Dietary Phosphate Consumption in Australians With Stages 3b and 4 Chronic Kidney Disease

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ABSTRACT

Objective: Dietary phosphate modification is a common therapy to treat hyperphosphatemia in individuals with chronic kidney disease (CKD). However, current dietary intake and common food sources of phosphate typically consumed by individuals with CKD are not well characterised. This study examined a cohort of CKD patients to determine total dietary intake and common food sources of phosphate, including phosphate additives.

Design and Methods: Participants with CKD stages 3b and 4 recruited to a sub-study of the "IMPROVE-CKD (IMpact of Phosphate Reduction On Vascular End-points in Chronic Kidney Disease) Study" completed a 7-day self-administered diet record at baseline. Diet histories were analysed and daily phosphate intakes determined using FoodWorks V.9 (Xyris). The proportion of phosphate contributed by each food group was determined using the AUSNUT 2011–2013 Food Classification System. Ingredient lists of packaged food items consumed were reviewed to determine frequency of phosphate-based additives.

Results: Ninety participants (mean eGFR 26.5ml/min/1.73m^2) completed this sub-study. Mean phosphate intake of participants was 1544±347 mg/day, with 96% of individuals exceeding the recommended daily intake of phosphate (1000mg/day). The highest sources of dietary phosphate were milk-based products (25%) and meat and poultry products/dishes (25%). Phosphate-based food additives were identified in 39% (n=331/845) of packaged foods consumed by participants.

Conclusion: Dietary phosphate intakes of Australians with CKD are high and come from a variety of sources. Managing dietary phosphate intake requires a patient-centered, tailored approach with an emphasis on maintaining nutritional adequacy and awareness of phosphate additives.

Key words: Chronic kidney disease, dietary phosphate, renal nutrition, phosphate additives.

INTRODUCTION

Chronic kidney disease (CKD) is an increasingly common problem, affecting approximately 10% of people worldwide.[1] CKD is associated with a high burden of cardiovascular disease (CVD), and increased mortality as individuals transition from early stages of CKD through to dialysis.[2, 3] Abnormalities of bone and mineral metabolism including hyperphosphatemia and hyperparathyroidism are closely associated with this increased cardiovascular burden,[4] as highlighted within the entity termed 'CKD - mineral and bone disorder (MBD).[5, 6]

Recommendations for the management of biochemical abnormalities of CKD-MBD relate to management of hyperphosphatemia and hyperparathyroidism.[7] Positive associations between serum phosphate and cardiovascular morbidity and mortality in both the general and CKD populations have resulted in clinical guidelines suggesting serum phosphate be targeted towards the normal range, although few randomized and placebo-controlled studies have addressed clinical outcomes using interventions to improve phosphate control.[7] Management of hyperphosphatemia has traditionally been characterised by two complementary strategies – oral phosphate binders to reduce absorption of dietary phosphate and dietary phosphate modification.

Dietary phosphate modification, with advice provided by renal dietitians, is recommended by international guidelines for patients with CKD. The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKFKDOQI) 2010 Guidelines recommended adults with stages 3-5 CKD should restrict daily phosphate intake to 800-1000 mg.[8] Similarly the 2017 Kidney Disease: Improving Global Outcomes (KDIGO) Guidelines, with level 2D evidence based on a few randomised controlled trials,[7] recommends adults with CKD should

limit dietary phosphate intake and consider phosphate source (e.g., animal, vegetable, additives) to assist with treatment of hyperphosphatemia. The source of dietary phosphate has a considerable impact on phosphate balance. Intestinal absorption is lower for phosphate of plant origin (~40%), compared to phosphate of animal origin (50-60%), whereas phosphate food additives may be 90-100% absorbed.[9] Furthermore, food additives are not accounted for in regular food composition tables and are known to increase the phosphate content of foods by 60-70%.[10, 11]

Although associations between high serum phosphate and adverse health outcomes such as mortality and CVD in individuals with CKD have been reported,[2, 12] whether dietary phosphate modification directly improves adverse patient outcomes remains unclear.[13] Despite this, there is general agreement that dietary phosphate modification is an important and inexpensive therapy to manage hyperphosphatemia in individuals with CKD.[7]

In Australia, it is currently recommended that individuals with stage 3 CKD see a renal dietitian every 6-12 months and those with stages 4 and 5 CKD every 1-3 months.[14] Despite these recommendations, local survey data suggests they are inequalities in service provision across Australian health services resulting in a number of patients not receiving expert nutrition advice[15] and instead possibly relying on other sources of dietary information to manage their CKD and phosphate levels.

Currently, dietary intake and common food sources of phosphate typically consumed by individuals with CKD is largely unknown. A better understanding of dietary phosphate consumption in individuals with CKD is necessary to facilitate more effective dietary modification and may contribute to better phosphate management. This study aimed to

examine a cohort of CKD patients to determine their dietary phosphate consumption, and the food sources, including additives, from which their phosphate intake was derived.

METHODS

Study Design

This cross-sectional study is a sub-study of the randomised controlled trial 'IMpact of Phosphate Reduction On Vascular End-points in Chronic Kidney Disease (IMPROVE-CKD).[16] The primary objective of the IMPROVE-CKD study is to determine if the phosphate binder lanthanum carbonate reduces the burden of CVD in patients with CKD stages 3b and 4 when compared to placebo. The primary end-point of the study is change in arterial compliance measured by pulse wave velocity over a 96-week period. Secondary outcomes include change in aortic calcification and biochemical parameters of serum phosphate, parathyroid hormone and fibroblast growth factor 23 (FGF-23) levels. The trial is registered with the Australia and New Zealand Clinical Trial Register (<trial number blinded>) and ethical approval for the IMPROVE-CKD trial was obtained by each local Institutional Ethics Committee for participating sites prior to commencement. Ethical approval for the dietary sub-study was obtained through the <Blinded> Human Research Ethics Committee and participants provided additional informed consent to participate in the sub-study.

Participants

Recruitment for the IMPROVE-CKD dietary sub-study occurred at eight different sites across Australia. Participants were eligible to participate if they were ≥18 years, had CKD stages 3b or 4 (estimated glomerular filtration rate [eGFR] 15-44 ml/min/1.73m^2), had a serum phosphate ≥3.10 mg/dL (≥1.00 mmol/L) on at least one occasion over the previous six months, and could give informed consent. Participants were excluded according to the

following criteria: medical conditions impacting calcium and phosphate metabolism other than CKD (e.g. primary hyper- or hypo-parathyroidism, gastrointestinal malabsorption disorders, liver dysfunction); kidney transplantation; presence of atrial fibrillation; and inability to obtain a pulse wave velocity measurement. The protocol for the IMPROVE-CKD study has previously been published.[16]

Data Collection

Participants recruited to the dietary sub-study undertook an extra dietary assessment, in addition to all baseline assessments for the IMPROVE-CKD study.[16] The dietary assessment involved a seven-day self-administered diet record prior to the baseline visit.[17] The record was then verified by a trained site dietitian in a face-to-face interview at the baseline visit. Dietitians at each site used the interview-administered open-ended methodology to verify reported dietary data.[17, 18] Verification included confirmation of types, portions and brands of all foods and beverages consumed, ensuring details of whether foods and beverages consumed were homemade or bought. Intakes of vitamin and mineral supplements and salt added during cooking or at the table were not included in the analysis. Participants' dietary intakes from the dietary questionnaire were converted into daily nutrient intakes using the nutrient analysis program FoodWorks V.9 (Xyris Software [Australia] PtyLtd). All individual food and beverage items consumed including fresh, packaged, frozen, processed or bought foods and separate ingredients that made up home meals were entered into FoodWorks for analysis. If the brand item for a particular food item was not present in the database, a generic brand was used in its place. Dietary phosphate density was defined as the ratio of daily dietary phosphate intake to the total average daily calorie intake. The percentage contributed by each food group to the total phosphate intake was determined using the AUSNUT 2011–2013 Food Classification System, grouping individual food items into major, sub-major and

minor food groups.[19] To minimize inaccurate self-reporting, participants with implausible intakes of < 500 or > 3,500 kcal/day were excluded.[20] To determine the prevalence of phosphate-based food additives consumed by participants, the ingredient list of all packed foods consumed were reviewed for the presence of any of the 22 phosphate-based food additives that have been approved for use in Australia, as well as added mineral phosphate.[21] The study did not record whether participants had received previous dietetic input. Previous dietary advice to modify dietary protein intake could have influenced phosphate intake.

Statistical Analysis

All statistical analyses were performed using SPSS for Windows (version 23.0, 2006; SPSS, Chicago, Illinois). Summary statistics of continuous variables were reported as means and standard deviations. The proportion of participants with dietary phosphate intakes > 1000mg/day were expressed as the proportion exceeding the upper intake for the management of hyperphosphatemia as recommended by KDOQI.[8] The population proportion method was used to calculate the contribution of phosphate from food groups.[22]

RESULTS

Participants

A total of 278 individuals across 17 sites were recruited to the IMPROVE-CKD study. Eight of these sites took part in the dietary sub-study. Individuals recruited at these sites (n=131) were invited to participate in the dietary study, of whom 117 individuals consented to participate (response rate 89.3%). Six participants withdrew from the IMPROVE-CKD study prior to baseline appointments, 20 did not complete baseline dietary assessments and three dietary histories were excluded due to implausible energy intakes, leaving 90 participants (completion rate 76.9%). Baseline characteristics of participants are shown in Table 1. The mean age of participants in our cohort was 63.6±12.3 years and 67.8% of participants were

male. Baseline eGFR was 26.5±7.7 ml/min/1.73m^2, and mean serum phosphate was 3.94±0.62 mg/dL (1.27±0.20 mmol/L) (Table 1). Participant characteristics for the dietary study were similar to the characteristics of the complete IMPROVE-CKD cohort (n=278), except for the proportion of Caucasians, with 83% in the dietary study compared to 64% in the entire IMPROVE-CKD study. This difference may relate to the recruitment of participants from Australia alone in the dietary sub-study, in contrast to participants recruited from Australia, New Zealand and Malaysia for the whole cohort.

Dietary phosphate intake

The mean daily dietary phosphate intake of our cohort was 1544±347 mg and the mean daily dietary phosphate density was 0.81±0.16 mg/kcal. Ninety-six percent of individuals (n=86) exceeded the Australian Recommended Dietary Intake of 1000 mg/day and best practice dietary guidelines for the management of hyperphosphatemia to limit dietary intake to 800-1000 mg/day[8]. The average daily energy, protein, sodium and potassium for our cohort were 1949±413 Kcal/day (8155±1728 kJ/day), 98.7±22.6 g/day, 2487±855 mg/day, and 3279±867 mg/day respectively. The mean dietary phosphate/protein ratio of our cohort was 15.8±2.6 mg of phosphate per gram of protein.

Food sources of phosphate

Figure 1 shows the contribution of phosphate from major food groups. The highest contribution of daily dietary phosphate intakes were milk products and dishes (25%), meat and poultry products and dishes (25%), and cereals and cereal products (14%). Other important food sources included cereal-based products and dishes 8%, fish and seafood products and dishes 5% and non-alcoholic beverages 5%. Supplementary Table 1 lists the contribution of participant phosphate intakes reported by sub-major food groups. The top five

sources were 1. Dairy milk (cow, sheep, goat), 2. Beef, sheep and pork, unprocessed, 3. Poultry, 4. Regular breads, and bread rolls and 5. Cheese. Collectively, these food groups accounted for 40.5% of all phosphate consumed. Supplementary Table 2 provides information on the food sources of phosphate at the minor food group level. When foods were grouped into 'animal-' or 'plant-' based foods, 51.6% of phosphate intake came from animal-based foods, 34.0% came from plant-based foods and 14.4% came from a combination (both animal- and plant-based foods).

Phosphate additives

Participants consumed a total of 1410 different types of foods and beverages. Of these, 845 (60%) were branded packaged food products and were reviewed for the presence of phosphate additives. Phosphate-based food additives were identified in 39% (n=331/845) of packaged foods consumed by participants. There was wide variation in the presence of phosphate additives across major food groups ranging from 0-87% (see Table 2). Additives were highly prevalent and in >85% of the following food groups: dry soup mix (packet soups), sausages, frankfurts, dairy blends, margarine and table spreads, pastries, processed meat, cakes, muffins, scones, cake-type desserts, chocolate and chocolate-based confectionery, cereal bars and sweet biscuits. Twenty of the 22 phosphate-additives approved for use in Australia, and added mineral phosphate, were found on more than one occasion in packaged foods consumed by participants (Supplementary Table 3). The most commonly used additive was 322 (lecithins) found in 16.4% (n=139) of food products followed by 450 (pyrophosphates) found in 11.4% (n=96) of food products. Food items that contained phosphate additives had a median of one additive and range of 1-6 additives.

DISCUSSION

To our knowledge this is the first study to look at dietary intakes and sources of dietary phosphate in a cohort of Australians with CKD. In this observational study we report a high mean daily phosphate intake of 1545 mg/day, with 96% of individuals exceeding the daily intake of phosphate recommended by KDOQI. These results are similar to the daily phosphate intake of 1573 mg in Australian adults aged 51-70 years reported in the 2011-2012 Australian Health Survey (AHS),[23] and slightly higher than American adults aged 50-69 years as reported in the National Health and Nutrition Examination Survey (NHANES)[24] with daily intakes of 1350 mg. The average daily energy (1949 Kcal [8155 kJ]), protein (99 g), sodium (2487 mg), and potassium (3279 mg) of our cohort were also very similar to the energy (2233 Kcal [9344 kJ]), protein (98 g), sodium (2510 mg), and potassium (3144 mg) of adults aged 51-70 years reported in the AHS; suggesting our cohort's daily nutrient intakes do not differ from the general Australian population aged 51-70. In a recent observational study looking at dietary intakes of Brazilian patients with CKD (mean age 68 years, eGFR 38.4 ml/min/1.73m^2), a daily dietary phosphate intake of 1184 mg/day, protein intake of 83 g per day and energy intake of 1916 Kcal/day (8015 kJ/day) was observed,[25] with phosphate and protein intakes lower than in the present study, despite daily energy intake being comparable. Variability may partially be accounted for by geographic and cultural differences in plant-based and processed foods. Dietary education could also be a factor contributing to varying dietary phosphate intake. CKD participants often receive medical nutrition therapy (MNT) to modify phosphate or dietary protein which may also influence phosphate intake. Whilst MNT is considered important it is not always routine practice in Australia, and often on a referral or sporadic basis only until individuals are close to commencement of dialysis.[14, 15] Our sample of CKD participants were predominantly normophosphatemic and therefore may have not received dietary education. This may explain the higher levels of daily

phosphate intake observed compared to other CKD populations, and with similar intakes to the general Australian population.

In the present study we found the majority of daily phosphate consumption came from animal-based food sources with milk products, and meat and poultry products contributing to 50% of phosphate consumed. These findings are consistent with those reported in the 2011-2012 AHS, which reported these same foods contributed 41% of the daily phosphate intake of Australians aged 51-70 years.[23] Previous studies have highlighted the importance of dark coloured sodas and other soft drinks as predominant dietary sources of phosphate,[26, 27] however similar to the NHANES we found a relatively minor contribution from sodas and soft drinks, which contributed <1% of the overall phosphate intake. While cereals and grains tend to have a relatively low phosphate content, in our cohort cereals, cereal products and cereal-based dishes contributed almost a quarter of daily phosphate intake due to their high frequency of consumption making them a major source of dietary phosphate.

Whilst our data provide important information of habitual dietary phosphate intakes of individuals with CKD, the impact on total phosphate absorption and overall phosphate balance cannot be accurately determined from these results. Plasma phosphate balance involves the combined effects of dietary intake, intestinal absorption, excretion, and the movement between sites of body storage. Phosphate bioavailability varies between dietary sources. The modern diet often contains high levels of phosphate in the form of preservatives and additive salts that are readily absorbed from the intestine, whereas phosphates in plants are often complexed in the form of phytates and have lower bioavailability than phosphate from animal products.[9-11] Given the considerable impact phosphate source can have on

intestinal phosphate absorption, better methods for assessment of dietary phosphate intake and its impact on overall phosphate balance are required.

Our study found that 39% of packaged food items consumed contained phosphate additives and these additives were most commonly found in processed meats (such as sausages), packaged ready-made soup mixes, margarines, processed cereals, cakes, biscuits, chocolate and confectionary items. These findings are similar to those reported by McCutcheon et al.[28] who identified the prevalence of phosphate-based additives in the Australian food supply to be 44% and Leon et al who found that 44% of the bestselling grocery items in northeast Ohio contained phosphate additives.[29] Our results confirm that phosphate additives are also commonly consumed by individuals with CKD.

Despite the high prevalence and bioavailability of phosphate additives in the food supply, there are currently no accurate ways to determine the amount of phosphate contributed by these additives in processed and packaged foods. Current Australian legislation does mandate the reporting of phosphate-based additives on nutritional labelling of food products but does not require food manufacturers to list the total phosphate content. Whilst this may help consumers identify whether a food item does contain phosphate additives (assuming that they can accurately recall the 22 phosphate-additives approved for use in Australia), it does not provide guidance to the quantity of phosphate, and consumers are likely to underestimate the total phosphate of these food sources, and in particular, the degree to which these foods will contribute to their overall phosphate balance. This lack of regulation makes following a lower phosphate nutrition plan much more challenging. The frequent use and high bioavailability of phosphate additives in association with our findings support the mandatory inclusion of

phosphate content on food labels, so that consumers and clinicians have a greater ability to identify phosphate additives.

Modification of dietary phosphate has traditionally focused on targeting foods naturally high in organic forms of phosphate such as meat, fish, dairy, legumes, nuts and grains. However, applying such dietary phosphate modifications may lead to reductions in dietary protein intake, and overall nutrition quality, and without close dietetic monitoring, can increase the risk of protein-energy wasting.[30] For patients with CKD, any reduction of dietary phosphate must be balanced against the maintenance of adequate dietary protein, energy and diet quality, and consideration must be given to including patient perspectives regarding dietary strategies that assist in managing phosphate balance.

High serum phosphate levels are associated with mortality in both healthy population and patients with CKD.[31] However, the association between dietary phosphate intake and cardiovascular mortality is less clear. A recent Cochrane review, with overall low quality and limited evidence, did not identify a significant change in mortality associated with dietary phosphate modification.[32] Another proposed benefit of dietary phosphate modification is to alleviate the dramatic rises in FGF-23 that occur as CKD progresses. The few dietary phosphate modification studies with a focus on changes in FGF-23 have produced differing results. Isakova *et al* showed no change in FGF-23 with dietary phosphate modification, despite a change in urinary phosphate,[33] whereas Di Iorio et al reported a reduction in FGF-23 following treatment with a very low phosphate and protein diet.[34] Moe et al examined the short term effects of vegetarian and meat diets on changes in FGF-23 in a cross-over trial in eight participants with CKD stages 3 and 4.[9] That study reported that following a vegetarian diet for one week led to lower serum phosphate levels and decreased FGF-23. These results

suggest that plant-based nutrition plans may be beneficial for phosphate balance in individuals with CKD, and add to a growing body of evidence suggesting possible benefits of healthy higher plant-based eating patterns such as diets rich in fruits and vegetables on improving kidney-related outcomes, delaying disease progression, and potentially lowering mortality risk in CKD populations.[35-39] Additional studies are needed to assess whether changes in phosphate balance result in improvements in CVD and mortality over the longer term, whether individuals can maintain nutrition adequacy, and whether benefits are observed in patients across all CKD stages.

Based on available evidence, the latest international KDIGO guidelines and the KDOQI commentary suggest dietary phosphate modification could be used as an adjunct rather than a primary intervention in patients with CKD-MBD to manage hyperphosphatemia; and have moved away from recommending a daily phosphate restriction. Our findings support the recommendation to encourage a reduction in consumption of phosphate additives and towards eating a higher proportion of plant-based meals with lower inorganic phosphate, whilst maintaining adequate dietary protein.

Our study highlights the potential contribution of various dietary substrates to phosphate balance in Australians with CKD and has important implications for clinical practice. Limitations of our study include difficulties in estimating dietary phosphate intake from participants. Missing data and incomplete questionnaires may also have contributed to inaccurate estimations of dietary phosphate. Therefore, total dietary phosphate intake measured in our cohort may be underestimated. Selection bias may have been another issue, given the opt-in nature of the additional dietary sub-study. Inclusion criteria for the IMPROVE-CKD study involved participants with serum phosphate ≥3.10 mg/dL (≥1.0

mmol/L) which may have selected patients with higher dietary phosphate intakes. Sodium intake may be underestimated as participants were not asked about salt added during cooking or at the table. Additionally, dietitian involvement with participants prior to participation in the study was not known and our results are not necessarily generalizable to patients with more advanced CKD who may have hyperphosphatemia or who have been advised to restrict daily protein.

In summary, high levels of dietary phosphate intake were reported in this sub-study cohort of the IMPROVE-CKD study, with the majority of phosphate intake from animal products and additives. Dietary phosphate intake was significantly above that which is recommended for patients with CKD, despite dietary phosphate modification considered part of the appropriate management of CKD-MBD. Overall, this gives impetus for more prospective studies assessing dietary phosphate intake and its impact on clinical outcomes, although several studies have previously addressed this area as summarized in a recent review article,[13] and further analyses of dietary phosphate intake in association with markers of mineral metabolism would also be important. Future studies exploring nutrition practices in the management of CKD in Australia would be useful to describe the current level of care, identify gaps in practice and to support changes to protocols for treatment.

Practical Application

Phosphate intakes of Australian CKD patients are high and come from a wide range of animal and plant food sources. We recommend individuals with CKD stages 3b-4 should see a specialist dietitian trained in renal nutrition for a patient-centred, tailored nutrition approach to managing phosphate; which emphasizes maintaining nutritional adequacy and awareness of phosphate additives.

Conflict of interest statement

The results presented in this paper have not been published previously in whole or part, except in abstract format.

Data Capture

Study data were collected and managed using REDCap electronic data capture tools hosted at < location blinded >[1,2]. REDCap (Research Electronic Data Capture) is a secure, web-based software platform designed to support data capture for research studies, providing 1) an intuitive interface for validated data capture; 2) audit trails for tracking data manipulation and export procedures; 3) automated export procedures for seamless data downloads to common statistical packages; and 4) procedures for data integration and interoperability with external sources.

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FIGURE LENGEND

Figure 1. Daily contribution (%) of phosphate from major food groups among participants (n=90), which includes major food groups providing ≥1% of daily intake of phosphate. Foods groups ranked in order of greatest contributor to phosphate intake.