

# Canine Hip Dysplasia Part V

## *Predicting the Abnormal Hip*

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*An evaluation method is needed that is not only diagnostic but which can predict the probability of canine hip dysplasia.*

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This article is the fifth in an eight-part series on canine hip dysplasia (CHD). What follows is written from the perspective that the readers are serious and conscientious breeders who are the guardians of the genetic pools that constitute their breeds. While this series of articles will not replace a stack of veterinary and medical texts, it is a relatively in-depth look at the whole problem of canine hip dysplasia. Furthermore, the series is designed to be retained as a reference. When you finish reading this series, you will have a sufficient background to make rational breeding choices and will be able to discuss the subject from an informed basis with your veterinarian. You may not like what you read, but you will be more competent to deal with the problem.

**Conclusions from part I:** Genetics is the foremost causative factor of canine hip dysplasia. Without the genes necessary to transmit this degenerative disease, there is no disease. Hip dysplasia is not something a dog gets; it is either genetically dysplastic or it is not. An affected animal can exhibit a wide range of phenotypes, all the way from normal to severely dysplastic and functionally crippled. Hip dysplasia is genetically inherited.

**Conclusions from part II:** While environmental effects, to include nutrition and exercise, may play a part in mitigating or delaying the onset of clinical signs and clinical symptoms, hip dysplasia remains a genetically transmitted disease. Only by rigorous genetic selection will the incidence rate be reduced. In the meantime, it makes sense to have lean puppies and to avoid breeding animals from litters that showed signs of hip dysplasia. It is probable that even normal exercise levels may increase the phenotypic expression of CHD of a genetically predisposed dog. Stay away from calcium supplementation of any kind; all it can do is hurt. There is no conclusive evidence that vitamin C can prevent hip dysplasia, but there is some evidence that vitamin C may be useful in reducing pain and inflammation in the dysplastic dog. Fortunately, large doses of vitamin C are readily excreted, but it is still possible to cause untoward side effects with megadoses.

**Conclusions from part III:** Canine hip dysplasia can be difficult to diagnose, as a number of other orthopedic neurological, autoimmune and metabolic problems may mimic it. Controversy surrounds the question of positioning for hip X-rays and what part joint laxity plays in hip dysplasia. Hip dysplasia may be more common in large and giant breeds and is one of the most over-diagnosed and misdiagnosed conditions.

**Conclusions from part IV:** Sadly, no breed registry in the United States requires genetic screening of parents as a prerequisite for litter registration or even offers a "fitness for breeding" certification. The current registries for hip dysplasia (and other genetically transmitted problems) cover so little

of the American Kennel Club-registered dog population that their impact so far has been minimal. The tools we need are there. Joint responsibility for failing to use the tools at hand lies with the AKC, United Kennel Club, parent clubs and individual breeders.

This article will cover the Orthopedic Foundation for Animals vs. PennHIP controversy, the requirement and desirability of an evaluation method that is not only diagnostic but also prognostic with an ability to predict the probability of phenotypic expression of hip dysplasia. Hand in hand with these methods goes the requirement for positive identification rather than the honor system currently in place and the concept of "open" genetic registries in order that genetic pedigree research can be done.

The first four articles in this series have generated many letters. In response, we restate that dogs of any recognizable breed, i.e., non-feral dogs, are inbred on a relatively small number of genes. Each breeding to members of the same breed constitutes continued inbreeding and thus further reduces the gene pool (genetic depletion), thus giving increased probability that recessive traits-desirable and undesirable-will match from each donor and will be expressed phenotypically in their get. We restate that it is desirable to inbreed (and line-breeding is inbreeding) to maintain breed characteristics. Unfortunately, over time this will cause more problems than it will solve, as virtually every dog (and human) carries several defective genes.

## **Preventing genetic depletion**

A basic fundamental fact of genetics is that genetic health decreases with every generation of breeding within a breed. This point must be made very clear. Only 10 to 30 genes distinguish one breed from the next, yet in the dog thousands of recessive and co-dominant genes also become fixed in the genetic makeup of a breed. The only way to prevent genetic depletion and its resultant inbreeding depression is to outcross for hybrid vigor.

The various registries will have to understand the genetics of the situation: To maintain genetic vigor, breeds will have to outcross. In the near term this heretical necessity can be temporarily staved off through restricting stud use, as is being done in some European breed clubs. AKC, in recent years, literally saved the Dalmatian from extinction (nobody wanted a breed of deaf dogs, regardless of other characteristics) by allowing breeding to non-Dalmatians. Similarly in Europe, Dutch Shepherd Dogs were outcrossed with the Belgian Tervuren, and Bernese Mountain Dogs were crossed with Newfoundlands. Some will decry this practice, calling it the blackest of heresy; others will rejoice in the genetic salvation. In the meantime, genetic screening and open registries of genetic traits could allow the identification and breeding to the least genetically related animals in a breed's gene pool.

"therefore, the breeder controls the occurrence of hip dysplasia in his/her breed."<sup>1</sup>

This is a quote from a recent memorandum from the OFA to the breed club representatives. So once again breeders must take the blame, yet how many of you have bred an OFA "normal" to another OFA "normal" and still produced dysplastic puppies? Some unscrupulous breeders commit fraud and offer a dog for OFA certification using the papers of another animal,, but most of us are conscientious breeders. We love our dogs and our breed and really want to eradicate this insidious

disease.

## **Orthopedic registries: Hip or hype?**

Could something be wrong with the current method of evaluating an animal for hip dysplasia? Where are the scientific papers that prove the efficacy of the OFA diagnostic method? Where are the peer reviews of these papers? From what population data do they base their conclusions?

What is population data? It is a term that statisticians use. It isn't feasible to check every single dog for a particular condition so one simply checks a sample population. However, to be accurate that sample must truly represent the entire population. It is our contention that the OFA is basing its conclusions on self-selected and therefore biased, data. The OFA does not require of veterinarians that all radiographs of client dogs taken for initial evaluation be submitted to OFA. Each breeder must answer this question: Do you send X-rays to the OFA that your own vet feels are from a dysplastic animal? We thought not. So, if the OFA is mostly seeing "normal" hips, on what does it base its claim that the incidence of the disease is decreasing in some breeds? It also claims to have evaluated a significant percentage of those breeds most likely to be affected.<sup>2</sup> In the last 20 years, less than one percent of all the dogs registered by the AKC have been evaluated by OFA, so what does OFA consider significant?

## **Predicting genotype**

Let us now consider the diagnostic method used by the OFA and its ability to predict genotype based on phenotype. In other words, does the physical appearance of the dog tell us what genes he is carrying? This is not the case, unfortunately, because the appearance of the animal shows only the genes he is expressing. The hip-extended view used by the OFA is good for evaluating an existing problem with degenerative hip disease when that diagnosis is based upon the specific radiographic signs of osteophyte formation, subchondral sclerosis and joint remodeling, and not subluxation. In a previous article in this series, it was demonstrated that the hip-extended radiographic view actually masked joint laxity or "looseness."<sup>3</sup> The hip-extended position actually "screws" the femoral head into closer congruity with the acetabular cup.

If there is also a correlation between joint laxity and the subsequent development of degenerative joint disease (and we feel that this already has been demonstrated), then a diagnostic method that conceals this fault may negate its predictive value.<sup>4,5</sup> We should also examine two other factors that can influence the effectiveness of a diagnostic method. These factors are the scoring procedure and the reproducibility of the scoring technique. The OFA uses a seven-point, subjective hip-scoring scheme that has an inherent flaw.<sup>6</sup> When evaluating a radiograph using this method it is possible to choose between Borderline and Mild Hip Dysplasia. Because of the problems associated with wide variation in interpretation among radiologists and even the agreement of an examiner with himself or herself, this scoring technique can introduce a false-negative into the breeding pool. For our purposes as breeders, this means that a dog that should not be used for breeding is allowed to propagate, further delaying the elimination of deleterious genes.

Since the first article in this series, we have been taken to task by a number of veterinarians,

anatomists and radiologists who feel that the variance in structure between breeds requires different definitions of normal hips. For example, the angle of the pelvis, flexion and elasticity of the spine and differing gaits among breeds all contribute to a separate definition of what should clinically constitute a good set of hips for a given breed. For example: the German Shepherd Dog, with its feet out somewhere in the lower 40 acres, experiences a lever and fulcrum action that exerts more force on the hip joint than if the legs were underneath the dog. It may well be-and is according to some of the veterinarians and breeders who have written in response to the earlier articles in this series-that the German Shepherd Dog must have tighter hips with deeper acetabular cups than other breeds if its hips are to be considered normal. These are issues that bring into question the practice of relying solely on radiographic evidence of hip dysplasia when there are no other clinical signs. He's 10 years old, moves like a dream, but bad hips by radiograph. Is this a dog that has bad hips, or is there some problem with the definition of good hips?

### **The role of hip laxity**

In 1982, a group of researchers and clinicians at the University of Pennsylvania School of Veterinary Medicine, who were concerned that the incidence of canine hip dysplasia did not seem to be decreasing began to investigate the role of passive hip laxity in the development of degenerative hip disease. Using mass-selection techniques, i.e., breeding "normal" to "normal," was still producing a greater incidence of CHD than would be expected. Since a genetic screening test for this disease is not available, the problem these researchers faced was to select a phenotypic trait that was most likely to reflect the dog's genotype with respect to CHD, one that would be the least effected by environmental factors. They concluded that functional hip laxity was the most likely condition that predisposed an animal to future degenerative joint disease due to biomechanical stress on the joint and the subsequent cartilage damage.<sup>7</sup> Herein lies the prediction capacity of the PennHIP system. Since it is impossible to measure functional hip laxity directly they proposed that passive hip laxity was a prerequisite for functional hip laxity, though not itself a causal event. "Some dogs, in fact, have a greater tolerance for passive laxity. That a well-muscled breed may have marked passive laxity yet be naturally protected from functional hip laxity by prominent hind limb musculature." Examples of exceptionally muscled dogs are the fighting, carting and freighting dogs.

What this means is that the biomechanical stresses on the joint due to the lateral displacement of the femoral head while the dog is standing in a normal stance are different from the supine animal, yet there remains a correlation. This correlation has been tested extensively for statistical significance.

"Passive hip laxity, then, may be considered a risk factor or perhaps loosely defined, a carrier state for HD in dogs"<sup>8</sup>

The OFA maintains that the issue of joint laxity as a predictor of CHD is neither new nor revolutionary.

"The [1972, author's note] symposium concluded and published that there was no scientific evidence to support the clinical application of palpation and/or stress radiography."<sup>9</sup> The methodology and the scoring techniques for these early diagnostic techniques were highly

subjective and depended largely on the skill and experience of the individual examiner. To address these concerns, the University of Pennsylvania researchers first determined what the normal range was for the degrees of freedom in the coxofemoral joint, where passive laxity is maximized.<sup>10</sup> This work was necessary in order to design a precise and accurate clinical stress-radiographic method that would hold up statistically.

The canine hip has four degree of freedom. Flexion/extension is when the leg moves forward toward the belly or back away from the body-what a breeder/exhibitor would call the "side gait." Abduction/adduction is when the dog moves the leg sideways away from the body or inward toward the belly. Internal/external rotation is the twisting motion the femur can make within the acetabulum until restrained by the round ligament and the joint capsule. Lateral translation is the sideways displacement or passive laxity. Maximal passive laxity, which approximates the neutral weight-bearing stance, was obtained at 10 degrees extension, 20 degrees of abduction and 10 degrees of external rotation.<sup>11</sup>

This early study also revealed the limitations of the hip-extended radiographic view. The magnitude of lateral displacement of the femur is concealed by this view, not only because of resultant forces on the joint capsule, but there appears to be a hydrostatic effect also. The hip-extended view lowers the pressure within the joint capsule, which causes it to invaginate. A sort of vacuum or "suction" effect occurs that when combined with the fixed synovial fluid volume limits the sideways movement of the femoral head.

## **Developing better diagnostic methods**

Using this information, the University of Pennsylvania researchers were able to design a radiographic protocol based on quantitative parameters.<sup>12</sup> The distraction index or DI is based on a compression radiographic view that determines where the center of the femoral head and the center of the acetabulum coincide. The distraction view then measures how far the femoral head can be moved away from the center. This view requires the use of a special device called a distractor. The proper positioning of a distractor and the amount of force is crucial. Clinicians wishing to become certified in the PennHIP method are required to attend a one-day training session. Prior to certification, in order to ensure consistency and repeatability they are also required to submit radiographs that demonstrate their proficiency to Dr. Gail Smith and his colleagues. This certification process is designed to enhance quality control and protects the all-important integrity of the PennHIP data base. Once the two views are taken, it is possible to derive a unitless variable by dividing the amount of sideways displacement from the center by the radius of the femoral head.

This variable or distraction index ranges from 0 to 1 and a later study indicated that dogs with a DI of 0.3 or lower were truly negative for CHD. Those animals with a DI of 0.7 or greater were associated with a high probability for developing dysplastic joints. A variety of statistical methods, including those that evaluate qualitative parameters, were used to evaluate their data.

The DI range between 0.3 and 0.7 is still a gray area and is most dependent on specific breed variability. In a recent publication the DI was shown to be the only statistically significant predictor of the risk of developing degenerative joint disease in Rottweilers.<sup>13</sup> When German Shepherd Dogs

were included, the results indicated they had a greater susceptibility to the disease. It is clear that further research must focus on elucidating the specific breed differences when correlating passive joint laxity and susceptibility to degenerative joint disease. As more dogs are added to the data base, it will be easier to quantify the specific DI range for each breed that indicates the disease-free phenotype. It is for this reason that every radiograph taken by a PennHIP-certified veterinarian will be submitted to PennHIP for evaluation. Breeders will not have a choice of whether to submit the radiographs or not, as is the case with veterinarians taking preliminary radiographs prior to submitting the case to OFA for interpretation and scoring. Not having this choice will make some breeders uncomfortable, but responsible breeders will be pleased to know they have contributed to the betterment of their breeds. Breeders can expect that some of their dogs that have "passed" OFA certification will not be deemed suitable for breeding using the PennHIP method.

The question needs to be answered whether it is less deleterious to breed to a dog that is genotypically positive for canine hip dysplasia than it is to lose the opportunity to breed an animal because it was a "false-positive" for canine hip dysplasia. At first such a question sounds a bit philosophical, but in practice where it hits the breeder, it has an operational answer. There will always be other dogs, other champions to be made and other suitable brood bitches and studs that can produce fine litters. It makes no sense whatsoever to risk doubling up on defective genes whether for hip dysplasia or any other known genetically transmittable disease. Once you introduce undesirable genes into your pedigree, you will have great difficulty getting them out-and it may take several human lifetimes to do so.

As we have seen previously the honor system in registries does not work. In fact it works so poorly in the AKC's registration of puppy mill animals that the Canadian government will not allow importation of AKC-registered animals if the claim is made that they are purebred. That is called fraud. It works so poorly that the U.S. Department of Agriculture found in 1992 that 70 percent of the licensed commercial dog breeders inspected did not track pedigrees accurately.<sup>14</sup> It works so poorly that in 1987 Mark Hyland, an AKC attorney, represented to a federal judge in Kansas City that the AKC does not revoke fraudulent dog registrations because of the "infinite back up" of such registrations.<sup>15</sup> How bad is the AKC situation?

## Identification methods

No one outside of AKC really knows how bad the pedigree situation is, but Alan Stern a former AKC vice president, is on record with a 1990 statement to the *Sacramento Bee* that fraud happens on half of AKC's registrations.<sup>16</sup> Other registries have a similar problem with dishonesty as do Greyhound and thoroughbred racetracks. What is needed is a foolproof method for identifying a particular animal. While several identification systems are available, the Destron-Fearing microchip, now distributed by Schering-Plough, and the Avid microchip are the two contenders for the market.

Much ado has been made about the AKC wanting action on genetic problems, but until the simple matter of pedigree is cleaned up, do not look to the AKC to solve genetic problems. In author Cargill's breed, Akitas, it has only been in the past few years that AKC has allowed the breeding to Akitas imported from Japan because three separate breed registries were there. No great intellect is

required to ascertain that the gene pool was artificially restricted by the AKC and that many genetic problems experienced now and that will surface in phenotype in the future will have resulted from a restricted gene pool.

Computer chip "passive responders" have been injected in dogs, cats, birds, horses fish, reptiles and exotic and endangered species since 1991. More than 2 million identification chips have been sold. These rice-size chips are injected without requiring anesthesia. They consist of a coil and a small circuit board with a one time programmable memory. The data programmed into the Avid chip's memory is encrypted, and thus not susceptible to tampering. A reader is a transceiver that transmits a radio frequency pulse (125KHz), which energizes the coil in the implanted chip, enabling it to transmit a message back to the reader.<sup>17</sup>

Although the implanted chips can be detected by X-ray, they have proven to be extremely difficult to remove, other than through advanced surgical techniques. There is one report that a staff of veterinarians were able to remove an injected chip in a horse using dual plane radiographic surgical techniques; however such imaging equipment is well beyond the reach of all but the most well-equipped veterinary centers. None of this wonderful technology has potential if costs are high, but they are not. A survey of veterinarians indicates that injection price (including the chip) is \$25 to \$50. Readers are available to veterinarians for less than \$300. "We have the technology."

The next step in the battle against CHD is to marry up PennHIP, OFA and other evaluations with an "open" genetic registry such as the one maintained by the Institute for Genetic Disease Control in Animals (GDC).<sup>18</sup> Unfortunately, OFA's registry is closed to outsiders, and does not require the submission of X-rays and pedigree data of all animals radiographed. PennHIP is also a closed registry, but does require submission of the cases of all animals radiographed. The authors feel so strongly about the requirement to collect and make available the phenotypical data on parents, siblings, progeny and other progeny of parents and siblings in a cross-referenced data base that they challenge both OFA and PennHIP to make their data available to some central genetic registry. The only one available and capable at present is the GDC.

**Conclusions:** The two major methods of diagnosing canine hip dysplasia available to the fancy in the United States are those followed by OFA and those followed by PennHIP. Both are diagnostic; however, the hip-extended protocol followed by OFA may produce false-negative results. The protocol followed by PennHIP has a prognostic or predictive capacity through the use of statistics and a carefully guarded data base that allows a prediction to be made with respect to the probability of phenotypic expression of canine hip dysplasia. No one has a clear quantification of the gray area between obviously clear and obviously dysplastic hips. Controversy still rages. Until there are open genetic registries, mandatory evaluation of all dogs registered and some assurance of pedigree validity, canine hip dysplasia will remain a common affliction of the domestic dog, especially of purebred dogs.

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