Bad Genes, Babies & Bathwater

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Everyone has heard the phrase, "Don't throw out the baby with the bath water." But do dog breeders ever stop to consider how this admonition applies to them? Certainly not the novice who righteously declares that he will never, ever, keep anything that has even the possibility of producing the smallest genetic defect. Not the experienced breeder who refuses to consider an otherwise excellent line because it sometimes throws cataracts. And most definitely not the individual who declares that all DNA-tested dogs found to be carriers of recessive disease mutations ought to be removed from breeding. This tendency toward genetic over-kill not only culls dogs that might have something to offer, it can exacerbate the very problems breeders are trying to avoid. The following is a real life example of what can happen when breeders exercise short-sighted culling in the name of genetic disease control.

In the early 1970s, breeders of Basenjis launched a campaign to wipe out a fatal genetic disease called pyruvate kinase deficient hemolytic anemia (HA). HA is caused by a recessive gene. Dogs with a single copy of the gene are healthy, but those with two copies die. A screening test was developed that would indicate carriers as well as affected animals. Breeders zealously screened their dogs, eliminating not only affected animals but the healthy carriers from the breeding population.

Today HA is rare in Basenjis, but the incidence of Progressive Retinal Atrophy is significantly higher. As is yet another fatal disorder, a kidney problem called Fanconi's Disease. At the time, neither of these diseases had a screening test that would indicate carriers. (A DNA test for Fanconi is now available.) Had breeders been less fanatical in their pursuit of HA, they might have retained the healthy carriers in the breeding population, breeding them only to non-carriers so they could avoid producing HA- affected puppies. By such a method they could have retained the good aspects of those carriers, including freedom from genes for PRA or Fanconi's, while gradually lowering the incidence of the HA gene. Now that a Fanconi test is available, they can use this approach for that disease.

Fortunately for the Basenji, there is still a native population of the breed in Africa. The Basenji club prevailed upon the AKC to allow them to re-open the stud book to admit some African-born Basenjis. This badly needed source of new genetic material comes at great trouble and expense for those breeders who make the effort acquire one of these imports. This option isn't even possible in some breeds, and even where it is, convincing a large registry like AKC to accept undocumented foreign imports is itself a daunting task.

In spite of what happened with the Basenji, this should not be viewed as an indictment of screening tests. The problem wasn't the HA test, but the drastic culling process that breeders undertook when using it. If there is a test which can identify carriers, make use of it. This is especially true of DNA tests which not only reveal the dog's genotype, they are not subject to the false positive or negative results of other types of testing. Breeders need to know as much as possible about the genetic potential of their breeding stock. Ideally, they should be willing to share the results, whether good or bad, with other breeders.

Knowledgeable dog people know there is no perfect dog. Even the best of them have faults. The faults are not only those conformation or behavioral problems you can readily observe, but also bad genes. Dogs have around 25-30,000 genes. No matter how high the standards for selection of breeding

stock or how strict the culling of offspring, every dog will have genes for unwanted traits. Experts agree that every individual dog, human or cauliflower probably carries, a few "lethal equivalents" as well as a batch of genes that are merely suboptimal. This may leave you wondering why we aren't seeing dogs and cauliflowers, not to mention each other, dropping like flies all around us.

Under normal circumstances, lethal genes remain rare. Natural populations breed randomly, maintaining a varied mix of alleles, or forms, of genes. Only occasionally will the right combination of bad alleles match up to produce an affected individual. In addition, the lethal nature of these diseases limits the ability of affected animals to pass them on to their offspring because affected individuals often don't live long enough to reproduce. But the breeding of purebred livestock, including dogs, is not natural or random. It is selective based on the wants and needs of breeders. As a result, the number of lethal equivalents in most breeds exceeds the average of three, the problem genes having been inadvertently concentrated through the standard inbreeding practices used to maximize production of desired traits. Two examples in Australian Shepherds are Pelger-Huet Anomaly and merle. Genes with lethal effects are only the tip of the iceberg. There unknown numbers of those suboptimal genes whose effects are anywhere from minor to extremely bad.

Breeders routinely evaluate breeding stock by studying conformation and/or performance attributes in minute detail. Virtues are weighed against faults and compared to the virtues and faults of prospective mates. If the overall analysis is positive, the breeder will proceed. Hereditary diseases and defects need to be given the same kind of consideration, in and of themselves and in combination with all the dog's other traits.

Some faults are severe enough to eliminate a dog from breeding consideration entirely, but even genetic defects and disease may not necessarily fall into this category in some circumstances. Remember the case of the Basenjis and HA. Dogs proven to be carriers of traits in which only homozygotes (those with two copies of the gene) are affected, can be used if care is taken never to mate one carrier to another and not to use them extensively. If a DNA test is available, preference can be given to the clear-tested offspring of carriers for the next generation. In time the number of carriers will be reduced.

If the mode of inheritance for a trait is unknown or complex, identifying carriers can be difficult. Individuals that repeatedly produce traits like hip dysplasia, Cushing's Disease, or severe allergies should be pulled from further breeding because of the serious and debilitating nature of those diseases. But their healthy relatives may be used if care is taken to select mates unlikely to carry the same defect. If at any point an individual proved to be a repeat producer of the defect, it could then be removed from the breeding program.

Many faults are variable in expression. This includes such genetic defects as hip dysplasia (HD) and missing teeth. In Clumber Spaniels, where HD was once almost universal, elimination of all affected animals was not an option if the breed was to be preserved. By selecting away from the most severely affected dogs, Clumber breeders have managed to improve their overall situation, producing more non-dysplastic dogs and fewer which are severely affected, even though HD is still common. A similar situation has occurred with Collies and Collie Eye Anomaly.

In the case of missing teeth, a fault common to show line Australian Shepherds, something similar could be done. There are sufficient quality dogs available with full dentition that dogs missing multiple teeth ought not to be bred. However, those missing one or two teeth could be bred to mates with full dentition which are out of families with full dentition. In the 1970s, missing teeth in Aussies were almost unheard of. Twenty years from now the situation could be to nearly its starting point if breeders would be conscientious about screening and mate selection and none of he good traits those dogs have need be lost along the way.

The overall size of a breeding population must be taken into account before making final decisions on whether a dog exhibiting or carrying a defect ought to be bred. Australian Shepherds are numerous, but certain sub-sets of the breed are not. In North America there are thousands of Aussies, but in some parts of the world national populations may number only a few hundred breeding animals at best. Opportunities to add new stock can be limited by the expense of importing, strict quarantine laws, or import restrictions. Even in North America a breeder's selection of potential mates may be limited if his breeding goals are very specific, such as producing a particular type of stock dog.

In small populations, breeders may have no choice but to use some defective animals. The only alternative is to resort to increased inbreeding which will narrow the available gene pool even further and bring other, possibly worse, defects to the fore. If defective dogs are to be used, breeders should take special care to avoid subsequent inbreeding on those dogs. Neither should such a dog be bred extensively. Among its offspring, only those which do not exhibit the defective trait should be considered for further breeding.

If breeders approach genetic disease with an objective eye and if they are honest with themselves and each other about the potential for producing genetic diseases and defects in any given cross, they can obtain healthy babies while the bath water full of bad genes drains slowly away.