., E.S., A.R., G.G. and W.V.B. and S.E.; supervision, S.E.; project administration, A.R. and S.E.; funding acquisition, A.R. and S.E. All authors have read and agreed to the published version of the manuscript.

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**Data availability** The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

**Code availability** Not applicable.

## **Declarations**

**Ethics approval** Approval was obtained from the ethics committee of Ghent University.

**Consent to participate** Written informed consent was obtained from the parents and children above the age of 12 years.

**Consent for publication** Not applicable.

**Conflict of interest** J.V.W. received lecture fees from Vitafo and is member of the European Society for Pediatric Nephrology (ESPN) nutritional task force (with Vitaflo grant). The other authors have no conficts of interest to declare.

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