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BRIEF INTRODUCTION TO GENETICS

by Fred Lanting

"Nature prevails enormously over nurture"
...English scientist Francis Galton in the late 19th Century.

Natural Selection & Survival of the Fittest

Hip dysplasia is not common in wild animals because of the continuous processes of natural selection and survival of the fittest. In an environment where society removes or inhibits these means of selection, the host of ills of which HD is but one example is denied full effect and allows the least fit to survive and breed as well, and in some instances makes it easier. The hunter bags the biggest game, we cut the best trees down, and so forth.

The hypothetical young elk with hip dysplasia will be one of the first ones caught by the wolf pack, and such will not live long enough to impregnate as many elk cows as stronger, healthier ones could. Conversely, any wolf with weakness such as HD will not have the stamina of its normal packmates and, bothered by arthritis and coxo-femoral pain, is probably going to lose any leadership challenge as well as the concurrent privilege of fatherhood to a younger, more agile, normal-hipped male. In human society and in those animal societies we influence, these natural safeguards are often lost. In the wild, there are no hospitals or welfare systems.

A colony of Dingos (wild dog breed in Australia) has been reported which had been bred, fed, reared, and protected in captivity for some 40 years. When radiographed, a substantial portion of these zoo animals were found to be dysplastic. However, newly-captured Dingos were free of the disorder. During one of my lecture-judging tours in that continent a couple of decades later, I visited a private Dingo breeding and preservation farm and found the operator practicing selective breeding which included rejection of dysplastic animals from the reproducing groups, just as any responsible breeder of more domesticated canines would do.

Canine hip dysplasia, a multifactorial, developmental, quantitative condition, is genetically controlled. Early in the 1960s and even before, HD was thought to be due to one or more dominant genes with incomplete penetrance, which meant it varied from individual to individual, or even between one hip and the other in the same dog. It is now widely held that HD is basically recessive (the word does not mean unnoticed or diminishing). To understand the difference between dominant and recessive, and to gain a working knowledge of the inheritable nature of HD, with an eye toward genetically controlling it in one's kennel or breed, a brief biology lesson and simplified examples of inheritance modes would be helpful.

Building Blocks

One of the principal tenets of education is that understanding is best attained if you start with the simple before progressing to the complex. Starting with atomic physics in order to understand breeding and orthopedic disorders may not seem like it fits that description, but let's start near there anyway, and try not to get lost. We'll go along in that direction a short distance before getting back to structures smaller than cells. You remember from your grade school science classes that "everything is made of atoms", a simplification, to be sure, but good enough for our purposes. An element is a substance that

cannot be separated into simpler parts, and whose atoms are all the same kind. Oxygen, gold, and diamonds (pure crystalline carbon) are examples of elements. A molecule is a new arrangement of atoms that are so "glued together" that they seem almost as indivisible and unique as atoms. A compound is a collection of identical molecules; water, salt, and carbon dioxide are examples of simpler compounds, while proteins, nylon, and perhaps even viruses are examples of complicated molecules or compounds. Some of the very highly developed molecules such as viruses and cell nucleus parts have the ability to reproduce themselves by building copies out of the raw materials available in the "cell soup" they are immersed in.

Getting back to the medium level of complexity, though, we see that most living organisms are composed primarily of a fantastic variety and innumerable combinations of compounds of oxygen and hydrogen atoms attached to carbon-chain "backbones" with smaller numbers of other elements such as sulfur, nitrogen, calcium, phosphorus, etc. Exactly how and where they are part of specific carbohydrate (carbon/hydrogen/oxygen) molecules determines their function. Proteins have more nitrogen atoms, bone has more calcium and phosphorus, blood has more iron, and so on, than other molecular structures in the various parts of the body.

Cells are units often likened to building blocks because in so many instances, they are arranged side-by-side to make up larger structures. The next step up in complexity as far as body organization is concerned is a collection of cells grouped together to perform a specific function; this is called a tissue, and examples include hair, bone, and muscle fiber. Tissues of similar or dissimilar types are associated in collections called organs, such as the heart, eye, and skin. Sometimes we use the same word to mean several things, as in the case of "bone", which could refer to specific types of bone cells; bone tissue as compared to cartilage; or bone as an organ containing marrow, cartilage, and harder tissues. Organs work together for the benefit of the organism (body and psyche).

Cell Division and Chromosomes

In every cell of every living thing, there are submicroscopic strands of protein-like material called "chromosomes", composed of long spiral macromolecules. Macro- means large, and molecules are fairly stable combinations of atoms. Examples of macromolecules include DNA, cellulose, rubber, and nylon. An electron microscope can differentiate not only the number but also the shapes and sizes of chromosomes when cells divide. The dog has 78 chromosomes, humans normally have 46; always an even number since chromosomes appear in pairs with each cell containing the same number of chromosomes except in sex cells which each have half the number so that the normal number reappears when they get together. The male sperm and female ovum (egg cell) each have just one of each pair that are found in the other cells of the body.

When cells perform that mathematical wonder of dividing to multiply, they create identical "daughter cells". The cell structure is basically cytoplasm, nucleus, and wall. During cell division, strands begin to appear in the nucleus, and soon are distinguishable as chromosomes. Before and after the phases of division, they are not identifiable, but persist in a "distributed" manner, becoming visible again only in the next division. Because each chromosome is composed of a double chromatids entwined about each other, they split lengthwise into two "pieces" which seem pretty identical on casual view. Only upon much closer investigation, such as with electron microscopes, other analytical instrumentation, and deductive reasoning, do we see that they are not truly identical, for the gene on a particular location on one chromosome of the pair could easily have a very slightly different chemical structure than that on the exact same location on the matching chromosome. Just as the chromosomes exist in pairs of strands, the genes on them obviously are paired, also. They too are strand-like in dimension, and are twisted and coiled like knobby rubber ladders made by someone on drugs or devoid of coordination.

Biochemists are able to stir up semi-dissolved cells with certain chemicals in order to separate these chromosomes and make them visible under high-power microscopes. They take pictures of the alphabet-soup dilution, and cut the photos apart so they can pair up "twins". You then have a karyotype and can see some abnormalities such as missing or extra chromosomes (severe defects such as Down's syndrome), or one chromosome fused to another (may produce either obvious or covert defects). Usually such fusions inhibit reproduction ability, so the results are seldom heritable. On the other hand, crossing-over is fairly common and is responsible for much of the variety you see in your litters. A dog can have a great multitude of different sperm cells (none of them quite the same, like snowflakes). The same is true in bitches' ova, but she releases far fewer sex cells at a time than does the male.

A chromosome is composed of numerous chemical units called "genes", and from the same root

we get the word genetics. An idea of how complex genetics can be is had when one realizes there may be from 10,000 to 100,000 pairs of genes in mammals. The interaction of many of these genes makes for an almost infinite number of variations, like snowflakes or fingerprints, and no 2 dogs are exactly alike. Genetic diseases such as HD are results of one or more abnormalities in deoxyribonucleic acid (DNA), the complex macromolecule/chemical of which genes are primarily made.

Most defects cannot be determined by just looking at the chromosomes. The aberrations are in individual genes, and since these are so much smaller, you can't tell much by looking, regardless of magnification. Just how small are these genes? They might be 3 to 7 microns long but only 2 thousandths of a micron long. (A micron is a thousandth of a millimeter, and a millimeter is about four hundredths of an inch). There are now about 6 billion humans on Earth, each of us having our physical beginning in a single fertilized ovum (egg). Genes are made of strands of DNA (deoxyribonucleic acid), and if you could measure all the DNA in 3 billion ova (nearly equal to the half the number of the world's entire population), you could fit it inside a single raindrop! Yet, end to end, the strands could make eight loops to the moon and back! We are talking about some pretty small, narrow strands of molecules.

Interaction

Certain genes have a primary effect on individual characteristics but other genes, especially nearby ones, influence them as well. Depending on the proximity on the chromosomes and the strength of the chemical attractions between different genes, minor to major differences will appear in genotypically very close relatives. One dog may have slightly larger white areas than a littermate with mostly similar gene arrangements, one pup may have a slightly different degree or appearance of HD than a littermate inheriting basically the same major genes. The nearest-neighbor theory holds that the greatest mutual effect may be between 2 adjacent genes.

Dominance and Polygenic Traits

We think of chromosomes as strings of pearls, with collections of genes up and down their lengths, and each gene having a specific location (locus) in relation to the others. These loci are given arbitrary letter designations, with the initial letter sometimes standing for a key word in the name or description of the condition. Usually, we use a capital letter to designate a dominant trait, and a subcase letter for a recessive (weaker) trait called for by that location's gene.

A characteristic governed "entirely" by one gene pair is commonly called a Mendelian trait, and those traits determined by a large number of genes are known as multifactorial or polygenic. What are commonly called simple Mendelian traits are handy characteristics to use in illustrating the nature of inheritance as regarding dominance, recessives, and other terms you've undoubtedly come across. Probably the easiest of these is the matter of coat color, because of everyone's familiarity with it.

Suppose that chromosome #28 is the one that carries the gene for coat color in a Labrador Retriever; when the sperm cell carrying a gene for black enters an ovum carrying a gene for yellow, the pup (fertilized egg) have the normal complement of chromosomes and genes. In this example, the pup will be black in color because the gene for "black Lab" is dominant or "stronger" than the gene for "yellow Lab". In the Shiba, the gene for red is stronger than (dominant over) the gene for black-and-tan (sometimes called "tan-point"). Once you gain some familiarity with inheritance of simple traits, which means those which are determined by a single pair of genes, you can at least appreciate the depth of complexity if not understand the combinations of many genes that we call polygenic traits. Most things breeders are concerned with are polygenic, such as temperament, joint quality, good ears, bite, hunting ability, length of bone, HD, and probably angulation and movement traits. The different numbers of genes involved in different traits gives the impression that some disorders are perhaps 50-100% genetic in expression, while others appear to be primarily due to environmental factors. However, this is an illusion, as environment determines only the expression of the genes, not their presence.

Polygenic traits, those determined by a few or many genes working in concert, are not as predictable as the Mendelian traits, but we can make a broad generalization that "defective genes" are usually recessive in nature. Enough "weaker" recessive genes can outnumber and overpower the dominant genes for correct joint conformation, for example. Thus, if you breed a dog with mostly genes for dysplastic hips to a dog with radiographically normal hips but perhaps 45% bad genes (not enough to make for bad hips), what will you get in the litter? Just maybe one or more that has radiographs like the OFA-certified parent, but more likely most of the offspring will have a preponderance of bad genes.

Further complicating this is the probability that one gene for depth of acetabulum is on one locus, the gene for growth plates' response to overnutrition is on a different locus, the gene for tightness of certain ligaments is on yet another, etc. As in dealing from a deck of cards, where you have a wide variation in the value of cards ace through king, and each player gets a random collection which may have a favorable or unfortunate combination, the puppies in a litter will have varied collections of genes. Some will have been dealt better hands than others, but if you have previously "stacked the deck" you can assure all of them getting better hands than those dealt by your less-careful competitor. If the high cards represent poorer quality genes (high percentage of defects), and the low cards optimum genes for joint quality, it is obvious that you will produce more "winners" if you start with decks that have fewer high cards and more low cards, then play your game without allowing yourself or anyone else to slip high cards into the deck. Removing high cards as they appear in hands is analogous to the process of culling: removing from the gene pool those dogs with lower-quality genes. Easy in principle, but it takes diligence to accomplish this in a breeding program.

You know that genes and chromosomes operate in pairs. When the half-number in sex cells are united at conception, the physical characteristic called for by one gene may be different than that of its corresponding gene partner, in which case the animal is said to be heterozygous for the trait exhibited (you can see the effect in simple, one-gene-pair, Mendelian traits). If the chemical nature (molecular structure) of the two members of that pair of genes is identical, the animal is said to be homozygous for the trait in question. The prefix homo- means "the same" and hetero- means "different, while "zygo" is the Greek root for "pair".

In a heterozygous situation, one gene will have a greater influence than its partner, but when many genes in many locations affect each other (polygenic), predicting the outcome is not as easy. Thus the importance of lowering the variation or number of different genes by culling.

Effects of the Bitch on HD in the Offspring

At the 1987 conference, "The Dog in Service of Humanity" which was held in Geilo, Norway, Swenson reported on test and retrospective breeding data, matching normal studs with grade-1 dysplastic bitches and vice versa, and compared that to the similar results found by the State Dog Training Centre eleven years earlier. It was discovered that the bitch had more influence on the eventual hip status of the offspring than did the sire, by about 10%. If the bitch were the dysplastic half of the union, about 10% more HD was found in the progeny. To explain, he offered a couple of possible reasons. It could be that the uterine environment or some factors prior to weaning, such as poor "general physiology" or poor milk/colostrum or other undiscovered factor affects HD in a negative direction from the beginning of conception or some time shortly thereafter.

It is also possible that a genetic factor plays a part. You know that the ovum is a cell with a nucleus surrounded by other substances collectively called cytoplasm. You also know that the DNA strands that make up the genes are located on chromosomes in the nucleus, and these chromosomes (and genes) match up with homologous (same shape, size and general make-up) ones from the entering sperm to make the nucleus of the first offspring cell. Well, there is also some DNA in the cytoplasm outside of the nucleus. While not thoroughly investigated yet, this "extra" DNA could explain the conventional wisdom that the bitch contributes more to the pups than the sire does, as well as explain the results of the Swedish studies noted above. The tiny sperm cell apparently does not carry cytoplasmic DNA to the union.

Early research in HD involved the effects of hormones, especially estrogens, and some thought they could initiate or prevent some evidence of dysplasia by manipulating estrogen levels but others found "maternal environment" including estrogens not to be important in the cause or etiology of HD. When I was on one of my lecture/judging tours in Australasia one of the lectures was sponsored by Uncle Ben's, the same company that makes Pedigree and Pal brand dog foods. During my incidental research at Murdoch University and elsewhere I came across work (also partially sponsored by Uncle Ben's) by Queensland and Victoria HD investigators (Goddard & Mason) which found "no differences" but rather a "lack of any breed-of-dam effect", which led them to conclude that "it is unlikely that maternal oestrogen concentration is an important cause of variation between dogs..." and that other maternal environments were likewise insignificant.

Polygenic Selection

That Australian work looked at the possible effect of heterosis (contribution to health or size, sometimes referred to as "hybrid vigor" even though matings within a species perhaps do not really produce hybrids in the usual semantic sense). They used 4 breeds with hip quality ranging from the

native Kelpie down through Boxer and Labrador Retriever to the German Shepherd Dog, and cross-bred them in almost all combinations, partly to see if better-hipped candidates for blind guides might be possible. Only the Kelpie and the Shepherd can liberally be considered anywhere near each other on the canine genealogical tree, and that very remote indeed. One breed known worldwide for its connection to relatively high incidence of HD (the GSD), one lightweight breed with generally low incidence (Kelpie), and two with moderate incidence (the Boxers were worse than expected and the Labs better, so they were roughly equivalent) made up the breeding stock. Pups were palpated, later radiographed, and the results analyzed using the least-squares estimate for breed effects. They found more variation within breeds than between breeds.

One indication was that selection against HD has occurred "naturally" in times past by selection for "working" qualities (Greyhound for speed, Kelpie for agility and stamina) but earlier reports by Pharr and others show no reduction in HD when Australian Shepherds were chosen on the basis of occupation as working stock dogs. Perhaps we don't work our "working" dogs as rigorously as they did a century ago! While significant heterosis is seen when cross-breeding Greyhounds and German Shepherds, no such significant improvement was seen by crossing the Shepherds to Kelpies.

Patterson illustrates polygenic inheritance with a balance: one pan contains the "good hip genes", the other contains the "bad hip genes". Each gene is on a different locus, and the pans probably involve more than one chromosome. Each pair of genes has one partner (allele) from the sire and one allele from the dam. Suppose young Rover had radiographically fair hips (but good enough to rate an OFA normal classification), and almost half of his hip genes were defective, the other o.k., and if he is bred to Lady who also has "normal X-rays" but many defective hip genes, what will the puppies of such a union inherit?

Mathematically, you might expect more possibilities than the dam would ever have time to produce, but suppose one of her pups designated "A" inherits all of Rover's bad genes and all of Lady's bad genes (actually half of all the pairs). "A" will then have the same genetic bank. But it is quite possible, even likely, that the hips of pup "A" will look worse on film if environmental forces (nutrition, especially) had more of an effect. Many people consider HD to be a "threshold condition" which means that if there are a certain number of bad genes (even if less than half), the condition will be seen unless covered up by such practices as deliberately keeping Rover and Lady very thin during growth. Also, some of the bad genes affect the acetabulum, some the ligaments, some the shapes of bones, etc., so the combination can be very complex.

Let's further suppose that Rover and Lady (both barely over borderline in hip quality genotype) were bred to each other by a math and statistics instructor (named Matthew Maddox, of course) and the professor kept one of the "average" pups (having both some bad and some good genes, like their parents). He named it "Gordo" and fed it a diet richly laced with calcium/phosphorus/Vitamin D tablets and beefed up that diet with ground meat to stimulate appetite, although keeping the meat supplement to 10% in accordance with advice from his colleague the biology professor who used to work for NRC. Another average pup, "Slim", was sold very early to a student who spent his summers at the Royal Veterinary College in Stockholm, helping researchers compile data on the effects of overnutrition. Knowing what you do about the effects of diet, especially overnutrition, guess which of these 2 average pups will have the greater chance of developing clinical and radiographic HD.

One more supposition: the rest of Lady's large litter (all average) went to loving homes whose masters all made sure they had their shots and vitamins, and ate well of their nice, rich puppy food especially formulated for growing dogs and reeking with appetizing flavors.

By now, you're getting ahead of me. Professor Matthew Maddox who spent a great deal of money for a fancy brood bitch and a stud service, both animals managing to have OFA numbers, has turned out a large litter of pups who have become badly dysplastic before the one-year guarantee ran out, and he has to borrow money to make refunds to many of the unlucky buyers, and stave off the others with a free replacement as soon as he can get another litter out of Lady, sired by some stud other than that nasty defective rascal Rover! Maybe when that student returns from Sweden this fall, just in time to breed Slim when Lady comes in season? What luck! The only dog not sold to a show home has great hips... well, actually, the OFA said "fair", but that's the same as his dam's, and they both have the same beautiful characteristics, so the breeding should be great! Besides, Lady's breeder had said, when he bought her as a pup, that they didn't have HD in their kennel, so it must have been carried by Rover. But you and I know something the professor doesn't: Slim has the same poor hip genotype or genetic bank that his dam, sire, and his moderately or severely dysplastic littermates have. And with such a precarious balance, such a small margin of total good genes over bad, the influence of environment can again be significant.

Unfortunately, "breeding programs" similar to the above extreme example are being conducted in like manner by "breeders" all over the world. The cripples go to the pound or are nursed by longsuffering, disappointed, or embittered owners, and the others like Slim or even some with not as good radiographs or none taken at all, are bred and bred and bred. There are nearly 90 million dogs and cats born each year, thousands every hour! (Dr. Faulkner says 2,000 to 3,000; Humane Society of the U.S. says 10,000; United Humanitarians say 12,500; Roger Caras estimates 15,0000.) We have a responsibility to encourage the breeding of genetically better dogs in preference to dogs with relatively more defective genes.

What's Ahead?

Perhaps someday the mysterious chemical nature of the inheritance of defects will be understood, and a simple chemical correction made or gene therapy performed for most of them. Until then, we must use what tools we have and do what can be done to control the genetic disorder known as hip dysplasia. OFA, GDC, and PennHIP are excellent diagnostic and control "helps", and AKC's slowly-awakening sense of responsibility to animal soundness and owner is commendable, partially making up for a history of neglect and greed in letting puppy mills flourish for the sake of litter registration dollars. The AKC is cooperating with OFA and the Morris Animal Foundation in a search for genetic markers in the chromosomes of dysplastic dogs and their (probably) carrier relatives. As a popular advertising phrase goes, "Watch This Space".

Are Orthopedic Disorders Such As HD Inherited?

Literally speaking, traits themselves aren't inherited; the bits of chemicals making up the genes are reproduced and distributed among sperm and egg cells for transmission to the next generation. But we can use the terminology just as we use the word "bloodlines" when blood has nothing to do with inheritance. Knowing that a trait is hereditary does not necessarily mean that equal knowledge is to be had about the manner in which it is so.

Numerous noted experts in the field have commented about the hereditary nature of HD, adding tons to the weight of evidence. Dr. U. V. Mostosky of Michigan State says, based on actual results in breeding programs involving many breeds, HD "is principally a genetic disease which may be modified by diet and exercise". Dr. Donald Patterson, chairman of Medical Genetics at University of PA School of Veterinary Medicine, states that some dogs with radiographically normal hips but a large number of hidden dysplasia-producing genes, if mated together, will produce at least some dysplastic offspring. Dr. Sheldon Gerstenfeld, author of "Taking Care of Your Dog", writes, "hip dysplasia has a polygenic mode of inheritance". Dr. George Lust of Cornell talks of "eliminating genes responsible for abnormal hip joint conformation". In another place he says flat out, "Canine hip dysplasia is a hereditary disease". Dr. E. A. Corley, in an OFA report, says in a section headed Findings of the OFA, "Hip dysplasia is an inherited trait". Dr. Frederick Hutt, author of a notable text on genetics, wrote of "genetic selection to reduce the incidence of hip dysplasia" and HD as "a good example of a defect that is polygenic". Allow me here also to quote Dr. Malcolm Willis who states the increase in proportion of better hips "provides ample proof to those doubters who still argue that HD is not inherited." Hedhammar, Olsson, and many scores of others have proven beyond any reasonable doubt that incidences of orthopedic disorders are reduced when genetic selection is used. The rare voice putting the blame for HD and other problems on a lack of vitamins or on some other nutritional or environmental cause will always find a few itching ears, but it is or should be absolutely astounding that anyone would give it the least credence.

Variation in Polygenic Traits

The reason you might not get a litter of exactly the ratio calculated when you breed two heterozygous dogs (Labs with genes for both black and yellow, for example) is that the mating of sperm and egg cells is a random event. It could easily be that the eggs are coincidentally surrounded by sperm cells carrying the black gene, in which case the "yellow genes" can't get to the eggs in time, and the litter will be all black, as if the sire was homozygous black. Well, if this can happen once in a while with only two alleles, imagine the random nature of mating perhaps dozens of eggs with millions of sperm cells. A great deal of variety exists in the wiggle-tailed suitors for Ms. Ovum. Some may have a bad gene for one specific trait in the hip joint design, some may have a bad gene for another trait, some may have both (in different genes or loci, of course, since there are no pairs in sex cells), and some sperm may have a whole bunch of defective genes (for many separate features in the joint). This is why you can get a very wide distribution of joint quality when you mate a dog with bad genotype to one with either good or bad overall

genotype (we're talking hips here, but it could apply to any polygenic disorder/trait). When there are more genes involved, and when the genotypes of the parents are disparate, you can expect much of what geneticists call "continuous variation".

To narrow the distribution of genotypes in the litters, and insure that it will be much closer to the ideal, "good end of the spectrum", mates must be chosen who have those properties themselves, and ideally ones who have already proven their genotype through their progeny.

Random Nature of Polygenic Disorders

To illustrate the culling aspect of the selection process in a polygenic disorder, let's start up that game of poker I alluded to earlier. Shuffle a deck of cards, then deal four hands, "up" so everyone can see all cards dealt. Assume that the "face cards" (king, queen, and jack) represent the highest numbers of genes for HD, and that the numbers on the rest of the cards are in proportion to the number of bad genes. Player A might get two face cards in his five "litters", and players C and D might get one face card each. Remove those face cards from play, set them aside, and reshuffle all other cards whether played or still in the deck. Repeat the procedure, and in the next couple of deals, you'll see the number of face cards decline. Become more selective, and remove from play the 10s, 9s, and 8s. After 2 to 4 more hands, which by now look pretty good, you might want to cull the 7s and 6s to get your score down really low in most hands (a hand representing either a bunch of litters or the pups in a single litter). Lo and behold, you'll still come up with an occasional face card or high-number card, which is the nature of our polygenic card game, but the odds against it are greater the longer you play the game and the more strict you become in culling individual dogs from a breeding program.

Because of the polygenic nature of HD and the heritability in most breeds and sub-populations, the breeder can expect much variation, a wide "spectrum" of hip qualities, when using phenotype as the deciding factor in his breeding program. Dogs with normal radiographs, mated together, will produce a range of pelvic quality in their offspring. However, if a dog has a normal (radiograph) phenotype, the chances of it having a genotype to match (normal, and expected to produce such phenotypes regardless of environments) is 1.4 times greater than the chances of it actually having a "dysplastic genotype". This figure comes from Lust's work at Cornell where it was found that, "of the dogs with normal phenotypes, the ratio of dogs with normal genotypes to dysplastic genotypes is 1.4 to 1".

The phenomenon of crossing-over mentioned earlier explains how a dog can inherit its father's ears and its mother's bite, just as in humans, and since there is so much crossing-over in the "plastic" nature of canine genes and chromosomes, we can see how a particular puppy might be unlucky enough to inherit all the bad-hip genes its dam contributes and if it doesn't get enough dominant good-hip genes from its sire to counteract them, can resemble the dam's worst ancestors in the hips, yet resemble the sire outwardly.

Since there are so many genes involved in polygenic traits such as HD, some few dogs with what we call a normal genotype (very few defective genes as evidenced by progeny results, for example) can be "forced" into dysplastic phenotypes if they are overfed and mineral-supplemented during their fast growth period. It is pretty hard to create more than mild signs of HD in a dog with very few bad genes, though, and my litter mentioned earlier which had been fed ad libitum is an example. Thus, some dogs with a certain mild grade of dysplasia have been known to produce better hips than they had themselves. And, as you have seen in the chapter on nutrition, a dog predisposed to HD because of the number of bad genes inherited may appear (phenotype) normal on radiographs if it has had restricted exercise and diet. Its genotype has been masked by manipulating the environment. Such a dog does not have as desirable a genotype as one which has been "forced" into signs of mild HD by overfeeding. Also, overfeeding often gives more signs of osteophytes than of laxity.

The very best dogs for breeding and work, given other things being equivalent, are those with the best pelvic radiograph phenotypes, raised in the most favorable environments. However, in order to establish a line or colony with great genotype, some believe, it may be worthwhile to expose the dogs in the program to the disadvantageous environments in order to force the few bad genes out into the open: feed liberally, especially. Then you could select for breeding only those dogs which, as pups, had such a "strong" genotype (fewest number of defective genes) that they were able to resist environmental forces, and develop normal phenotypes in spite of them. Some have termed this an extreme approach to eugenics.

How Much Is Genetic and How Much Environmental?

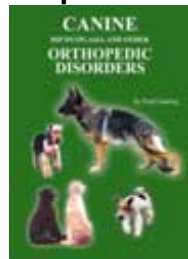
Most people like to put labels on others, and those who study characteristics of animals are often categorized as either hereditarians or environmentalists (not to confuse this word with the other meaning, that of conservationist). The latter believe more of an animal's actions are due to culture (training or environment) than do the hereditarians. I always like to exaggerate just a little for the sake of emphasis, and tell people that everything is genetic, even getting run over by a car (because traits of fear or avoidance in dogs who survive would tend to keep them away from the road). A more scientific phrasing might be that genetically-determined diseases and traits are modified to some degree between zero and 99% by forces other than the genes themselves; it has been my observation through many years of intensive experience in dogs that this percentage is usually far lower than the average dog owner believes. . One reason you can't find most of the wild animals in the woods is that they have a genetic shyness or flight response to the stimulus of human presence in their home. Of course, environmental forces such as the opening days of hunting season serve to greatly reinforce such inherited traits, so no one is a *total* hereditarian.

Fred Lanting is an internationally respected show judge, approved by many registries as an all-breed judge, has judged numerous countries' Sieger Shows and Landesgruppen events, and has many years experience with SV. He presents seminars and consults worldwide on such topics as Gait-&-Structure, HD and Other Orthopedic Disorders, Anatomy, Training Techniques, and The GSD. He conducts annual non-profit sightseeing tours of Europe, centered on the Sieger Show (biggest breed show in the world) and BSP. Check out his website: www.MrGSD.com



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Canine HD and Other Orthopedic Disorders by Fred Lanting.



It covers all joints plus many bone disorders and includes genetics, diagnostic methods, treatment options, and the role that environment plays. This new Hip Dysplasia and Other Canine Orthopedic Disorders\' book is a comprehensive (nearly 600 pages!), amply illustrated, annotated, monumental work that is suitable as a coffee-table book, reference work for breeders and vets, and a study adjunct for veterinary students, for the dog trainer and the general dog owner of any breed.

The Total German Shepherd Dog by Fred Lanting



This is the expanded and enlarged second edition, a "must" for every true GSD lover. It is an excellent

alternative to the "genetic history" by Willis, but less technical and therefore suitable for the novice, yet very detailed to be indispensable for the reputable GSD breeder. Chapters include: History and Origins, Modern Bloodlines, The Standard, Anatomy, The German Shepherd in Motion, Shows, Showing, and Training, The Winners, Nutrition and Feeding, General Care and Information, Health and First Aid, Parasites and Immunity, Diseases and Disorders, The Geriatric German Shepherd, Breeding, Basics of Genetics, Reproduction, Whelping, The First Three Weeks, Four to Twelve Weeks, Trouble-shooting Guide

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