Evaluation of the effect of limited food consumption on radiographic evidence of osteoarthritis in dogs

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Objective—To determine prevalence of radiographic evidence of osteoarthritis in 4 diarthrodial joints of dogs with restricted feed intake, compared with dogs without restricted feed intake.

Design—Paired feeding study.

Animals—48 Labrador Retrievers.

Procedure—Dogs in litters from 7 dams and 2 sires were paired by sex and weight within litters and randomly assigned to a control-fed group or a limit-fed group that received 25% less food than the control-fed group. Radiographic evaluation of prevalence and severity of osteoarthritis in the hip, shoulder, elbow, and stifle joints was performed when dogs were 8 years of age.

Results—Radiographic evidence of osteoarthritis that affected multiple joints was significantly more common in the control-fed group than in the limit-fed group. Prevalence of lesions in the hip joint was 15/22 in the control-fed group and 3/21 in the limit-fed group. Prevalence of lesions in the shoulder joint was 19/22 in the control-fed group and 12/21 in the limit-fed group; lesions in this joint were generally mild. Severity, but not prevalence, of osteoarthritis in the elbow joint was greater in the control-fed group than in the limit-fed group.

Conclusions and Clinical Relevance—Prevalence and severity of osteoarthritis in several joints was less in dogs with long-term reduced food intake, compared with control dogs. Food intake is an environmental factor that may have a profound effect on development of osteoarthritis in dogs. (*J Am Vet Med Assoc* 2000;217:1678–1680)

Osteoarthritis (OA) is a common clinical condition in veterinary medicine that seriously affects the quality of life of many dogs. Cause of the disease has not been fully explained, and treatments are, at best, palliative. Multiple joint involvement has been recognized in OA in humans[1-4] and in osteochondrosis in several animal species.[5] Hip, shoulder, stifle, and lumbar vertebral joints of dogs of many different breeds commonly had OA, and approximately 30% of dogs had 2 or more affected joints.[6] Whether multiple joint involvement indicates a systemic or generalized disease of bone or other joint tissues is unknown.

Results of previous studies indicate that limiting food consumption to 75% of that fed to control dogs from 8 weeks of age in a matched group of Labrador Retrievers resulted in a 67% reduction in the prevalence of hip dysplasia at 2 years of age[7] and substantially reduced the prevalence and severity of hip joint osteoarthritis at 5 years of age.[8] That dietary study was continued, and the purpose of the study reported here was to determine the prevalence of radiographic evidence of OA in 4 diarthrodial joints in the same dogs at 8 years of age.

Materials and Methods

Dogs and housing—Forty-eight Labrador Retrievers, the progeny in litters from 7 dams and 2 sires, were allotted to a paired feeding study when they were 8 weeks of age. Dogs were paired by sex and body weight within the litter prior to random assignment to dietary treatment. Dogs were housed in 2 × 19-m indoor-outdoor kennel runs with concrete floors for 8 years. The amount of exercise the dogs received was not controlled.

Diet and feeding regimen—Samples from each of 25 batches of food prepared during the 8-year period were analyzed for moisture, protein, fat, fiber, ash, calcium, phosphorus, sodium, potassium, and chloride according to methods approved by the Association of Official Analytical Chemists. Control-fed dogs were maintained on a paired feeding regimen, as described.[7,8] The control feeding method was instituted at approximately 3.25 years of age to prevent obesity in dogs that previously were fed ad libitum. At that time, ad libitum-fed pairmates were changed to a control-fed regimen defined as 62.1 kcal of **metabolizable energy (ME)**/kg (28.2 kcal of ME/lb) of ideal body weight/d. Limit-fed dogs received 25% less food than control-fed pairmates. Whenever it was judged that this feeding methodology may impair the health of the limit-fed pairmate, upward adjustments consisting of 10% increments were made until the limit-fed pairmate maintained body weight. The control-fed pairmate received the same proportionate upward adjustment so that the limit-fed dog always received 25% less food.

When a control-fed dog developed a clinical disorder that resulted in decreased food intake, the limit-fed pairmate was maintained at the level of food intake it had been receiving prior to development of clinical signs. When a dog died or was euthanatized, its pairmate was maintained on the amount of food it had been receiving prior to that time.

Dog health and monitoring—All dogs were maintained under veterinary supervision, and physical examinations were performed annually. Dogs were given standard vaccinations and antiparasite treatments. When a dog developed clinical signs of a treatable disorder, as determined by the attending clinician (DFL), the dog was treated accordingly,

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and the feeding regimen for both pairmates was maintained. Hematologic and serum biochemical analyses were obtained when dogs were 6, 11, 16, 26, 30, 42, 54, 78, and 102 weeks old and then annually. Body weight was recorded at the same intervals from 6 to 102 weeks of age, at 3 and 3.25 years of age, and then weekly until dogs were 8 years of age.

Evaluation of OA—Radiographic evaluation of prevalence and severity of OA in hip, shoulder, elbow, and stifle joints was performed when dogs were 8 years of age. Joints were evaluated without knowledge of experimental treatment. The OA scoring methodology for hip joints was conducted, as described.[8]

Shoulder joints—By use of the lateral radiographic view of each shoulder, degree of OA was scored subjectively on the basis of severity of osteophyte formation on the margins of the glenoid cavity of the scapula and the humeral head, mineralization in the region of the bicipital groove of the humerus, and presence or absence of an intra-articular fragment.

Elbow joints—By use of flexed lateral and craniocaudal radiographic views of each elbow, degree of OA was scored subjectively on the basis of severity of osteophyte formation on the radial head, the humeral condyles and the nonarticular margin of the anconeal process, presence or absence of joint congruity, and presence or absence of subchondral sclerosis. The degree of osteophytosis was scored on the basis of the International Elbow Working Group's classification of mild, moderate, and severe arthrosis.

Stifle joints—By use of the lateral radiographic view of each stifle joint, degree of OA was scored subjectively on the basis of severity of osteophytosis on the patella, femoral trochlea, tibial plateau, and fabellae, presence or absence of a normal or abnormal infrapatellar fat pad, and presence or absence of popliteal sesamoid bone displacement.

Within each joint, each category of OA was subjectively scored on radiographs as follows: disease-free, 0; mild OA, 1.0; moderate OA, 2.0; and severe OA, 3.0. Increasing scores in each category represented progressive increases in osteophyte formation, subchondral sclerosis, and joint remodeling. The highest score among the scoring categories within a joint was used as the estimate of OA in that particular joint. When joints were involved bilaterally, the joint with the highest OA score was used in data analysis. All radiographs were evaluated at the same session to provide consistency of scoring.

Statistical analyses—Differences between dietary regimen for evidence of OA (present vs absent) within each joint were analyzed by use of the McNemer test for matched pairs.[9] A score test for marginal homogeneity was used to test for treatment differences for the ordinal severity of OA (none, mild, moderate, severe).[10] Differences were considered significant at $P < 0.05$.

Results

Results of laboratory analyses indicated that diets continued to meet nutritional criteria, as reported.[7,8] Mean (± SD) body weights after 8 years were 33.7 ± 6.02 and 24.2 ± 3.79 kg (74.1 ± 13.2 and 53.2 ± 8.3 lb) for control-fed and limit-fed dogs, respectively. Range in body weights was 24.7 to 44.4 kg (54.3 to 97.7 lb) for control-fed dogs and 16.6 to 29.7 kg (36.5 to 65.3 lb) for limit-fed dogs. Mean daily caloric intake during the 8-year period was 1,786 and 1,359 kcal of ME for control-fed and limit-fed dogs, respectively.

Five dogs died during the 8-year study period; 4 of these dogs were euthanatized. Causes of mortality in the control-fed group included invasive squamous cell carcinoma of the maxilla and fibrocartilaginous embolism of the spinal cord, whereas mortality in the limit-fed group was attributed to ileal infarction, megaesophagus, and mammary adenocarcinoma. One limit-fed dog and 5 control-fed dogs required daily treatment for signs of pain attributed to OA.

Osteoarthritis that affected multiple joints was significantly ($P = 0.01$) more common in the control-fed group, compared with the limit-fed group. Ten of 22 control-fed dogs had OA in 2 different joints, whereas 1 of 21 limit-fed dogs had OA in 2 joints. Seven of 22 control-fed dogs had OA in 3 joints, whereas only 1 of 21 limit-fed dogs were similarly affected. Five of the limit-fed dogs had normal radiographic findings in all evaluated joints, whereas 2 of the control-fed dogs had normal radiographic findings in all joints.

Hip joints had the most severe lesions of OA (**Fig 1**). Prevalence of hip joint lesions in control-fed dogs was 15/22, compared with 3/21 in limit-fed dogs ($P = 0.01$). Severity of lesions was significantly ($P = 0.01$) greater in the control-fed group. The most common lesion was periarticular osteophyte formation in the proximal aspect of the femur, which developed in 14 of 22 control-fed dogs and in 3 of 21 limit-fed dogs.

Lesions in the shoulder joint, although more common than lesions in other joints, generally were mild (Fig 1). Prevalence of OA in shoulder joints was 19/22 for control-fed dogs and 12/21 for limit-fed dogs ($P < 0.05$). The most common lesion was a small osteophyte on the caudal aspect of the humeral head.

Prevalence of OA in the elbow joint was moderate overall, with 8 of 22 control-fed dogs and 4 of 21 limit-fed dogs affected (Fig 1). Severity, but not prevalence, of OA was significantly ($P < 0.05$) greater in the control-fed group. The most common lesion observed was an osteophyte on the proximal nonarticular margin of the anconeal process, which developed in all affected dogs. Only 2 cases of mild OA were observed in stifle joints in the control-fed group. Osteoarthritis of the stifle joint was not detected in the limit-fed dogs.

Bilateral OA was most common in the hip and shoulder joints (**Table 1**). For these 2 joints, the ratio of bilateral lesions to unilateral lesions was approximately 2:1. The ratio of bilateral to unilateral lesions in

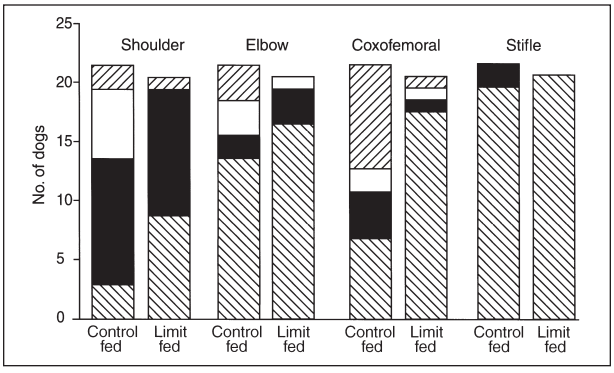

Figure 1—No. of dogs with severe (▨), moderate (□), mild (■), or no (▧) radiographic lesions of osteoarthritis in various joints. Limit-fed dogs received 25% less food than control-fed dogs during an 8-year study.

Table 1—Distribution (No.) of dogs without osteoarthritis (OA) or with unilateral or bilateral osteoarthritis in various joints

Joint	No OA	Unilateral	Bilateral
Shoulder	12	10	21
Hip	25	6	12
Elbow	31	7	5
Stifle	41	1	1

the elbow joint was approximately 1:1. One dog had unilateral lesions in the stifle joint, and 1 had bilateral lesions. The ratio of unilateral to bilateral lesions in joints did not differ significantly between groups.

Prevalence of multiple joint involvement in declining order for various joint combinations in control-fed versus limit-fed dogs, respectively, was as follows: shoulder-hip, 15/22 versus 2/21; shoulder-elbow, 7/22 versus 1/21; hip-elbow, 5/22 versus 1/21; and shoulder-hip-elbow, 5/22 versus 1/21. Three other multiple-joint combinations were detected in only 2 dogs. The shoulder-hip combination was identified in 40% of all dogs.

Discussion

The major new finding of the study reported here was that when food intake was limited by 25%, compared with control dogs, from 8 weeks to 8 years of age, prevalence and severity of OA was low in the shoulder, hip, and elbow joints.

Radiographic signs of OA were observed in the 4 diarthrodial joints we examined, either individually or in combinations. The combination of affected shoulder and hip joints was observed in 68% of dogs in the control-fed group and 10% of dogs in the limit-fed group. In the control-fed group, 77% of dogs had either 2 or 3 different joints with OA, compared with 10% in the limit-fed group. Previously, multiple joint lesions of OA were reported in 38% of 16 Labrador Retrievers and 32% of 100 adult dogs of various breeds.[6] In the study reported here, the number of dogs with lesions in multiple joints was substantially lower in the limit-fed dogs, compared with control-fed dogs.

Multiple joint involvement in a dog may be attributable to mechanical damage that develops subsequent to effects of arthritis at a primary site such as the hip joint. Development of OA at a primary site theoretically could result in alterations of biomechanical forces acting on other joints, altered weight-bearing, and altered ambulation as a compensatory response. In our study, hip joints, however, were not always affected first, because some of the dogs had shoulder or elbow joint lesions without hip joint involvement. It may be hypothesized that OA has a systemic cause, with variable expression in different joints; this may partially explain the development of disease in joints that are not subject to large forces associated with weight-bearing such as lumbar intervertebral joints in dogs and joints in the skeleton of sharks.[1] Alternatively, a humoral substance from an affected joint may affect tissues of other joints. This concept is supported by the recent finding that an extract of human arthritic bone tissue induced abnormalities in disease-free articular cartilage.[11] Additional support for the concept of variability in tissue susceptibility is derived from a recent report that shoulder joints have articular cartilage abnormalities similar to osteoarthritic joints in young adult dogs at high risk for hip dysplasia.[12]

Results of our study of 8-year-old Labrador Retrievers confirm our previous reports that limited food intake is an environmental factor that may have a profound effect on the prevalence of OA in the hip joint in dogs. In addition, our results indicate that prevalence of OA in other joints is lower in dogs with restricted food intake.

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