



# Nutritional management of feline chronic kidney disease



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## KEY POINTS

The goals of dietary therapy in chronic kidney disease depend on the IRIS stage:

- ◆ In Stage 2 and early Stage 3 dietary changes are made to counteract the mal-adaptations that lead to intrinsic kidney damage in an attempt to slow progression
  - Management of hyperphosphatemia by dietary phosphate restriction
  - Management of proteinuria
  - Management of hypokalemia
  - Management of hypertension
- ◆ In later stages of CKD (late Stage 3 and Stage 4), the priority changes to correcting imbalances that give rise to the uremic syndrome and influence quality of life of the animal. These include:
  - Minimizing azotemia
  - Limiting hyperphosphatemia by dietary restriction and intestinal phosphate binders
  - Fighting against anorexia to maintain sufficient energy intake
  - Managing metabolic acidosis

## ■ Introduction

The composition of the diet is important for maintaining homeostasis in cats suffering from chronic kidney disease (CKD) and helps to improve the animal's quality of life. In some cases, dietetic measures can prevent the progression of CKD to the stage where the development becomes fatal unless a renal substitution treatment is performed.

## ROYAL CANIN VIEWPOINT

Recommendations for dietetic and other forms of medical treatment and supportive care should be adapted to the needs of each patient according to the clinical presentation and the laboratory test results. CKD is progressive and dynamic, it is therefore necessary to conduct regular clinical examinations and laboratory analyses and adapt the treatment to the changes observed for it to remain effective.

### ■ Fighting against anorexia and maintaining sufficient energy consumption

In the advanced stages of CKD, the high accumulation of nitrogen waste products has an irritant effect on the mucous membranes. The cat suffers from nausea and vomiting and tends to lose its appetite. If this situation persists for a while, the animal undergoes major weight loss and its life expectancy is shortened (*Figure 1*).

The animal's energy intake should be adapted to its needs and therefore its weight and body condition score should be assessed regularly. Cats generally need between 50 and 60 kcal/kg/day. Since lipids provide around twice as much energy as carbohydrates, per gram consumed, they increase the food's energy density, which makes it possible to decrease the volume of the ration and thus reduce the risks of nausea and vomiting.

It may be necessary to try several different foods before selecting the one the cat prefers. It is

sometimes worthwhile heating up the food (in the case of moist food) and giving it to the animal in small quantities at very regular intervals. The cat's appetite can also be stimulated by adding flavoring substances to the basic diet.

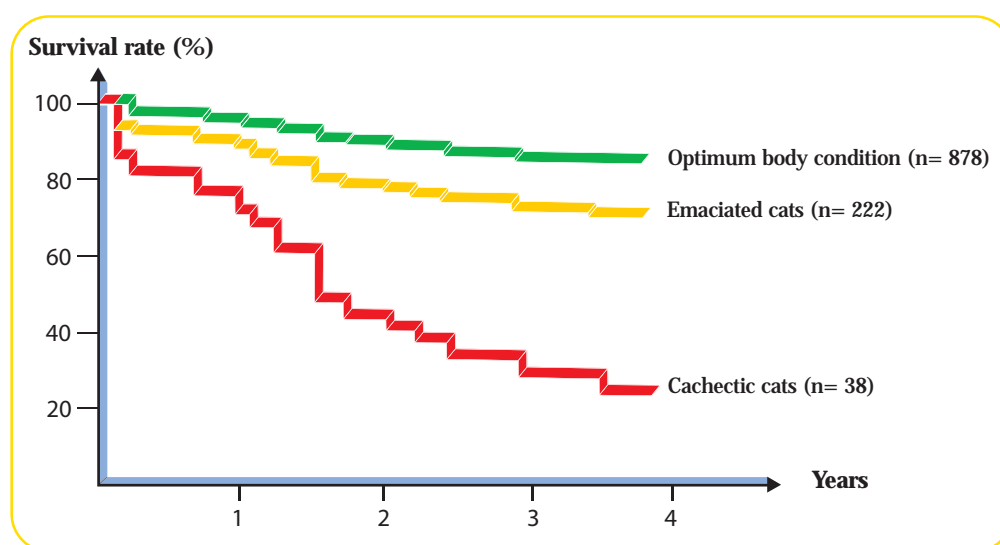
### ■ Reasons for restricting protein intake in chronic kidney disease

There are two reasons for restricting the protein level in a diet formulated for kidney disease:

- To minimize the azotemia/uremia - most appropriate for late Stage 3 and Stage 4 CKD cats.
- To decrease proteinuria mediated by glomerular hyperfiltration, a mal-adaptive response to CKD which contributes to progressive renal injury. This is the reason for reducing protein intake in Stages 2 and 3 of CKD.

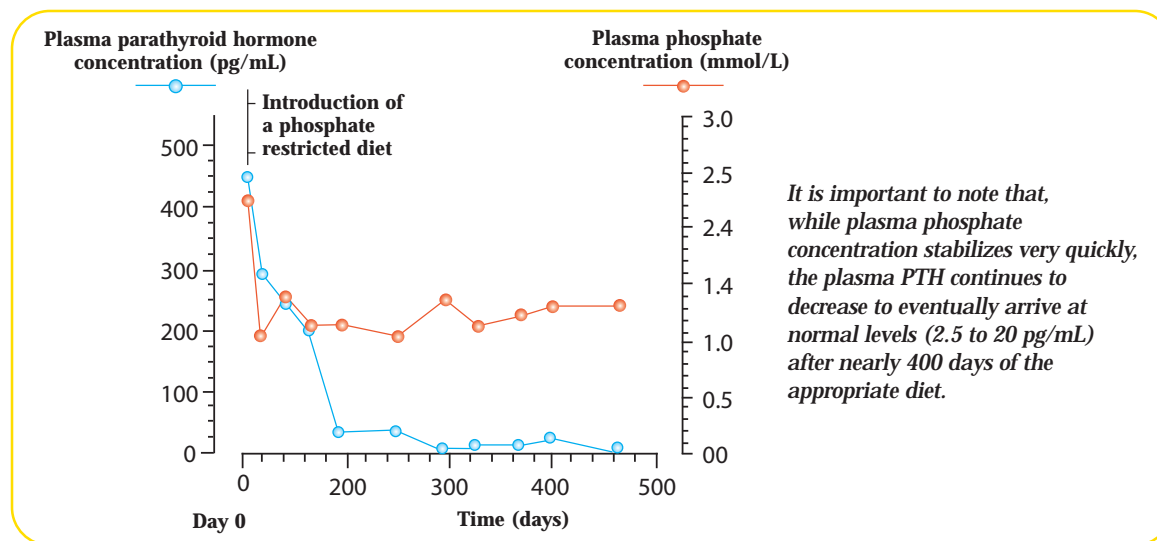
Once the CKD reaches the uremic phase (end of Stage 3/beginning of Stage 4 in the IRIS classification), it is recommended to reduce the protein intake to ensure that the cat's well-being is not too adversely affected by uremia. Measuring the plasma urea to creatinine ratio is useful in assessing the animal's response to protein restriction (decrease in the production of nitrogen waste products). In dogs, reference values have been recommended depending on protein intake, but none have been published for cats.

The efficacy of reducing protein intake as a treatment for proteinuria is highly controversial



**Figure 1.** Body condition and life expectancy in the cat (1).

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**Figure 2.** Impact of a dietetic renal food on plasma phosphate concentration (red dots) and on plasma PTH (blue dots) in a cat presenting with CKD (2).

in the cat and dog. In experimental rat studies, this strategy has been shown to help to slow the progression of renal lesions (3), so that protein restriction has also been recommended for other species. Similar studies have been conducted in cats, but those whose protein intake was most limited (2.7 g/kg/day) presented with signs of protein malnutrition and a decrease in albuminemia at the end of the study (4). A subsequent study failed to highlight any beneficial effect of protein restriction (5.2-5.3 g/kg/day) when azotemia is limited (Stage 1 or 2 of CKD according to the International Renal Interest Society or IRIS classification) (5).

It seems clear from the feline model studies published that avoidance of high protein diets, particularly those formulated with animal protein would be advisable in cats with CKD.

Another dietary approach to limiting proteinuria is through dietary supplementation with n3-PUFAs. In dogs with CKD, the administration of a diet greatly enriched with long-chain omega-3 polyunsaturated fatty acids (PFA) slows the progression of deterioration in the GFR (glomerular filtration rate) (6). Although other studies will be required to determine the impact of omega-3 PFA on the progression of CKD in cats, it is no doubt very important to provide the animal with an intake of long-chain omega-3 PFA

(eicosapentenoic acid [EPA] and docosahexenoic acid [DHA]), since cats are deficient in delta-6-desaturase, which compromises their synthesis. Fish oil should therefore be contained in foods for cats with CDK.

#### ■ Preventing secondary renal hyperparathyroidism by controlling hyperphosphatemia

When the GFR decreases, if phosphorus consumption remains the same, there is a discrepancy between the amount of phosphate excreted daily in the urine and the amount consumed and phosphate accumulates in the body, thus promoting hyperparathyroidism and the progression of renal lesions.

Initially, the task is to reduce the dietary consumption of phosphorus by means of an appropriate food to control PTH secretion (**Figure 2, Table 1**). At Stage 3/4, it is, however, unlikely that a feline renal diet will be sufficient to attain this objective and it may be worthwhile introducing intestinal phosphorus chelators (**Table 2**) in order to reduce the bioavailability of dietary phosphorus. The phosphorus chelators interact with the food and it is therefore important to mix them into the food for maximum efficacy. Undesirable effects linked to dietary phosphorus restriction are rare. It is recommended to measure a cat's plasma phosphate concentration and plasma calcium concentration

## ROYAL CANIN VIEWPOINT

**Table 1.**  
Tolerable levels of plasma phosphate concentration depending on stage of Chronic Kidney Disease (CKD)

- **During Stage 2 CKD**, post-treatment plasma phosphate concentration must be less than 1.45 mmol/L (4.5 mg/dL), but no less than 0.8 mmol/L (2.5 mg/dL).
- **For Stage 3 CKD**, post-treatment target value is < 1.61 mmol/L (5.0 mg/dL). In advanced Stage 3 cases, it may be necessary to combine intestinal phosphorus chelators with a phosphorus-poor diet to achieve the targeted value.
- **For Stage 4 CKD**, post-treatment plasma phosphate concentration should remain at < 1.93 mmol/L (6.0 mg/dL) and it is unlikely that this result may be obtained by solely restricting dietary phosphorus intake.

*Ranges recommended by IRIS*

(preferably ionized calcium) regularly, once every two or three months. Cases of hypercalcemia have occasionally been reported (7).

### ■ Adapting sodium levels to the risk of hypertension

Most diets aimed at cats with CKD contain less sodium than maintenance foods for adult cats (**Table 3**). This formulation is based on the hypothesis that with a reduced functional renal mass, it is more difficult to maintain sodium homeostasis and sodium retention can increase systemic arterial blood pressure. However, some observations have raised doubts as to the value

**Table 2.**  
Phosphorus chelators currently available

- Aluminum carbonate
- Aluminum hydroxide
- Aluminum oxide
- Calcium carbonate (+/- Chitosan)
- Calcium acetate
- Lanthanum carbonate
- Sevelamer hydrochloride (polyallylamine hydrochloride polymeric hydrogel)

of a systematic restriction in dietary sodium in cats presenting with spontaneous CKD:

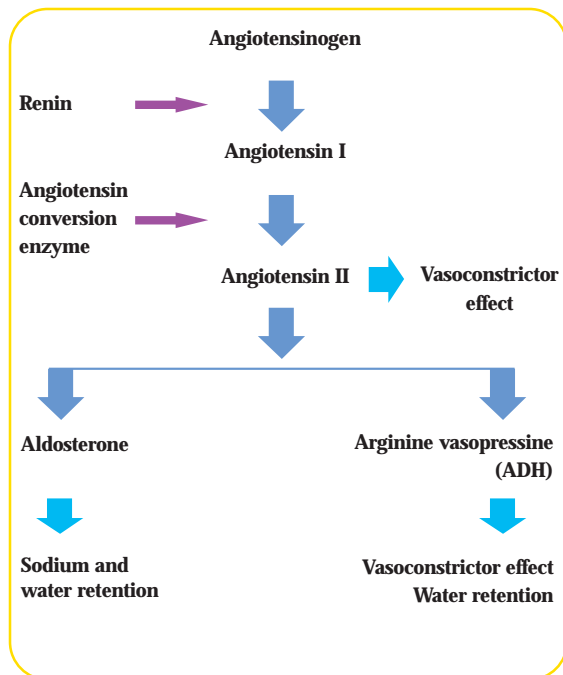
- Cats suffering from CKD tolerate an increase in ingested sodium chloride of up to 200 mg/kg/day body weight for 7 days (food containing 1.27% of sodium, *i.e.* 2.8 g sodium for 1000 kcal) with no increase in arterial blood pressure (8).
- In experimental models of hypertension, a reduction in sodium consumption leads to an increase in urinary potassium losses and slight hypokalemia with a more pronounced activation of the renin-angiotensin-aldosterone system (RAAS) (**Figure 3**); the pathological activation of the RAAS can have harmful effects on the renal function and exacerbate renal fibrosis in some feline renal disease models (9).

**Table 3.**  
Dietary sodium intake in adult cats during maintenance (*National Research Council, 2006*)

Minimal requirements (mg)			Recommended requirements (mg)			Upper safety limit (g/kg dry matter)
(mg/kg DM)	(mg/1000 kcal ME)	(mg/kg BW <sup>0.67</sup> )	(mg/kg DM)	(mg/1000 kcal ME)	(mg/kg BW <sup>0.67</sup> )	
650	160	16	680	170	16.7	> 15 g

mg/kg DM: Amount per kg of dry matter, assuming that the food's energy density is 4000 kcal of metabolizable energy/kg  
 BW: Body Weight: values for amounts per body weight<sup>0.67</sup> are calculated for a lean cat with an energy intake of 100 kcal per kg of body weight<sup>0.67</sup> ME: Metabolizable energy

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**Figure 3.** Activation of the renine-angiotensin-aldosterone system (RAAS).

Studies should be conducted to determine whether the reduction in ingested sodium helps minimize the slight chronic increase in systemic arterial blood pressure detected in most cats suffering from CKD and whether dietary sodium restriction has a beneficial effect in cats receiving anti-hypertensive medication in terms of the degree of blood pressure control achieved.

### ■ Preventing hypokalemia

The association between CKD and hypokalemia is relatively specific to cats. (In dogs or in human beings, the loss of functional nephrons brings with it a greater risk of hyperkalemia). In 20 to 30% of cats with CKD, the functional adaptation of remaining functioning nephrons leads to excessive losses of potassium in the urine resulting in hypokalemia (10). Correcting these electrolyte abnormalities, especially when the plasma potassium concentration is less than 3 mmol/L, is clinically beneficial. Severe hypokalemia and the associated myopathy can be avoided in cats by not feeding acidifying diets and ensuring a diet is fed that is replete in potassium and magnesium. For the majority of cats with CKD, if a diet formulated for kidney disease can be fed, the use of potassium supplements should not be

necessary once the initial problem of hypokalemia has been treated and the cat is eating well again.

### ■ Fighting against the risk of metabolic acidosis

Objective signs of metabolic acidosis generally become visible at advanced Stage 3 and early Stage 4 of CKD. The part played by metabolic acidosis in the bone pathology associated with CKD is well known in human medicine, but has not yet undergone study in cats.

The management of metabolic acidosis is centered round administering an alkalinizing agent by the oral route. The animal's response to the treatment can be monitored by repeat measurements of the plasma bicarbonate concentration, which should ideally lie within the physiological ranges.

The choice of alkalinizing agent depends on several parameters: its palatability, the possible presence of hypertension (in which case sodium supplements are contra-indicated), hypokalemia (for which potassium salts are recommended) and hyperphosphatemia; in this latter case calcium salts may be prescribed because of their ability to bind phosphorus in the food and intestinal secretions. Metabolic acidosis increases the risk of hypokalemia: a treatment using potassium gluconate or potassium citrate is therefore indicated.

### ■ Other dietary strategies aimed at slowing the progression of renal lesions

Dysfunctioning endothelial cells are involved in the pathogenesis of CKD. In human medicine, a field in which active research is conducted to fight against dysfunctioning endothelial cells, some strategies mentioned below have proven worthwhile. It remains to be determined whether these measures are beneficial in cats with CKD and when they should be applied.

- **Enriching the diet with anti-oxidants** (vitamin E, vitamin C, taurine, lutein, lycopene, beta-carotene, etc.) to minimize oxidative stress, which contributes to the progression of CKD lesions. Flavanols, for example, can play a protective role in areas of necrosis occurring in the glomeruli, because of alternating periods of ischemia and reperfusion, owing to the circulation problems that accompany CKD.

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- **L-arginine supplements** to stimulate the production of nitric oxide (NO), which promotes local vasodilatation.

### ■ Role of fiber

Fermentable fibers have appeared recently in the dietetic treatment of CKD. They represent a source of carbohydrates for the gastrointestinal bacteria, which use urea as a source of nitrogen for their growth. Since the excretion of nitrogen in the feces increases in accordance with the bacterial mass, it has been proposed that an increase in bacterial mass may help reduce uremia. However, classic uremic toxins, unlike urea nitrogen, are medium-sized molecules, too large to easily pass through the membrane barrier. It is therefore unlikely that these toxins may be used by the bacteria to cater for their nitrogen needs. Conversely, the beneficial effects of fermentable fibers can help to regulate the digestive disorders that accompany CKD.

### ■ Conclusion

The diet has an important role to play in the treatment of feline CKD. It is essential to adapt the diet to the animal's needs and to understand the objectives behind the dietetic treatment at the different stages of the disease.

**Early stages (Stage 2 and 3 of CKD)\*** the principles behind a dietetic treatment are:

- Minimizing the phosphorus intake: this prevents

the risk of abnormal phosphorus retention and slows the progression of renal lesions.

- Limiting proteinuria by avoiding ingestion of high quantities of protein from animal sources and dietary supplementation of n3-PUFAs. The beneficial effects of omega-3-PUFA supplementation in cats has yet to be studied but the theoretical basis of this approach is sound.
- A potassium supplement: necessary in cats presenting with hypokalemia.

**Advanced Stage 3 and Stage 4 of CKD\***, the diet aims above all to improve the cat's quality of life during the uremic phase.

- The protein intake must be minimized further to reduce the accumulation of nitrogen waste products. It is important to take into consideration the source of the protein: highly digestible proteins minimize the release of nitrogen by-products into the blood.
- Alkalinizing agents may help to prevent metabolic acidosis, which contributes to secondary renal osteodystrophy (leading to bone pain) and the animal's loss of appetite.
- It may become necessary to treat hypokalemia with potassium supplements.
- The use of intestinal phosphorus chelators helps to minimize the extrarenal effects of hyperphosphatemia and hyperparathyroidism, especially renal osteodystrophy and vascular calcification, which affect the animal's quality of life.

\* Refer to the IRIS staging system on page 21.

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