

# Progressive Retinal Atrophy and Borzoi eye lesions

by Bonnie Dalzell, MA Silkenwift Borzoi

**A** brief review of what is known about Borzoi eye lesions from Dr Gerg Acland, who has worked not only with Borzoi recently but also with some of the original material from the California 'PRA' Borzoi in the 1960s.

The lesions commonly seen in Borzoi eyes are also seen in Border Collies and other herding dogs, Siberian Huskies, Samoyeds and Greyhounds. In most cases the lesioned dogs are athletes coming from situations in which a fairly large population of dogs is housed on property that has had numerous dogs for a long while. While there may be an inherited component to the development of these lesions the environmental effect is so overwhelming that without detailed studies it is impossible to see a clear cut pattern of inheritance. In addition, the fact that the lesions are seen in the most athletic individuals—not the passive non-athletic individuals—makes it difficult to study dogs kept in typical research environments where the dogs are mostly in runs and they never participate in high levels of athletic performance.

These eye lesions are frequently unilateral and also they are more common in males than in females (in the Borzoi sample the frequency was 4 times more common in males than females). Often they do not progress or if they do progress they progress slowly and vision may not be lost until the dog is 10 or more years of age.

In dogs in general two major patterns of Progressive Retinal Atrophy are observed. Early onset PRA leads to blindness by 2 or 3 years of age or earlier and has been identified in Irish Setters, Norwegian Elkhounds and Rough Collies. In each breed it is due to a different defective gene and the degeneration is visible in puppyhood. Late onset PRA exists in a great many breeds including Poodles, Labrador Retrievers, Cocker Spaniels, Portuguese Water Spaniels, etc. The retinal degeneration is not visible until 2 to 6 years of age and the disease seems to be due to a defect of the same gene in all the different breeds. It is thought that this gene mutated independently in all the different breeds. Since this particular gene is so easy to mutate there is no reason to think that it could not have happened in Borzoi but at present Dr Acland has not seen any cases of eye lesions in Borzoi that he thinks could be due to this gene.

What Dr Acland has seen in Borzoi (and what was originally mistaken for PRA in the California Borzoi) is a condition, probably environmentally induced, that in later stages

looks just like late state PRA. However the early stages are quite different from progressive retinal atrophy. The Borzoi retinopathy can be observed by fundic exam (a examination of the internal portion of the eye with a lens and a light source in which the iris is prevented from contracting by the presence of atropine-containing eye drops). It first appears as multifocal spots of hyperreflectivity on the retina. These spots may be rings of hyper reflectivity surrounding a tiny black dot on the tapetal area of the retina. There may be few lesions or many but generally they are present in or worse in the left eye and four times more common in males than females. They are also more severe in males than females.

If the eye accumulates a threshold level of these lesions the retina will degenerate and die—the retina may detach and cataracts will develop. Severely affected dogs may be blind by 6 years of age.

There is a strong suspicion that the agent causing these lesions is a parasitic agent of some sort. It would have to be smaller than a red blood cell and be more likely to be picked up by dogs living in situations where they are on grass or dirt and there have been large numbers of dogs present over a period of time. Both toxoplasmosis and round worm larvae have been proposed but neither has ever been actually found to be present in a lesioned eye. At this stage all that has been seen is the scar that is left behind.

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