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Management of Natural and Added Dietary Phosphorus Burden in Kidney Disease

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Abstract

Phosphorus retention occurs from higher dietary phosphorus intake relative to its renal excretion or dialysis removal. In the gastrointestinal tract the naturally existing organic phosphorus is only partially (~60%) absorbable; however, this absorption varies widely and is lower for plant-based phosphorus including phytate (<40%) and higher for foods enhanced with inorganic-phosphorus-containing preservatives (>80%). The latter phosphorus often remains unrecognized by patients and health care professionals, even though it is widely used in contemporary diets, in particular low-cost foods. In a non-enhanced mixed diet, the digestible phosphorus is closely correlated with total protein content, making protein-rich foods a main source of natural phosphorus. Phosphorus burden is more appropriately limited in pre-dialysis patients who are on low protein diets (~0.6 g/kg/day), whereas dialysis patients who require higher protein intake (~1.2 g/kg/day) are subject to a higher dietary phosphorus load. An effective and patient-friendly approach to reduce phosphorus intake without depriving patients of adequate proteins is to educate patients to avoid foods with high phosphorus relative to protein such as egg yolk and those with high amounts of phosphorus-based preservatives such as certain soft drinks and enhanced cheese and meat. Protein-rich foods should be prepared by boiling, which reduces phosphorus as well as sodium and potassium content, or by other types of cooking induced demineralization. The dose of phosphorus-binding therapy should be adjusted separately for the amount and absorbability of phosphorus in each meal. Dietician counselling to address the foregoing aspects of dietary phosphorus management is instrumental for achieving reduction of phosphorus load.

Keywords

Dietary phosphorus; preservatives; enhanced food; digestibility; phosphorus-binder; cooking; food processing; Chronic Kidney Disease

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Introduction

An important aspect of the management of chronic kidney disease (CKD) patients is the mineral and bone disorders (MBD), the surrogates of which are predictors of clinical outcomes, including higher risk of vascular calcification and mortality [1–3]. This is clearly evident in dialysis patients, where hyperphosphatemia is associated with increased cardiovascular morbidity and mortality.[4] Phosphorus retention happens early in CKD and worsens as CKD progresses. In a recent study, Zoccali et al [5] reported that in patients with proteinuric nephropathies serum phosphorus is an independent risk factor for renal disease progression and may limit or even blunt the renoprotective effect of ACE inhibitor therapy. In this article we review the pathophysiology of CKD-MBD as it pertains to dietary phosphorus load and examine different types of phosphorus in food.

Early Phosphorus Retention in CKD

The pathogenesis of CKD-MBD is multifactorial and, in addition to hyperphosphatemia, includes changes in calcium, calcitriol, PTH and FGF-23.[1] However, the tendency to phosphorus retention, based on an excessive dietary intake relative to residual renal function, plays a central role [6–10]. Higher dietary phosphorus load may inhibit the renal 1α-hydroxylase directly and also indirectly through the increase in FGF-23; the decline in the circulating levels of natural calcitriol leads to relative hypocalcemia and stimulates the synthesis and secretion of PTH. In addition, evidence suggests that the increase in circulating phosphorus level, irrespective of changes in serum calcium and calcitriol, can enhance the synthesis and secretion of PTH leading to hyperplasia of the parathyroid glands. The recently discovered FGF-23 axis also affects calcium-phosphorus metabolism leading to even more complex interactions between the above-mentioned pathways. However, a key and early role player in CKD-MBD is still believed to be the retention of phosphorus.[10–11] It is now evident that FGF-23 increases in early stages of CKD especially in response to dietary phosphorus load leading to higher fractional excretion of phosphorus via kidneys; hence, an apparent “normophosphatemia” ensues at the expense of heightened FGF-23 pathways. The foregoing mechanisms explain the need for early dietary control of phosphorus load [12]. In addition to these renal and endocrine pathways, a gut-kidney axis may contribute to short-term regulation of phosphorus by “sensing” the load of intestinal phosphate, which induces phosphaturia even before an increase in circulating phosphorus occurs [13]. Hence, hyperphosphatemia, which is quite common in well-nourished dialysis patients and which is a marker for dietary phosphorus load in these patients, may not be evident in earlier stages of CKD, where increased renal fractional excretion of phosphorus through increased FGF23 and PTH plays an important role. Combined assessment of serum phosphorus and FGF-23 levels and fractional excretion of phosphorus may become the future approach in therapeutic decision making for non-dialysis dependent CKD patients [14,15].

Dietary Phosphorus

Phosphorus (P) is a non-metal element with atomic weight of 31 Daltons. It is widely present in nature as phosphate (PO_4). Phosphorus is also an essential constituent of all living

organism, as it plays a role in many fundamental biological and enzymatic reactions to maintain life due to its high chemical reactivity. Most of the total body phosphorus, i.e., about 80%, is found in bones and teeth in form of calcium salts, whereas about 20% is present in soft tissues and body fluids, where it is in the form “organic” phosphorus and as a constituent of nucleic acids, phospholipids and phosphoproteins, as well as such energy modulating molecules as ATP, AMP, and GMP. The “inorganic” phosphates in the skeletal system serves as the buffering system to not only maintain the acid–base homeostasis but also to harbor phosphorus reserves.

Given the wide-spread presence of phosphorus in animal and plant based foods, the daily dietary requirement for phosphorus is generally adequately satisfied. The recommended intake of phosphorus is approx. 800 mg for adults and 1000 to 1200 mg for adolescents and pregnant or breast-feeding women. In the natural food the main source of phosphorus is from protein, whereas some degree of phosphorus absorption also happens from non-protein based phosphorus such as nucleic acids and, of a lesser degree, in form of phytates in plant foods (see below).

Circulating Level of Phosphorus

In plasma, the normal level of phosphorus is 2.5–4.5 mg/dL. Under normal physiologic conditions with an extracellular fluid pH of 7.4, hydrogen ion is bound to phosphate either as one (HPO_4^{2-}) or di-hydrogen structure ($H_2PO_4^-$) at a 4:1 ratio, which explains why the average phosphate valence is 1.8 in body fluids. Serum phosphorus measurement usually yields same concentration as plasma. Serum phosphorus level remains within normal range until the late stages of CKD; therefore, as discussed above, lack of hyperphosphatemia must not be interpreted as normal renal function [16]. Instead, even a slight increase in serum phosphorus levels towards borderline high levels but still within the “normal range” may suggest phosphorus retention in early stages of CKD. This trend may happen when glomerular filtration rate falls below 50 ml/min per 1.73 m^2 and may be accompanied by the increase in PTH and FGF-23 as described above [17–19].

Phosphorus Digestibility and Intestinal Absorption

The absorption of phosphate in the intestinal epithelial cells occurs via a co-transport mechanism through active sodium/phosphate (Na^+/P_i) co-transporters, which involves at least three different types of Na^+/P_i, i.e., NPT2a, b and c. This mechanism can be inhibited by nicotinamide, so that the administration of niacin may be used as an effective approach to reduce the intestinal absorption of phosphorus and to lower circulating phosphorus level [20]. Upon intake of natural (non-enhanced) food, approximately 60% of the dietary phosphorus is absorbed in the intestine as phosphate. This proportion may increases to 80% when the circulating level of calcitriol increases. In normal adults, dietary phosphorus load is counterbalanced by a phosphorus excretion in urine that equals its net intestinal absorption. The so-called phosphatonines, in particular FGF-23 as described above, reduce the expression of co-transporters NPT2a, b and c but, but unlike PTH they also inhibits the renal 1-alpha hydroxylase leading to a reduction in the in-vivo synthesis of calcitriol by the kidneys [21]. Hence, the net phosphorus load from food intake is a function of phosphorus

content of the ingested food, phosphorus bioavailability from different sources of diet, the food preparation modality (see below), and of vitamin D status.

Phosphorus from unprocessed food

In natural foods phosphorus is present both as inorganic ions and as constituent of phosphoproteins but also as membrane phospholipids, ATP, ADP, DNA, and RNA. In a mixed diet, the phosphorus content is usually proportional to the amount of protein content. Indeed protein-rich foods are historically and naturally the main source of dietary phosphorus. Several equations have been advanced to estimate the content of phosphorus based on the amount of protein of a mixed diet, including the following by Boaz and Smetana [22]:

$$\text{Dietary phosphorus (mg)} = 128 + 14 \times \text{protein (g)}$$

Another similar equation was recently developed in a dietary assessment study of the US American dialysis patients by Kalantar-Zadeh et al [26]:

$$\text{Dietary phosphorus (mg)} = 78 + 11.8 \times \text{protein (g)}$$

Finally, in a cohort of 260 Italian subjects, the following equation has recently been developed by Cupisti et al. [23]:

$$\text{Dietary phosphorus (mg)} = 102 + 12.9 \times \text{protein (g)}$$

However, it is important to note that these equations should be used for a typical meal intake consisting of different types of food and not for a single type of food exclusively. This relatively constant relationship between the content of phosphorus and protein in a mixed diet can be significantly different by a deliberate choice and food restricted to certain nutrients, e.g. mostly egg whites as will be discussed later.

Table 1 provides an overview of the content of phosphorus in major foods. Most foods that are marked with no added phosphorus are supposed to be natural and raw. The highest concentrations of phosphorus are found in cereal grains (120–360 mg/100 g), cheese (220–700 mg/100 g), egg yolk (586 mg/100 g) and legumes (300–590 mg/100 g). The phosphorus content in the flesh and meat can vary from 170 to 290 mg/100 g, while in fish it varies from 190 to 290 mg/100 g, as the natural type of inorganic phosphorus is contained in phosphoproteins and phospholipids and can be up to 40% of the total natural dietary phosphorus [24]. Phosphopeptides usually contain clusters of phosphoserines, which can effectively bind calcium and iron. They inhibit the formation of insoluble calcium phosphates or iron complexes. They are present in milk protein casein but more abundantly in egg yolk [25].

The proportion of phosphorus which is absorbed throughout the gastrointestinal tract is 60% of the ingested phosphorus on average. The bioavailability of phosphorus varies widely,

however, depending on level of 1,25 (OH)2 vitamin D and activation of the intestinal vitamin D receptors as well as the degree of digestibility of the food and its phosphorus, and the presence of compounds that can bind to phosphorus by inhibiting the gastrointestinal absorption (e.g. calcium, magnesium, aluminum)[26]. Phosphorus in animal based foods such as meat or fish is more readily bioavailable than that found in foods of plant origin such as legumes (Table 1). The former is present as inorganic salts or as part of organic compounds, which are cleaved by hydrolases in the intestinal tract releasing inorganic phosphorus upon absorption. The latter phosphorus is largely in the form of phytate [27]. Phytates are found mainly in cereals and legumes, where they are concentrated in the seeds and fibrous parts. Hence, they are abundant in whole grain products, and virtually absent in refined products. In monogastric animals, including humans, the enzyme phytase is not expressed; therefore phytate degradation occurs only partially, if at all, by the intestinal bacterial flora or to some extent by non-enzymatic hydrolysis reaction [27]. This results in lower bioavailability of dietary phosphorus of plant origin, which remains below 50% (usually 30% to 40%), which is in contrast to the digestible proportion of meat, chicken or fish, which can be as high as 60% to 80%. Interestingly, according to some experts phytates are considered as the so-called “anti-nutritional factors” due to their ability to bind divalent cations and thus chelating and preventing their absorption. This addresses the agro-food research towards addition of the enzyme phytase in products intended for human or animal breeding with the aim of reducing the chelating action and at the same time increasing the bioavailability of phosphorus. Indeed soaking and extraction in aqueous solutions are able to remove some phytic acid through the activation of plant endogenous phytase [28]. This is the area of major knowledge gap in the field of renal nutrition as it pertains to dietary phosphorus management. Hence, it is possible that the traditional approach to CKD patients by restricting their intake of legumes (beans, peas, lentils, etc.), nuts, seeds and chocolate is indeed of no major relevance. A recent study by Moe et al [29] showed that, provided the same amount of dietary phosphorus content, one week of a vegetarian diet led to lower serum phosphorus levels and decreased FGF-23 when compared with a meat-containing diet. Hence, not only the total amount but also the plant-vs.-animal type of dietary phosphorus intake plays a significant role in phosphorus management of CKD patients [29].

In the field of dietetics, various procedures have been implemented such as the addition of phytase or phosphatase enzymes of plant origin to the whole wheat flour, or inducing the activation of endogenous phytase by prolonged soaking and germination of wheat. All these procedures, promoting the degradation of phytic acid, improve the nutritional level of the food in general, but not for patients with kidney disease, who need to limit the dietary phosphorus load and its digestibility. Some studies have examined the ability of several bacteria in degrading phytic acid with the aim to improve mineral availability and to provide nutritive benefits to the consumer [30–31]. In livestock and agriculture related nutrition, bacteria are often added to mixture of cereals to degrade phytic acid in order to increase phosphorus bioavailability and, hence, the so-called “nutritional value” of vegetable foods, but this approach could pose a significant risk to CKD patients as the degradation of phytic acid produces free phosphorus that can be more readily absorbed. Similar concerns may exists with probiotics that may enhance phosphorus availability in gut.

Fruits and vegetables generally contain small amounts of phosphorus, but this is not true for certain plant seeds, nuts and legumes. Chocolate is another food that is exceptionally rich in phosphorus with an average content ranging from 355 to 540 mg per 100 g. The dark chocolate is rich of phytates, hence, given low bioavailability of this type of phosphorus the apparent high phosphorus content of chocolate should not cause a real dietary burden, which is in sharp contradistinction to the traditional dietary recommendations to CKD patients. On the other hand, in phosphate-bodied milk that is added to some types of chocolate a much higher and more readily absorbable phosphorus may exist, which is mainly in form of phospholipids (lisofosfaditilcholine, fosfaditilcholine, fosfaditil ethanolamine and fosfaditil-inositol).

The direct correlation between the content of phosphorus and proteins of raw foods, as shown by equations developed by Boaz et al [22], Kalantar-Zadeh et al [26] and Cupisti et al [23] can also be taken advantage of for dietary management of pre-dialysis CKD patient, in whom a low protein diet is usually recommended [32]; however, high protein diet represent a serious obstacle to the patient on dialysis who would otherwise limit their intake of phosphorus in the face of an increased protein requirement [33,34]. Since dialysis patients need to limit the intake of dietary phosphorus while maintaining high protein, the choice of food should fall back on foods with a lower amount of phosphorus per gram of proteins [35] and lower phosphorus digestibility [36]. The use of food composition tables that show the amount of phosphorus per 100 g of food or even more importantly per each gram of protein may be of great benefit for both the patient and the health care providers including dietitians and nephrologists [26, 35].

A typical example of differential phosphorus to protein ratio is the egg and its components:, egg is rich in protein and in phosphorus. However, yolk contains most of the phosphorus, largely as phospholipids, with some amount of phosphoproteins. Indeed egg yolk based phosvitin is known as the most phosphorylated protein found in nature and contains a much greater proportion of phosphates in the molecule than casein [25]. Instead, the egg white contains higher portion of egg proteins (3.7 g per egg white) with an extremely low amount of phosphorus and virtually no cholesterol. Hence, the implementation of recipes involving egg whites allows an increase in intake of protein with a negligible supply of phosphorus. Indeed a recent study by Taylor et al.[37] found that substituting one meal per day with pasteurized egg white lowered serum phosphorus while serum albumin tended to increase in dialysis patients.

Changes of Phosphorus Intake by Industrial Processing: Food Preservatives

In modern society food and beverages are often consumed a long time after their production or in places far away from the production site. Notwithstanding these facts, the dietary product must satisfy safety guidelines and taste characteristics. This is why the food industry uses progressively more “food additives”, also known as “preservatives”, i.e., substances intentionally added to food for dietetic-preservative and financial purposes (Table 2). A food additive is a substance not normally consumed as a food in itself and not naturally present as

an inherent ingredient of food. It is intentionally added to the enhanced food during production, processing, preparation, packing, transport or storage.

In the past three decades, the use of food preservatives has been increasingly expanded, although the use of additives has its origins in ancient times. Salting of meat and fish, adding lemon juice to fruit and vegetables to prevent browning, use of vinegar in the preparation of canned vegetables, addition of saltpeter in meat sausage, and the sulfidation of musts and wines are forms of additives derived from tradition and experience but not science, albeit not without any risk. The exponentially increased addition of preservatives to food is a consequence of modern changes in the cycle of food production, distribution and consumption. Currently used additives and preservatives are substances widely studied and documented with rather extensive toxicological and dietetic profiles, and their use is constantly under the control of national and international authorities within each jurisdiction, although to date no globally effective regulation appears to exist.

Phosphorus is the main component of most additives and is usually in form of phosphoric acid, phosphates or polyphosphates in the processed foods. In the European Community, the phosphorus-containing additives are used as acidity regulators (E338-E343), and as emulsifiers and thickeners (E442, E450-E452, E544-E545) (Table 2). The food industry uses them in food processing for a variety of reasons, i.e., to extend conservation, enhance color, improve flavor and retain moisture. No limit is given with regards to the amount of phosphorus containing-preservatives; currently only a “technological” limit of 5 grams (as P_2O_5) per kg of food exists. In other words, higher than 5 g concentrations are not useful and thus are usually not used, but one cannot exclude the possibility that higher concentrations may exist in certain types of food products.

The European Community (EC) Regulations obligate producers to report the presence of phosphates or polyphosphates on the food label, but the amount is not required. In addition, food labeling reports the preservatives as full name or initials, the so-called “E” series. Table 2 lists some of the most widely used additives containing phosphorus and the corresponding initials in the European Union countries. It is easy to appreciate that subjects who are unaccustomed to paying attention to the food composition labels can hardly avoid the extra phosphorus load coming from processed products.

Preservatives are largely used in meat products (e.g. chicken nuggets and cooked hotdogs), processed cheese spreads, pasta, or cooked and frozen dishes, puddings, sauces, bakery products, partially cooked and frozen foods, and soft drinks and beverages. Phosphorus is abundantly present as inorganic salts in these additives and thus more easily absorbed in the intestine 90% or higher rate [26, 29]. The major public health implications arising from these considerations is that the load of phosphorus derived from inorganic phosphorus in food additives is disproportionately high compared to phosphorus naturally present in non-processed foods. In the United States the dietary phosphorus burden from phosphorus-containing preservatives has increased dramatically from an average of 470 mg/day in 1990’s to over 1000 mg/day for a typical American diet in recent years [38].

In the United States, but not in Europe, the addition of preservatives even to the fresh meat is allowed, in that the meat is literally injected with solutions containing water, salt, sodium phosphate and other natural flavors, in order to improve its appearance, flavor and tenderness, and to extend shelf life [38]. Most beverages contain little to no protein and, hence, any phosphate content is almost entirely from additives. As a consequence, patients who consume beverages with high phosphate content had serum phosphate levels that are often quite high whereas their nutritional status may be inferior [9]. Enhanced food with additives such as many fast food items are generally of lower quality and lower cost compared to the fresh products with no additives. Hence, poorer segments of the population are at greater risk of excessive dietary phosphorus exposure that is even more easily absorbable. Gutierrez et al [39] demonstrated that low socioeconomic status was associated with higher serum phosphate concentrations, despite a lower protein intake irrespective of race. A likely explanation was that low income patients usually eat relatively inexpensive processed and fast foods that are enriched with highly absorbable phosphorus additives.

With the exception of a recent study that has introduced a novel method to measure differential dietary phosphorus [24], to date there is no other accurate and reproducible method for distinguishing the natural phosphorus content of a food from the phosphorus added as preservative. It must be taken into account, however, that all the polyphosphates added to a food over time degrade to phosphate ions becoming virtually indistinguishable from natural phosphate ions in the food. Usually, the presence of phosphorus-containing preservatives is quantified indirectly by the difference between the total amount of phosphorus contained in food and the expected one, based on its protein content. This practice has major limitations especially in the case of homogeneous mixtures (for example, mechanically deboned chicken sausage) or products including several phosphate-containing ingredients (e.g., combinations of milk, egg, meat) [39, 40]. In practice, the standard methodologies are used to evaluate the amount of phosphate (P_2O_5) and total protein nitrogen on dry matter. From the measurement of P_2O_5 (mg) to protein (g) ratio the phosphorus index is derived; for instance it is “25” for cooked hams. Above that value the addition of phosphorus containing preservative is rather certain [40].

A recent study has shown that there is no difference between food without or with phosphorus-containing preservatives with regard to the percentage of dry matter and of total nitrogen, but total and inorganic phosphorus was markedly increased in the processed food [41]. This was largely expected, but the interesting point was about the quantity: ~100 mg of extra phosphorus was found per 100 grams of processed food [41]. Recalling that the phosphorus-containing preservatives are widely used and that this phosphorus is in inorganic form and therefore almost completely absorbed in the intestine, the dietary phosphorus load is exceptionally high and beyond what is estimated by a diet diary and/or dietary interview that provides results in terms of dietary composition of raw natural foods [42]. The importance of this point becomes particularly significant in CKD, transplanted and dialysis patients, in particular in those with low socio-economic status or those who receive no dietary education [43]. All this can undermine the effects of phosphorus-binder therapy that is expected to remove 200–300 mg of phosphorus per day [44].

In summary, given the widespread presence of phosphorus-containing preservatives in food and beverages (Table 2), one can assume an extraordinarily high phosphorus exposure from additives. Indeed food additives can contribute more than 30% of the daily dietary phosphorus intake [45]. This type of dietary phosphorus burden is even more significant because it is almost completely absorbed, and because it interferes with phosphorus-binder therapy and the treatment costs and effectiveness, as these patients may require extra amounts of phosphorus-binder medications to achieve the same effect on phosphorus balance. Clinically, a lower efficacy of phosphorus binders and/or an increase in the number of doses and costs are expected. This scenario is amplified in the case of a typical American diet that seems to have a much higher content of phosphorus-containing preservatives than in many other nations [38].

Changes of Phosphorus Intake by Home-Made Processing: Cooking

The considerations made thus far are based on the composition of foods and refer to the phosphorus content per 100 g of uncooked food, or on the recognition of the presence of phosphorus-containing additives in food. However, many foods are consumed after cooking, and this can result in changes in the natural composition of the nutrients [46]. Among the different methods of cooking, some are more favorable for health because they maintain or even improve the nutritional properties of food by increasing the bioavailability of the nutrients while creating lesser amount of unwanted substances Examples of more favorable cooking are steaming, baking and stewing. From a bioavailability preservation point of view, however, boiling is generally the least preferred because this procedure removes many nutrients (mainly minerals) from the food; nevertheless, this may be seen as a favorable approach for CKD patients because boiling reduces phosphorus content of the food along with lowering the content of sodium, potassium, calcium, and several other minerals in both plants and in animal based food.

After a prolonged boiling in water, a significant reduction in the phosphorus content of food occurs. Evidence suggests that a phosphorus reduction of up to 51% for vegetables, 48% for legumes, 38% for meat, 70% for the flour and 19% for the cheese (“cheddar”) can be achieved [47]. In the case of vegetables, it is possible that favorable effect of phosphorus removal by boiling be partly counteracted by an increase in digestible phosphate derived from phytate processing; this is a clinically and dietetically impotant knowledge gap that needs further investigations. However this type of cooking may not result in the most appealing food for the patients because it reduces the palatability and taste of various components of the diet and because it requires relatively long preparation times [47]. The loss of minerals by boiling is a function of the amount of liquid used for cooking, time period, and the degree of food shredding. The interesting fact is that boiling allows a reduction in the phosphorus content of foods of animal origin, which are indispensable as a source of dietary proteins for dialysis patients who should receive high protein intake [48]. Boiling for 10–20 minutes reduces the phosphorus content of the meat or poultry by 30–50%, whereas the dry matter and protein are reduced by only 5–8% and 9–17%, respectively (Figure 1). Hence, the phosphorus content per gram of protein is reduced significantly (Figure 2) As a result this procedure offers an effective way to constructively manipulate the positive relationship between dietary protein and phosphorus intake [46].

Management of dietary phosphorus in clinical practice: Need for education

Dietary assessment of patients with CKD is possible through different methods [42]. The calculation of phosphorus intake from the dietary recall or interview may be inaccurate and indeed misleading. These estimates are generally based on the nutrient contents of the raw product and do not include any loss occurring via the cooking process. Moreover, they usually not consider the phosphorus from preservatives, nor do they distinguish between the phytates and other components with differential digestibility. Therefore it is difficult to define the actual dietary phosphorus load to the body from a standard food diary. Management of dietary phosphorus requires a multidisciplinary approach including input from nephrologists, renal dieticians and nurses, while the patient has a central role in decision-making. These challenges highlight the need for a highly professional team and a carefully thought-out multi-disciplinary approach, which includes dieticians [49–52] and psycho-social support [53]. It has been estimated that it would take a full-time dietitian for every 100 to 180 CKD patients, a situation unlikely to be achieved in many countries [54]. A kidney disease trained dietician should counsel dialysis patients on restricting dietary phosphate intake with the assumption that 30% to 50% of patients starting dialysis therapy may not have ever met a dietician previously and hence would not have received any counseling [55].

In a recent study of 191 hemodialysis patients and 105 dialysis nurses, phosphorus nutrition knowledge was lower than that of other nutrients, namely protein, potassium and sodium, even in hyperphosphatemic patients [56]. Although higher than in the general population, nutritional knowledge was unsatisfactory but well in keeping with those in a study by Pollack et al [57] who found a lack of appropriate nutritional education in hemodialysis patients, particularly regarding dietary phosphorus. Even though in this study nurses showed the best knowledge scores, the rate of correct answers for phosphorus items was only 55% on average [56]. Increased training and better information provided to nurses and to patients with poor control of serum phosphate, may facilitate more effective control of dietary phosphorus, reduction of patient care costs and improvement of the quality of care and possibly survival in dialysis patients.

One of the barriers to controlling dietary phosphorous intake is patient education; indeed up to 50% of patients receiving treatment at a dialysis clinic in the United Kingdom believed that dietary phosphate restriction was no longer necessary when they were taking phosphate binders [58]. A systematic review of the educational strategies for phosphorus reduction in CKD patients with hyperphosphatemia showed an average reduction of serum phosphorus levels of 0.72 mg/dL, and that the reduction increased to 1.07 mg/dl in the case of educational intervention for 4 months or longer [59].

The net decrease in intestinal absorption of phosphorus is the first measure to be implemented at the very early stages of CKD, in order to prevent or correct the initial alterations of calcium-phosphorus metabolism, namely the increase of FGF-23, the inhibition of 1α-hydroxylase leading to reduced calcitriol synthesis, and the increase in the synthesis and secretion of PTH. The control and the reduction of the dietary protein and phosphorus, small amounts of calcium and the correction of any deficiency of vitamin D

represent the first measures to be implemented in CKD. In dialysis patients diet alone is not able to control the phosphorus balance given the high protein requirement; hence, efforts are undertaken to limit the dietary phosphorus load by increasing the efficacy of intestinal phosphorus binding, The ideal phosphorus-binder would ardently bind dietary phosphorus, have minimal systemic absorption, have few side effects, have a low pill burden, and be inexpensive; however, none of the currently available oral phosphorus-binders meet all these criteria at the same time. As a result, dietary control of phosphorus burden and real nutritional education may help in reducing the pill burden and in control the excessive costs for this kind of drugs [41, 60]. Even a high dose of phosphorus binder agents can remove only up to 200–300 mg of dietary phosphorus while the daily dietary phosphorus load may surpass 1000 mg per day. Although these considerations appear obvious, there is a significant degree of nihilism in this sector with regard to dietary and nutritional counseling [44]. The use of phosphorus-binders has limited efficacy in the face of uncontrolled dietary phosphorus load. Recent evidence in healthy volunteers suggest that the amount of phosphorus that can be bound by lanthanum carbonate [61] is 135 mg per gram of Lanthanum (that is for 1g tablet), sevelamer binds 63 mg phosphorus per 2.4 g (namely 21 mg per 800 mg capsule) [62], and calcium carbonate 45 mg and calcium acetate 44 mg per gram of substance [61]. Table 3 summarize the quantity of common food supplying an amount of phosphorus that equals the phosphorus binding capacity of 1 g of lanthanum carbonate tablet, 2.4 g sevelamer powder, a 667 mg tablet of calcium acetate, or a 1.25 g tablet of calcium carbonate. As shown Table 3, even small amounts of phosphorus-containing cheese or processed food can blunt the phosphorus binding capacity, thus reducing efficacy, or increasing costs or pill burden of the binder therapy.

In a recent review study, Daugirdas calculated the Phosphate-Binding-Equivalent-Dosage (PBED) among the available phosphorus-binder modalities [61]. For a 60 to 80-kg hemodialysis patient on a conventional thrice weekly treatment schedule and on a 1.0 g⁄kg b.w. protein diet, a daily phosphorus-binding need of 290 or 500 mg has been calculated. Given an average mg phosphorus bound per gram of 45 mg, it follows that 11 g⁄day of PBED will be needed to maintain phosphorus balance. Unfortunately, this analysis does not fit with the average PBED reported in studies on dialysis patients that is in the range of 4–7 g⁄day [61]. Hence, it is possible that positive phosphorus balance persists despite apparent adequate control of serum phosphorus concentrations. These data suggest that adequate phosphorus balance is possible only when protein restriction is associated with P binder therapy.

A patient-friendly educational approach has recently been suggested by Kuhlmann et al to estimate the so-called “phosphorus unit” (100 mg phosphorus per serving size) in each meal, so that patient can determine how many phosphorus-binder pill he or she requires upon each meal intake.[63] We strongly believe that phosphorus-binder pills may not be suggested as a fixed number per meal but that the number of pills per each meal or snack should be dynamic and pro-actively adjusted for the amount and type of phosphorus in the given meal. As an example, a patient who eats cheeseburger with soft-drink should take more pills, while the same patients may need less pills upon eating egg-whites and boiled chicken. Furthermore, the timing of pill intake has a bearing on phosphorus-binding, in that the pills should be taken with the meals and towards the end of the meal intake. Many dialysis

patients may take the pills with empty stomach, for instance early morning before having breakfast or only upon drinking a coffee or tea with no solid food as their breakfast. In our opinion such wrong phosphorus-binding pill regimens could cause more harm than help and may lead to gastrointestinal complaints such as nausea, vomiting and bloating, which may severely compromise patient adherence to phosphorus-binding therapy.

Conclusion

A simple and effective approach to reduce the load of dietary phosphorus without reducing protein supply consists of educating patients to mitigate consumption of foods high in absorbable phosphorus (e.g., processed cheese and egg yolk), to avoid foods containing additives based on polyphosphates (such as certain types of soft-drinks), and to prefer vegetable based foods that have lower phosphorus–absorption, and to prefer boiling to other common methods of cooking. Table 4 provides an overview about three types of dietary phosphorus relevant to the management of patients with kidney disease. The multi-disciplinary approach including close collaboration with the dietitian is essential to the work of nutrition education, to help the patient in the choice of foods, and promote the effective adherence to dietary rules, a crucial aspect of an integrated approach to CKD-MBD. Areas of knowledge gap about different sources of dietary phosphorus and their differential digestibility need to be examined in expert meetings that should identify and propose pathways to urgently needed research to this end.[64]

References

1. Kestenbaum B. Phosphate metabolism in the setting of chronic kidney disease: significance and recommendations for treatment. Semin Dial. 2007; 20:286–94. [PubMed: 17635817]
2. Cozzolino M, Ciceri P, Volpi EM, Olivi L, Messa PG. Pathophysiology of calcium and phosphate metabolism impairment in chronic kidney disease. Blood Purif. 2009; 27:338–44. [PubMed: 19295196]
3. Mizobuchi M, Towler D, Slatopolsky E. Vascular Calcification: The Killer of Patients with Chronic Kidney Disease. J Am Soc Nephrol. 2009; 20:1453–64. [PubMed: 19478096]
4. Kalantar-Zadeh K, Kuwae N, Regidor DL, et al. Survival predictability of time-varying indicators of bone disease in maintenance hemodialysis patients. Kidney Int. 2006; 70:771–80. [PubMed: 16820797]
5. Zoccali C, Ruggenenti P, Perna A, et al. Phosphate may promote CKD progression and attenuate renoprotective effect of ACE inhibition. J Am Soc Nephrol. 2011; 22:1923–30. [PubMed: 21852581]
6. Messa P. Impact of Ca/P disorders on risks and comorbidities. Contrib Nephrol. 2008; 161:215–21. [PubMed: 18451680]
7. Galetta F, Cupisti A, Franzoni F, et al. Left ventricular function and calcium phosphate plasma levels in uraemic patients. J Intern Med. 2005; 258:378–84. [PubMed: 16164578]
8. Burke SK. Phosphate Is a Uremic Toxin. J Ren Nutr. 2008; 18:27–32. [PubMed: 18089440]
9. Noori N, Kalantar-Zadeh K, Kovesdy CP, Bross R, Benner D, Kopple JD. Association of dietary phosphorus intake and phosphorus to protein ratio with mortality in hemodialysis patients. Clin J Am Soc Nephrol. 2010; 5:683–92. [PubMed: 20185606]
10. Gutierrez OM. Fibroblast Growth Factor 23 and Disordered Vitamin D Metabolism in Chronic Kidney Disease: Updating the “Trade-off” Hypothesis. Clin J Am Soc Nephrol. 2010; 5:1710–6. [PubMed: 20507957]

11. Cozzolino M, Mazzaferro S. The fibroblast growth factor 23: a new player in the field of cardiovascular, bone and renal disease. Curr Vasc Pharmacol. 2010; 8:404–11. [PubMed: 20180772]
12. Eddington H, Hoefield R, Sinha S, et al. Serum phosphate and mortality in patients with chronic kidney disease. Clin J Am Soc Nephrol. 2010; 5:2251–7. [PubMed: 20688884]
13. Kumar R. Phosphate sensing. Curr Opin Nephrol Hypertens. 2009; 18:281–4. [PubMed: 19352177]
14. Ketteler M. Phosphate Metabolism in CKD Stages 3–5: Dietary and Pharmacological Control. Int J Nephrol. 2011; 2011:970245. [PubMed: 21660261]
15. Wetmore JB, Quarles LD. Calcimimetics or vitamin D analogs for suppressing parathyroid hormone in end-stage renal disease: time for a paradigm shift? Nat Clin Pract Nephrol. 2009; 5:24–33. [PubMed: 18957950]
16. Moranne O, Froissart M, Rossert J, et al. Timing of Onset of CKD-Related Metabolic Complications. J Am Soc Nephrol. 2009; 20:164–71. [PubMed: 19005010]
17. Martinez I, Saracho R, Montenegro J, Llach F. The importance of dietary calcium and phosphorous in the secondary hyperparathyroidism of patients with early renal failure. Am J Kidney Dis. 1997; 29:496–502. [PubMed: 9100037]
18. Stubbs J, Liu S, Quarles LD. Role of Fibroblast Growth Factor 23 in Phosphate Homeostasis and Pathogenesis of Disordered Mineral Metabolism in Chronic Kidney Disease. Semin Dial. 2007; 20:302–8. [PubMed: 17635819]
19. Oliveira RB, Cancela A, Graciolli FG, et al. Early Control of PTH and FGF23 in Normophosphatemic CKD Patients: A New Target in CKD-MBD Therapy? Clin J Am Soc Nephrol. 2010; 5:286–91. [PubMed: 19965540]
20. Berns JS. Niacin and Related Compounds for Treating Hyperphosphatemia in Dialysis Patients. Semin Dial. 2008; 21:203–205. [PubMed: 18363600]
21. Prie D, Beck L, Urena P, Friedlander G. Recent findings in phosphate homeostasis. Curr Opin Nephrol Hypertens. 2005; 14:318–24. [PubMed: 15930998]
22. Boaz M, Smetana S. Regression equation predicts dietary phosphorus intake from estimate of dietary protein intake. J Am Diet Assoc. 1996; 96:1268–70. [PubMed: 8948388]
23. Cupisti A, D'Alessandro C. The impact of known and unknown dietary components to phosphorus intake. G Ital Nefrol. 2011; 28:278–88. [PubMed: 21626496]
24. Cupisti A, Cupisti A, Benini O, Ferretti V, Gianfaldoni D, Kalantar-Zadeh K. Novel Differential Measurement of Natural and Added Phosphorus in Cooked Ham With or Without Preservatives. J Ren Nutr. 2012 Mar 8. [Epub ahead of print].
25. Samaraweera H, Zhang WG, Lee EJ, Ahn DU. Egg Yolk Phosvitin and Functional Phosphopeptides-Review. J Food Sci. 2011; 76:R143–50. [PubMed: 21806612]
26. Kalantar-Zadeh K, Gutekunst L, Mehrotra R, et al. Understanding Sources of Dietary Phosphorus in the Treatment of Patients with Chronic Kidney Disease. Clin J Am Soc Nephrol. 2010; 5:519–30. [PubMed: 20093346]
27. Bohn L, Meyer AS, Rasmussen SK. Phytate: Impact on environment and human nutrition — A challenge for molecular breeding. J Zhejiang Univ Sci B. 2008; 9:165–91. [PubMed: 18357620]
28. Hurrell RF. Phytic acid degradation as a means of improving iron absorption. Int J Vitamin Nutr Res. 2004; 74:445–52.
29. Moe SM, Zidehsarai MP, Chambers MA, et al. Vegetarian compared with meat dietary protein source and phosphorus homeostasis in chronic kidney disease. Clin J Am Soc Nephrol. 2011; 6:257–64. [PubMed: 21183586]
30. Raghavendra P, Halami PM. Screening, selection and characterization of phytic acid degrading lactic acid bacteria from chicken intestine. Int J Food Microbiol. 2009 Jul 31; 133(1–2):129–34. [PubMed: 19481282]
31. Anastasio M, Pepe O, Cirillo T, Palomba S, Blaiotta G, Villani F. Selection and use of phytate-degrading LAB to improve cereal-based products by mineral solubilization during dough fermentation. J Food Sci. 2010; 75(1):M28–35. [PubMed: 20492182]
32. Barsotti G, Cupisti A. The role of dietary phosphorus restriction in the conservative management of chronic renal disease. J Ren Nutr. 2005; 15:189–92. [PubMed: 15648032]

33. Fouque D, Vennegoor M, ter Wee P, et al. EBPG guideline on nutrition. Nephrol Dial Transplant. 2007; 22(Suppl 2):ii45–87. [PubMed: 17507426]
34. Shinaberger CS, Greenland S, Kopple JD, et al. Is controlling phosphorus by decreasing dietary protein intake beneficial or harmful in persons with chronic kidney disease? Am J Clin Nutr. 2008; 88:1511–8. [PubMed: 19064510]
35. Cupisti A, Morelli E, D'Alessandro C, Lupetti S, Barsotti G. Phosphate control in chronic uremia: don't forget diet. J Nephrol. 2003; 16:29–33. [PubMed: 12649532]
36. Karp H, Ekholm P, Kemi V, Hirvonen T, Lamberg-Allardt C. Differences among Total and In Vitro Digestible Phosphorus Content of Meat and Milk Products. J Ren Nutr. 2012; 22:344–9. [PubMed: 21978846]
37. Taylor LM, Kalantar-Zadeh K, Markewich T, et al. Dietary egg whites for phosphorus control in maintenance haemodialysis patients: a pilot study. J Ren Care. 2011; 37:16–24. [PubMed: 21288313]
38. Karalis M, Murphy-Gutekunst L. Enhanced Foods: Hidden Phosphorus and Sodium in Foods Commonly Eaten. J Renal Nutr. 2006; 16:79–81.
39. Gutiérrez OM, Anderson C, Isakova T, et al. Low socioeconomic status associates with higher serum phosphate irrespective of race. J Am Soc Nephrol. 2010; 21:1953–60. [PubMed: 20847142]
40. Cantoni C, Beretta G. Würstel e polifosfati. Eurocarni. 2006; 7:139.
41. Benini O, D'Alessandro C, Gianfaldoni D, Cupisti A. Extra-Phosphate Load From Food Additives in Commonly Eaten Foods: A Renal and Insidious Danger for Renal Patients. J Ren Nutr. 2011; 21:303–8. [PubMed: 21055967]
42. Bross R, Noori N, Kovesdy CP, et al. Dietary assessment of individuals with chronic kidney disease. Semin Dial. 2010; 23:359–64. [PubMed: 20673254]
43. Sherman RA, Mehta O. Dietary phosphorus in dialysis patients: potential impact of processed meat, poultry, and fish products as protein sources. Am J Kidney Dis. 2009; 54:18–23. [PubMed: 19376617]
44. Daugirdas JT. Proposed controlled trials of phosphate reduction in CKD: which whey should we go? Kidney Int. 2010; 77:929–30. [PubMed: 20431578]
45. Mathewson AM, Fouque D, Toft AJ. Dietary Phosphate Assessment in Dialysis Patients. J Ren Nutr. 2010; 20:351–8. [PubMed: 20850345]
46. Uribarri J. Phosphorus homeostasis in normal health and in chronic kidney disease patients with special emphasis on dietary phosphorus intake. Semin Dials. 2007; 20:295–301.
47. Jones WL. Demineralization of a wide variety of foods for the renal patient. J Ren Nutr. 2001; 11:90–6. [PubMed: 11295029]
48. Cupisti A, Comar F, Benini O, et al. Effect of Boiling on Dietary Phosphate and Nitrogen Intake. J Ren Nutr. 2006; 16:36–40. [PubMed: 16414439]
49. Poduval RD, Wolgemuth C, Ferrell J, Hammes MS. Hyperphosphatemia in Dialysis Patients: Is There a Role for Focused Counseling? J Ren Nutr. 2003; 13:219–23. [PubMed: 12874747]
50. Reddy V, Symes F, Sethi N, et al. Dietitian-Led Education Program to Improve Phosphate Control in a Single-Center Hemodialysis Population. J Ren Nutr. 2009; 19:314–20. [PubMed: 19539185]
51. Cupisti A, D'Alessandro C, Baldi R, Barsotti G. Dietary habits and counseling focused on phosphate intake in hemodialysis patients with hyperphosphatemia. J Ren Nutr. 2004; 14:220–5. [PubMed: 15483782]
52. Sullivan C, Sayre SS, Leon JB, et al. Effect of food additives on hyperphosphatemia among patients with end-stage renal disease: a randomized controlled trial. JAMA. 2009; 301:629–35. [PubMed: 19211470]
53. Quintaliani G, Cappelli G, Lodetti L, et al. Chronic kidney disease certification process manual by the Italian Society of Nephrology (SIN): Part I: clinical care delivery and performance measurements and improvement. J Nephrol. 2009; 22:423–38. [PubMed: 19662596]
54. Fouque D. Should we still prescribe a reduction in protein intake for CKD patients? (abs). Am J Nephrol. 2006; 26(S1):7–9.
55. Locatelli F, Fouque D, Heimburger O, et al. Nutritional status in dialysis patients: a European consensus. Nephrol Dial Transplant. 2002; 17:563–72. [PubMed: 11917047]

56. Cupisti A, Ferretti V, D'Alessandro C, et al. Nutritional Knowledge in Hemodialysis Patients and Nurses: Focus on Phosphorus. J Ren Nutr. 2012 Jan 31. [Epub ahead of print].

57. Pollock JB, Faffery JB. Knowledge of Phosphorus Compared with Other Nutrients in Maintenance Dialysis Patients. J Ren Nutr. 2007; 17:323–8. [PubMed: 17720101]

58. Ramal G, Chandra L, Harnett P, et al. Assessing knowledge of haemodialysis patients on low phosphate diet and binders. (abs). J Ren Nutr. 2008; 18:S5.

59. Caldeira D, Amaral T, David C, Sampaio C. Educational strategies to reduce serum phosphorus in hyperphosphatemic patients with chronic kidney disease: systematic review with meta-analysis. J Ren Nutr. 2011; 21:285–94. [PubMed: 21429764]

60. Tonelli M, Pannu N, Manns B. Oral Phosphate Binders in Patients with Kidney Failure. N Engl J Med. 2010; 362:1312–24. [PubMed: 20375408]

61. Daugirdas JT, Finn WF, Emmett M, Chertow GM. Frequent Hemodialysis Network Trial Group. The phosphate binder equivalent dose. Semin Dial. 2011; 24:41–9. [PubMed: 21338393]

62. Martin P, Wang P, Robinson A, et al. Comparison of dietary phosphate absorption after single doses of lanthanum carbonate and sevelamer carbonate in healthy volunteers: a balance study. Am J Kidney Dis. 2011; 57:700–6. [PubMed: 21354682]

63. Kuhlmann MK, Hoechst S, Landthaler I. Patient empowerment in the management of hyperphosphatemia. Int J Artif Organs. 2007 Nov; 30(11):1008–13. [PubMed: 18067103]

64. Kalantar-Zadeh K, Burrowes JD, Franch H, Gutekunst LA, Ikizler TA, Iseki K, Kim YL, Wanner C. Nephrology and nutrition leaders coming to hawaii for the World Renal Nutrition Week: why is the 16th Congress in Renal Nutrition and Metabolism in Honolulu, Hawai'i, June 2012, worth attending? J Ren Nutr. 2012 Jan; 22(1):1–3. [PubMed: 22200416]

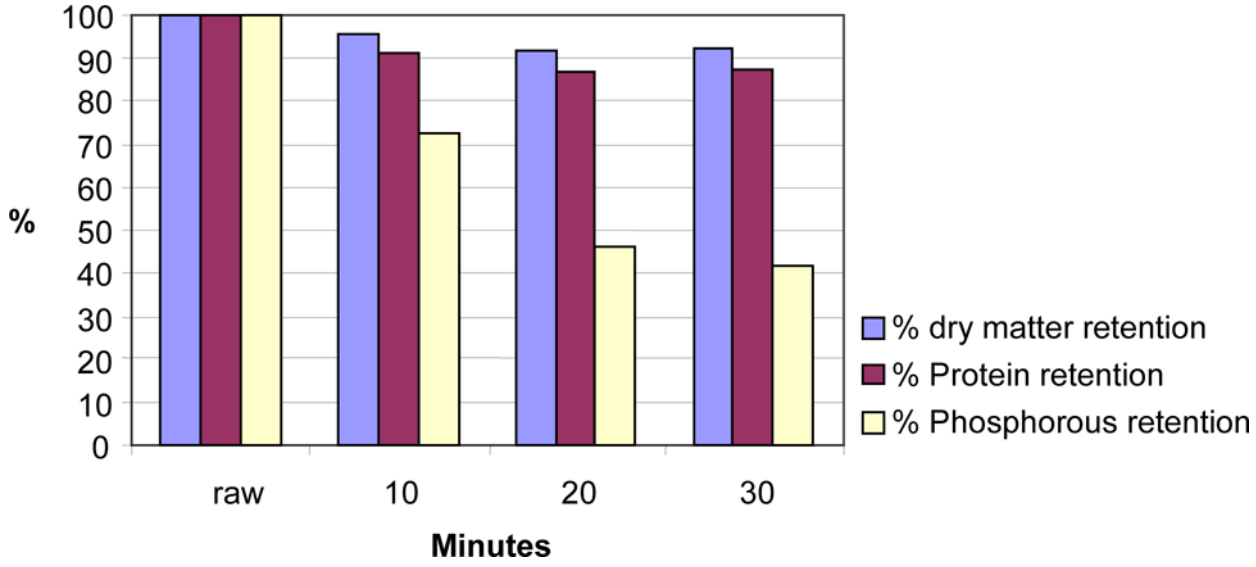

Figure 1.
Average retention values at 10′, 20′ e 30′ boiling, as percentage of raw edible part (beef) (ref. 48)

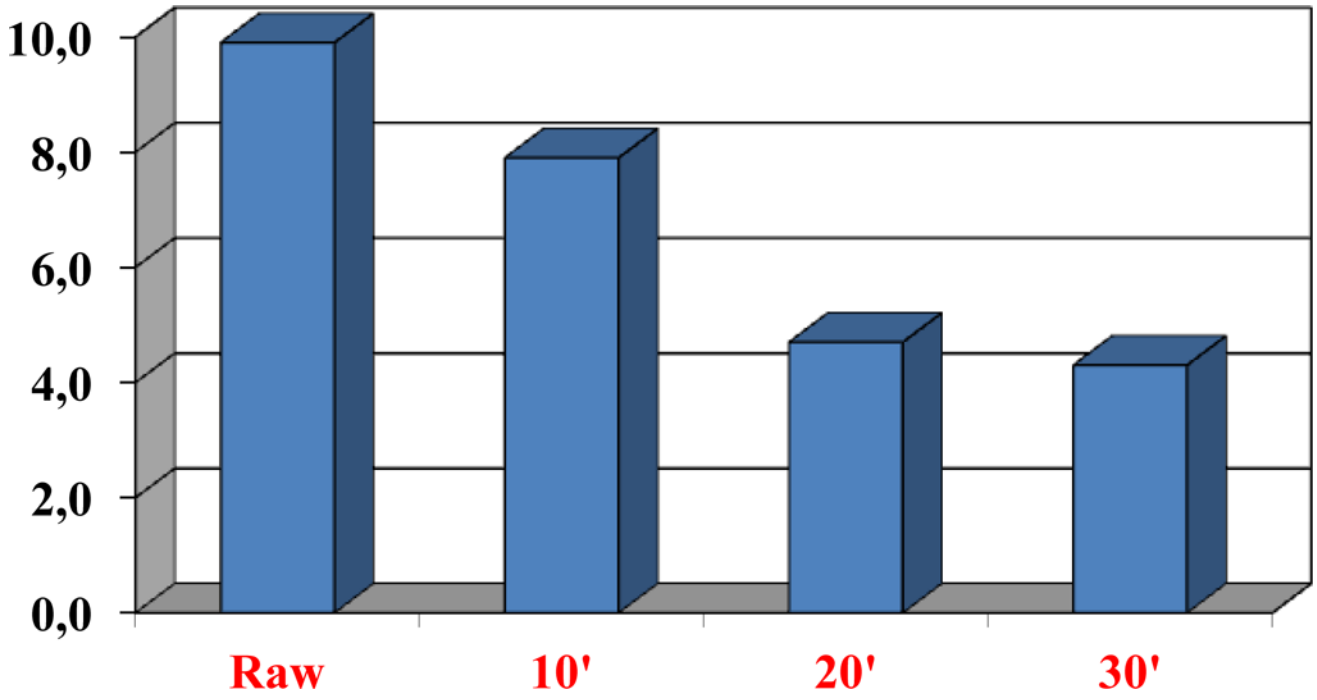

Fig. 2.

Average Phosphorus to Protein (mg/g) retention values at 10′, 20′ e 30′ (minutes) of boiling (example of beef) (ref. 48)

Table 1

Food and beverages content of Phosphorus (given as mg P per 100 g of edible part, as mg P per gram of protein, and as mg P per serving) according to the different groups of foods. They are referred to standard servings for Italian people (Italian Society of Human Nutrition).

Food	P mg in 100 g food	P to protien mg/g	P mg per serving	Presence of “added” P	Intestinal P absorption
Milk and dairy products					
Milk	94	26,9	118	−/+	++
Fresh cheese (ricotta)	237	19,4	237	−	++
Seasoned cheese (Parmesan)	702	19,8	351	−/+	++
Spreadable cheese	650	58,0	325	++	+++
Meat and fish					
Beef	172	9,0	172	−/+	++
Turkey	257	12,3	257	−/+	++
Sole	195	11,5	293	−	++
Shrimp	349	25,7	524	−/+	++
Cooked ham	185	9,8	93	− to ++	++/+++
Cooked ham with preservatives	270	16	135	++ to +++	+++
Roast turkey (organic*)	170	9	170	−	++
Roast turkey with preservatives	314	16	314	++	+++
Roast chicken (organic*)	193	9	193	−	++
Roast chicken with preservatives	257	12	257	++	+++
Natural Egg					
Yolk	586	37,1	99,6	−	++
Egg white (organic vs. prepared)	15	1,4	5,1	vs. ++	++
Legumes					
Dry bean	437	18,5	131	−	+
Cereals and tubers					
Biscuits	157	23,8	79	−/+	+/+++
Pasta	165	15,3	132	−/+	+
Potatoes	54	25,7	108	−	+
Rice	120	17,1	96	−	+

Food	P mg in 100 g food	P to protien mg/g	P mg per serving	Presence of “added” P	Intestinal P absorption
White bread	77	9,5	62	−/+	+
Whole bread	180	24,0	90	−/+	+
Whole bread made with yeast§	180	24,0	90	−	++
Fresh Fruit and vegetables					
Spinach	62	18,2	155	−	+
Tomato (natural vs. canned)	26	21,7	65	− to ++	+
Apple/Pear	12	60,0	18	−	+
Dried fruit					+
Almond	508	31,8	254	−/+	+
Walnut	238	15,1	119	−/+	+
Beverages					
Beer	28	140	70	−/+	+
Soft drinks (Cola)	41	N.A	135	++ to +++	+++
Wine (red)	28	N.A.	56	−/+	+
Miscellaneous					
Dark chocolate	186	32	56	−	+
Milk chocolate	207	23	62	−/+	+/+++
Pre-cooked soups (mean)	55	12,8	132	−/+	+/+++
Ready-made sauces (mean)	25	83	4	−/+	+/+++

The presence of added P by preservatives is also estimated as rare (−), possible (+), likely (++), certain (+++). Estimation of percentage of Gastro-intestinal (GI) absorption is also given as ≤ 40% (+); from 40 to 80% (++); > 80% (+++)

§natural yeast contains phytase so that phosphorus from phytate is more available.

Table 2

P-containing preservatives most commonly used in the trade and food industry.

Initials	Full Name	Food
E 338	Phosphoric acid	Cola and similar; fruit-flavoured soft drinks; jellies
E 339a	Sodium dihydrogen orthophosphate	Candid fruit; soft drinks
E 339b	Disodium hydrogen orthophosphate	Processed fruit products
E 339c	Trisodium orthophosphate	Partly dehydrated milk containing at least 28% of dry matter
E 340a	Potassium orthophosphate	Partly dehydrated milk containing more than 28% of dry matter
E 340b	Dipotassium hydrogen orthophosphate	Dehydrated milk and skimmed milk
E 340c	Tripotassium orthophosphate	Dehydrated milk and skimmed milk
E 341a	Calcium tetrahydrogen diorthophosphate	Soft drinks, cola in particular; jellies
E 341b	Calcium hydrogen orthophosphate	Soft drinks, cola in particular; jellies
E 341c	Tricalcium diorthophosphate	Soft drinks, cola in particular; jellies
E 343	Magnesium phosphate	Fresh cheese, except mozzarella cheese
E 450	Polyphosphates	Bread, matl, toasted barley, coffee, chocolate, processed cheese, ice cream and dessert, potato flour, cooked ham, canned meat, cooked sausages, breaded products
E 540	Calcium diphosphate	Baked products
E 541	Sodium aluminum phosphate	Dehydrated milk, processed egg products, various flours
E 544	Calcium polyphosphates	Sauces, soups and broth, infusions made with instant tea, chewing gum, alcoholic beverages except wine and beer, powdered sugar, frozen fillets of unprocessed fish, spreadable fats (except butter), beverages made from coffee for vending machines, flavours
E 545	Ammonium polyphosphate	Cocoa and products made with chocolate

Table 3

Quantity of common foods supplying an amount of phosphorus that equals the estimated P binding capacity of one administration of 1 g of Lanthanum carbonate (as elemental lanthanum), 2,4 g Sevelamer carbonate, 0,667 g tablet Calcium Acetate or 1,25 g tablet Calcium carbonate.

Phosphorus	**Lanthanum Carbonate (1g cp *) 135 mg**	**Sevelamer Carbonate (2,4 g p) 63 mg**	**Calcium acetate (0,667 g cp.) 33 mg**	**Calcium carbonate (1,25 g cp.) 56 mg**
Dairy products				
Parmesan cheese	19	9	5	8
Ricotta	57	27	14	24
Soft cheese	36	17	9	15
Whole milk	144	67	35	60
Fish			0	0
Dentex	51	24	12	22
Shrimp	39	18	9	16
Sole	69	32	17	29
Tuna in oil	57	27	14	24
Meat				
Beef	78	37	19	33
Chicken	70	32	17	29
Pork	61	28	15	25
Turkey	53	25	13	23
Ham	76	36	19	32
Miscellaneous				
Almond	27	12	6	11
Walnut	36	17	9	15
Boiled ham	70	32	17	29
Added boiled ham	46	21	11	19
Roasted turkey	80	37	19	33
Added roasted turkey	43	20	10	18
Roasted chicken	70	33	17	30
Added roasted chicken	53	25	13	23

Table 4

Overview of the types of dietary phosphorus

Type	Source	Examples	GI absorption	Phos/Protein Ratio	Advantage
Organic Plants	Plant proteins	Nuts, beans, chocolate	20–40%	5–15 mg/g	Protein gain
Organic Animals	Animal proteins	Fish, meat, chicken	40–60%	10–20 mg/g Egg whites <5 Egg yolk >20	High value protein & amino acids
Inorganic	Additives	Soft drink, fast food	~100%	Very high (>>50 mg/g)	No gain (Teenagers?)