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# Balanced Feeding Assists Healthy Growth in Dogs

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## Abstract

The main factors affecting the healthy growth of dogs, such as proper calcium and phosphorus supplies as well as energy consumption and its impact on the growth curve, are discussed in the context of nutrient requirements of dogs compared to those in man. Published data on the consequences of excessive or deficient mineral supply and growth intensity also will be reviewed.

**Glossary of Abbreviations**

**BCS:** Body Condition System
**BW:** Body Weight
**Ca:** Calcium
**DOD:** Developmental Orthopedic Diseases
**NRC:** National Research Committee
**P:** Phosphorus
**PTH:** Parathyroid Hormone

## Calcium and Phosphorus

Dogs have higher needs for some minerals, such as calcium (Ca) and phosphorus (P), and seem to be more sensitive to deficient or excessive nutrient supplies when compared to other species, especially man. This is true for adult dogs, but specifically for dogs during growth.

The existing requirements for dogs and humans may differ in format and heights according to the recommendations you choose, but one thing becomes obvious: Adult dogs seem to need considerably more calcium than adult humans (Table 1).

## Calcium Requirements and Calcium Deficiency

Why do dogs need so much more Ca?

It is a fact that the body needs to keep the blood Ca levels in narrow ranges. Therefore, a sensitive regulation is mandatory to avoid far-reaching, and possibly life-threatening, hypo- or hypercalcaemia. Next to increasing digestibility, which does not seem to occur at least in adult dogs, a deficient supply of Ca and P may be counteracted using the body stores, i.e., activating bone resorption. Currently, it is not fully known how long it takes a dog to efficiently activate bone resorption. It also is within the realms of possibility that skeletal reservoirs with different availability, i.e., for *ad hoc* or for long-term use, leave the dog widely unaffected by day-to-day variability of mineral supply. However, even adult dogs are susceptible to osteomalacia as a consequence of prolonged Ca deficient feeding.[4, 5]

What happens to humans with a long-term calcium deficient diet?

It is not really farfetched to declare a widely spread unbalanced and incomplete nutrient supply in some Western countries with increasing consumption of food rich in (animal) fat and carbohydrates but low in vitamins and certain minerals. For example, the 2008 nutrition report of the Deutsche Gesellschaft für Ernährung documented deficient intake of some vitamins, fiber and Ca in most children and adolescents. However, reports of clinically and obviously relevant developmental problems due to mineral-deficient nutrition are scarce. Even the Ca deficiency in combination with the often (too) high body weight and too steep growth curve do not lead to a relevant incidence of developmental orthopedic diseases (DOD) in children comparable to dogs.

A growing dog will most probably develop severe clinical skeletal problems when fed amounts of Ca sufficient for children of a similar body weight. Growing dogs may be more sensitive due to the higher speed of growth and, therefore, higher needs for tissue accretion. Dogs are multipar and, therefore, it is comprehensible that reproducing bitches have higher needs for Ca and P. But even the recommended allowance for maintenance for an adult man, including safety margins, is far less than the net requirement of a dog of the same body weight. The question is not if a dog provided with the amount of Ca sufficient for humans (calculated on BW or per energy unit basis) will develop clinical problems but rather how fast this will happen.

**Table 1: Daily Calcium Requirement Based on the Same Body Weight During Maintenance, Growth and Reproduction in Dog and Man**

| **Maintenance** | **Adult Dogs** | **Adult Man** | **Factor** |
|---|---|---|---|
| mg/d (70kg BW)* | 3146 | 1000 | 3.1 |
| **Growth** | **Growing Dogs (<14w)** | **Children (~2y)** | **Factor** |
| mg/d (12kg BW)# | 4384 | 600 | 7.3 |
| **Reproduction** | **Adult Dogs** | **Adult Women** | |
| mg/d (70kg BW)* | 19844 | 1000 | 19.8 |

* Calculated for a dog with a body weight of 70kg due to only one recommendation for an average adult man (estimated mean body weight of 70kg)
\# Calculated for an actual body weight of 12kg and an adult body weight of 70kg

Recommendations from Deutsche Gesellschaft für Ernährung,[1] NRC[2] and FEDIAF.[3]

Do we have enough knowledge and data to postulate whether such a huge difference exists? If so, is this difference due to a less efficient Ca digestibility in the dog? How about regulation and adaptation to the respective form of the diet or the origin of the existing data, i.e., the design of digestion trials? Do we have to take into account how the data were established?

First, we should have a look into the existing requirements including safety margins and safe upper limits. Even though many papers in this field are published and on first sight the information status seems to be excellent or at least sufficient due to limited information, for example, on bioavailability of Ca and P, the deriving recommendations on Ca and P supply remain rather speculative. How is that possible?

Presumably many factors may affect the bioavailability of the major minerals, predominantly Ca and P, in dogs. Among these are age, performing stage (growth, maintenance, reproduction, etc.), body weight, and breed of the animal. Another affect comes from dietary factors, such as diet composition, source of minerals, concentration of the respective mineral in the diet, as well as the concentration of other minerals known to influence the digestibility of the mineral in question (interactions determined, for example, by the Ca/P ratio), processing of the diet and last, but not least, the duration of feeding the respective diet, the least understood and realized factor. The compilation of sound data on Ca and P bioavailability, therefore, includes the uniformity of trials or, better, the knowledge and quantification of all factors that may modify the results.

A recently submitted work comprising a meta-analysis of digestibility trials in dogs and cats on Ca and P digestibility[6] yields much better insight on the bioavailability of Ca and P in dogs and cats. The Ca metabolism in adult cats and dogs is regulated by parathyroid hormone (PTH), calcitonin and metabolites of vitamin D. Unlike hindgut fermenters, such as horses or rabbits, dogs and cats are thought to balance their Ca status through the regulation of digestibility and not, for example, through excretion of excess Ca via the kidneys. According to the current scientific consensus, cats and dogs are able to decrease the intestinal mineral absorption in case of a dietary excess and to increase it in case of deficient supply[7] in order to maintain equilibrium. However, the meta-analysis showed that this is not the case.[6] Two explanations are possible in this case: The duration of the trials commonly used to determine the Ca balance is too short to initiate regulation of Ca digestion, or the dogs (at least at maintenance) are completely unable to adapt the digestibility of Ca comparably to other species, such as horses and rabbits.

First: The duration of the trials and therefore the exposure to the diet may be too short for the dog to modulate the digestibility. Normally, digestion trials last four to eight weeks. If there is no adaptation of Ca digestibility in this period, as shown from Mack, et al.,[6] the blood levels must be regulated through bone resorption in case of deficient Ca supply and Ca excretion via urine in case of Ca excess, respectively. In this case, data of those "normal" balancing trials are quite useless in regard to the resulting data of Ca digestibility — just because they are too short.

Second: Dogs are not able to adapt the digestibility of Ca, even after a longer period of a deficient or excessive Ca supply. This would cause depletion of the skeletal stores and result sooner or later in possible clinical problems.

Using the wolf as progenitor as a basic model for understanding the background of Ca metabolism may help to understand that the availability of Ca in the diet is important. All primal, natural diets consisted of whole prey animals. Therefore, there was no need to upregulate the efficiency of Ca digestibility as more than enough bony material was available. Even the much higher need for Ca of growing and reproducing dogs may be explained by this image: The alpha dogs feed on the more valuable meaty parts, whereas the bitches are next and the youngsters feed on a higher proportion of skeleton, thereby (involuntarily) increasing their Ca intake. The same background may be responsible for the inability to synthesize vitamin D in dogs and cats or better the lack of need to be able to do so.

As a consequence of these factors affecting the bioavailability of the major minerals Ca and P, it is mandatory to establish the maximum possible uniformity (for comparability reasons) regarding the digestibility trials taken into account (animals, diet, trial design, etc.) in order to have valid data to base the recommendations for these minerals. If dogs are able to adapt the digestibility of Ca, and if so, how long it takes to do so, needs to be clarified, and therefore, more research is required. This knowledge is the basis for a healthy feeding of dogs, especially in case of insufficient or excess Ca intake.

## Calcium Excess

Rearranging the point of view on research papers and clinical cases dealing with diet-induced developmental skeletal diseases may help to understand the complex situation and sometimes seemingly conflicting information. In the literature, plenty of papers describe the detrimental effect of excess Ca on the skeletal development in growing dogs.[8,9,10-14, etc.] What the underlying trials of most of those papers have in common is an excessive Ca supply to the trial animals (puppies) without a concomitant increase of P in the diet, resulting in a wide Ca:P ratio > 2:1 that seems to trigger signs of developmental orthopedic diseases in growing dogs of certain breeds, mostly Great Danes. It is a well-known fact that the amount of Ca in the diet has an impact on the P digestibility.[15] Also, Mack, et al.[6] stated that the fecal P excretion was strictly correlated to fecal Ca excretion in dogs and cats. In other trials with more balanced Ca:P ratios through elevated P supply, no or definitely less-severe clinical signs of DOD were caused.[13,14,16,17]

Therefore, not the Ca excess itself seems to be the only and major problem for the skeleton, but the consequences of a wide Ca:P ratio on the bioavailability of P and, accordingly, a possible

clinical consequence of a secondary P deficiency triggered by a Ca excess.[18] This explanation is not really new. It was first published in 1931 by Marek and Wellmann[19] who were summarizing their findings about the detrimental effects of excess Ca, concluding that the signs, such as lameness or deviation of limb axes, were more detrimental when the P content in the diet was not increased concurrently. It has been reported that two German Shepherd Dog puppies developed severe clinical signs of skeletal problems, confirmed by radiological and histological findings, as well as hypophosphataemia after being fed excess Ca and a normal amount of P. Severe clinical signs of DOD were observed in a Fox Terrier puppy after adding $CaCO_3$ to its diet, and these signs disappeared after $CaCO_3$ was exchanged with bone meal, which led to an increase in the P supply and a balanced Ca:P ratio.[19]

Taking that information into account, it is possible to conclude that mainly the combination of a Ca excess and a marginal P supply, i.e., a secondary P deficiency, may cause DOD, especially in puppies of large and giant breeds. However, a certain sensitivity of some breeds against a Ca excess supposedly exists alongside a co-factor to breed size. Such a breed difference may help to explain the practical experience that some dogs develop the multifactorial-caused DOD when others stay (clinically) healthy under the same conditions. This also is shown by a study on the effects of a Ca excess combined with an increased P supply on skeletal development in two different dog breeds during growth: The measurements of bone lengths and widths in radiographs of the forearm of Beagles and Foxhound-crossbred dogs at 6 weeks of age, and again after a period of overexposure to Ca at about 27 weeks of age, revealed a growth-reducing influence only in Beagles, without influence on clinical parameters of skeletal health.[16]

The hypothesis that DOD in growing dogs is related to the co-factor "P supply" was reinforced by the results of a study of P deficiency in growing Beagles and Foxhound crossbreds.[20] The puppies received a diet providing approximately 40 to 50% of the recommended P allowance[2] (approximately 3.5% DM) while the Ca supply met the requirements, resulting in a Ca:P ratio above the recommended ratio. In this trial, some puppies of both breeds developed severe clinical signs of DOD showing extremely bowed legs. These signs were reversible by phosphorus repletion.

That excess Ca is much or may be only more hazardous to growing dogs when it is accompanied by a low or a marginal P supply also makes sense in respect to the natural diet of the ancestors of the modern dog: In a pack of hunting dogs or wolves, those low in the hierarchy, such as puppies and young dogs, are feeding on the remains of larger prey, i.e., mainly connective tissue and bones. A certain sensitivity against Ca excess would only make sense here if the digestibility of minerals from bones would be quite low or the dogs would be able to excrete excessive amounts via urine without greater harm (possibly after a period of extra storage of Ca in the skeleton).

Forming a hypothesis, it is more likely that dogs, including growing dogs over wide ranges, are quite unsusceptible against Ca excess because their natural diet contains high quantities of Ca and P. However, a certain degree of Ca excess, maybe after a certain period of time, may have a negative effect on its own, especially on the healthy skeletal development influenced by breed, growth curve, micro-trauma, training intensity, the source and bioavailability of Ca and P, supply of other nutrients, and possible other factors. On the other hand, dogs seem to have only a limited ability to increase the digestibility of Ca and P and, therefore, may be prone to exhibit clinical signs of deficiency. The latter part of the hypothesis also would explain why dogs need so much more Ca compared to humans. As in many other areas, the warning to draw conclusions based on knowledge from human physiology is justified. Dogs are no barking humans!

## Energy and Growth Development

Another main factor to consider in the healthy upbringing of dogs is energy supply. It is common knowledge that next to age and body weight, the energy requirements in growing dogs are influenced by a number of factors, such as breed, activity, health status, etc. This leads to the main conclusion that the correct energy supply for the individual puppy can only be determined through monitoring the individual weight development. If the body weight lies within a range of the recommended growth curve, the risk of overfeeding and excessive body weight is minimal. In this context, it is necessary to emphasize that substantial limitations exist in puppies with regard to a common body condition scoring (BCS) system. Not only that the body fat content measured by DEXA is not necessarily in accordance with the predicted content using the BCS system,[21] a low BCS may easily be found in a puppy that is too heavy for its age. This is due to the fact that especially puppies of breeds with a high growth potential use excess energy for growth and not fat accretion. Here, all detrimental effects of a too high body weight on a growing skeleton may act while the puppy itself has a skinny appearance. Because the size of an animal is often mistaken for beauty, good health and strength, especially in large and giant breeds, growing dogs of those breeds often are too heavy.[22] Here, restrictive feeding is required to let the dog grow more slowly but healthfully.

The recommendations for energy supply during growth given in the NRC[2] overestimate the *de facto* needs,[23] which may lead to wrong feeding recommendations. Dog owners following feeding guides based on existing predictions for energy needs, therefore, may overfeed their puppies. Additionally, most of the owners add a lot of treats and snacks to their puppies' daily rations for training or bonding reasons. But not only an excessive energy supply and a resulting forced growth development with detrimental effects may result. When the average commercial diet is designed to meet all nutrient requirements, presuming an

energy consumption that in reality is overestimated, the nutrient supply will be insufficient when the puppy eats less. If, for example, the puppy requires only 70% of the presumed average daily energy to grow according to the recommendation, it will consume 70% or less (also subject to the amount of snacks and treats) of the presumed amount of diet and therefore only 70% of the nutrients. Even when safety margins are incorporated, the nutrient supply is probably marginal or insufficient. This also is the reason it is not recommendable to use diets meant for maintenance/adult dogs during growth. In most of those products, the nutrient density is not sufficient for puppies let alone for those individuals with below-average energy requirements. The consequences of feeding such inappropriate diets to growing dogs, such as signs of DOD, is something we regularly see in our nutrition consultation practice.[24]

## Other Nutrients with Impact on Skeletal Development

Other nutrients, such as vitamins A and D and trace elements, including zinc, copper, etc., have a possible negative impact in case of deficiency or excess, respectively, and therefore have to be considered, especially in diets for growing dogs. Other nutrients, such as protein, are overrated in regard to their impact. Often a warning is expressed that a protein excess impairs the healthy growth of puppies, and there are products on the market advertised by indicating the restricted protein concentration for a healthy growth, although it was demonstrated that this effect does not exist.[24] This is partly true for the presumed detrimental effect of a vitamin A excess on skeletal development. We learned recently[25] that the safe upper limit in puppies is 26fold higher than expected.[2]

As a consequence of these factors, the recommendation for practical feeding remains that one should try to meet the requirements at least for energy, Ca and P in a puppy as accurately as possible. This leads to the question if it is possible to create a diet that is perfect for all breeds, ages, activities, life stages, etc., but also to the insight that special requirements need to be addressed with matching products. A careful choosing of a suitable product is crucial especially during the very sensitive life stage of growth.

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