

THE USE OF HEALTH DATABASES AND SELECTIVE BREEDING

A Guide for Dog & Cat Breeders & Owners



Seventh Edition • 2018

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In memory of all our special animal friends

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Dr. George M. Strain—Genetic Deafness

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INTRODUCTION

Breeders have an inherent responsibility to protect the comfort and well-being of the animals they produce, yet the dog and cat owning public spends hundreds of millions of dollars each year on diagnosis and treatment of genetic diseases. These factors justify placing continued emphasis on prevention of these diseases. Responsible breeders and the more progressive breed clubs are, and have been, responding to the challenge of improving the genetic health of our companions through better breeding practices.

OFA—The Canine Health Information Center is a private non-profit foundation which formed a voluntary dysplasia control database in 1966. Today OFA-CHIC operates under the following objectives:

- To collate and disseminate information concerning orthopedic and genetic diseases of animals
- To advise, encourage and establish control programs to lower the incidence of orthopedic and genetic diseases
- To encourage and finance research in orthopedic and genetic disease in animals
- To receive funds and make grants to carry out these objectives

The OFA's voluntary databases serve all breeds of dogs and cats and have the world's largest all-breed data bank on radiographic evaluations of the hip and elbow. The testing methodology and the criteria for evaluating the test results for each database were independently established by veterinary scientists from the respective specialty areas. These standards are accepted throughout the world, and the results are used to evaluate prevalence and progress in controlling the respective diseases in the breeding population. The OFA serves as a central source of information for breeders and owners based on the standards for evaluation, and as a major source of funding for studies directed at animal wellness.

The purpose of this monograph is to assist the breeder, dog owner and veterinarian in accomplishing their goals by providing a summary of information on the OFA databases, their methodology and a reference source for further study. Data on individual animals may be obtained at www.ofa.org. This data can be useful for the breeder to determine the status of potential breeding animals and their family lines.

RECOMMENDATIONS FOR PUPPY BUYERS

To verify health information when considering a purchase from a particular breeder, the buyer should obtain a pedigree of the animal in question. Health information can then be verified on the sire, dam, various siblings and other close relatives at the OFA website, www.ofa.org. Information in the OFA's database can be used as a tool to increase the probability for obtaining a normal dog when choosing dogs for breeding, competition or as healthy pets. Overall, if there are a substantial number of relatives that do not have OFA numbers in the pedigree, they should be assumed to be abnormal until proven otherwise. The more animals in a pedigree with OFA numbers, and the greater the percentage of their siblings with OFA numbers, the better the genetic probability for healthy animals from a given breeding. Breedings for which two to three generations of this depth and breadth of information is available and normal will usually demonstrate significantly reduced incidence of disease.

It also may be helpful to consider whether the breeding in question is a repeat breeding, a line breeding, or an outcross. With repeat breedings, there may be health information available on puppies from the previous litter resulting from the same genetic combination. In the case of line breedings, experienced, knowledgeable breeders often have extensive information about the phenotypes present in their lines, and therefore can make more informed breeding choices. Longtime health conscious breeders often have greatly reduced the incidence of disease in their breeding programs, and

this will be reflected in their track record (as verifiable on the OFA website). Outcross breedings require more diligence from the breeder to fully investigate the new lines that are brought into the pedigree, and again, information available on the OFA website may greatly aid in this effort.

GENERAL OFA APPLICATION INFORMATION

The owner or agent should complete and sign the appropriate OFA application form. Animal information is best obtained directly from the animal's certificate or registration papers. It is also important to record the animal's tattoo or microchip number and registration numbers of the sire and dam. Application forms are available online at www.ofa.org.

Completed and signed application forms should include the owner's choice of open or semi-open database. Applications for radiographic evaluations should accompany the radiographs sent to OFA, whether by mail or email. Digital radiographs, whether on a CD or via email, are only accepted directly from the attending veterinary clinic. All other applications can be mailed to OFA, 2300 E. Nifong Blvd., Columbia, MO 65201-3856, or emailed to ofa@offa.org. All radiographic images are retained by the OFA for research and reference purposes.

OFA OPEN DATABASE

Historically only normal results from data submitted to the OFA were reported in the public domain (semi-open databases). All normal results are still reported in the database; the OFA now provides owners the choice of whether or not they want to report abnormal information in an open database. **The open database provides all information, normal and abnormal, in the public domain.** All OFA applications include a section where the owner or agent can initial to authorize the release of abnormal results.

If an owner wishes to retroactively release abnormal results into the public domain, there is a "Form to Change from Semi-Open to Open Database" available on the OFA website for this purpose.

GENETICS

Inherited traits, desirable or not, are controlled by the genetic makeup (genotype) of an individual animal. The genotype is determined by the genes received from the parents, one-half from the sire and one-half from the dam. Most inherited traits in animals are polygenic (controlled by two or more genes). Some examples are: conformation, type, size, longevity, disease resistance, temperament, speed, milk and egg production, growth rate, maturation and sexual maturity rate, and numerous inherited diseases.

Intuitively, it is recognized that these traits do not follow inheritance patterns based on simple Mendelian genetics. Mendelian genetics usually uses one pair of genes to explain basic genetic principles. For example, assume that: 1) The color black is dominant to brown, 2) The black gene is represented by B and the brown gene by b, and 3) a homozygous black (BB) is mated with a brown (bb). All of the offspring will be black, but will have the heterozygous Bb genotype. If two heterozygous blacks (Bb) are mated, Mendelian genetics predicts the offspring are expected to be three black (1 BB and 2 Bb) and one brown (bb). The ratio of 1:2:1 for the genotypes is based on probability. If only a small number of offspring are available from this type of mating, they may not fall within the ratio, but larger numbers will produce the predicted results. In addition, the finding of one brown offspring from the mating of black parents indicates that both parents are carriers (heterozygous Bb) of the recessive brown gene. In such a case, two out of three black offspring are also carriers, but until they are bred it is uncertain which are the carriers. In the above example of simple Mendelian genetics, the probable genotype of the parents can be determined by examination of the progeny.

However, polygenic traits, such as most characteristics that breeders are concerned with, are defined as those affected by multiple gene pairs. An oversimplified example is two genes affecting the same trait. Assume the mating of two dogs with genotypes of AaBb, where the dominant alleles “A” and “B” are desirable. The expected genotypic outcome is nine different genotypes with the following frequencies:

Genotypes	AABB	AABb	Aabb	AaBB	AaBb	Aabb	aaBB	aaBb	aabb
Frequency	1/16	2/16	1/16	2/16	4/16	2/16	1/16	2/16	1/16

Only 25% of the progeny from this mating are expected to have the same genotype for the trait as the parents. Some of the remaining progeny will have a

more desirable genotype (AABB, AABb, AaBB) while others will have a less desirable genotype for the trait (Aabb, AAbb, aaBB, aaBb, aabb). As the number of genes involved increases, the possible combinations soar. The problem is further magnified if each gene pair exerts a different degree of influence on a trait to produce an “additive” result. It is currently impossible to precisely predict the specific outcome of a particular mating with regard to polygenic (additive) traits, and probabilities can only be generally estimated.

However, animal geneticists have developed successful breeding programs to improve milk production in cows, egg production in hens, speed in horses, growth rate in food animals, etc. They use basic genetic principles that have also been demonstrated effective in the dog. Some of the aspects of polygenic traits considered in arriving at these principles include:

Polygenic traits have a range of manifestations from the most desirable to the least desirable characteristic under consideration.

For example, mating two dogs of ideal conformation can be expected to result in a larger number of offspring with ideal conformation when compared with offspring of a mating where one or both parents have less than ideal conformation. However, both litters will present a range of conformational characteristics.

Polygenic traits are influenced by environmental factors which may minimize or maximize genetic potential.

For example, a horse with a respiratory infection will not be able to achieve its genetic speed capability, nor will a cow on a starvation diet produce milk to its full genetic potential.

Heritability measures the phenotypic expression of multiple genes as possibly modified by environmental influences, and the degree to which the resulting phenotype predicts the genotype. The equation P (phenotype) = G (genetics) + E (environment) is a starting point. This equation means the variation in phenotype presented comes about from the complex interaction of the animal’s own inherited genotype with the environment to

which it has been exposed. Using hip dysplasia (HD) as an example, some environmental factors include, but are not limited to, overweight, rapid growth rate, early maturation, sex of the animal, etc. The most studied environmental influence on HD is caloric intake.

It is important to understand that heritability estimates do not refer to the degree of inheritance, but rather to the degree that the additive genetic component is reflected in the phenotype. This is easier to understand using a trait for which most people have a greater intuitive grasp. In dogs, wither height is a polygenic trait that may be modified by the environment. Height may be influenced by restricting calorie or vitamin intake, certain environmental effects on hormones (such as early spay/neuter), and other environmental factors. Despite those potential environmental influences, height is recognized to be an inherited trait. However, one cannot accurately predict the height of an offspring by knowing the height of parents or siblings. This is because polygenic traits have many complex genetic interactions, in addition to their interactions with the environment. Thus, when one is only able to measure the height of parents or siblings, one is measuring their phenotypes, and not able to consider their genotypes and the various possible interactions of those genes. It may be helpful to substitute “predictability” for “heritability” to further clarify this concept.

Heritability estimates are usually determined through mid-parent offspring analysis using statistical methods, and express the reliability of the phenotype as a guide to the predictive breeding value of the animal. Heritability estimates are reported on a scale from 0 to 1.0 (0-100%). These are expected to vary depending on the genetic background of the studied breed population and will change over time through selective breeding.

If the heritability estimate for a given trait is 0.1, it is generally considered low and the animal’s phenotype is not a good indicator of the genotype (breeding value). Genetic selection based on a single phenotype would yield poor results. Although difficult to obtain for most hobby breeders, phenotypic information on many offspring raised in different environments (progeny testing) would offer additional insight into the parent’s genotype.

If it is between 0.2 and 0.3, the heritability estimate is generally considered moderate. The animal’s phenotype predicts its genetic makeup to a reasonable degree, and genetic selection based on the individual animal’s phenotype

Table 1: Progeny results of matings between parents with known hip scores

Scores on 490,966 progeny from sires and dams with known hip scores

		Dam Rating							Sire Rating
		Excellent (1)	Good (2)	Fair (3)	Borderline (4)	Mild (5)	Moderate (6)	Severe (7)	
Excellent (1)	Dysplastic (%)	3.6	6.1	9.6	12.3	13.4	18.7	18.5	
	Total	17,972	52,784	9,039	155	1,271	729	65	
Good (2)	Dysplastic (%)	5.8	9.6	14.6	17.5	18.9	23.0	31.5	
	Total	50,485	217,938	49,212	811	6,930	3,973	461	
Fair (3)	Dysplastic (%)	9.4	14.1	19.8	22.8	26.5	32.2	37.1	
	Total	6,241	41,628	13,513	263	2,301	1,328	167	
Borderline (4)	Dysplastic (%)	8.9	17.7	20.2	22.2	30.8	50.0	50.0	
	Total	79	532	168	9	39	30	4	
Mild (5)	Dysplastic (%)	16.4	18.3	27.2	36.2	29.6	41.4	45.0	
	Total	807	4,531	1,532	47	459	239	40	
Moderate (6)	Dysplastic (%)	18.9	22.8	31.6	34.4	35.0	38.0	65.3	Sire Rating
	Total	428	2,618	896	32	266	213	49	
Severe (7)	Dysplastic (%)	22.0	24.2	36.0	44.4	39.6	55.8	44.4	
	Total	59	360	136	9	48	52	18	
Total		76,071	320,391	74,496	1,326	11,314	6,564	804	

is expected to yield slow yet substantial results. However, more rapid results can be achieved if phenotypic information on relatives (pedigree depth and breadth) is also considered. This also increases the accuracy in predicting the animal’s breeding value and aids in identifying carrier animals.

If the heritability estimate is between 0.4 and 1.0, it is generally considered high and the animal’s phenotype is a good predictor of its genetic makeup. In this case, rapid results can be obtained with genetic selection based on phenotype.

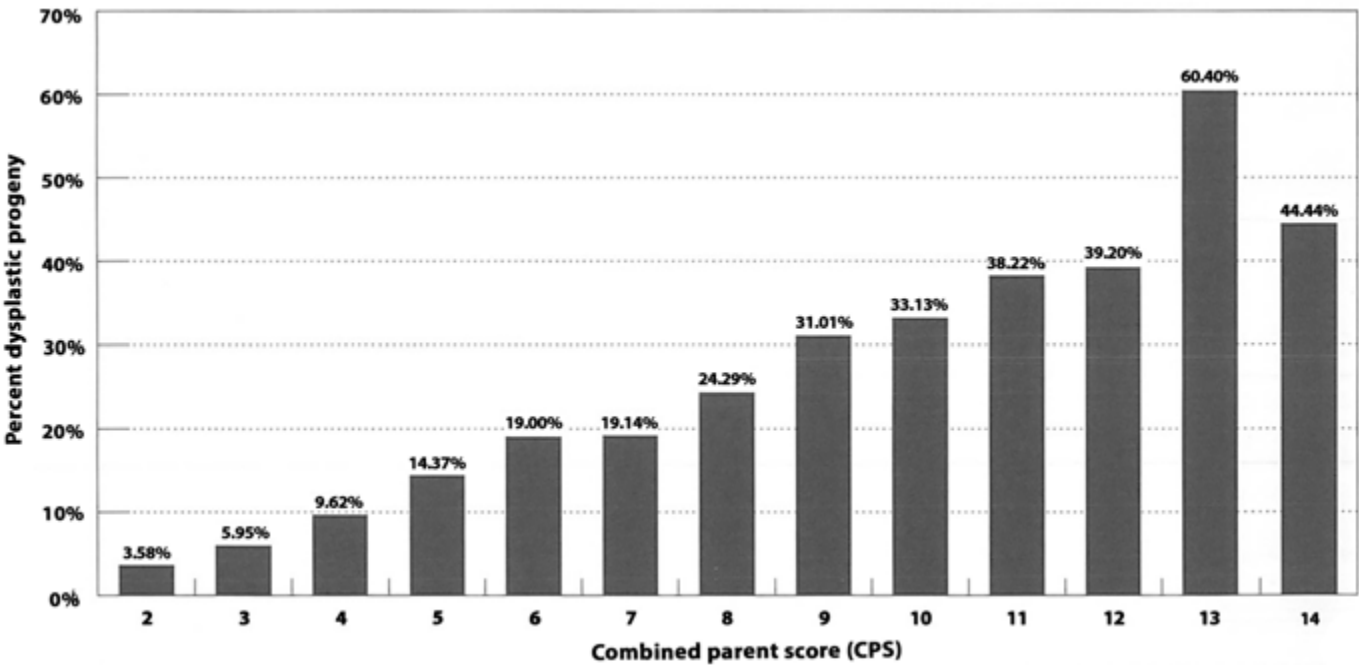
Breeding based on individual phenotypes appears to be the method used by most breeders, as available information on relatives is somewhat limited. For traits considered to have moderate heritability, this approach will reduce the frequency of an undesirable trait in the progeny, but progress, while substantial, will be slow.

Information on siblings of an individual animal, plus information on the siblings of parents and grandparents, makes it possible for the breeder to apply greater selection pressure against the disease. This results in selection of animals with more ideal breeding values and provides a more rapid reduction of the undesirable trait in the breeding program. The vertical pedigree feature accessed on individual dog pages on the OFA website allows the user to take advantage of additional pedigree depth and breadth information when available.

The following breeding selection criteria have been demonstrated to more rapidly and effectively reduce the frequency of undesirable traits:

- 1. Breed only normal dogs to normal dogs**—using hip dysplasia as an example, **Table 1** and **Figure 1** illustrate the outcome of matings based on information extracted from the OFA database. A total of 490,966 progeny were identified where both parents had hip conformation ratings (Keller). The percentage of dysplastic progeny increased as the sire’s and dam’s phenotypic hip ratings decreased from excellent through dysplastic. Reed reported equal genetic contribution on progeny hip scores from the sire and dam.
- 2. Breed normal dogs that come from normal parents and grandparents**—this employs the traditional horizontal pedigree with emphasis on the most immediate three generations (50% genetic contribution from each parent, 25% from each grandparent and 12.5% from each great grandparent).

Figure 1: Relationship of Combined Parent Score to percentage of HD progeny



Hip grades were assigned a numerical value: excellent–1, good–2, fair–3, borderline–4, mild hip dysplasia–5, moderate hip dysplasia–6 and severe hip dysplasia–7. For example, two excellent parents have a CPS of 2 and two severe parents would have a CPS of 14. Also see number associations in Table 1.

3. **Breed normal dogs that have more than 75% normal siblings**—this information is usually not available, since most animals in a litter become pets and are not screened for undesirable traits. Breeders can add incentives to purchase contracts in an attempt to gather this information, such as offering reimbursement for a preliminary hip radiograph taken when the pet dog is spayed/neutered.
4. **Select a dog that has a record of producing a higher than breed average percentage of normal progeny**—if known, the comparison of production performance between individuals is an important criterion. For example, a stud dog with a track record of producing 90% normal progeny is far superior to another dog producing only 50% normal progeny.
5. **Choose replacement animals that exceed the breed average**—exert constant, consistent pressure to ensure overall breed improvement.

In summary, achieving goals in a breeding program depends upon the ability to assess an animal's predictive breeding value. Important information to assist breeders in achieving their goals is available on the OFA website through the database search option (www.ofa.org).

DNA GENETIC DATABASES

DNA testing based on identification of a specific genetic mutation is the most accurate method of identifying an animal's genotype, and knowledge of the genotypic status is the breeder's most powerful tool for elimination of a genetic disease. There are several broad categories into which DNA tests may fall. The most straightforward are tests which definitively predict whether or not a dog will manifest a certain disease, and also predict the risk to its offspring. These include tests for simple recessive genes without complex modifiers (incomplete penetrance) or environmental interaction. Most current DNA tests fall into this category. Such tests can identify affected/at risk (homozygous for the disease alleles), carrier (phenotypically normal, but heterozygous with one disease allele and one normal allele), or clear/normal (homozygous normal) dogs, both in adults for breeding purposes, and within litters to help determine the appropriate placement of a puppy. In the case of diseases caused by recessive genes, careful and knowledgeable breeding decisions and strategies may permit the use of carrier, or rarely, affected/at risk, animals in breeding programs for a short period of time. This enables the breeder to maintain desirable breed traits within a breeding program, while being assured of producing offspring that are phenotypically normal, and making rapid progress toward the goal of producing offspring that are genotypically normal.

DNA tests can also be used to detect genes that do have modifiers such as incomplete penetrance or environmental influences. In the case of dominant genes with modifiers, DNA tests could definitively detect which dogs have the abnormal gene, but this would not predict with certainty whether such a dog would actually develop the disease. It would, however, give accurate information with regard to the odds of the disease gene being passed to the next generation (although once again, not with certainty whether offspring will develop the disease). Perhaps equally as important, DNA tests could be done on puppies in litters. This would enable breeders to place only pups who do not carry the disease gene at all into potential breeding situations, while those who test positive could be placed into non-breeding homes. Remember, however, that when a dominant gene is involved, such a pup may develop the disease and have a compromised quality of life. The ability to use DNA testing to keep pups with abnormal genes out of breeding homes should never be thought of as a way to excuse breeding a dog that is capable of producing clinical disease, and the possibility of producing affected pups with these types of breedings must be given appropriate and compassionate weight. In the rare instance where desirable traits of an individual capable of producing disease outweigh the undesirable genetic trait, a breeding should only be undertaken with a clear commitment to eliminate the disease gene in the next generation through diligent DNA testing.

DNA testing by linkage is not as accurate as that for identification of a specific genetic mutation, but it is still more desirable than existing tests based on phenotypic evaluations. While some minor degree of false positives and false negatives is possible, accuracy rates are usually above 95%.

The financial advantages of DNA testing and associated DNA profiling are clear. Tests are accurate, can be done at an early age and only one test is required. Progeny can be cleared by parentage for one generation when DNA profiles are available for determination of parentage.

There are numerous genetic testing facilities that utilize DNA based testing. All of these tests are more sensitive and specific for the detection of genetic disease traits than are phenotypic based tests. Check the OFA website for breed specific DNA test availability.

OFA DNA TESTS

The OFA offers DNA testing in conjunction with the University of Missouri. For the current list of OFA/MU DNA tests, go to www.ofa.org/diseases/dna-tested-diseases.

OTHER DNA TESTS

The OFA provides a comprehensive listing of all known DNA testing facilities, breeds and labs at www.ofa.org/diseases/dna-tested-diseases/all-dna-tests. This list is updated regularly as new tests from recognized labs become commercially available.

DNA APPLICATION INFORMATION

The OFA provides a central repository of DNA test results from approved laboratories for purposes of monitoring disease and as a source of information for breeders, breed clubs, owners, prospective owners and researchers. DNA application forms can be downloaded from the OFA website (www.ofa.org/veterinarian/application-forms). There is a minimal cost to enter a clear/normal or carrier in the data bank; litter and kennel discount rates are available for non-OFA DNA tests. Submitting results for the same DNA test for three or more dogs from the same litter, or five or more dogs owned or co-owned by the same person, qualifies that submission for the litter and kennel rates, respectively. There is no charge to enter an affected/at risk individual, as it is important for scientific analysis that affected information be entered into the database.

HIP DYSPLASIA

Hip dysplasia (HD), literally defined as an abnormal development of the hip joint, was first reported in the dog in 1935 by Dr. G.B. Schnelle. Little to no further information was added to his report over the following decade, due primarily to limited availability of radiographic equipment and radiographic expertise within the veterinary profession.

Popularity of the working dog, particularly the German Shepherd Dog, increased greatly in the late 1940s and the importance of HD became evident to breeders, dog owners and the veterinary profession. Unrelated, but concurrently, veterinary education underwent an explosion in numbers of veterinary colleges and in quality of specialized education. Rapid advances in the veterinary profession made it difficult for most general practicing veterinarians to remain current with expanding knowledge in animal diseases. To provide the best possible diagnosis and patient care multiple specialty colleges were formed, including the discipline of radiology, which became a recognized specialty in 1966 through the American College of Veterinary Radiology (ACVR).

Hip dysplasia has been reported in man and in most domestic species of animals. In some breeds of dogs and cats, it is the most common cause of osteoarthritis (degenerative joint disease). In recent years, interest in canine HD research has been at an all-time high, as evidenced by the number of conferences focusing on the subject and by the number of new publications in scientific journals and popular magazines.

We now know that HD is a more complex disease than what was first believed. The complexity of the problem is expected to, and has produced, research findings that appear to be contradictory. These research reports, and anecdotal writings that continually appear in the popular press, contribute to confusion and frustration in breeders and veterinarians not familiar with the scientific literature. Thus, few diseases in animals have resulted in such extreme emotional reactions, controversy or monetary expense as HD.

While it is useful to summarize results from the scientific literature, in the final analysis more research is needed to find answers to the many unresolved questions about HD.

Hip dysplasia is currently accepted to be an inherited disease caused by the interaction of many genes (polygenic). In animals that are genetically predisposed, there are unknown complex interactions of genes with the environment that bring about the degree of phenotypic expression (mild, moderate, or severe hip dysplasia) of these genes within an individual.

At this time, selectively breeding for normal hips is the only means to reduce the genetic frequency of HD.

Radiography is currently the accepted means for evaluating the hip status and it is well documented that the frequency of HD can be significantly reduced using the standard hip extended view.

It is expected that future research studies will refine these currently accepted tenets. For example, advances in molecular genetics may bring about DNA tests to replace radiography as the primary diagnostic tool, or environmental factors such as medical or nutritional treatments may be identified that will overcome the genetic expression of HD in an individual animal.

There are many debates surrounding the myriad of possible factors that may influence or initiate one or more aspects of HD. While interesting to consider, the breeder and veterinarian can most successfully pursue their mutual goals by maintaining their focus on current knowledge without becoming mired in the debate. The responsible breeder attempts to produce the best possible representatives of the breed. The veterinarian assists the breeder in accomplishing this objective by encouraging breeder education, maintaining the general health of the dog and/or cat, and providing the best possible treatment when appropriate.

DEVELOPMENT OF THE HIP JOINT

The embryonic hip joint and its supporting structures begin to develop from an undifferentiated mass of embryonic tissue. The differentiation of this tissue into the distinct parts of the hip joints is predetermined by a genetic code. Embryonic tissues form muscles, a specialized connective tissue that encases the joint (the joint capsule), and joint ligaments. A cartilage mold forms the unique parts of the ball and socket joint, with the acetabulum functioning as the socket and the head of the femur functioning as the ball. These structures continue to grow and differentiate as the embryo matures. Ossification (bone formation) begins at approxi-

mately 49 days of pregnancy, but the degree of skeletal maturity at birth appears to be breed dependent. That is, ossification in some breeds is more advanced than in others, which contributes to the continued difference in rates of skeletal growth after birth.

The surfaces of the femoral head and acetabulum are covered with smooth articular cartilage. A thin layer of fluid (synovial fluid) serves as a lubricant for the joint, carries nourishment for the articular cartilage, and separates the opposing surfaces. The head of the femur is attached to the depth of the acetabulum by a ligament (round ligament). The joint capsule encases the joint by attaching to the neck of the femur and to the rim of the acetabulum and is lined by a specialized tissue, the synovial membrane, which produces the synovial fluid. Muscles encase the entire hip structure and serve to stabilize and move the joint. The major pelvic muscles exert a forward and upward pressure on the femoral head during movement, and the head of the femur is held in the acetabulum by the pelvic muscles, the joint capsule, surface tension and the round ligament. Proper development of the joint depends upon the head of the femur being held firmly within the acetabulum.

The hip joint of the dog is reported to be normal at birth. After birth, a complex interaction of multiple genetic and environmental factors can initiate incorrect fit or function of one or more of the parts of the hip joint, although the exact pathogenesis of these interactions is not fully understood at this time. It is likely that these factors may differ between genetic lines, since HD is caused by the interaction of many genes. Currently, any attempt to define the process in an exact sequence of events is speculative.

Regardless of what the initiating interaction of factors may be, abnormal looseness (joint laxity) is generally accepted to be the most common abnormality that results in the pathologic changes of HD. However, some dogs with tight hips but shallow acetabula have also been reported to develop dysplastic changes.

Many of the early (2-14 weeks) pathologic changes are not readily detectable by clinical or radiographic examination. These include: swelling, fraying and possible rupture of the round ligament; inflammation of the synovial membrane (synovitis) resulting in synovial fluid changes; stretching of the joint capsule; and damage to the cartilage mold of the acetabulum and femoral head. These structural alterations result in joint instability and subluxation, which are followed by erosion of the articular cartilage; changes in the bone beneath the articular cartilage; micro fractures of the dorsal acetabular rim; filling in of the acetabulum; remodeling (change in size, shape or architecture) of the femoral head, neck and acetabular rims; and production of osteophytes (bone spurs) around the joint.

Depending on the individual dog and the initiating factors of joint instability, the changes occur at varying rates and to differing degrees. Severe cases can be detected radiographically as early as 8 to 12 weeks of age, while others may not be evident until later in life (greater than 2 years of age).

CLINICAL FINDING OF HIP DYSPLASIA

While animals with HD may not exhibit clinical signs, those that do are usually first affected between 3 and 15 months of age. In some, the signs may not be observed until later in life. The signs 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 animal warms up, changes in disposition due to pain, lameness after exercise, a wobbly gait, a clicking sound when walking, and many others. Many animals will shift their center of gravity forward in an effort to relieve weight and pressure on the hips, thereby developing disproportionately greater muscle mass in the front limbs as compared to the rear limbs.

The hip joint is a weakened structure in dysplastic animals and is more prone to injury from normal activities such as jumping off a couch or roughhousing with a playmate. Frequently this results in an acute lameness that appears as if it might have been caused by injury, whereas the underlying dysplasia actually made the joint more susceptible to injury. Obviously the normal hip can be injured, but radiographic examination can usually distinguish between a hip problem due to dysplasia and one due to other causes.

HD cannot be diagnosed by observing how the animal moves, acts, lies down, etc. Clinical signs may have other causes, and therefore a complete orthopedic and radiographic examination is required before arriving at the conclusion that the signs are caused by HD.

RADIOGRAPHIC ASSESSMENT OF THE HIP JOINT

Modern breeds vary widely in body size, shape and pelvic conformation. Because of these differences, OFA classifications are based on comparisons among individuals of the same breed and age. Knowledge of hip phenotype can be valuable for the breeder in selection against hip dysplasia and in estimating the potential for an active working life. It is assumed that radiographs submitted to OFA are generally screened by the veterinarian, and many of the more obvious cases of HD are probably not submitted. Therefore, the actual frequency of HD in the general population is not known, but has been approximated by Corley and Rettenmaier to be higher than reported by OFA. However, the main objective of the OFA is to identify phenotypically normal animals as potential breeding candidates. Thus, the OFA-reported breed frequency of HD can be used as a benchmark for breeders to gauge their breeding program's relative position.

Historically the diagnosis of HD has been determined by radiographic examination of the hips according to the protocol established by the American Veterinary Medical Association in 1961. In this standard hip extended position (ventrodorsal view), the animal is placed on its back with the pelvis symmetrical, both femurs extended and parallel, and with the stifles (knees) rotated internally, placing the patellas (knee caps) on the midline. The radiograph should include the last two lumbar vertebra and the stifle joints. It is essential, particularly in marginal cases, to obtain proper position and radiographic technique.

The radiographic criteria of subluxation, shallow acetabula, remodeling and/or secondary degenerative joint disease are well documented. However, interpretation and application of these criteria differ between breeds, age of evaluation and veterinarians. **Figure 2** provides the nomenclature of the hip structures that are evaluated by the veterinary radiologist. The veterinary radiologist is concerned with deviations in these structures from the breed normal, and with evidence of subluxation and degenerative joint disease (also called arthritis, osteoarthritis or osteoarthrosis).

MULTIPLE ANATOMIC AREAS OF THE HIP ARE EVALUATED (FIGURE 2) INCLUDING:

1. **Craniolateral acetabular margin**—Area where abnormal bone spurs (osteophytes) develop as the dysplastic joint attempts to stabilize the biomechanically unstable femoral head.
2. **Cranial acetabular margin**—Area visualized in conjunction with the hip ball to assess the degree of congruity and confluence of the hip joint.
3. **Femoral head (hip ball)**—Assessed to determine its fit into the socket and degree of congruity with the cranial acetabular margin forming the joint space.
4. **Fovea capitis**—Normal flattened area on ball for attachment of the round ligament; can be mistaken for degenerative changes if there is lack of familiarity or inexperience in interpretation of hip radiographs.
5. **Acetabular notch**—Area visualized to help assess depth of socket or "degree of fit."
6. **Caudal acetabular rim**—Area where bone spurs can form.
7. **Dorsal acetabular margin**—Area visualized to assess the depth of the hip socket (acetabulum) and percent coverage of the femoral head.
8. **Junction of femoral head and neck**—Area visualized to assess size, shape and architecture of the femoral head/neck. The neck of the hip ball is usually the earliest and most commonly affected area where degenerative changes occur in a dysplastic joint. In the dysplastic joint, new bone builds up at the site of attachment of the joint capsule and muscular attachments. This is a result of abnormal stress created by incongruent articulation of the ball with the acetabulum during movement.
9. **Trochanteric fossa**—Area to assess for any microtrabecular bone changes or new bone proliferation.

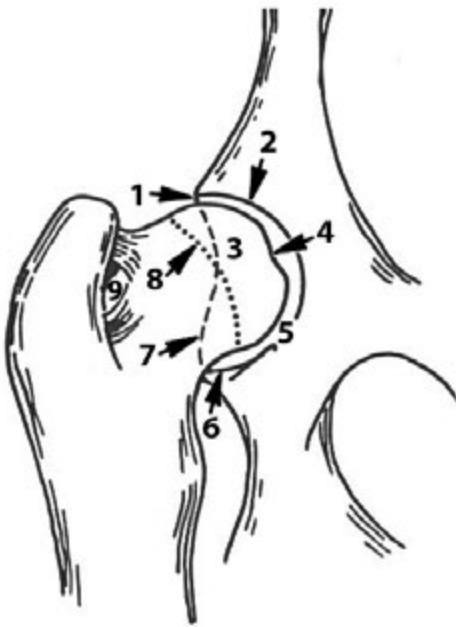


Figure 2

UNILATERAL HIP DYSPLASIA

Hip dysplasia may occur in only one hip (unilateral). In man, the left hip is reported to be involved more frequently than the right at a ratio of 10:1. Unilateral dysplasia in dogs follows a similar pattern, but the predominantly affected side is breed dependent. It occurs more frequently in the left hip of the Labrador Retriever, Newfoundland, Akita and Golden Retriever, but more frequently in the right hip of the Rottweiler. The German Shepherd Dog does not appear to have a side (left or right) predilection. Frequency of unilateral HD is also independent of the frequency of HD in a breed.

Chase (2004) identified quantitative trait loci (QTL's) associated with hip joint laxity; one for the left hip and the other for the right hip in the Portuguese Water Dog.

The reported frequency of unilateral HD varies from 3% to more than 30% of the dysplastic dogs depending on the population studied. It appears that frequency of unilateral HD is higher in some genetic lines within a breed, than in other lines within the same breed. Furthermore, the same hip (right or left) is repeatedly involved within the line. That is, when several or influential ancestors have unilateral HD in, for example, the left hip then the progeny that are unilaterally affected will almost invariably show the abnormality in the left hip.

HIP DYSPLASIA DATABASE

The OFA hip dysplasia control database functions as a voluntary screening service and as a database of hip status for dogs and cats of all breeds. Information intended to aid breeders in reducing the incidence of this polygenic problem is made available from this resource. The necessity for such a central repository was recognized by the Golden Retriever Club of America and the German Shepherd Dog Club of America, which provided the impetus for formation of the OFA.

The owner or agent should notify the veterinarian, before the x-ray examination, that the purpose is for OFA evaluation. This is best done at the time of making an appointment in order to ensure that application forms are available and that the required procedures are followed. The owner also should provide the animal's registration certificate (or copy of this information) and the animal's tattoo or microchip number at the time of radiography.

GENERAL PROCEDURES

Age—Only dogs and cats that are 24 months of age or older at the time of radiography can qualify for an OFA breed registry number. The hip joint status of younger animals will be evaluated, but only a preliminary consultation report will be issued.

Restraint—Obtaining a properly positioned film may require chemical restraint and is recommended by OFA. The type of restraint used—physical, sedative, tranquilizer or general anesthesia—is best determined by the veterinarian. The dog should not be fed on the day of radiography.

Positioning—Dorsal recumbency with the rear legs extended and parallel to each other and the stifles rotated internally is the prescribed position (**Figure 3**). This standard ventrodorsal view is accepted worldwide as the basis for evaluation of hip joint status with respect to hip dysplasia. Care should be exercised to be sure the patient is positioned correctly.

Film size—For traditional (non-digital) x-rays of large and giant breeds of dogs, 14 X 17 inch film size is recommended. Smaller film sizes can be used for smaller breeds if the area between the sacrum and stifles can be included.

Film Identification—To be eligible for OFA evaluation all radiographs submitted must include permanent animal identification in the film emulsion (traditional x-rays) or overlaid on the image (digital x-rays), using the dog's full registered name, if applicable, registration number OR microchip number/tattoo. Lead letters, radio opaque tape or digital overlay can be used to identify the film with: a) the hospital or veterinarian's name, b) date of radiograph and c) dog ID as noted above.

If the above required information is illegible or missing the OFA cannot accept the film for evaluation purposes, and the clinic will be contacted and asked to resubmit radiographs with the required information.

Exposure—Good contrast is essential. Technique settings (low kVp and high mAs), film-screen combinations and use of grids are all considered in producing the desired contrast. Film contrast should be such that the micro-trabecular pattern of the femoral head and neck are readily seen. The dorsal-lateral margin of the acetabulum must also be visible.

Radiation safety—Proper collimation and protection of attendants are the responsibility of the veterinarian. Gonadal shielding is recommended for male dogs. Radiography of females in season or pregnant should be avoided.

Application information—The owner or agent should complete and sign the appropriate OFA application form. Animal information is best obtained directly from the animal's certificate or registration papers. It is also important to record the animal's tattoo or microchip number, and registration numbers of the sire and dam. Application forms are available online at www.ofa.org.



Figure 3

A standard position radiograph of the pelvis that has been appropriately positioned will have symmetrical obturator foramina (long arrow), symmetrical wings of the ilium (arrowhead) and knees that are centered over the knees (short arrow) with the legs extended parallel to one another.

Completed and signed application forms should include the owner's choice of open or semi-open database. Applications for radiographic evaluations should accompany the radiographs sent to OFA, whether by mail or email. Digital radiographs, whether on a CD or via email, are only accepted directly from the attending veterinary clinic, and can be mailed to OFA, 2300 E. Nifong Blvd., Columbia, MO 65201-3856, or emailed to applications@offa.org. All radiographic images are retained by the OFA for research and reference purposes.

OPERATIONAL PROCEDURES

When a radiograph arrives at the OFA, the information on the radiograph is verified against information on the application form. The age of the dog in months is calculated and the submitted fee is recorded. The veterinary radiologist on staff at the OFA then evaluates the radiograph for diagnostic quality. If it is not of suitable diagnostic quality (the hip is tilted, too light or too dark, etc.) it is returned to the referring veterinarian with a written request that it be repeated (**Figure 4**). If the required dog ID is not present on the radiograph, or does not match information on the Hip/Elbow application, the clinic is contacted with a request to add or correct the ID. Once the radiograph is accepted for evaluation it is assigned an application number and given a "quality control" hip rating.

There is a pool of 20 to 25 board certified veterinary radiologists throughout the USA in private practice and academia that consult for the OFA. The radiographic images are randomly assigned and forwarded to three radiologists. Each evaluation is independent—that is, no radiologist knows what interpretation was given by another. The only information they have is the radiograph, OFA application number, breed, sex and age. The breed, age and sex of dog are important for the radiologists to know so that normal conformational differences among and within breeds, and differences related to degree of skeletal maturity, can be taken into consideration. Each radiologist grades the hips into one of seven phenotypic hip conformation categories: excellent, good or fair (which are normal and receive an OFA hip number); borderline; or mild, moderate or severe (which are abnormal and do not receive an OFA hip number).

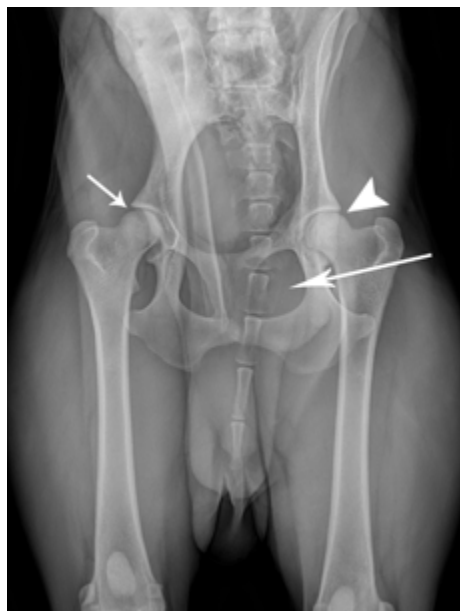


Figure 4

A standard position radiograph of the pelvis that has been inappropriately positioned will have asymmetrical obturator foramen and asymmetrical wings of the ilium (long arrow). The distortion caused by poor positioning can inaccurately make one hip look worse than it actually is by creating a more shallow appearing hip socket (short arrow) and the opposite hip appear better than it actually is by creating more depth to the hip socket over the hip ball (arrowhead). The OFA will routinely send poorly positioned films back to the referring veterinarian and request a repeat of the study.

When results of over 1.5 million radiographic evaluations by 35 radiologists were analyzed, it was found that all three radiologists agreed as to whether the dog/cat should be classified as having a normal phenotype, borderline phenotype or HD 94.9% of the time. A retrospective study using both digitized and conventional images on the same dog yielded identical results. In addition, 73.5% of the time, all three radiologists agreed on the same hip phenotype (excellent, good, fair, borderline, mild, moderate or severe).

When the final evaluation is completed, the consensus of the three evaluations is formulated. Two evaluations of the same phenotype result in a consensus of that phenotype; three different evaluations (i.e., excellent, good and fair) result in a consensus of the middle phenotype. If the consensus is phenotypically normal (excellent, good or fair) an OFA registry number is assigned. The owner of record and the referring veterinarian are notified of the evaluation results. Dysplastic results are not in the public domain unless the owner of record gives explicit direction for the release of such information by initialing the appropriate space on the application form.

Normal results for dogs that are AKC registered and whose permanent ID was verified by the attending veterinarian at the time of x-ray are forwarded to AKC monthly.

The time it takes to obtain three independent evaluations, arrive at the consensus, and generate the final OFA report is dependent on a number of factors. It may take a week to 10 days for the film to arrive at OFA via the mail service. Depending on the case load it typically takes two to three weeks from the time that OFA receives radiographs to completion and mailing of the consensus report.

HIP JOINT CONFORMATION

The OFA consulting radiologists make subjective evaluations of the hip status

based on criteria previously described in **Figure 2**. Although the radiologists apply the criteria subjectively, a study demonstrated good correlation between the consensus grade assigned and two objective measurements used to assess hip phenotype. These measurements are percent coverage (PC) of the femoral head within the acetabulum and Norberg angle (NA), which also estimates degree of fit. The higher the numeric value the better the degree of fit. A retrospective study of OFA hip phenotypes by Tomlinson (2000) reported a distinct difference in both percent coverage and Norberg angle values between OFA hip grades and between breeds.

The following numerical values (*) for each OFA classification are averages derived from that study.

Excellent—This classification is assigned for superior hip conformation in comparison to other animals of the same age and breed. There is a deep seated ball (femoral head) which fits tightly into a well-formed socket (acetabulum) with minimal joint space width.
*PC=63% NA=110

Good—The most common normal grade reported (**Figure 5**) regardless of breed is slightly less than superior but a well-formed congruent hip joint is visualized. The ball fits well into the socket and good coverage is present.
*PC=58% NA=108

Fair—Assigned where minor irregularities in the hip joint exist. The hip joint space is wider than a good hip phenotype. This is due to the ball slipping slightly out of the socket, causing a minor degree of joint incongruency (called subluxation). There may also be slight inward deviation of the weight-bearing surface of the socket (dorsal acetabular rim) causing the socket to appear slightly shallow. This can also be a normal finding in some breeds, such as the Chinese Shar Pei, Chow Chow and Poodle.
*PC=49 NA=104

CATEGORIES INELIGIBLE FOR AN OFA HIP NUMBER

Borderline—There is no clear cut consensus among the radiologists to place the hip into a given category of normal or dysplastic. There is usually more incongruency present than the minor amount found in a fair, but there are no arthritic changes present that definitively diagnose the hip joint as dysplastic. There also may be bony changes present on any of the areas of the hip anatomy that cannot be accurately evaluated as either an abnormal arthritic change or a normal anatomic variant for that individual dog. To increase the accuracy of the diagnosis, it is recommended the radiographs be repeated at a later date (usually 6 months). This allows the radiologist to compare the initial film with the most recent film and assess for progressive changes that would be expected if the dog is dysplastic. Most dogs (over 50%) with this grade that show no interval change in hip conformation receive a normal hip rating upon resubmission, usually a fair hip phenotype.

Mild Hip Dysplasia—There is significant subluxation present wherein the ball is partially out of the socket, causing an incongruent and increased joint space. The socket is usually shallow, only partially covering the ball. There are usually no arthritic changes present with this classification. If the dog has other superior traits and/or a great deal of time and investment has been placed into training, there is an option to resubmit a radiograph when the dog is older so it can be reevaluated. Most dogs will remain dysplastic, showing progression of the disease with early arthritic changes. There are a few dogs, however, that show improved hip conformation with increasing age. Since HD is a chronic, progressive disease, the older the dog, the more accurate the diagnosis of HD (or lack of HD). At 2 years of age, the reliability for a radiographic diagnosis of HD is 95%, and the reliability steadily increases as the dog ages. Radiographs should definitely be resubmitted if they were initially taken during times of possible detrimental environmental effects such as periods of physical inactivity, or high hormone levels related to time of a heat cycle which could lead to a “false” diagnosis of mild hip dysplasia.
*PC=40% NA=97



Figure 5.

Good hip.



Figure 6

Moderate hip dysplasia.

Moderate HD (Figure 6)—There is significant subluxation present wherein the ball is barely seated into a shallow socket, causing joint incongruency. There are secondary arthritic bone changes, usually along the femoral neck and head (termed remodeling), acetabular rim changes (termed osteophytes or bone spurs), and various degrees of trabecular bone pattern changes (called sclerosis). Once arthritis is reported, there is only continued progression of arthritis over time, and the dog may or may not be lame. The onset of lameness is unpredictable and some dogs may go most of their lives without showing any signs of lameness whatsoever. *PC=30% NA=92

Severe HD—Assigned where radiographic evidence of marked dysplasia exists. There is significant subluxation present, where the ball is partially or completely out of a shallow socket. Like moderate HD, there are also significant secondary arthritic bone changes along the femoral neck and head, acetabular rim changes, and significant abnormal bone pattern changes. *PC=21% NA=83

In addition to assessing the dog/cat hip conformation, the veterinary radiologist reports other radiographic findings that could have familial, inherited causes, such as transitional vertebra or spondylosis. Transitional vertebra is a congenital malformation of the spine that occurs at the junctions of major divisions of the spine (usually at the thoracic and lumbar vertebral junction or the lumbar and sacral vertebral junction). Transitional vertebra take on anatomic characteristics of the two divisions of the spine between which it occurs. The most common transitional vertebra reported by OFA is in the lumbo-sacral area. Transitional vertebra are usually not associated with clinical signs and the dog/cat can be used in a breeding program, but the OFA recommends breeding to a dog/cat that does not have transitional vertebra.

Spondylosis is an incidental radiographic finding in which smooth new bone production is visualized on vertebral bodies at the intervertebral disc space margins. The new bone production can vary in extent from formation of small bone spurs to complete bridging of adjacent vertebral bodies. Spondylosis may occur secondary to spinal instability but often it is of unknown cause and clinically insignificant. A familial basis for its development has been reported. As with transitional vertebra, dogs/cats with spondylosis can be used in a breeding program, but again, OFA recommends breeding to an animal that does not have spondylosis.

THE EFFECT OF AGE AND THE USE OF PRELIMINARY RADIOGRAPHS FOR EARLY DETECTION OF HIP DYSPLASIA

Frequently breeders want early knowledge of the hip status on puppies/kittens in a given litter. This allows early selection of animals for use as show/performance/breeding animals or animals that would be best suited for pet homes. The OFA accepts preliminary consultation radiographs on puppies and kittens as young as 4 months of age for evaluation of hip conformation. If the dog or cat is found to be dysplastic at an early age, the economic loss from cost of training, handling, showing, etc., can be minimized and the emotional loss reduced. Preliminary radiographs are read by the OFA staff veterinary radiologist and are not sent to outside radiologists as are the 24-month-old examinations. The same hip conformation grading scheme is used.

The OFA has performed a retrospective analysis of the reliability of early radiographic evaluation for canine hip dysplasia, using information in their database obtained from the standard ventrodorsal radiographic projection. Corley (1997) reported on a population of over 2,000 dogs from the four breeds with the greatest number of OFA

TABLE 2: RELIABILITY OF NORMAL PRELIMINARY EVALUATIONS BY HIP GRADE

	Excellent	Good	Fair	Total
Number	71	1,369	360	1,800
No Change	71	1,340	277	1,688
Normal to Dysplastic	—	24	75	99
Normal to Boderline	—	5	8	13
Reliability	100%	97.9%	76.9%	93.8%
Confidence Level Upper	100%	98.5%	81.2%	94.8%
Confidence Level Lower	94.9%	96.9%	72.2%	92.6%

TABLE 3: RELIABILITY OF DYSPLASTIC PRELIMINARY EVALUATIONS BY HIP GRADE

	Mild	Moderate	Severe	Total
Number	390	38	1	429
No Change	329	37	1	367
Dysplastic to Normal	47	1	—	48
Dysplastic to Borderline	14	—	—	14
Reliability	84.4%	97.4%	100%	85.5%
Confidence Level Upper	87.8%	99.9%	—	88.7%
Confidence Level Lower	80.4%	86.2%	—	81.9%

TABLE 4: RELIABILITY OF NORMAL PRELIMINARY EVALUATIONS BY AGE

	< 6 mo.	7-12 mo.	13-18 mo.	Total
Number	278	714	808	1,800
No Change	249	670	769	1,688
Normal to Dysplastic	25	43	31	99
Normal to Boderline	4	1	8	13
Reliability	89.6%	93.8%	95.2%	93.8%
Confidence Level Upper	92.9%	95.5%	96.5%	94.8%
Confidence Level Lower	85.4%	91.8%	93.5%	92.6%

TABLE 5: RELIABILITY OF DYSPLASTIC PRELIMINARY EVALUATIONS BY AGE

	< 6 mo.	7-12 mo.	13-18 mo.	Total
Number	102	150	177	429
No Change	82	126	159	367
Dysplastic to Normal	18	15	15	48
Dysplastic to Borderline	2	9	3	14
Reliability	80.4%	84.0%	89.8%	85.5%
Confidence Level Upper	87.6%	89.5%	93.9%	88.7%
Confidence Level Lower	71.4%	77.1%	84.4%	81.9%

submissions (Labrador Retrievers, Rottweilers, German Shepherds and Golden Retrievers). The reliability of the preliminary evaluation (3 to 18 months) was determined by comparing the initial evaluation to a follow-up evaluation (> 24 months) of the same dog. The reliability of a normal preliminary hip joint phenotype was 100% for excellent, 97.9% for good and 76.9% for fair (**Table 2**). The reliability of a preliminary evaluation of canine hip dysplasia was 84.4% for mild, 97.4% for moderate and 100% for severe (**Table 3**). Reliability of preliminary evaluations increased significantly as age at the time of preliminary evaluation increased, regardless of whether dogs received a preliminary evaluation of normal phenotype or canine hip dysplasia (**Tables 4 & 5**).

For normal hip conformations, the reliability was 89.6% at 3-6-months, 93.8% at 7-12 months and 95.2% at 13-18 months for the four main breeds. Pooled data comparing preliminary OFA evaluations at various ages and in various breeds with final OFA evaluations at 24 months or older resulted in a similar reliability factor for preliminary evaluations of approximately 90%. The false positive rate (defined as a preliminary evaluation of HD for a dog with a follow-up evaluation of a normal phenotype) of OFA preliminary evaluations ≤ 6 months of age was 18%; and the false negative rate (defined as a preliminary evaluation of normal phenotype for a dog with a follow-up evaluation of hip dysplasia) of OFA preliminary evaluation ≤ 6 months of age was 9%. This suggests that OFA

preliminary evaluations of hip joint status in dogs are generally reliable. However, dogs that receive a preliminary evaluation of fair or mild hip joint conformation should be reevaluated at an older age (24 months).

JOINT LAXITY

Laxity is generally considered to be one of the earliest pathologic findings in HD. The fact that joint laxity plays a role, but is not the only factor, in development of hip dysplasia and its secondary changes of degenerative joint disease has been recognized for over 30 years.

Joint laxity (looseness of the joint) is a dynamic state that may not be determined by routine radiography. The joint may appear radiographically normal, but in actual use it may be loose.

Some dogs demonstrate abnormal laxity (subluxation) radiographically, but do not develop the more definitive degenerative changes of dysplasia.

Some dogs demonstrate radiographically tight hips, but later develop the degenerative changes of dysplasia.

In 2005 Chase identified quantitative trait loci (QTLs) associated with joint laxity but not osteoarthritis/degenerative joint disease and a separate QTL associated with osteoarthritis/degenerative joint disease.

Palpation of the hips to demonstrate looseness is not generally accepted as a single diagnostic feature of HD. Stress radiography using a fulcrum or wedge (placing an object between the thighs and bringing the stifles together to force the head of the femur out of the acetabulum) has been investigated as a technique to demonstrate the degree of radiographic subluxation that is possible. Some measurement criterion such as Norberg angle, millimeters of displacement, distraction index (DI), or dorsal lateral subluxation measurement (DLS) is usually employed to calculate the amount of displacement of the femoral head when compared to a fixed anatomic structure or to a standard radiograph taken without the fulcrum or wedge. The differences in the measurements indicate the range of possible motion or joint laxity. Different devices, measurements and positions have been developed at the University of Pennsylvania (PennHIP®), Cornell University and Michigan State University. Use of the fulcrum technique has demonstrated that some laxity is expected in the normal joint, but that many dogs with laxity beyond a certain amount later show the more definitive characteristic radiographic changes of dysplasia. The specific degree of laxity that is acceptable at a given age, and in various breeds of dogs and cats, has not been determined and represents a major unanswered question.

Table 6 is a comparison of different early screening procedures, and with the exception of palpation, all yield similar false-negative results (initially reporting a dog as normal that is later evaluated as dysplastic). There is, however, a major difference in the comparison of false-positive results (initially reporting a dog as dysplastic that is later evaluated as normal). A later publication by Lust (2001) suggested that the strength of the hip extended view (OFA view) is its specificity. Specificity refers to the ability to correctly identify dogs without hip dysplasia and this study also noted that this is dependent on the expertise of the evaluator.

The degree of joint laxity—as demonstrated by forcing the head of the femur away from the acetabula either by palpation or by using a fulcrum/stress device—that can be normal, and what degree is abnormal (eventually leading to degenerative joint changes) is unknown.

A primary reason this is unknown is that stress radiographic techniques measure artificially forced laxity in a non-weight bearing position. A report using such a device on 367 OFA normal dogs (excellent, good and

TABLE 6: FALSE-NEGATIVE AND FALSE-POSITIVE RESULTS FOR DYSPLASIA FROM FOUR STUDIES

Method	False-Negative	False-Positive
Palpation (1)	25%	33%
DI at 4 months @ .3 (2)	12%	48%
DI at 4 months @ .3 (3)	0%	45%
DI at 4 months @ .4 (3)	13%	43%
OFA Prelims @ < 6 months (4)	9%	18%

1 = Reviewed by Willis; 2 = Smith et al.; 3 = Lust et al.; 4 = Corley et al.

fair) indicated that 80% had a DI \geq 0.3 (Powers). Meaning that if PennHIP® employed a DI of 0.3 as their gold standard, approximately 80% of otherwise normal potential breeding dogs could be eliminated. Improved accuracy using laxity as the diagnostic finding might be possible with a technique that measures dynamic laxity (laxity that occurs during normal movement).

There is currently no explanation to account for adult animals with substantial joint laxity that do not develop degenerative joint disease.

There is no pathologic evidence available to determine what processes are occurring in the hips that are lax but do not develop degenerative joint disease, or in hips that are tight yet develop degenerative joint disease. Without this information there is a deficiency of necessary data to support breeding or treatment recommendations based on laxity alone. It is obvious that dogs with “tight” hips tend to be normal and those with markedly “loose” hips tend to be abnormal. What happens between the two extremes remains unknown. Further research using carefully controlled scientific methods is needed to understand the full implication of joint laxity.

However, breeders have a phenotypic screening method (standard hip extended radiograph) readily available that is safe, accurate, of modest cost and effective. As an example of effectiveness, Leighton reported that while the mean DI did not change, the incidence of hip dysplasia at The Seeing Eye Inc. was dramatically reduced over five generations using the standard hip extended position and a subjective hip score similar to OFA's. That breeding program also illustrates the importance of obtaining and considering information on the hip status of siblings as well as on the dam and sire with regard to selection of potential breeding animals.

PHYSICAL RESTRAINT OR CHEMICAL RESTRAINT

Chemical restraint permits easier, and as a rule more accurate, positioning and reduces potential radiation exposure risk to the patient and veterinary personnel. The types of chemical restraint, depth of general anesthesia, or use of manual restraint only are environmental variables that can affect the radiographic evaluation.

Preliminary OFA data indicates that chemical restraint does affect the radiographic appearance of the hip joints in some dogs. Current information, observations made on large numbers of dogs, and experience with follow-up studies on large numbers of dogs supports the recommendation that chemical restraint to the point of relaxation, or general anesthesia, be used. This appears to give a truer evaluation of the hip status, but more research is needed on this controversial subject, as there is an absence of controlled scientific data.

NUTRITION

Kasstrom, and later Kealy, reported that a higher than needed caloric intake during the rapid growth phase may result in earlier and more severe dysplastic changes when the genetic potential for dysplasia is present. Lower caloric intake may minimize or delay the evidence of dysplasia in the same dog, but will not change the genotype. Without genetic predisposition however, environmental influences alone will not create hip dysplasia.

There is no evidence in the scientific literature that megadoses of vitamin C (Bennett, 1987) or any other multi-vitamin/mineral supplement is beneficial in reducing the effects of or preventing hip dysplasia.

HORMONAL EFFECTS

Estrus appears to affect the reliability of diagnosis in some females. Some animals in season demonstrate a degree of subluxation (laxity) that is not present when the bitch is out of season, possibly due to the relaxation effects of estrogens on the ligaments and joint capsule. Radiography of these bitches may result in a false diagnosis of HD.

It is recommended that bitches not be examined for HD when in season and radiographs should be obtained one month prior or one month following the heat cycle. In addition, following a pregnancy the OFA recommends that the bitch's radiographs be taken at least one month after weaning the offspring.

PHYSICAL INACTIVITY

Periods of prolonged inactivity may affect the reliability of diagnosis. A few animals exhibit subluxation after prolonged periods of inactivity due to illness, weather conditions, etc. On later examination, when the animal is in good muscular tone, the hips appear normal. Therefore radiography is recommended when the animal is in good health and muscle tone.

IMPACT OF OFA HIP EVALUATIONS THROUGH MULTIPLE GENERATIONS

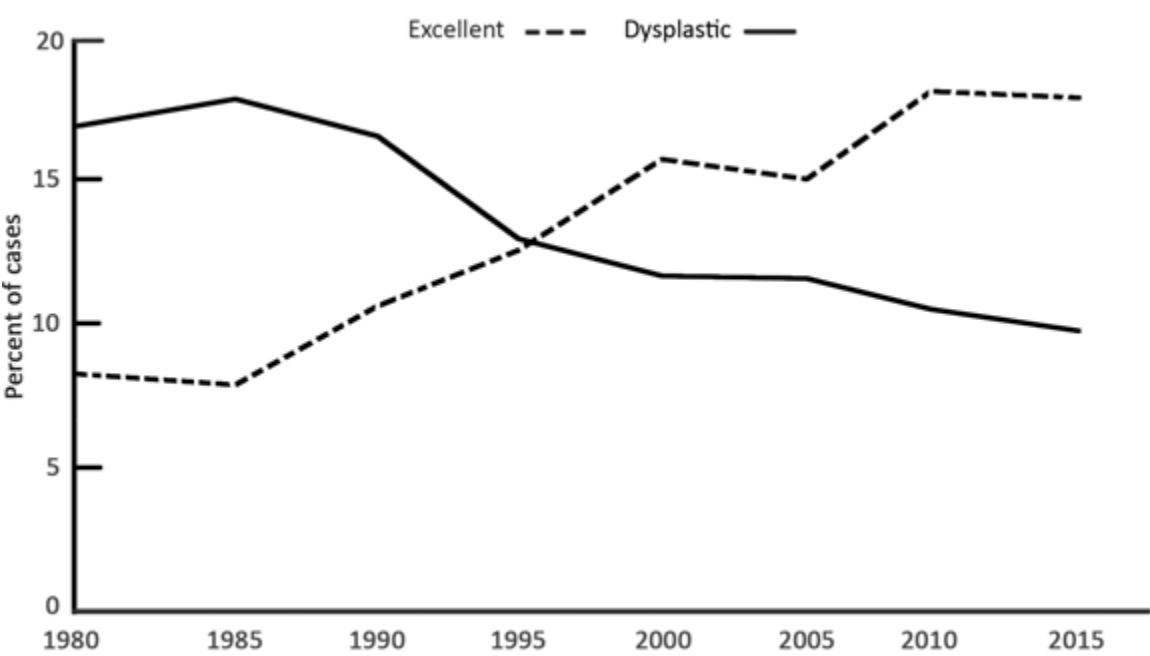
Retrospective studies covering the period of 1972–2003 have demonstrated steady and encouraging progress as a result of the collaborative efforts of responsible breeders and the OFA (Kaneene). The OFA database population represents a specific subset of the general population of animals, primarily show dogs and cats, and working/ hunting dogs. Accumulated data clearly illustrates the impact that the focused efforts of conscientious breeders can have on reducing the frequency of HD, and further indicates that the hip status of progeny follows that of parents (Table 1).

Success in reducing HD in a breed depends first on breeders recognizing that a problem exists. This must then be followed by a commitment to solve the problem and dedication to consistent use of a standard hip evaluation protocol.

HD has been reported in all breeds of dogs and some cat breeds that have been evaluated by the OFA. The OFA database is an important tool that can provide breeders with information regarding changes in hip status of specific breeds over time. The frequency of HD in most breeds has steadily declined. Concurrently, the percentage of animals with excellent hip conformation has steadily increased (Graph 1) in most breeds. Within the OFA population of animals with normal hip conformation, there has been a steady decrease in the percentage of fair and an increase in the percentage of excellent (Graph 2). Within the OFA population of dysplastic animals, there has been a steady decrease in the percentage of moderate with a leveling off in the percentage of mild dysplasia (Graph 3).

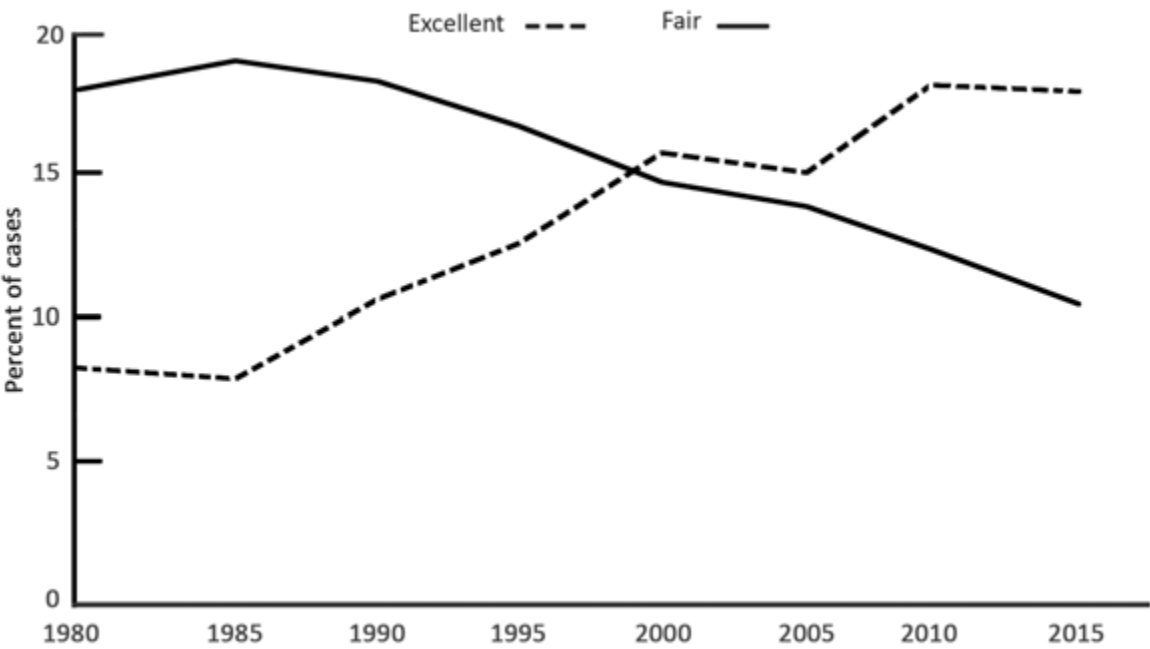
While this may be surprising to some, it is also important to realize that some of the smaller sized breeds and mixed breeds have as high a percentage of HD as the larger breeds and purebreds. Generalizations that claim that dysplasia is limited to, or more common in, large dogs and purebred dogs are misleading. HD appears to be perpetuated by breeder imposed breeding practices. However, when breeders and their breed clubs recognize HD as a problem and establish HD reduction as a priority, improvement of breed hip status can be accomplished without jeopardizing other desirable traits.

GRAPH 1: PERCENT DYSPLASTIC VS EXCELLENT BY BIRTH YEAR

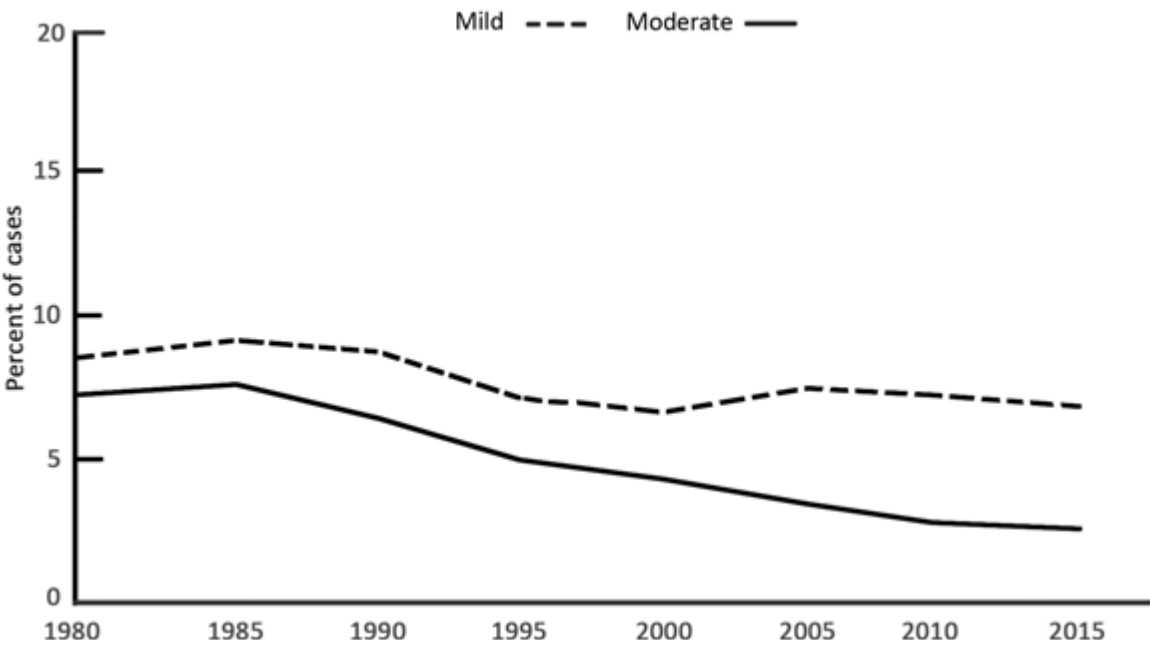


Although it is clear from the graphs that breeders have made steady progress toward reducing the frequency of hip dysplasia, some are concerned that this decline may reach a plateau. As with any polygenic disease, it is anticipated that HD will decline in an exponential manner. Therefore, after several generations it may appear that progress has leveled out. This is to be expected when phenotypic data is used to place selection pressure against polygenic disease traits with moderate to high heritability estimates. However, Leighton has shown that rapid progress can be expected in the first three or four generations, and is followed by slower but continued progress in subsequent generations. In the future a DNA-based genetic test might overcome this, but meanwhile breeders can continue to make significant progress by committing to careful selective breeding practices.

GRAPH 2: PERCENT EXCELLENT VS FAIR BY BIRTH YEAR



GRAPH 3: PERCENT MILD VS MODERATE BY BIRTH YEAR



LEGG-CALVE-PERTHES DATABASE

Legg–Calve–Perthes (LCP) disease, or avascular necrosis of the femoral head, is a disorder of the hip joint(s) which occurs in both humans and dogs. LCP is an inherited disease, but in most breeds the mode of inheritance is unknown. LCP is most often seen in miniature and toy breeds between 4 and 12 months of age. A decrease in vascularization to the immature femoral head results in death of chondrocytes beneath the articular cartilage, resulting in necrosis. Subsequent collapse of the affected area causes pain and lameness. In untreated dogs, revascularization can occur, resulting in a malformed femoral head and secondary degenerative joint disease (**Figure 7**).

OFA submission procedures are the same as for HD, outlined in the hip dysplasia section, except for the age requirement and restraint recommendations.

AGE

Only dogs 12 months of age or older at the time of radiography can qualify for an OFA LCP number. The hip joint status of younger animals will be evaluated, but only a preliminary consultation report will be issued.

RESTRAINT

Obtaining a properly positioned film may require chemical restraint, but with many small breed dogs it may be possible to obtain a well positioned image with the dog awake. The dog should not be fed on the day of radiography.

BREEDS AT RISK INCLUDE:

Affenpinscher	Fox Terrier	Poodle
Australian Terrier	Havanese	Pug
Bichon Frise	Lakeland Terrier	Schipperke
Border Terrier	Manchester Terrier	Scottish Terrier
Boston Terrier	Miniature Pinscher	Shetland Sheepdog
Cairn Terrier	Miniature Schnauzer	Silky Terrier
Chihuahua	Parson Russell Terrier	Welsh Terrier
Cocker Spaniel	Pekingese	West Highland White Terrier
Dachshund	Pomeranian	Yorkshire Terrier

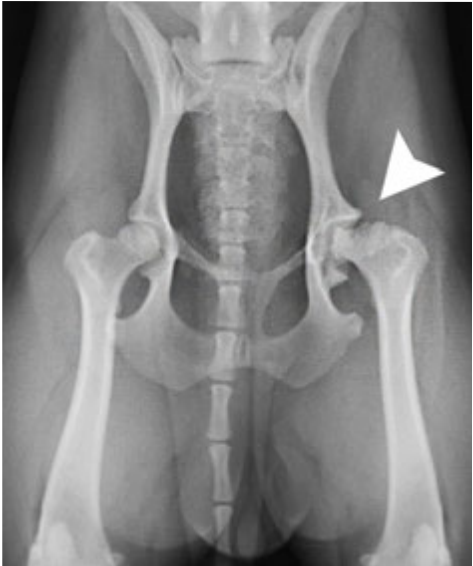


Figure 7
Note the collapse of the femoral head.

ELBOW DYSPLASIA DATABASE

Elbow dysplasia was originally described as a developmental disease manifested as degenerative joint disease (DJD) with or without an ununited anconeal process (UAP). Over time two other inherited diseases, osteochondrosis (OCD) and fragmented medial coronoid process (FCP), were identified as part of the DJD complex collectively referred to as elbow dysplasia.

ETIOLOGY

Multiple theories on the cause of these abnormalities have been proposed. Olsson suggested a unitarian theory that UAP, OCD and FCP were all due to osteochondrosis. Osteochondrosis is a disturbance in endochondral ossification (the process by which bone is formed from a cartilage mold). Osteochondrosis results from a reduction in nutrients to the chondrocytes of the cartilage mold beneath articular cartilage. This loss of chondrocytes produces a weakened foundation under the articular cartilage, resulting in fracturing of the cartilage.

Wind suggested that asynchronous growth of the ulna and radius, or insufficient development of the ulnar trochlear notch, results in abnormal loading forces on the anconeal process or medial coronoid process. Numerous studies suggest that the three diseases (UAP, OCD and FCP) are independent, inherited diseases.

CLINICAL PRESENTATION

The radiographic evidence of elbow dysplasia (ED), the presence of secondary degenerative joint disease (DJD), and the clinical presentation do not correlate directly. Grondalen reported on a population of 207 Rottweilers of which 141 were not lame. Yet 68% of the non-lame dogs had degenerative joint disease of the elbow. Another study by Read reported on serial radiographic and physical examination of 55 Rottweilers at 6 and 12 months of age. At 6 months of age the majority of lame dogs did not have radiographic evidence of ED; however, by 12 months of age the radiographic changes were apparent. But the majority of dogs in this population remained sound.

The elbow is a complex joint with overlapping osseous structures, which often makes a definitive diagnosis difficult, especially when dealing with pathology involving the medial coronoid process. To increase the probability of achieving an accurate diagnosis, the routine radiographic examination of the elbow (cranial-caudal and neutral medial-lateral projections) can be supplemented with the craniolateral caudomedial oblique and an extreme flexed mediolateral projection. Even then, a definitive diagnosis can be difficult without linear tomography, computerized tomography or surgical exploration of the joint.

OFA ELBOW PROTOCOL

The International Elbow Working Group, (IEWG) a consortium of experts from around the world, was founded in 1989 to lower the incidence of elbow dysplasia by coordinating worldwide efforts. The OFA started its elbow database in 1990 using a modified protocol of the IEWG. The diagnosis of elbow dysplasia is based on the presence of degenerative joint disease/osteoarthritis. Radiographically, the primary finding is sclerosis in the area of the trochlear notch and a periosteal response on the anconeal process which is best visualized on the extreme flexed mediolateral projection (**Figure 8**). Although in and of itself secondary degenerative joint disease is not an inherited disease, it is the end result found in dogs with elbow dysplasia.

Therefore, OFA requires one view of each elbow clearly labeled left and right in the extreme flexed medial-lateral position (**Figure 8**). Inclusion of additional views is at the discretion of the attending veterinarian. A permanent clearance can be obtained at 24 months of age, and dogs between 5 and 24 months of age can receive a preliminary evaluation. Non-grid, table top technique using high MaS and low Kvp is recommended (see hip dysplasia protocol).



Figure 8

Note the remodeling of the proximal surface of the anconeal process (upper arrow) and sclerosis in the area of the trochlear notch (lower arrow).

As with hip x-rays, to be eligible for OFA evaluation all radiographs submitted must include permanent animal identification in the film emulsion (traditional x-rays) or overlaid on the image (digital x-rays), using the dog's full registered name, if applicable, registration number OR microchip number/tattoo. Lead letters, radio opaque tape or digital overlay can be used to identify the film with: a) the hospital or veterinarian's name, b) date of radiograph and c) dog ID as noted above. Please see page 17, "General Procedures," for more information about radiographic procedures.

If the above required information is illegible or missing the OFA cannot accept the film for evaluation purposes, and the clinic will be contacted and asked to resubmit radiographs with the required information.

ELBOW CLASSIFICATIONS

The OFA reports elbows as normal or dysplastic. While there is no subdivision classification of normal, dysplastic elbows are graded 1 through 3, with grade 3 being the most severe. Differences between dysplastic grades are based on the severity of degenerative joint disease present.

Normal—No evidence of inherited pathologic change

Dysplastic

Grade 1—mild DJD; osteophytes less than 2 mm in height

Grade 2—moderate DJD; osteophytes 2 to 5 mm in height

Grade 3—severe DJD; osteophytes greater than 5 mm

There can be pathology involving the medial coronoid process without a distinct fracture fragment. As seen in **Figure 9** the malformed medial coronoid process and a fissure fracture of the articular cartilage could not be ascertained from the radiographic image, but created sufficient joint instability to produce secondary degenerative joint disease (**Figure 8**).

RATIONALE FOR SELECTIVE BREEDING

There are multiple studies supporting the theory that the various components of ED have a polygenic mode of inheritance. Further, it appears that environmental factors also contribute to expression of the disease. Selective breeding of phenotypically normal dogs has been shown to reduce the incidence of elbow dysplasia. In 1965, Corley reported on the inheritance of ununited anconeal process. Swenson reported on a study which included 4,515 dogs registered by the Swedish Kennel Club. As selective pressure was applied toward identifying and breeding dogs with normal elbows, there was a corresponding increase in the percentage of normal progeny.

There are a number of papers reporting on the inheritance of osteochondrosis and fragmented medial coronoid process. A report by Padgett classifies these as separate diseases that may occur alone or in combination. In this study, the initial breeding pair of Labrador Retrievers had surgically confirmed osteochondrosis and fragmented medial coronoid process in both elbows. The male dog was subsequently bred to two of his first and second generation daughters. There was a total of 31 progeny produced of which 83.9% had osteochondrosis, fragmented coronoid process or both.

Table 7 and **Figure 10** illustrate the outcome of matings based on information extracted from the OFA database. A total of 67,599 progeny were identified in which both parents had elbow dysplasia evaluations. The percentages of progeny with elbow dysplasia more than doubled if either parent had ED, and more than tripled if both parents had ED, as compared to when both parents were normal. Results of selective breeding practices indicate that elbow dysplasia should be considered in the moderate to high heritability estimate category (See Genetics section).

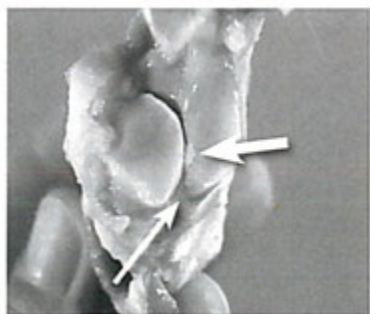
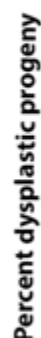


Figure 9

Irregularly formed medial coronoid process (thin arrow) and fracture of the articular cartilage (thick arrow). These changes are difficult, if not impossible, to visualize radiographically.

Scores on 67,599 progeny from sires and dams with known elbow scores.

Sire Rating



OSTEOCHONDROSIS (OCD) OF THE SHOULDER

While the exact mode of inheritance is unknown, osteochondrosis is considered to be an inherited disease. In affected individuals there is a disruption in ossification of the cartilage mold beneath the articular cartilage of the joint. This results in aseptic necrosis (cell death), and when the weakened area collapses the articular cartilage fractures, resulting in lameness and secondary degenerative joint disease/osteoarthritis.

OCD has been reported to occur most often in the shoulder, but can also be found in elbow, stifle, hock and spine and can be unilateral or bilateral. Most affected dogs that develop clinical signs are less than one year of age.

OCD is seen in many breeds but appears to be more common in breeds with larger torsos. It is also more frequent in males than females.

SHOULDER PROTOCOL

Radiographically, the primary finding is a radiolucent defect in the caudal articular surface of the humeral head (**Figure 11**). This flattening may be accompanied by sclerosis of adjacent bone and in some cases a cartilage flap may contain subchondral bone. Chronic cases may also have secondary DJD changes associated with glenoid fossa of the scapula and humeral head.

OFA requires one view of each shoulder clearly labeled left and right in the neutral medial-lateral position. The radiographs are required to contain permanent dog identification as noted under "Film Identification" in the Hip Dysplasia Database section of this book. The shoulder of interest should be pushed cranial to the thorax and the opposite shoulder pulled caudal. Inclusion of additional views is at the discretion of the attending veterinarian. A permanent clearance can be obtained at 12 months of age, and dogs between 4 and 12 months of age can receive a preliminary evaluation.



Figure 11

Note radiolucent defect in caudal articular surface of the humeral head.

SHOULDER CLASSIFICATIONS

Normal—No evidence of inherited pathologic change

Abnormal—Osteochondrosis (OCD)—radiographic changes consistent with inherited disease

Degenerative joint disease/osteoarthritis—the presence of degenerative joint disease without a definite diagnosis of, but probably due to, prior osteochondrosis.

EYE DATABASE

The OFA Companion Animal Eye Registry (CAER) provides breeders with information about eye diseases so that they may make informed breeding decisions. CAER registers those dogs seeking certification as free of heritable eye disease by board certified veterinary ophthalmologists (ACVO), and collects data on all dogs examined by ACVO Diplomates. The latter data are used to form the Clinical Research Database, which provides information on trends in eye disease and breed susceptibility. Clinicians and students of ophthalmology, as well as interested breed clubs and individual breeders and owners of specific breeds, will find this useful.

EVALUATION CRITERIA

OFA eye certification examinations are annual exams performed by board certified veterinary ophthalmologists. The exams can take place either in the veterinary office, or at a special clinic held in conjunction with another event (such as a dog show).

The CAER forms used for OFA eye certification are triplicate forms, and are only available in the ophthalmologists' offices. After the examination is completed, the pink copy will be retained by the ophthalmologist, the white copy will be given to the dog owner to submit for certification, and the third, yellow copy will be sent to the OFA for research purposes. Owners should bring their dogs' registration information, date of birth, breed/variety and a permanent form of ID (microchip number or tattoo) to the exam for use in filling out the CAER forms.

The eye certification exam is performed after drops are placed in the eyes to dilate the pupils. The exam consists of indirect ophthalmoscopy and slit lamp biomicroscopy. It is not a comprehensive ocular health examination, but rather an ocular screening exam. For example, eye certification exams do not entail measuring tear production, staining the eyes for the presence of corneal ulcers or measuring intraocular pressures. Gonioscopy, tonometry, Schirmer tear test, electroretinography and ultrasonography are not routinely performed; thus, dogs with goniodysgenesis, glaucoma, keratoconjunctivitis sicca, early lens luxation/subluxation or some early cases of progressive retinal atrophy might not be detected without further testing. If a serious ocular health problem (such as glaucoma) is suspected during the eye certification exam, the exam may need to be "converted" to a comprehensive ocular examination. The diagnoses obtained during an ophthalmic examination refer only to the phenotype (clinical appearance) of an animal. Thus it is possible for a clinically normal animal to be a carrier (abnormal genotype) of genetic abnormalities.



OFA eye certification examinations must be performed by board certified veterinary ophthalmologists.

The following breeds are recommended to have a preliminary examination prior to initial pharmacological dilation to best facilitate identification of these disorders:

Dalmatian—iris hypoplasia/sphincter dysplasia

Australian Shepherd—iris coloboma

Mastiff—persistent pupillary membrane

Basenji—persistent pupillary membrane

Pembroke Welsh Corgi—persistent pupillary membrane

Miniature Australian Shepherd and Toy Australian Shepherd—iris coloboma

Louisiana Catahoula Leopard Dog—iris coloboma, persistent pupillary membrane

GENETICS OF INHERITED EYE DISEASE

Genetic diseases are those that are passed on from parent to offspring through genes that carry the codes for each specific trait. Many of the diseases and disorders that affect the eyes have genetic factors. Some diseases are linked to a single gene pair and are either dominant, meaning it only requires one gene copy to express the trait, or recessive, which requires two copies of the gene. Other diseases are known to run in families but are affected by a number of genetic factors (more than one gene pair).

IDENTIFYING INHERITED EYE DISEASE

Although there are noteworthy exceptions, most of the ocular diseases of dogs which are presumed to be hereditary have not been adequately documented. Genetic studies require examination of large numbers of related animals in order to characterize the disorder (age of onset, characteristic appearance, rate of progression) and to define the mode of inheritance (recessive, dominant). In a clinical situation, related animals are frequently not available for examination once a disorder suspected to be inherited is identified in an individual dog. Maintaining a number of dogs for controlled breeding trials through several generations is a long and costly process. Both of these obstacles are compounded by the fact that many ocular conditions do not develop until later in life. Until the genetic basis of an ocular disorder is defined in a peer-reviewed published report, we rely on what statistical information is available from registry organizations, informed opinions and consensus from ACVO diplomates. We must satisfy ourselves with terms like “presumed inherited” and “suspected to be inherited.” Several companies provide information on genetic testing and greatly assist in providing more information and data to aid in defining the canine genetics of ocular diseases.

There are eye diseases in the dog for which there is evidence of a genetic or heritable cause. The American College of Veterinary Ophthalmologists has listed 10 of these diseases as automatic “fails” (this means the affected dog is ineligible to receive an eye certification) because of the significance of the condition to vision and/or the very strong evidence of heritability.

CONDITIONS THAT PREVENT ELIGIBILITY FOR OFA EYE CLEARANCE

There are currently 10 disorders for which there is an unequivocal recommendation against breeding in all breeds. These diagnoses also result in ineligibility for an OFA Eye Certification number. These are conditions which frequently result in blindness and for which there is definite evidence of heritability in one or more breeds. *Note: The prudent approach to these disorders is to assume they are hereditary except in cases specifically known to be associated with trauma, other causes of ocular inflammation, specific metabolic diseases or nutritional deficiencies.

1. *Keratoconjunctivitis sicca (KCS)*—Breeding is not recommended for any animal demonstrating keratitis consistent with KCS. The prudent approach is to assume KCS to be hereditary except in cases suspected to be non-genetic in origin. See above *note.
2. *Cataract*—Breeding is not recommended for any animal demonstrating partial or complete opacity of the lens or its capsule unless the examiner has also checked the space for “significance of above cataract unknown” or unless specified otherwise for the particular breed. See above *note.
3. *Lens luxation or subluxation*—see above *note.
4. *Glaucoma*—see above *note.
5. *Persistent hyperplastic primary vitreous*
6. *Retinal detachment*—see above *note.
7. *Retinal dysplasia*—geographic or detached forms see above *note.
8. *Optic nerve coloboma*
9. *Optic nerve hypoplasia*
10. *Progressive Retinal Atrophy (PRA)*—Breeding is not advised for any animal demonstrating bilaterally symmetric retinal degeneration (considered to be PRA unless proven otherwise).

BREEDER OPTION CODES

Two categories of advice regarding breeding have been established:

No: Substantial evidence exists to support the heritability of this entity AND/OR the entity represents a potential compromise of vision or other ocular function. (See Conditions That Prevent Eligibility for OFA Eye Clearance)

Breeder Option: Entity is suspected to be inherited but does not represent potential compromise of vision or other ocular function. Please note: although the dog will "pass," it will have additional documentation on its OFA eye certificate with a category listing the problem.

When the breeding advice is "No," even a minor clinical form of the entity would make this animal unsuitable for breeding. When the advice is "Breeder Option," caution is advised. In time it may be appropriate to modify this stand to "No" based on accumulated evidence. If it becomes apparent that there is insufficient evidence that an entity is inherited, it may be deleted from the list.

PATELLAR LUXATION DATABASE

The patella, or kneecap, is part of the stifle joint (knee). In patellar luxation the kneecap luxates, or pops out of place, either in a medial or lateral position. Bilateral involvement is most common, but unilateral patellar luxation is not uncommon. Animals can be affected by the time they are 8 weeks of age. The most notable finding is a knock-knee (genu valgum) stance. The patella is usually reducible, and laxity of the medial collateral ligament may be evident. The medial retinacular tissues of the stifle joint are often thickened, and the foot can be seen to twist laterally as weight is placed on the limb.

GENERAL PROCEDURES

Purpose—To identify those dogs that are phenotypically normal prior to use in a breeding program, and to gather data on the genetic disease of patellar luxation.

Examination and classification—Each dog is to be physically examined awake and classified by an attending veterinarian according to the general information instructions.

Clearance issued—A breed registry number will be issued for all dogs found to be normal at 12 months of age or older. There is an initial OFA fee and no charge for recertification at a later age. The breed registry number will contain the age at evaluation, and it is recommended that dogs be periodically reexamined as some luxations will not be evident until later in life.

Preliminary evaluation—Evaluation of dogs less than 12 months of age is encouraged if the owner desires to breed at this age. A very opportune time to gather this data is at 6-8 weeks of age, prior to the puppy's release to the new owner.

Dogs with patellar luxation—The attending veterinarian and owner are encouraged to submit all evaluations, whether normal or abnormal, to help assure accuracy of the database. There is no OFA fee for entering an abnormal evaluation of the patella in the data bank. Abnormal information will not be released into the public domain unless the owner gives permission for this release by initialing the appropriate line on the application form.

CLASSIFICATIONS

A method of classifying the degree of luxation and bony deformity is useful for diagnosis, and can be applied to either medial or lateral luxations by reversing the medial-lateral directional references. The position of the patella can easily be palpated starting at the tibial tubercle and working proximal along the patellar ligament to the patella.

Grade 1

The patella easily luxates manually at full extension of the stifle joint, but returns to the trochlea when released. No crepitation is apparent. The medial, or very occasionally, lateral deviation of the tibial crest (with lateral luxation of the patella) is only minimal, and there is very slight rotation of the tibia. Flexion and extension of the stifle is in a straight line with no abduction of the hock.

Grade 2

There is frequent patellar luxation, which in some cases becomes more or less permanent. The limb is sometimes carried, although weight bearing routinely occurs with the stifle remaining slightly flexed.

Especially under anesthesia it is often possible to reduce the luxation by manually turning the tibia laterally, but the patella reluxates with ease when manual tension of the joint is released. As much as 30 degrees of medial tibial torsion and a slight medial deviation of the tibial crest may exist. When the patella is resting medially the hock is slightly abducted. If the condition is bilateral, more weight is thrown onto the forelimbs.

Many cases in this grade live with the condition reasonably well for many years, but the constant luxation of the patella over the medial lip of the trochlea causes erosion of the articulating surface of the patella and the proximal area of the medial lip. This results in crepitation becoming apparent when the patella is luxated manually.

Grade 3

The patella is permanently luxated with torsion of the tibia and deviation of the tibial crest of between 30 degrees and 50 degrees from the cranial/caudal plane. Although the luxation is not intermittent, many animals use the limb with the stifle held in a semi flexed position. The trochlea is very shallow or even flattened.

Grade 4

The tibia is medially twisted and the tibial crest may show further deviation medially with the result that it lies 50 degrees to 90 degrees from the cranial/caudal plane. The patella is permanently luxated. The patella lies just above the medial condyle and a space can be palpated between the patellar ligament and the distal end of the femur. The trochlea is absent or even convex. The limb is carried, or the animal moves in a crouched position with the limb partly flexed.

PATELLAR LUXATION CATEGORIES

Patellar luxations fall into several categories:

1. Medial luxation in toy, miniature, and large breeds.
2. Lateral luxation in toy and miniature breeds.
3. Lateral luxation in large and giant breeds.
4. Luxation resulting from trauma in any breed, and of no importance to the certification process.

Categories 1, 2 and 3 are either known to be heritable or strongly suspected to be heritable.

MEDIAL LUXATION IN TOY, MINIATURE AND LARGE BREEDS

These luxations are often termed “congenital” because they occur early in life and are not associated with trauma. Although the luxation may not be present at birth, the anatomical deformities that cause these luxations are present at that time and are responsible for subsequent recurrent patellar luxation. Patellar luxation in these dogs should be considered an inherited disease. Medial luxation is far more common than lateral luxation in all breeds, representing 75-80% of cases, with bilateral involvement seen 20-25% of the time.

Clinical signs

Three classes of patients are identifiable:

1. Neonates and older puppies often show clinical signs of abnormal hind-leg carriage and function from the time they start walking; these generally present as grades 3 and 4.
2. Young to mature animals with grade 2 to 3 luxations usually have exhibited abnormal or intermittently abnormal gaits all their lives, but are presented when the problem symptomatically worsens.
3. Older animals with grade 1 and 2 luxations may exhibit sudden signs of lameness because of further breakdown of soft tissues as a result of minor trauma or because of worsening of degenerative joint disease pain.

Signs vary dramatically with the degree of luxation. In grades 1 and 2, lameness is evident only when the patella is in the luxated position. The leg is carried with the stifle joint flexed but may be touched to the ground every third or fourth step at fast gaits. Grade 3 and 4 animals exhibit a crouching, bowlegged stance (*genu varum*) with the feet turned inward and with most of the weight transferred to the front legs. Permanent luxation renders the quadriceps ineffective in extending the stifle. Extension of the stifle will allow reduction of the luxation in grades 1 and 2. Pain is present in some cases, especially when chondromalacia of the patella and femoral condyle is present. Most animals, however, seem to show little irritation upon palpation.

LATERAL LUXATION IN TOY AND MINIATURE BREEDS

Lateral luxation in small breeds is most often seen late in the animal's life, from 5 to 8 years of age. The heritability is unknown. Skeletal abnormalities are relatively minor in this syndrome, which seems to represent a breakdown in soft tissue in response to obscure skeletal derangement. Thus, most lateral luxations are grades 1 and 2, and the bony changes are similar to, but opposite, those described for medial luxation. The dog has more functional disability with lateral luxation than with medial luxation.

Clinical signs

In mature animals, signs may develop rapidly and may be associated with minor trauma or strenuous activity. A knock-knee or genu valgum stance, sometimes described as seal-like, is characteristic. Sudden bilateral luxation may render the animal unable to stand and so simulate neurological disease. Physical examination is as described for medial luxation.

LATERAL LUXATION IN LARGE AND GIANT BREEDS

Also called genu valgum, this condition is usually seen in the large and giant breeds, with Great Danes, St. Bernards and Irish Wolfhounds being the most commonly affected. Components of hip dysplasia, such as coxa valga (increased angle of inclination of the femoral neck) and increased anteversion of the femoral neck, are related to lateral patellar luxation. These deformities cause internal rotation of the femur with lateral torsion and valgus deformity of the distal femur, which displaces the quadriceps mechanism and patella laterally.

Clinical signs

Bilateral involvement is most common. Animals appear to be affected by the time they are 5 to 6 months of age. The most notable finding is a knock-knee (genu valgum) stance. The patella is usually reducible, and laxity of the medial collateral ligament may be evident. The medial retinacula tissues of the stifle joint are often thickened, and the foot can often be seen to twist laterally as weight is placed on the limb.

CANINE THYROID DATABASE

Autoimmune thyroiditis is the most common cause of primary hypothyroidism in dogs. The disease has variable onset, but tends to clinically manifest itself at 2 to 5 years of age. Dogs may be clinically normal for years, only to become hypothyroid at a later date. The markers for autoimmune thyroiditis, autoantibody formation (autoantibodies to thyroglobulin, T4 or T3), usually occur prior to the occurrence of clinical signs. The majority of dogs that develop autoantibodies have them by 3 to 4 years of age. Development of autoantibodies at any time in the dog's life is an indication that the dog probably has the genetic form of the disease. Using current technology, only a small fraction of false positive tests occur.

As a result of the variable onset of the presence of autoantibodies, periodic testing is necessary. Dogs that are negative at 1 year of age may become positive at 6 years of age. Dogs should be tested every year or two to be certain they have not developed the condition. Since the majority of affected dogs will have autoantibodies by 4 years of age, annual testing for the first four years is recommended. After that, testing every other year should suffice. Unfortunately, a negative test at any one time will not guarantee that the dog will not develop thyroiditis.

This data can be used by breeders in determining which dogs are best for their breeding programs. Knowing the status of the dog and the dog's lineage, breeders and genetic counselors can decide which breedings are most appropriate for reducing the incidence of autoimmune thyroiditis in the offspring. The Animal Health Diagnostic Laboratory at Michigan State University has the largest pooled database on breed prevalence of autoimmune thyroiditis.

GENERAL PROCEDURES

Purpose—To identify those dogs that are phenotypically normal for breeding programs, and to gather data on the genetic disease of autoimmune thyroiditis.

Clearance issued—A breed registry number will be issued for all dogs found to be normal at 12 months of age or older. Age will be noted on the certificate since the classification can change as the dog ages. There is an initial OFA fee and no charge for recertification at a later date. It is recommended that re-examination occur at ages 2, 3, 4, 6 and 8 years of age.

Preliminary evaluation—Dogs less than 12 months of age can be evaluated for the owner's information, however few dogs are positive at that age.

Dogs with autoimmune thyroiditis—All data, whether normal or abnormal, should be submitted to help assure accuracy of the database. There is no OFA fee for entering an abnormal evaluation of the thyroid into the data bank. Abnormal information will not be released into the public domain unless the owner gives permission for this release by initialing the appropriate line on the application form.

Thyroid abnormalities fall into several categories, and two types are defined by the registry:

- Autoimmune Thyroiditis—Known or suspected to be heritable.
- Idiopathic Hypothyroidism—Of unknown origin.

Dogs with laboratory results that are not definitive will be considered as equivocal. It is recommended that the test be repeated in three to six months.

CLASSIFICATION

Thyroid classifications are based on the most current and scientifically validated tests available.

Indices of thyroiditis

Free T4 (FT4)—This test is considered to be the "gold standard" for assessment of the thyroid's production and cellular availability of thyroxine. FT4 concentration is expected to be decreased in dogs with thyroid dysfunction due to autoimmune thyroiditis.

Canine thyroid stimulating hormone (cTSH)—This test helps determine the site of the lesion in cases of hypothyroidism. In autoimmune thyroiditis, thyroid gland function is reduced, while the pituitary gland continues to function normally. Therefore, when FT4 levels fall due to a malfunctioning thyroid gland, the pituitary pro-

duces elevated levels of cTSH in an attempt to stimulate thyroid production. Thus, the cTSH concentration is expected to be abnormally elevated in dogs with thyroid atrophy from autoimmune thyroiditis.

Thyroglobulin Autoantibodies (TgAA)—This test measures the level of thyroid autoantibodies (antibodies directed against normal body tissue). Positive levels of thyroid antibodies are an indication that an autoimmune process is damaging the dog's thyroid gland.

- | | |
|--|--|
| <p>a. Normal
FT4 within normal range
cTSH within normal range
TgAA is negative</p> <p>b. Positive autoimmune thyroiditis
FT4 less than normal range
cTSH greater than normal range
TgAA is positive</p> <p>c. Positive compensative autoimmune thyroiditis
FT4 within normal range
cTSH greater than or equal to normal range
TgAA positive</p> | <p>d. Idiopathically reduced thyroid function
FT4 less than normal range
cTSH greater than normal range
TgAA negative</p> <p>e. All other results are considered equivocal</p> |
|--|--|

LABORATORY CERTIFICATION

In an attempt to standardize testing methodology, laboratories are required to pass a certification process. Laboratories may apply, and if successful will be approved to perform analysis for OFA thyroid certification.

The following laboratories are approved, and should be contacted directly for the appropriate submission forms (other than the OFA application form), sample handling procedures and laboratory service fee before collecting the sample. Contact information is always subject to change; for the most current list of available laboratories, visit www.ofa.org.

Animal Health Diagnostic Center (AHDC)

Endocrinology Laboratory, Cornell University, 240 Farrier Rd., Ithaca, NY, 14853; (607) 253-3673

Animal Health Laboratory

Laboratory Services Division, University of Guelph, Specimen Reception, 419 Gordon St., NW corner Gordon/McGill-vray St., Guelph, Ontario, N1G 2W1, Canada; (519) 824-4120 x 54530, Fax (519) 827-0961

Antech Diagnostics*

1111 Marcus Ave., Suite M28, Lake Success, NY, 11042; 1-800-872-1001

**Only the Lake Success, NY, location of Antech has been certified to process OFA thyroid panels.*

Michigan State University Veterinary Diagnostic Laboratory

4125 Beaumont Rd., Room 122, Lansing, MI, 48910; (517) 353-1683, Fax (517) 353-5096

IDEXX

1345 Denison St., Markham, Ontario, L3R 5V2, Canada; 1-800-667-3411

Texas A&M Veterinary Medical Diagnostic Laboratory

483 Agronomy Rd., College Station, TX, 77840; (979) 845-3414

**Submissions to this laboratory should not include OFA payment; OFA will invoice the owner.*

University of California-Davis Veterinary Medical Teaching Hospital

Central Laboratory Receiving, VMTH Room 1033, 1 Garrod Dr., Davis, CA, 95616; (530) 752-VMTH, Fax (530) 752-5055

INSTRUCTIONS FOR TESTING

1. The veterinarian or owner must obtain the "Application for Thyroid Database" from OFA (phone 573-442-0418) or online at www.ofa.org/veterinarian/application-forms.
2. The veterinarian and owner must complete their respective portions of the form.
3. Two milliliters (2 ml) of serum are needed for testing, and the serum sample must be from freshly collected blood. Use a plain "red-top" tube for blood collection. Do not use a serum separator tube with clot additives or any other type of plasma collection tube.
4. After collection, place the blood sample in the refrigerator for 60 to 90 minutes to allow clotting. Centrifuge, collect the serum, and transfer to a plain plastic or glass tube suitable for shipping. Clearly label the sample with the owner's name, animal's identification, date of blood collection and "OFA Thyroid Panel." If the specimen is to be stored for more than 12 hours prior to shipping, frozen storage is recommended.
5. Ship to the approved laboratory of choice via an overnight courier service. It is recommended that all specimens be packaged properly and shipped so they are received either chilled or frozen. Serum samples arriving unchilled or at room temperature within 48 hours of the collection date will be accepted. However, samples arriving after this time must be received either chilled or frozen in order to be accepted for registry testing. Contact the laboratory for further information as necessary.
6. Overnight fasting prior to the blood draw is preferred.
7. Female dogs should not be tested during an estrus cycle.
8. The date of last routine vaccination should be noted and dogs should not be tested within 30 to 60 days of vaccination.
9. Please do not submit whole blood, clotted blood or plasma.
10. Severely lipemic or hemolyzed specimens are also unacceptable.
11. Test results will be mailed or faxed only to the submitting veterinarian and the Orthopedic Foundation for Animals, Inc. Results will not be available from the laboratory by telephone. The testing laboratory will report results to the veterinarian, and OFA will send a report to the owner.

SEBACEOUS ADENITIS DATABASE

Sebaceous Adenitis (SA) is a hereditary skin disease in which the sebaceous glands become inflamed, often leading to progressive loss of hair. Diagnosing SA can be difficult as the symptoms vary by breed, the symptoms are similar to those of other diseases such as hypothyroidism or allergies, and the disease can vary greatly in its severity. Visible symptoms include excessive dandruff or scaling, hair loss, lesions, a musty odor and even secondary skin infections. On the other hand, dogs affected with SA can be subclinical and show no outward signs of the disease. As a result, diagnosis requires microscopic examination of tissue samples. The disease is primarily seen in Standard Poodles, Akitas and Samoyeds, although there have been reported cases in a number of other breeds and mixed breeds as well.

Two factors make SA particularly difficult for breeders to control: the possible late onset of the disease, and the subclinical state of the disease. With late onset, the dog may have already been bred long before it ever shows clinical signs of the disease. In its subclinical state, an owner may be unaware that the animal is affected since it shows no visible signs of the disease.

Sebaceous adenitis is believed to be a simple autosomal recessive in its mode of inheritance. The challenge in controlling the disorder is in identifying dogs as clear, carriers or affecteds. DNA testing remains the “gold standard” in terms of identifying a dog’s genotype, however, at present there is no DNA test to determine a dog’s status with regard to SA. Today’s best alternative is the phenotypic evaluation through the skin biopsy. As enough phenotypic information on families of dogs is entered into the database, breeders will be able to make educated assumptions on a dog’s genotype. This will allow breeders to apply greater selective pressure in controlling and reducing the incidence of the disease.

GENERAL PROCEDURES

Purpose—To identify phenotypically normal dogs prior to breeding, and to gather data on the genetic disease of sebaceous adenitis.

Examination—Dogs are to be screened using skin biopsies examined by a dermatopathologist. (See Examination Procedures below.)

Clearance issued—A breed registry number will be issued for all dogs found to be normal at 12 months of age or older. There is an initial OFA fee and no charge for recertification at a later age. The breed registry number will contain the age at examination and it is recommended that dogs be periodically re-examined as some affected dogs may not be evident until later in life.

Dogs with sebaceous adenitis—There is no OFA fee for entering an abnormal examination into the sebaceous adenitis data bank. Abnormal information will not be released into the public domain unless the owner gives permission by initialing the appropriate line on the application form.

EXAMINATION PROCEDURES

The attending veterinarian examines the dog for clinical symptoms of the disease and notes any findings on the application form. A minimum of two 6mm punch biopsy samples are taken from the skin of the dog’s neck between the top of the head and the withers (**Figure 12**). If there are areas of scaling and hair loss, samples should be taken from those areas.

To procure the sample, a local anesthetic such as lidocaine may be used. The area should not be scrubbed or otherwise cleaned, however gentle clipping of the area may be necessary. The specimen should not be squeezed with the forceps while placing in a crush-proof container containing 10% buffered formalin in preparation for shipment to the lab.

The biopsy sites may be closed with one or two sutures. The sample, the completed OFA application, and both the lab fee and

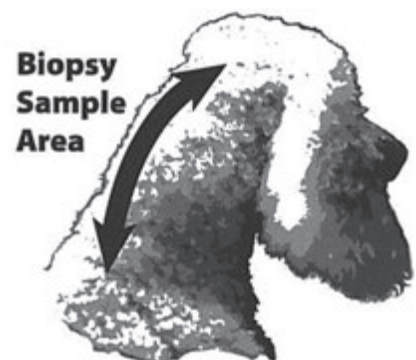


Figure 12

OFA fees are shipped to one of the approved dermapathology labs for evaluation. The lab results and final diagnosis are returned to the OFA and to the owner.

CLASSIFICATIONS

There are several classifications:

Normal—no evidence of sebaceous adenitis at the time of the examination.

Equivocal—some inflammation is present, but the cause cannot be determined.

Affected—dogs with clinical signs and histopathologic evidence of inflammatory skin disease with destruction of hair follicles, especially the sebaceous glands.

APPROVED DERMATHOLOGISTS/LABORATORIES

For the most current list of available laboratories, see the OFA website (www.ofa.org/diseases/other-diseases/seba-ceous-adenitis).

CARDIAC DATABASE

The OFA maintains two separate and distinct cardiac databases: the Congenital Cardiac Database and the Advanced Cardiac Database. Their purpose is to gather data regarding heart diseases in dogs, and to identify dogs which are phenotypically normal prior to use in a breeding program. For the purposes of the registry, a phenotypically normal dog is defined as:

- One without a cardiac murmur.
- One with an innocent heart murmur that is found to be otherwise normal by virtue of an echocardiographic examination which includes Doppler studies.

CONGENITAL CARDIAC DATABASE

Examination and classification—Each dog is to be examined and classified by a veterinarian with expertise in the recognition of canine heart disease, in accordance with procedures outlined under Identification and Classification.

Clearance issued—A breed registry number will be issued for any dog found to be normal for congenital cardiac disease at 12 months of age or older. The exam must include auscultation. There is an initial OFA fee and no charge for recertification at a later age. The breed registry number will indicate the age at evaluation and the type of examiner (C-cardiologist, S-specialist or P-practitioner).

Preliminary evaluation—Dogs under 12 months of age can be evaluated for the owner's information. The most opportune time to gather this data is at 8–10 weeks of age, prior to the puppy's release to the new owner.

Dogs with heart disease—The veterinarian and owner are encouraged to submit all evaluations, whether normal or abnormal, to help assure accuracy of the database and to assist in the analysis of patterns of inheritance in important canine congenital heart disease. There is no OFA fee for entering an abnormal cardiac evaluation into the database. Abnormal information will not be released into the public domain unless the owner gives permission for this release by initialing the appropriate line on the application form.

ADVANCED CARDIAC DATABASE

Examination and classification—Each dog is to be examined and classified by a veterinary cardiologist.

Veterinary cardiologists are defined as licensed veterinarians with diplomate status in either the American College of Veterinary Internal Medicine (ACVIM) cardiology sub-specialty, or the European College of Veterinary Internal Medicine (ECVIM) cardiology sub-specialty.

Clearance issued—The Advanced Cardiac Database examination results in a two-tiered clearance: congenital cardiac disease and adult-onset cardiac disease. A breed registry number will be issued for any dog found to be normal for cardiac disease (congenital disease and/or adult-onset disease) at 12 months of age or older. The congenital clearances are considered permanent. The adult-onset clearances are valid for one year from the date of the exam. In order for an adult-onset clearance to remain current, exams must be repeated periodically. The exam must include auscultation at a minimum. Echocardiograms may be recommended following the auscultation results, or for breeds susceptible to adult-onset cardiac diseases requiring an echo for an accurate diagnosis. Additionally, for an adult-onset clearance, Boxers and Doberman Pinschers require a Holter test within 90 days of the cardiologist's examination.

Preliminary evaluation—Dogs under 12 months of age can be evaluated for the owner's information. The most opportune time to gather this data is at 8 to 10 weeks of age, prior to the puppy's release to the new owner. Preliminary exams do not result in OFA certification.

Dogs with congenital or adult-onset heart disease—The veterinarian and owner are encouraged to submit all evaluations, whether normal or abnormal, to help assure accuracy of the database and to assist in the analysis of patterns of inheritance in important canine congenital and adult-onset heart disease. There is no OFA fee for entering an abnormal cardiac evaluation into the database. Abnormal information will not be released into the public domain unless the owner gives permission for this release by initialing the appropriate line on the application form.

IDENTIFICATION AND CLASSIFICATION

General Instructions

Congenital heart disease in dogs is a malformation of the heart or great vessels. The lesions characterizing congenital heart defects are present at birth and may develop more fully during perinatal and growth periods. Many congenital heart defects are thought to be genetically transmitted from parents to offspring; however, the exact modes of inheritance have not been precisely determined for all cardiovascular malformations.

The most common congenital cardiovascular defects can be grouped into several anatomic categories. These anatomic diagnoses include:

- **Malformation of the atrioventricular valves.**
- **Malformation of ventricular outflow leading to obstruction of blood flow.**
- **Defects of the cardiac septa (shunting defects).**
- **Abnormal development of the great vessels or other vascular structures.**
- **Complex, multiple, or other congenital disorders of the heart, pericardium, or blood vessels.**

Adult-onset cardiac diseases are also considered to have a genetic component and include, for example, hypertrophic and dilated cardiomyopathy; pulmonic and subaortic stenosis; and mitral and tricuspid valve dysplasia.

A careful clinical examination that emphasizes cardiac auscultation is the most expedient and cost-effective method for identifying heart disease in dogs. While there are exceptions, virtually all common congenital heart defects are associated with the presence of a cardiac murmur. Consequently, it is recommended that cardiac auscultation be the primary screening method for initial identification of heart disease and the initial classification of dogs.

Murmurs related to heart disease may at times be difficult to distinguish from normal, innocent (also called physiologic or functional) murmurs. Innocent cardiac murmurs are believed to be related to normal blood flow in the circulation. Innocent murmurs are most common in young, growing animals. The prevalence of innocent heart murmurs in mature dogs (especially in athletic dogs) is undetermined. A common clinical problem is the distinction between innocent murmurs and murmurs arising from heart disease.

Definitive diagnosis of heart disease usually involves one or more of the following methods:

- Echocardiography with Doppler studies.
- Cardiac catheterization with angiocardiography.
- Post-mortem examination of the heart (necropsy).

Other methods of cardiac evaluation, including electrocardiography and thoracic radiography, are useful in evaluating individuals with heart disease, but are neither sufficiently sensitive nor specific enough to reliably identify or exclude the presence of heart disease.

- The non-invasive method of echocardiography with Doppler is the preferred method for establishing a definitive diagnosis in dogs when heart disease is suspected from the clinical examination. Echocardiography is an inappropriate screening tool for the identification of congenital heart disease and should be performed only when the results of clinical examinations suggest a definite or potential cardiovascular abnormality.
- Two-dimensional echocardiography provides an anatomic image of the heart and blood vessels. While moderate to severe cardiovascular malformations can generally be recognized by two-dimensional echocardiography, mild defects (which are often of great concern to breeders of dogs) may not be identifiable by this method alone.
- Doppler studies, including pulsed-wave and continuous-wave spectral Doppler, and two-dimensional color Doppler demonstrate the direction and velocity of blood flow in the heart and blood vessels. Abnormal patterns of blood flow are best recognized by Doppler studies. Results of Doppler studies can be combined with those of the two-dimensional echocardiogram in assessing the severity of heart disease. Color Doppler echocardiography is used to evaluate relatively large areas of blood flow and is beneficial

in the overall assessment of the dog with suspected heart disease. Turbulence maps employed in color Doppler imaging are useful for identifying high velocity or disturbed blood flow but are not sufficiently specific (or uniform among manufacturers) to quantify blood velocity. It is emphasized that quantification of suspected blood flow abnormalities is essential and can only be accomplished with pulsed- or continuous-wave Doppler studies. Pulsed-wave and continuous-wave Doppler examinations provide a display of blood velocity spectra in a graphical format and are the methods of choice for assessing blood flow patterns and blood flow velocity in discrete anatomic areas.

- Cardiac catheterization is an invasive method for identification of heart disease that is considered very reliable for the diagnosis of heart disease. Cardiac catheterization should be performed by a cardiologist, usually requires general anesthesia, carries a small but definite procedural risk, and is generally more costly than non-invasive studies. While cardiac catheterization with angiocardiology is considered one of the standards for the diagnosis of heart disease, this method has been supplanted by echocardiography with Doppler for routine evaluation of suspected heart disease.
- Necropsy examination of the heart should be done in any breeding dog that dies or is euthanized. The hearts of puppies and dogs known to have cardiac murmurs should always be examined following the death of the animal. A post mortem examination of the heart is best done by a cardiologist or pathologist with experience in evaluating heart disease. While it is obvious that necropsy cannot be used as a screening method, the information provided by this examination can be useful in guiding breeders and in establishing the modes of inheritance of heart disease.

Each of the methods of evaluation indicated above may be associated with false positive and false negative diagnoses. It must be recognized that some cases of heart disease fall below the threshold of diagnosis. In other cases, a definitive diagnosis may not be possible with currently available technology and knowledge. These limitations can be minimized by considering the following general guidelines:

- The results of the examinations described above are most reliable when performed by an experienced individual with advanced training and experience in cardiovascular diagnosis. Echocardiography with Doppler, cardiac catheterization, and post-mortem examination of the heart for heart disease require advanced training in cardiovascular diagnostic methods and the pathology and pathophysiology of heart disease.
- Examinations performed in mature dogs are most likely to be definitive. This is especially true when considering mild congenital heart defects. Innocent heart murmurs are less common in mature animals than in puppies and are less likely to be a source of confusion. Furthermore, the murmurs associated with some mild congenital malformations become more obvious after a dog has reached maturity. While it is quite reasonable to perform preliminary evaluations and provide provisional certification to puppies and young dogs between 8 weeks and 1 year of age, final certification prior to breeding should be obtained in mature dogs at 12 months of age or older.
- Examination conditions must be appropriate for recognition of subtle cardiac malformations. Identification of soft cardiac murmurs is impeded by extraneous noise and by poorly restrained, anxious or panting dogs.
- A standardized cardiac clinical examination must be performed according to a predetermined and clearly communicated protocol. Physical examination and cardiac auscultation should be used as the initial method of cardiac evaluation. If the clinical examination is normal, no further diagnostic studies are recommended. If the clinical examination is abnormal, a tentative diagnosis may be made, but the definitive diagnosis generally requires other diagnostic studies (as indicated above).
- Examiners who perform echocardiography with Doppler must use appropriate ultrasound equipment, transducers and techniques. Such individuals should have advanced training in non-invasive cardiac diagnosis and should follow diagnostic standards established by their hospital and by the veterinary scientific community, including standards published by the American College of Veterinary Internal Medicine, Specialty of Cardiology (J Vet Internal Med, 1993;7:247-252).

Examination of dogs for heart disease is aimed at the identification and classification of the phenotypic abnormalities. Heritable aspects of heart disease cannot be addressed unless suitable genetic studies have been conducted.

METHODS OF EXAMINATION

Clinical Examination

The clinical cardiac examination should be conducted in a systematic manner. The arterial and venous pulses, mucous membranes and precordium should be evaluated. Heart rate should be obtained. The clinical examination should be performed by an individual with advanced training in cardiac diagnosis. Board certification by the American College of Veterinary Internal Medicine, Specialty of Cardiology is considered by the Veterinary Medical Association as the benchmark of clinical proficiency for veterinarians in clinical cardiology, and examination by a Diplomate of this specialty board is recommended. Other veterinarians may be able to perform these examinations, provided they have received advanced training in the subspecialty of congenital heart disease.

Cardiac auscultation should be performed in a quiet, distraction-free environment. The animal should be standing and restrained, but sedative drugs should be avoided. Panting must be controlled and if necessary, the dog should be given time to rest and acclimate to the environment. The clinician should be able to identify the cardiac valve areas for auscultation. The examiner should gradually move the stethoscope across all valve areas and also should auscultate over the subaortic area, ascending aorta, pulmonary artery and the left craniodorsal cardiac base. Following examination of the left precordium, the right precordium should be examined.

- The mitral valve area is located over and immediately dorsal to the palpable left apical impulse and is identified by palpation with the tips of the fingers. The stethoscope is then placed over the mitral area and the heart sounds identified.
- The aortic valve area is dorsal and one or two intercostal spaces cranial to the left apical impulse. The second heart sound will be most intense when the stethoscope is centered over the aortic valve area. Murmurs originating from or radiating to the subaortic area of auscultation are evident immediately caudoventral to the aortic valve area. Murmurs originating from or radiating into the ascending aorta will be evident craniodorsal to the aortic valve and may also project to the right cranial thorax and to the carotid arteries in the neck.
- The pulmonic valve area is ventral and one intercostal space cranial to the aortic valve area. Murmurs originating from or radiating into the main pulmonary artery will be evident dorsal to the pulmonic valve over the left hemithorax.
- The tricuspid valve area is a relatively large area located on the right hemithorax, opposite and slightly cranial to the mitral valve
- The clinician also should auscultate along the ventral right precordium (right sternal border) and over the right craniodorsal cardiac border.
- Any cardiac murmurs or abnormal sounds should be noted. Murmurs should be described as indicated below.

DESCRIPTION OF CARDIAC MURMURS

A full description of the cardiac murmur should be made and recorded in the medical record.

- Murmurs should be designated as systolic, diastolic or continuous.
- The point of maximal murmur intensity should be indicated as described above. When a precordial thrill is palpable, the murmur will generally be most intense over this vibration.
- Murmurs that are only detected intermittently or are variable should be so indicated.
- The radiation of the murmur should be indicated.

Grading of heart murmurs

Grade 1—A very soft murmur only detected after very careful auscultation.

Grade 2—A soft murmur that is readily evident.

Grade 3—A moderately intense murmur not associated with a palpable precordial thrill (vibration).

Grade 4—A loud murmur; a palpable precordial thrill is not present or is intermittent.

Grade 5—A loud cardiac murmur associated with a palpable precordial thrill; the murmur is not audible when the stethoscope is lifted from the thoracic body wall.

Grade 6—A loud cardiac murmur associated with a palpable precordial thrill and audible even when the stethoscope is lifted from the thoracic wall.

Other descriptive terms may be indicated at the discretion of the examiner; these include such timing descriptors as: proto- (early) systolic, ejection or crescendo-decrescendo, holo-systolic or pan-systolic, decrescendo, and tele- (late) systolic; and descriptions of subjective characteristics such as: musical, vibratory, harsh and machinery.

EFFECTS OF HEART RATE, HEART RHYTHM AND EXERCISE

Some heart murmurs become evident or louder with changes in autonomic activity, heart rate or cardiac cycle length. Such changes may be induced by exercise or other stresses. The importance of evaluating heart murmurs after exercise is currently unresolved. It appears that some dogs with congenital subaortic stenosis or with dynamic outflow tract obstruction may have murmurs that only become evident with increased sympathetic activity or after prolonged cardiac filling periods during marked sinus arrhythmia. It also should be noted that some normal, innocent heart murmurs may increase in intensity after exercise. Furthermore, panting artifact may be a problem after exercise.

It is most likely that examining dogs after exercise will result in increased sensitivity to diagnosis of soft murmurs but probably decreased specificity as well. Auscultation of the heart following exercise is at the discretion of the examining veterinarian.

At this time the OFA does not require a post exercise examination in the assessment of heart murmurs in dogs; however, this practice may be modified should definitive information become available.

ECHOCARDIOGRAPHY

The echocardiographic examination should be conducted in a systematic matter. The examiner must be able to perform two-dimensional, pulsed-wave Doppler and continuous-wave Doppler examinations of the heart. The availability of color Doppler is valuable but not essential for most examinations. The echocardiographic examination should be performed and interpreted by individuals with advanced training in cardiac diagnosis. Board certification by the American College of Veterinary Internal Medicine, Specialty of Cardiology is considered by the Veterinary Medical Association as the benchmark of clinical proficiency for veterinarians in clinical cardiology, and examination by a Diplomate of this Specialty Board is recommended. Other veterinarians may be able to perform these examinations provided they have appropriate equipment and have received advanced training in echocardiography.

The pericardial space, both atria, both ventricles, the great vessels and the four cardiac valves should be imaged using long axis, short axis, apical and angled image planes as necessary to perform a complete examination of the heart. Nomenclature should follow that recommended by the American College of Veterinary Internal Medicine, Specialty of Cardiology. An anatomic diagnosis may be possible based on two-dimensional imaging; however, the origin of cardiac murmurs should also be evaluated using Doppler methods.

Doppler examination of all cardiac valves should be performed and recorded. Abnormal flow should be quantified using pulsed-wave or continuous-wave Doppler techniques. Values obtained should be compared to reference values. The depressant effects of any tranquilizers or sedative must be considered when measuring peak flow velocities. Color Doppler echocardiography should be employed if available to assess normal and abnormal blood flow patterns. Identification of abnormal flow across the cardiac septa or shunts at the level of the great vessels is best done by a combination of color and pulsed-wave Doppler techniques.

Special attention should be directed to the assessment of flow patterns and velocities in the left ventricular outlet and descending aorta. Optimal alignment with blood flow should be sought for accurate velocities to be reported. This may require the use of sub-xiphoid (subcostal) transducer positions as well as left apical (caudal parasternal) transducer placements. In addition to measurement of peak velocity using pulsed- or color wave Doppler, the pulsed-wave sample volume should be gradually advanced from the subaortic area into the ascending aorta in order to identify sudden accelerations in flow velocity, turbulence or aortic regurgitation.

Echocardiographic studies should be reported on videotape for subsequent analysis and a written record of abnormal findings should be entered into the medical record.

DENTITION DATABASE

The Dentition Database was established in late 2011 at the specific request of the American Rottweiler Club. Full dentition is an element of breed-specific health, form and function for a number of breeds. The purpose of the database is to certify dogs with all adult teeth fully erupted. The database does not certify overall dental health, misaligned teeth or dentition in accordance with a breed standard.

EXAMINATION AND CLASSIFICATION

Each dog is to be examined and classified by a licensed veterinarian. The examining veterinarian will determine whether all adult teeth are fully erupted, and identify and persistent (retained) deciduous teeth as well as any missing teeth.

The exam form includes a dental chart, and any retained or missing teeth should be marked "P" (persistent) or "M" (missing). If the owner authorizes release of any abnormal information, the dental chart identifying the specific missing or persistent deciduous teeth will be included on the dog's OFA webpage.

CLEARANCE ISSUED

A breed registry number will be issued for any dog found to be normal (all adult teeth fully erupted) at 12 months of age or older.

DEAFNESS DATABASE

GENETIC DEAFNESS IN DOGS

Printed with permission of Dr. George M. Strain, Louisiana State University—School of Veterinary Medicine, www.lsu.edu/deafness.deaf.htm

Congenital deafness in dogs (or other animals) can be acquired (caused by intrauterine infections, ototoxic drugs like gentamicin, liver disorders or other toxic exposures before or soon after birth) or inherited. Inherited deafness can be caused by a gene defect that is autosomal dominant, recessive, sex-linked or may involve multiple genes (more on this later). It is usually impossible to determine the cause of congenital deafness unless a clear problem has been observed in the breed, or carefully planned breedings are performed. In this article I will discuss what is currently known about the genetics of deafness in dogs so that breeders can make the best informed decisions possible when attempting to reduce or eliminate deafness.

Congenital deafness has been reported for approximately 80 breeds, with the list growing at a regular rate; it can potentially appear in any breed but especially those with white pigmentation. Deafness may have been long-established in a breed but kept hidden from outsiders to protect reputations. The disorder is usually associated with pigmentation patterns, where the presence of white in the hair coat increases the likelihood of deafness. Two pigmentation genes in particular are often associated with deafness in dogs: the merle gene (seen in the Collie, Shetland Sheepdog, dappled Dachshund, harlequin Great Dane, American Foxhound, Old English Sheepdog and Norwegian Dunkerhound, among others) and the piebald gene (Bull Terrier, Samoyed, Greyhound, Great Pyrenees, Sealyham Terrier, Beagle, Bulldog, Dalmatian and English Setter). However, not all breeds with these genes have been reported to be affected. The deafness, which usually develops in the first few weeks after birth while the ear canal is still closed, usually results from the degeneration of part of the blood supply to the cochlea (the stria vascularis). The nerve cells of the cochlea subsequently die and permanent deafness results. The cause of the vascular degeneration is not known, but appears to be associated with the absence of pigment producing cells (melanocytes) in the blood vessels. All of the function of these cells is not known, but one role is to maintain high potassium concentrations in the fluid surrounding the hair cells of the cochlea; these pigment cells are critical for survival of the stria. Deafness in the Doberman, which is also accompanied by vestibular (balance) disturbance, results from a different mechanism, where hair cell death is not the result of degeneration of the stria. Deafness may also occur later in life in dogs from other causes such as toxicities, infections or injuries, or due to aging (presbycusis); these forms of deafness almost never have a genetic cause in animals and thus do not present a concern in breeding decisions.

The prevalence of congenital deafness in different breeds is seldom known because of the limited number of studies (see the table on Breed Specific Deafness Prevalence in Dogs at www.lsu.edu/deafness/incidenc.htm). In the Dalmatian, where the incidence is highest, 8% of all dogs in the U.S. are bilaterally deaf and 22% are unilaterally deaf. In the English Setter, English Cocker Spaniel, Australian Cattle Dog and Bull Terrier, where fewer numbers of dogs have been hearing tested, the incidence appears to be about one-third to one-half that of Dalmatians. Unilateral or bilateral deafness is found in 75% of all white Norwegian Dunkerhounds, but the incidence in normal-color dogs is unknown. Other breeds with a high incidence are the Catahoula and Australian Shepherd. The incidence of all types of deafness in the general dog population is low, reported to be 2.56 to 6.5 cases per 10,000 dogs seen at veterinary school teaching hospitals, but these data predate the availability of hearing testing devices and so are much lower than actual values. Recognition of affected cases is often difficult, because unilaterally deaf dogs appear to hear normally unless a special test (the brainstem auditory evoked response, BAER) is performed; facilities to perform the BAER are usually only available at veterinary schools. It should be noted that a unilaterally deaf dog can be as great a genetic risk for transmission of deafness to its offspring as is a bilaterally deaf dog.

The method of genetic transmission of deafness in dogs is usually not known. There are no recognized forms of sex-linked deafness

TABLE 8

Dog Breeds with Relatively High Incidence of Reported Deafness

Australian Cattle Dog	Dalmatian
Australian Shepherd	English Cocker Spaniel
Bull Terrier	English Setter
Catahoula Leopard Dog	

Note: dogs of any breed can have congenital deafness, from a variety of causes. Breeds with white pigmentation are most affected.

TABLE 9

	Dd	
Dd	DD	Dd
	Dd	dd

Theoretical outcomes of breeding of two carriers of a recessive deafness gene (d).

TABLE 10

	DD	
Dd	DD	DD
	Dd	Dd

Theoretical outcomes of breeding a carrier and a dog free of the recessive deafness gene.

TABLE 11

	Dd	
dd	Dd	dd
	Dd	dd

Theoretical outcomes of breeding a carrier and an affected dog with the recessive deafness gene.

TABLE 12

	DD	
dd	Dd	Dd
	Dd	Dd

Theoretical outcomes of breeding an affected dog and a dog free of the recessive gene.

in dogs, although this does occur in humans. The disorder has been reported to have an autosomal recessive mechanism in the Rottweiler, Bull Terrier and Pointer, but this suggestion is not reliable because the reports were before the availability of BAER testing and the ability to detect unilaterally deaf dogs. References usually state that deafness transmission in most other breeds is autosomal dominant, but this is false, as will be discussed below. Pigment-associated inherited deafness is not restricted to dogs. Similar defects have been reported for mice, mink, pigs, horses, cattle, cats and humans. Deafness in blue-eyed white cats is common and is known to be passed on as an autosomal dominant defect. Blue eyes, resulting from an absence of pigment in the iris, is common with pigment-associated deafness but is not, in and of itself, an indication of deafness or the presence of a deafness gene; however, in several breeds (Dalmatians, English Setters, English Cocker Spaniels and Bull Terriers) dogs with blue eyes are statistically more likely to be deaf. Waardenburg's syndrome, a human condition, presents with deafness, a stripe of white in the hair and beard, blue or different colored eyes (even in blacks and Asians), no pigment behind the retina, and minor structural deformities around the nose and eyes. This is an autosomal dominant disorder with incomplete penetrance, which means that individuals that inherit the disorder may not show all components of the syndrome - i.e., they may not be deaf. Incomplete penetrance of a defect greatly complicates the determination of mode of inheritance. At present there is no documentation that incomplete penetrance is a factor in any canine deafness.

In simple Mendelian genetics, each dog carries two copies of each gene, one from each parent. The possible outcomes of breedings can be demonstrated with tables showing the genotype of both parents and the possible combinations in their offspring. If deafness is carried as a theoretical simple autosomal recessive gene (d), the breeding of two hearing carriers (Dd) (**Table 9**) will result, on average, in 25% affected dogs (dd), 50% hearing carriers (Dd), and 25% free of the defect (DD). The breeding of a carrier to a dog free of the defect (**Table 10**) will result in no affected dogs but 50% carriers and 50% free. The breeding of an affected dog to a carrier (**Table 11**) will result in 50% affected, 50% carriers, and no free. Finally, the breeding of an affected dog to a dog free of the defect (**Table 12**) will result in 100% carriers and no affected or free.

If instead the deafness is carried as a simple autosomal dominant gene (D), the breeding of an affected dog (Dd) to a free dog (dd) (**Table 11**) would result on average in 50% affected and 50% free. Dogs with the genotype DD would be unlikely to occur unless two deaf dogs had been bred. All of the above assumes that incomplete penetrance is not acting. If more than one gene (recessive and/or dominant) is involved in producing the deafness, the possible combinations become much more complicated. In humans more than 50 different autosomal recessive or dominant deafness genes or loci have been identified. The children of two deaf parents with two different recessive deafness genes can be unaffected, but carry both genes. If deafness in dogs results from more than one recessive gene, the possible outcomes of breedings are more numerous and determination of the mechanisms of transmission will be difficult.

As stated above, deafness is often associated with the merle (dapple) gene, which produces a mingled or patchwork combination of dark and light areas. This gene (M) is dominant so that affected dogs (Mm) show the pattern, which is desirable in many breeds. However, when two dogs with merle are bred, 25% will end up with the MM genotype (i.e., **Table 11**). These dogs usually have a solid white coat and blue irises, are often deaf and/or blind, and are sterile. Breeders of these dog breeds know not to breed merle to merle. In this case the deafness is neither dominant nor recessive, but is linked to a dominant gene that disrupts pigmentation and secondarily produces deaf dogs.

Genetic transmission of deafness in dogs with the piebald (sp) and extreme white piebald (sw) pigment genes, such as the Dalmatian, is less clear. These genes affect the amount and distribution of white areas on the body. Deafness in Dalmatians does not appear to be autosomal dominant, since deaf puppies result from hearing parents. It does

not appear to be a simple recessive disorder; we have bred pairs of deaf Dalmatians and obtained bilaterally hearing and unilaterally hearing puppies, when all should have been deaf if the disorder was recessive. These findings might be explained by a multi-gene cause, the presence of two different autosomal recessive deafness genes, or a syndrome with incomplete penetrance. Further studies (in progress) will be required to determine the mechanisms. Several candidate genes known to cause pigment-related deafness in humans or mice have been eliminated as the possible cause of pigment-associated deafness in Dalmatians. Whole-genome screens will hopefully identify the cause in this and other breeds.

Recent studies have shown that deafness in Dobermans, which do not carry the merle or piebald genes, results from direct loss of cochlear hair cells without any effects on the stria vascularis. Vestibular (balance) system signs, including head tilt and circling, are seen, and the deafness is transmitted by a simple autosomal recessive mechanism. A similar pathology has been described for the Shropshire Terrier.

So what should breeders do when deafness crops up? The most conservative approach would be to not breed the affected animal and not repeat the breeding that produced deafness. It is frequently recommended (i.e., Dalmatian Club of America) that bilaterally deaf puppies should be euthanized, since they make poor pets, are difficult to train, are prone to startle biting, frequently die from misadventure (cars), and require excessive care. There is considerable controversy on this point, and there is no question that many people have successfully raised deaf dogs. For every story of a problem deaf dog there seems to be a story of one that was successfully raised. Unfortunately, there is no way to predict how a deaf puppy will turn out. Unilaterally deaf dogs can make good pets but should not be bred. When deafness is uncommon in a breed, affected dogs should not be bred, but this does not mean that all related dogs are a risk and must be retired from breeding. An understanding of simple autosomal recessive and dominant patterns, as explained above, can allow the breeder to make better informed decisions and likely avoid future deaf animals without sacrificing a breeding line that has been shaped over many years. However, extreme caution must be used when line-breeding dogs related to deaf dogs, whether the deafness is unilateral or bilateral. To make these decisions in an informed manner for breeds with known deafness, it is important that advantage be taken of hearing testing facilities at veterinary schools. Unilaterally deaf dogs cannot be detected by other means, and these dogs will pass on their deafness genes.

OTHER OFA SERVICES

PRELIMINARY HIP AND ELBOW EVALUATIONS

This service is offered to evaluate the hip status of an animal as young as 4 months of age. Many owners choose to breed their animals prior to 24 months or need to know the hip status of progeny produced by a particular sire and dam before using them in a repeat breeding. The evaluation is performed by one radiologist, and the response time is usually five days. Use the same application procedure as described in the introduction.

OTHER SCREENINGS

OFA offers radiographic screening for tracheal hypoplasia for Bulldogs and other brachycephalic breeds only. Dogs must be over 12 months of age to receive an OFA tracheal hypoplasia number. The OFA Spine Database is an anecdotal database for Bulldogs, French Bulldogs and Boston Terriers. Information from OFA spine evaluations are maintained in this database for research purposes only.

OFA also offers a Serum Bile Acid Database as a screening tool for liver shunt and hepatic microvalvular dysplasia.

FUNDING OF ANIMAL WELLNESS STUDIES

The OFA has funded over \$3 million in research aimed at reducing the incidence and prevalence of inherited companion animal disease. The OFA funds projects through the AKC Canine Health Foundation (AKC CHF), the Morris Animal Foundation (MAF) and occasionally through direct grants. The OFA has achieved Ruby Donor status with MAF, and Millennium Founder status with the AKC CHF. OFA supported research is not limited to orthopedic disease, and has included cancers, heart disease, thyroid disease and various other diseases. Some research has been breed specific, some for all breeds, some for multiple species, and all is done at leading universities and research institutions. With the recent completion of the mapping of the canine genome, the OFA is focusing more of its research dollars toward research at the molecular level.

Details of the research the OFA has funded are available on the OFA website. In addition to the specific grants outlined there, the OFA has directly funded whole genome sequencing on numerous dogs and regularly supplies DNA samples to research projects worldwide.

WWW.OFA.ORG

The primary function of the OFA website is found in the searchable disease database holding the records of every dog certified by the OFA since 1974. An indispensable tool for breeders, owners and puppy buyers, the online database allows searches by individual dog, breed, disease and/or result and contains over 1 million animals. Search results list not only the animal, but also sire, dam, siblings, half-siblings and offspring, either in list or pedigree format. All information on the OFA website is free of charge and open to the public. In addition to the OFA disease databases, OFA website resources include:

- Information about the OFA
- OFA/MU DNA testing information
- Breaking health and OFA news
- Upcoming health clinics across the U.S. and Canada
- Breed health surveys
- Downloadable quarterly reports by breed
- The Blue Book—Ocular Disorders Presumed to Be Inherited in Purebred Dogs, updated monthly
- Many additional OFA publications (including this one), all in downloadable formats
- All OFA disease applications (which can be filled out online)
- The latest disease statistics and data
- Up-to-date lists of genetic disease databases and DNA-linked diseases and laboratories

VETERINARY STUDENT OUTREACH

The OFA regularly funds veterinary student outreach projects, and maintains an endowed veterinary student scholarship at the University of Missouri.

SEMINARS

The OFA underwrites travel expenses to present seminars to clubs when the audience size is 100 or more. No honorarium is charged. Arrangements must be made at least 6 months in advance; contact the OFA for more information.

BREED SPECIFIC DATA

OFA provides breed-specific summaries and CHIC reports to breed parent club designated liaisons on a quarterly basis.

CANINE HEALTH INFORMATION CENTER (CHIC) DNA REPOSITORY

Mission Statement

The OFA-CHIC DNA Repository collects and stores canine DNA samples along with corresponding genealogic and phenotypic information to facilitate future research and testing aimed at reducing the incidence of inherited disease in dogs.

Objectives

- Facilitate more rapid research progress by expediting the sample collection process
- Provide researchers with optimized family groups needed for research
- Allow breeders to take advantage of future DNA-based disease tests as they become available
- Foster a team environment between breeders/owners and the research community, improving the likelihood of genetic discovery

Submission by Blood Sample

Blood is the gold standard for genetic material; the yield of DNA is sufficient for all research methods, including technologies on the horizon. Moreover, the stability and purity of the DNA is of the highest caliber, which offers many benefits. The drawback of banking blood samples is cost; drawing, shipping, storing and extracting DNA from blood are more expensive endeavors than the alternative.

Submission by Cheek Swab

Cheek swab-derived DNA is a viable option for DNA banking. Although the yield and purity of this DNA is inferior to that obtained from blood, the material is suitable for most genetic approaches. The swabs are inexpensive, and the samples can be taken by the owner of the dog without the necessity of a veterinary office call. Swabs are easily shipped in standard envelopes using the postal mail, and they can be stored for at least a decade at room temperature, so long as they are stored under conditions of low humidity. The success rate for obtaining DNA from a swab in the laboratory is roughly 98%, so multiple swabs should be submitted for each dog to ensure representation in the archive.

Laboratories

The CHIC DNA Repository has partnered with the Veterinary Genetics Lab at the University of California–Davis and the Animal Molecular Genetics Lab at the University of Missouri. UC Davis will receive and store all swab samples, and Missouri will receive and store all blood samples.

For health surveys, application forms, and instructions on how to participate in the DNA repository visit www.ofa.org/about/dna-repository.

REFERENCES

- Report of Panel on Canine Hip Dysplasia: AVMA Council on Veterinary Service. JAVMA, October 1, 1961, p. 792.
- Bennett D: Hip Dysplasia and Ascorbate Therapy: Fact or Fancy? Seminars in Vet. Med. And Surg., 2:2, 1987, pp. 152-157.
- Chase, K et al: Bilaterally asymmetric effects of quantitative trait loci (QTL's): QTL's that affect laxity in the right versus left coxofemoral (hip) joints of the dog (canis familiaris). Am. J. Med. Genet., 124A: 239-247, 2004.
- Chase, K et al: Genetic regulation of osteoarthritis: A QTL regulating cranial and caudal acetabular osteophyte formation in the hip joint of the dog (canis familiaris). Am. J. Med. Genet., 135A: 334-335, 2005.
- Corley EA, Carlson W: Radiographic, Genetic, and Pathologic Aspects of Elbow Dysplasia. J Am Vet Med Assoc, 1965;147:1651.
- Corley EA, et al: Reliability of Early Radiographic Evaluation for Canine Hip Dysplasia Obtained from the Standard Ventrodorsal Radiographic Projection. JAVMA, 211:9, 1997, pp. 1142-1146.
- Essman S: Comparison of Digitized and Conventional Radiographic Images for Assessment of Hip Joint Conformation in Dogs. AVVR, 67:9, 2006, pp. 1549-1551.
- Genevois JP et al: Prevalence of Hip Dysplasia According to Official Radiographic Screening among 31 Breeds of Dogs in France. Vet. Comp. Ortho Traumatol., 1, 2008, pp. 21-24.
- Grondalen J, Grondalen T: Arthrosis in the Elbow Joint of Young, Rapidly Growing dogs. Nordish Veterinarmedicin, 1981;33, pp. 1-16.
- Grondalen J: Arthrosis in the Elbow Joint of Young, Rapidly Growing Dogs: Interrelation between Clinical Radiological, and Pathoanatomical Findings. Nordish Veterinarmedicin, 1982; 34:65-75.
- Kaneene JB, et al: Retrospective Cohort Study of Changes in Hip Joint Phenotype of Dogs in the United States. JAVMA, Vol. 211, No. 12, Dec. 1997, p. 1442-1544.
- Kaneene JB et al: Update of a Retrospective Cohort Study of Changes in Hip Joint Phenotype of Dogs Evaluated by the OFA in the United States, 1989 – 2003. Veterinary Surgery, 38: 398-405, 2009.
- Kasstrom H: Nutrition, Weight Gain, and Development of Hip Dysplasia: An Experimental Investigation in Growing Dogs with Special Reference to the Effect of Feeding Intensity. Acta Radiol. Suppl., Vol 344: 135-179, 1975.
- Kealy RD, et al: Effects of Limited Food Consumption on the Incidence of Hip Dysplasia in Growing Dogs. JAVMA, Vol. 201, No. 6, 1992, p.857-863.
- Kealy RD, et al: Effect of Diet Restriction on Life Span and Age-related Changes in Dogs. JAVMA, 2002; 220: p.1315-1320.
- Keller GG, et al: How the Orthopedic Foundation for Animals (OFA) is tackling inherited disorders in the USA: Using hip and elbow dysplasia as examples. The Veterinary Journal (2011), Vol: 10.1016/j. tvjl.2011.06.19
- Leighton EA: Genetics of Canine Hip Dysplasia. JAVMA, Vol. 210, No. 10, 1997, pp. 1474-1479.
- Lust G et al: Joint Laxity and its Association with Hip Dysplasia in Labrador Retrievers. AJVR, Vol. 54, No. 12, 1993, p.1990-1999.
- Lust, G et al: Comparison of Three Radiographic Methods for Diagnosis of Hip Dysplasia in Eight-month Old Dogs. JAVMA, 2001; 219: p.1242-1246.
- Oberbaur, AM et al: Long Term Genetic Selection Reduced Prevalence of Hip and Elbow Dysplasia in 60 Dog Breeds. PLOS One, Feb 24, 2017.
- Olsson SE: Osteochondrosis in Domestic Animals. ACTA Radiologic Suppl., 358, 1978, pp.299-305.
- Olsson SE: The Early Diagnosis of Fragmented Coronoid Process and Osteochondritis Dissecans of the Canine Elbow Joint. JAAHA, 1983;19(5):616-626.
- Padgett GA, et al: The Inheritance of Osteochondritis Dissecans and Fragmented Coronoid Process of the Elbow Joint in Labrador Retriever. JAAHA, 1995; 31: 327-330.
- Powers MY, et al: Evaluation of the relationship between Orthopedic Foundation for Animals hip joint scores and PennHIP distraction index values in dogs. JAVMA, Vol 237:5, Sept 1, 2010, pp 532-541.

- Read RA, et al: Fragmentation of the Medial Coronoid Process of the Ulna in Dogs: A Study of 109 Cases. *J. Sm. Anim. Prac.*, 1990; 32(7), 330-334.
- Reed AL, et al: Effect of Dam and Sire Qualitative Hip Conformation Scores on Progeny Hip Conformation. *JAVMA*, 2000; 217: 675-680.
- Rettenmaier JL, Keller GG, et al: Prevalence of Canine Hip Dysplasia in a Veterinary Teaching Hospital Population. *Vet. Rad. & Ultrasound*, Vol. 43, No. 4, 2002, pp. 313-318.
- Smith, GK et al: 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 Radiograph in Dogs. *AJVR*, Vol 54: 1021-1042, No. 7, July, 1993.
- Swensen L et al: Prevalence and inheritance of and Selection for Hip Dysplasia in Seven Breeds of Dogs in Sweden and Benefit:Cost Analysis of a Screening and Control Program. *JAVMA*, 210:2, 1997, pp. 207-214.
- Swenson L, Audell L, Hedhammar A: Prevalence and Inheritance of and Selection for Elbow Arthrosis in Bernese Mountain Dogs and Rottweilers in Sweden and Benefit: Cost Analysis of a Screening and Control Program. *JAVMA*, 210:2 1997, pp. 215-221.
- Tomlinson JL: Quantification of Measurement of Femoral Head Coverage and Norberg Angle within and among four breeds of dogs. *AJVR*, 2000; 61: pp.1492-1498.
- Willis MB: Practical Genetics for Dog Breeders. H. F. & G. Witherby Ltd, Great Britain, 1992.

ADDITIONAL READING

- Ackerman L: The Genetic Connection. AAHA Press, Lakewood, CO, 1999.
- Bardens JW: Palpation for the Detection of Dysplasia and Wedge Technique for Pelvic Radiography; *Proc AAHA*; pp. 468-471, 1972.
- Belkoff SM, et al: Development of a Device to Measure Canine Coxofemoral Joint Laxity. *VCOT*, Vol 1: 32-36, 1989.
- Berry CR: Evaluation of the Canine Elbow for Fragmented Medial Coronoid Process. *Vet. Radiol. & Ultrasound*, 1992;33(5): pp. 273-276.
- Carpenter LG, Schwarz PD, Lowry JE, Park RD, Steyn PF: Comparison of Radiographic Imaging Techniques for Diagnosis of Fragmented Medial Coronoid Process of the Cubital Joint in Dogs. *J Am Vet Med Assoc*, Vol 203, No 1, July 1, 1993.
- Corley EA: Role of the Orthopedic Foundation for Animals, Inc. in the Control of Canine Hip Dysplasia. *Veterinary Clinics of North America: Small Animal Practice*, Vol 22: 579-593, May 1992.
- Dixon RT: The Effect of Limb Positioning on the Radiographic Diagnosis of Canine Hip Dysplasia. *Vet. Rec.*, Vol 91: 644-646, 1972.
- Guthrie S, Pidduck HG: Heritability of Elbow Osteochondrosis within a Closed Population of Dogs. *J. Sm. Anim. Pract.*, 1990: 31: 93-96.
- Hedhammar A, Olsson SE, et al: Study of Heritability in 401 Litters of German Shepherd Dogs. *JAVMA*, Vol. 1974; 1012-1016, 1979.
- Keller GG, et al: Influence of the Estrus Cycle on Coxofemoral Joint Subluxation. *Canine Pract.*, Vol. 18, No. 1, 1993, p.19-22.
- Keller GG, et al: Correlation of Radiographic, Necropsy and Histologic Findings in 8 dogs with Elbow Dysplasia. *Veterinary Radiology & Ultrasound*, Vol.38 No. 4, 1997, pp. 272-276.
- Larsen JS: Lumbosacral Transitional Vertebrae in the Dog. *J Amer. Vet. Soc.*, Vol. 18, No. 3, 1977, pp. 76-79.
- Lust G: Overview of the Pathogenesis of Canine Hip Dysplasia. *JAVMA*, Vol. 210, No. 10, 1997, pp. 1443-1445.
- Mackenzie SA: Canine Hip Dysplasia; Why Heritability Estimates Differ. *Canine Pract.*, Vol 12: 19-22, 1985.
- Padgett GA: Control of Canine Genetic Diseases. Howell Book House, New York, NY, 1998.
- Wind A: Elbow Incongruity and Development Elbow Dysplasia in the Dog (Part 1). *J Amer Anim Hosp Assoc* 1986;22:711-724.



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