



Nutritional effects of calcium and phosphorus on chronic kidney disease in cats: A literature review

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Abstract. The relevance of this study is conditioned by the significant prevalence of chronic kidney disease in cats and the prominent role of calcium-phosphorus metabolism disorders in its progression and the development of complications, which requires further scientific attention to improve approaches to animal treatment. The purpose of this review was to summarise modern scientific data on the influence of calcium and phosphorus on the course of chronic kidney disease in cats and to formulate recommendations for nutritional support. A systematic approach was employed to search and analyse scientific literature for 2020-2025. The review analysed the results of modern studies on the diagnostics of chronic nephropathy in small carnivores, clinical recommendations, and expert opinions in the field of veterinary medicine. The study found that the excess phosphorus content in the diet of animals directly correlates with the acceleration of disease progression, the development of secondary hyperparathyroidism, soft tissue calcification, and deterioration of the functional state of the kidneys. At the same time, excessive decrease in phosphorus concentration can cause hypercalcemia, loss of appetite, vomiting, and other metabolic disorders. The optimal diet for animals with chronic kidney disease is a calcium to phosphorus ratio within the range of 1.1:1-1.5:1. The study considered role of modern biomarkers, such as symmetrical dimethylarginine

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and fibroblast growth factor-23, in the early diagnosis and monitoring of chronic kidney disease. The study also analysed the efficacy of dietary therapy, particularly the use of phosphate binders, active forms of vitamin D, probiotics, antioxidants, and agents for the correction of acid-base homeostasis. A review of the literature confirmed that efficacious nutritional management is a key factor in the control of chronic kidney disease. The findings obtained emphasised the significance of developing optimal dietary strategies that account for both the phosphorus level and the calcium to phosphorus ratio in the blood serum. This will inhibit the progression of the disease and improve the quality of life of cats with chronic kidney disease

Keywords: mineral homeostasis; veterinary dietetics; electrolyte metabolism; metabolic disorders; hypercalcemia; hyperphosphatemia; phosphate binders

Introduction

Chronic kidney disease is one of the most widespread pathologies in cats, especially among older animals. It substantially reduces the quality of life, as it is accompanied by a gradual and irreversible loss of kidney function. According to R.C.C. Costa *et al.* (2024), the disease can occur due to a variety of causes, including genetic predisposition, past infections, or toxicity. The kidneys play a key role in maintaining homeostasis: eliminating metabolic products, regulating electrolyte balance, and fluid volume. If these functions are insufficient, toxic substances accumulate in the blood, which adversely affects all body systems.

One of the most effective tools for managing chronic kidney disease (CKD) is dietary therapy, which is aimed at reducing the metabolic load on the kidneys. According to V.J. Parker (2021), controlling phosphorus, protein, and calcium levels in the diet is key to slowing disease progression, reducing the risk of mineral disorders, and improving the overall condition of the animal. Diets moderately reduced in phosphorus and protein, supplemented with essential nutrients, can reduce uraemia, stabilise parathyroid hormone levels, and improve quality of life. Although protein restriction is a classic component of dietary therapy for chronic kidney disease, recent

literature reviews in humans suggest that too severe protein reduction (<0.6 g/kg/day) may not have an additional nephroprotective effect and increases the risk of protein-energy deficiency, especially in frail patients. According to J.W. Kim & S.J. Yang (2025), excessive protein restriction also contributes to the development of sarcopenia – loss of muscle mass and functional activity, which is especially dangerous for elderly patients. This emphasises the need for a balanced and individualised approach to protein correction in the diet, which is likely to be relevant for cats with CKD, considering the similarity of pathophysiological processes and sensitivity to muscle loss in older animals.

One of the most prominent aspects of the pathogenesis of chronic kidney disease is the disturbance of calcium and phosphorus metabolism. Under physiological conditions, these minerals ensure the strength and integrity of bone tissue. However, cats with this pathology often develop calcium and phosphorus imbalances, which, according to S.-H. Tsai *et al.* (2024), contributes to the development of secondary hyperparathyroidism, bone resorption, soft tissue mineralisation, and even the development of cardiovascular complications. The level of phosphorus in the diet plays a

crucial role in maintaining kidney health in cats. An excess of this mineral is associated with an increased risk of structural damage to renal tissue and accelerated disease progression. Apart from the phosphorus content, a significant factor is its ratio with calcium.

Olad J. Stockman (2024) highlighted modern aspects of nutritional therapy for chronic kidney disease in cats, particularly the effects of phosphorus and the calcium to phosphorus ratio on disease progression. The researcher noted that an imbalance between these elements may contribute to the development of mineral disorders, although their exact impact on clinical outcomes is still understudied. Stockman also emphasised the variability of phosphorus bioavailability in diets, the potential risks of excessive restriction, and the lack of clear recommendations for target intakes. Previous studies showed that specialised diets with controlled phosphorus content can be an effective component of therapy, helping to reduce clinical manifestations of the disease and maintain patients' stable condition. The researcher emphasised the significance of a comprehensive nutritional approach that includes not only phosphorus regulation, but also a balanced ratio of protein and other macronutrients to prevent secondary disorders such as hypoproteinaemia or hypophosphataemia.

According to W. Zhang *et al.* (2024), a high dietary calcium to phosphorus ratio is associated with a greater prevalence of nephroliths, which can cause ureteral obstruction and create risks for urinary infections, which worsens the development of kidney disease. Although these values may vary between cats, focusing on calcium/phosphorus ratios rather than absolute concentrations appears reasonable for the prevention of mineral disorders, particularly in patients with chronic kidney disease. This emphasises the expediency of dietary adjustment regarding the calcium to phosphorus

ratio as one of the key nutritional parameters in the treatment of CKD.

R.F. Geddes *et al.* (2021) also noted that excessive phosphorus restriction can lead to hypercalcaemia, especially in the early stages of renal dysfunction. The researchers found that mitigating phosphorus restriction in cats with stage 2 kidney disease helped to normalise ionised calcium levels, suggesting the need for a cautious approach to dietary correction.

Considering the significance of improving approaches to the treatment of chronic kidney disease in cats, the purpose of the current study was to analyse current research on the role of calcium and phosphorus in the progression of CKD to develop recommendations for optimal dietary nutrition of animals. For this, the objectives were to scientifically substantiate the role of calcium and phosphorus in the pathogenesis of chronic kidney disease in cats, to analyse the efficacy of dietary therapy and means of correcting mineral imbalances, and to identify diagnostic approaches to monitor the state of mineral metabolism to optimise therapeutic tactics and improve the quality of life of animals.

To fulfil this purpose, a systematic literature review was applied, the search for which was conducted through the scientific databases PubMed, Scopus, and Web of Science. The initial search yielded over 100 publications, of which 41 scientific sources were selected for analysis after filtering by inclusion criteria. The inclusion criteria included publications in peer-reviewed journals, studies of the effect of calcium and phosphorus on the functional state of the kidneys in cats, availability of empirical data, or a detailed review of the results of clinical application of dietary approaches. The results of clinical and experimental studies, official veterinary recommendations, and expert opinions on metabolic changes in chronic nephropathy in cats were analysed. Particular

attention was paid to biomarkers of early diagnosis, mechanisms of metabolic disorders, and the efficacy of nutraceutical and pharmacological approaches to correcting phosphorus-calcium imbalance.

Mineral metabolism disorders in chronic kidney disease

In cats, the course of chronic kidney disease is accompanied by a disturbance in mineral metabolism, particularly calcium and phosphorus levels, which affects its progression. New studies confirmed that these changes can substantially affect the further development of the disease and the quality of life of animals. D.J. Chew (2022) noted that in cats with chronic kidney disease, the mechanism of phosphorus excretion through the kidneys is impaired, which causes its accumulation in the blood. This can lead to the development of hyperphosphatemia, which is a trigger for secondary hyperparathyroidism. Dietary therapy is the key method of inhibiting the progression of chronic kidney disease in cats. V.J. Parker (2021) indicated that key aspects of dietary restrictions include controlling phosphorus levels, balancing protein, regulating sodium intake, maintaining acid-base homeostasis, and correcting essential nutrient deficiencies.

C.J. Vorland *et al.* (2020) investigated the effects of chronic kidney disease progression on intestinal phosphorus absorption using rats, which have become a model for chronic kidney disease and mineral metabolism disorders. The researchers found that, despite the deterioration of kidney function, disease progression does not reduce the efficiency of phosphorus absorption in the intestine. This study provided new insights into the role of the gut in regulating phosphorus levels when suffering from CKD, as it was previously thought that renal dysfunction directly reduced the body's ability to absorb phosphorus.

D.E. Jewell *et al.* (2022) studied metabolic changes in cats with chronic kidney disease and calcium-oxalate uroliths, finding marked changes in metabolic processes that are essential for the development of these diseases. Their findings highlighted that the impaired calcium and phosphorus metabolism that often accompanies CKD may be associated with metabolic changes that contribute to the development of uroliths. The study also pointed to the significance of metabolic analysis as a tool for understanding the pathophysiology of kidney disease in cats. Specifically, it enables only a more accurate diagnosis of mineral metabolism disorders, but also the creation of personalised treatment and prevention strategies that include the correction of metabolic changes in these patients.

J. Stockman (2024) indicated that elevated blood phosphorus levels are associated with faster progression of chronic kidney disease in cats. This is conditioned by the fact that hyperphosphatemia stimulates the production of parathyroid hormone, which causes bone resorption, increasing calcium mobilisation. Long-term exposure to this mechanism can lead to calcification of kidney and cardiovascular tissues. J. Lin *et al.* (2021) noted that early monitoring of calcium and phosphorus levels can predict the risk of complications in cats with chronic kidney disease. The researchers found that cats with a high phosphorus to calcium ratio had a significantly shorter life expectancy compared to those with balanced levels in the early stages of the disease. Assessment of calcium and phosphorus levels is a crucial element of the diagnosis and monitoring of chronic kidney disease in cats, as their imbalance affects the progression of the disease and the effectiveness of dietary correction. P.-K. Tang *et al.* (2021) noted that monitoring these parameters helps to assess the risk of renal calcification or secondary hyperparathyroidism.

Diagnosics of chronic kidney disease in cats

Serum biochemical parameters such as creatinine, urea, and symmetrical dimethylarginine are used to determine the stage of chronic kidney disease and assess the functional state of the kidneys. According to J.A. Hokamp & M.B. Nabity (2016), SDMA is a more sensitive biomarker than creatinine, particularly in animals with low muscle mass, where creatinine levels may stay within normal limits. The combined use of SDMA and creatinine enables a more precise assessment of glomerular filtration rate, especially in the early stages of CKD. This approach provides greater diagnostic accuracy by compensating for the limitations of each marker when used in isolation.

Another element of the diagnostics is urine analysis. V. Pedrinelli *et al.* (2020) emphasised the significance of assessing urine specific gravity and proteinuria. A decrease in the concentration capacity of urine indicates tubular damage, while the presence of protein in the urine indicates glomerular damage. Proteinuria is an independent predictor of the progression of chronic kidney disease and is associated with a shorter life expectancy. R.C.C. Costa *et al.* (2024) noted that basic diagnosis includes determination of urine specific gravity, serum creatinine and SDMA levels. Creatinine continues to be a standard marker of renal function, but it does not always enable early detection of pathology due to its dependence on muscle mass. In this context, SDMA is a more sensitive indicator, because its increase may precede the increase in creatinine. This was confirmed by L. Hahn & C. Callaband (2022), who demonstrated that SDMA can diagnose chronic kidney disease in the preclinical phase and has a high prognostic value. Specifically, the level of ionised calcium in the blood serum is a more accurate indicator of the body's supply of this mineral than the total calcium level.

R.F. Geddes *et al.* (2021) emphasised the significance of the ionised fraction in assessing mineral metabolism disorders, especially in the setting of reduced albumin levels, which often accompany renal pathology. In their study, the researchers emphasised that monitoring the level of active calcium enables a more accurate interpretation of the clinical situation and the selection of suitable correction tactics.

Laboratory analysis of urine, particularly the determination of specific gravity and protein-creatinine ratio, helps to identify early signs of proteinuria and reduce the concentration capacity of the kidneys. Furthermore, L. Hahn & C. Callaband (2022) emphasised that a comprehensive urine assessment together with renal ultrasound allows not only detecting early functional disorders but also visualising structural changes in tissues. Specifically, urine analysis, including the determination of specific gravity, protein-creatinine ratio and sediment, allows assessing the concentration capacity of the kidneys and the degree of proteinuria, which are vital indicators of glomerular damage. Ultrasound diagnostics of the kidneys allows detecting morphological changes such as increased parenchymal echogenicity, reduced organ size, mineralisation, or cystic formations. Particularly significant is the detection of calcification, which may indicate a long-term disorder of mineral metabolism and the progression of a chronic process.

Another promising method for assessing renal function is renoscintigraphy, a radioisotope test that provides information on perfusion, filtration capacity, and symmetry of the kidneys. This method is based on the injection of a radiopharmaceutical followed by recording its uptake, transport, and excretion by the kidneys using a gamma camera. Renoscintigraphy enables a separate assessment of the functional activity of each kidney, which is particularly valuable in cases of asymmetric lesions.

It also helps to differentiate between a decrease in glomerular filtration rate and obstructive changes or vascular disorders. Despite its limited availability in routine veterinary practice, it is considered to be extremely informative and promising, especially in specialised centres or research institutions. Thus, the combination of general clinical, visual, and functional examination methods, such as urinalysis, ultrasound, and renosciintigraphy, provides a multidimensional assessment of the functional state of the kidneys and enables the prompt detection of both structural and functional changes, which is critical for choosing the optimal tactics in the treatment of animals.

Role of FGF-23 in the diagnosis of chronic kidney disease and mineral metabolism disorders in cats

Apart from conventional biomarkers, recent studies have pointed to the significance of fibroblast growth factor-23 in the pathogenesis of chronic kidney disease. This hormone, which is produced by osteocytes and osteoblasts, is instrumental in the regulation of phosphate homeostasis and vitamin D metabolism. Its level is significantly increased in chronic kidney disease, which can have both compensatory and pathological effects on the animal's body. S. Seiler *et al.* (2009) found that in cats with chronic kidney disease, FGF-23 levels increase markedly, especially in the later stages of the disease. This is because the kidneys lose their ability to effectively excrete phosphorus, and the body tries to compensate by increasing the secretion of FGF-23. However, this compensation mechanism can have negative consequences:

- ◆ inhibition of calcitriol: FGF-23 blocks the synthesis of active vitamin D, which reduces intestinal calcium absorption;

- ◆ exacerbation of secondary hyperparathyroidism: calcitriol deficiency stimulates the production of parathyroid hormone, which can

lead to osteodystrophy and soft tissue calcification;

- ◆ accelerating the progression of chronic kidney disease: elevated levels of FGF-23 correlate with a decrease in glomerular filtration rate, which means a rapid deterioration in kidney function.

J. Lin *et al.* (2021) showed that FGF-23 levels can be used as a predictor of survival in cats with chronic kidney disease. Cats with elevated levels of this hormone have a significantly worse prognosis, as it indicates a critical disruption of phosphate balance and a systemic inflammatory response. Monitoring of FGF-23 levels in patients with chronic kidney disease can be useful for: early detection of phosphorus imbalance before clinical signs of hyperphosphatemia appear; evaluation of the effectiveness of dietary therapy (a decrease in FGF-23 levels after the introduction of a low-phosphorus diet or phosphate binders may indicate improvement in kidney health); and prediction of complications (high FGF-23 levels may signal the need for more aggressive phosphorus control). This study showed that the use of low-phosphorus diets and phosphate binders can help reduce FGF-23 levels in cats with chronic kidney disease. This confirms the hypothesis that FGF-23 is a sensitive indicator of phosphorus load.

L. Rodríguez-Osorio *et al.* (2015) found that the administration of phosphate binders such as sevelamer significantly reduced FGF-23 levels, which helps to reduce the manifestations of secondary hyperparathyroidism. This highlights the value of phosphorus control as a key factor in the management of chronic kidney disease. The researchers emphasised the role of phosphorus control at all stages of CKD as a key element of treatment. However, FGF-23 is not a routine biochemical marker available for widespread use in clinical practice. Its determination requires specialised laboratory methods, which may limit its use in veterinary medicine.

Nevertheless, further research in the field of nephrology may help to expand the possibilities of using FGF-23 as part of a comprehensive approach to the diagnostics and treatment of chronic kidney disease in cats.

Ca:P balance – clinical significance of the ratio and dietary impact

Nutrition plays a key role in the management of CKD in dogs and cats. A properly formulated diet can not only stabilise mineral metabolism but also reduce the burden on the kidneys, which helps to inhibit the progression of the disease. Specifically, controlling the content of phosphorus and calcium in the diet helps to reduce the risk of developing hyperphosphatemia and hypercalcaemia, factors that can worsen kidney function and the general condition of the animal. According to L.A. Nagode *et al.* (1996), increased phosphorus concentrations promote the development of calcium-phosphate complexes that are deposited in tissues, particularly in the kidneys, which accelerates their damage. Controlling phosphorus levels is an essential part of the treatment of chronic kidney disease in cats. However, dietary phosphorus restriction requires careful monitoring of blood calcium levels to prevent hypercalcaemia. According to scientists, a significant reduction in phosphorus levels reduces the stimulation of parathyroid hormone secretion, which can lead to increased intestinal calcium absorption under the influence of activated vitamin D (calcitriol). As a result, blood calcium levels may increase, which potentially contributes to calcification of tissues, including renal parenchyma.

P.-K. Tang *et al.* (2021) noted that phosphorus plays a key role in the pathogenesis of CKD, affecting both renal haemodynamics and systemic processes. Elevated blood phosphorus levels are associated with a decrease in glomerular filtration rate, activation of parenchymal fibrosis, and chronic inflammation, which

contributes to the progression of nephron damage. The researchers also noted that impaired phosphorus and calcium metabolism in CKD affects the musculoskeletal system. Specifically, a decrease in calcitriol levels and the development of hypocalcaemia stimulate the secretion of parathyroid hormone, which leads to bone resorption and decreased bone mineralisation. Although clinical manifestations in the form of fractures are rare, these changes indicate a systemic nature of the disease. Therewith, an excess of calcium and phosphorus contributes to vascular calcification and a subsequent decrease in their elasticity, which can have dire consequences for both the renal and cardiovascular systems. A combination of dietary restrictions and the use of phosphate binders is effective in reducing blood phosphorus levels. L. Danks (2015) pointed out the risk of hypophosphatemia in case of excessive restriction of this element, which can cause muscle weakness and impaired energy metabolism. A balanced ratio of protein and phosphorus in the diet is significant, as insufficient protein intake can lead to muscle loss, which is a negative factor for the animal's health.

Calcium has vital physiological functions, including nerve conduction, muscle contraction, and blood coagulation. R.F. Geddes *et al.* (2021) noted that cats with chronic kidney disease often have disturbances in calcium homeostasis due to phosphorus imbalance. When phosphorus levels in the blood increase, the amount of free (ionised) calcium decreases, which is a stimulus for increased secretion of parathyroid hormone. As pointed out by S.A. Brown *et al.* (2008), the decrease in ionised calcium levels is caused by its binding to excess phosphorus in the blood, which leads to the development of insoluble complexes. Elevated levels of parathyroid hormone cause calcium to be mobilised from bones, which can lead to osteodystrophy and bone resorption.

This process is widespread in cats with chronic kidney disease and requires careful monitoring. Dietary phosphorus restriction requires careful monitoring of blood calcium levels to prevent hypercalcaemia. Therefore, A.J. Felsenfeld *et al.* (2015) recommended the use of calcium-free phosphate binders, such as lanthanum carbonate or sevelamer, in cats with chronic kidney disease, which enables effective control of phosphorus levels without increasing calcium levels. These scientists stated that hypercalcaemia can also occur in chronic kidney disease, especially with prolonged use of active forms of vitamin D or impaired calcium excretion mechanisms due to reduced renal function.

Hypercalcaemia is an essential factor that can contribute to the development and progression of chronic kidney disease in cats. One of the key mechanisms of renal damage in hypercalcaemia is the deposition of calcium-phosphate complexes in the renal parenchyma. L.A. Nagode *et al.* (1996) pointed to an increased risk of calcification in the renal tubules and interstitium with elevated plasma calcium levels, which impairs renal function and contributes to the progression of chronic nephropathy. R.F. Geddes *et al.* (2021) noted that elevated calcium levels lead to renal vasoconstriction, which can reduce renal blood flow and impair renal perfusion. This contributes to the development of ischaemic nephron damage and the progression of chronic kidney disease. Furthermore, hypercalcaemia can cause nephrogenic diabetic polyuria, which is accompanied by impaired renal tubular concentration. According to S.A. Brown *et al.* (2008), excessive plasma calcium concentration reduces the sensitivity of the renal tubules to the antidiuretic hormone (vasopressin), leading to polyuria, water loss, and secondary dehydration. R. Pusoonthornthum *et al.* (2011) recommended that calcium levels in cats with chronic kidney disease should be carefully monitored, as even a moderate increase in calcium

concentration can negatively affect renal function. This is especially true for animals receiving calcium-based phosphate binders or activated forms of vitamin D. M. Coady *et al.* (2019) noted that the severity of ionised hypercalcaemia and hypocalcaemia is associated with the aetiology of this condition in dogs and cats.

The optimal ratio of calcium and phosphorus in the diet is crucial for maintaining mineral homeostasis in cats with chronic kidney disease. R.M. Korman & J.D. White (2013) noted that the balance between these elements in the diet affects their plasma levels, which is critical for preventing mineral metabolic disorders. A study conducted by S.A. Brown *et al.* (2008) showed that even with controlled phosphorus intake, excessive levels of calcium in the feed can lead to an increase in its concentration in the blood serum. This phenomenon is especially evident in cats with chronic kidney disease, as impaired renal function affects the calcium excretion mechanisms. Increased calcium intake in conditions of reduced phosphorus levels in the feed can increase hypercalcaemia, which negatively affects renal function. L.A. Nagode *et al.* (1996) emphasised that in animals with chronic kidney disease, the Ca:P ratio in the feed should be stable, as fluctuations in this ratio can lead to instability of calcium and phosphorus levels in the blood. Specifically, diets with a ratio below 1:1 can contribute to the development of hypocalcaemia, while excess calcium in the presence of phosphorus deficiency can increase the risk of calcium salt deposition in tissues. J. Stockman (2024) recommended maintaining a calcium to phosphorus ratio of 1.1:1-1.5:1, as this helps to maintain a stable level of these elements in the blood. The researcher also noted that it is not only the absolute amount of these minerals that is vital, but also their balance, as even with optimal levels of phosphorus and calcium, their incorrect ratio can cause undesirable changes in the kidneys.

Furthermore, according to R.F. Geddes *et al.* (2021), controlling the calcium-phosphorus ratio in the diet is essential to prevent the development of secondary complications. Scientists addressed the need to balance these minerals depending on the stage of chronic kidney disease, as a change in one of them can lead to compensatory mechanisms that further complicate the pathological process. Recent research by H. Kramer (2019) indicated the potential use of probiotics to reduce phosphorus absorption in chronic kidney disease. This approach has not yet been validated for veterinary practice but may be the subject of further research. This area may be especially useful for animals that do not tolerate phosphate binders or have concomitant digestive system diseases that complicate the uptake of medications.

Role of phosphate binders, ketosteroids, and probiotics

Phosphate binders are key pharmacological agents for controlling phosphorus levels in cats with chronic kidney disease. Their use helps to reduce the risk of secondary hyperparathyroidism and soft tissue calcification, which are frequent complications of mineral imbalance in renal failure. These medications work by binding phosphorus in the gastrointestinal tract, preventing its absorption into the bloodstream. According to V.J. Parker (2021), controlling blood phosphorus concentrations in cats with chronic kidney disease involves primarily reducing its intake from the diet, and in case of insufficient effectiveness, adding oral phosphate binders administered with food, such a restriction of phosphorus intake from the diet helps to reduce its systemic level and improve the metabolic state of the animal.

There are several key types of phosphate binders, which have different mechanisms of action, efficacy, and safety profiles:

- ◆ Calcium binders. They effectively reduce the concentration of phosphorus, however, as noted by G.J. Ider & J. Center (2017), their excessive use can lead to hypercalcaemia and increased soft tissue calcification, which can complicate the course of the disease.

- ◆ Aluminium hydroxide-based binders. These compounds have a high affinity for phosphorus, but their prolonged use is accompanied by a risk of aluminium intoxication. K.G. Beita *et al.* (2024) highlighted the potential toxicity to the nervous system, which limits the long-term use of this group of medicines.

- ◆ Non-complex binders. For example, sevelamer, which does not contain calcium, shows a lower risk of hypercalcaemia. At the same time, according to L. Rodríguez-Orsorio *et al.* (2015), its effectiveness in reducing phosphorus may be somewhat lower compared to calcium medicines, especially in patients with high hyperphosphatemia.

Overall, the efficacy of phosphate binders depends on the type of medicine, dosage regimen, dietary background, and the stage of renal dysfunction. They can be a vital element of complex therapy aimed at stabilising phosphorus levels and reducing the risk of calcifications. At the same time, according to E. Biasibetti *et al.* (2018), their use requires an individual approach, consideration of the animal's clinical condition, and regular monitoring of possible side effects.

This approach may be complemented by the use of ketosteroids, nitrogen-containing substitutes for essential amino acids that do not contain nitrogen but are converted into amino acids in the body. H.-Y. Chen *et al.* (2021) showed that combining ketosteroids with phosphate binders in people with renal impairment can reduce the burden on nitrogen metabolism, lower phosphorus levels, and contribute to an overall improvement in metabolic status. The researchers noted that this combination has a

synergistic effect, slowing down the progression of the disease by reducing the production of uremic toxins, improving protein balance, and controlling phosphate metabolism. This approach can also be considered as a promising one in veterinary medicine for animals with signs of hypoproteinaemia, which are not recommended to have a high protein intake but need amino acid support.

F. Perondi *et al.* (2025) investigated the effect of a probiotic feed supplement on stool and clinical parameters in dogs with chronic kidney disease and gastrointestinal disorders. They used a combination of probiotics, including *Lactobacillus* spp., *Bifidobacterium* spp., and *Enterococcus faecium*, which are widely known for their gut health support properties. According to the study, the use of the probiotic supplement helped to improve stool consistency and reduce the frequency of diarrhoea episodes. Thus, the researchers concluded that probiotic support is a promising additional component of therapy in dogs with kidney disease, especially in the presence of gastrointestinal complications.

G. Meineri *et al.* (2021) confirmed the potential role of nutraceutical support in the comprehensive treatment of chronic kidney disease in dogs. The use of combined supplements containing prebiotics, probiotics, and antioxidants was associated with a statistically significant reduction in urea, creatinine, phosphorus, and SDMA levels. Furthermore, patients with concomitant gastrointestinal disorders showed improvements in faecal score, appetite, and overall clinical condition. Although the results are encouraging, the researchers emphasised the need for further investigation to examine the mechanisms of action and confirm the long-term effectiveness of this approach. E. Ephraim & D.E. Jewell (2020) investigated the effects of dietary supplements such as betaine and soluble fibre

on metabolic changes and faecal microbiome in dogs with early chronic kidney disease. The findings revealed a positive effect of these supplements on metabolic parameters and gut microbiota. However, the researchers noted that these findings required further study to better understand the effectiveness of such dietary adjustments in the long-term management of chronic kidney disease.

Role of vitamins, nutrients, and electrolytes in maintaining mineral homeostasis

Vitamins, trace elements, and other nutrients play a significant role in the regulation of mineral metabolism and metabolic state of the body. In cats with chronic kidney disease, abnormalities in calcium, phosphorus, magnesium, potassium, and acid-base homeostasis are widespread changes that substantially affect the prognosis. Their prompt detection and correction can reduce the risk of complications and stabilise the course of the disease. R.V.A. Zafalon *et al.* (2020) studied vitamin D metabolism in dogs and cats, emphasising its significance in regulating calcium and phosphorus metabolism. The researchers noted that disorders in vitamin D metabolism can contribute to the development of various pathologies, including chronic kidney disease, due to mineral imbalances and tissue calcification.

Vitamin D is a key regulator of phosphorus and calcium metabolism. It stimulates the absorption of calcium and phosphorus in the intestines and contributes to normal bone mineralisation. According to the researchers, in cats with renal failure, the level of active vitamin D decreases, which leads to the development of secondary hyperparathyroidism and tissue calcification. This study showed that optimising vitamin D levels may be significant for controlling the progression of CKD and maintaining normal calcium and phosphorus levels in

the animal's body. Disorders of vitamin D-dependent calcium-phosphorus metabolism in CKD have not only biochemical but also structural consequences. A. Shipov *et al.* (2014) demonstrated that cats with this pathology show a marked deterioration in bone microstructure, i.e., a decrease in the density and strength of both trabecular and cortical bone. Such changes may indicate the development of renal osteodystrophy and an increased risk of pathological fractures.

Vitamin K also plays a significant role in the regulation of calcium metabolism, specifically, through the activation of osteocalcin and other Vitamin K-dependent proteins. M. Cozzolino *et al.* (2019) pointed out that deficiency of this vitamin in people with renal impairment is associated with an increased risk of vascular calcification and a deterioration in the coagulation profile. Studies showed that Vitamin K deficiency reduces the activity of matrix Gla protein, one of the key inhibitors of vascular calcification. Furthermore, an inverse relationship between Vitamin K levels and the severity of cortical artery calcification was found in patients with renal insufficiency.

Another aspect that was studied is the effect of potassium levels in cats with chronic kidney disease. D.J. Polzin (2013) noted that hypokalaemia is a frequent complication in cats with stage 2-3 chronic kidney disease, with a prevalence of up to 20%-30%. It can be accompanied by polyuria, polydipsia, muscle weakness, lethargy, and in severe cases, the development of hypokalemic myopathy. V.J. Parker (2021) recommends the addition of potassium supplements, such as potassium gluconate or citrate, to the diet to correct this imbalance. Furthermore, an inverse relationship between blood potassium concentration and the risk of developing systemic hypertension is indicated, which emphasises the significance of monitoring potassium status as part

of the comprehensive treatment of chronic kidney disease. The study found that maintaining optimal potassium levels in the diet can reduce the manifestations of hypertension in cats with chronic kidney disease, which is a crucial factor in preventing further deterioration of kidney function. The researcher also described the role of antioxidant therapy in maintaining kidney function in chronic kidney disease. Parker found that the addition of vitamin E and omega-3 fatty acids to the diet of cats with chronic kidney disease can help reduce inflammation and improve renal glomerular function. Antioxidants are essential for reducing oxidative stress, which is one of the key mechanisms of nephron damage in chronic kidney disease.

In a study on mice with induced chronic kidney disease caused by a high-fat diet, H. Lu *et al.* (2025) demonstrated that supplementation with magnesium hydride nanoparticles reduced renal fibrosis and improved renal function. These findings suggest the potential of innovative forms of magnesium as a nutraceutical to influence the progression of CKD under conditions of metabolic stress but require further evaluation in clinical settings.

The approach to the treatment of cats with chronic kidney disease also includes monitoring of acid-base homeostasis. R. Pusoonthornthum *et al.* (2011) showed that metabolic acidosis is a widespread problem in chronic kidney disease in cats and needs to be corrected by dietary intervention or the addition of alkaline agents such as sodium bicarbonate. Metabolic acidosis adversely affects calcium and phosphorus metabolism, contributing to bone demineralisation. The researchers revealed that controlling blood pH may be critical to preventing osteodystrophy in cats with chronic kidney disease. Thus, chronic metabolic acidosis can exacerbate bone health and cause catabolic processes in the body, which is critical to consider when planning treatment.

Comparison of veterinary and medical approaches

The treatment of chronic kidney disease in cats is largely based on the principles developed in human medicine. At the same time, there are substantial differences between veterinary and humane protocols conditioned by both physiological features and the availability of medical technologies and drugs. In human medicine, chronic renal failure treatment protocols include a wide range of pharmaceuticals, such as phosphate-binders, angiotensin-converting enzyme inhibitors (ACE-inhibitors), diuretics, electrolyte balance agents, and immunosuppressants for the treatment of secondary hyperparathyroidism. According to S.-H. Tsai *et al.* (2024), the use of immunosuppressive therapy can reduce parathyroid hormone activity and prevent osteodystrophy. Furthermore, L. Hahn & C. Callaband (2022) noted that medical protocols often include a kidney transplant or dialysis programme, which is the standard of care in end-stage disease. In veterinary practice, the treatment of renal dysfunction in cats is based on somewhat simplified but effective approaches. Transplantation or dialysis are rarely used due to the prohibitive cost, complexity, and lack of adequate infrastructure. The key focus is on dietary therapy with a limited phosphorus and protein content, the use of phosphate binders, both calcium and non-complex, and hydration support. Both approaches have a series of shared features. Specifically, both medicine and veterinary medicine actively use biomarkers to assess the functional state of the kidneys, monitor blood electrolyte composition and acid-base homeostasis, and prescribe vitamin D and K supplements to regulate mineral metabolism. However, according to R.F. Geddes *et al.* (2021), veterinary protocols still have less pharmacological variability and less often include invasive interventions. A promising area for both fields is the introduction of the latest technologies, such

as cell therapy. According to S. Clark (2024), the use of stem cells opens new opportunities for the regeneration of renal tissue and the potential restoration of its function. Such approaches, as well as the expansion of the panel of biomarkers for early diagnosis, can bring veterinary nephrology closer to the standards of evidence-based human medicine and substantially improve the quality of life of patients.

Promising areas of research and future interventions

Chronic kidney disease in cats continues to be one of the most pressing problems in veterinary medicine. Despite the advances made in its diagnostics and treatment, effective control over the course of the disease is often complicated by the limitations of modern methods. In this regard, promising areas of research are being developed to improve early detection, introduce new interventions, and improve the quality of life of patients. One of the key factors in successful treatment is prompt diagnostics. Current markers, such as creatinine and symmetrical dimethylarginine concentrations, have limited sensitivity in the early stages. Therefore, special attention is paid to the search for new biomarkers. S. Seiler *et al.* (2009) pointed out the potential of fibroblast growth factor-23, whose level increases before the onset of clinical symptoms. This opens opportunities for early intervention and better prognosis.

The use of stem cells is also of considerable interest. N. Tavakolidakhrabadi *et al.* (2024) demonstrated that the introduction of mesenchymal cells can promote renal tissue regeneration, reduce inflammation, and stabilise functional parameters. This approach gives hope not only for maintenance but also for partial restoration of the affected structures. Another development vector is molecular technologies, specifically gene therapy. J.L. Peek & M.H. Wilson (2023) described the possibilities of gene

modification aimed at normalising metabolic processes in the nephron. Genome editing and targeted molecular interventions are considered as a future alternative or complement to conventional approaches. Additionally, new pharmacological agents are being developed to optimise mineral metabolism. According to J. Stockman (2024), phosphatase inhibitors and stimulators of active vitamin D synthesis can reduce the risk of secondary hyperparathyroidism, improve calcium-phosphorus balance, and slow the course of the disease.

Most approaches to the treatment of chronic kidney disease in cats focus on supportive care and slowing the progression of the disease. However, recent studies are increasingly drawing attention to the possibility of actively influencing the pathogenesis of the disease through immunological mechanisms. Among the innovative strategies, the development of vaccines that can not only modulate the immune response but also potentially improve kidney function by reducing inflammation and fibrosis is of particular interest.

R. Sugisawa *et al.* (2016) found that the structure of the AIM (Apoptosis Inhibitor of Macrophage) protein in cats may be a key factor in their susceptibility to developing chronic kidney disease. Specifically, it was shown that in cats, AIM binds extremely strongly to IgM, which prevents its release in response to renal insult or toxin-induced damage. Under normal conditions, free AIM promotes the removal of necrotic cells and prevents the development of tubular obstructions, but this mechanism is impaired in cats. The use of exogenous AIM in model animals improved the clearance of cellular residues and reduced the severity of renal damage, demonstrating the potential of this molecule as a therapeutic agent for the prevention or treatment of CKD in cats. If successfully completed, this approach could be a prominent breakthrough in the prevention and

treatment of animals with chronic kidney disease, especially in case of its early detection. Thus, the future treatment of renal failure in cats is associated with an integrated approach: early diagnostics using sensitive biomarkers, cell and gene therapy, as well as pharmacological modulation of metabolism, which forms a new paradigm of veterinary nephrology.

The results of the literature analysis showed that the ratio of calcium and phosphorus plays a key role in the development and progression of chronic kidney disease in cats. An optimal balance of these elements is necessary to maintain a stable mineral metabolism, reduce the risk of secondary hyperparathyroidism, avoid hypercalcaemia, and prevent renal tissue calcification. Studies revealed that elevated blood phosphorus levels correlate with the progression of chronic kidney disease and impaired renal function. According to V.J. Parker (2021), phosphorus restriction is one of the key measures in the treatment of chronic kidney disease. Reducing phosphorus levels through dietary therapy or the use of phosphate binders can effectively regulate the level of this element and reduce the risk of complications. However, according to L.A. Nagode *et al.* (1996), excessive phosphorus reduction can cause hypercalcaemia, especially when using calcium-containing phosphate binders.

Evaluation of the impact of calcium showed that its elevated level contributes to the development of nephrocalcinosis, which increases structural damage to the nephron. According to A.J. Felsenfeld *et al.* (2015), hypercalcaemia in cats with chronic kidney disease can aggravate renal failure due to impaired glomerular filtration capacity. On the other hand, M. Coady *et al.* (2019) noted that reduced calcium levels caused by hyperphosphatemia stimulate the activity of parathyroid hormone, which leads to bone loss and impaired overall metabolism.

The use of modern diagnostic methods, such as determining the concentration of creatinine, urea, SDMA, calcium, and phosphorus, allows for effective monitoring of kidney function in cats with chronic kidney disease. This enables not only the accurate assessment of the intensity of disease progression, but also prompt adjustment of therapy, including diet and the use of phosphate binders. Complete (clinical) urinalysis and ultrasound are significant additional tools for detecting structural changes in the kidneys, which contribute to more accurate prognosis and treatment of CKD. Considering these factors in combination is a prerequisite for achieving optimal results in controlling the course of the disease and improving the quality of life of animals.

Conclusions

In the context of the purpose of this study, the current literature data on the role of the serum calcium-phosphorus ratio in chronic kidney disease in cats were analysed and the optimal approaches to the effective correction of this indicator were determined. As a result of the literature analysis, the data on the influence of mineral metabolism on the course of chronic renal failure in animals were systematised and the main mechanisms of complications were outlined. The study examined the physiological role of calcium and phosphorus, analysed the mechanisms of their imbalance in kidney damage, and determined the consequences of hyperphosphatemia and hypercalcaemia, as well as the effectiveness of their correction with dietary therapy and phosphate binders. Particular attention was paid to the evaluation of diagnostic approaches, including laboratory and visual monitoring of changes in mineral metabolism and renal function. The presented results confirmed the

significance of maintaining an optimal calcium to phosphorus ratio to prevent the development of secondary hyperparathyroidism, tissue calcification, and glomerular filtration disorders in cats with renal pathology. It was found that the interval of 1.1:1-1.5:1 is the safest and most suitable in the long-term dietary therapy of sick animals. The study systematised the methods for evaluating the effectiveness of therapy, including monitoring of biochemical parameters of blood and urine, as well as changes in the renal parenchyma by ultrasound examination.

The synthesis of available data allows forming a modern idea of the pathophysiological role of calcium-phosphorus metabolism in chronic kidney disease in cats and confirms the effectiveness of an integrated approach to the diagnostics and correction of mineral imbalance. The use of individualised dietary strategies in combination with the monitoring of marker indicators can slow down the progression of chronic renal failure and improve the quality of life of animals. Promising areas for further research include studying the long-term results of using different types of phosphate binders, the effect of vitamins D and K on calcium and phosphorus metabolism in the chronic course of nephropathy in cats, and the search for new biomarkers to assess the effectiveness of therapy in the early stages of renal failure in animals.

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Conflict of Interest

None.

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Нутріціологічний вплив кальцію та фосфору на хронічну хворобу нирок у котів: огляд літератури

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Анотація. Актуальність дослідження зумовлена значною поширеністю хронічної хвороби нирок у котів та важливою роллю порушень кальцій-фосфорного обміну в її прогресуванні та формуванні ускладнень, що потребує подальшої уваги науковців для вдосконалення підходів до лікування тварин. Метою цього огляду було узагальнення сучасних наукових даних щодо впливу кальцію та фосфору на перебіг хронічної хвороби нирок у котів та формування рекомендацій щодо нутріціологічної підтримки. Використовувався систематичний підхід до пошуку та аналізу наукової літератури. Проведено аналіз результатів сучасних досліджень з питань діагностики хронічної нефропатії в дрібних м'ясоїдних тварин, клінічних рекомендацій та експертних висновків у галузі ветеринарної медицини. Встановлено, що надлишковий вміст фосфору в раціоні тварин прямо корелює з прискоренням прогресування хвороби, розвитком вторинного гіперпаратиреозу, кальцифікацією м'яких тканин та погіршенням функціонального стану нирок. Водночас надмірне зниження концентрації фосфору може спричиняти гіперкальціємію, втрату апетиту, блювання та інші метаболічні порушення. Оптимальним для раціону тварин із хронічною хворобою нирок є співвідношення кальцію до фосфору в межах 1,1:1-1,5:1. Розглянуто роль сучасних біомаркерів, таких як симетричний диметиларгінін та фактор росту фіброblastів-23 у ранній діагностиці та моніторингу хронічної хвороби нирок. Проаналізовано ефективність дієтичної терапії, зокрема застосування фосфатзв'язуючих препаратів, активних форм вітаміну D, пробіотиків, антиоксидантів і засобів для корекції кислотно-лужного балансу. Огляд даних літератури підтверджує, що ефективний нутріціологічний менеджмент є ключовим фактором у контролі хронічної хвороби нирок. Отримані результати підкреслюють важливість розробки оптимальних дієтичних стратегій, що враховують як рівень фосфору, так і співвідношення кальцію до фосфору в сироватці крові. Це дозволить уповільнити прогресування захворювання та покращити якість життя котів із хронічною хворобою нирок

Ключові слова: мінеральний гомеостаз; ветеринарна дієтологія; електролітний обмін; метаболічні порушення; гіперкальціємія; гіперфосфатемія; фосфатбіндери