

Reevaluating Phosphate Use in Pet Food: Nutritional & Environmental Concerns

Britta Dobenecker, Dr.med.vet., DECVCN, Ludwig-Maximilians-Universität München, Munich, Germany

Phosphate (P) is an essential nutrient for plants and animals. Both deficient and excess intake can cause severe health issues in plants and animals. The precondition for absorption, again in both plants and animals, is the solubility of the phosphate source.

Phosphates are usually categorized according to their source: On the one hand there is P from animal tissues and plants. Here, the term organic phosphate (oP) is often used as a synonym even if it is not entirely correct from a technical point of view. In oP sources P is associated with a carbon-based molecule and bound in tissues or cell structures, in plants it is stored in the form of phytic acid. Organic P has predominately a low or moderate solubility. On the other hand, most inorganic phosphate (Pi) sources, i.e., P from minerals or rock, are highly soluble and therefore highly available. Pi is a key fertilizer and source of phosphoric acid (ortho- and polyphosphates, i.e., derivatives of H_3PO_4) which is important to the feed, food, and beverage industry due to the many functional properties Pi exhibits. To this day, Pi containing additives are still categorized as GRAS (Generally Recognized As Safe) substances despite the potential harm they can cause. Overall, P, especially Pi, is a most relevant element in agriculture and livestock farming. The discussion about the limited global resources of this element should also be included in the assessment of the extensive use of inorganic phosphate because phosphate rock is ranked one of the 20 most scarce raw materials globally. In Brazil, over 90% of the mined phosphate ores come from igneous complex carbonatite deposits, accounting for 2% of the estimated worldwide reserves.

The high amount of mineral phosphate used, obviously has an impact not only on the available resources but also on the input into the environment, i.e., pollution. Regarding environmental input (eutrophication), pet food has to be taken into account: it is ranked as third major P input in certain urban regions, accounting for 14% of total P input, and therefore more than twice as much as turf fertilization and atmospheric deposition combined. Globally, the annual amount of P from dog and cat waste is estimated to be as high as 670.000.000.000.000 kg. In contrast to human waste (water) there is a direct input in soil and water in this case, which makes recycling more difficult. Overall, possible measures to reduce the high consumption of P resources and emissions are being discussed, such as recycling, re-use, and recovering (from waste, especially urine), restricted use, less meat production, and fertilizing with ash from waste and bones etc.

Unlike in livestock feeding, where efforts are made to minimize the use of P, the use of Pi is not spared in the production of commercial food for humans and pets. High quantities of Pi with above average solubility and therefore availability are used predominantly due to the many positive properties, e.g., palatability enhancement, water binding, texturizing, and preservation. Processed food and pet feed (including snacks, beverages, dental hygiene products) contains surplus of added Pi due to the use of P-containing additives such as phosphoric acid and its derivatives, ortho- and polyphosphates. Due to existing regulations on labeling (US, EU), a conclusive estimate of the Pi content is impossible. However, in human and pet nutrition, an excessive P intake is frequently seen: already a decade ago, at least 1/3 of the U.S. adult population consumed more than recommended. Due to the correlation between the amount of added Pi and the fraction of highly soluble P in food and feed, the fractionation of P is a valid method to estimate the amount of added Pi. Using the method of P fractionation (Lineva et al. 2019), it has been demonstrated that most examined pet food products purchased in the EU supplies an excessive amount of highly available P. That a high intake of P can disrupt the

phosphate metabolism with adverse effects especially on kidneys, skeleton, and cardiovascular system has been demonstrated repeatedly since ~100 years ago. Among other things, high amounts of soluble, i.e., available, dietary P can cause an increase of postprandial serum P concentrations, renal P excretion, and bone resorption.

Chronic kidney disease (CKD) has a high and increasing prevalence in our human, canine and feline populations. It is even ranked as the main cause of death in (senior) cats. It can be hypothesized that cats are susceptible for kidney damage because of their low water consumption and production of concentrated urine, which increases the urinary P concentrations even further. As CKD can unfortunately only be diagnosed at an advanced disease stage, it has to be accepted that a severe damage of functional renal tissue exists even in „early“ diagnosis and that there is a high prevalence of undiagnosed individuals with severe kidney damage in the populations. Because in CKD the renal P excretion is increasingly compromised but crucial to limit hyperphosphatemia and to slow down disease progression, a limited P load is the major goal of dietary treatment. It is not only the absolute amount of P in the daily ration that is decisive here, but also the phosphate sources used. The complete avoidance of Pi is recommended for individuals with kidney damage. If CKD is diagnosed, total dietary P concentration should be reduced to such an extent that hyperphosphataemia is avoided. If the serum P level remains elevated despite eliminating Pi from the feed and lowering the amount of total dietary P, P binders may have a positive effect. To avoid P deficiency, which can occur in growing dogs, for example, and possibly as a consequence of excessive dietary CKD treatment, it is recommended to use organic sources such as bone meal instead of Pi to meet daily P requirements. Because an excessive supply with soluble Pi but not oP can cause adverse effects, the use of highly soluble Pi should be avoided especially in CKD patients and seniors. It remains questionable if the use of up to 1g highly available Pi per Mcal metabolizable energy is unconditionally safe for long-term use in our pet populations, which includes individuals with undiagnosed CKD. To enable an informed assessment of P supply at all, labelling of Pi sources and amounts in processed food and feed (incl. dental health products) should be mandatory.

References

- Baker, L. A. (2011). Can urban P conservation help to prevent the brown devolution?. *Chemosphere*, 84(6), 779-784.
- Besterekov, U., Nazarbek, U., Zhuldyzbaeva, S., Pochitalkina, I., & Raiymbekov, Y. (2021). Resource indicators of phosphate raw materials in the world. *Industrial technology and engineering*. (4), 37-40.
- Böswald, L. F., Kienzle, E., & Dobenecker, B. (2018). Observation about phosphorus and protein supply in cats and dogs prior to the diagnosis of chronic kidney disease. *Journal of animal physiology and animal nutrition*, 102, 31-36.
- Calvo, M. S., & Uribarri, J. (2013). Public health impact of dietary phosphorus excess on bone and cardiovascular health in the general population. *The American journal of clinical nutrition*, 98(1), 6-15.
- Chang, A. R., Lazo, M., Appel, L. J., Gutierrez, O. M., & Grams, M. E. (2014). High dietary phosphorus intake is associated with all-cause mortality: results from NHANES III. *The American journal of clinical nutrition*, 99(2), 320-327.
- Cordell, D., Rosemarin, A., Schröder, J. J., & Smit, A. L. (2011). Towards global phosphorus security: A systems framework for phosphorus recovery and reuse options. *Chemosphere*, 84(6), 747-758.

Cowan, N., Brownlie, W., Tomlinson, S., Carnell, E., Drewer, J., Dragosits, U., ... & Spears, B. M. (2024). A global assessment of nitrogen and phosphorus generated in the waste streams of domesticated cats and dogs. *Sustainable Environment*, 10(1), 2415181.

Dobenecker, B., Webel, A., Reese, S., & Kienzle, E. (2018). Effect of a high phosphorus diet on indicators of renal health in cats. *Journal of feline medicine and surgery*, 20(4), 339-343.

Dobenecker, B., Hertel-Böhnke, P., Webel, A., & Kienzle, E. (2018). Renal phosphorus excretion in adult healthy cats after the intake of high phosphorus diets with either calcium monophosphate or sodium monophosphate. *Journal of Animal Physiology and Animal Nutrition*, 102(6), 1759-1765.

Dobenecker, B. (2021). Phosphate intake with complete food and diets for chronic kidney disease available on the German market. *Tierärztliche Praxis Ausgabe K: Kleintiere/Heimtiere*, 49(04), 247-254.

Dobenecker, B., Kienzle, E., & Siedler, S. (2021). The source matters—effects of high phosphate intake from eight different sources in dogs. *Animals*, 11(12), 3456.

Dobenecker, B., Reese, S., & Herbst, S. (2021). Effects of dietary phosphates from organic and inorganic sources on parameters of phosphorus homeostasis in healthy adult dogs. *PLoS One*, 16(2), e0246950.

Erem, S., & Razzaque, M. S. (2018). Dietary phosphate toxicity: an emerging global health concern. *Histochemistry and cell biology*, 150(6), 711-719.

Georgievskiy A.F., Bugina V.M. *RUDN Journal of Engineering Researches*. 2020;21(3):197–207

Kiefer-Hecker, B., Kienzle, E., & Dobenecker, B. (2018). Effects of low phosphorus supply on the availability of calcium and phosphorus, and musculoskeletal development of growing dogs of two different breeds. *Journal of animal physiology and animal nutrition*, 102(3), 789-798.

Kiefer-Hecker, B., Bauer, A., & Dobenecker, B. (2018). Effects of low phosphorus intake on serum calcium, phosphorus, alkaline phosphatase activity and parathyroid hormone in growing dogs. *Journal of animal physiology and animal nutrition*, 102(6), 1749-1758.

Lineva, A., Kirchner, R., Kienzle, E., Kamphues, J., & Dobenecker, B. (2019). A pilot study on in vitro solubility of phosphorus from mineral sources, feed ingredients and compound feed for pigs, poultry, dogs and cats. *Journal of animal physiology and animal nutrition*, 103(1), 317-323.

MacKay, E. M., & Oliver, J. (1935). Renal damage following the ingestion of a diet containing an excess of inorganic phosphate. *The Journal of experimental medicine*, 61(3), 319.

Ramos, S. J., Dinali, G. S., de Carvalho, T. S., Chaves, L. C., Siqueira, J. O., & Guilherme, L. R. (2016). Rare earth elements in raw materials and products of the phosphate fertilizer industry in South America: Content, signature, and crystalline phases. *Journal of Geochemical Exploration*, 168, 177-186.

Steffen, C., & Dobenecker, B. (2023). The phosphate additives phosphoric acid and sodium phosphate lead to hyperphosphatemia as well as increased FGF23 and renal phosphate excretion in healthy cats. *J. Urol. Ren. Dis*, 8, 1338.

Steffen, C., & Dobenecker, B. (2024). The Dietary Cation Anion Balance Exacerbates the Effects of Inorganic Phosphates on Parameters of Phosphate Metabolism in Cats. *Journal of Veterinary Healthcare-3* (3), 1-13.