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NUTRITION AND DEVELOPMENTAL ORTHOPEDIC DISEASES IN LARGE BREED DOGS

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INTRODUCTION

Large breed dogs are of particular interest relative to orthopedic concerns since they are at the upper end of the body weight spectrum. Breeding of the large and giant breed dogs continues to emphasize increased mature body size as this is often considered advantageous in the show ring and can be an important owner preference. While an increased genetic potential for growth rate is not in itself detrimental to the large breed dog, management practices that allow growth rate to be maximized can result in negative consequences. It is well documented that the incidence of skeletal disease, including osteochondroses, hypertrophic osteodystrophy and hip dysplasia, is markedly increased in the growing large breed dog if management practices are such that this maximal genetic potential for rate of growth is realized.

The predominant management consideration impacting growth rate, and ultimately skeletal disease, is nutritional support. Although many nutritional considerations have been implicated in skeletal disease in the growing large breed dog, three predominant factors, the dietary concentrations of protein, energy and calcium (and vitamin D) are most often indicted.

The association between a high-protein diet and skeletal abnormalities was studied in Great Dane puppies from weaning to 14 weeks of age.1,2 No evidence of dietary protein (31.6%, 23.1%, or 14.6%) on calcium metabolism or skeletal development was found. In another study, growing Great Dane puppies fed free choice had a dramatic increase in both growth rate and incidence of hip dysplasia compared to puppies fed only 66% of the intake of the dogs fed free choice.3 The free-choice intake group experienced subchondral spongiosa, osteopenia, and biomechanically weak subchondral bone which could not adequately support the articular cartilage of the hip. It has further been observed that a high calcium diet (3.3% Ca/0.9% P) fed to Great Dane puppies (weaning through 6 months of age) resulted in poorer mineral mass, delayed bone remodeling, increased radiographic irregularities, and more osteochondritic lesions. Finally, a more recent study in the growing Great Dane documented that either a high (2.70% Ca/2.20% P) or low (0.48% Ca/0.40% P) calcium diet resulted in poorer conformation, increased clinical lameness and gait asymmetry compared to a medium calcium diet (0.80% Ca/0.67% P).4-6

Another potential contributor to skeletal health is dietary vitamin D. Either a deficiency or excess of dietary vitamin D can promote negative effects on skeletal development in the large breed dog. A recent report has demonstrated that excessive but non-toxic dietary concentrations of vitamin D3 can result in decreased skeletal remodeling and focal enlargement of growth plates without clinical abnormalities, or produce radius curvus syndrome with higher excesses of vitamin D3.7 These skeletal abnormalities were the result of a direct effect of vitamin D3 metabolites rather than an increase in Ca absorption.8,9

It is also important to recognize that nutritional support is also a critical component impacting the development of skeletal problems in adulthood. For example, excessive body weight is a key contributor to the development of orthopedic problems through the application of stresses on the joint surfaces. It is important to manage body weight and develop nutritional strategies for weight reduction in those cases where body weight is greater than ideal.

Common treatment programs for weight reduction include the use of high-fiber diets10-12 which are thought to promote satiety. The control of satiety is complex and other components of the diet, such as protein and carbohydrate intake, may affect satiety by influencing brain serotonin precursors.11 Problems associated with high-fiber diets include constipation, excessive frequency and volume of defecation, poor skin and coat quality, decreased nutrient digestibility,12 decreased mineral absorption, and reduced palatability.12

Alternative approaches to high-fiber diets for obesity include nutrients designed to address the underlying physiology of the animal. Obesity is generally associated with abnormal glucose metabolism, altered lipoprotein profile, and altered hormonal status. Starch sources such as barley and sorghum,13 minerals such as chromium tripicolinate,14 and vitamin/vitamin-like substances such as vitamin A and L-carnitine15-17 have been shown to be important nutrients in helping achieve a normal physiological state for obese individuals.

The importance of weight reduction on lameness associated with hip osteoarthritis was recently reported.18 Dogs which were at least 10% over ideal body weight and expressing lameness due to osteoarthritis of the hip joint were provided with 60% of maintenance calories to promote weight loss. A loss of 11 to 18% of initial body weight resulted in a significant improvement in body condition score and lameness over time. These results demonstrate that moderate weight loss can positively impact the signs of hip arthritis in medium to large breed dogs.

The adult large breed dog is also subjected to skeletal disease resulting from normal age-related physiological changes to articular cartilage and the consequence of traumatic injury. Just as changes occur in the articular cartilage during the process of maturation, normal age-related changes continue to occur throughout life. Data from porcine articular cartilage has shown a decrease in hydration, a decrease in collagen on a dry matter basis, a decrease in chondroitin sulfate, and a decrease in proteoglycan size with age.19,20 Although the total glycosaminoglycan concentration may not vary much with increasing age, the ratio of keratan sulfate to chondroitin sulfate increases.20 With respect to chondroitin sulfate it was also noted that the 4-sulfated compound decreased while the 6-sulfated compound increased. The alteration of proteoglycan composition and size is most likely the result of proteolytic cleavages that are not limited to the period of growth and maturation but appear to occur throughout the aging process in all species.21 The link proteins are also subject to proteolytic cleavage as aging progresses.22 This is likely a normal component of aging and could contribute to the destabilization of the proteoglycan component of the extracellular matrix. The inherent result of these normal age-related changes of the articular cartilage...
will be a matrix of reduced capability to withstand the forces associated with normal joint functioning.

Articular cartilage injuries are characterized by the degree of involvement of the extracellular matrix composition and resultant damage to the chondrocytes. The following are general types of injury are described involving the articular cartilage: microdamage or blunt trauma, chondral fractures, and osteochondral fractures. Microdamage may be caused by a single impact or repetitive blunt trauma. It is characterized by a loss of matrix components, most notably proteoglycans, without chondrocyte damage. If the traumatic event is short in duration, the chondrocytes may be able to repair the cartilage by restoring the lost proteoglycans and matrix components. Damage resulting from sustained blunt trauma, however, may eventually become irreversible.

Chondral fractures result from a penetrating traumatic event disrupting the articular surface but sparing the subchondral plate. The pathophysiological response of articular cartilage surrounding the injury results in chondrocyte proliferation and synthesis of extracellular matrix protein. Unfortunately, since chondrocytes cannot migrate to the lesion, these efforts do not result in complete repair. The third type of injury is described as a full thickness defect or osteochondral fracture. These injuries are characterized by an insult crossing the tidemark into the underlying bone resulting in chondrocyte damage and marrow cell involvement. Full thickness injuries invoke an inflammatory process since vascular structures are now involved. This is in contrast to the lack of inflammatory response to less traumatic articular cartilage injuries resulting from the inherent avascular nature of this tissue. Following a full thickness injury, fibroblasts differentiate into chondrocytes and repair of the tissue is attempted, but the fibrocartilaginous repair tissue produced is not “normal” articular cartilage.

After several phases of remodeling, the repair tissue has a lower proteoglycan content and a substantial component of type I collagen rather than type II. Therefore, the resulting repair is often of suboptimal quality resulting in compromised joint function.

Oral chondroprotective agents (nutraceuticals and slow-acting, disease-modifying agents) may modulate joint structure and physiology and thereby mitigate the problems associated with cartilage aging and trauma depending on the severity of the insult. Two such agents which are included in the majority of nutritional supplements are glucosamine and chondroitin sulfate. Glucosamine is a ubiquitous amino sugar used in the synthesis of the disaccharide units of glycosaminoglycan (GAG). In humans, an oral dose of glucosamine sulfate is 90% absorbed and 26% bioavailable. Another study found the bioavailability, pharmacokinetics and excretion pattern in the human to be consistent with those of the dog. Chondrocyte studies in vitro found that glucosamine has a stimulatory effect causing increased production of normal collagen and proteoglycans. Chondroitin sulfate is a GAG which can be sulfated on the fourth or sixth carbon. Chondroitin-4-sulfate is the predominant GAG in growing mammalian hyaline cartilage but, with age, production of chondroitin-4-sulfate decreases and more production of other types of GAGs is increased by chondrocytes. Two chondroprotective activities produced by chondroitin sulfate but not by glucosamine are prevention of thrombi formation in microvasculature and inhibition of metalloproteases via the modulation of interleukin-3.

Considerable evidence exists documenting the safety of oral administration of glucosamine and chondroitin sulfate. In considering oral supplementation of glucosamine and chondroitin sulfate for management of joint problems one must be cognizant of the route of supplementation. If capsules or pills are utilized owner compliance may be an issue of concern. Inclusion of these compounds in a total diet will eliminate any concerns for compliance. Several other chondroprotective compounds are presently marketed with varying degrees of supportive data regarding their efficacy.

CONCLUSION

In conclusion, skeletal disease must be managed nutritionally in the large breed dog both during growth and during adulthood. Failure to provide proper nutritional management and support during both of these life stages can result in less than optimal skeletal health of the large breed dog.

REFERENCES


