

Factors that modify the effect of excess calcium on skeletal development in puppies

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Abstract

Developmental skeletal problems are often connected to mistakes in feeding, i.e. a deficiency or excess supply of nutrients, especially minerals. The energy consumption, the resulting growth intensity and the breed of animals are also known as factors that influence developmental orthopaedic diseases. In most clinical trials, excess Ca was connected to alterations in normal, healthy skeletal development, which led to severe clinical symptoms in some dogs, subclinical changes in the radiographic or histological examinations in other animals, or even no observable symptoms. These results correspond to practical experience in the field of clinical nutrition consultation, since excess Ca has been shown to have various impacts in growing dogs. The present review of results published in the literature and from my own trials led to the hypothesis that P is an important co-factor that can alleviate or aggravate the development of skeletal problems in growing dogs.

Key words: Dogs: Growth: Calcium: Phosphorus

Excess Ca is considered detrimental to the growing skeletons of dogs⁽¹⁾. Practical experience in nutrition consultation shows that not all dogs under Ca excess develop skeletal problems. The same is true for own research work where the effects of feeding diets with excess Ca to growing dogs were not identical in all individuals⁽²⁾. The present review was undertaken to identify co-factors that alleviate or aggravate the effect of excess Ca on developmental orthopaedic diseases (DOD). Therefore, a literature review was performed to investigate the effects of excess Ca in growing dogs^(1–14). The parameters of special interest were the Ca and P intakes, Ca:P ratio, clinical and subclinical symptoms of DOD, breed, age and plasma P concentration, if available.

Literature review and discussion

In 1931, Marek & Wellmann published their historical data on the effects of Ca and P supply in growing dogs. They summarised their findings about the detrimental effects of excess Ca, concluding that the symptoms 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 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 (calculated: 2.2% Ca; 0.9% P⁽¹⁰⁾). Severe clinical signs of DOD were observed in a Fox Terrier puppy after adding CaCO₃ to its diet, and these signs disappeared after CaCO₃ was exchanged with bone meal, which led to an increase in the P supply and a balanced Ca:P ratio. The underlying reason for these clinical signs might be the decreased availability of P in cases with a wide Ca:P ratio caused by a high Ca content of the diet, as described previously⁽¹⁵⁾. This effect of selective Ca excess on the apparent digestibility of P was also observed in Beagles during the own experiments⁽¹⁶⁾. The results of Nap *et al.*⁽¹¹⁾ can help explain the sometimes inconsistent findings published more recently by various laboratories (Table 1).

Hazewinkel and his colleagues^(4–7,12) also observed detrimental effects in Great Danes (GD) after feeding the animals excess Ca in a diet with a P supply that fulfilled the minimum requirements (0.9% DM; wide Ca:P ratio)⁽¹⁷⁾. In some studies of Fox Terrier, German Shepherd, and GD puppies fed an excess amount of Ca, clinically severe symptoms were observed^(5,7,10,12). In other studies, mild symptoms have been reported in puppies fed excess Ca, and only subclinical effects were measured in radiographs or through histology⁽³⁾, or no symptoms have been observed or reported^(6,14). In another trial using Miniature Poodles (MP), the combination of excess Ca and a marginal P supply did not cause signs of

Abbreviations: DOD, developmental orthopaedic diseases; GD, Great Danes; MP, Miniature Poodles.

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Table 1. Effects of excess calcium on growing dogs

Author and year	Breed	Ca (% DM)	P (% DM)	Age (week)	Number of dogs	Effect (X-rays or histology)	Clinical symptoms	Feed intake
Hazewinkel <i>et al.</i> (1991) ⁽⁷⁾	Great Dane	3.3	0.9	5(10) – (11)26	6	n.m.	Yes*	Reduced (week 14)
	Great Dane	3.3	3	5(10) – (11)26	6	n.m.	n.m.	Normal
Hazewinkel <i>et al.</i> (1985) ⁽⁵⁾	Great Dane	3.3	0.9	5(10)–25	6	Yes	Yes	Reduced
Hazewinkel <i>et al.</i> (1987) ⁽⁶⁾	Great Dane	3.3	0.9	5(10)–25	6	Yes	n.m.	Reduced
Voorhout & Hazewinkel (1987) ⁽¹⁴⁾	Great Dane	3.3	0.9	7–21	6	Yes	No	n.m.
Goedegebuure & Hazewinkel (1986) ⁽³⁾	Great Dane	3.3	0.9	6–26	6	Yes	Yes	Bulging of osteochondral junction ribs
Schoenmakers <i>et al.</i> (2000) ⁽¹²⁾	Great Dane	3.1	0.9	6–27	9	Yes	Yes	n.m.
	Great Dane	3.1	2.8	6–27	6	(Yes) not severe	(Yes)	Not severe
Lauten <i>et al.</i> (2002) ⁽⁸⁾	Great Dane	2.7	2.2	5–24	32	No	No	Normal
Goodman <i>et al.</i> (1998) ⁽⁴⁾	Great Dane	2.7	2.2	5–24	13	yes	(Yes)	Increased incidence (lameness, 1 × mandibular osseus proliferation)
Lepine (1998) ⁽⁹⁾	Great Dane	2.7	2.2	6–72	15	Yes	Yes	Six out of fifteen pups transient lameness. Three out of fifteen HOD
Nap <i>et al.</i> (1993) ⁽¹¹⁾	Miniature Poodle	3.3	0.9	6–18	21	n.e.	No	Reduced (week 11)
Stephens <i>et al.</i> (1985) ⁽¹³⁾	Beagle	2.8–3.9†	1.1	7–17	9	Yes	No	n.m.
Marek & Wellmann (1931) ⁽¹⁰⁾	Small Mongrel	2.2	0.9	6–18	6	Yes	Yes‡	Normal
Dobenecker <i>et al.</i> (2006) ⁽²⁾	Foxhound crossbred	3.6	1.25	6–27	35	No	No	Normal
	Beagle	3.6	1.25	6–27	25	Yes	No	Normal
Dobenecker & Kienzle (2008) ^{(19)§}	Foxhound crossbred	1.2	0.35	6–12(14)	19	n.e.	Yes	(Four out of nineteen bowed legs)
	Beagle	1.2	0.35	6–12(14)	15	n.e.	Yes	(Three out of fifteen bowed legs)

n.e., not examined; n.m., not mentioned.

* In one sentence, severe skeletal abnormalities were mentioned.

† Constant supplementation of 2.3 g Ca/d per dog.

‡ Clinical symptoms disappeared when one of the dogs received P in addition to excess Ca.

§ Supply with low P and normal Ca for comparison.

Modifying factors on excess calcium

DOD⁽¹¹⁾. Therefore, excess Ca alone without a concomitant increase in the P supply (i.e. a wide Ca:P ratio >2/1) seems to trigger signs of DOD in growing dogs of certain breeds, especially GD, and increase the number of severely affected puppies. In dogs with severe clinical symptoms, some authors have reported hypophosphataemia^(7,10), which supported the hypothesis that P deficiency is a major co-factor in the development of skeletal problems in puppies fed excess Ca. Accordingly, trials were performed where P levels were elevated approximately 25% or more above the minimum requirements or were provided at 0.9% of the DM to ensure a more balanced Ca:P ratio. These trials led to less severe skeletal changes or, more frequently, to no clinical symptoms at all (GD^(4,7,9,12); Beagles^(2,13)). When Foxhound crossbreeds were fed a P supply above the previously published recommended allowance⁽¹⁸⁾, the animals developed no clinical or subclinical symptoms⁽²⁾. Lauten *et al.* did not state their clinical findings clearly enough to evaluate their results in this context⁽⁸⁾.

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 crossbreeds⁽¹⁹⁾. The puppies received a diet providing approximately 40–50% of the recommended P allowance (approximately 0.35% DM⁽¹⁸⁾) 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 symptoms of DOD.

Along with evaluating the impact of the nutrient supply, especially the supply of Ca and P, it is important to consider other factors in the study of complex nutritionally triggered DOD. The adult body weight alone might influence the effects of excess Ca through body-weight development or the growth rate, which involves the corresponding hormonal status of the animal. Another potential factor is the susceptibility or sensitivity of the breed to certain nutritional factors. From the literature and from clinical experience, we know that large or giant dog breeds are more likely to develop skeletal problems during growth when they ingest insufficient or excess nutrients. Overall, dogs with an adult body weight of about 30 kg or more are more vulnerable to excess Ca. However, too intensive growth alone can lead to a greater frequency of developmental skeletal disease even when all nutrients are supplied according to the requirements⁽²⁰⁾. The breeding of some dog varieties seems to be problematic even when the required energy and nutrient supplies are met. For example, in radiographic examinations⁽²¹⁾, there were diagnosed skeletal changes in GD but not in MP after feeding the animals a balanced diet without an excess of Ca. The authors have reported that the majority of the GD showed abnormally flattened distal ulnar metaphyses with increasing incidence of cartilage cones, and the growth plates were more irregular in the GD than in the MP puppies.

The evidence collected in the present review supports the hypothesis that excess Ca is much more hazardous to growing dogs when it is accompanied by a low or a marginal P supply. Moreover, large and giant dogs are particularly prone to nutritionally triggered developmental skeletal diseases.

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