

GRUNFELD GERMAN SHEPHERDS

Heritability

by Fred Lanting, edited by John Cole

Perhaps no term is more misunderstood by dog fanciers when talking about inherited characteristics and environmental effects than the concept of heritability. It is necessary for you to take a little time and effort to get this idea fixed in your mind before applying it to practical breeding programs. I will try to give the major ways of looking at it as expressed by some of the leading authorities, in both veterinary medicine and genetics. Try to differentiate between the similar terms of heritability and inheritance. Think of HD as being 100% transmitted by genes, while its expression (obvious or radiographic signs) is not as predictable as that 100%. The first is how the trait is obtained, while the second is how much of the trait is seen (either obvious or radiographic signs). Pin this on your wall:

INHERITANCE = HOW; HERITABILITY = HOW MUCH.

Dr. Corley of the OFA describes heritability as a percentage measurement of "how much of the phenotypic variation in a studied population is of genetic origin." This means the differences caused by the genes, not the disease itself. In the late 1950s and early '60s it was thought that heritability of HD was around 0.6 (60%), but experiments in Sweden and elsewhere gave the another heritability index value, that of 0.2 to 0.3 for the same breed. Thus, a heritability index of 0.25 means that 25% of the phenotypic variance (the expression of HD) is due to gene action. (This does not mean, however, that the environment causes 75% of dysplasia in dogs.)

When Olmstead says, "70% of the development of hip dysplasia is influenced by genetics" (personal correspondence, 1992), the key word is development. Environment, then, would affect 30% of the development, including the severity, age at which HD became apparent, and the specific nature of the lesions (osteophytes, laxity, etc.). Environment would not have anything to do with the actual existence (genotype) of the disorder. Therefore, to measure the total phenotypic variance of HD, you must evaluate the pelvic radiographs of a large population of dogs maintained in the same environment (identical nutritional intake per pound of body weight, for example). Dr. Lust of Cornell University maintains that the usefulness of heritability estimates (indexes) is due to the fact that they are reliable predictors of future progeny when certain phenotypes are combined in a breeding program.

Malcolm Willis, professor of genetics at the University of Newcastle-Upon-Tyne, and one of Britain's leading HD/genetics authorities, reminds us that heritability, strictly speaking, "relates to a specific character calculated on a specific population of dogs at a specific point in time". You can more accurately predict hip quality in your dogs' offspring, by decreasing the environmental variation. Or you could outcross more often (always to dogs with good joint backgrounds) to raise the genetic variance. This is like bringing in more musicians to audition for spots in the orchestra. The more you have from which to choose, the more you can cull of the less desirable musicians and enhance the quality of your orchestra. You can also positively direct heritability by fine-tuning diagnostic and rating systems. With the old BVA scheme, in which hips were classified as normal, near-normal, and dysplastic, HD heritability in the German Shepherd Dog was said to be around 0.25, but when their much more detailed 0-to-106-point scoring system was applied to the same dogs, heritability increased to nearly 0.4, which supposedly represents greater success in eliminating (or, at least, reducing) the HD genotype from your line. If this is an accurate estimate, then it means that the presence of good and bad genes have been more closely identified by the newer British scheme than they had been by using the old one, and the effects of environment are of lesser importance now that diagnosis was supposedly more accurate. The OFA, using the AVMA protocol, is reported to use a seven-feature approach, also, but in practice two things are of utmost importance in a diagnosis: laxity and any sign of remodeling or DJD. This is not something they publicize to the breeder.

Continuing with the idea of directing heritability and changing the indexes, remember that HD is polygenic. When many genes are involved, more accuracy might be had in "looking" at them by more methods or aspects. Using an evaluation scale with many levels for example, would allow the geneticist to determine how much of the trait is under the control of genetics, and how much is under the control of the environment. When you put all individuals into two or three groups in order to evaluate them in terms of heritability, you do a poor job of describing "reality" either statistically or verbally. Consider one equation for heritability:

$h^2 = s^2A / (s^2A + s^2E)$. The better job we do of estimating the environmental and genetic variances in the equation, the better estimate we get of heritability, h^2 . Usually when a scale with few categories is used to measure a trait that exhibits continuous variation, genetic variance is underestimated and environmental variance is overestimated (this has been going on in the field of HD for a long time), and h^2 index appears to be smaller than it should be.

Good advice has been given that breeders perhaps should not use the reported and variable heritability figures either as a tool in breeding or as an excuse to avoid using good genetic sense. We should not interpret heritability to be anything more than an aid in predicting results from a breeding program. Stick to the basics: HD (and other joint disease) is a genetic problem and must be attacked by better selection of genes. You can raise the heritability of your own lines by using the right mix of outcrossing and linebreeding on dogs with the best phenotypes.

LINEBREEDING, OUTCROSSING, AND INBREEDING

Linebreeding (or its extreme application, inbreeding), while perpetuating desirable characteristics, also limits the gene pool. Another way of saying this is that when you linebreed/inbreed, you end up with a smaller pedigree (contains fewer unique individuals). This results in a reduction of additive genetic variance because you have fewer genes to put together, and you have many individuals with similar genotypes. That is why puppies from linebreeding practices look so much more uniform than do pups resulting from outcrossing. Since there is less variability and lower heritability in linebred dogs, one can expect much less progress through phenotypic selection when the hips of all the near ancestors have not been carefully "chosen." The typical German Shepherd Dog is an example of extremely close linebreeding. In recent decades this has been as true of the German dog as it has long been of the phenotypically quite-different AKC version. So, it is not surprising that breeders who have not "culled the ancestors" and selected strongly for good hips far back in the pedigree, will often find a "percentage plateau" (a point at which no further progress can be made because of no more variability in the line).

It is imperative that when you buy an outcrossed pup, you make sure that the parents' hips are as good as possible. Of course, you know that the term "outcross" is also relative. By obtaining all the information you can on the parents and their littermates and litters, especially the dam's, the less likely you will be to encounter the "outcross surprise." The partial reliance on an index such as BV (breed value) also known as ZW (Zuchtwert) would be very helpful.

The October and December 1992 issues of Shiba Journal carried an excellent piece on inbreeding by Susan Houser, an attorney with another degree in zoology and direct work experience in genetics. She points out that inbreeding (breeding of very close relatives) "brings about a decline in characters concerned with fitness (viability, fecundity, and growth)", quotes Darwin in saying that prolonged inbreeding brings about "loss of size, constitutional vigor, and fertility", and quotes Ehrlich and colleagues in saying, "a loss of fitness referred to as inbreeding depression occurs . . . [when] . . . inbreeding is imposed on populations that are usually outbreeding". Willis also warns against unwise inbreeding.

Inbreeding limits small populations to small numbers of genes, and too often, many of these are the "wrong" genes being paired because there is no dominant "good" allele there. While inbreeding concentrates certain desired characteristics such as milk production, rapid weight gain in pigs, more ears per cornstalk, grain yield per acre, rear leg angulation, short legs, chiseled head features, etc., it also causes the breed of animal to lose certain other genes through attrition or prevention of being reintroduced to the pool. At least, the result is a loss of availability of genes that are not easily recovered.

The idea that genes are "lost" with increasing homozygosity is referred to by population geneticists as "fixation".

Inbreeding depression is the result of achieving homozygosity in recessive genes. This could partly explain the reported rise in HD (9.7% in one fairly recent analysis) and other defects among American German Shepherd Dogs while the rest of the world population in that breed is steadily (but slowly) improving in hips, without losing other historic qualities of type. Other breeds were reported by the OFA to have increased in frequency of HD in the 1980s compared to the 1970s: Great Dane 3.8%, Golden Retriever 1.3%, and Akita 1.1%. The Labrador Retriever showed no change in HD, according to their statistics, but had marginally more dogs graded Excellent.

In the light of this knowledge (or perhaps it is in spite of this knowledge being available!) why do breeders use so much inbreeding, since there is voluminous evidence that it badly affects health? Because they are focusing on one or a few specific traits and, seeing some short-range success with perhaps a litter of champions, continue to inbreed or heavily linebreed beyond the invisible margin of safety. Then they wonder why their lines are notorious for intussusception, pancreatic deficiency, and other organ problems

as well as shortened lifespans. The puppy buyers, rather than the breeders, are too often forced to pay the price of inbreeding.

Fortunately, breeders can improve general health by selecting ancestors for good joints without inbreeding. However, those who inbreed or linebreed without regard to x-ray knowledge of their dogs' joints will undoubtedly add dysplasias to the list of complaints of their doggy descendants.

PHENOTYPE SELECTION AND VARIABILITY IN REPORTING h2 INDEX

Heritabilities vary, both by the method of evaluating the trait and by which particular traits are considered (e.g., HD's average heritability may be different from heritability of shoulder angulation or temperament). Heritability of HD varies also between breeds, so that the Samoyed, for example, may have an estimated index variability of 0.8 and the German Shepherd Dog may have one of 0.25 (we shall see later that this might be an inaccurate and too-low figure) even if calculated by the same people. In Sweden, where it is generally believed that heritability of HD is around 0.4 to 0.5 across the board, you will still have differences between breeds. For example, choose a German Shepherd Dog pup for breeding purposes from a group of "normal" German Shepherd Dogs there and you might find, on average, more HD in its pups (and perhaps the individual itself, when it grows up) than you would if you did the same thing in Labradors. Swenson explains this by the higher incidence of HD in their German Shepherd Dog population, but I think it might have to do with differences in heritability between breeds or colonies of dogs. HD heritability can also be a function or property of a colony or population of dogs selected over a period of time so that they have a different percentage than other groups not so selectively bred. A conscientious breeder who has been applying the best principles of HD prevention to his program for generations may have a different index in his dogs than exists in the breed at large. Heritability indices for HD in purebred dogs have been estimated at levels as low as 0.2 and as high as 0.6 or even as high as 0.8 in Samoyeds, with most of the data supporting these numbers having been gathered before much breeding progress in specific colonies. Reasons for why heritability estimates vary have been more recently analyzed and they are as much dependent on the particular population (selected subgroup) as on the environment, if not more.

Heritability differences between breeds can reflect the numbers of dogs and families used by the amount of linebreeding and by differences in selection practices. They may also reflect the number of grades and, hence, the accuracy of the rating/scoring system used. Heritability differences within a breed can be influenced by the same things. The reasons heritability of HD in American guide dogs for the blind is so high (0.54) probably include conscious pre-selection for better hips and possibly the use of other breeds in addition to the "historic" German Shepherd Dog blind guide dog. It has been found that the much more accurate diagnostic technique of stress radiography known as PennHIP has a significantly higher heritability index than that associated with the hip-extended view used by OFA, SV, and other "traditional" registries. A higher index means progress is potentially accelerated; that breeders who use PennHIP, for example, can produce pups with much greater assurance of joint soundness.

Traits that are high or moderately high in heritability (perhaps over 0.35, as an arbitrary level) respond comparatively rapidly to phenotype selection (i.e., selection of breeding partners based on no radiographic evidence of abnormalities in the joints). High heritability means that environmental differences will have less effect on the expression of bad genes, because there are fewer bad genes to be affected. In such animals, phenotype is a better predictor of genotype than is the case in those dogs with low heritability indexes. This way, the polygenic trait of HD, after generations of phenotypic breeding (i.e., exclusion of dysplastic dogs from the program) begins to act more like a "simple" trait created by one or two gene pairs.

For example, no matter what you do with environment, be it nutrition, exercise, or anything else, you cannot change the phenotype of a black Labrador Retriever to a yellow Labrador Retriever. Heritability of coat color, then, could be said to be 1.0 (100% controlled by the genes, zero by environment). The yellow Lab doesn't have any genes for black and cannot produce black puppies if bred to another yellow Lab. The goal of the breeder seriously fighting HD is to develop a line of dogs with so few bad hip genes that in a somewhat similar manner, it cannot produce differently than their own phenotypes. At least, we should go as far in this direction as is feasible. If you are dealing with a trait or a breed with low heritability (maybe 0.1 to 0.2 or so), selecting parental stock by phenotype (normal hips, in this context) will yield slower progress in better hips in the first few generations. However, if you keep at the selection process for normal hips, you will see some progress in your line. Stick with dogs that have been selected for normal phenotype in your own and others' lines and you will raise the heritability in your stock above that of the breed in general. Use improved diagnosis, and the advance will be greater and even higher. Do you want to lower the influence of environmental factors such as overnutrition on your production of dogs? You can

do so by selecting dogs that have hips that are relatively unaffected by such factors, which is another way of saying you should choose dogs with higher heritability.

On the other hand, the example used above might not be the best, since it really does not make a lot of sense to talk about heritability for a qualitative trait such as coat color. For one reason, you would have a hard time trying to measure color on an empirical scale in many breeds such as the Bloodhound, Airedale, and German Shepherd, all of which are saddle-marked dogs but with differences in expression of the pattern. Such dogs are hard, if not impossible, to refer to as "36% black" or "4% saddle". Since there is no good numerical value measured, you would not be able to compute the variances that form the mathematical expression for heritability.

If all breeds are considered together, HD is a less-heritable (and in most cases more polygenic) trait than many other characteristics. So, compared to those others, we would expect to see a less-rapid change in frequency when using strictly phenotypic breeding. This includes X-ray pictures as well as regular visual signs and traits. When we speak of phenotype in the context of a discussion on HD, we are usually referring to the evaluation of the "standard" pelvic radiograph. If we say that a breed or population within a breed has a heritability of 0.25, we are saying that the difference in genes between two dogs is responsible for 25% of the x-ray picture differences between them. That means that differences in environments (feeding practices, road work, etc.) account for the other 75% of the phenotypic differences as seen on the radiographs. You can therefore see how a higher h^2 index can lower the effect of environment on what you think you see in the dog. Higher h^2 gives you a better indication of genotype.

One reason for the fact that heritabilities can vary from one breed to another is the history of intense linebreeding/inbreeding that is most often used to found a breed. Look at the books on your own favorite breeds, and you will find a very few individuals in all of the early pedigrees. A natural consequence of such inbreeding is that there will be differences between breeds in regard to genetic variation for the same trait. In dairy cattle, heritabilities for traits such as volume of milk or level of butterfat produced are quite similar, but that is because the major dairy breeds, while heavily selected, have not undergone the same kind of pedigree-linebreeding that many dog breeds have. The Border Collie, as soon as it was "accepted" by the AKC as a "showdog", was doomed to eventual loss of abilities, the same as happened to many a Spaniel, Setter, Shepherd, and "Non-Sporting" breed. Once breeding is based on pedigrees and a narrow slice of the phenotype spectrum, instead of production records or verifiable working ability, the gene pool becomes more limited, perhaps very much so.

The interplay of genetic and environmental components of the expression of pelvic phenotype is complex and understandably misinterpreted by vets and breeders alike. But the bottom line is still: environment may have a great effect on the expression of genes an individual dog has inherited, especially if the diagnostic method is inferior, but no effect at all on the genes it will pass along to its progeny.

Note: copyright 1999, Fred Lanting Thanks to geneticist John Cole for editing and some suggestions. e-mail: Mr.GSD@juno.com for permission to use. Fred Lanting presents seminars worldwide on canine orthopedic disorders, gait-and-structure, schutzhund problem-solving, history of the GSD, and other topics.

GRUNFELD GERMAN SHEPHERDS

For the Best in German Shepherds visit
www.grunfeldshepherds.com