Deconstructing the Diehard:

The Genetic Plight of the Scottish Terrier

Part Two:

Pet Dollar$ and Deconstruction: Fundamentals of Scottish Terrier Gene Pool Peril

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"As a 'companion human' I probably think way too little about the breeding aspects of our kids and just assume the breed will always be around and be as wonderful 20 years from now as they are today."
Rebecca Collins, Ft. Worth, TX., letter to the editor

This candid insight from a thoughtful and devoted Scottie owner brings into the open unrecognized dimensions to the genetic peril facing the Scottish Terrier. Although rarely acknowledged, the jeopardy now facing our breed arises from both the supply side of breeders and from the demand side of buyers, for when pet owners do not understand their influence on breeding, genetics, and proliferation of genetic diseases, and when breeders pursue private agendas isolated from the public good, a harmful synergy envelopes the Scottish Terrier leaving the dogs and those who love them in serious trouble.

My argument here is that *both* buyers and breeders have been blind to deeper issues of genetics and the marketplace and therefore both are driving the deconstruction of the Diehard. Both sides of that Scottie equation, therefore—both breeders and buyers—must take responsibility for a new kind of informed partnership aimed at healthier Scotties, better stewardship of our breed, and unity in the Scottie community.

Pernicious Myth

In Part One of this series (*"Troubled Breed: How Did We Get Here?*," ***GSM***, Jul/Aug 2003) I referenced the 58 known genetic diseases in our breed plus the lethal cancers killing our dogs in increasing numbers, including the eye-opening data from Purdue

University's bladder cancer research which identified Scottish Terriers as 18 times more likely to develop bladder cancer than other purebreeds. I argued that because our dogs' world is the same polluted environment other modern breeds inhabit our breed's massive disproportionate risk of lethal cancer points to genetic predisposition in the Scottie gene pool and to unmapped consequences of the genetic deconstruction of the Diehard.

My analysis of how our dogs reached this predicament began at the top of the hierarchy of Scottie stewardship looking at our century-old purebreed system built on pedigrees and rigid appearance standards and exposed the pernicious myth which drives show-breeding culture, viz., that phenotype = genotype, or in laymen's terms, that the best-looking dog is the superior strain. The flaw in that premise is that looks do not equate with genetics, so a handsome, 'typy' Scottie can be a carrier of recessive traits which, when mated to another carrier of that recessive gene, can produce a health disaster, and on the other hand, a dog with less-than-perfect ear-set, who is rejected from the gene pool as "non-breed stock" because of ears, may have longevity and fitness genes the gene pool badly needs.

Today's pet owner who wishes to understand our breed's genetic predicament and wants to be part of the solution needs a lively sense of breed history—not merely the received story of illustrious kennels and dominant champions, but our breed's history viewed through the lens of population genetics which maps deleterious effects of loss of genetic diversity across whole populations. As shown in Part One, a genetics-framed look at Scottie history flags (1) our breed's small 'founder event' stock, (2) our century-old fixation on phenotype, or appearance, (3) our sustained inbreeding ("linebreeding" is genetically a form of inbreeding), and (4) our cyclic constriction of the Scottie gene pool to gene puddles by genetic bottlenecks due to overuse of celebrated popular sires. These four 'virtues' of the purebreed establishment amount to *diversity-reducing breeding practices* that add up over time to what geneticists call "inbreeding depression." As genetics expert Jerold Bell, of Tufts University, warns:

> "Inbreeding can exacerbate a tendency toward disorders controlled by multiple genes. Unless you have prior knowledge of what milder linebreedings on the common ancestors have produced, inbreeding may expose your puppies (and puppy buyers) to extraordinary risk of genetic defects. Research has shown that inbreeding depression, or diminished health and viability through inbreeding, is directly related to the amount of detrimental recessive genes present." (*"The Ins and Outs of Pedigree Analysis, Genetic Diversity, and Genetic Disease Control."*)

Classic signs of inbreeding depression are manifest in our Scotties: (1) shorter lifespans (2) weakened immunolgy (3) smaller litters (4) increased whelping problems, and of course, (5) spread of genetic diseases (see: Lisa Paddock, *"Our Mr. Holmes: Poster Dog for a Scottish Terrier Open Health Registry—The Face of Chronic Illness In Well-Bred Scotties,"* ***GSM***, May/June 2003, and also 'Letter To Editor' from Elaine Weigle, ***GSM***, Jul/Aug 2003).

My whole argument regarding unintended deconstruction of the Diehard is we cannot continue doing business as usual making the same breeding decisions based on the same discredited genetic thinking we've used in the past and expect to change the future health of our breed. So long as our Scotties are officially measured by glamor-quotient rather than fitness, so long as official recordkeeping is myopically fixed on championship records instead of health records, our 'well-bred' Scotties will continue to devolve into genetically unfit 'Barbie Dogs' for whom no amount of medical research will cure their inbreeding depression. We must have a paradigm shift in our thinking as to what 'purebred' could and should mean, redefining our notions of 'well-bred' and our choices of breeding stock in terms of genetic fitness and vigor, and that shift must inform all of us from top levels of our national breed club to the newest pet owner in our breed.

Genetics Primer

But what about those of us who do not "make breeding decisions," who are

GENETICS GLOSSARY

Allele: Alternate form of a gene possessing a unique nucleotide sequence.

Autosome: Chromosome not a sex chromosome.

Chromosome: Thread-like DNA structure carrying genes, found in nuclei of cells.

Consanguinity: Related through at least one common ancestor.

Effective population size: Equivalent to population in which equal number of males and females contribute to next generation.

Gamete: Mature, haploid reproductive cell.

Gene: Unit of heredity comprising a segment of DNA found at a particular locus on a chromosome.

Genetic Drift: Random fluctuation in gene frequencies noticeable in small populations.

Genetic Load: Average number of lethal alleles per individual in a population.

Genotype: Full complement of genes influencing the phenotype for particular trait.

Hardy-Weinberg Law: Principle in population genetics that (ideal) gene frequencies remain constant from one generation to the next.

Heterozygous: Organism has two different gene-alleles for the same trait; said to be hybrid for that particular trait.

Homozygous: Having like gene-alleles for the same trait at same locus.

Inbreeding Coefficient: Probability that both gene-alleles at a particular locus will be identical by descent.

Linebreeding: Mating which maintains close breeding relationship in descendant generations to a particular ancestor.

Locus: Position of a single gene on a chromosome.

Mendel: Gregor Johann Mendel (1822-1884), Austrian Monk and founder of principles of genetics.

Mutation: Heritable change in DNA that alters genetic information carried by cell.

Oncogene: Cancer-causing gene.

Phenotype: Observable characteristics determined by genotype + environment.

Polygenic: Trait for which variation in phenotypes is influenced by genes at many loci.

Population Genetics: Study of genetics in populations and how frequencies of genes and genotypes change from one generation to the next.

Wright: Sewall Wright, pioneer in population genetics, formulator of 'Wright's Coefficient,' a statistical estimate of genetic homozygosity by descent in a gene pool.

companion owners, not breeders? What influence can we possibly have on the future genetic health of the breed we love?

It is essential for pet owners to understand that we *do* make breeding decisions, at least our dollars influence and finance breeding decisions, and therefore we are crucially important in the larger picture of the Scottish Terrier gene pool. Our numbers and the combined economic muscle we represent in today's marketplace identify us collectively as not only a player in the contemporary drama of breeding and the deconstruction of the Diehard, but as prime movers.

Before turning to the larger picture of breeding and the marketplace, it is important to touch on a minimal survey of genetics fundamentals. Genetics is complex and most of us get glazed eyes at the mention of gametes, chromosomes, and heterozygosity. To help our Scotties it is not necessary that pet owners become geneticists, but it is necessary for us to know more about genes, breeding, and genetic disease than we do, in order to become the advocates our dogs need.

Genes & Reproduction

Genetics refers to the encoded 'instructions' carried within the cells of all living organisms which control all biological processes. Analogous to the software which 'tells' computer hardware what to do, these encoded instructions are responsible for maintaining the continuity of an individual and its species or breed and also for expressing the differences between individuals within a breed.

These encoded instructions are located within *genes*, which are long double strands of *DNA* (*deoxyribonucleic acid*) made up of two long sugar *molecules* linked by pairs of smaller molecules called 'bases' or '*nucleotides*.' It is the sequence of these nucleotides that encodes the instructions carried in the gene. The gene is the basic unit of inheritance and the general rule is that many genes control a single trait.

In canine reproduction, the inherited genetic material consists of a chromosome from the mother and one from the father which, when combined in fertilized sperm and egg, then generates a new complete mixed set of 39 chromosomes—the full number in the canine species (wolves also have 39 chromosomes in their genetic 'set' and therefore can breed with domestic dogs; foxes, however, have only 19 chromosomes and cannot).

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Defective genes as well as good ones are passed on in breeding. Every individual, whether man, mouse or Scottish Terrier, carries a few such dark secrets in its genetic closet, according to geneticist John B. Armstrong, of the University of Ottowa. Defective genes are mutations caused whenever something disrupts the exact replication of the DNA during cell division. Not all mutations are harmful but only a few changes in the base sequence of nucleotides can render the 'instructions' in that gene useless or impared, and no matter how slight the modification, it changes the information carried in that gene so it is a different version. Different versions of the same gene are distinguished as *alleles*. When two alleles are the same, the individual is said to be *homozygous* for that gene. When the alleles are different, it is said to be *heterozygous*.

Though there are potentially a large number of instruction-sets, or alleles, possible for each gene, by far the most common are those that prevent function entirely. Therefore, typically there are normal genes, called *wild-type*, and 'no-function,' or *null* genes. But no matter how many versions there are in a population, an individual can carry only two of them as instruction-sets per location on the genetic strand—one from the sire and one from the dam. Since each pup inherits blueprints from each parent, if one gene in a combined pair is defective the good gene paired with it is usually sufficient to provide normal genetic function.

When someone speaks of a genetic abnormality being "carried" by an individual or line of dogs they mean that a mutant gene is there, but it is recessive. Unless there is a genetic-marker test available to identify the gene itself you cannot tell by looking at the carrier that it is any different from an individual with two normal copies of the gene. Nevertheless, the mutant gene will be passed to some of its offspring. This is why breeding which confounds phenotype (appearance) and genotype (full genetic makeup) is breeding blind.

Genes & 'Tossed Saled'

The jeopardy comes when two individuals carrying the same defective gene, perhaps inherited from a common ancestor, mate. In large, natural populations which randomly breed without interference, Nature minimizes the risk of such matings by constantly tossing the genetic salad to achieve optimal genetic distribution and diversity. In addition, the brutal non-survival of the unfit cleans up the gene pool.

Nature's genetic 'tossing of the salad' in a species to maximize genetic diversity (*heterozygosity*) is the benchmark of genetic fitness calculated mathematically in the *Hardy-Weinberg Law*, first formulated in 1910. That baseline genetics theory says that in an ideal natural population that is large, breeds randomly without restraining forces, genes lost in the population through natural genetic drift and genes gained by natural mutation balance out across the gene pool and so the ratio of 'plus' and 'minus' accumulations in fitness remain in equilibrium.

Our problem is Scotties are not a Hardy-Weinberg population (no actual population is; H-W is an hypothetical ideal). In fact, what we refer to as breeds do not exist in Nature. Our different breeds of dogs, including Scottish Terriers, are genetically engineered 'unnaturals' created and sustained by interfering with Mother Nature's normal tossing of the canine genetic salad.

Mother Nature's 'revenge' comes when we discover that the same diversity-reducing breeding practices that double up on coveted 'typy' genes also simultaneously double up on whatever particular hidden defects those dogs carry in common.

> "Each individual within a breed also carries it's own kind of load—four or five genes for potentially fatal diseases or defects. These are called 'lethal equivalents.' In most cases they will not affect the individual carrying them because a single allele, or form of the gene, will be insufficient to cause the problem. But when relatives are mated, the odds of matching up those alleles increases as does the frequency of the disease." (C.A. Sharp, editor of ***Double Helix Network News***, *"The Downside of Inbreeding: It's Time For a New Approach"*)

All this survey of reproduction and genetics adds up to one thing for the pet owner: all Scottish Terrier breeding is risk management because it is an assault on Nature. Natural genetics moves constantly towards diversity (*heterozygosity*); Scottie breeding moves constantly towards uniformity (*homozygosity*). Our dogs' future depends on whether we can find ways to maintain our breed while nurturing genetic diversity in our gene pool.

Immunity and Loss of Diversity

To understand the natural process of gene-pairing in Scottie reproduction and to grasp the risks of pairing two disease-genes in the genetic makeup of offspring, is to see at a glance why the essence of Scottie breeding is risk management and why anyone who breeds our dogs without knowledge and risk-assessment skills, detailed health information, and deep commitments to long-term breed welfare, is a menace to the Scottish Terrier gene pool.

But seeing the risk of pairing disease-genes through inbreeding is only the more obvious of our deconstruction worries. Less obvious, but arguably more damaging in the long run, is the loss of genetic diversity as individuals acquire the same genes from both parents as a result of the parents' common ancestry.

Nowhere is the impact of loss of genetic diversity more telling than in the immune system. Among the newer frontiers of genetic research is study of the Major Histocompatibility Complex, MHC, which in dogs is a complex (mapped as DL-

> ***Now we can better understand why our dogs succumb in rising numbers in a toxic world. We've weakened their immunologic system by stripping it of genetic diversity over a century of inbreeding.***

A) of closely linked genes best known for its role in the immune system, the production of antibodies, and the rejection of infectious agents. The basis of an organism's immunology is the ability or inability to discern self from non-self and to regulate a normal immune response. With the advent of DNA profiling these MHC genes have been mapped to a tightly linked continuous gene region which generally segregate as an entity in reproduction.

What we know is that the immune system of all animals is dependent on genetic diversity. The environment and its pathogens are constantly evolving so a species must have a highly diverse and adaptible immune system to survive. There is not enough room on the chromosomes for a ready-made complement of pre-formed antibody genes to meet every infection contingency. The amazing answer to this crisis is our dogs' immune system (MHC) shuffles its own DNA (the only cells in the body which do this), each B-cell and T-cell (the response engines in the immune system) splicing together out of tiny gene pre-elements the whole gene required for immunological self-defense.

What is crucial for us to understand is that the more genetic diversity there is at the MHC site the wider the immunologic response the individual is capable of mounting, on the other hand, the more duplicate alleles at the MHC site the narrower range of response an individual can mount when challenged immunologically.

Genetic diversity at the MHC site in the canine genome was recently analyzed for the first time using microsatellite markers at University of California, Davis. 28 Dog breeds, representing the seven recognized breed groups of the AKC, were tested for genetic diversity. Their findings: (1) the smaller the breed population and (2) the longer registered by a kennel club, the more depleted the gene pool (D.N. Irion, A.L. Schaffer, T.R. Famula, M.L. Eggleston, S.S. Hughes, N.C. Pedersen, *Analysis of Genetic Variation in 28 Dog Breed Populations with 100 Microsatellite Markers*).

All of this is directly relevant to our Scotties because now we can better understand why our dogs succumb in rising numbers in a toxic world. We've weakened their immunologic system by stripping it of genetic diversity over a century of inbreeding. Like the Bernese Mountain Dog—one of the breeds analyzed in the University of California study—our breed population is small and like the Berners, Scotties have high incidence of cancer. Most important, like the Bernese Mountain Dog, Scotties are closely inbred and it is inbreeding which robs dogs of vital diverse alleles. However, we're worse off than the Berners, in two regards: Scotties are one of the early breeds in the AKC, hence much longer subjected to deleterious effects of inbreeding for rigid phenotype, and unlike Bernese Mountain Dogs, Scotties have no open health registry to help

us breed against known diseases.

How Inbred Are We?

Inbreeding produces animals that acquire the same allele or gene from both parents as a result of their common ancestry. While inbreeding does not itself produce disease, it increases the probabilities of pairings of suboptimal alleles and worse, of doubling up on any seriously deleterious traits carried by a shared ancestor.

Geneticists can predict negative health effects in a population based on its degree of inbreeding. Dr. John Armstrong, researching Standard Poodles, found that the least inbred group (cumulative coefficient of inbreeding lower than 6.25% over 10 generations) lived longer by more than three years than the most highly inbred group of Poodles (COI 35%), and that a 10% increase in COI will likely reduce litter size by about 7% (John Armstrong, *Population Genetics and Breeding*, July 2000).

> ***[We need] an era in which pet owners pull the plug on irresponsible breeding and in return responsible breeders give more to an eager Scottie public than the crumbs from their private breeding agendas by producing healthy, genetically diverse, long-lived Scotties in quantities modulated to public needs.***

Geneticist Sewall Wright, one of the pioneers in population genetics who first investigated 'genetic drift,' formulated an equation for estimating degree of relatedness or inbreeding in a population. Wright's "*coefficient of inbreeding*" is the probability that at a specific locus on the genetic strand both alleles are identical and were obtained as replicates of a gene from the same common ancestor. Said another way, Wright's Coefficient is a statistical estimate of how much or how little genetic diversity there is in a gene pool. COI is a complicated statistical formula, but essentially it assigns a value between 0 and 1 as the degree of inbreeding, called the *inbreeding coefficient*, where 0 indicates that the animals have no common ancestors.

Knowing a proposed breeding pair's degree of shared ancestry is crucial to breeding with knowledge instead of breeding blind, crucial to breeding which seeks in managed ways to promote genetic vigor in a gene pool through nurturing diversity. This is accomplished by choosing matings which represent least consanguinity, or shared ancestry. Swedish geneticist, Per-Erik Sundgren, Canadian geneticist, John Armstrong, and German geneticist, Helmut Wachtel, target the ideal purebreed population inbreeding coefficient at less than 10% over 10 generations, and Sundgren proposes a five-generation rule for breeders: the COI increase across five generations should be held to a maximum of 2.5%, or 0.5% per generation.

Trouble is, today's Scottish Terrier breeder cannot calculate whether a proposed breeding raises or lowers our breed's inbreeding coefficient and hence helps or hurts our gene pool's relative degree of inbreeding, *because our breed's average coefficient of inbreeding has never been calculated*.

German geneticist, Dr. Helmut Wachtel (1997), estimates that across all purebreeds the average inbreeding coefficient has reached 14%, with some breeds and breed lines reaching 50% or more. A 14% COI is roughly the equivalent of direct half-sibling mating, and such a level carries about six times higher probability of progeny with hereditary defects than non-related matings, and that prediction assumes only 5% of the population carries the defect.

While we do not know our breed's average inbreeding coefficient because it has never been calculated, we do know from Dr. George Padget's estimates based on STCA's 1995 Health Survey and national veterinary databases that ratios of Scottie carriers of known genetic diseases run higher than Dr. Wachtel's warnings based on a 5% population carrier rate: 8.4% of the Scottie population are carriers of von Willebrand's Disease (bleeding disorder similar to haemophilia in humans), 19.5% carriers of Patellar Luxation (joint disease) and Deafness, 28.6% carriers of Epilepsy, to 29.3% of the population as carriers of Scottie Cramp.

These numbers approach a third of our breed population as carriers—nearly one out of three—for certain genetic diseases. And these numbers from STCA's 1995 survey do not even address Scottie cancers.

My point in all this is that inbreeding is at the heart of the deconstruction of the Diehard and unless we change our thinking about how we breed so as to lower our breed's degree of kinship we're dooming our dogs. A conservation biologist would consider the Scottie with the lowest mean of kinship to be the most genetically valuable in terms of managing diversity in the population. If we would do no harm, we must learn to think as conservationists, too.

But that raises an awkward fact. Demanding that breeders "do no harm" and then failing to provide the conscientious breeder in the trenches with one of the crucial tools for avoiding breeding blind is bad faith. In our case we've left our breeders' toolbox lacking a crucial tool: an accurate benchmark calculation of the average Scottish Terrier inbreeding coefficient, and a computer program such as ***CompuPed***, by Man's Best Friend Software, designed to give quick calculations of Wright's COI on any proposed mating. We've demanded that our best breeders 'do it right' without knowledge of or access to Wright, and that means we've left our best breeding blind in terms of genetic diversity.

We could all wish that a small piece of the current funding the national club is dedicating to genetic marker research

at ever rising costs could be earmarked to do something immediate and of far-reaching health impact: to set up a centralized, accessible database for serving all Scottie breeders with crucial inbreeding coefficient calculations. It could be done without years of waiting for research-results to the benefit of all breeders and all Scotties for under $5,000 dollars.

Pet Dollar$ and Deconstruction

Earlier I referred to the fact that the deconstruction of the Diehard is double-sided, that it is fueled by both the supply side of breeders who breed blind, but also by the demand side of buyers who buy blind.

Scottie dollars in the marketplace represent a prime factor in the ruin or rescue of our breed's gene pool. To make this point dramatically consider this American Kennel Club statistic: 5% of purebred dogs are bred by purebreed clubs; 10% are bred by commercial breeders; 85% are bred by pet owner backyard breeders.

In the context of our breed's genetic predicament these numbers are revelatory. First, they reveal what a small proportion of Scottie breeding is subject to STCA ethics and control—fully 95% of Scotties come from non-STCA sources. This means, if our dogs face a problem from blind-breeding at highest levels as I have shown here and in Part One, they face an even greater jeopardy from 'the blind breeding the blind' when large numbers of casual breeders produce large numbers of our dogs in total ignorance of breed health risks and of genetics.

Now, it might be argued from the perspective of genetic diversity that backyard breeding puts diversity into the gene pool by expanding the breed stock beyond highly inbred lines. There is theoretical merit to that argument, but before we embrace it we need to think very carefully and clearly on this matter. Do we wish to abdicate our Scotties' future to the lowest common denominator of knowledge and wisdom among us, or do we wish to manage our breed's health risks by intelligence and the most informed decision-making? I cannot speak for others but to me, my dogs are family and that means the very thought of leaving their future in casual, uninformed hands is out of the question. As I have shown, anyone who breeds our dogs without knowledge and risk-assessment skills, without detailed health information, and without deep commitments to long-term breed welfare, not just short-term personal goals, is a menace to the Scottish Terrier gene pool and a real harm to the dogs we love.

I need to be explicit. We who do not breed are not off the hook when we congratulate ourselves, "My dogs are neutered; I'm not breeding a litter or two for puppy-fun; I'm not part of the problem"—and then as soon as our beloved Scottie dies we buy another from a newspaper ad or at a pet store, thus fueling the production of Scotties bred in total genetic blindness. *Consumer dollars finance the deconstruction of the Scottie gene pool* and no amount of special pleading changes that fact—our dollars subsidize show breeders whose inbreeding contributes to genetic bottlenecks and lost fitness, and our dollars keep in business puppymills and irresponsible backyard breeding. We are very much a part of the deconstruction of the Diehard, and we need to be a large part of his restoration to genetic vigor and health.

But the AKC statistics of purebreed dog production point also in another less obvious direction, one which reveals the dynamics of marketplace demand which is another key element in our Scotties being bred blind. Looked at one way we see exigenses of production; looked at another way we see the abdication of responsible breeders from the marketplace.

AKC's numbers are based on identities of sources who make puppy litter registrations. Working backwards by way of 2002 annual breed rankings and numbers, the AKC ranks Scotties for 2002 at 44th in breed popularity with 3516 new puppies. Their ratio of breeder sources suggests that the percentage of those new dogs coming from our best breeders amounted to 176 dogs (5%).

These numbers uncover a hidden dimension to the deconstruction of the Diehard. Even if, as Carole Fry Owen insists, the AKC ratios are misleading when applied to a small population breed like the Scottish Terrier, and even if the more accurate 'guesstimate' is that our best breeders produced fully 1000 responsibly bred Scotties in 2002, that adjusted number is still less than one-third of the market demand.

There is in this disjunction of demand and supply a fateful breakdown between responsible breeders and pet buyers which amounts to dismissal of the pet marketplace by our best producers. Even putting the most positive spin on the AKC's 5% estimate it is manifest that our best breeders are breeding with little or no reference to the larger Scottie public's needs. This is a serious problem because you cannot fault buyers of pet store Scotties and then breed with no regard to supplying those buyers' eagerness for a Scottie. Railing against backyard breeders and commercial breeding and all who subsidize them is hollow when those who know and understand what is genetically at stake choose to isolate themselves behind private breeding agendas, thus driving an abandoned Scottie public into the backalleys of puppy production to meet their passion for a Scottie.

Meeting one-third of the public demand for Scottish Terriers patronizes the public by requiring them to subsist on the Scottie-crumbs that fall from the show bench table. That is not right, and is another subtle form of breeding blind—blind to the public interest—to which I referred earlier. Although unacknowledged, this is a large but hidden part of the dynamics now driving the deconstruction of the Diehard, for this abdication from the pet market by our best breeders amounts to an indirect franchise to the very puppymills and casual breeders we all decry.

What is needed is a new era of collaboration and coordination between informed pet buyers and responsible Scottie breeders, a new sense of community built on mutual commitments to do what is best for the dogs we all love. We need pet

owners to pull the plug on irresponsible breeding by putting an end to irresponsible buying, and we need responsible breeders to give back to an eager Scottie public more than crumbs from private breeding agendas by producing healthy, genetically diverse, long-lived Scotties in quantities modulated to public needs.

Conclusion

So what does it all mean? Where do we go from here? How do we make a difference for the dogs we love?

Surely it is clear by this point that we have very real problems in our Scottie gene pool. The important truth on this matter, however, is that irresponsible breeding is no more harmful to the breed we love than is irresponsible buying. The deconstruction of the Diehard belongs to all of us, both breeders and buyers, and all must be part of the genetic reconstitution of the terrier we love.

We need to write on our hearts certain genetics fundamentals, such as the profound difference between phenotype and genotype, the crucial importance of diversity in a population, and the jeopardy of inbreeding. These fundamentals must inform our thinking as we move towards reframing our Scotties so we begin to see and act as biological conservationists and not merely as 'users' of our breed. Our new thinking, informed by principles of population genetics, must turn into advocacy in Scottie circles for health and fitness over glamor.

We must speak up and speak out for an open health registry for our breed in which all Scotties are registered, the good, the bad, and the ugly. At every turning in my investigation of our dogs' genetic predicament, whether one is calculating inbreeding coefficients or making risk-assessments for breeding against known diseases, everything depends on complete and accurate information on the dogs involved. No one disputes such registries are imperfect. In a finite world every 'solution' is imperfect. But whatever its inadequacies, the blank alternative is worse: patch-work ignorance and groping blind. The truth is, a Scottie open health registry has not been tried and found wanting; it has been found difficult and left untried.

Finally, we must resist contemporary pressures to balkanize the Scottish Terrier community into factious self-interest groups nursing private agendas and public suspicions. Divided we will not conquer; we will squander our Scottish Terrier heritage. Breeders need informed, committed pet owners and pet owners need breeders who relate to the public and "do no harm."

Most of all, our dogs whose future is in our hands need friends who will work together for the good of the breed. Facing killer cancers and tough odds, our Scotties need all the MHC genetic diversity they can get to bolster their immunity against tomorrow.

Our Scotties did not ask to fight the fight of their life at 18 to 1 odds against them. We put them in that predicament with the best intentions in the world.

True friends of the Diehard will not abandon him to face those odds alone. We will do what it takes to give him a brighter genetic tomorrow.

[*This is Part Two of a three-part series on Scottish Terrier Genetics and Health. Part Three will focus on an 'action plan' for the future, with special reference to the pet owner's tool kit for contributing to the improvement of Scottie genetic health.]

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