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REVIEW

CLINICAL SPOTLIGHT

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DIETARY PHOSPHORUS AND RENAL DISEASE IN CATS
Where are we?

Jonathan Stockman

Practical relevance: Phosphorus is an essential nutrient required for the normal function of every cell in the body and a deficiency in dietary phosphorus may lead to adverse effects. Conversely, high dietary phosphorus may cause kidney damage in otherwise healthy adult cats, particularly when provided in highly bioavailable forms and when the calcium-to-phosphorus ratio is low. For cats that have chronic kidney disease (CKD), phosphorus is the most important mineral in its pathogenesis and morbidity. As the disease progresses, elevated phosphorus may increase the risk of complications such as soft tissue mineralization, which can lead to a further decrease in renal function. Additionally, the hormones secreted in response to increased circulating phosphorus have harmful effects, such as bone resorption, and can cause cardiovascular pathology. Very low phosphorus diets can also be problematic in cats with early CKD, potentially leading to hypercalcemia.

Clinical challenges: There is currently a lack of maximum safety limits for dietary phosphorus in accepted nutritional guidelines in North American and Europe, which makes it difficult to assess the safety of some higher phosphorus cat foods. Additionally, information regarding phosphorus bioavailability is unknown for many diets and there are no commercially available tests. Similarly, there is no consensus regarding phosphorus requirement and recommended intake in cats with International Renal Interest Society stage 1–4 CKD despite there being targets for serum phosphorus.

Aims: This review evaluates dietary phosphorus in healthy cats and cats with renal disease, and describes how newer research is informing evolving clinical approaches in feline nutrition.

Audience: The article is aimed at general practitioners, internal medicine specialists and veterinary nutritionists.

Evidence base: Information provided in this article is drawn from the published literature.

Keywords: Phosphorus; calcium; kidney; renal; diet; parathyroid hormone; fibroblast growth factor-23

Homeostasis and metabolism of this essential nutrient

Phosphorus is an essential nutrient present in all cells and has varied roles in the body. The main store of phosphorus is skeletal tissue. Circulating phosphorus, its excretion and its absorption from the gastrointestinal tract are regulated by several hormones and also impacted by calcium in the diet and circulation. Dietary phosphorus requirements are highest during growth when skeletal development and maturation occur; however, phosphorus remains an essential nutrient throughout life.

Phosphorus homeostasis is maintained by similar mechanisms across mammalian species. These include the regulation of phosphorus absorption in the gastrointestinal tract, filtration and absorption in the kidneys, and shifts in and out of the bone. Phosphorus is absorbed throughout the intestinal tract, although most absorption occurs in the small intestines. Intestinal absorption is mediated by the type IIb sodium phosphate cotransporters Npt2b, which are regulated by phosphorus intake as well as cholecalciferol – the active form of vitamin D. The sites of active intestinal absorption of phosphorus in cats are unknown and cannot be presumed as they appear to vary depending on the species; in rats and humans, for example, the sites of absorption are thought to be primarily the duodenum and jejunum with little absorption in the ileum, whereas phosphorus absorption occurs primarily in the ileum in mice.[1,2] The kidneys also have an important role in phosphorus regulation where filtered phosphorus may be reabsorbed. Most renal reabsorption occurs in the proximal tubule, where the density of sodium phosphorus cotransporters Npt2a, Npt2c and PiT-2 determines how much phosphorus is reabsorbed. Factors such as dietary phosphorus, calcitriol levels, parathyroid hormone (PTH), fibroblast growth factor-23 (FGF-23), metabolic acidosis and glucocorticoids all impact phosphorous reabsorption.

The kidneys have an important role in phosphorus regulation where filtered phosphorus may be reabsorbed.

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Effects of CKD

CKD results in disturbances in calcium and phosphorus homeostasis, which can lead to complications such as soft tissue mineralization and progression of CKD. Phosphorus retention may occur because of a decline in renal function and glomerular filtration rate with CKD. Despite the increase in serum phosphorus and the decline in metabolic transformation of vitamin D to cholecalciferol in advanced CKD, the absorption of phosphorus from the intestines is hardly affected, however (as shown in rats).[3] PTH, which is secreted in response to hypocalcemia with or without hyperphosphatemia, leads to the release of calcium stores from the bone and in the long term results in bone remodeling.[4] Elevated serum phosphorus and calcium may result in soft tissue mineralization, which can exacerbate CKD if the kidneys are affected. FGF-23, which is secreted from the bone in response to hyperphosphatemia, has the primary role, in combination with a factor named Klotho, of decreasing the density of sodium phosphorus cotransporters in the kidney, thereby having a phosphaturic effect.

Elevated phosphorus due to CKD may lead to soft tissue mineralization and a resultant further decrease in renal function.

Phosphorus excess and deficiency

Both excesses and deficiencies of phosphorus can cause adverse effects. When there is increased circulating phosphorus, the hormones secreted in response have harmful effects, such as bone resorption, and can cause cardiovascular pathology. In relation to CKD, a high intake of phosphorus may induce kidney disease in healthy cats (see 'High phosphorus intake and risk of kidney damage in healthy cats').

Nutritional phosphorus deficiency is detrimental in growing kittens as it can lead to hypophosphatemic rickets and skeletal deformities. Adult cats rarely suffer from clinical phosphorus deficiency but low levels can lead to hemolytic anemia, decreased mobility and metabolic acidosis; this was demonstrated in one study where adult cats were fed a diet with severe phosphorus restriction of 0.13 g per 1000 kcal and a calcium-to-phosphorus ratio (calcium:phosphorus) of 4:1.[5] Considering CKD specifically, recent data suggest that a low-phosphorus diet may lead to hypercalcemia in cats with early CKD (see 'Veterinary therapeutic diets through the renal disease stages' box).[6]

High phosphorus intake and risk of kidney damage in healthy cats

High dietary soluble phosphorus (see 'Dietary phosphorus and its varied bioavailability' box) may induce kidney disease in otherwise healthy adult cats. This may be of higher concern in senior cats, which are at a greater risk of CKD, and so clinicians should especially consider phosphorus intake in this age group. The impact of dietary soluble phosphorus on kidney health in cats has been evaluated in several feeding studies in adult cats:

- Two studies documented a reduction in endogenous creatinine clearance as a measure of glomerular function and, in some cases,

Dietary phosphorus and its varied bioavailability

Dietary phosphorus may be provided as organic and inorganic phosphorus. Organic phosphorus is often water-insoluble and present in animal- and plant-derived ingredients included in the diet. Inorganic phosphorus is found in compounds added to commercial cat food for processing, palatability, leavening and other food properties or to meet nutritional requirements. Water-soluble inorganic phosphoric salts are generally much more bioavailable than insoluble phosphorus.[7] Moreover, phosphorus absorption may be impacted by factors such as the content of other minerals, including magnesium and calcium.[8] Therefore, total dietary phosphorus provides limited information when assessing the possible impact on feline health.

The calcium:phosphorus in the diet appears to have great importance for absorption. A diet that includes soluble phosphoric salt (such as sodium dihydrogen phosphate) with a low calcium:phosphorus may lead to a gradual increase of phosphorus and PTH post-prandially. Conversely, a diet with a higher calcium:phosphorus and insoluble phosphorus as the main form of phosphorus has been found to cause a slight decrease in serum phosphorus.[7,9]

There are currently no stipulations regarding a maximum level of phosphorus in European or American guidelines (for total or inorganic phosphorus), although the European guidelines (FEDIAF) allow for the calcium:phosphorus to be between 1 and 2, and in a footnote explain that there are changes in renal function parameters when cats are fed diets with high inorganic phosphorus.[10]

How much phosphorus is in feline diets?

Two diet surveys analyzed how much phosphorus is found in feline diets commercially available in North America. For 59 adult cat diets, the phosphorus amounts ranged from 0.6 to 5.8 g per 1000 kcal and calcium:phosphorus ranged from 0.8 to 1.7;[11] for 31 senior diets, the phosphorus range was 1.5–4.4 g/1000 kcal and calcium:phosphorus was 0.9–1.6.[12] While the analysis did not differentiate between soluble and insoluble phosphorus compounds, some commercial cat foods may be high in soluble phosphorus, and further regulation is needed to ensure safety. While the total amount of phosphorus often does not reflect the total amount absorbed, the author suggests that new guidelines include a limit on total dietary phosphorus (eg, 4 g/1000 kcal), as well as a calcium:phosphorus above 1 and a limit on soluble phosphorus of up to 1 g/1000 kcal. This could be controlled during the diet formulation step, although there is currently no available test to accurately measure this.

glucosuria and albuminuria, when healthy adult cats were fed diets supplemented with soluble phosphorus for a total intake of 3.0–3.6 g in 1000 kcal and a calcium:phosphorus of 0.3–0.4.[8,13]

- In a more recent study, adult cats were fed extruded dry foods with phosphorus exceeding 4.8 g/1000 kcal, with 3.6 g primarily provided by a soluble phosphorus salt, and a calcium:phosphorus of 0.6, for 4 weeks. This resulted in a significant increase in creatinine, a decline in glomerular filtration rate and ultrasonographic abnormalities, including nephrolithiasis.[14]
- When a different cohort of cats in the same study was fed a diet with 3.6 g of phosphorus per 1000 kcal, with 1.5 g coming from inorganic phosphorus, and a calcium:phosphorus of 0.9, for 28 weeks, this resulted in renal echogenicity changes on ultrasound and nephrolithiasis, with one cat developing an acute uremic crisis leading to euthansia.[14]
- A further trial included feeding diets with 4 g or 5 g of phosphorus per 1000 kcal, with 1 g provided by inorganic phosphoric salt and a calcium:phosphorus above 1.0, for 30 weeks.[15] Cats in this study maintained good overall health, without evidence of glucosuria or albuminuria, despite a temporary increase in FGF-23 in one of the diet groups.

A low-phosphorus approach to nutritional management of cats with CKD

The clinical benefit of dietary phosphorus reduction in animals with CKD has been well established for many years. One of the first studies to demonstrate this was performed in nephrectomized cats where a diet with reduced phosphorus resulted in fewer pathologic renal changes, such as tissue fibrosis, compared with the higher phosphorus control diet.[16] Dietary phosphorus reduction can bring a decrease, or even normalization, of serum phosphorus and PTH in cats with naturally occurring CKD.[17] In rats, a lower phosphorus diet may also prevent parathyroid hyperplasia and hyperparathyroidism.[18]

The benefits of dietary phosphorus reduction in cats with IRIS stage 2 or higher CKD specifically have been known for decades, while data for cats with early kidney disease is only recent and limited (see 'Veterinary therapeutic diets through the renal disease stages' box). Some cats lose weight and muscle mass while being fed low-phosphorus veterinary therapeutic diets, however, which raises the question of whether they are of clinical benefit when cats have a low appetite and are frail.

Veterinary therapeutic diets through the renal disease stages

CKD stages 2 and higher

The dietary modifications present in veterinary therapeutic diets for cats with kidney disease (described in the 'Veterinary therapeutic diet composition' section) have been shown to reduce serum urea and increase bicarbonate in cats with naturally occurring International Renal Interest Society (IRIS) CKD stages 2 and 3. More importantly, cats that were fed a non-therapeutic diet had 26% more uremic episodes compared with cats that were fed the veterinary therapeutic renal diet. The strongest evidence regarding the benefits of veterinary therapeutic kidney diets was demonstrated in cats with IRIS stage 2 CKD or higher.[19]

CKD stage 1

Some newer research has also shown a potential benefit of feeding a therapeutic diet to cats with IRIS stage 1 CKD. One study showed that a moderately reduced phosphorus and protein diet can help normalize creatinine, total calcium and phosphorus, and decrease FGF-23 in cats with earlier stages of renal disease.[20] Another study demonstrated that the levels of uremic solutes such as p-cresol sulfate and 3-indoxyl sulfate are increased in IRIS stage 1 CKD cats when fed a higher protein diet compared with those fed a reduced protein diet, even as both diets were comparable in the amount of total phosphorus.[21] While these findings are intriguing, there is still a paucity of clinical evidence for the benefits of feeding cats with early kidney disease a veterinary therapeutic kidney diet. Instead, a clinician may therefore opt to rely on early detection of abnormalities in phosphorus metabolism to decide if a lower phosphorus diet is warranted (see 'Monitoring serum phosphorus and its metabolism' box). If some phosphorus reduction is indicated, new diets labeled for early-stage CKD (IRIS stages 1–2) are now available, which are higher in phosphorus and protein relative to the diets formulated for the advanced stages of CKD but lower than typical maintenance diets.

As mentioned earlier, there are also recent data that suggest transition to a low-phosphorus diet (0.7–1.1 g/1000 kcal) is a risk factor for hypercalcemia in some cats with early CKD[6] and that changing back to a higher phosphorus diet with 1.5 g of phosphorus per 1000 kcal can help resolve the hypercalcemia.[24] A recently published case series in cats with idiopathic hypercalcemia or early CKD included 10 cats fed diets with either 2 g of calcium per 1000 kcal and/or a calcium:phosphorus of 1.4 or greater. Nine of the ten cats improved 3–20 weeks after having changed to a lower calcium and calcium:phosphorus diet.[25] Calcium:phosphorus could therefore be an important factor, in addition to the total phosphorus and calcium. It is recommended that clinicians monitor both calcium and phosphorus in feline patients with early CKD, particularly if they recommend feeding a low-phosphorus diet.

Monitoring serum phosphorus and its metabolism

Detection of abnormalities in phosphorus metabolism can be a helpful tool for determining whether reducing dietary phosphorus may be beneficial for a patient. This can be achieved by measuring serum phosphorus and using commercial tests for PTH and FGF-23, which may indicate early shifts in normal phosphorus metabolism.[22,23] A high serum phosphorus relative to the patient's IRIS stage of disease would be a negative prognostic indicator and warrants a diet change to a lower phosphorus diet.

Serum phosphorus results should be interpreted with caution, however, as they may not reflect abnormalities in calcium and phosphorus metabolism due to the activity of phosphatonins, which are factors that have a major role in regulating phosphorus homeostasis, or due to circadian fluctuations in serum phosphorus (unpublished data).

Clinicians should monitor both calcium and phosphorus in feline patients with early CKD, particularly if they recommend feeding a low-phosphorus diet.

Veterinary therapeutic diet composition

Veterinary therapeutic diets for cats with kidney disease are typically reduced in phosphorus and sodium, supplemented with B vitamins and long-chain omega-3 fatty acids, and are alkalizing. These diets also have less protein in order to reduce the accumulation of uremic toxins, which result from protein metabolism and are not cleared effectively because of decreased renal filtration. Protein reduction can additionally help mitigate proteinuria when present and prevent the progression of tubular injury.[26]

While lower in protein, these diets are often highly digestible, and the provided protein is of high quality, meaning that amino acid composition and ratios approximate the cat's requirement. This allows for maximal utilization of amino acids to minimize endogenous protein catabolism, amino acid oxidation and protein fermentation in the colon. Moreover, the protein amount in these diets usually meets the recommended allowance by the National Research Council (2006) for healthy adult cats, meaning that while veterinary therapeutic renal diets are reduced in protein, they are not considered deficient in protein.[27]

Meeting the protein and calorie requirements of cats with renal disease

While veterinary therapeutic diets meet the recommended allowance for protein in a healthy cat, as described earlier, protein requirements for cats with kidney disease are unknown and may be higher due to increased muscle catabolism. Moreover, these requirements may not be met when the energy intake is below a cat's energy requirements due to poor appetite associated with disease, poor diet palatability or food aversion. Therefore, some cats with kidney disease may suffer from an exacerbated decrease in lean body mass following a diet change to a veterinary therapeutic diet (Figure 1). It may then be hard to discern whether the reduction in muscling is due to primary disease, protein malnutrition or insufficient calorie intake. The new diets for cats with early CKD (see 'Veterinary therapeutic diets through the renal disease stages' box) can be a good option for individuals at this stage without the significant proteinuria that would indicate further protein reduction.

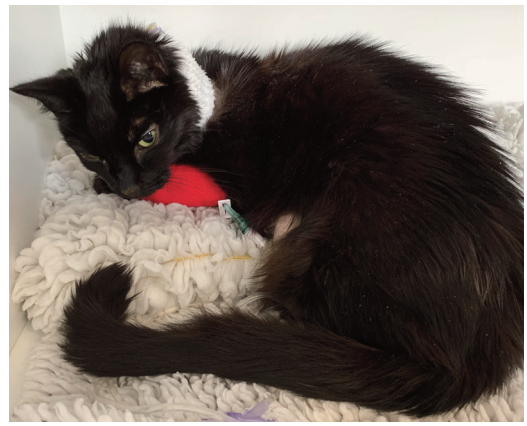

Figure 1 **Adult cat suffering from chronic kidney disease and sarcopenia receiving supportive care in a veterinary clinic.** *Courtesy of Sam Taylor*

Kidney disease often results in decreased appetite and many cats with advanced disease present with sarcopenia and poor body condition. The dilemma many clinicians therefore face is whether to prescribe a therapeutic veterinary kidney diet and risk poor intake with resultant decreased muscling and body condition, or to opt for an 'over-the-counter' commercial maintenance diet with or without a phosphate binder such as aluminium hydroxide (see 'Veterinary therapeutic diets vs phosphate binders' box). Monitoring serum phosphorus can be a helpful

Veterinary therapeutic diets vs phosphate binders

The benefits of therapeutic diets for kidney disease are not limited to phosphorus reduction as they are also formulated to benefit kidney health by, as mentioned earlier, having highly digestible and high-quality protein in reduced amounts, and being supplemented with long-chain omega-3 fatty acids, fortified with B vitamins and are alkalinizing. Phosphate binders are unlikely to be as effective in reducing phosphorus absorption as a diet low in phosphorus and have possible adverse effects; therapeutic kidney diets are therefore preferred when possible. The variety of aromas and flavour profiles available with veterinary therapeutic diets also allow owners to try several options with their cat or to rotate between a few different diets to minimize food aversion, which can develop in some azotemic cats.

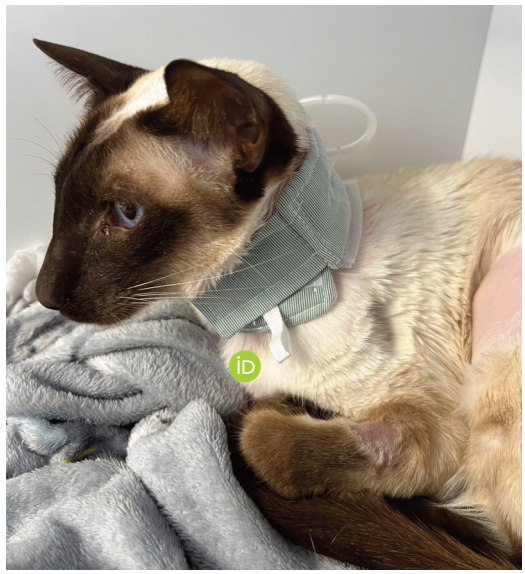

Figure 2 **Adult cat suffering from chronic kidney disease with an esophageal feeding tube.** *Courtesy of Sam Taylor*

tool (see 'Monitoring serum phosphorus and its metabolism' box). Appetite-enhancing medications and even feeding tubes for assisted enteral feeding may be considered in patients that are reluctant or unable to meet their energy needs with an appropriate diet (Figure 2).

Conflict of interest

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Ethical approval

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Informed consent

This work did not involve the use of animals (including cadavers) and therefore informed consent was not required. For any animals or people identifiable within this publication, additional informed consent for publication was obtained.

KEY POINTS

- Dietary phosphorus intake is a paramount consideration during the management of feline CKD.
- While phosphorus is an essential nutrient, decreased kidney function causes a shift in normal phosphorus homeostasis, which promotes disease progression.
- New information highlights that excessive soluble/inorganic phosphorus intake in healthy cats may lead to kidney injury and CKD.
- Clinicians should consider phosphorus intake, particularly in senior cats and in cats with CKD. An important limitation, however, is that phosphorus solubility and bioavailability are rarely known and no commercial assays are available.

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