Relationship Between Nutrition and Bone Growth in Large and Giant Dogs

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ABSTRACT The pathogenesis of the osteochondrosis syndrome was studied in large and giant breeds of dogs. Spontaneous cases of osteochondrosis were examined in large breed dogs, and experimental disease was investigated in Great Dane puppies fed ad libitum or restricted diets until 6 mo of age. This investigation concluded that the primary lesion occurring in osteochondrosis of dogs from the large and giant breeds is an acquired pattern of osteopenic and biomechanically weak subchondral spongiosa that cannot provide adequate bony support for the articular cartilage of joints. Excessive biomechanical loading of the inadequately supported epiphyseal articular cartilage leads to secondary disturbances in the nutrition, metabolism, function and viability of the chondrocytes in the developing joint surface. The primary lesion in the subchondral spongiosa develops when overnutrition (ad libitum feeding) overstimulates skeletal growth and cancellous bone remodeling mechanisms in those breeds of dogs who already have an inherent capacity for rapid skeletal growth. The epiphyseal spongiosa of rapidly growing dogs of the larger breeds is inherently less dense and less strong per unit area than epiphyseal spongiosa of dogs from the smaller breeds. Overnutrition in dogs from the larger breeds exaggerates this tendency to create osteopenia by increasing the rates of skeletal growth and remodeling of the newly formed cancellous bone. Accelerated rates of bone growth and bone remodeling act in concert to produce a pattern of epiphyseal spongiosa that is composed of fine trabecular systems that are spaced relatively far apart and that are ultimately less strong per unit area than in small breeds of dogs. Overnutrition also predisposes to development of lesions of osteochondrosis by promoting increases in muscle mass and body weight that further overload inadequately supported joint surfaces. J. Nutr. 121: S114–S121, 1991.

INDEXING KEY WORDS: symposium, dogs, osteochondrosis syndrome, bone growth

The incidence of growth disturbances in the skeletons of dogs of the large and giant breeds is continuously increasing. There is disagreement among investigators regarding the nomenclature and causes of these disturbances. There is concern whether all or only some of these growth disturbances are osteochondroses, and there is lack of agreement as to the roles that nutrition, genetics and biomechanics play in their pathogenesis. Despite these problems, many of the lesions have structural similarities, implying that they may share common basic pathogenic pathways.

Primary lesions of these growth disturbances are localized to one or more of the growth zones of the cartilage tissue located in the growth plates of endochondral bones and/or in the deep layers of the articular cartilages of the epiphyses of synovial joints. Disturbances of growth plates in long bones are manifested as angular limb deformities, and in the vertebral column, especially of the cervical spine, as spondylolisthesis. Disturbances resulting in incongruity of joint surfaces may predispose to intra-articular fracture or, as occurs in the elbow joint, to the production of an ununited anconeal process. Complete or partial epiphysiolysis may occur in the growth plates of the distal ulna and/or radius or in the tibial tuberosity (Osgood-Schlatter-syndrome). Otherwise deformed or misshapened joint surfaces are caused during skeletal growth by disturbances within growth zones of the deep layers of the articular cartilage. One of these latter lesions, chondrosis dissecans, is a characteristic lesion that develops on convex joint surfaces, e.g., caudal aspect of the articular surface of the proximal humerus or on the femoral condyles.

Currently, the osteochondrosis syndrome is considered by some to be a biomechanical disease. Lesion development requires two essential elements:...
rapid skeletal growth and an overloading of the growing skeleton due to increasing muscle mass and body weight. Some observations support this theory. Rapidly growing males are more frequently affected than female dogs. The incidence of osteochondrosis increases with preferential selection for increased body size (1–3).

Rapid skeletal growth can be produced in dogs by overnutrition [ad libitum feeding]. The combination of rapid skeletal growth and prominent muscle development work together to concentrate biomechanical forces on forming joint surfaces while they are still structurally weak. Thus, this combination increases the incidence of developmental bone disease in large rapidly growing dogs as well as increases the severity of the lesions when they are present. The initial studies demonstrating the role of nutrition in initiating biomechanical disease of the skeleton were done while investigating the causes of hip dysplasia. In studying canine hip dysplasia, workers found that immature, heavy dogs fed high energy diets had a higher frequency and increased severity of lesions than did young dogs of normal weight fed nonsupplemented diets (4, 5).

It seems reasonable to propose that the pathogenesis of osteochondrosis occurring spontaneously in dogs of the large and giant breeds is also due to the effects of nutrition in initiating biomechanical disease. Evidence supporting this hypothesis is found in reports by two investigators that indicate that osteochondrosis affects young, heavy puppies whose bone growth and muscle development have been accelerated by overnutrition (1, 2, 6).

There is some debate as to whether development of osteochondrosis is predisposed by high energy intake alone, or by the combined effects of a diet high in energy and calcium. Studies of osteochondrosis in other species, i.e., swine and cattle, using high energy diets otherwise controlled for levels of protein, vitamins, calcium and phosphorous have shown that the frequency and severity of the lesions of osteochondrosis were directly related to the energy intake (2). However, there are two experimental studies of osteochondrosis in Great Dane puppies in which lesions of osteochondrosis were thought to be caused by diets that were either high in both energy and calcium (7) or in calcium alone (8). In these experiments the workers reported that the high calcium diet caused an initial hypercalcemia leading to hypercalcitoninism and hypoparathyroidism. They proposed that hypercalcitoninism played a role in the development of the lesions of osteochondrosis by the interference of calcitonin on the normal maturation process of cartilage matrix. While Hedhammer et al (7) believed that the dogs fed the diet high in both calcium and energy eventually developed hyperparathyroidism, Hazewinkel (8) found that there was a direct correlation between the amount of calcium consumed in the diet and the degree that endochondral ossification was disturbed. He also reported that bone remodeling was retarded at sites of the articular lesions of osteochondrosis in puppies.

Despite the different points of view regarding the roles that overnutrition (ad libitum feeding), excessive levels of dietary calcium resulting in hypercalcitoninism and biomechanical overloading of joint surfaces have in the pathogenesis of spontaneous osteochondrosis in the dog, all investigators of osteochondrosis, including myself, agree that the initial lesions are located in the deeper cartilage tissues of the developing joint surfaces.

Pathogenesis of the osteochondrosis syndrome. In our laboratory we have studied the pathogenesis of the osteochondrosis syndrome by gross and histologic examination of bone specimens collected from spontaneous cases of osteochondrosis in immature dogs of large breeds presented to the clinic for routine autopsy and by experimentally producing osteochondrosis in Great Dane puppies. Experimental disease was produced by feeding an experimental diet (9) ad libitum or in restricted amounts [70–80% of ad libitum] to Great Dane puppies until 6 mo of age.

The cartilage lesions in both the spontaneous and experimentally induced lesions of osteochondrosis in our studies were found in both the articular cartilages of the epiphyses and the growth plates of long bones as has been previously reported (1, 2, 6). Lesions in these cartilaginous growth sites included delayed chondrocyte proliferation and maturation, irregular alignment of chondrocyte columns, degenerative and necrotic chondrocytes, foci of chondrocyte proliferation, formation of hyaline stripes between chondrocyte columns, regressive changes in cartilage matrix including loss of fibrillar structure and edema and disturbances in endochondral ossification (Fig. 1).

The pathogenesis of the cartilage lesions are due to a disturbance in the metabolism of chondrocytes, which are actively proliferating and synthesizing cartilage matrix for subsequent replacement by bone tissue in the process of endochondral ossification. Since the cartilage tissue is without an intrinsic blood supply, the metabolic needs of the chondrocytes must be met by diffusion of nutrients and metabolic wastes through the water-rich ground substance of the cartilage matrix. Movement of these substances in cartilage tissue is facilitated by the massaging effect of repetitive forces of compression and decompression that occur during passive joint movement and locomotion. Normal nutrition of articular cartilage depends upon the maintenance of a normal pattern of diffusion in cartilage tissue covering the entire joint surface, and this is dependent in large part upon the biomechanical properties of the subchondral spongiosa.

Because the cartilage tissue of the developing joint surface is relatively soft and easily deformed, the subchondral spongiosa must provide firm support for the
overlying joint cartilage. To protect the articular cartilage from trauma the biomechanical forces of locomotion must be transferred across the entire joint surface and through the articular cartilage to the underlying subchondral bone in a uniform manner. The formation of a normal articular surface in a joint requires that the mechanisms of skeletal growth, endochondral ossification and bone remodeling must work in harmony at all stages of joint development to ensure the construction of subchondral spongiosa that meets the biomechanical requirements of the particular joint surface during all stages of joint development. The complex, closely integrated processes of skeletal growth, endochondral ossification and bone remodeling are vulnerable to a variety of potential insults, including those having a nutritional, metabolic, genetic, vascular and biomechanical basis. Because these processes take place at an accelerated rate in the subchondral spongiosa, insults first manifest their presence as disturbances in the growing cartilage.

**Predisposition.** Growing dogs of large and giant breeds are predisposed to develop multiple lesions in cartilage tissue at active sites of endochondral ossification in the skeleton, i.e., lesions of the osteochondrosis syndrome. It is well recognized that the frequency of osteochondrosis is higher in male than female dogs and is highest in canine breeds that have the greatest rate of skeletal growth. If one compares relative bone density with skeletal size in individual dog skeletons of different canine breeds during the most rapid growth period of the particular breeds, one finds that the bones of large breeds of dogs are relatively less dense than bones of small breeds of dogs. For example, the long bones of larger breed dogs have a relatively thinner cortex, larger medullary cavity and a less dense spongiosa made of thinner body trabeculae having larger intertrabecular spaces than are present in small breeds of dogs (Fig. 2).

The differences in relative bone structure between large and small breeds of dogs is a reflection of differential rates of skeletal growth and bone remodeling in breeds of disparate sizes (10, 11). Biomechanical implications are that the skeletons, and especially the developing joint surfaces, of large growing dogs are less strong than comparable sites in small growing dogs. The wide-meshed or relatively osteopenic, subchondral spongiosa that develops beneath and supports
the articular cartilage of developing joint surfaces of growing dogs of large breeds is not only less sound but also potentially more vulnerable to biomechanical stress than is the subchondral bone of joints of smaller dogs.

**Influence of overnutrition (ad libitum feeding).** While the final skeletal height is a genetically determined factor and is not primarily determined by nutrition, the time it takes for the dog to achieve adult height can be influenced by nutrition. For example, intake of a diet high in energy and protein will shorten the time for completion of skeletal development that diets deficient in energy and protein will lengthen the time it takes the dog to attain adult stature.

In growing animals the increase in body weight is in balance with the rate of skeletal growth that is taking place in cartilaginous growth centers located in the deep layers of the articular cartilages of joints and in the growth plates of bones. During growth and as the body mass increases, the architecture of the articular cartilage changes from that of a primarily growth-oriented structure to one that permits both growth activity and resistance to biomechanical stress. Fine-caliber primary spongiosa initially formed by endochondral ossification in the deep layers of the articular cartilage must be strong enough to support the articular cartilage from which it arose. These thin spicules of bone are subsequently remodeled to form thicker and stronger bony spicules of secondary spongiosa. Biomechanical forces caused by increasing body weight and weight-bearing during locomotion promote fusion of the bony trabeculae of the secondary spongiosa. This secondary or adaptive remodeling process forms bony arcades that align themselves along the trajectories of biomechanical stress being exerted on the skeleton. At skeletal maturity and under the influences of the biomechanical loading of joint surfaces, the vertically-oriented primary spongiosa of the subchondral bone changes into a solid subchondral plate of bone.

Overnutrition accelerates the rate of skeletal growth. In our experiments with Great Danes puppies fed ad libitum and followed until 6 mo of age, the male puppies weighed twice as much as male puppies on a restricted-diet regimen (70–80% of ad libitum). These differences were less marked in females puppies on the same two feeding regimens (Table 1). These results are in accordance with the clinical findings in natural cases of osteochondrosis in which the incidence of osteochondrotic lesions is higher in male than female dogs. In our experiments the greater body weight was accompanied by an accelerated rate of skeletal growth (Table 2). A comparison of femoral measurements between male puppies in the two feeding groups showed a difference in femoral length (ad libitum: 244 mm; restrictive: 210 mm) and in femoral shaft cross-sectional area (ad libitum: 359 mm²; restrictive: 318 mm²). There were no significant differences between female puppies of the two feeding groups; however, similar trends were present, i.e., femoral length (ad libitum: 240 mm; restrictive: 221

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**Table 1**

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<tr>
<th></th>
<th>Ad libitum</th>
<th>Restrictive</th>
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<tr>
<td></td>
<td>kg</td>
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<tr>
<td><strong>Males</strong></td>
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<tr>
<td>[n = 4]</td>
<td>42.4 ± 4.1</td>
<td>21.8 ± 3.9</td>
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<tr>
<td><strong>Females</strong></td>
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<td>[n = 5]</td>
<td>37.6 ± 3.9</td>
<td>22.3 ± 3.7</td>
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*Values are means ± SD.

**Table 2**

<table>
<thead>
<tr>
<th>Femur</th>
<th>Nutrition</th>
<th>Length mm</th>
<th>Ratio of length to body weight mm/kg</th>
<th>Diaphyseal transverse section mm²</th>
<th>Ratio of diaph. transv. section to body weight mm²/kg</th>
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<tbody>
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<td></td>
<td>Ad libitum</td>
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<tr>
<td><strong>Males</strong> [n = 4]</td>
<td>244 ± 19.5</td>
<td>6.2 ± 0.6</td>
<td>359 ± 2.8</td>
<td>8.6 ± 1.2</td>
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<tr>
<td><strong>Females</strong> [n = 5]</td>
<td>240 ± 7.4</td>
<td>6.4 ± 0.9</td>
<td>340 ± 58.6</td>
<td>9.1 ± 1.4</td>
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<tr>
<td><strong>Restrictive</strong></td>
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<tr>
<td><strong>Males</strong> [n = 3]</td>
<td>210 ± 17.3</td>
<td>9.6 ± 0.8</td>
<td>318 ± 10.4</td>
<td>15.0 ± 3.5</td>
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<tr>
<td><strong>Females</strong> [n = 6]</td>
<td>221 ± 10.4</td>
<td>10.0 ± 1.2</td>
<td>324 ± 14.5</td>
<td>14.9 ± 3.1</td>
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*Values are means ± SD.
mm) and femoral shaft cross-sectional area (ad libitum: 340 mm²; restrictive: 324 mm²).

The accelerated growth rate caused by overnutrition affects both intrinsic and extrinsic factors involving the skeleton. Bone tissue structure making up a bone organ is dynamic because of an intrinsic bone remodeling mechanism that can make an adaptive response to biomechanical forces exerted on the skeleton. The bone remodeling mechanism modifies the microscopic architecture of the cancellous and compact bone structure in a manner that resists biomechanical forces exerted on the bone organ or on the subchondral bone of joint surfaces. The two different feeding regimens used in the Great Dane puppies in our experiment produced different patterns of bone structure in the two groups of puppies.

Differences were noted in the character of the subchondral bone of joint surfaces and in the cancellous bone produced by growth plates of long bones. In the developing joint surfaces of dogs in the ad libitum group, endochondral ossification was still very active in the deep layers of the articular cartilage and there was, as yet, no attempt to form an intact plate of subchondral bone (Fig. 3). However, in developing joint surfaces of puppies on the restricted-diet regimen, endochondral ossification was slower, and a partially complete subchondral bony plate had been established.

In the growth plates and adjacent metaphyseal spongiosa of long bones of dogs in the ad libitum group, endochondral ossification was intense. Primary and secondary spongiosa was formed of relatively thin bony trabeculae that were set relatively far apart, and all of the spongiosa was undergoing very active remodeling, particularly beneath the periosteum of the metaphyseal surface (Fig. 4). By comparison with similar sites in the long bones of dogs on the restricted-diet regimen, endochondral ossification was less intense. Primary and secondary spongiosa was formed of relatively thicker bony trabeculae that were set relatively close together. Bone remodeling was less active in the spongiosa and on the periosteal surface of the metaphysis.

From our experiment in Great Dane puppies it was concluded that overnutrition accelerates the rate of bone growth. The accelerated rate of bone growth causes both an increase in the size and volume of the bone organs and an increase in the accompanying rate of bone remodeling within the bone organs. The end result of overnutrition on the dog skeleton is the creation of enlarged bone organs formed of cancellous and compact bone having relatively low density and, consequently, having lower resistance to biomechanical loading. Dogs subjected to overnutrition frequently develop focal lesions of cartilage deformation.

**FIGURE 3** Articular cartilage of the humeral head and supporting subchondral bone tissue: Great Danes, 6 mo old. left: complete support; restrictive feeding; right: incomplete support, ad libitum feeding (H & E, ×75).
at sites on joint surfaces and beneath growth plates of long bone where spongiosa of abnormally low density cannot support the local biomechanical load [Fig. 5].

The accelerated growth rate caused by overnutrition can also have effects on the skeleton that are initiated by extrinsic factors, such as the development of a large muscle mass and increased body weight. Under normal conditions there is a close correlation between the rate of increase in body weight and in the rate of skeletal growth. Skeletal growth occurs at physeal centers of endochondral ossification, and attainment of the adult weight coincides with completion of physeal growth.

Under abnormal conditions such as in our experiment, the Great Dane puppies fed ad libitum not only had greater skeletal growth rates, greater body weights and larger muscle masses than pups fed a restricted diet but also increased their body weights at a rate that was greater than their skeletal growth rate. This conclusion was based on finding that the difference in body weights between ad libitum and restricted groups was greater than the differences in the femoral lengths or in the cross-sectional areas of the femoral diaphyses (Table 2).

Overnutrition in the growing puppy creates a mismatch between the rates of body weight increase and skeletal growth, and this eventually leads to overload-
layers of the articular cartilages of synovial joints of growing dogs. Occurrence of lesions is influenced by the capacity for rapid skeletal growth inherent in large and giant breeds of dogs, the capacity of dogs of these breeds to attain great skeletal size and by growth characteristics of male members of the breeds.

How overnutrition (ad libitum feeding) causes and promotes the osteochondrosis syndrome is controversial. According to Olsson (1) and Schulze Schleithoff (12), the articular lesions in spontaneous cases of osteochondrosis are biomechanical in nature. They are caused by increased body weight from a high energy diet and subsequent overloading of the cartilage tissue of immature joint surfaces. Others conducting experimental studies of the effects of overnutrition on skeletal development implicate the role of increased amounts of calcium in the diet. According to them, chronic hypercalcemia leads secondarily to hypercalcitoninism and hypoparathyroidism, which are directly responsible for the development of the cartilaginous lesions in osteochondrosis (7, 8).

Our experimental investigations into the pathogenesis of osteochondrosis in large dogs due to overnutrition indicate that the articular lesions develop secondary to faulty subchondral bony support of the articular cartilage, which interferes with cartilage tissue nutrition, metabolism, production of ground substance, viability and function in endochondral ossification. The primary problem, i.e., faulty subchondral bony support, results from the effects of overnutrition on promoting rapid skeletal growth and rapid bone remodeling of newly formed cancellous bone. These two mechanisms result in the formation of a delicate primary spongiosa beneath the articular cartilage, which is quickly remodeled and replaced by a secondary spongiosa composed of relatively thin bony trabeculae spaced relatively far apart. The resulting subchondral bone formed by a wide meshwork of relatively thin bony trabecular systems fails to provide adequate biomechanical support for the articular cartilage.

In addition to overnutrition of immature dogs of the large breeds causing rapid skeletal growth and the development of bone organs that are large but low in bone density and strength, overnutrition also produces an even greater acceleration in the rate of growth of the musculature of the body. This combination of mismatched growth rates results in the formation of a skeleton that cannot always meet the biomechanical demands of the attached muscle mass. This mismatch leads to focal failures in joint surfaces and growth plates, which are recognized as the lesions of osteochondrosis.

In none of the dogs in our study, either dogs with spontaneous lesions of osteochondrosis or with experimentally induced lesions due to overnutrition, were there any indications of hypercalcitoninism. Two main histologic manifestations of hypercalcitoninism on bone, i.e., decreased osteoclastic activity and the formation of abnormally dense, bluish-stained bone tissue, were absent.

In our experience some dogs that have mild spontaneous cases of nutritional secondary hyperparathyroidism will also have mild articular lesions indistinguishable from those of osteochondrosis. Parathyroid hormone production stimulates osteoclastic bone resorption, particularly in cancellous bone. In moderately severe cases of hyperparathyroidism, osteoclastic bone destruction may be focal and severe enough in the subchondral spongiosa to cause a loss of support.
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for the articular cartilage. Secondary changes resembling those seen in osteochondrosis subsequently develop in the cartilage. We consider that such findings support our belief that the primary lesions in the osteochondrosis syndrome are caused by inadequate subchondral bony support for the articular cartilage during skeletal development.

LITERATURE CITED


