

## **Nutritional Influences on Hip Dysplasia**

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### **INTRODUCTION**

Hip dysplasia (HD) is a series of developmental conditions of polygenetic nature, allowing for coxofemoral subluxation. Secondary changes may develop including shallow acetabulum and osteoarthritis (OA). From follow-up studies it is known that not all dogs with joint laxity develop subluxation or these secondary signs. There are young dogs with joint laxity which will never develop OA of the hip joints and have still joint laxity at mature age, whereas young dogs which develop OA in their hip joints will develop joint capsule thickening and as a consequence stable hips. From a variety of studies it was demonstrated that a combination of clinical and radiological finding, or a combination of different radiological techniques increase the predictability of screening methods at a young age to develop OA of the hip joints at maturity. Nevertheless is the predictability not close to 100%, and therefore there might be a variety of factors of importance which make hip joints with laxity to hip joints with OA. Since the hereditary coefficient for HD varies between 0.2 and 0.5, important influence exist of environment, including nutrition and activity, on the occurrence of subluxation and OA development.

There are a variety of cellular activities and cell differentiation processes involved in skeletal growth and modeling in the growing puppy. Some of them can be influenced by nutrition.

### **NUTRITION**

Different nutrients have influence on different aspects of hip joint development and on the development of OA. In addition, the diet may influence the severity the dog will suffer from HD. These different aspects are overviewed:

1. During bone growth, the bones keep their typical form due to new bone formation by osteoblast working in concert with removing of "old bone" by osteoclasts to adapt the contours to the new situation. During growth the femoral collum will differ in position to the femoral shaft. Subluxation due to abnormal development of the collodiaphyseal angle during growth can be due to lowered activity of osteoclasts in the case of high food and thus high Ca intake, as was demonstrated by Hedhammar *et al* (1974) in G.Danes. The surgical technique developed to normalize this situation (Prieur *et al*) might be prevented by a normal osteoclast activity. Osteoclasts are stimulated by parathyroid hormone, which is increased by low calcium intake and decreased in case of high calcium intake. In addition, calcitonin which is increased in case of high calcium intake will decrease osteoclast activity. For that reason, high calcium intake should be prevented.
2. Since joint cartilage is much more plastic than bone, delayed endochondral ossification at a given age and weight may facilitate joint deformities when compared with dogs with a more advanced stage of skeletal development. We demonstrated that G. Danes raised on food

with high Ca content had delayed endochondral ossification (i.e., retarded skeletal maturation), and hyper-calcitoninism with decreased bone remodeling as a result. For that reason, high calcium intake should be prevented in order not to disturb the process of maturation of the young skeleton.

3. Excessive energy intake causes excessive body weight gain with negative influences on hip joint development in those dogs which are prone to HD. **Ad lib fed Labradors developed significantly more severe HD, assessed by Norberg angle measurement than restricted fed matched group.** Subluxation of the femoral head together with overweight may cause the acetabular rim to become deformed as is seen in young dogs with severe HD, together with increased pressure on the limited loaded joint cartilage. Significantly overweight in Labradors coincided with severe OA in hips and elbows and significant differences in plasma levels of GH and IGF-I compared to the restricted fed and slim genetically related Labradors. This indicates that adiposity might cause differences in regulatory hormones with influence on cartilage cell function even without the influence of overloading.
4. **Excessive intake of carbohydrate, protein or fat per se has no proven influence on skeletal development as long as it does not go together with adipositas and is part of a balanced diet covering the required nutrients.**
5. An excess of Vit A might increase osteoclastic and decreased osteoblastic activity; since vitamin A is not a limiting factor in dog food for dogs, this should not be of any concern for dog owners. Hypervitaminosis C can cause hypercalcaemia, probably by changing the pH of the microenvironment in the bone, and thus induce hypercalcitoninism and thus may hamper modeling. Moderate elevated levels of dietary Vit D (<4000 IU, normal is 500 IU per kg food) will not have severe consequences for skeletal development.
6. A relative increase in unsaturated omega 3 fatty acids will decrease the synthesis of inflammatory mediators in dogs, and might decrease inflammation of joints as is described of skin. This has been demonstrated to occur in dogs with OA of the elbow joints, however without improvement of the locomotion analyzed with ground reaction forces.
7. Chondroitin sulphate in combination with glucatamin-HCl used as supplement in, or separate from, food or given as injection, has been investigated in different groups of dogs with natural occurring or induced OA. Several investigators claim that there is indication for efficacy in dogs with OA; the effect can be noticed after several weeks, but may continue after cessation of the therapy. In addition, it is claimed that the dosage of NSAIDs can be reduced.
8. Reduction in body weight in obese dogs causes dramatic improvement in case of OA especially when combined with strict cage rest for a period of 3 months allowing for regeneration of cartilage lesions.

## CONCLUSION

In conclusion, nutrition plays an important role in the (the prevention of the) development of HD, and as part of the conservative treatment of HD.

The best guidelines for owners of a young dog of a breed at risk for HD is a food with balanced mineral contents at a lower level to increase osteoclast activity and thus increase skeletal remodeling, but not too low to induce alimentary hyperparathyroidism with pathological fractures. It is important the food is of high quality, including high quality of proteins. A balanced diet with high protein content has NOT proved to have negative effects on skeletal development in carnivores. Excessive energy intake, even as part of a balanced diet, increases both the incidence of OA, its severity, and the degree of lameness it will cause. Weight reduction is both a part of the prevention as of the OA-programme.

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#### **SPEAKER INFORMATION**