

# Canine Hip Dysplasia Part II

## Causative Factors of Canine Hip Dysplasia

*Owners must separate fact from myth when examining theories on genetic, nutritional and environmental factors that influence CHD. By John C. Cargill, MA MBA, MS and Susan Thorpe-Vargas, MS*

This is the second part in a series on canine hip dysplasia. What follows is written from the perspective that the readers of the series are 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 medical texts, it is a relatively in-depth look at the whole problem of a canine hip dysplasia. Furthermore, the series is designed to be retained as a reference. When you finish reading it 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 either is 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.

In this article we will address the issue of genetic, nutritional and environmental factors. We hope to debunk some of the myths and introduce some recently developed theories.

Other diseases, infections or trauma can produce clinical signs suggestive of canine hip dysplasia. In some breeds the animals learn to live with pain and are stoic about letting anyone know of their pain. This stoicism seems to be especially prevalent in terriers and northern breeds and is the case - not the exception - in the fighting breeds. Those fanciers who participate in pulling, freighting, carting or sledding events with their dogs should always be aware that tendonitis or pulled muscles can cause a gait change reminiscent of hip dysplasia. Anyone involved in lure chasing or coursing for real needs to understand that on occasion, an animal will twist or turn the wrong way while in full chase. In the older dog, trauma from younger years may manifest itself as arthritic deterioration. A little bit more unusual is to have viral penetration of the joint capsule with resultant damage to articular cartilage, or the epiphyseal surfaces of the femur. Absent such unusual occurrences, the reality of hip dysplasia is that it is a genetically linked condition--always was, always will be.

## The role of growth

In the first article we said that the first six months of a puppy's life seem to be a critical time of development. The rate of growth can be astonishing. When one thinks of the number of things that could go wrong as an Akita puppy, for instance, goes from a birth weight of slightly more

than 1 pound to 60 to 70 pounds in six months and then adds another 30 to 40 pounds by year end, it is amazing that most dogs mature without serious problems. It is during this period that dogs are most active. There is evidence to suggest that exercise is necessary to retain the depth of the acetabulum. How much exercise and of what type is unknown.

One Norwegian anecdotal study published in England in 1991 concluded that German Wirehaired Pointer, English Setter, Irish Setter, Gordon Setter and Labrador Retriever puppies growing up during the spring and summer had a lower incidence rate of hip dysplasia than puppies growing up during autumn and winter. Oddly enough, Golden Retrievers and German Shepherd Dogs did not manifest the same seasonal pattern of incidence of hip dysplasia. <sup>1</sup>

While this study may lack strict experimental protocol, it raises many questions. The first question is whether there was an exercise differential between the dogs due to weather in Norway. The second question was whether there was different availability of sunlight necessary for vitamin D production and utilization. The list of questions could go on, but this study is brought up to show that there may be exercise and diet factors at play, and that various breeds may respond to these factors in different ways. It would be reasonable to conclude that there is probably an amount of exercise during a genotypically dysplastic puppy's rapid growth period where phenotypic expression is mitigated, delayed, or both. Without taking the time, cost and effort to conduct a rigorous scientific study, it is still sometimes possible to glean valuable information from existing, i.e., available data. Therefore, do not shy away from creating working hypotheses from anecdotal studies; conversely, do not lock their findings in concrete as inviolate fact.

With respect to the published scientific literature, we found nothing in Medline (an online listing of medical and biological articles) referencing any journal article addressing the subject of surfaces and their effects on the incidence of hip dysplasia. While we know of breeders who write into their sales contracts that animals must be kept on a specific surface and fed a specific feed, these demands seem to be without scientific basis.

There is some evidence that preventing rapid growth reduces the extent to which the adult dog will manifest hip dysplasia. Decreasing the dog's food consumption during its growth period seems to correlate well with normal hips. <sup>2</sup>The Kealy study published in 1992 was based upon 48 8-week-old Labrador Retriever puppies. These puppies were sex-matched littermates randomly assigned to two groups: the first group was fed *ad libitum* (as much as they wanted, when they wanted to eat); the second group was fed the same feed until they were 2 years old, but in amounts of only 75 percent of what the first group consumed *ad libitum*. Thus for every puppy fed *ad libitum*, there was a same-sex littermate on a restricted diet. This rigid protocol gives this study great respectability and credence. The accompanying chart gives the findings in tabular form. Note the tremendous increase in normal animals at two years of age when kept on a restricted diet for those two years. This ought to more than suggest that overweight animals are at risk for phenotypic expression of canine hip dysplasia.

Radiographic	Group 1	Group 2
--------------	---------	---------

Evaluation Method	Ad Libitum Feeding			75% of Ad Libitum Feeding		
	Dysplastic	Normal	% Normal	Dysplastic	Normal	% Normal
OFA	16	8	<b>33%</b>	7	17	<b>71%</b>
Swedish	18	6	<b>25%</b>	5	19	<b>79%</b>

Many researchers conclude that early fusion may lead to bone and cartilage deviations which then could predispose the animal to future dysplasia. An important point that these studies illustrate is that it is possible to improve the individual phenotype of dogs whose parents carried the gene for hip dysplasia (genotypically dysplastic).

In the first article we alluded to joint laxity as being present whenever there is canine hip dysplasia. Given that joint laxity is at least one of the factors governing the onset of hip dysplasia, then any process that retards this condition could possibly minimize the severity of the disease. It also is conceivable that retardation of joint laxity could delay the onset of the physical appearance of the disease.

### Feed for health

A recent study (1993) showed that coxofemoral joint stability was improved in dogs that were fed increased levels of chloride and decreased levels of sodium and potassium.<sup>3</sup> In the eight-part "Feed That Dog!" series (*Dog World*, July 1993 through February 1994) we emphasized repeatedly the importance of the ratio of sodium and chlorine, with a ratio of 1.5 sodium to chlorine being accepted as the dietary requirement.<sup>4</sup> We noted also that "sodium chloride deficiency is manifested by fatigue, decreased utilization of protein, decreased water intake, inability to maintain water balance, retarded growth, dryness of skin and loss of hair."<sup>5</sup> "Potassium deficiency" results in poor growth, restlessness, muscular paralysis, a tendency toward dehydration, and lesions of the heart and kidney."<sup>6</sup> We cautioned that "prednisone, a steroid commonly prescribed for various skin allergies, causes a loss of potassium and retention of sodium, and retention of sodium can cause further loss of potassium."<sup>7</sup>

Calcium (Ca), sodium (Na), and potassium (K) are the electrolytes considered most important, as they are necessary to many biological functions. Electrolytes are atoms or molecules that carry either a negative or a positive charge. Anions have an extra electron, and thus carry a negative charge. Cations are missing an electron, thus they carry a positive charge. In the study cited, Kealy et. Al. Introduced the theory of "dietary anion gap" or DAG.<sup>8</sup> The researchers explained DAG as the amount of chloride ion subtracted from the sum of sodium ion and potassium ions:

$$\text{DAG} = [(\text{K}^+ + \text{Na}^+) - \text{Cl}^-]$$

This experiment, consisting of the raising of 167 puppies, included puppies from five different breeds. They were placed on three different diets that varied only in their DAG content.

Examples of low DAG ingredients are rice with a DAG of 6 and corn gluten meal with a DAG of 5. The result of this experiment showed that except for some breed-specific exceptions, those dogs that were fed a lower DAG diet had better hips at 30 weeks than those fed a diet with a higher DAG content. Differences in DAG balance did not result in different rates of weight gain. This is important, for it allowed elimination of weight gain as a causative factor in the study. Hips were evaluated by their degree of subluxation as measured by the Norberg angle. The Norberg angle is the "angle included between a line connecting the femoral head centers and a line from the femoral head center to the craniodorsal acetabular rim." <sup>9</sup>The greater the Norberg angle, the less the subluxation. Norberg angles are commonly measured as <90 DEGREES FOR LOOSE HIPS AND>105 degrees for tight hips. Those dogs with better hips at 30 weeks also had good hips at 2 years of age.

Unfortunately, the researchers were unable to explain the mechanism or the "why" of how they got the results they did. One of the theories proposed was that a lower DAG somehow affected the pH or "acidity" of the synovial fluid. This in turn affected the osmolality or "thickness" of the synovial fluid. The osmolality of a fluid depends upon the number of dissolved particles in it, and is the measure of the osmotic pressure. In previous studies, a higher osmolality was associated with the greater synovial fluid volume found in dysplastic dogs. Note, of course, that there is a normal range of DAG values in a balanced diet. Leaving that range while formulating a dog food, for example, could cause serious problems.

## Calcium

The question of calcium supplementation while controversial among breeders, is fairly easy to answer: don't do it. It is not necessary to add extra calcium to your dog's diet. Not only is calcium an essential skeletal component, it is also necessary for blood coagulation, hormonal release and muscle contraction. The three biological systems involved in controlling the amount of calcium in the blood are bones, kidneys, and the intestine.

Calcium is constantly being recycled in and out of living bone. In the adult dog, under balanced conditions, both accretion (calcium uptake) and resorption (calcium loss from bone) values vary from 0.1 to 0.2 mmol per kilogram of body weight per day. [A millimole is a minute measure of molecular weight.] For the rapidly growing puppy these values are at least 100 times higher. <sup>10</sup> Another difference between an adult dog and a puppy is their relative abilities to absorb calcium from the food they ingest. In the adult dog, the percentage of calcium assimilated from food varies from 0 to 90 percent, depending upon the composition of the food and its calcium content. <sup>11</sup>

A 1985 study which examined the physical, biochemical and calcium metabolic changes in growing Great Danes, showed that young puppies do not have a mechanism to protect themselves against excessive calcium feeding. Under the influence of certain hormones, the calcium excess is routed to the bones. This results in severe pathological consequences for the patterning for the growing skeleton and the subsequent impairment of gait. Strongly correlated with high calcium intake is disturbed enchondral ossification (growth plate anomalies) causing the clinical appearance of radius curvus syndrome and osteochondrosis (a

disturbance of bone formation within the cartilage, occurring during periods of maximum growth).<sup>12</sup> Chronic, high calcium intake in large breed dogs has also been associated with hypercalcemia, elevation of the liver enzyme alkaline phosphatase, retardation of bone maturation, an increase in bone volume, a decrease in the number of bone resorption cells, and delayed maturation of cartilage.<sup>13</sup> We can safely conclude that calcium plays a significant role in skeletal disease. The giant breed dogs, because of their rapid and intense growth, are sentinels for nutritionally influenced diseases. These changes, while exaggerated in the giant breeds, are just as real-though they may be slower to surface and not as easily identified-in the smaller breeds.

## Vitamin C

Vitamin C (L-ascorbic acid) has frequently made it into the literature along with calcium. At one time or another vitamin C has been touted by somebody as a cure-all for virtually any malady known to man and beast. This is not discount the requirements for vitamin C, for it is absolutely necessary. Fortunately for dogs, they produce an enzyme called L-gulonolactone oxidase, which allows them to synthesize vitamin C from glucose without having access to a dietary form of vitamin C. (A deficiency could only be the result of either a problem with absorption or an increased need.) Interestingly, canines produce only 40mg of ascorbate per kilogram of body weight, which is far less than other mammals with the ability to synthesize their own vitamin C. There is no established minimum daily requirement for vitamin C in canine nutrition. That said, let's look at the function of the vitamin C the dog manufactures.

Vitamin C figures prominently in the biosynthesis of collagen.<sup>14</sup> Collagen is an important structural protein in the body. There are different types of collagen, but it is Type I collagen that appears most often in connective tissue, particularly in bone and ligaments. Vitamin C adds an -OH group to the two amino acids proline and lysine. Without this functional group there is a decrease in the number of cross-links in collagen. Without this cross-linking, the melting temperature of the protein is reduced from about 39 degrees to 23 degrees centigrade. In other words, without the cross-links this protein can be denatured at body temperatures.

There is experimental evidence that vitamin C may play a role in bone mineralization by stimulating bone resorption. What has been shown by one researcher to be efficacious in treating the physical manifestations of canine hip dysplasia (CHD) is a form of vitamin C called polyascorbate.<sup>15</sup> Calcium ascorbate, used in conjunction with vitamin E, also is considered helpful in reducing the inflammatory processes that accompany the disease. In this form, vitamin C is taken up by the bone along with calcium, and this acts like a time release factor that keeps the blood plasma concentration high and the cells constantly "bathed" with vitamin C.

With all the continuing fuss about vitamin C in the fad literature, it was inevitable that it would be tried for treatment of hip dysplasia. Belfield (1976) conducted a somewhat anecdotal study on eight German Shepherd Dog litters of puppies from dysplastic parents or parents known to have produced dysplastic puppies.<sup>16</sup> Megadoses of ascorbate were given to dams (2

to 4 grams of sodium ascorbate crystals per day) and to the pups (birth to 3 weeks-calcium and vitamin E supplement; 3 weeks to 4 months-500 grams ascorbate per day; 4 months to 1.5 to 2.0 years-1 to 2 grams ascorbate per day). Belfield claimed that none of the pups developed hip dysplasia, and breeders involved with the research were so convinced that they guaranteed dysplasia-free puppies if the ascorbate therapy was followed by the new owner. It is significant to note that no follow-up studies were published. While this is interesting, there is little accepted hard evidence to suggest that supplementation with ascorbate can prevent or ameliorate canine hip dysplasia. Readers are cautioned that large doses of vitamin C are not considered mainstream prophylaxis or therapy. The truth of the matter is that it is in the genes, not the diet, though diet may play a minor part.

A recent study (1993) observed that synovial fluid volume as related to osmolality correlated highly with the incidence of hip dysplasia.<sup>17</sup> This suggested that the swelling of the joint capsule from excess fluid pressure might be forcing the femoral head out of position in the acetabulum.

## **Tissue changes**

Before any radiographic indications appear, there are structural changes at the tissue level of muscles, ligaments and cartilage. Cellular changes and molecular changes occur both in the joint capsule and in the synovial fluid. One study suggested that one of the first observable changes of the disease process is hypertrophy or swelling of the pectineus muscle fibers.<sup>18</sup> This hypertrophy is thought to be a compensatory adaptation to extreme contractile tensions and may be the result of the muscle mass trying to hold the acetabulum and the femoral head in the proper position.

Another study showed that the composition of the pectineus muscle was significantly different between 2-month-old puppies that eventually developed normal hips, and those that were dysplastic by 24 months.<sup>19</sup> The two groups differed by the size of the muscle fibers, but this time, the dysplastic animals had smaller than normal muscle fibers (hypotrophy) and the ratio between contractile tissue and non-contractile tissue was lower. Thus, not only did the affected animals have diminished capacity to contract their muscles, their muscles were also less elastic. This study begs the question of joint laxity: Once stretched, would the muscles tend to remain stretched, thus resulting in a looser hip joint? Unfortunately, it cannot be said with any certainty whether these differences are causal or correlative.

It is certain, however, that hip dysplasia is characterized by joint laxity.<sup>20,21,22,23,24</sup> Whether such laxity is the result of the pathological processes involved in the disease, or whether the laxity is the cause of the disease, cannot be determined. Remember, however, that loose joints and hip dysplasia are found together. We will be coming back to this point in later articles. There is a little twist to what we find: All dogs that have hip dysplasia have loose hips, but not all dogs with loose hips have hip dysplasia. It is not known which comes first: remodeling of the bony surfaces leading to abnormal wear of articular surfaces and joint instability or vice versa. It may very well be that both processes are concurrent and/or iterative processes. Other changes that can precede either clinical signs, like pain and gait abnormalities, or radiographic

evidence of hip dysplasia include thickening of the joint capsule and swelling of the round ligament. Subtle and early changes in articular cartilage structure also precede clinical signs. Specifically, in affected animals, the ratio between Type A cells and Type B cells differs from the norm. Type A cells are macrophages, i.e., large mononuclear cells produced by the immune system which ingest damaged cells and blood tissue. Type B cells are fibroblasts which are precursors of connective tissue. In one study, the population of Type A cells increased.<sup>25</sup> Conceptually this makes sense, as the function of macrophages is to scavenge damaged cells, which would be the case if articular cartilage is being damaged. Note that these changes can only be observed after dissection and examination under an electron microscope. While diagnostic and predictive, such examination is without use to the clinician who is trying to diagnose the disorder. What is important to remember is that these changes are found in dogs whose x-rays showed them to be perfectly normal at the time of radiographic study. As a concerned breeder or fancier of dogs, this should alarm you. Do not be too alarmed, however, because there is hope for predictive techniques. These will be covered in later articles in this series.

### Significant studies

The major study demonstrating the polygenic and multifactorial aspects of canine hip dysplasia is probably the 1991 German study on German Shepherd Dogs.<sup>26</sup> Unfortunately this article is in German and we know of no translations available. While this poses no problem for co-author Thorpe-Vargas, as she used to be at the Max Planck Institute in Germany, it is a real problem for co-author Cargill, as he has to take her word for it, supported only by Medline abstracts in English! The importance of this study is that it covered 10,595 dogs. Furthermore, this study attempted to quantify both environmental influences and genetic influences on the frequency of hip dysplasia. Models were developed using the following variables-independent random variables: age at X-raying, birth year, season, litter size, percent of X-rayed dogs in each litter and sex ratio of litter; independent fixed variables: sire and dam.

Through multiple linear and non-linear regression methods it was shown that sire, dam, sex and age at X-raying all showed statistically significant influence on the occurrence of hip dysplasia. The heritability indices ( $H^2$ ) were-Relationship: full siblings,  $H^2 = 0.30$ ; maternal half-siblings,  $H^2 = 0.48$ ; and paternal half-siblings,  $H^2 = 0.11$ .

The researchers' caveat at the end of the study was that only the paternal half siblings' heritability index should be accepted because kennel and breeder effects are confounded with the dam effect. Their overall conclusion was that the frequency of hip dysplasia could be reduced if selection for breeding based upon the estimation of breeding values ( $H^2$ ) with respect to the frequency of hip dysplasia in *all* relatives was implemented.

Many of the world's militaries are good sources of information on German Shepherd Dogs. The goals of such organizations have been to improve behavioral traits and to reduce the frequency of CHD. One of the more interesting studies in the literature is the one based upon

information provided by the US Army's division of Biosensor Research on the German Shepherd Dogs bred between 1968 and 1976.<sup>27</sup> Detailed records were available for 575 animals representing 4 years, 18 sires, 71 dams and 48 human handlers. Variance component estimates were made, which allowed estimates of the heritabilities for both temperament and CHD scores to be made. The heritability index ( $H^2$ ) for temperament was 0.51 and for CHD was 0.26. Interestingly, in this population the genetic correlation between good temperament and bad hips was -0.33. Given the selection process of the U.S. Army, it was not surprising to find that dogs with good temperaments also had good hips. Because of the extremely high heritability index for temperament, records of the animal being evaluated can be used for repeat breeding selection rather than the records of the progeny.

A 1993 Austrian dissertation looked at a population of 10,750 Hovawarts from 1962 to 1988, out of which CHD findings were available for 4,387 dogs.<sup>28</sup> The goal of the dissertation was to statistically calculate two parameters. The first was a prediction coefficient based upon the CHD findings of all the ancestors of a specific animal. The second was a "taint" coefficient calculated on the basis of the CHD findings of all ancestors as well as of the individual CHD finding as well as those of any offspring already checked for CHD. The conclusions of this dissertation were that both the "prediction" and "taint" coefficients were useful in calculating the relative CHD risk of the prospective offspring when selecting breeding partners. A connection was found between the CHD findings and the inbreeding level of an animal as calculated from the "ancestor loss coefficient" and Malecots "coefficient de parente." Thus, increasing levels of inbreeding increase the risk of CHD. There was no difference between males and females for risk of CHD. Detailed coverage of the various genetic coefficients is beyond the scope of this article. Readers are directed to modern comprehensive texts, dissertation abstracts and the like in genetics should more than a passing familiarity with the intricacies of these coefficients be required.

**Conclusions:** 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 that are exercised regularly and to avoid breeding any 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. Let your conscience and your veterinarian be your guides in supplementing with vitamin C. Fortunately, large doses of vitamin C are readily excreted, but it is still possible to cause untoward side effects with megadoses.

The next article in the series will address the abnormal hip, to include differential diagnosis, observation, palpation fluid sampling and sedated and unsedated radiographic studies.

## **CREDITS**

## References

1. Hanssen I. "Hip Dysplasia in dogs in relation to their month of birth." *Vet Rec.* 1991 May 4;128(18):425-6.
2. Kealy R.D., Olsson S.E., Monti K.L., Lawler D.F., Biery D.N., Helms R.W., Lust G., Smith G.K. "Effects of limited food consumption on the incidence of hip dysplasia in growing dogs." *J Am Vet Med Assoc.* 1992 September 15;201(6):857-63.
3. Kealy R.D., Olsson S.E., Monti K.L., Lawler D.F., Biery D.N., Helms R.W., Lust G., Smith G.K. "Effects of dietary electrolyte balance on subluxation of the femoral head in growing dogs." *Am J Vet Res.* 1993 April;54(4):555-62.
4. Cargill J.C. "Feed That Dog! Part II." *Dog World.* 1993 August;75(8):12.
5. Ibid.
6. Ibid.
7. Ibid.
8. "Effects of dietary electrolyte balance." Pp. 555-62.
9. Smith G.K., Gregor T.P., Rhodes W.H. Biery D.N. "Coxofemoral joint laxity from distraction radiography and its contemporaneous and prospective correlation with laxity, subjective score, and evidence of degenerative joint disease from conventional hip-extended radiography in dogs." *Am J Vet Res.* 1993 July;54(7):1023.
10. Hedhammer A., Wu F.M., Krook L., Schryver H.F., de Lahunta A., Wahlen J.P., Kallfelz F.A., Nunez E.A., Hintz H.F., Sheffy B.E., Ryan G.D. "Overnutrition and skeletal disease. An experimental study in growing Great Dane dogs." *Cornell Veterinarian* 1974;64 supp15:11-160.
11. Hedhammer A., Krook L., Schryver H.F., Kallfelz F. "Calcium balance in the dog." In "Nutrition of the Dog and Cat" ed. Anderson R.S.; Pergamon Press, Oxford 1980:119-27.
12. Hazewinkle H.A.W. "Influence of different calcium intakes on calcium metabolism and skeletal development in young Great Danes." PhD Thesis Utrecht State University 1985.
13. Hazewinkle H.A.W., Goedegebuure S.A. Poulos P.W., Wolvekamp W.ThC. "Influences of chronic calcium excess of the skeletal development of growing Great Danes." *J Am An Hosp Assoc.* 1985;21:377-91.
14. Berg R.A., Prockop B.J. "The thermal transition of a non-hydroxylated form of collagen: Evidence for a role for hydroxyproline in stabilizing the triple helix of collagen." *Bio Chem Bio Phys Res Commun.* 1973; Vol. 52:115-129.

15. Berge, G.E. "Polyascorbat, et behandlings-alternativ ved kroniske forandringer I støtte og bevægelsesapparatet hos hund" ("Polyascorbate, an interesting alternative by problems in the support and movement apparatus in dogs.") *Norsk Veterinaertidsskrift (Norwegian Vet J)*, August/September 1990;102:581-582.
16. Belfield, W.O. "Chronic subclinical survey in canine hip dysplasia." *Vet Med Sm An Clin.* 1976; Vol. 71:1399-1403.
17. Lust G., Beilman W.T., Rendano V.t. "A relationship between degree of laxity and synovial fluid volume in coxofemoral joints of dogs predisposed for hip dysplasia." *Am J Vet Res.* 1980,41:55-60.
18. Cardinet, G.H. III, Wallace L.J., Fedde M.R. "Developmental myopathy in the canine." *Arch Neurol.* 1969, 21:620-630.
19. Lust G., Craig P.H., Ross G.E. "Studies on pectineal muscles in canine hip dysplasia." *Cornell Vet.* 1972, 62:628-645.
20. Henricscon B., Norberg I., Olsson S.E. "On the etiology and pathogenesis of hip dysplasia: a comparative review." *J Small Anim Pract.* 1966;7:673-687.
21. Smith G.K., Biery D.N., Gregor T.P. "New concepts of coxofemoral joint stability and the development of a clinical stress radiographic method for quantitating hip joint laxity in the dog." 1990 January 1;196(1):59-70.
22. "Coxofemoral joint laxity." Pp.1021-1042.
23. Morgan S.J. "The pathology of canine hip dysplasia." *Vet Clin N.Am Sm Anim Pract.* 1992 May;22(3):541-50.
24. Alexander J.W. "The pathogenesis of canine hip dysplasia." *Vet Clin N.Am Sm Anim Pract.* 1992 May;22(3):503-11.
25. Greisen H.A., Summers B.A., Lust, G. "Ultra Structure of the Articular Cartilage and Synovium in the Early Stages of Degenerative Joint Disease in Canine Hip Joints." *Am J Vet Res.* 1982; 43:pp. 1963-1971.
26. Distl O., Grussler W., Schwarz J., Karusslich H. "Analyse umweltbedingter un genetischer Einflüsse auf die Häufigkeit von Hüftgelenkdysplasie beim Deutschen Schäferhund." ("Analysis of environmentally conditioned and genetic influences on the frequency of hip joint dysplasia in German Shepherd Dogs"). *Zentralbl Veterinarmed A.* 1991 Jul;38(6):460-71.
27. Mackenzie S.A. "Inheritance of temperament and hip dysplasia scores in German Shepherd Dogs." 1984 March; Dissertation Abstracts International-B 44/09, p.2652.

28. Potscher L.A. "Selektion gegen huftegelenkdysplasies (HD) in einer Hovawart population" ("Selection criteria concerning hip dysplasia (HD) in a Hovawart population.") 1993 Winter; Dissertation Abstracts International-C 54/04, p.1069.

[NEXT SEGMENT](#)