Canine Hip Dysplasia

by Tom Phillips, D.V.M., MS, PhD.

Associate Dean, Research Professor Virology College of Veterinary Medicine Western University of Health Sciences Pomona, CA 91766 Phone: 909-469-5551 Fax: 909-469-5635 tphillips@westernu.edu

Canine Hip Dysplasia (CHD) is a complicated and often misunderstood disease. The purpose of this article is to provide a basic understanding of Canine Hip Dysplasia (CHD), particularly concentrating on its causes. Hopefully, this information will be helpful in reducing the incidence of this disease in the Briard.

The term dysplasia means "improper growth". Thus, Canine Hip Dysplasia simply means improper growth or development of the canine hip joint. This improper growth is usually characterized by lax or loose hips, which allows excessive movement in the hip joint which in turn leads to arthritis and lameness. Hip dysplasia is common in many of the large breeds of dogs. In the Briard, it is the most common cause of osteoarthritis. CHD is not an "all-or-nothing" disease, but is a syndrome that can present in a variety of clinical forms that range from clinically undetectable to a severe crippling disease. Dogs that are afflicted with CHD can have problems with going up or down stairs, difficulty in rising, a bunny-hopping gait when walking or running, early morning stiffness, personality changes from the pain, a wobbly gait, or lameness after exercise. Dogs often will carry more of their weight on their front legs as a result of the pain in the hips, resulting in the front end of these dogs being better developed than the rear. Dysplastic dogs are at a greater risk of incurring injury by normal activity such as jumping or rough housing. Thus, CHD can present as a spectrum of clinical diseases. It is possible for a dog to have CHD yet have no signs of the disease or for the disease to become so severe that the dog can not use it rear legs.

The clinical signs listed above are not diagnostic as they may also be caused by other conditions. The only way to correctly diagnose CHD is to have a radiographic (X-ray) examination performed by your veterinarian. The classic way of determining if a dog has CHD is to have the animal radiographed with the hips extended outward. In other words, the dog lying on its back with its legs extended straight

back. This position is required for the Orthopedic Foundation for Animals (OFA) to correctly evaluate the dog's hips. This is often an uncomfortable position for the dog and sometimes an anesthetic or tranguilizer is required for the animal to remain in this position long enough for the radiograph to be taken. Some veterinarians believe that placing the dog under anesthesia gives a better evaluation of the hip, as the muscles around the hip joint are relaxed, while other veterinarians do not believe that the anesthetic enhances the evaluation process. OFA will evaluate hips regardless of whether an anesthetic was used or not. OFA is a non-profit organization that consists of several veterinarians specially trained to evaluate the canine hip for CHD. The OFA is the oldest and best known registry in the United States. It grades hips, as severe, moderate, fair, good, and excellent. The OFA certifies dogs as acceptable for breeding if they are over two years old and receive a passing grade (fair, good, or excellent).

PennHip is a new organization for evaluating dogs for CHD. Their technique requires that the dog be radiographically examined with the hips flexed (frog like position). They also require that the animal must be anesthetized during the procedure. One of the advantages of PennHip evaluation over OFA is that animals as young as 16 weeks can be evaluated. Currently, there is no prevailing evidence as to which system is better. There are only a few veterinarians that advocate the PennHip system, but it does appear to be gaining in popularity.

As with people, dogs appear to differ in their ability to adapt to a disease. Some dogs may have severe radiographic evidence of the disease and show little in the way of clinical signs, while others may only have mild radiographic changes yet show severe clinical disease. This may be due to differences in the ability of individual dogs to adapt to the presence of chronic pain. If a dog develops CHD it is not necessarily a death sentence as most cases, with the exception of the severest forms, symptoms can be relieved by the use of pain medication. However, even in its mildest forms this disease may limit the canine athlete from attaining his full potential.

Both heredity and environmental factors are important in the development of CHD. CHD has an estimated inheritability that ranges from 0.2 to 0.6, with 0.0 being non-inherited condition and 1.0 meaning that a condition is completely under genetic control. Thus, demonstrating that CHD is a condition where both the environment and the genetics of the dog play a role in the development of this disease.

The interaction between the dog's genetic makeup and environment determine whether an individual dog will develop CHD. The genetics of the dog, to a large part, determine if a given dog has the potential to allow the environmental factors to act in such a way that CHD is produced. However, even dogs that are not genetically predisposed to develop CHD can contract the disease if they are pushed too hard when young by hyper-nutrition and excessive exercise.

During puppy hood, diets that are low in protein combined with low activity levels markedly reduced the severity of CHD in animals that were genetically predisposed to developing this condition. However, even dogs that are not at a genetic risk of developing CHD, if exercised too strongly early in life and/or are fed diets that are too high in calories and protein can develop CHD, since hyper-nutrition and excessive exercise may interfere with proper joint growth and development. Thus, it is best to prevent a puppy from jumping or undergoing sustained exercise until at least a year old. Also, the premium dog foods that are so often recommended may also contribute to CHD development by increasing the growth rate. A protein percentage of 22% (dry food) is generally all that is required for normal growth. However, we all want our dogs to reach their full potential, so premium dog foods are often fed. Here in lies the problem - consider this information as food for thought (sorry).

In addition to the environmental influences that confound our understanding of CHD, the genetics of this condition are very complicated and not completely understood. CHD is inherited as a polygenetic trait. In other words, many genes are involved. This makes sense when we consider that that the hip joint is composed not only of bone but also of muscles, tendons, ligaments, and a joint capsule which together hold the bones in the proper position for a normally functioning hip. Coordinating all of these parts into a normal functioning joint requires many different genes. But it is even more complicated, if we consider that this joint during the first year of life is continuing to change and adjust to the tremendous growth of the dog. If the parts of the joint grow at different rates or are out of synchrony with each other, then this will lead to a joint that is loose which predisposes the dog to develop CHD. Additionally, other seemingly unrelated factors such as rear leg angulation could also exacerbate or alleviate CHD. Dogs that have straight rear legs have less shock absorption, while well angulated dogs would not transmit as much of the movement stress to the hip joint, as more of the trauma is absorbed by the hock and knee. When all other factors are equal, the dog with well angulated rear legs is less likely to develop CHD than a

dog with straight rear legs. Thus, it is easy to see that the genetic control of CHD is complicated and difficult to understand.

Currently the reported incidence of CHD in Briards in the U.S.A. is about 17%. However, in reality the incidence may well be twice that percentage. The incidence of CHD is reported by the OFA, and represents only those radiographs (X-rays) that are submitted to OFA for evaluation. It has been demonstrated, in other breeds, that many of the radiographs that are taken for evaluation are never submitted to OFA. It is believed that this occurs because the radiographs of animals that have little or no chance of being certified are not sent to OFA for evaluation. Thus, there is a pre-selection by both the veterinarian that takes the radiographs and the dog's owner, so that only those animals with the best hips are evaluated. It is very likely that the above figure is an under estimate of the prevailing problem in the breed.

Selective breeding is the only way we can reduced the incidence of CHD in the Briard. However, knowing which animals to breed is not a simple question. Both environmental factors and genetics determine whether an individual dog will develop CHD. Genetics alone is not the only cause of CHD. Therefore we will never be able to completely eliminate CHD from the breed by selective breeding. However, we can realistically reduce the incidence of CHD through appropriately selecting the correct dogs to breed. Obviously a dysplastic dog should not be bred even if there is reason to think that environmental factors may have contributed to the animal developing CHD, such as trauma to the rear end when the dog was young. In such an animal, it is impossible to determine the exact role genetics played in CHD development, and it is far too easy to make excuses for breeding a dysplastic animal once one starts down this road. Thus, only breed animals that do not have CHD and all should be fine - right? Unfortunately this is not the case. The polygenetic control of the hip joint structure greatly complicates the situation. It is entirely possible for a dog with an OFA rating of excellent to produce puppies that develop CHD. How can this occur?

Without going too deeply into the genetics, some basic genetic background is needed to explain how an OFA certified animal can produce off-spring that develop CHD. The following is a gross over simplification of the genetics of the CHD; but does provide a framework for understanding a problem with multiple gene control over a single trait. Dogs have two copies of each gene that controls CHD (the total number of genes that control or contribute to the development of CHD is unknown; but it is believed that many are

involved). They get one gene from their mother and the other from their father. Some of the "good genes" can mask the presence of a "bad gene". In other words some of the "good genes" are dominant over the recessive "bad genes". Thus, the physical expression of the "good genes" will be result in good hips, even though the animal is carrying a number of hidden (recessive) "bad genes". When the sperm or egg is formed the two copies of each gene are separated so that sperm and the egg end up with only one copy of each gene. In a case where an animal had a copy of one good gene and one copy of the bad gene, 50% of the eggs or sperm would get the "good gene" and 50% will get the "bad gene". Now recall that many genes are involved in the development of CHD, so this separation occurs for each of the genes that control hip joint development. Thus, a very few of the eggs and sperm will get all of the "good genes" and a very few will get all of the "bad genes" but most will get a mixture of both "good" and "bad" genes that control CHD. The outcome of a particular mating will be determined by the match up between the genetic make -up of the parents. If the bad recessive genes line up incorrectly then more of the puppies could have hip dysplasia. However, if the good dominate genes line up well then many of the pups will have good hips, but may still carry the hidden bad genes. Complicating the situation even further is that some genes may be good when combined with one set of genes and bad when combined with different set of genes. It is also possible for some genes to have a greater or lesser influence on CHD than others, adding to the complexity of the problem. Thus, one can see that the genetics of CHD is **very complicated**.

Some puppies will have the same genetic combination as the parents. Some may have a more desirable genetic make-up than either parent; and others may have a less desirable genotype. Predictions of a specific CHD outcome from a particular mating is currently very difficult if not impossible.

However, it is OFA's opinion, that a dog with fair hips and less than 25% of its littermates exhibiting dysplasia is a better breeding prospect than a dog with excellent hips having more than 25% of its brothers and sisters affected by hip dysplasia. In other words, less of the bad genes are likely to be present where a smaller percentage of the littermates have CHD. Ideally, we would want to achieve a homogeneous gene pool composition where the dogs carry two copies of the good genes for each gene that controls CHD. In other words eliminate the bad hidden (recessive) genes from the pool. This could be achieved by careful line breeding, making good hips part of that line's type. However, to accomplish this it is important to look beyond

the individual animals of the proposed mating and consider the OFA status of the littermates as well as other relatives on both sides of the mating before proceeding. It is also important to point out that a good dog is more than just good hips. Care must be taken that the breeding of dogs is not based only on one trait. Otherwise we may end up with dogs that have excellent hips but have poor temperaments, bad coats, no herding instincts, or other genetically related health problems. The whole animal should be considered with hips status being important but not the only consideration

From this article it is easy to see why CHD is such a difficult problem to control. However, through careful breeding selections of OFA certified animals that have a low incidence of CHD in their littermates and in their pedigrees, we should be able to reduce, although not eliminate, CHD as a problem in the Briard.

This article first appeared in "Briard Tales", the newsletter of the Briard Club of California in 1998.