our experts speak on current Saluki health issues

I realize that Classic hasn't had any health info specific to Saluki concerns in quite awhile. I receive many e-mails with health-related questions - both personally, as a long-time breeder, and as editor of the Classic. On many of these topics I have only the most basic knowledge. So, rather than answer these inquiries myself, I decided to ask some of the veterinary professionals within our breed. Dr. Van Arsdale, Dr. Hinsch, Dr. Sist, and Dr. Gonda have been kind enough to address the issues of monorchidism and mange - neither related problems, but both topics I have been asked about constantly this past year.

In ensuing annuals, I will try to get professional fanciers to answer about some other issues. If you have a specific question, please write to me and perhaps they will address the problem. –Sue Ann

An e-mail conversation between Sue Ann (SA, *italics*) and Jo-Ann Van Arsdale, DVM (JA)

SA: Monorchidism seems to be appearing more and more in many lines . . . I realize that there has been no definitive answer as to transmission.

JA: IMO, the genetics are well known, but lots of people prefer to blame other "causes".

SA: However, when one stud dog or brood bitch produces this in a number of litters, one can guess this is a hereditary problem . . . what do you think? What would you do?

JA: Since I've just had my first one in 40 years I made serious inquiries to the breeder of a line I had never used and much admired but was now in the pedigree of a litter much desired by me. I'll shelve the affected male and then breed the litter sisters to a pedigree known to be free of the problem.

SA: And what should a one-time breeder or novice do when selecting breeding stock?

JA: Stay away from dogs with a known history. Inquire about all the littermates of their chosen dogs that they have never seen. If they learn about the problem in littermates or offspring, "run" as fast as they can from those bloodlines if they really care about testicular problems in dogs. A monorchid dog is usually just as fertile as a normal male, and if he can win big in other venues than the show ring, some people probably don't care about the testicle count. Since dogs "litter," breeders may decide to take the chance that their favorite dogs (for whatever reason) are worth breeding and then "sorting". That's okay as long as everyone who gets the pups knows the history, so they can make informed decisions.

SA: Secondly, mange . . . I realize this also is an after-effect of immune system problems, however, my own vet screamed at me never to use the puppy I got that developed a spot of demodex at seven months after a trip to the National. The specific spot was treated never to return. None of his offspring have ever had another problem. However, it has come to my attention about a stud dog that produces mange in every litter. What is your feelings on this?

JA: A lot of pups under stress get transitory demodex lesions. No biggy. It's the pup or dog that has severe generalized intractable demodex that is a huge problem. You can easily assess your dog's immune system with a simple lab test: immunoglobin diffusion. It's imperative to breed healthy dogs that produce healthy dogs, otherwise why bother? We now know a lot more about immune-mediated disease in dogs, as well as other species. IMO, no dog with a proven immune-mediated disease should ever be bred. Doesn't matter if hyper vaccination is suspected as a part of the problem. Once they're diagnosed, out of the gene pool. I include immune-mediated hypothyroidism on this list.

There are a lot of serious problems in the purebred dog arena. IMO the show ring demands that dogs be bred who have defective and crippled skeletal systems, too much hair to survive without intense grooming, stupid ears that will always be a problem, eyes asking for damage, and teeth that require a huge investment in veterinary care. And, because of "dog shows," people flock to pay serious money for a pet that is going to cost them a lot over it's lifetime just because they wanted something that looks cute on AKC TV.

My latest soapbox issue is dogs with allergies. Food allergies, pollen allergies, contact allergies. With the advent of Frontline and Advantage, we can no longer blame everything on fleas. So what's up with this problem? Would you breed a dog with asthma?

Sue Ann,

You asked the question about monorchidism . . . problems with testicular non-descent. First off, everything in our lives ultimately can be traced back in an individual (human and/or dog) to their genes. There are those out there, professionals and fanciers, who will categorically state that monorchidism is a genetic recessive. It seems to act so in some breeds. One of the major problems is that it is sex-limited and therefore seen only in the male. To state that it is a simple recessive condition seems to belie the facts associated with the process of testicular descent. If one goes through the literature, one finds that some of the latest work on development of the male reproductive system describes the formation of the testes and associated ligaments holding them in place. In some genetic studies done of mice, the genes associated with testicular descent were "knocked" out. When they studied descent of the testes, they found that there was a gene which was involved in the dissolution of the anterior ligament and another in the formation of the posterior ligament (gubernaculum). It is the latter which shortens to pull the testes down into the scrotal sac. Both dissolution of the anterior and shortening of the posterior must occur for the descent to occur. This then is part of the genetic control.

BUT, for descent to occur, the juvenile testes must produce a certain amount of testosterone and there must also be present some insulin-like growth factor. Thus, there are a multiple of factors involved in testicular descent.

Do we breed monorchids? According to AKC rules, we cannot show such but nowhere does it state we can't breed them. As long as there has been the descent of at least one testicle, the animal is capable of producing sperm. Sperm plus eggs = puppies. The fact that some lines seem prone to produce males in which one/neither testicle descends should make one take notice and decide if such a characteristic is worth considering as an exclusion factor.

Ultimately, it is genetic . . . its means of transmission we cannot categorically state in Salukis.

Gertrude Hinsch, PhD

Cryptorchidism in Dogs

by Casey Gonda, DVM, MS, Diplomate Am College of Veterinary Internal Medicine

Cryptorchidism is the term used to describe the failure of one or both testicles to properly descend into the scrotum. The defect is genetic and inherited as a sex limited recessive trait.

In most puppies, one or both testicles can be palpated by 10 days of age and, in most cases, both are present by 45 days. There are two "rings" through which the testicles must pass during their descent into the scrotal sac. In very young puppies these rings are still open wide enough to allow the testicles to move back and forth, especially if the pup is cold or excited. In general, by six months of age these rings are closed and the testicle large enough to prevent ascent through them. Because of this, most puppies would not be considered true cryptorchids until this age.

Drugs and surgical techniques have been used in an attempt to "pull" an un-descended testicle down, but such methods are highly discouraged in breeding or show dogs.

The difficulty in screening for cryptorchidism, if there is no direct evidence of a source (affected sire), is two-fold. First, because it is a sex-limited trait, it remains "hidden" in the carrier bitch due to the fact that she has no testicles TO descend. Secondly, because it is thought to be a simple recessive, the trait can remain hidden and be passed for generations without a breeder's knowledge. Remember, it takes both sire and dam to have a pup that demonstrates the trait. To determine the carrier state in a female, she would have to produce at least 40 male puppies who live to be six months of age to clear her as a carrier for the trait!

Demodectic mange

by Casey Gonda, DVM, MS, Diplomate: Am College of Veterinary Internal Medicine

What is it?

Demodectic mange, or demodicosis, is a skin disorder caused by the mite Demodex Canis. The mite is considered a normal inhabitant of the dog's skin and can be seen incidentally in skin scrapings taken from healthy dogs. Overgrowth of this ectoparasite may result in small, circumscribed lesions, with hair loss, crusty patches of skin, redness and itching. There are two distinct forms of the disease, localized (or juvenile form) and generalized.

The localized form typically presents as one or two small lesions, often on the face or legs. Any dog can acquire the localized form of the disease, but it is most commonly seen in puppies less than 18 months of age (average 3-4 months). In most cases (50%), the lesions regress and disappear without treatment, but in a small percentage of dogs (10%) the lesions fail to heal, eventually become worse and spread to multiple sites. It is this form of the disease that is referred to as generalized demodicosis. It is the most difficult of the forms to treat and is considered to be inherited.

How is it transmitted?

The parasite is transmitted at birth from the dam to her suckling pups.

How do dogs get the disease?

All dogs have demodex mites on their skin, but healthy dogs have the ability to keep the number of mites at a level that prevents the development of heavy infestations. Dogs with underlying diseases, parasitism, cancer, or those who may otherwise be immune compromised (i.e. allergic dogs receiving corticosteroid therapy) have an increased incidence of acquiring demodicosis. The pathogenesis of the generalized form of demodicosis is somewhat complex and still not well understood. In the simplest of terms, there appears to be a relationship between some unidentified "factor" produced by the mite, and the dog's immune system. When the number of mites is low, the dog's own immune system is able to maintain that population and prevent disease. But, as the mite population multiplies in greater and greater numbers, these "factors" are produced in abundance and overwhelm the dog's immune system. These "factors" actually suppress the function of certain cells in the immune system making it all but impossible for the dog to fight the disease. In addition to the damage produced by the mite, many dogs experience secondary bacterial and mycotic (ringworm) infections, which likely contribute to the severity of the disease and complicate treatment.

What dogs are at risk for generalized demodicosis?

Dogs with underlying health issues and purebred dogs with a history of demodicosis in their family are at greatest risk. There are certain breeds (Afghan hound, Doberman Pinscher, Shar-pei and others) with a predisposition to demodicosis. Dr. Ristic, a clinical dermatologist from the Virginia-Maryland School of Veterinary Medicine, stated that the worst case that she had ever seen was in an Azawakh!

Although a hereditary predisposition to the disease has been observed regularly in breeding kennels, the exact mode of inheritance has not been determined. Those kennels that have maintained a strict culling program have substantially reduced or eliminated the disease. It is the opinion of the American Academy of Veterinary Dermatology that all affected dogs and bitches are neutered to prevent the perpetuation of the disease.

How do I know if my dog has demodicosis?

Any angry looking, non-healing spot on your dog should be examined by your veterinarian, in particular, a puppy or adult with multiple spots. He or she will take a careful history and scrape the lesion for examination under the microscope. In addition, your veterinarian will likely do a thorough physical examination and depending upon the age of your dog, may recommend certain blood tests or imaging studies (i.e. radiographs/ultrasound). Demodicosis can affect dogs of any age, especially those who may have underlying diseases.

Can it be treated?

In most cases the demodicosis, especially the localized form, can be controlled and/or eradicated with miticide rinses (amitraz), parasiticides (ivermectin, milbemycin), antibiotics (when warranted) and good owner compliance. However, treatment of generalized demodicosis can be frustrating and last for the life of the dog.

How do you prevent it?

If you have a dog that has been diagnosed with demodicosis, never give your dog any medication without first speaking to your veterinarian. Certain medications may result in a relapse.

Most importantly, because there appears to be a genetic predisposition to generalized demodicosis in purebred dogs, one should not use affected dogs or bitches, littermates of affected individuals, or direct sons or daughters of affected parents for breeding purposes. In addition, affected bitches should be spayed because estrus can trigger a relapse in certain individuals.

Special thanks to Thomas Manning, DVM, Diplomate: American College of Veterinary Dermatology and Dr. Zahranna Ristic who assisted me in producing this article.

Additional comments

by MaryDee Sist, DVM

Dr. Casey Gonda has written excellent articles discussing cryptorchidism and demodicosis in response to Sue Ann's inquiry. I want to stress that both conditions have a hereditary component. The following excerpts are from current veterinary reference texts.

Cryptorchidism

The incidence of cryptorchidism in the dog has been reported to be between 0.8% and 9.8%. There is a definite breed predisposition which supports a genetic base. In general, small-breed dogs are at increased risk compared to medium and large-breed dogs. Since it is an autosomal recessive trait, both parents of the affected offspring are carriers, but it is not known if the female carrier is homozygous or heterozygous for the trait, which makes it difficult to determine her carrier status. The mode of inheritance, including the number and penetrance of genes involved, is undefined.

Other congenital defects that have been reported with increased frequency in cryptorchid dogs include inguinal hernia, umbilical hernia, hip dysplasia, patellar luxation and penile/preputial defects. Retained testes are predisposed to neoplasia. Unilaterally cryptorchid dogs should be castrated to decrease possible transmission of this hereditary defect, and to decrease the predisposition of the retained testis to neoplasia and torsion of the spermatic cord.

Medical therapy for cryptorchidism includes the use of testosterone and human chorionic gonadotrophin (hCG). Dogs younger than 16 weeks of age have the best chance of responding to serial injections of hCG. In one study, 21 of 22 treated pups younger than 16 weeks of age responded with complete descent of the testis, while 28 untreated dogs of a similar age remained cryptorchid if the testes had not descended by 8 weeks of age.

I have known of litters that did not have descended testicles by 10 weeks of age which were treated with hCG and all had descended testicles by six months of age. I question whether the treatment really made a difference. In another study of young pups diagnosed with undescended testicles, nearly half showed spontaneous descent by six months of age. No dog, however, showed testicular descent after six months of age. This suggests that the diagnosis of cryptorchidism should not be made before six months of age. Delayed descent seems to be prevalent in some lines.

Most reproductive experts feel that canine cryptorchidism is best controlled by removing cryptorchid dogs and, ideally, their sire and dam, from breeding programs.

Demodicosis

Demodicosis is an inflammatory parasitic skin disease characterized by the presence of a larger than normal number of demodectic mites. The earliest sign is fur loss only. The initial proliferation of mites may be due to a genetic or immunological disorder. Most localized cases resolve spontaneously. Many treatments that receive credit for curing demodicosis are in reality spontaneous recoveries. Internal disease or malignant neoplasia is often diagnosed in adult-onset demodicosis patients within a year of the onset of generalized demodectic lesions.

Purebred dogs have a much higher incidence than mongrels and certain breeds are predisposed to the development of generalized disease. Factors such as short coat, poor nutrition, estrus, stress, endoparasites and debilitating diseases may precipitate the disease. Elimination of affected or "carrier" dogs (both parents and siblings) from a breeding program greatly reduces or eliminates the incidence of demodicosis. Studies have shown that dogs with chronic generalized demodicosis have severely depressed T-cell lymphocyte responses. The immunodeficiency state is secondary to the disease, but not the cause of the disease. More than half of the dogs with generalized demodicosis have a nonregenerative anemia as well as low levels of thyroid hormones. These changes are generally secondary to the chronic dermatitis and are not autoimmune anemia or hypothyroid disease states.

Lymphocytic thyroiditis is clearly an inherited disorder. This condition, however, is not an indicator of other immune defects. Hypothyroid dogs routinely live very normal lives when appropriately supplemented with thyroid hormones and never develop other immune conditions. The rare polyglandular syndrome, which is an inherited autoimmune human disease where multiple endocrine organs are affected, has not been well documented in dogs.

The American Academy of Veterinary Dermatology recommends neutering all dogs who have had generalized demodicosis so that the incidence of the disease is decreased and not perpetuated. It is strongly recommended not to breed dogs that have recovered from generalized demodicosis or those that have produced affected pups.

*Reference*s

Feldman & Nelson, Canine and Feline Endocrinology and Reproduction

Johnston, Kustritz & Olson, Canine and Feline Theriogenology

Muller, Kirk & Scott, Small Animal Dermatology