THE CHOW CHOW

Genetics & Recommended Breeding Guidelines

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The Chow Chow Club of Victoria

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CHOW CHOW

Genetics & Recommended Breeding Guidelines

INTRODUCTION

In response to Dogs Victoria's 2008 submission to the State of Victoria regarding the proposed amendments to the *Prevention of Cruelty to Animals Act 1986*, and the publication thereof in the March Gazette, each breed club was asked for its response to the amendments and for recommended breeding programs for each breed.

On the following pages are listed those disorders identified in the Chow Chow breed. While this list is not exhaustive, it is one for which there is a general consensus among international researchers and veterinary practitioners that the conditions may have some hereditary significance in this breed.

There are three categories defined by the above mentioned Act and these are outlined below. The last category lists conditions that have been reported sporadically and may possibly be inherited in this breed.

Following within the outline of the three categories are listed the noted diseases and disorders. Information is provided on each disorder/disease within a construct provided by Dogs Australia. Recommendations regarding testing (where possible) and breeding accompany a brief explanation of the disorder/disease and its mode of inheritance.

At the very end of this paper are the source documents used for the information contained herein, a glossary of terms and the genetic inheritance charts provided by Dogs Victoria and the Department of Primary Industries, under which this legislation will be enacted.

These testing and breeding recommendations were adopted by the Chow Chow Club of Victoria in April 2008 as the Recommended Breeding Guidelines to ensure that breeder members of the club were cognizant of both the legislation and the genetics involved and had the necessary information to enable compliance with the often confusing and/or vaguely worded legislation noted above.

This document was re-confirmed by the Chow Chow Club of Victoria's Management Committee in August 2010 for re-submission to Dogs Victoria, in compliance with a request from Dogs Victoria for all breed clubs to provide such guidelines.

The 2010 Management Committee recommends a review and updating of these guidelines every 3-5 years, or as required.

Judith-Ann Robertson, Editor

CATEGORIES OF GENETIC IMPORTANCE

CATEGORY 1. MOST SIGNIFICANT

These are disorders considered relatively common in a breed, and where possible, efforts are being made to eradicate them. These disorders can seriously affect the health of the dog and may require medical or surgical intervention.

CATEGORY 2. OTHER DISORDERS WITH A KNOWN INCIDENCE IN THE BREED

Disorders that occur less commonly or are less devastating than those in the most significant category.

CATEGORY 3. OTHER DISORDERS THAT MAY BE INHERITED IN THE BREED

These disorders have been reported sporadically in the breed, and may be inherited in this breed.

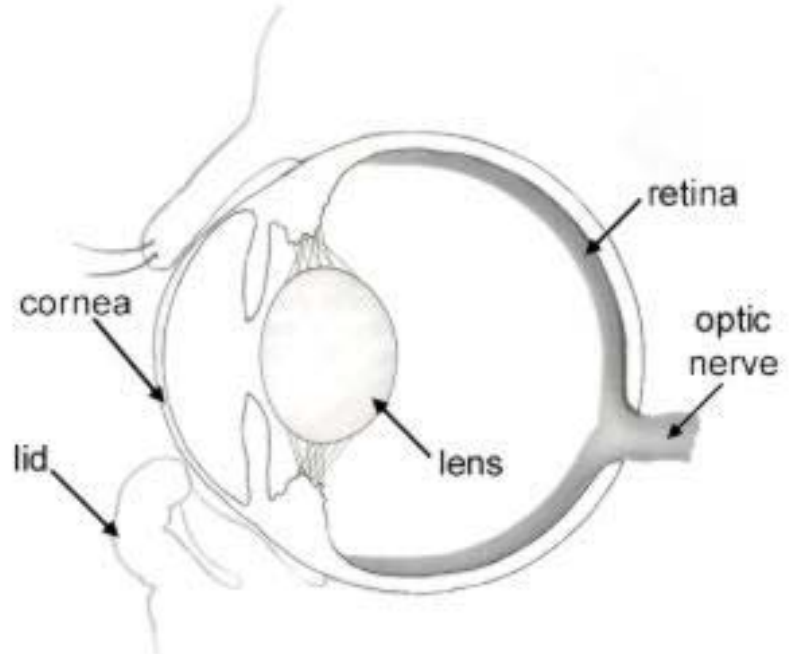
CATEGORY 1: MOST SIGNIFICANT

These are disorders considered relatively common in the breed, and where possible, efforts are being made to eradicate them. These disorders can seriously affect the health of the dog and may require medical or surgical intervention. All three of the disorders identified as most significant for Chows are considered to be ***polygentic,*** indicating that more than one gene is necessary to cause the problem. At this time, there is no reliable method to predict its inheritability.

ENTROPION:

This is a disorder most correctly associated with conformation. Entropion is often incorrectly described as a disease; and while it may lead to diseases of the eye if left unaddressed, in actuality, entropion is a conformational disorder of the muscle that operates the opening and closing of the eyelids (*orbicularis oculi* muscle).

Entropion is the inward rolling of the eyelid, most commonly the lower lid. This irritates the surface of the eye (the cornea) and may ultimately cause visual impairment.



Adapted from D Slatter, Fundamentals of Veterinary Ophthalmology. 1990. Philadelphia. WB Saunders

Entropion is a common hereditary disorder in dogs. In some breeds, entropion results from the selection by breeders and a demand by the public for certain features for prominent eyes or heavy folds of skin on the head. To some extent this may be true in the Chow. However, historical evidence

shows that in spite of the physical attributes of the facial features, both the heavier headed Chow and the very open faced Chow have experienced entropion since the early 20th century.

Furthermore, Chows with perfectly conformed eyelids may develop entropion later in life. This adult onset form of entropion is considered to be caused by environment or trauma and not inheritance; although the shape of the orbital socket lends itself to this condition occurring either early or later in life.

Mode of inheritance

Evidence appears to indicate that entropion is influenced by several genes ***(polygenic inheritance)*** that affect the skin and eyelid musculature, the way the skin covers the face and head, and the conformation of the skull and in particular the orbital socket construction.

Numbers Affected/Rate of Occurrence

Entropion is known to occur in puppies as young as four weeks of age and as late as ten years of age. Rough estimates from the United States indicate that upwards of 50% of the breed may be affected at some stage in the life cycle.

The proportion of carrier and clear individuals in the breed as a whole is unknown and presently undeterminable.

Severity of the end disease

If left untreated, entropion may lead to eye diseases.

Testing/Screening

DNA tests for entropion (and ectropion) do not exist for the Chow Chow at this time.

Periodic veterinary observation is required to determine whether or not the disorder has occurred since the last observation and clearance.

Treatment

If detected early in young puppies, the eyelids can be tacked to allow for a stretching of the eyelid/s and growth in the puppy's head structure. In a large percentage of puppies, this early intervention can circumvent the actual development of entropion in the adult dog. In other cases, entropion is corrected surgically. In young dogs, if possible it is best to delay surgery

until the dog is an adult since the involved facial structures are still growing and changing.

Breeding advice

The genetics behind entropion in Chows appears to be both polygenetic and dominant. Encouragement of breeders to engage in breeding programs that, wherever possible, use non-affected animals for breeding; however, considering the dominance of the disorder, this will require many successive generations to alter the dominance pattern of inheritance.

Caution must be noted that rapid changes to a breeding program to eliminate entropion in a line may actually cause changes in the skull's conformation.

Recommendations

- Breeders be encouraged to have puppies' eyes checked, and tacked where necessary, at about six weeks of age.
- Breeders be further encouraged where possible to use unaffected dogs in their breeding program.
- Breeders be encouraged to provide puppy buyers with information regarding entropion (and other possible eye disorders) and whether or not the puppy's eyes have been tacked.

ELBOW DYSPLASIA

Related terms: *OCD, osteochondrosis of humeral condyle, fragmented medial coronoid process, ununited anconeal process, incongruent elbow*

The term elbow dysplasia refers to several conditions that affect the elbow joint: osteochondrosis of the medial humeral condyle, fragmented medial coronoid process, ununited anconeal process, and incongruent elbow. More than one of these conditions may be present, and this disease often affects both front legs. An affected dog shows forelimb lameness and elbow pain.

These conditions may actually be different manifestations of a single disease process, osteochondrosis dissecans (OCD). OCD is abnormal maturation of cartilage (the specialized connective tissue from which bone develops). While this in an inherited defect, environmental factors such as diet, activity, and trauma also have a role in the development and progression of the disease.

Osteochondritis dissecans (OCD): A fragment of cartilage peels away from the bone, within the joint. Current research indicates that this can be the result of restricted blood flow with the bones particularly affect the joint/s and leading to a death of the cartilage, which in turns peels off into the joint capsule. There is no known cause at this time as to why blood flow is restricted.

Osteochondrosis of medial humeral condyle: OCD develops on the elbow end of the humerus (the long bone in the front leg above the elbow).

Fragmented medial coronoid process and ununited anconeal process: The coronoid and anconeal processes are small bones which fuse with the main part of the ulna as the animal matures. (The ulna and the radius are the two bones which make up the front leg between wrist and elbow). These terms describe the condition where those processes either break off from the ulna, or fail to fuse normally.

Incongruent elbow: The bones which form the elbow joint grow at different rates and do not fit together properly.

Mode of Inheritance & Heritable Disease group

This is a ***polygenic condition*** (although it is not currently known how many or which genes are responsible). Environmental factors such as fast weight-gain and growth, can also affect the development of this condition in dogs that are genetically predisposed to it.

In very few cases, trauma may be the cause, but generally this form of elbow dysplasia is found in previously tested and cleared adults.

Numbers Affected/Rate of Occurrence

Unknown at present – insufficient numbers of the breed have been screened in Australia to make this determination.

Severity of the end disease

Lameness usually starts insidiously at 7 to 10 months of age. It is present every day, and may be most obvious when the dog first gets up, or starts to walk or run. The prognosis (the likely outcome) depends on how far the disease has progressed when treatment begins. Good clinical results (i.e., the dog will not be in pain) are usually seen if treatment starts early, before osteoarthritis (degenerative changes in the joint) has developed. If left untreated, the dog's pain and lameness will gradually get worse.

Testing/Screening

DNA testing for this disease is not yet available.

The initial lameness may be very subtle with this condition, and it may be some time before it can be documented or diagnosed. Elbow dysplasia may be suspected in a young, fast-growing dog with forelimb lameness and elbow pain. This is confirmed by a veterinary physical exam and observation of the dog walking or running to confirm which limb and which joint is painful.

X-rays are necessary to diagnose elbow dysplasia. Both elbows should be x-rayed because the hereditary form of this disease is usually bi-lateral (present in both sides even if the dog is only lame on one side). If possible, a CT scan may be done to further determine the amount and location of bone fragments in the joint/s.

The AVA maintains a panel of assessors qualified to determine the presence and/or severity of elbow dysplasia and the data collected allows a breed average to be determined.

Treatment

Surgery is usually recommended to remove a bone or cartilage fragment. If unequal bone growth is the problem, surgery may help to relieve the pressure at the joint.

With the success in the United States with stem cell therapy (extracted from the affected dog itself) in the treatment of elbow dysplasia, hip dysplasia and other forms of arthritis in dogs, this may become the recommended therapy in Australia in years to come, rather than invasive surgery.

Medical management recommendations include monitoring the diet (to avoid excess weight gain and fast growth), and controlling exercise. Alternative therapy such as acupuncture may be recommended. Medications such as non-steroidal anti-inflammatory drugs may help with pain relief. "Chondroprotective agents" or nutraceuticals such as pentosan polysulfate injections or glucosamine supplements may also be prescribed.

Breeding advice

The best attempts at control are based on a grading scheme for identification of the defect and a breed policy of recording and publishing the results for as many dogs as possible.

Breed organizations and veterinarians in various countries have developed control programmes that rely on radiographic evaluation and a central registry of dogs. Dogs should be evaluated by accepted screening

programmes before breeding, and should be bred based on the guidelines of that programme.

Essentially, the best way for breeders to prevent elbow dysplasia is to breed only dogs that have relatively disease-free joints, based on appropriate radiographic evaluation, and over time that come from families with disease-free joints.

Either dogs with any degree elbow dysplasia over that of the breed average (these averages are kept as part of the statistics from the screening program/s) or dogs whose offspring have elbow dysplasia should NOT be used in a breeding program.

Recommendations:

All breeding stock should be x-rayed and scored by an AVA approved veterinarian prior to breeding and only dogs below the current breed average be used in a breeding program.

HIP DYSPLASIA

What is hip dysplasia?

The hip joint is a "ball and socket" joint: the "ball" (the top part of the thigh bone or femur) fits into a "socket" formed by the pelvis. If there is a loose fit between these bones, and the ligaments which help to hold them together are loose, the ball may slide part way out of the socket (subluxate). With time, as this occurs repeatedly, other degenerative changes in the joint occur (also called osteoarthritis) and the dog will become painful, lame and weak in the hind end.

This disease is progressive; that is, it gets worse with time.

Mode of Inheritance & Heritable Disease group

The mode of inheritance of this disease is a ***polygenic condition*** (caused by many different genes). Scientists do not yet know which genes are involved, or how many genes. Factors that can make the disease worse include excess weight, a fast growth rate, and high-calorie or supplemented diets.

Numbers Affected/Rate of Occurrence

Unknown at present – insufficient numbers of the breed have been screened in Australia to make this determination.

Severity of the end disease

While there is a severe form of hip dysplasia that affects young dogs (less than one year of age), signs of this disease are most common in older dogs. The loose fit at the hip joint will be present in young dogs, but it may take years for the other changes (such as osteoarthritis) to cause pain. The dog may be painful after exercise, have difficulty with stairs, or even have difficulty getting up. This may only be noticed once in a while, but over time will worsen.

Testing/Screening

X-rays are taken to evaluate the general fit of the femur and pelvis and to look for any osteoarthritic changes in the hip joint.

There are several established scoring systems to evaluate radiographs for the presence of hip dysplasia. The Australian Veterinary Association uses a system by which nine aspects of the hip joint are scored. This system is generally not used on dogs under 12 months of age. The system is similar to that used in the United Kingdom and New Zealand.

Recently, Australia has seen the introduction of the he PennHip method which uses a quantitative measure of joint laxity (based on distraction and compression views) to determine the Distraction Index (DI), as well as the standard hip-extended view, to evaluate a dog for hip dysplasia (see Smith and McKelvie,1995, in the Resource list below). Dogs may be evaluated by this technique as young as 16 weeks of age.

The Orthopedic Foundation for Animals in the United States evaluates a standard ventrodorsal view with hips extended and stifles rotated internally. Radiographs are scored based on degenerative joint changes and evidence of subluxation. Dogs must be 2 years of age in order to be certified by the OFA.

Treatment

The degree to which the hips are dysplastic does not always correlate with the amount of pain. Some dogs with very bad hips radiographically have less pain than others who's x-rays show only minor changes.

With the success in the United States with stem cell therapy (extracted from the affected dog itself) in the treatment of elbow dysplasia, hip dysplasia and

other forms of arthritis in dogs, this may become the recommended therapy in Australia in years to come rather than invasive surgery.

Pain management treatments include anti-inflammatory drugs and/ or alternative therapies such as acupuncture. Nutraceuticals such as pentosan polysulfate injections and/or glucosamine supplements may also be helpful.

Breeding advice

The best attempts at control are based on a grading scheme for identification of the defect and a breed policy of recording and publishing the results for as many dogs as possible.

Breed organizations and veterinarians in various countries have developed control programmes that rely on radiographic evaluation and a central registry of dogs. Dogs should be evaluated by accepted screening programmes before breeding, and should be bred based on the guidelines of that programme.

Essentially, the best way for breeders to prevent elbow dysplasia is to breed only dogs that have relatively disease-free joints, based on appropriate radiographic evaluation, and over time that come from families with disease-free joints.

Dogs with any degree of hip dysplasia over that of the breed average (these averages are kept as part of the statistics from the screening program/s) and dogs whose offspring have hip dysplasia should NOT be used in a breeding program.

Recommendations:

All breeding stock should be x-rayed and scored by an AVA approved veterinarian prior to breeding and only dogs with normal or near normal hips be used in a breeding program.

CATEGORY 2: OTHER DISORDERS WITH A KNOWN INCIDENCE IN THE BREED

These disorders occur less commonly or are less devastating than those mentioned above.

CATARACTS

Congenital cataracts are those that are present when the eyes open or before eight weeks of age and have been identified in the breed in North American and Europe.

Mode of Inheritance & Heritable Disease Group

This is a **recognised inherited disease.** The genetics have not yet been defined although there is some thought that the mode of inheritance may be autosomal recessive, autosomal dominant, or with incomplete dominance.

Numbers Affected/Rate of Occurrence

Uncommon occurrence – numbers unknown at present

Severity of the end disease

This depends on whether the cataracts are localized to a small area or are more general, and whether they affect one or both eyes. A small cataract in one eye will not affect the dog's vision at all. At the other end of the spectrum, cataracts may progress rapidly or slowly to cause complete blindness.

Congenital cataracts or those that develop at a young age may mature and be reabsorbed, resulting in improved vision. This is unpredictable. In the process of resorption, liquefied lens material may leak into the eye causing inflammation and possibly glaucoma.

With their acute senses of smell and hearing, dogs can compensate very well for visual difficulties, particularly in familiar surroundings. In fact owners may be unaware of the extent of vision loss.

Testing/Screening

Opthamological screening by a veterinarian.

Breeding advice

Dogs to be used in breeding programmes be screened and cleared for cataracts prior to breeding. Where congenital cataracts are identified, affected animals, their parents and littermates should not be used for breeding.

Recommendations

Puppies have eye clearances at about eight weeks of age and prior to sale.

CEREBELLAR HYPOPLASIA

Mode of Inheritance & Heritable Disease Group

Although inconclusive, there is some evidence that the mode of inheritance is ***autosomal recessive*** and may be categorised as ***simple recessive that may take years to develop.***

Numbers Affected/Rate of Occurrence

Whilst identified in the breed, this is a very uncommon disorder.

Severity of the end disease

The cerebellum is the part of the brain that regulates the control and coordination of voluntary movement. The clinical signs of cerebellar dysfunction in affected puppies range from mild to severe, and may include poor balance, a wide-based stance (feet planted far apart), stiff or high-stepping gait, apparent lack of awareness of where the feet are (standing or walking with a foot knuckled over), and head or body tremors. Affected pups have normal mental alertness.

In this condition, signs are evident at birth or by 2 weeks thereafter, and do not get worse as the pup ages. Other than the abnormalities in balance and coordination, the animal's general health is unaffected.

Testing/Screening

The clinical signs (relating to uncoordinated movement and lack of balance) are suggestive of a cerebellar disorder. Intention tremor (of both head and limbs) is common. The tremors worsen with stress or excitement and subside

when the dog is at rest. Diagnosis is based on the clinical signs, lack of progression, and lack of significant findings on other diagnostic tests.

Treatment

There is no treatment for this condition. Affected dogs will not get any worse (or better) and, especially where the signs are mild, may be able to lead a relatively normal life, particularly if owners can adjust their expectations to the dog's limitations.

Breeding advice

Affected dogs, their parents and their siblings should not be used for breeding.

CERVICAL VERTEBRAL INSTABILITY

Related Terms: *Wobbler syndrome, cervical spondylomyelopathy, cervical vertebral deformity*

There is compression of the spinal cord in the neck (cervical) region. With this condition, there are abnormalities in the structure of the vertebrae, of the ligaments that connect them, and/or of the disks between them. The reasons for these abnormalities are not clear; inheritance is a factor, and overfeeding in rapidly-growing large breed dogs is also thought to play a role.

The result is instability between adjacent vertebrae, narrowing (stenosis) of the spinal canal, and pressure on the spinal cord. The consequences of compression of the spinal cord in the neck region are weakness and incoordination in all 4 legs - hence the name "wobbler".

Mode of Inheritance & Heritable Disease Group

Unknown. It has been suggested that it **may** be autosomal recessive in some breeds and therefore may be categorised as ***simple recessive which may take years to develop.***

Numbers Affected/Rate of Occurrence

Whilst identified in the breed, this is a very uncommon disorder.

Severity of the end disease

The main signs with this disease are weakness and incoordination (ataxia); these signs begin insidiously and worsen slowly over several months. It may look like the dog doesn't know where his or her feet are. This will be most obvious when s/he is rising from lying down, or negotiating a turn or stairs. Over time, the dog may develop a stiff, high-stepping, exaggerated gait that gradually worsens.

The signs are bilateral and symmetrical. All four legs are eventually affected, with the hind legs affected first, and more severely. Sometimes there is a sudden change for the worse as a result of minor trauma.

This is a chronic, progressive disease. Without treatment, the dog's condition will gradually deteriorate. With therapy (either medical management or surgery) the prospect for recovery remains guarded.

Testing/Screening

Based on characteristic clinical signs which include slowly progressive, bilateral, symmetrical hind leg weakness and ataxia; x-rays are taken to show structural abnormalities in the vertebrae. Nevertheless, **myelography** is necessary to determine if there is spinal cord compression. Other imaging techniques, such as CT scans and MRI, may also be used.

Treatment

The type of treatment chosen for this condition will depend on a number of factors, including the severity and duration of the dog's signs, and the extent of spinal cord compression apparent on radiography. The goals of medical management are to minimize neck movement (through confinement and use of a neck brace) and use anti-inflammatory medications to prevent further damage to the spinal cord. Medical management may be effective for weeks to years, although it does not address the underlying problem of spinal cord compression. A variety of surgical techniques have been developed (and more are being developed) which attempt to both alleviate the spinal cord compression and stabilize the vertebrae.

Breeding advice

Although the exact mechanism of inheritance is not known, dogs with cervical vertebral instability should not be bred. (Unfortunately, because this condition often has a later onset, dogs may be bred before any problems appear). It is best to avoid breeding their parents or siblings as well, who are considered potential carriers of the trait.

COLOUR DILUTION ALOPECIA

Related Terms: *colour mutant alopecia, blue dog disease*

This condition develops in some but not all dogs that have been bred for unusual coat colour, in particular "fawn" or "blue". Alopecia means hairlessness - affected dogs have a poor, patchy hair coat progressing to widespread permanent hair loss. At the cellular level, there are abnormalities of the hair follicles and uneven clumping of pigment (melanin) granules in the hair shafts in affected areas

Mode of Inheritance & Heritable Disease Group

The inheritance is unclear. The condition is thought to be due to the interaction of different factors at the gene position for colour. It is not simply determined by the genes at that locus, because not all dogs with colour dilution develop coat problems.

Coat colour genetics indicate that this is a ***polygentic disorder.***

Numbers Affected/Rate of Occurrence

Uncommon

Severity of the end disease

The dog will experience hair loss and dry skin. Hair loss is usually first apparent on the back and by 2 or 3 years has spread over all the light coloured areas of the body. The exposed skin is often scaly and is susceptible to sunburn or extreme cold. The dog's health is not otherwise affected by this condition.

Testing/Screening

The diagnosis is confirmed through microscopic examination of plucked hairs or a skin biopsy. Careful microscopic examination of plucked hairs will show large clumps of melanin distributed unevenly along the hair shaft.

In young dogs, demodicosis or other inherited hair defects should be considered while in dogs with a later onset (2 to 3 years of age), endocrine disorders (particularly hypothyroidism) should be ruled out.

Treatment

The dog can lead a normal healthy life with periodic symptomatic treatment as needed - moisturizing rinses for dry scaly skin or antibiotics for bacterial infections.

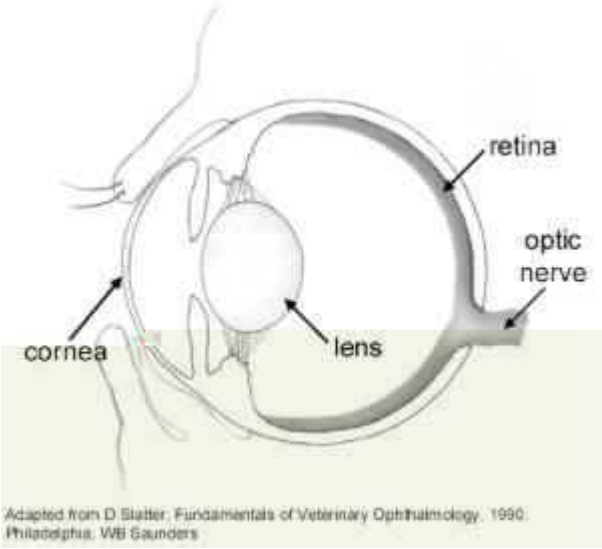
Since early hair loss occurs due to breakage, you may be able to slow the rate of loss by avoiding harsh shampoos and vigorous grooming.

There have been some early reports of hair regrowth using etretinate treatment.

Breeding advice

Affected dogs, their parents and siblings should not be used for breeding.

CORNEAL DYSTROPHY



Related Terms: boxer corneal ulceration, indolent/persistent ulcer, epithelial/stromal corneal dystrophy, endothelial corneal dystrophy, corneal lipid dystrophy, epithelial erosion

Corneal dystrophy is an inherited abnormality that affects one or more layers of the cornea. Both eyes are usually affected, although not necessarily symmetrically. Chronic or recurring shallow ulcers may result, depending on the corneal layers affected:

Endothelial dystrophy is the type that has been documented as affecting the Chow Chow. It affects the function of the endothelial cells. The result is a build-up of fluid in the cornea (corneal edema) which clouds the normally transparent cornea and may decrease vision. Edema may cause the eye to appear blue. Recurring non-healing shallow corneal ulcers occur as well.

Mode of Inheritance & Heritable Disease Group

In some breeds, corneal dystrophy has been shown to be an autosomal recessive trait, with variable expressivity. In some breeds, inheritance is sex-linked. The mode of Inheritance in Chow Chows has not been identified.

The disease group MAY be ***simple recessive that may take years to develop.***

Numbers Affected/Rate of Occurrence

Uncommon

Severity of the end disease

Endothelial dystrophy: Over time, the fluid build-up causes inflammation of the cornea and reduced vision. "Water blisters" (bullous keratopathy) may develop which can rupture and cause painful erosions or ulcers.

Testing/Screening

One or several small white to gray areas in one or both of the dog's eyes may be noticed. Magnification may reveal crystalline deposits within the deeper layers of the cornea or simply a haze.

Veterinary examination of the eye for erosions or, in the case of oedema, for bullous keratopathy. A fluoroscein dye test is used to check for corneal ulcers.

Treatment

In **endothelial dystrophy**, no treatment is necessary in the early stages of the disease. As the oedema (or fluid build-up) in the cornea increases, dogs may develop "water blisters" (bullous keratopathy) which can rupture and cause painful erosions. There is prescription eye medication appropriate for bullous keratopathy (hyperosmotic solutions) as well as treatment for ulcers if present. There are surgical treatments which can be performed by a veterinary ophthalmologist if the erosions persist or recur frequently despite medical therapy.

Breeding advice

Affected dogs and their close relatives should not be used for breeding.

DIABETES MELLITUS

Diabetes mellitus is a disruption of the body's ability to use carbohydrates/sugars. Normally, certain cells in the pancreas produce the hormone insulin which regulates the uptake of sugars into cells throughout the body. In some forms of diabetes mellitus, the cells are dysfunctional and do not produce insulin, while in other forms insulin is produced but body tissues do not respond appropriately.

Genetics is only one of many factors that may be involved. In some dogs there seems to be a genetic predisposition to to the destruction by the immune system of the insulin-producing cells. In other dogs, less severe genetic-based changes in the cells may make the dog more susceptible to the development of diabetes mellitus - in association with another illness or obesity or exposure to certain drugs.

Mode of inheritance

In some breeds the mode of inheritance is as an ***autosomal recessive*** ***and may be categorised as simple recessive that may take years to develop.trait.***

The mode of inheritance has not been determined in this breed.

Numbers Affected/Rate of Occurrence

Uncommon

Severity of the end disease

In dogs with the most severe form of inherited diabetes mellitus, signs are usually apparent by 6 months of age. Pups drink and eat more than normal, and yet grow very slowly. They urinate frequently, and their stools are soft.

In other dogs, diabetes mellitus does not develop until middle age. The signs of uncomplicated diabetes are typical - increased eating, drinking, and urination, with weight loss - all of which are a result of increased levels of glucose in the blood and urine. Over the long term, this can lead to the development of cataracts, increased susceptibility to bacterial infections (especially of the urinary tract), liver disease, and pancreatitis. Eventually,

untreated diabetic dogs will develop ketoacidosis, a very serious condition. Signs of ketoacidosis include depression, weakness, vomiting, and irregular breathing patterns.

Testing/Screening

The diagnosis is made based on the typical clinical signs - increased eating, drinking, and urination, with weight loss - together with persistently elevated levels of glucose in both the blood and the urine. Ketones may also be present in the urine..

Fasting hyperglycemia with glucosuria; if the dog has ketoacidosis may see metabolic acidosis, hyponatremia, hyperkalemia, and hypochloremia.

Treatment

In the diabetic dog without any other illness, the goals of therapy are to achieve near-normal blood glucose levels and minimize the daily variation in those levels. This is important to prevent the complications that develop over time in poorly controlled diabetic patients. Treatment includes insulin administration, diet, and exercise, all of which your veterinarian will discuss with you.

Emergency treatment for dogs with ketoacidois includes intravenous fluids and fast-acting insulin. Once the animal is stable, a regular regime of longer-acting insulin, diet, and exercise can be established.

Breeding Advice

Affected dogs should not be bred, and parents and siblings should be considered potential carriers.

ECTROPION

Ectropion is a defect of conformation in which there is a sagging or rolling-out (eversion) of the eyelids. This results in abnormal exposure of the eye, which often leads to irritation.

Mode of Inheritance/

This is a conformational disorder caused by polygenic inheritance. It affects the structures that make up the eyelids and affects the way the skin covers the face and head.

Numbers affected/Rate of Occurrence

Unknown. It is often the mistaken diagnosis given to dogs with simple laxity in the lower eyelid.

Severity of the end disease

Because of increased exposure of the eye, dogs with ectropion are prone to develop allergic or bacterial conjunctivitis. Affected dogs may develop keratoconjunctivitis sicca because of reduced efficiency at wetting and cleaning the cornea.

Testing/Screening

In addition to the sagging of the eyelids, dogs with ectropion commonly have a mucopurulent discharge in the eye, reddening of the exposed conjunctiva, and decreased tear production. To check the latter, a Schirmer tear test is needed.

Treatment

With mild entropion, no treatment may be necessary. If secondary problems such as conjunctivitis develop, these are treated as required.

More severe ectropion can lead to chronic problems associated with eye irritation. In these cases, surgery is performed to remove a small wedge of tissue from the margin of the eyelid.

Breeding advice

Encouragement of breeders to engage in breeding programs that, wherever possible, use non-affected animals for breeding.

GLAUCOMA

Related terms: *goniodysgenesis, pigmentary glaucoma*

What is glaucoma?

Glaucoma is a leading cause of blindness in dogs. It is the result of increased fluid pressure within the eye (elevated intraocular pressure or IOP). If the pressure can not be reduced, there will be permanent damage to the retina and optic nerve resulting in visual impairment. Complete blindness can occur within 24 hours if the IOP is extremely elevated or can occur slowly over weeks or months if the the elevation is mild. Glaucoma is usually very painful.

Glaucoma may be primary (inherited) or secondary to a number of eye disorders including luxation of the lens, tumours of the eye, and uveitis (inflammation of the eye).

Primary/inherited glaucoma causes an elevation of pressure within the eye because of abnormal drainage of fluid through the iridocorneal angle. When the angle at which the iris and cornea join is wide, the glaucoma is classified as open angle. If the base of the iris is pushed forward, the glaucoma is described as narrow angle.

Goniodysgenesis is characterized by an abnormal sheet of tissue in the angle where drainage normally occurs. This may or may not cause an elevation in IOP and glaucoma.

In **pigmentary glaucoma**, the obstruction to fluid drainage is caused by an abundance of pigmented cells within the iridocorneal angle and sclera. The increase in IOP is progressive and often results in blindness.

How is glaucoma inherited?

Inherited open angle glaucoma is an autosomal recessive trait in beagles. Narrow angle glaucoma is inherited as an autosomal dominant trait in the Welsh springer spaniel. The mode of inheritance for glaucoma in other breeds has not been identified.

Narrow/closed angle glaucoma is the form of glaucoma identified in the Chow Chow, as well as other breeds.

What does glaucoma mean to the dog & its owner

Glaucoma is moderately to extremely painful. The eye may be red and the dog may paw at it, or rub his or her head along the carpet. The eye may look cloudy due to swelling of the cornea and the dog will be very sensitive to light. The affected eye may seem larger, or appear to bulge out, relative to

the other eye. Other more general signs of pain include loss of appetite and depression.

Glaucoma is an emergency. Treatment must be started as soon as possible if the dog's sight is to be saved. Irreversible damage to the retina and optic nerve occur within a few hours of significant elevation of the intraocular pressure.

How is glaucoma diagnosed?

Glaucoma is one of the conditions your veterinarian will suspect if the dog has a painful eye. It is diagnosed by measuring the intraocular pressure with a tonometer. This can usually be done with local anaesthetic drops placed in the dog's eye. To determine the type of glaucoma, gonioscopy is used to measure the iridocorneal angle.

How is glaucoma treated?

Preserving vision in an eye with glaucoma is difficult and requires aggressive medical and surgical therapy. Your veterinarian may choose to provide initial emergency medical therapy and refer you immediately to a larger veterinary centre.

Treatment depends on several factors - the type of glaucoma present, the degree of elevation of IOP, and the extent of visual impairment. Primary open angle glaucoma tends to be slower in onset and may, at least initially, be controlled by medical therapy (drugs) alone. With closed angle glaucoma, which is much more common, there is usually a sudden, rapid elevation in IOP. Ultimately, most forms of glaucoma require surgery.

If vision is present or has just recently been lost, a combination of medical and surgical therapy will be used to try and maintain the dog's sight . Aggressive medical therapy (meaning a combination of anti-glaucoma drugs administered frequently and monitored closely) is used to reduce IOP prior to surgery to prevent further damage to the eye. Some of these drugs will be used as well for additional minor IOP reductions following surgery. The aim of surgery in an eye that is still visual (or potentially visual) is to decrease the production of fluid within the eye, and to improve the drainage from the eye. There are a few different methods that a veterinary ophthalmologist can use to achieve this.

If the eye is irretrievably blind, glaucoma can be treated by removing the globe of the eye (enucleation). This will eliminate the pain for the dog. There are also procedures that can be done that preserve the globe such as placing a prosthesis.

Inherited glaucoma usually occurs in both eyes eventually. Your veterinarian will monitor the pressure in the other eye regularly, and discuss with you recognition of early signs of glaucoma. He or she may also recommend preventive medication for the unaffected eye.

Breeding advice

Screening of breeding stock for glaucoma before being used for breeding. Affected dogs and their close relatives should not be bred.

Unfortunately, glaucoma does not generally become apparent until after breeding age has been reached, usually 3 years of age or greater.

FOR MORE INFORMATION ABOUT THIS DISORDER, PLEASE SEE YOUR VETERINARIAN.

GROWTH HORMONE-RESPONSIVE AND ADRENAL SEX-HORMONE DERMATOSES

***Related Terms**: Hyposomatotropism, acquired growth hormone deficiency, adrenal sex-hormone dermatosis*

What is growth hormone-responsive dermatosis?

With this condition there are skin changes due to a lack of growth hormone (somatotropin). This hormone is secreted by the pituitary gland and is necessary for hair growth and the maintenance of normal elasticity of the skin. Affected dogs have varying degrees of hair loss and darkening of the skin, but are otherwise healthy.

Adrenal sex-hormone dermatosis is a clinically similar syndrome that appears to be due to abnormal sex hormone production by the adrenal glands, causing secondary changes in growth hormone levels.

How is growth hormone-responsive dermatosis inherited?

A mode of inheritance has not been established; however the disorder is mostly seen in the breeds listed below.

The Chow Chow has been identified as one of several breeds affected by growth hormone-responsive dermatosis.

What does growth hormone-responsive dermatosis mean to the dog & its owner?

This condition is more common in male dogs between 1 and 5 years of age, with hair loss usually starting at puberty. Adrenal sex-hormone dermatosis is seen in males and females, either neutered or intact.

Hair loss (called alopecia) is symmetrical over the trunk of the dog and the skin is markedly darker in colour due to increased pigmentation. Without treatment, hairlessness and hyperpigmentation will eventually spread over the dog's body except for head and feet. The condition does not affect the health of the dog, only his/her appearance.

How is growth hormone-responsive dermatosis diagnosed?

There are several possible hormonal causes of hair loss in dogs. Your veterinarian will do tests to determine the cause in your pet.

How is growth hormone-responsive dermatosis treated?

As mentioned, this condition does not affect the health of the dog, only his/her appearance. It can be treated with growth hormone (GH), but this is expensive and can be difficult to obtain. Treated dogs must be monitored for the development of diabetes mellitus which is a potential side effect of GH therapy. Castration may resolve the condition in male dogs.

Similarly, neutering of affected male or female dogs may help to treat adrenal sex-hormone imbalance. If ineffective, there are medical treatments that may be beneficial (opDDD or mitotane, and ketoconazole).

Breeding advice

Although little is known about the inheritance of this disorder, it is prudent to avoid breeding affected animals or their close relatives.

FOR MORE INFORMATION ABOUT THIS DISORDER, PLEASE SEE YOUR VETERINARIAN.

HYPO-/DYSMYELINOGENESIS

Related Terms: *hypo-/dysmyelination, "shaking pup"*

What is hypo-/dysmyelinogenesis?

Myelin is a fatty substance that coats nerve cells. It serves as an electrical insulator and speeds the conduction of nerve impulses. The formation of myelin begins mid-way through pregnancy and continues for a short period after birth.

In this disorder, there is a lack of ("hypo"), or abnormal ("dys") myelination, primarily in the spinal cord but also in parts of the brain. Most affected is the general proprioceptive system, which is important for the coordination of body movements and positioning.

The condition is most severe in the springer spaniel and samoyed. In other breeds, puppies often gradually improve, perhaps because of continued slow myelination of cells after birth.

How is hypo-/dysmyelinogenesis inherited?

The condition is an x-linked trait in the springer spaniel ("shaking pup") and possibly the samoyed. In other breeds it is believed to be autosomal recessive.

What breeds are affected by hypo-/dysmyelinogenesis?

This uncommon disorder occurs in the Springer Spaniel ("Shaking Pup"), Chow Chow, Weimaraner, Bernese Mountain Dog ("Trembler"), And Samoyed.

What does hypo-/dysmyelinogenesis mean to the dog & its owner

Because the proprioceptive system is most affected, pups with this condition have problems with balance, coordination, and positioning. They have a wide-based stance, and appear clumsy (but without weakness). Pups may have what is described as a rocking horse gait. There are pronounced tremors of the head and legs, which are stronger when the pup is excited or moves, and subside at rest or during sleep. Affected pups are normally alert and responsive to their surroundings.

Signs are first noticed after birth or when the pups first walks. With the exception of the Springer spaniel and Samoyed, these signs do not worsen but instead improve gradually over several weeks or months, often to normalcy by 1 year.

Because the trait is sex-linked in the Springer spaniel, males are most severely affected. By about 2 weeks of age, male pups develop a severe tremor of body, head and legs that decreases during rest and worsens with excitement. They are unable to stand, walk, or eat and generally do not survive unless intensively hand-reared. Females will be carriers of the trait and may show mild signs as puppies that disappear by 4 to 6 weeks of age.

How is hypo-/dysmyelinogenesis diagnosed?

There are other conditions, primarily affecting the cerebellum, that cause similar signs in newborn puppies. Your veterinarian will do tests to rule out other possible causes.

How is hypo-/dysmyelinogenesis treated?

There is no treatment, but affected puppies tend to improve over time, often to complete recovery by 1 year of age. Samoyeds and male Springer spaniels show little if any improvement.

Breeding advice

The mother of any affected pup is a carrier and should not be used again for breeding. Breeding of his sisters should also be avoided as they have a 1 in 2 chance of being a carrier, and passing this severe disorder to any male offspring.

FOR MORE INFORMATION ABOUT THIS DISORDER, PLEASE SEE YOUR VETERINARIAN.

HYPOTHYROIDISM

Related Terms: *familial thyroiditis, lymphocytic thyroiditis, congenital hypothyroid dwarfism*

What is hypothyroidism?

The clinical signs of hypothyroidism are caused by a decrease in normal thyroid hormone activity. The disorder may be acquired (a progressive deficiency of thyroid hormone) or congenital (meaning the animal is born with the disorder). The acquired form is the most common disorder of the endocrine system in dogs. It occurs as a result of gradual atrophy of the thyroid gland or of gradual infiltration and replacement of the thyroid gland with lymphocytes due to an autoimmune process (lymphocytic thyroiditis).

Acquired hypothyroidism is generally seen in middle-aged (4 to 10 years) mid - to large breed dogs. Congenital hypothyroidism is very rare.

How is hypothyroidism inherited?

Unknown

What does hypothyroidism mean to the dog & its owner

The changes due to gradually decreasing levels of circulating thyroid hormone are slow and insidious. Early signs (which are usually not recognized as being related to hypothyroidism) include lower energy levels and increased susceptibility to infections. As the disease progresses, you will likely notice changes in the dog's hair coat - symmetrical hair loss with or without darkening of the skin, and dry or greasy hair. Other signs of hypothyroidism include a slow heart rate, lethargy, mental dullness, intolerance to cold, infertility in males and females, constipation, and weight gain. Less commonly, a dog with hypothyroidism may experience heart disease, a bleeding disorder, profound muscular weakness associated with abnormalities in the muscles or nerves, or another endocrine disorder such as diabetes mellitus.

Congenital hypothyroidism

Thyroid hormones are essential for normal growth and maturation of the nervous and skeletal systems. Puppies with congenital hypothyroidism will have stunted growth as well as many other abnormalities. Severely affected puppies most likely die before weaning.

How is hypothyroidism diagnosed?

Because there is such a broad range of possible clinical signs, hypothyroidism can be quite difficult to diagnose. Blood tests may show certain suggestive (but non-specific) abnormalities. If this condition is suspected, laboratory tests to assess thyroid function are run.

1. CLINICAL PATHOLOGY: Suggestive findings include hypercholesteremia, mild nonregenerative anemia, elevated serum creatine kinase (CK), and hypoglycemia
2. CONFIRMATION: Measurement of free T4 (FT4) and canine thyroid-stimulating hormone (cTSH) are the tests of choice to diagnose hypothyroidism, and to differentiate the primary from the secondary form. [Secondary hypothyroidism is much less common.] Some laboratories can also test for thyroid autoantibody levels (TgAA) which is helpful in the early diagnosis of autoimmune thyroiditis. This may provide useful information for breeders.
3. RADIOGRAPHS: In congenital hypothyroidism, there are typical radiographic changes including epiphyseal closure, shortened vertebral bodies, kyphosis, and

arthritis. Epiphyseal dysgenesis (ragged epiphyses with a few foci of calcification) is pathognomonic for congenital hypothyroidism.

How is hypothyroidism treated?

The standard treatment is levo-thyroxine given once a day. This must be continued for life. Within a week of starting treatment, the dog's attitude and activity levels should improve. It can take up to 6 weeks before there is noticeable improvement in the skin and haircoat, but eventually all abnormalities should completely resolve.

Dogs on thyroid replacement can live a normal life.

Congenital hypothyroidism is treated the same way.

Breeding Advice

Although inheritance of this disorder has not been determined, it is advisable not to breed affected dogs.

Recommendation

The Club request, through the VCA, that AVA or a similar agency be requested to maintain a thyroid registry based on assessment of FT4, cTSH, and TgAA, for all affected breeds. This would prove useful for breeders attempting to choose dogs free of hypothyroidism for a breeding programme.

MYOTONIA

This disease affects skeletal (voluntary) muscle, such as the muscles of the limbs. Muscles are unable to relax normally following contraction (or electrical stimulation). This results in a stiff, awkward gait, difficulty in rising, and stiff joints.

How is myotonia inherited?

In Chow Chows, myotonia is believed to be inherited as an autosomal recessive trait.

What breeds are affected by myotonia?

Myotonia is seen most often in Chow Chows, although it is not a common disease and relatively unheard of in Australia. Isolated cases have also been reported in other breeds.

What does myotonia mean to the dog & its' owner

While there is no cure for this disease, affected dogs will often remain stable, without worsening of clinical signs, with drug therapy.

How is myotonia diagnosed?

The dog may have difficulty rising and a stiff gait (although gait will become more normal as the dog 'warms up'). Muscles may be hypertophied (large and well-developed) and it may be difficult to flex the limbs. Diagnosis is confirmed by analyzing muscle response to electrical stimulation (electromyography) and by evaluation of muscle biopsies.

How is myotonia treated?

There is no cure for this disease, although drug therapy (eg. procainamide) helps many dogs.

Breeding advice

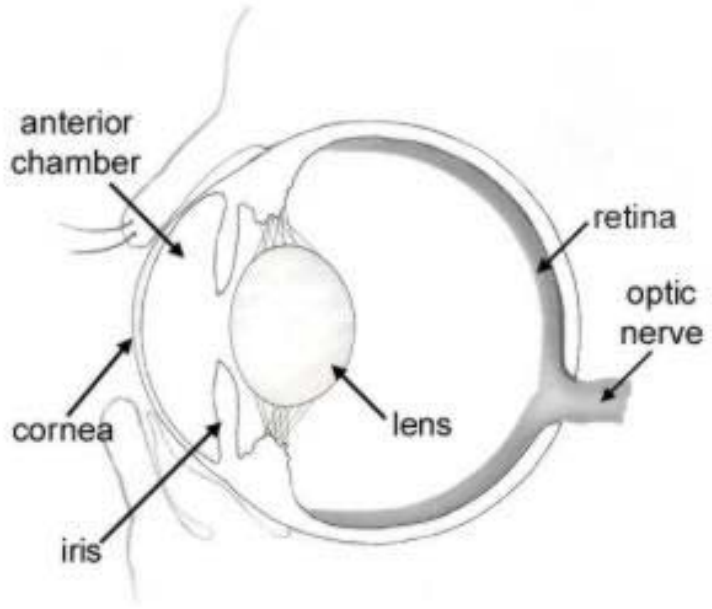
Affected dogs should not be bred. Parents of affected Chow Chows are carriers, and siblings are suspect carriers, and so should not be used for breeding.

PERSISTENT PUPILLARY MEMBRANES

Persistent pupillary membranes are strands of tissue in the eye. They are remnants of blood vessels which supplied nutrients to the developing lens of the eye before birth. Normally these strands are gone by 4 or 5 weeks of age.

Depending upon the location and extent of these strands, they may interfere with vision. They may bridge from iris to iris across the pupil, iris to cornea (may

cause corneal opacities), or iris to lens (may cause cataracts), or they may form sheets of tissue in the anterior chamber of the eye. In many dogs these tissue remnants cause no problems.



Adapted from D Slatter, Fundamentals of Veterinary Ophthalmology, 1990, Philadelphia, WB Saunders

Mode of Inheritance & Heritable Disease Group

Inheritance is not defined.

What breeds are affected by persistent pupillary membranes?

PPM are known or strongly suspected to be inherited in the Chow Chow amongst many other breeds.

What do persistent pupillary membranes mean to the dog & its owner

Generally persistent pupillary membranes cause no problems. However, if attached to the cornea or lens, the strands can cause opacities which may interfere with vision. The cataracts that can occur with PPM usually don't worsen.

How are persistent pupillary membranes diagnosed?

PPM are seen in young dogs. Small white spots in the dog's eyes may be noticeable to the naked eye or there may be some suspicion that the dog's vision is impaired. In other cases, strands may not be observable without an ophthalmoscope.

How are persistent pupillary membranes treated?

There is no treatment for the membranes themselves and in most cases there are no associated problems. If there is significant oedema or "bluing" of the cornea due to adhesions, hyperosmotic eye drops may help. Surgery may be required if there are extensive cataracts.

Breeding advice

Iris to iris PPM is not uncommon in Chows. Ideally, only unaffected dogs would be used in breeding programs. Where PPM appears to be an isolated incident, breeders may use their discretion.

Recommendation

Careful ophthalmic examinations for PPM be undertaken before dogs are used in a breeding programme.

SEBACEOUS ADENITIS

This is a perplexing condition in which the sebaceous glands in the skin become inflamed for unknown reasons, and are eventually destroyed. These glands normally produce sebum, a fatty secretion that helps prevent drying of the skin.

Clinical signs vary with the severity of the condition, and between different breeds.

How is sebaceous adenitis inherited?

It appears that the disorder is inherited as an autosomal recessive trait; however the wide variation in clinical signs suggests that inheritance is not straightforward, and breeding studies continue.

Rates of occurrence

Uncommon

What does sebaceous adenitis mean to the dog & its owner?

Sebaceous adenitis is usually first noticed in young adult dogs (1 to 5 years of age). The condition can appear differently in different breeds, and there is also marked variability depending on the clinical severity.

One form of the disorder is seen in long-coated breeds. Typically affected dogs have dry scaly skin with patches of hair loss along the top of the head, back of the neck, and back. Silvery scales tightly adhere to tufts of remaining hair. Very mildly ("sub-clinically") affected standard poodles have a normal hair-coat, but abnormalities typical of the condition are seen on microscopic examination of skin biopsies. More severely affected dogs will have areas of thickened skin ("hyperkeratosis"), extensive hair loss and often a musty or rancid odour. Secondary skin infections often occur as well.

Sebaceous adenitis is primarily a cosmetic disorder - that is it affects the appearance of the dog rather than his/her general health.

How is sebaceous adenitis diagnosed?

Diagnosis of this disorder based on the dog's clinical signs. To differentiate this condition from other skin disorders, many of which are also associated with increased scaling, a skin biopsy is necessary. The biopsy will show changes in the skin consistent with this condition.

How is sebaceous adenitis treated?

This disorder requires long term management, which can be frustrating for both owners and veterinarians because the response to treatment is highly variable. There may be periods of spontaneous improvement or worsening of the condition, independent of treatment.

Retinoid therapy and cyclosporine have been used with variable results in refractory cases.

Breeding advice

Although the genetics have not been determined, the condition does appear to be inherited in those breeds studied, including the Chow Chow. It is thus preferable to avoid breeding affected dogs of any breed, their siblings, and their parents.

CATEGORY 3. OTHER DISORDERS THAT MAY BE INHERITED IN THIS BREED

DERMATOMYOSITIS

Related Terms: *familial canine dermatomyositis, ulcerative dermatosis*

This condition is one of inflammation (itis) of the skin (dermato) and muscle (myo). There appears to be a defect in the immune system that predisposes dogs to this disorder. The skin lesions typically develop first with variable muscle problems occurring later. There are many similarities to dermatomyositis in people.

Ulcerative dermatosis may be a variant of this condition. It is a rare disorder that occurs in middle-aged to older dogs of the same breeds, and is manifest by skin lesions (blisters, crusting) that are seen primarily in the groin and underarm regions. Occasionally there are muscle abnormalities.

How is dermatomyositis inherited?

The trait is believed to be autosomal dominant with variable expressivity. This means that if either parent is affected, all puppies have a susceptibility to the disorder, but not all will be affected equally. The variability suggests there is more involved than simple inheritance, including internal factors such as the individual's immune system (also affected by heredity) and external factors (including possibly viral infection). The most severely affected dogs may be homozygous for the trait.

Rates of occurrence

Uncommon

What does dermatomyositis mean to the dog & its owner

With this condition, the skin is almost always affected before, and worse than, muscle. Typically, skin lesions occur by 6 months of age. There is reddening, hair loss, blisters or small bumps, crusting and where severe, ulceration of the skin. Most often affected are the face (especially the muzzle and ear tips, and around the eyes), the tip of the tail, bony prominences (over the elbows for instance) and the toes. Over time, the affected skin becomes scarred.

The muscles are not always affected in dermatomyositis, or the abnormalities may be so slight as to go unnoticed. When there is muscle involvement, the puppies may be weak and lethargic and have a slow rate of growth. Muscles (especially of the face and head) may appear smaller due to muscle

atrophy (shrinkage and loss of use). The most severely affected dogs may have difficulty in chewing or swallowing. The leg muscles may also atrophy.

The degree to which pups are affected varies considerably.

Generally the clinical signs fluctuate over time for no apparent reason, and many mildly affected dogs will outgrow the condition before a year of age, although some may have permanent scars on their face or legs. In severely affected dogs, the condition is progressive and these dogs may have to be euthanized due to severe muscle atrophy and associated problems such as an inability to eat and drink properly, which may be complicated by pneumonia.

How is dermatomyositis diagnosed?

This disorder is usually suspected in a young dog with crusting facial skin lesions, with or without muscle weakness. A skin biopsy will pinpoint the diagnosis. The biopsy will show changes in the skin consistent with this condition.

CBC, biochemical profile and urinalysis are usually normal, and the results of standard tests for autoimmunity are usually negative. In addition to history and physical exam findings, diagnosis is made by biopsy (affected skin and muscle), electromyography (EMG), and ruling out other conditions. The main differential diagnosis, especially where the muscle component is mild, is epidermolysis bullosa. The skin lesions have a similar age of onset and clinical progression, but with dermatomyositis, erythematous plaques or vesicles can not be induced in normal skin by applying mild friction.

Dermatomyositis may be complicated by localized or generalized demodicosis. Megaesophagus (+/- aspiration pneumonia) may occur in dogs with severe muscle involvement.

How is dermatomyositis treated?

Skin lesions are exacerbated by trauma and by exposure to ultraviolet light, so these should be avoided (by the use of sunscreens for example). This may be all that is required in mildly affected dogs, who are likely to outgrow the condition with time.

Dermatomyositis can usually be managed fairly well in moderately affected dogs, with the above precautions and the use of Vitamin E and occasional use of corticosteroids for flare-ups. Your veterinarian will work with you to determine how best to manage the condition in the dog. Unfortunately, it is very difficult to maintain the health and comfort of severely affected dogs, and euthanasia is sometimes the best option.

Pentoxifylline may help by improving microvascular blood flow. A response may take 2 or 3 months. Short term use of glucocorticoids may be necessary for acute flare-ups of skin or muscle inflammation, but long term use should be avoided as it will exacerbate skin and muscle atrophy.

Breeding advice

Affected dogs should not be bred. Also, because it is difficult to identify dogs that have only a mild form of this condition, close relatives of affected dogs (siblings and parents) should not be used for breeding.

GASTRIC DILATAION VOLVULUS SYNDROME

Related Terms: *bloat, stomach torsion, gastric torsion, GDV*

What is Gastric Dilatation Volvulus Syndrome?

Gastric Dilatation Volvulus Syndrome, best known amongst lay people as bloat, is a life-threatening condition characterised by an over-distension of the stomach with gas, fluid, or ingested food, with a resulting rotation of the stomach on its long axis, thus cutting off both entry into the stomach and out of the stomach.

How is bloat inherited?

Unknown

Numbers Affected/Rate of Occurrence

Unknown

What does bloat mean to the dog & its owner

If treated immediately, the dog's life may be saved and it's health can be managed; if left untreated, the dog will die.

How is bloat diagnosed?

GDV is a potentially life-threatening condition that causes severe abdominal pain, acute retching without vomiting, excessive salivation and depression or restlessness. The inflation of the stomach compresses the veins leading from the hindquarters to the heart. This causes evidence of cardiovascular failure such as rapid and difficult breathing, pale gums and a weak pulse. Some

dogs are reluctant to stand, whereas others lie on their side, unable to get up. These dogs can rapidly enter a state of shock and death.

How is bloat treated?

Surgical/Medical

Surgical treatment should be undertaken as soon as the patient has been stabilized with fluid therapy and decompression. A gastrectomy might be required if the stomach is becoming necrotic. A gastropexy is required to prevent recurrence.

Breeding advice

None

PEMPHIGUS FOLIACEUS

Related Terms: *pemphigus foliaceus, pemphigus erythematosus*

What is pemphigus?

Pemphigus covers a group of uncommon disorders that occur in dogs. With these conditions, there is an abnormal immune response to normal components of the skin, resulting in separation of cells. This leads to blisters, pustules, and crusting erosions in the skin. There are some similarities to pemphigus in humans, but many significant differences as well.

Breed predispositions are recognized for two forms only - pemphigus foliaceus and the milder pemphigus erythematosus.

How is pemphigus inherited?

Unknown

Numbers Affected/Rate of Occurrence

Uncommon

What does pemphigus mean to the dog & its owner

Pemphigus foliaceus and erythematosus develop around 4 years of age. P. erythematosus is thought to be a milder form of p. foliaceus. Both conditions begin with pustular, crusty lesions on the face and ears. However with p.

foliaceus, the lesions spread to the feet, the groin and other areas; there may be itching and pain; and severely affected dogs may lose their appetite and become depressed.

Loss of pigment in the nose is common with both forms of pemphigus, and this results in photodermatitis - increased sensitivity to the sun's rays so that the condition is worse in sunny weather.

How is pemphigus diagnosed?

P. foliaceus is the most commonly seen form of pemphigus; however it is still uncommon. The diagnosis is based on the history of how the condition developed, physical examination of the dog, and tests such as skin scrapings and smears, skin biopsy, and immune testing to rule out other causes of similar skin lesions such as a bacterial or fungal skin infection, mites, seborrhea, dermatomyositis, and lupus erythematosus.

Multiple skin biopsies will increase the chances of finding diagnostic histologic changes. Direct immunofluorescence or immunohistochemical testing may or may not be helpful. These tests have relatively low sensitivity and specificity.

How is pemphigus treated?

Treatment is based on suppressing the inappropriate immune response. For pemphigus foliaceus, steroids (as a cream, or orally as a tablet - prednisone) are used to accomplish this. Long term treatment is generally necessary. Where prednisone is not effective (as is sometimes the case), immunomodulating drugs or chrysotherapy (gold salts) will be tried.

For dogs that have lost pigment in the nose, protection against the sun is important to prevent flare-ups of the condition. Keep the dog out of the sun between 10:00 and 3:00 and/or use suncreens on the nose with SPF of 15 or higher.

The milder form, pemphigus erythematosus, may be successfully treated with sun avoidance and glucocorticoids applied to the skin. If this is ineffective, oral glucocorticoids or other drugs may be required.

A combination of tetracycline and niacinamide has been used with some success in dogs with p. erythematosus (Scott, 1995).

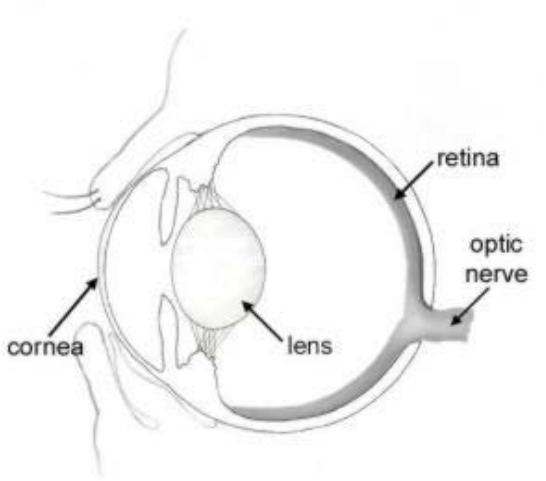
Breeding advice

It is advisable not to use affected dogs in breeding programmes, even though inheritance for these conditions has not been worked out.

PROGRESSIVE RETINAL ATROPHY

Related Terms: *central/generalized progressive retinal atrophy, retinal degeneration, early retinal degeneration, progressive rod-cone degeneration, rod-cone dysplasia, hemeralopia (day blindness), nyctalopia (night-blindness)*

The cells of the retina receive light stimuli from the external environment and transmit the information to the brain where it is interpreted to become vision. In progressive retinal atrophy (PRA), deterioration of the retinal cells causes blindness.



Adapted from D Slatter, Fundamentals of Veterinary Ophthalmology, 1990. Philadelphia. WB Saunders

The term progressive retinal atrophy covers several types of inherited degeneration (deterioration) of the retina. Sub-classifications of PRA are based on the age at which dogs show signs of the disease and the type of retinal cell which is affected.

Generalized PRA:These diseases affect primarily the photoreceptor cells. Both eyes are similarly affected and dogs eventually become totally blind.

i) Early onset photoreceptor dysplasias: In these conditions, the photoreceptor cells develop abnormally in the first few weeks after birth, and then degenerate along with the inner layers of the retina.

ii) Later onset photoreceptor degeneration (progressive rod-cone degeneration): Here the retina matures and functions apparently normally for varying periods of time before degenerating. Dogs are not usually clinically affected until 1 year of age or more, although abnormalities can be seen in the eye and on the electroretinogram (ERG) long before owners notice signs of visually impairment.

Progressive rod-cone degeneration has similarities to retinitis pigmentosa in people.

Central PRA:(also called RPE dystrophy) The abnormality is in the retinal pigmented epithelium (RPE). The photoreceptor cells will also degenerate eventually. The rate of vision loss is much slower than with generalized PRA, and not all dogs become totally blind.

How is progressive retinal atrophy inherited?

In most breeds studied to date, PRA is inherited as an autosomal recessive and may be categorised as simple recessive that may take years to develop and some research indicates that it may also be sex linked on the X chromosome.

Numbers Affected/Rate of Occurrence in the Chow Chow

Uncommon to rare

What does progressive retinal atrophy mean to the dog & its owner

Generalized PRA - early onset: The first sign is generally failing night vision, as early as 6 weeks of age, and this progresses to complete loss of vision by about 1 - 2 years of age. Collies may retain some vision until the age of 2 - 3 years. In miniature schnauzers, poor night vision usually develops later (6 months to a year) and there is advanced loss of vision by 3 to 4 years. Affected Alaskan malamutes are day-blind (hemeralopia) at 8 to 10 weeks of age; night vision is never affected.

Generalized PRA (progressive rod-cone degeneration) - late onset: Generally night blindness is noticed between 2 and 5 years of age (depending on the breed) progressing to total blindness within a year or so. Peripheral vision is lost first.

Central PRA (CPRA) - retinal pigment epithelial dystrophy (RPED): Loss of vision occurs much more slowly than in generalized PRA, without initial night blindness. Affected dogs may not lose vision completely. Because the changes are in the centre of the retina, affected dogs initially have trouble locating still objects in bright light.

How is progressive retinal atrophy diagnosed?

There are no obvious external changes to the eyes. It may be noticed that the dog has difficulty getting around when the lights are turned off, or when outside at night.

Generalized PRA

1. Ophthalmoscopic exam: retinal thinning is seen as hyper-reflectivity of the tapetal fundus, attenuation of the retinal vessels, and shrinking and pallor of the optical disc; cataracts and/or retinal detachment may occur late in the disease.

2. Electroretinogram: Generalized PRA can be detected by ERG long before it is apparent clinically.

3. DNA testing: Rod-cone dysplasia or rcd1 can be detected in Irish Setters by polymerase chain reaction. DNA testing for PRA is also available for Chesapeake Bay retrievers, Labrador retrievers and Portuguese water dogs. For more information, see resources below.

CPRA

1. Ophthalmoscopic exam: initially central multiple light to dark brown spots within tapetal fundus, varying in size, shape and density, due to accumulation of lipopigment within the photoreceptor layer. You will also see hyper-reflectivity and retinal vessel attenuation as the disease progresses.

2. Electroretinogram: The ERG has not been found useful in the early diagnosis of CPRA because the photoreceptor cells are only affected later in the course of the disease.

Genetic testing is quickly becoming available for different forms of PRA in different breeds. The advantage of such testing is that it can identify dogs whose sight is unaffected, but who are carriers of the disorder (heterozygotes).

How is progressive retinal atrophy treated?

There is no treatment for PRA. The degree of visual impairment varies with the breed and specific type of retinal degeneration as described above, but most affected dogs will ultimately be completely blind. With their acute senses of smell and hearing, dogs can compensate very well, particularly in familiar surroundings, to the point where owners may be unaware of the extent of vision loss.

Breeding advice

Breeding is not advised for any dog with PRA, or for the parents (assumed to be carriers). Siblings should be carefully screened by electroretinogram if they are considered for breeding. Generalized PRA can often be detected by electroretinography at least a year before clinical signs are apparent.

For some disorders, blood-based DNA tests may be available which can distinguish normal animals from those who are clinically normal but are carriers, and from those that are affected but are not yet showing any signs.

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GLOSSARY OF GENETIC TERMS

Alleles: different versions of the same gene (found at the same locus but in homologous chromosomes or in different individuals) that may produce different phenotypes.

Allele frequency: the fraction of all the alleles of a gene in a population that are of one type.

Assortative mating: a mating scheme that relies on the pairing of unrelated individuals with similar phenotypes to obtain consistency of type and reinforce desirable traits.

Codominant alleles: two alleles that have different effects that are distinguishable in a heterozygous individual (e.g. AB blood groups)

Cross-breeding: crossing two different breeds.

Dominant allele: one that determines the phenotype even when there is only one copy (i.e. in a heterozygous individual).

Drift: changes in allele frequencies over time due to chance (as opposed to selection or mutation).

Effective population size (Ne): the size of a hypothetical stable, randomly-mating population that would have the same rate of gene loss or increase in inbreeding as the real population (size N). As all finite populations are inbred to some degree and generally do not choose mates at random, Ne is typically 1/10 N or less, particularly if fewer males breed than females.

Epistasis: used to describe the situation where one gene's expression prevents the expression of another (e.g. you cannot determine whether an albino would have had black or brown hair, though these two traits are controlled by separate genes.)

Fitness (relative): The reproductive success of individuals of a particular genotype relative to the most fit genotype.

Fixation: loss of all alleles of a gene but one.

Founder: an individual drawn from a source population who contributes genetically to the derived subpopulation.

Founder effect: changes in allele frequencies that occur when a subpopulation is formed from a larger one. Typically many rare and usually undesirable alleles are excluded while a few carried by the founders get a big boost in frequency.

Founder equivalents: the number of hypothetical founders that would have the same diversity as the descendant population. Generally much smaller than the actual number due to unequal use and allele loss (gene dropping).

Gene: that portion of the genome that carries the information for a single protein. (In cases of proteins with multiple subunits, there may be a gene for each.)

Gene dropping: loss of alleles due to genetic drift.

Genetic bottleneck: when population numbers are temporarily reduced to a level insufficient to maintain the diversity in the population.

Genetic diversity: usually expressed in terms of percentage of genes that are polymorphic and/or are heterozygous.

Genome: the total genetic makeup of an organism.

Heritable: passed on from parents to progeny through the chromosomes/DNA.

Heritability: the fraction of the variability in a trait that is caused by genetic differences.

Heterozygous: carrying two different alleles of a gene.

Heterozygous advantage: a situation where the heterozygous genotype for a particular gene shows the highest relative fitness.

Heterozygous insufficiency: when the heterozygous genotype lacks sufficient gene product to have the normal phenotype. (Approximately equivalent to partial dominance.)

Heterosis: a situation where crossing two inbred lines yields progeny that are more healthy/vigorous than their parents. (More commonly used in plant breeding.)

Homologous chromosomes: in higher plants and animals, chromosomes are found in nearly identical "homologous" pairs, one coming from the sire and the other from the dam. A dog has 39 pairs or 78 in total. Only one of each, chosen at random, is passed on through eggs or sperm to the progeny.

Line-breeding: a scheme that attempts to maintain a high contribution of one or two ancestors through successive generations. Often used by breeders for any inbreeding less intensive than between first-degree relatives.

Linkage: a measure of how frequently two genes found on the same chromosome remain together during gamete (egg or sperm) formation.

Locus: the location of a gene on a chromosome.

Map (a.k.a., linage map): a drawing showing the location of and relative distances between genes on a chromosome.

Mean kinship (mk): a measure of how related an individual is to the other members of a population. Generally computed as the average IC (inbreeding coefficient)for the hypothetical progeny of the individual mated to all other members of the population (both sexes). A low average mk for a population indicates that most of the diversity carried by the founders has been retained.

Monomorphic genes: have only one common allele (rare alleles with frequencies of less than 0.001% may still occur).

Mutation: a change in the sequence of the base pairs in a DNA molecule.

Mutation rate: the number of new mutations that occur per gene per gamete per generation.

Outcrossing: mating two individuals of the same breed that are sufficiently unrelated that the IC of the progeny is lower than the average of the parents.

Polymorphic genes: have 2 or more common alleles in the population.

Recombination: the reciprocal exchange of portions of two homologous chromosomes (usually equivalent) during gamete formation.

Recombinant frequency (RF): how often two linked genes are separated by recombination, generally expressed as a percentage of total progeny.

GENETIC INHERITANCE CHARTS

The charts on the following pages have been taken directly from Section 7 of the Prevention of Cruelty to Animals Act: Code of Practice for the Responsible Breeding of Animals With Heritable Disease -14 Jan 2008 Consultation Draft

7 Heritable disease groups

7.1 Heritable disease caused by a simple dominant defective gene

Carrier (is affected) = heterozygote (ie. 1 clear gene and 1 defective gene), displays degrees of disease
Affected= homozygous for heritable defect genes (ie 2 defective genes) displays severe form of disease
Clear = homozygous for clear genes (ie. 2 clear genes) and is free of the disease

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Parent combination	Theoretical status of progeny	Heritable disease requirements
Clear x Clear	100% Clear	No restriction
Clear x Carrier (& Clear x Unknown)	** 50 % Clear 50 % Carrier (that may be affected to some degree)	1. All progeny should be DNA tested for the heritable disease by an approved collection officer. 2. The degree of disease in carrier animals should be assessed by a veterinary practitioner and the animal managed in accordance with the instructions of a veterinary practitioner. 3. Carrier animals must not be disposed of to another person without advice of the animals heritable disease status 4. Carriers should be de-sexed prior to sale unless to be used in an aproved breeding program
Carrier x carrier (&Carrier x Unknown)	** 25% Clear 50% Carrier (is affected to some degree) 25% Affected (usually seriously)	1. **Prohibited unless** as part of a planned long term breeding program approved by an approved organisation listed in this code for the type of animal. 2. All progeny must be DNA tested for the heritable disease by an approved collection officer 3. Records of carrier and affected progeny must be marked with their test status 4. The severity of disease in carrier animals must be assessed by a veterinary practitioner and the animal managed in accordance with the instructions of a veterinary practitioner. 5. Carrier and affected animals must not be disposed of to another person without advice of the animals heritable disease status. 6. If kept alive affected animals must be de-sexed prior to disposal, must not be permitted to suffer from their condition by their owner and must be under the supervision and monitoring of a veterinary practitioner.
Affected x Clear	100% Carrier (all will have a degree of the disease)	1. **Prohibited unless** as part of a planned long term breeding program approved an approved organisation listed in this code for the type of

		animal. 2. Records of progeny must be marked as carrier status unless otherwise certified by a veterinary practitioner 3. The degree of disease in carrier animals must be assessed by a veterinary practitioner and the animal managed in accordance with the instructions of a veterinary practitioner. 4. Carrier animals must not be disposed of to another person without advice of the animals heritable disease status 5. All progeny not being used in a breeding program must be de-sexed.
Affected x Carrier & Affected by Affected	50% carrier , 50% Affected(all be affected to some degree) 100% Affected(usually seriously)	**1. Prohibited** 2. Intentional or reckless use of this combination is an offence under the Act. **3.** If kept alive affected progeny must be de-sexed prior to disposal, must not be permitted to suffer from their condition by the owner and must be under the supervision and monitoring of a veterinary practitioner.
Unknown X Unknown		1. **Prohibited unless** all progeny are tested for the heritable disease by an approved collection officer 2. Records of carrier and affected progeny must be marked with their status 3. This is considered reckless breeding in view of the definition of 'unknown'

**Testing is preferred as in practice the unpredictable nature of the process of gene inheritance in these combinations may cause variation in the actual % outcomes per generation. As carriers may express varying degrees of the heritable disease they must be tested, assessed and monitored by a veterinary practitioner experienced with the disease to determine the impact on the animal.

Carrier and affected animals should be de-sexed if not to be used in a breeding program.

7.2 Heritable disease caused by a simple recessive defective gene resulting in severe disease

Carrier = heterozygote (ie. 1 clear gene and 1 defective gene) and does not exhibit the disease
Affected= homozygous for heritable defect genes (ie, 2 defective genes) and is affected by the disease
Clear = homozygous for clear genes (ie. 2 clear genes) and is free of the disease

Parent combination	Theoretical status of progeny	Heritable disease requirements
Clear x Clear	100% Clear	No restriction
Carrier x Clear (& Clear x Unknown)	** 50 % Clear 50 % Carrier	1. All progeny will be unaffected by the disease 2. All progeny to be used for ***breeding purposes*** must be DNA tested for the heritable disease by an approved collector officer prior to use in a

		breeding program. 3. Records of carrier progeny must be marked with their status. 4. Carrier animals that are not to be used for breeding purposes should be desexed.
Carrier x Carrier	** 25% Clear 50% Carrier 25% Affected	1. **Not recommended.** Must only occur as part of a planned long term breeding program approved by an approved organisation listed in this code for the type of animal. 2. All resultant progeny must be DNA tested for the heritable disease by an approved collection officer. 3. Records of carrier and affected progeny must be marked with their test status. 4. Affected animals must not be disposed of to another person without advice of the animal's heritable disease status. 5. If kept alive affected progeny must be de-sexed prior to disposal, must not be permitted to suffer from their condition by their owner and must be under the supervision, advice and monitoring of a veterinary practitioner.
Carrier x unknown status	Outcome may be one of the above two options depending on the status of the untested parent	1. **Not recommended**. All progeny must be tested for the heritable disease by an approved collection officer. 2. Records of carrier and affected progeny must be marked with their status.
Affected x Clear	100% Carrier	1. **Not recommended.** Must only occur as part of a planned long term breeding program approved an approved organisation listed in this code for the type of animal. 2. Records of progeny must be marked with carrier status.
Affected x Carrier Affected x Affected	50% Carrier 50% Affected 100% Affected	**1. Prohibited** 2. Intentional or reckless use of this combination is an offence under the Act. 3. Affected animals must not be disposed of to another person of to another person without advice of the animal's heritable disease status 4. If kept alive affected progeny must be de-sexed prior to disposal, must not be permitted to suffer from their condition by the owner and must be under the supervision and monitoring of a veterinary practitioner.
Unknown X Unknown		**1. Prohibited unless** all progeny are tested for the heritable disease by an approved collection officer **2.** Records of carrier and affected progeny must be marked with their status

**Testing is required as in practice the unpredictable nature of the process of gene inheritance in these combinations may cause variation in the actual % outcomes per generation.

Carrier animals should be de-sexed if not to be used in a breeding program.
Affected animals must be de-sexed if not to be used in a breeding program.

7.3 Heritable disease caused by simple recessive gene that may take years to develop symptoms of the disease

Dogs:
Progressive Retinal Atrophy (in those breeds affected by the *pcrd* form, also *rcd 1,2,3)*

Hereditary Cataract (in breeds where a simple recessive mode has been scientifically established).

Parent combination	Theoretical status of progeny	Heritable disease requirements
Clear x Clear	100% Clear	No restriction
Clear x Carrier Clear x Unknown	50 % Clear 50 % Carrier	1. All progeny will be unaffected by the disease. 2. Any progeny that is to be used for ***breeding purposes*** should be DNA tested for the heritable disease by an approved collection officer. prior to sale or use in a breeding program (which ever occurs first) 3. Records of tested progeny must be marked with their DNA status.
Carrier x Carrier	** 25% Clear 50% Carrier 25% Affected	a. **Not recommended.** Must only occur as part of a planned long term breeding program approved an approved organisation listed in this code for the type of animal. b. All resultant progeny must be DNA tested for the heritable disease by an approved collection officer. c. Records of carrier and affected progeny must be marked with their test status. d. Affected animals must not be disposed of to another person without advice of the animal's heritable disease status. e. If kept alive, ***affected*** progeny (or any juvenile offspring confirmed as 'Affected' on genetic test) should be de-sexed prior to disposal, must not be permitted to suffer from their condition by their owner and must be under the supervision, advice and monitoring of a veterinary practitioner.
Carrier x unknown status	Outcome may be one of the above two options depending status of untested parent	1. Not recommended unless all progeny are tested for the heritable disease by an approved collection officer. 2. Records of carrier progeny must be marked with their status.
Affected x Clear	100% Carrier	a. **Not recommended**. Should only occur as part of a planned long term breeding program approved by an approved organisation listed in this code for the type of animal. b. Records of resultant progeny must be marked with carrier status.

Affected x Carrier Affected x Affected	50% Carrier 50% Affected 100% Affected	**1. Prohibited** 2. Intentional or reckless use of this combination is an offence under the Act. 3. Affected animals must not be disposed of to another person of to another person without advice of the animal's heritable disease status **4.** If kept alive affected progeny must be de-sexed prior to disposal, must not be permitted to suffer from their condition by the owner and must be under the supervision and monitoring of a veterinary practitioner.
Unknown X Unknown		**1. Prohibited unless** all progeny are tested for the heritable disease by an approved collection officer. **2.** Records of carrier and affected progeny must be marked with their status

**Testing is required as in practice the unpredictable nature of the process of gene inheritance in these combinations may cause the actual % outcomes per generation to vary from the theoretical outcomes.

Carrier and affected animals should be de-sexed if not to be used in a breeding program.

7.4 Heritable disease caused by simple recessive genes that are sex linked (or show weak penetrance or limited expression resulting in only a few affected individuals)
See section 3.4.

7.5 Heritable disease caused by a simple recessive defective gene that is dependant on over-riding or modifying genetic effects for full expression of disease.

This includes conditions where the vast majority of genetically affected individuals do not exhibit the full range of clinical signs of the disease unless modifying factors are present - factors that directly influence the degree to which the disease is ultimately expressed

Parent combination	**Theoretical status of progeny**	**Heritable disease requirements**
Clear x Clear	100% Clear	No restriction
Clear x Carrier (& Clear x Unknown)	 50 % Clear 50 % Carrier	1. All progeny will be unaffected by the disease. 2. Any progeny that is to be used for ***breeding purposes*** should be DNA tested for the heritable disease by an approved collection officer prior to sale or use in a breeding program (which ever occurs first) 3. Records of tested progeny must be marked with their DNA status.
Carrier x Carrier	** 25% Clear 50% Carrier 25% Affected	1. Should only occur as part of a planned long term breeding program approved by an approved organisation listed in this code for the type of animal.

		2. All resultant progeny should be DNA tested for the heritable disease by an approved collection officer 3. Records of carrier and affected progeny must be marked with their test status 4. Affected animals must not be disposed of to another person without advice of the animal's heritable disease status 5. If kept alive, adversely ***affected*** animals should be de-sexed, must not be permitted to suffer from their condition by their owner and must be under the supervision, advice and monitoring of a veterinary practitioner.
Carrier x unknown status	Outcome may be one of the above two options depending status of untested parent	1. All progeny should be tested for the heritable disease by an approved collection officer 2. Records of carrier progeny must be marked with their status. 3. Affected animals must not be disposed of to another person without advice of the animal's heritable disease status 4. If kept alive, adversely ***affected*** animals should be de-sexed prior to disposal, must not be permitted to suffer from their condition by their owner and must be under the supervision, advice and monitoring of a veterinary practitioner.
Affected x Clear	100% Carrier	1. Should only occur as part of a planned long term breeding program approved by a relevant applicable organisation or an organisation listed in this code for the type of animal. 2. Records of resultant progeny must be marked with carrier status.
Affected x Carrier Affected x Affected	50% Carrier 50% Affected 100% Affected	1. **Prohibited** unless as part of a planned long term breeding program approved by an approved organisation listed in this code for the type of animal. 2. Intentional or reckless use of this combination outside of an approved breeding program is an offence under the Act. 3. Affected animals must not be disposed of to another person of to another person without advice of the animal's heritable disease status 4. If kept alive, adversely **affected** progeny must be de-sexed prior to disposal, must not be permitted to suffer from their condition by the owner and must be under the supervision and monitoring of a veterinary practitioner.

**Testing is required as in practice the unpredictable nature of the process of gene inheritance in these combinations may cause variation in the actual % outcomes per generation.