An overview of the pathogenesis of canine hip dysplasia

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Canine hip dysplasia (CHD) is an inherited, developmental condition that involves a lack of conformity between the femoral head and acetabulum and invariably leads to osteoarthritis (OA). Canine hip dysplasia is diagnosed radiographically, but it is expected that dogs with radiographic evidence of CHD will have pathologic or clinical signs of OA (Fig 1). It seems unlikely that OA per se is inherited in dogs with CHD, because OA is usually thought of as a sequela of the malarticulation of the joint components. However, the inherited part of the disease process is currently unknown, and despite many attempts to control the disease in purebred dogs through selective breeding programs, CHD is still a common orthopedic disease, especially in larger dog breeds.

Age for Diagnosis

Time of appearance of radiographic signs of CHD in a group of Labrador Retrievers of known age has been studied (Fig 2). A similar time course has been reported for German Shepherd Dogs and dogs of mixed breeding. Mean age at which CHD was first evident on a standard, legs extended, radiographic projection was about 6 months. The disease frequently first appears in susceptible dogs when they are between 4 and 12 months old, although in some dogs, the disease is not evident radiographically until they are ≥ 24 months old. It is reasonable to assume that milder pathologic soft-tissue changes are already developing in these joints in the weeks and months before the disease is radiographically detectable. In fact, excessive joint laxity, a putative precursor and risk factor for OA, can be measured at 4 months of age. However, in 1 study, degree of laxity at 8 months was a more accurate predictor of the risk of OA than was degree of laxity at 4 months of age.

Role of Joint Laxity

Laxity of the hip joint has been recognized as a constant feature of CHD. Degree of joint laxity (i.e., looseness or instability) appears to have a hereditary basis, in that progeny of dogs without CHD had less laxity than did those from dysplastic parents. To date, joint laxity has been measured most often by veterinarians.
narians or dog owners as a means to predict which young puppies will develop OA. However, whereas puppies with low laxity (ie, tight joints) are unlikely to develop OA, not all puppies with high laxity (ie, loose joints) will (Fig 3). Therefore, a practical use for measurements of hip joint laxity is in selecting breeding stock with low laxity, which should reduce the prevalence of OA in the progeny. This has been proposed by several investigators3,5,6 as a method of reducing the prevalence of CHD and, thus, OA in populations of dogs, but the concept must be substantiated in controlled breeding programs. Furthermore, an explanation must be sought to account for adult dogs with substantial joint laxity that do not develop OA.7,9

**Genetics**

Evidence in several reports gives credence to the notion that CHD has a hereditary basis. Many offspring from parents with CHD also become dysplastic; often several or all siblings in a litter are dysplastic. Likewise, dogs without CHD are more likely to be born to parents without CHD. Willis8 summarized results of a number of breeding programs and concluded that in various dog populations, between 64 and 81% of the progeny of normal dogs (as determined on the basis of a standard radiographic examination) would also be normal, and between 19 and 36% would be dysplastic. On the other hand, between 17 and 37% of the progeny of dysplastic dogs would be normal, and between 63 and 93% would be dysplastic (Table 1). Conventional wisdom holds that the genetic basis of CHD is a continuously varying (ie, quantitative) trait. Evaluations of the inheritance of CHD suggest that >1 gene is involved, but the number is unknown.8,9 The expression of the disease (ie, phenotype) appears to be determined by an interaction between genetic heritage (ie, genotype) and environmental factors (eg, food consumption).10

**Growth of Dogs**

Canine hip dysplasia, in contrast to a similar disease in human beings,11 cannot be diagnosed at birth. The condition appears to be in its earliest stages during a puppy's rapid growth phase during the first 6 months of life. The relationship between growth and the appearance of CHD has been studied by several investigators10,12 who have concluded that growth rate and the associated weight of the dog influence the CHD phenotype. In 2 studies,12 for instance, data were presented supporting the view that abundant (super-optimal) food consumption (overfeeding) shortened the time to first appearance and also increased severity of CHD. In contrast, in a recent controlled study,13 the frequency and the severity of CHD in Labrador Retrievers were substantially reduced by limiting food consumption when dogs were between 6 weeks and 1 year of age.

**Involvement of Multiple Joints**

Canine hip dysplasia most often is thought to be an abnormality involving only the tissues in the region of the hip joint. But studies indicate that the shoulder and stifle joints and the lumbar vertebral joints in dogs with CHD often have similar patho-

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**Figure 2**—Age when radiographic signs of CHD were first detected in 102 Labrador Retriever puppies from 17 litters born to dysplastic parents. Puppies were radiographed at monthly intervals.

**Figure 3**—Relationship between degree of coxofemoral joint laxity (ie, distraction index) at 8 months of age and the probability that a dog would have normal hip joints at 2 years of age. Distraction indexes range from 0 (no laxity) to 1 (joint luxation). * = 95% confidence interval. (Reprinted with permission from Lust G, Williams AJ, Burton-Wurster N, et al. Joint laxity and its association with hip dysplasia in Labrador Retrievers. Am J Vet Res 1993;54:1990-1999).
logic changes, although the hip joints more frequently have osteoarthritic changes than do these other joints.12,13 In 1 study,13 multiple joint involvement was recorded in 30 to 40% of dogs, and the tissue and biochemical changes were similar, if not identical, in the shoulder and hip joints of dogs that had CHD and shoulder OA. Thus, CHD and OA may simply be the most conspicuous manifestations of a more generalized abnormality affecting several joints in dogs.

Conclusion

In the past few years, interest in CHD research has been at an all-time high, as evidenced by the number of conferences focusing on the subject14 and by the number of new publications in scientific journals3,4,10,13-17 and popular magazines.10-20 It is useful to summarize results from the scientific literature at conferences and in reviews, but in the final analysis, more research is needed to find answers to unresolved questions about CHD (Appendix). It is encouraging that a number of current grant-supported research programs are addressing this need.

References


Appendix

Unanswered questions regarding the pathogenesis of canine hip dysplasia (CHD)

- What is the essential cause of CHD and osteoarthritis? What roles do hormonal effects, enchondral ossification, growth plate abnormalities, the joint capsule, and muscle metabolism have?
- What is the cause and role of excessive joint laxity?
- How can we explain the association between food consumption and prevalence of CHD?
- How can we explain instances of unilateral CHD?
- Are multiple joints involved in dogs with CHD? If so, why is the disease most obvious in the hip joint?
- Are birth order and presentation (i.e., breech birth) important?
- Why is the prevalence so high in large dogs?
- How many genes are involved?
- Can molecular genetic markers help in the detection of CHD? Would genetic markers be useful for detection of CHD in mixed-breed dogs?
- What cellular and biochemical mechanisms lead to osteoarthri

Table 1—Percentages of normal and dysplastic puppies expected from matings of normal and dysplastic dogs

<table>
<thead>
<tr>
<th>Type of mating</th>
<th>Normal × normal</th>
<th>Normal × dysplastic</th>
<th>Dysplastic × dysplastic</th>
</tr>
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<tbody>
<tr>
<td>Progeny Normal (%)</td>
<td>75 (64-81)</td>
<td>50 (41-66)</td>
<td>25 (7-37)</td>
</tr>
<tr>
<td>Dysplastic (%)</td>
<td>25 (19-36)</td>
<td>50 (34-59)</td>
<td>75 (63-93)</td>
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