HIP DYSPLASIA AND THE CARDIGAN CORGI

The hip joints may be the most important joints of the dog’s skeleton. All the power that moves the dog forward is generated by the hind legs, and is transmitted to the rest of the body via the hip joints. Therefore, dysfunction of the hips is truly crippling.

The important issue is to decide whether available evidence indicates that hip dysplasia is sufficiently under genetic control within the breed that breeders can select stock to produce sounder hips.

WHAT IS HD?

Hip dysplasia (HD) simply stated means an "abnormal formation" of the hip joint. Think of the condition first as looseness in a joint that should be snug – then most of the problems attendant to hip dysplasia are a result of this "looseness". The normal anatomy of the hip joint is a classic ball and socket joint. The head of the femur (the ball) is supposed to fit smoothly and exactly into the acetabulum (the socket). If the head of the femur does not fit snugly, it will be slipping and bumping around somewhere in the neighborhood of the acetabulum, and this will damage the relatively delicate surfaces of the joint. A perfect joint has living layers of tissue on the surface of both bones; if that is damaged, there may be bone rubbing on bone, which is both destructive and painful.

The overt signs of HD vary from decreased exercise tolerance to severe crippling. They include: a reluctance or inability to go up or down stairs, difficulty in rising from a sitting or prone position, bunny-hopping gait when running, stiffness early in the morning that improves as the dog warms up, change in disposition due to pain, lameness after exercise, wobbly gait, a clicking sound when walking, and many others.

Research on hip dysplasia suggests that HD is a more complex disease than was first thought. There are five biological factors that contribute to HD, and there are often severe interactions between those factors.

[3] Exercise as the puppy is growing
[4] Floor surfaces the puppy is exposed to.
[6] To this list of biological causes, we should add a catch-all to include technical, policy, and technique difficulties, essentially artifacts of the evaluation systems.

There are no simple answers or solutions to the CHD problems. However, many aspects of the disease have been repeatedly and independently documented and are generally accepted by the scientific community. Five important ones are:

[i] Two physical features contribute to CHD, the structure of the bone in the joint, and the tightness or laxity of the ligaments which hold the joint together. The latter is less known and studied.

[ii] The genetic component of canine hip dysplasia is caused by the presence of many genes (polygenic), although recent studies provide weak support for the idea of a major gene which might control the whole range. While no environmental cause has
been demonstrated experimentally, many environmental factors contribute to HD’s expression in a particular dog (phenotype).

[iii] One other feature of selection in polygenic systems has never been examined for HD, but must not be ignored. Many genes have more than one function, so that when we select for a particular allele, we may be affecting systems that we do not know are related. Then, genes are on chromosomes which carry many genes. If we are severe with selection, as we wish we could be for HD, we may inadvertently eliminate some desirable genes as well, by discarding individuals for one undesirable trait on a particular chromosome. Good and bad genes for HD do not exist in isolation, and we must expect that other systems will be affected by selection for good hips.

[iv] The only current means for reducing the occurrence of HD at the breed level is by selectively breeding for normal hips. The problem with that approach is that the heritability of HD may be relatively low.

[v] Radiography is the accepted means for evaluating the status of the bone in the joint, and there are recent methods for measuring laxity of ligaments.

We like to think of genes as "blueprints", which they are. Also, the final result, in the case of hips, is a relatively hard structure. But the mechanical analogy breaks down when we consider that, as a puppy grows, the bones are not calcified, they are cartilage. Also, not all parts of a puppy grow at the same rate, in terms of time and size, and that relative growth can be affected by nutrition. That can mean, for example, that a puppy whose hips would be fine if he grew up on a wolf diet of raw prey, might, if on another diet, have his bones growing faster than his muscles and ligaments in the 3 - 8 month-old period when growth is most rapid. At this point it is possible that hips that are genetically OK could be seriously damaged if growth and exercise get out of whack with each other. When the transition was made from feeding dogs home made rations, to commercial pet foods, the first dog foods were developed by livestock feed manufacturers. In livestock, young animals are fed so as to grow as fast as possible, to reach market size as young as possible. By 1990 it had become evident that this approach was not appropriate for dogs, especially for larger breeds. Therefore, modern puppy foods are not as rich, and differ from adult rations mostly in vitamins and minerals. So, the bad effects of puppy food on hips may be less now (2007) than they were decades ago.

Add to that too much or too little exercise during this critical period, and you easily see that further damage could occur, through changes in muscle mass and muscle tone. That is called interaction by statisticians, where the combined effect of two factors is either greater or less than the sum of the effects of the two factors studied separately. Add to the mixture the concept that raising dysplasia-prone breeds on surfaces such as linoleum, which become slippery when wet, and you observe that a puppy which is growing too fast, and stressed by too much exercise, may have its legs slip out sideways, thereby stretching the ligaments. So there is large potential for disruption of the normal growth of healthy hips by a variety of environmental factors, and the potential for destructive interaction is significant.

Policies and policy differences between the two major systems affect the estimates of heritability of the results. You may have your dog X-rayed for submission to OFA in the USA, but the comment is equally valid for other national hip registries), but, between you and your veterinarian, you may decide not to submit that dog. That should mean that OFA estimates of the prevalence of good hips in a breed will be
overestimated, if obviously bad hips are not submitted. PennHip requires that every
dog examined by their procedure must be submitted to the database. However, while
that may be a better approach, there is no requirement that all individuals in a litter
be examined. The latter requirement would put estimates of heritability on a sounder
basis. The onus here rests with breeders and fanciers of a breed. National veterinary
registers, and OFA, provide well-honed techniques for assessing hips, but unless all
members of a breed or breeding line are submitted, progress in elimination of hip
dysplasia will be slow.

Statistically, it is true that the PennHip Distraction Index has a higher heritability
than OFA ratings. But even that heritability is relatively low. Another advantage of
PennHip is that their lead-up studies indicated that the technique produces reliable
measures even at less than six months of age. Unfortunately, several veterinarians
familiar with PennHip say that there are problems in applying the technique to
dwarf-legged dogs.

Examination of radiographs of hips has a considerable subjective component, so hip
scores may vary depending on who reads the X-rays. Most of the national hip rating
schemes recognize and react to that, by requiring that more than one expert read the
pictures.

Several veterinarians have suggested that owners may mask some of the heritability
of CHD by taking extra care with puppies. For example, there are some who carry
puppies up and down stairs for the first year of their lives, to help prevent them from
becoming dysplastic. In doing so, they may mask genetic dysplasia by trying to keep
the puppies „normal“.

There are at least two ways of thinking about hip dysplasia. One is that it is largely a
genetic condition. If that were true we should be able to improve dog breeds by
careful selection. The consensus is that we have made some improvement, but not
nearly as much as we want to. The second way is to admit that the growth process is
dynamic, and the structures are relatively fragile during some periods of growth. In
that picture, dysplasia is, partly at least, the result of events during growth. Call them
accidents, call them environmental, call them what you will, they are not solely the
results of genetics. Strictly speaking, they are probably what a statistician would call
the result of interactions between genetic potential and environmental factors. But,
these environmental influences can make the dysplasia many times worse than it
might have been under more favorable circumstances. If you admit to that, then
saying that the environmental factors "caused" hip dysplasia is not entirely
exaggeration. Damage to a delicately growing joint can be from a variety of
environmental sources. On the other side of the fence, if the individual had grown up
on the right regime of food, floors and exercise, it would never have shown
dysplasia.

What do we know about hip dysplasia in the wild ancestors of dogs? There do not
seem to have been any studies of the incidence of dysplasia among wolves, coyotes
and jackals.

The most recent DNA studies suggest that all modern dogs come from a single wild
ancestor, the wolf species that still exists in northern Iran and Iraq. Dogs diverged
from that stock some 30,000 years ago. The wolf stock itself has been around for an
estimated 7,000,000 years, and must have been subject to a lot of natural selection.
And from that basic wolf stock came Chihuahuas, Irish Wolfhounds, etc. and corgis.
So we have taken a single wild blueprint, and manipulated it into a huge variety of
different forms. And, we have done that in a very short time, that is, roughly the last
200 years. If you strip away the bunk and fantasy of most breed histories, you can't
find much solid evidence that many breeds actually existed before 1850. And for
those that did, many have changed dramatically since then. So what are the
implications of such rapid changes in size and relative weight for the hip joints?

The generalization is that small breeds rarely suffer from dysplasia, giant and large
breeds almost all have dysplasia, and in between you have to look breed by breed. In
that middle range the rule of thumb is that breeds that are heavier built are more
likely to suffer from dysplasia. This is item [5] on the list above, and it represents an
important contributor to hip dysplasia which cannot be affected by breeder selection.
That means, that if we want to maintain the characteristics of a particular breed as
declared by the standard, we cannot change the 'damage' to the hip joints when we
made the transition from the ancestral wolf to the modern breed. Put another way,
strict selection for hip health alone would result in all dogs having the general size
and proportions of the ancestral wolf.

That may be an indicator of why it has been so difficult to get rid of dysplasia.
Breeds which have muscle to skeleton relationships similar to the small wolves of
Iran are relatively free of CHD, so the greyhound is the 'perfect' breed as far as hips
are concerned. The further you deviate in basic design, the more likely it is that your
breed will be subject to dysplasia. That observation leads to the prediction that
progress in reducing the incidence of serious dysplasia will not be the same in
every breed. Many breeds have deviated too far from the basic design. Small breeds
have gone a beneficial way, while large breeds have traveled a dangerous path.

The concept of heritability comes from population genetics. In all large populations,
any particular characteristic shows variation. The population geneticist studies
individuals linked together by their pedigrees, and estimates by careful statistical
analysis, the proportion of all that variation which appears to be under genetic
control. That can be done without knowing which, or how many, genes affect the
character, as long as there are reliable and repeatable means for measuring the trait.

**IS HD A PROBLEM IN CARDIGAN CORGIS?**

Across breeds in general, some individuals suffer from bad hips, but there are trends.
Smaller breeds are less subject to hip dysplasia. Among larger breeds, those which
are light in weight in relation to their skeleton are less prone to hip dysplasia than
breeds which are relatively heavier.

That brings us to Cardigans. They are on the small end of middle sized, but their
weight seems to have increased by over 40% in the past 40 years or so. They are
heavily muscled, but on a sturdy skeleton. But then, they have dwarf limbs, and no
one seems to know the implications of dwarf legs on dysplasia. They have a broader
muscle mass about their hips than most breeds, and that may help stabilize the joints.
Some veterinarians have stated that they are not sure how to X-ray dwarf-limbed
breeds, because the muscles hold the joint differently than in longer-legged breeds.
PennHip specialists have expressed similar reservations. That makes it uncertain
whether current methods of studying hip dysplasia actually apply to dwarf-legged breeds. Much more study specific to dwarf-legged breeds is required before we can be sure that current X-ray techniques are fully applicable to Cardigans.

Cardigans in North America are much more involved in activity sports than they were 30 years ago. Is that showing up deficiencies in hip structure that would not have shown up in the kennel dogs and couch potatoes of 1950-1980?

Generally speaking, serious cases of hip dysplasia seem to be uncommon (less than 1%) in Cardigans. It has often been claimed that dogs that can lie flat with their hind legs stretched out behind them do not have HD. That, however, is no proof. Photos of one young dog show that he can do this. He does not express any clinical signs of having HD, yet when tested his Pre-lim was MILD and the X-ray showed hardly any socket. This dog lives on a farm where he exercises all day and his tight muscles enable him to keep his legs from falling out of his hips. He may yet prove to show signs later in life when he slows down and isn't so active and physically fit. His litter brother, on the other hand, could not walk at nine months. In another example, a Cardigan had his hip joint removed due to damage after being hit by a car. He soon learned to walk, and run, and jump, and to be a normal Cardigan, except for a slight limp.

As mentioned above, there are six classes of phenomena which contribute to the development of hip dysplasia. The logical point here is that few Cardigans have crippling hip dysplasia, and with six basic 'contributing causes', only one of which is genetics within the breed, the chances are that, even with large numbers in a formal experiment, it would be next to impossible to estimate the extent of the contribution of genes to the problem. The fact that decades of breeding only X-ray cleared stock has not, in breeds like German Shepherd Dogs, reduced the incidence of hip dysplasia by very much indicates that the heritability of CHD may be quite low.

OFA CONTRA PENNHIP

In North America, there are two different methods available for assessment of hips, OFA (Orthopedic Foundation for Animals) - http://www.offa.org/hipinfo.html and PennHip - http://www.pennhip.org/

The two methods are trying to measure different things. OFA asks the question "Does this animal have hip dysplasia?" In other words, OFA cares about the anatomy as it stands and measures laxity, depth of socket and shape of ball and socket.

PennHip asks "Will this animal develop degenerative joint disease?" What hurts dogs with bad hips? Arthritis. So can we find a method that predicts whether the dog will develop clinical arthritis in its lifetime? And they found a very good correlation between laxity of the ligaments and eventual degenerative joint disease. Therefore PennHip also measures the tightness of the ligament.

The OFA system of examining X-rays was established a long time ago, based on the best scientific principles of the day. Similar systems are available all over the world. (Note that the systems may be looking at the same features, but their rules and
procedures differ enough that the results are not always comparable. Note also that OFA is the oldest system, and the more recent systems have tried to improve on its methods.) But, OFA results, in terms of reducing the prevalence of hip dysplasia, have been disappointing. That suggests that environmental variation is more important than genetics. The PennHip system was born out of that frustration with OFA results. It should be better, its formal results are better, but the jury is still out with respect to Cardigan corgis, because not enough of our breed have been examined and entered into the database. We should note that part of PennHip is an OFA style examination of the X-rays. We also note that it is much more expensive, per dog. Has that influenced the small size of the Cardigan database? Has it influenced our thinking about the breeding value of the PennHip system?

There is one important policy difference between OFA and PennHip. Owners and their vets can look at X-rays taken for OFA, and decide whether to submit them. That may weed out some obviously bad hips before they get to OFA. With PennHip, the owner must sign a form before the dog is examined, and one requirement is that the test results will be submitted for every dog tested.

One feature of the whole hip dysplasia picture which is not covered by OFA is the range of changes which can occur during puppyhood. A major reason for OFA not rating hips of dogs under 18 months is that such changes can be observed!! OFA says that the joint has not matured before 18 months. In the research that led up to the PennHip system, testing at even 4 – 6 months of age proved to be an accurate predictor of results later in life. The same is obviously not true for OFA.

There is an additional relevant subject. OFA and comparable organizations have defined the shape of a good hip joint. But, we see many dogs that don't have ideal structure, yet live long, pain-free lives. PennHip added the concept that you need good tight ligaments. It would appear that tight ligaments may hold an 'iffy' structured joint together such that the dog will not damage the joint. Are there any exercises to make ligaments tighter? In Cardigans, it is even possible that the very heavy muscling around the hips joint helps to stabilize the joint, and reduce CHD.

There is no test for a "good hip" gene. OFA results (such as they are) can show us which dogs are a better bet for sound hips than others, but it's very subjective; how a dog's hip status translates to hips in the offspring is not clear. Neither is the relationship between OFA hip reading and functional ability in the adult dog except where results show gross dysplasia.

There may be an important difference between dysplasia as seen on X-rays, and functional dysplasia, which hurts the dog. In other words, many dogs with mediocre hips from X-rays, live long, happy and pain-free lives. For example, Norwegian elkhounds tend to have relatively shallow hip sockets, so many do not pass OFA. But that does not reduce their performance in the field. Perhaps the socket depth is compensated for by tighter ligaments? That is not yet known. Dogs with poorly formed hip sockets may suffer a lot, and don't move well. So clinical dysplasia is very real. Some X-ray dysplasia does not hurt enough to slow the dog down. Again, this will be affected by the overall size and bulk of the breed, compounded by whether the individual is heavier than he/she should be. Many dogs live comfortably with hips that are not the best. However, such a dog competing in competitive agility weekend after weekend may incur damage that a couch potato would avoid. Surely
damage from exercise is more likely when the hips do not have ideal structure. Slippery floors under 6-16 week old puppies cause the dogs to stretch the ligaments. Once the joint is loosened, it can pound the articular surfaces during heavy exercise, and damage will result that would not have happened if the ligaments were tight. In the 1970s and 80s, puppy foods were formulated to maximize growth, in the manner of livestock rations. That resulted in dogs that grew so fast their skeletons outgrew the ability of muscles and tendons to hold the hip joints together, and the prevalence of CHD was high in heavier breeds such as Labradors. Dog food manufacturers have noted this, and modern puppy foods do not spur very rapid growth.

The hips are, in computer speak, a whole system. They are not just a joint subject to damage. You need a feeling for all the forces which help or hinder development and good function of the hip joint. You need to remember that almost all the force which moves the dog forward, at whatever speed, is transmitted through the hip joint. Especially, you need to think about whether these factors might interact. So, raise a Labrador puppy on a wet linoleum floor, feed it lots of old style puppy food, and have it do 100 retrieves a day on an asphalt playground, what is the forecast for its hips? Would a Corgi raised on grass and carpet, with moderate play, fare better?

We have to further consider that HD involves a *system*, made up of bone, ligament, cartilage, and muscle as well as their very complex interactions as they grow, mature, and later deteriorate. Students of systems theory refer to this as a "tightly coupled" system in that a problem with one aspect of it can affect the performance of the system as a whole. Because the system is tightly coupled, problems with different aspects of the system can cause similar-appearing failures. In other words, the system can fail in a similar way, i.e. lameness can occur from HD, due to problems with any of its components: bone, ligament, cartilage, or muscle. No one would argue that one or even a few genes control all the aspects of this system. And that is one good reason why we see the variance numbers: the problem is complex and multi-factorial. Failures are only partially explained by the factors that OFA screening is capable of identifying. The rest of the variance has to be explained in other ways.

An OFA screening (even the most objective), if it only predicts 20-40% of variance, should **not** be taken as completely predictive of hip dysplasia. In the very **best** of all cases, 60% of the variance is explained by other factors. OFA results must be considered in light of family history, environmental factors, nutrition, and importantly, the structural components of the system which OFA screening is not capable of assessing, like soft tissue. Just because OFA results *seem* to have precision, they should not be given more weight than they deserve. To do so commits a form of the "false precision" fallacy.

We also need to remember the Hardy-Weinberg Law of population genetics. It states that, in the absence of selection, the frequency of particular genes does not change from generation to generation. For CHD that means that failure to X-ray breeding stock will not cause the prevalence of CHD to rise, unless other factors are involved. Of course, if one hopes that X-raying, and breeding only from cleared stock will reduce CHD, then failure to X-ray will have a negative influence. However, in the real world, where heritability of CHD is apparently low, the difference in frequency of CHD in puppies of X-ray cleared stock, compared to non-X-rayed stock, may be disappointingly small.
Many older dogs, and older humans, become subject to painful arthritis, and the hip joints, due to their functional importance in dogs, are among the most frequently affected. Hip arthritis is painful and crippling to older dogs. However, it is wrong to assume that the only cause of arthritis of the elderly dog is hip dysplasia. We must also remember that, in the wild, very few wolves live to be more than 8 years old, so there was little to be lost to the wolf genome if the elderly slowed down, because those elderly were not here! Severe hip dysplasia can slow down a younger dog, but that is a much rarer event than a mediocre X-ray result.

PROBLEMS IN ASSESSING CARDIGAN HIPS

Now, where both methods are problematic, we must consider the Cardigan/hypochondroplastic hip. It will be shallower and looser than a non-dwarfed hip. If you showed a Cardigan film to an orthopedist and told him/her it was a Labrador the dog would promptly fail on either method. The mean distraction index in the corgi breed group is around 0.6; that would be a horrifying score to a breeder of tighter-hipped longer-legged dogs. In fact, if you got a 0.6 on a Lab, you'd be told to expect joint disease in the future. So how can we have these loose-hipped shallow-socketed little dogs that somehow seem to keep going for well into their second decade?

One the other side of this picture, some Europeans report that Cardigans with the highest possible hip scores have restricted, somewhat stilted movement. Is it possible that the very quick changes of direction that are characteristic of Cardigans moving cattle require a more flexible hip joint? That might have a shallower socket, as a majority of the breed seems to show.

Unfortunately, OFA will not discuss the issue of testing dwarf breeds. One vet was on the OFA evaluators list for years, that was until he started voicing his opinion that you can't evaluate a dwarf breed the same way you do a normal growth breed. He was asked to retire from the evaluation list by OFA. He also quit PennHip X-raying when two healthy and happy show Shi Tzus came in for PennHip evaluations. He was certified, and had performed several films for PennHip prior to these two dogs. Both dogs went home sore and never recovered from their ordeal. It was later found both dogs had failed severely with OFA on their hips. PennHip claimed their method was not at fault as any dog with severely dysplastic hips most likely will suffer damage when the technique required for proper PennHip evaluation is done.

However, the biggest problem with either method is that it is totally based on a human OPINION. Vets are human and they tend to prefer A over B. If your Cardigan just happens to get an evaluator (be it for OFA, or PennHip) who FEELS what they are viewing is dysplastic, or indicating joint problems your dog gets a VOTE that it is indeed a client for developing dysplastic conditions.

SHOULD CARDIGAN CORGIS BE X-RAYED FOR HD?

While the basic science is sound, the results do not live up to the promise and despite the science, X-raying has not made dysplasia go away.
First of all, we need to decide whether Cardigans are large dogs or small dogs. And we need all the help we can get to decide whether medium sized dwarf-legged dogs are subject to special dysplasia rules because of their dwarfness. X-rays only scratch the surface of the subject. The breed has seen some remarkable changes in the 80 years since the first Cardigans were “rescued” from Welsh farms. Until 1967, the Cardigan standard called for dog to weigh 18-26 pounds, bitches 15-22 pounds. In 2007, the average Cardigan weighed almost twice that. The reasoning presented above suggests that such a large increase in size might affect the incidence of hip dysplasia. Fortunately, it still seems to be rare, but there are no data from the 1930s to allow us to estimate whether the incidence is increasing. Vigilance is justified.

We need a lot more information about whether CHD causes problems in Cardigans. With increasing emphasis on activity sports, will the frequency of real hip problems rise? How many older Cardigans show pain and slowness that probably relates to CHD? So far it appears that Cardigan Corgis are only rarely troubled by CHD, but the size of the breed, the nature of puppy foods, rearing conditions and other factors are all dynamic. The problem may not be serious this year, but may worsen next year.

We also need to inform ourselves about the relative importance of feeding, floors, and exercise. All three of these environmental factors have been shown to either cause or aggravate hip dysplasia. These are what a geneticist calls environmental, or non-genetic influences. They make analysis of the genetic side more difficult, because we do not have standard food, exercise and flooring regimes in puppies, right across any of the breeds mentioned. Dog owners' variable habits and habitats make an organized multivariate statistician tear his/her hair!

Tests are made to provide information. The honest sharing of the information is what is important. That gives others the opportunity to make good-faith-based decisions when breeding, buying a puppy and providing honest information to those that end up with most of the puppies produced, namely the pet owners.

It is clear from various statistics that carefully breeding for good hips can indeed lower the incidence of CHD in a breed, and may also help raise the average hip score. BUT it does not seem to indicate that CHD can be eradicated, or even reduced beyond some limit. This is NOT PRA where one gene is identified, and by avoiding all PRA affected dogs and all PRA carriers (now identified by test), it would actually be theoretically possible to eradicate PRA in one generation [if everyone tested and everyone avoided the gene]. HD is not like that. It is clearly polygenetic + strongly affected by environment factors as well as nurturance issues. PRA is not! It seems to be purely a genetic issue -- you either have the gene or you don't! And therefore, you either have PRA (homozygous for the gene), or you are a carrier (heterozygous) or you are clear (no copies of the gene).

None of us can guarantee a dog free of genetic disorders but we can guarantee that we have done the best that we can do to provide healthy dogs. It is our responsibility as breeders to do the best we can do with the tools we have. We are dealing with human lives and canine lives, not just stocks and bonds.

Every responsible breeder in this breed should therefore applaud the advent of good, reliable testing, and the improved knowledge that this brings to the very difficult
breeding decisions we face as we work to advance this breed. However, the decision to use an expensive test must be tempered by the evidence for heritability of the trait. If testing will only improve the situation by a maximum of 10%, is it worth the cost?

Unfortunately, hip dysplasia was one of the earliest conditions for which a specific test was developed. At the time the original X-ray techniques were developed, it was convenient to assume that this structural problem must be entirely controlled by genetics. It has been downhill since then, as environmental factors became better understood. But, one unfortunate result has been a mantra that, to be a responsible breeder, you simply must have hips evaluated. It is further implied that, if you do not X-ray, you will contribute to the rapid decline of hip quality in the breed. Hopefully the evidence presented above will tell you that both statements must be evaluated carefully, because neither is fully true, nor fully false. In the end, each breeder must make their own decision, based on careful study of the evidence.

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Further information will follow when available.