# Research on Canine Longevity & Dog Breeding

By: Liz Sullivan

Many Boxer breeders are concerned about the apparent decrease in the average life span of our breed. But has the average life span of the Boxer really decreased and if so, what can breeders do to reverse this trend? John Armstrong, Ph.D., of the University of Ottawa dept. of Biology, a geneticist and a dog fancier, is currently engaged in a research project on canine longevity and its genetic components. Dr. Armstrong is head of the Canine Diversity Project and the list owner of the Cangen list (Canine Genetics list). I had an opportunity to speak with him recently about his research.

**LS:** Dr. Armstrong, many breeders, not just of Boxers but of a number of purebreds, are concerned by an apparent decrease in their breeds average life span. Is this what prompted your research and how do you go about proving if this assumption is true and if it is, what may be causing it?

**JA:** First, we need to establish what the reasonable life expectancy for a domestic dog is. This is not information readily available, as neither owners nor veterinarians are obliged to report deaths, and the kennel clubs keep no records. Where such data exists, it has been collected by surveys, and the results depend on how representative the sample is of the population. For example, a Swedish study (Bonnett et al., 1997) based on insurance claims ignores all past age 10 as they are no longer insurable, while an American study (Patronek et al, 1997) is based on deaths at veterinary teaching hospitals and will likely not include many that die of old age.

Breed health surveys avoid these biases, but may be biased by either under-reporting of dogs that lived relatively problem-free lives or, particularly when it comes to looking at longevity earlier in the century, "selective memory" -- i.e. ones that died very young have been forgotten. Furthermore, most breed surveys of health that have led to longevity estimates don't analyze the data correctly. In fact, they generally underestimate the median lifespan because they do not account for the dogs that are still living while including contemporaries that have died.

Some of the questions I would like to attempt to answer include the one of whether mixed breed dogs live longer and also whether smaller dogs live longer. Patronek's study seems to support these beliefs. We started with the Standard Poodle (because I am a poodle owner and the subject came up on one of the discussion groups), and expanded to include Clumber Spaniels and Australian Shepherds because of interest expressed by individuals active in these breeds. I won't claim that these are representative of all purebred dogs, but they are all approximately in the same size range while having quite different breed histories. Clumbers and Poodles are probably among the least likely large dogs to be maltreated. (When was the last time you heard of a Poodle being chained outside during bad weather? It may happen, but not often, and probably not by people who are involved with their dogs to the extent that they participate in Internet discussion groups or would visit the Diversity web site.) Both are old breeds, but the Clumber has never been as numerous and went through a serious bottleneck at the end of WW II. In contrast, the SP has a much broader base, but a serious problem with popular sires (and a popular kennel) that has reduced genetic

diversity considerably. Aussies are a much more recent breed, at least with respect to kennel club recognition and one might hope that they are more diverse.

My working hypothesis is that the major factor affecting purebred longevity is inbreeding depression. The history of the breed will dictate the inbreeding and thus the lifespan to a greater extent than other factors, including size. One reason large dogs live shorter lives may be that they are often less numerous than the smaller dogs and are more subject to abuses such as the overuse of popular sires.

Once the stud book is closed, inbreeding goes up -- even if breeders are not deliberately inbreeding -- simply because there are not enough dogs in a breed for every dog to have unique ancestors. If dogs are living shorter lives than they once were, this is likely part of the reason.

**LS:** How have you conducted your survey to collect relevant information?

**JA:** Data on these three breeds has been collected almost exclusively over the Internet with the project being advertised on breed-specific discussions groups and in breed newsletters. There is no fool-proof way of establishing whether this sample is representative, but we now have on the order of 10% of the registered Clumbers from the last 25-30 years and probably a similar percentage of Standard Poodles. (The exact number of SPs is unknown because the poodles are not registered by variety. The number of Aussies is also uncertain because there are several registries.)

We have also been collecting data on a potential control group of mixed breed dogs. This last group is not intended to be representative of mongrels, but rather the potential life expectancy of a mixed breed dogs owned by a caring and responsible pet owner. Although this is an assumption, the responses come from visitors to the Diversity site and from those who own one of the survey breeds.

**LS:** Have you reached any conclusions regarding the data you have already studied?

**JA:** My expectation is that the mixed-breed dogs should live the longest and the Clumbers the shortest as they represent the lowest and highest inbreeding, respectively. The data collected to date supports this expectation.

Perhaps the most significant discovery, however, was that the survivorship curve for Poodles inbred to less than 10% (10 gen. calculation) is a close match to that for the Mixed group, with a median of 14 yrs. In contrast, those over 30% have a median of about 11 years. This is longer than the average Clumber (COI 24%) lives, but there are undoubtedly other factors involved.

The other trend of interest is the reduction of 1-1.5 yrs from the recent to the early poodles. However, there could be reasons for this in addition to higher inbreeding. For one thing, current dogs are likely exposed to more environmental toxins. Another factor could be "selective memory". People may have forgotten

some of the dogs that died young and tend to remember the ones that lived a long time.

**LS:** There seems to be a rather distinct correlation between inbreeding level and longevity, has this held true for all the breeds you have studied?

**JA:** The Clumbers do not have a wide enough range -- 15-40% compared to 1-70% for the poodles -- for a trend to be apparent. Unfortunately, we don't have enough pedigree data yet for the Aussies.

When you compare the recent to the older poodles, the inbreeding actually appears to go down, but this is misleading. We are comparing pedigrees for their last 10 generations rather than from a fixed time point. If I go back 15 generations from the '90s I get back about as far as 10 generations from the '70s and the average IC increases from ~ 17% to 22%. However, I do not feel this is large enough to account for the difference.

A preliminary analysis suggests that longevity can be inherited. This is independent of the inbreeding effect.

**LS:** Dr. Armstrong, a lot of breeders struggle when it comes to the genetic implications of conditions we see in our dogs, especially if we don't have a background in scientific training. Earlier books on breeding that were directed to dog breeders advocated inbreeding and linebreeding as the preferred method. Almost every author stated that "doubling up" on the preferred traits would "fix" them and that by doing so, you would bring problems to the surface, where you could work to eliminate them. The stated goal was to "purify" your line in order to be successful. People followed this advice to a large degree because it seemed to make sense. But now, if you look around, breeds every where are in trouble. Genetic diseases seem rampant and even the popular press is full of articles criticizing purebred dog breeders. Why didn't this work? Could you give us a brief explanation of how traits are inherited?

**JA:** When Mendel established the basic rules of genetics, he based his conclusions on a study of the inheritance of seven easily-recognized morphological traits in the pea -- things like height, flower color and seed color. There were two alternatives for each trait (tall vs. short, purple vs. white, etc), but neither was deleterious, and there were no other complications such as multiple alleles or incomplete dominance.

If we were breeding peas instead of poodles (or Boxers), the popular "type" might be tall with yellow seeds and purple flowers. If this were the overwhelmingly preferred type, some of the other phenotypes might eventually disappear. Would it matter if green peas disappeared? You might think "no" (unless you were particularly fond of green), but the answer is not that simple. It depends on what other genes are closely linked to these traits, how large the population is, and how rapidly this occurs.

All populations of sexually reproducing organisms have some genetic variation. In mammals, around 10% of the genes are estimated to be polymorphic. By that we mean that they have more than one common allele. Many of these are like the traits Mendel studied in the pea, resulting in phenotypic differences that do not

impair survival and reproduction. Lets call these Group A variants (or mutants, if you prefer). The remaining 90% or so of the genes are also susceptible to mutation, but the mutant alleles are generally found at frequencies below 0.1% (1 in 1000) and are frequently deleterious (lets call these Group B)

All animals are believed to carry several deleterious recessive alleles. This is called "genetic load." The unanswered question is whether this is in the form of 3 or 4 recessive lethals, or a greater number of genes with alleles that diminish fitness but are not individually lethal when homozygous. This group may include subtle differences detectable only with sophisticated biochemical techniques.

When breeders, be they of peas or dogs, attempt to create a line (variety, breed), they are focused on a particular set of the visible (group A) traits. Ideally, none of these traits reduce fitness. If a breed is founded by 100 individuals, we should eliminate 90% or more of the group B mutations, but will boost the frequency of those carried by the founders by 10 fold or more. At frequencies now on the order of 1%, these should still not be a major problem. However, most populations are genetically smaller than the actual number of individuals due to unequal contributions. For example, if one popular sire services 10% of the available bitches, 10% of the males will not be bred, and their genes will be lost. If they include some alleles that are not well-represented in the breed, there is a risk these alleles may be lost. Even at the replacement level -- 2 progeny replacing 2 parents -- there is a 50% probability that one allele from each parent will not be carried by either of the progeny. If there is only one son or daughter, half of each parent's collection of genetic recipes will be lost. Of course if all were either lethal or harmless, we would not miss the former and the loss of the latter would only matter esthetically.

The need to maintain relatively large, diverse populations arises largely from the existence of mutations that diminish fitness by a small amount. The smaller the population, the greater the chance of one allele being fixed (taking over). If a mutation reduces fitness by only a small amount, the loss of the better allele may not be noticed. As suboptimal mutations accumulate over time, we may, however, begin to notice that our dogs don't live as long and are not as healthy as in the "old days". In a large population, there is less chance of any allele being fixed. Consequently, some individuals will be healthy, and some may not be, but as long as we do not favor the latter, the survival of the population should not be at risk.

**LS:** Right now a great many Boxer breeders are trying to deal with a very serious heart condition in our breed that is certainly affecting it's longevity. Although there are other cardiac conditions that are present, the one most troubling is Boxer Cardiac Conduction disease. Until recently it was referred to as Boxer Cardiomyopathy but as more information about it has come to light many in the Boxer community are using this alternative term to distinguish it from Dilative Cardiomyopathy. There are three things that have made this particularly difficult to deal with; first, many dogs are asymptotic until they drop dead unexpectedly. Second, there wasn't any good screening procedure available until last year when the researchers at Ohio State University told us that a 24 hour Holter monitor test was the best way to screen for it. And third, it is often a late onset disease, which appears after a dog has been bred.

Additionally, Dr. Kate Meurs (the head of the research project) told us this year that they aren't sure that a Holter reading of clear this year will accurately predict future health. Furthermore, no one is sure exactly how many ectopic beats in a 24-hour period are indicative of the condition. Because we don't yet have any concrete answers many breeders are understandably worried about the best way to deal with this disease.

As a geneticist, if you were advising the American Boxer Club, what would you tell it's members?

**JA:** Share information.

Get numbers. If the Holter test looks promising, pursue it. Get a second opinion on the inheritance. (It may make no difference to how you proceed, but it can't hurt. I would suggest this no matter who did the initial analysis.)

Share information. One approach may be to not breed progeny until parents are an age where they should have developed the problem. If it normally appears at 4 or 5, don't breed before they are 3. By the time the progeny are 3 the parents will be 6. (Obviously this doesn't work if a substantial number don't show a problem till 8 or 10.) Whenever everyone rushes to breed to the currently popular stud, and many are two generations down the road by the time he is 4, there is a potential for disaster.

Share information. I can't say it too many times. When breeders start accepting that there are no "perfect" dogs, that genetic problems can occur in anyone's line, and that they don't have to hide it or face ruin, then they will have the power to bring any problem under control.

**LS:** Some people have suggested the best course of action would be to get as many Boxers holtered as possible so we can find what the breed average is. And then, to try to use those dogs that are in the upper 50% percentile for breeding (I believe this is the approach advocated with things like hip dysplasia). They are concerned that eliminating too many dogs from the gene pool would cause more problems in the future. How do you feel about this?

**JA:** I agree that testing a fairly large and representative sample (different ages, both sexes, and different lines) would be a good idea. If there is an indication that clear now does not mean clear forever, then it should be an ongoing study, where the same dogs are assessed yearly. I do not think it would be wise to eliminate those who do not score in the top 50%, especially if there are no long-term guarantees.

I suspect that if there is a range in scores, and breeders start publicizing the results, you won't have to do much else. The conscientious ones will look for mates with the best scores, while those who breed carelessly will continue to do so. The problem may be preventing the overcautious from using only the top 10%

If the score turns out to be a reliable indicator, then setting a suggested cutoff score should be dictated to some extent by the effects it would have on breed

diversity. I would proceed very cautiously and 50% seems high. You really need to do a study on genetic diversity in the breed before you get carried away with extensive culling.

**LS:** There seems to be increasing evidence that heterozygosity, especially with regard to immune system function, may be optimal for the overall health of an animal. How can breeders try to get the most diversity in things like the Major Histological Complex (MHC) without losing breed specific traits or traits particular to their own line that they value?

**JA:** With respect to the latter, the advice I always give Poodle breeders who ask which dog to breed to is to evaluate their own dog as objectively as possible with reference to the breed standard (or their vision of the ideal poodle). Then look at as many potential mates as possible evaluating them in the same way. Rank them in order of closest match to the most distant (which may not be the same as best to worst). It is alright to try for improvement, but avoid many and/or large differences. (You can't make a silk purse...). Give me your list and I will determine how closely they are related. Then the breeder has to decide how much weight to place on type and how much on health. In my opinion, little weight should be given to show titles and none to popularity or how impressive the pedigree looks. (If you want evidence that it works, read "Inbreeding and Diversity" at the Diversity web site.)

Breed specific traits should have been fixed when the breed was established. If they prove difficult to maintain, then something is wrong. You are probably trying to do something that goes against thousands of years of canine evolution. The look you want may be due to an inherently unstable combination of genes, particularly if the breed was established from dissimilar founders over a short period.