

# Poker

Breed: Labrador Retriever  
Microchip number: 900115001638680  
Birth date: 2024-09-29

Registration number: X  
Test date: 2025-02-25  
ID kit: DCSWMFT



## Poker’s Profile

### Pet information

Registered name	Sex
Poker	M
Owner reported breed	Date of birth
Labrador Retriever	2024-09-29
Microchip number	
900115001638680	

### Genetic Diversity

Poker’s Percentage of Heterozygosity  
32%

### Health summary

- At Risk0 conditions
- Carrier1 condition
  - Exercise-Induced Collapse
- Clear271 conditions

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## Genetic Diversity

### Heterozygosity

#### Poker's Percentage of Heterozygosity

32%

Poker's genome analysis shows an average level of genetic heterozygosity when compared with other Labrador Retrievers.

#### Typical Range for Labrador Retrievers

31% - 40%

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## Health conditions known in the breed

Exercise-Induced Collapse	Gene	Risk Variant	Copies	Inheritance	Result
	DNM1	G>T	1	AR	Carrier

**Information about the genetic condition**

Affected dogs appear normal during low to moderately strenuous activity, but they develop a wobbly, uncoordinated gait that is most severe in the hind limbs after brief bouts of strenuous activity. Typically the dogs remain conscious and are not in pain during an episode. In some cases, however, the signs are severe with full body weakness and low muscle tone (flaccid paralysis), confusion, loss of consciousness, and seizures. Very rarely, death can occur. The episodes typically last 5 to 10 minutes and most dogs will recover completely within 15 to 30 minutes.

**Breeder recommendation**

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to be shown. A carrier dog with one copy of the EIC mutation can be safely bred with a clear dog with no copies of the EIC mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the EIC mutation. A dog with two copies of the EIC mutation can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the EIC mutation could develop due to a different genetic or clinical cause.

Alexander Disease	Gene	Risk Variant	Copies	Inheritance	Result
	GFAP	G>A	0	AR	Clear

**Information about the genetic condition**

Clinical signs of this disorder emerge around three months of age and begin with weakness of movement in the limbs, causing a spastic swimming-puppy-like position of the front legs. The dog can also develop other neurological signs, such as mild vestibular signs and myoclonic jerks. The chest of affected puppies can be flat and regurgitation can occur. The disorder is progressive and in a few weeks, the limb weakness during voluntary movement progresses to where the dog is unable to stand. The disease should be a differential diagnosis based on breed history and clinical presentation when other possible causes have been ruled out. MRI and electrodiagnostic tests can be used for further diagnostics.

**Breeder recommendation**

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to develop. A carrier dog with one copy of the Alexander Disease mutation can be safely bred with a clear dog with no copies of the Alexander Disease mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Alexander Disease mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the Alexander Disease mutation could develop due to a different genetic or clinical cause.



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## Health conditions known in the breed

Centronuclear Myopathy (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	PTPLA	Insertion	0	AR	Clear

### Information about the genetic condition

In Labrador Retrievers, weakened or non-existent peripheral reflexes of the legs can occasionally be observed as early as 1 month of age, although more typically not until the puppies are 2 to 5 months old. At the age of 5 months, the affected puppies manifest decreased movement and exercise intolerance. The condition does not usually progress after the first year of life. In adult dogs, the most common signs are severe skeletal muscle atrophy especially in the areas of head, neck, and legs that limit the dog's ability to keep his head raised (ventroflexion) as well as abnormalities in posture and movements. Although the disorder limits the dog's athletic potential, it can still have a normal life span as a pet.

### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to develop. A carrier dog with one copy of the CNM mutation can be safely bred with a clear dog with no copies of the CNM mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the CNM mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Please note: It is possible that disease signs similar to the ones caused by the CNM mutation could develop due to a different genetic or clinical cause.

Congenital Cornification (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	NSDHL	Deletion	0	XD	Clear

### Information about the genetic condition

Congenital Cornification, also known as inflammatory linear verrucous epidermal nevi (ILVEN), is an X-linked dominant disorder characterized by symmetrical, sharply demarcated alopecia, hyperkeratosis (thickening of the outer layer of skin), brown-black scaling, and dilated, keratin-filled follicles that can appear in stripes over the head, trunk and limbs in a distinct patterning known as Blaschko's lines. The lesions appear shortly after birth and can be quite painful. Secondary skin infections are common in affected individuals, and paw pad hyperkeratosis can lead to significant lameness. Affected dogs may also show stunted growth. Histological examination can reveal hyperplastic epidermis, hyperkeratosis and parakeratosis while the follicular infundibula may appear distended with parakeratotic keratin. Unfortunately, this disorder is considered lethal in affected males, and death tends to occur in utero or shortly after birth (with mouse models showing abnormally thin placental tissue and fewer blood vessels). Thus, current research describes affected females as having one copy of the X-linked variant. More research is needed to understand the possibility of and effects on females with two copies of this variant.

### Breeder recommendation

This disorder is X-linked dominant, meaning the genetic variant is found on the X chromosome. However, since it is dominant in nature, males and females only require one copy of the variant to be at an elevated risk for being diagnosed with this condition. Due to the severity of the disorder, use of dogs with one or two copies of the Congenital Cornification (Discovered in the Labrador Retriever) variant are not recommended for breeding, as there is a risk that the resulting litter will contain affected puppies. Please note: It is possible that clinical signs similar to the ones associated with the Congenital Cornification (Discovered in the Labrador Retriever) variant could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

Congenital Myasthenic Syndrome (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	COLQ	T>C	0	AR	Clear

### Information about the genetic condition

Initial signs of congenital myasthenic syndrome can be observed in 12 to 16 weeks old puppies. Affected dogs suffer from exercise intolerance and collapse after 5 to 30 minutes of exercise. Before collapsing, affected dogs will start to take shorter and shorter strides and eventually fall down. Affected dogs are able to recover from the transient paralysis after resting for a few minutes, but the signs reappear if the dog continues to run.

### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to be shown. A carrier dog with one copy of the CMS mutation can be safely bred with a clear dog with no copies of the CMS mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the CMS mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the CMS mutation could develop due to a different genetic or clinical cause.

Ehlers-Danlos Syndrome (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	COL5A1	Deletion	0	AD	Clear

### Information about the genetic condition

Ehlers-Danlos Syndromes are a group of inherited connective tissue disorders characterized by skin hyperextensibility, tissue fragility and generalized joint hypermobility. Affected dogs can show clinical signs of EDS from a young age. Initial signs may be thin, hyperextensible skin that tears and bruises easily. Lacerations can present following even minor trauma, and affected dogs tend to demonstrate poor wound healing. Additional signs may include hypermobile joints and seroma-like swellings following trauma. Affected dogs can otherwise appear clinically healthy and have normal skeletal development.

### Breeder recommendation

This disorder is autosomal dominant, meaning dogs with one or two copies of the Ehlers-Danlos Syndrome (Discovered in the Labrador Retriever) variant are at an elevated risk for being diagnosed with this condition. Use of dogs with one or two copies of the Ehlers-Danlos Syndrome (Discovered in the Labrador Retriever) variant are not recommended for breeding, as there is a risk that the resulting litter will contain affected puppies. For example, if a dog with one copy of the Ehlers-Danlos Syndrome variant is bred with a clear dog with no copies of the Ehlers-Danlos Syndrome variant, about half of the puppies will have one copy and half will have no copies of the variant. Please note: It is possible that clinical signs similar to the ones associated with this Ehlers-Danlos Syndrome variant could develop due to a different genetic or clinical cause.

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Health conditions known in the breed

Hemophilia A (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	Confidential	-	0	XR	Clear

Information about the genetic condition

Hemophilia A is an inherited bleeding disorder characterized by a deficiency in Factor VIII protein, which is necessary in the blood coagulation process. Clinical signs of Hemophilia A vary depending on the activity of Factor VIII in the blood. Signs may include excessive and prolonged bleeding following traumas or when shedding deciduous teeth, lameness and swelling of the joints, and subdermal hematoma formation. Bleeding into the chest or abdominal cavity may be observed in severely affected dogs and often carries a poor prognosis. The disorder follows an X-linked recessive mode of inheritance and, therefore, is more commonly observed in male dogs as males only have one X chromosome. Additionally, large or active dogs are usually associated with more severe signs.

Breeder recommendation

This disorder is X-linked recessive, meaning the genetic variant is found on the X chromosome. Given males only have one X chromosome, a single affected copy will increase the risk of being diagnosed with Hemophilia A (Discovered in the Labrador Retriever). Females typically require two copies to be at an elevated risk. Use of dogs with one or two copies of the Hemophilia A variant is not recommended for breeding as there is a risk that the resulting litter will contain affected puppies. Please note: It is possible that clinical signs similar to the ones caused by this variant could develop due to a different genetic or clinical cause.

Hereditary Elliptocytosis	Gene	Risk Variant	Copies	Inheritance	Result
	SPTB	C>T	0	AD	Clear

Information about the genetic condition

Only one genetic mutation associated with elliptocytosis has been previously described. In a case study on one dog, a Labrador and Chow Chow mixed breed, the patient presented with persistent elliptocytosis, decreased mechanical deformability of erythrocytes and decreased erythrocyte membrane stability. Molecularly, elliptocytosis was found to be due to a defect in the erythrocyte membrane protein beta-spectrin. The studied dog was found to carry one copy of a mutation in the beta-spectrin encoding gene. Further information on the mutation is needed to examine whether dogs with two copies of the mutation have a more severe hemolytic, elliptocytic anemia.

Breeder recommendation

This disease is autosomal dominant meaning that one copy of the mutation is needed for disease signs to occur. Use of dogs with one or two copies of the disease mutation for breeding is not recommended, as there is a risk that the resulting litter will contain affected puppies. For example if a dog with one copy of the Hereditary Elliptocytosis mutation is bred with a clear dog with no copies of the Hereditary Elliptocytosis mutation, about half of the puppies will have one copy and half will have no copies of the Hereditary Elliptocytosis mutation. Please note: It is possible that disease signs similar to the ones caused by the Hereditary Elliptocytosis mutation could develop due to a different genetic or clinical cause.

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Hereditary Nasal Parakeratosis (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	SUV39H2	A>C	0	AR	Clear

Information about the genetic condition

Clinical signs of the disease typically emerge at 6-12 months of age as dry, rough crusts develop on the nose. Dryness of the skin can lead to cracks in the skin of the nose. These painful, bloody cracks can cause chronic irritation and inflammation of the noses skin. Depigmentation of the nose occurs over time and the nose changes from dark to a lighter color. The condition can be managed with life-long care and medication.

Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to develop. A carrier dog with one copy of the HNPk mutation can be safely bred with a clear dog with no copies of the HNPk mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the HNPk mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the HNPk mutation could develop due to a different genetic or clinical cause.

Hyperuricosuria	Gene	Risk Variant	Copies	Inheritance	Result
	SLC2A9	G>T	0	AR	Clear

Information about the genetic condition

HUU predisposes affected dogs to the formation of urate stones. Clinical signs of urolithiasis include hematuria, pain while urinating, and blockage of the urinary tract. Patients with urinary stones are more susceptible to urinary tract infections. Blockage of the urinary tract is a life-threatening condition that requires immediate veterinary care. In Dalmatians, the clinical signs are more common in males than in females. As many as 34% of all male Dalmatians are diagnosed with urate stones.

Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the HUU mutation can be safely bred with a clear dog with no copies of the HUU mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the HUU mutation. A dog with two copies of the HUU mutation can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. In some breeds, such as the Dalmatian, the frequency of the disease mutation is very high. Carriers and dogs with two copies of the disease mutation (genetically affected dogs) should be used for breeding purposes, with the aim of gradually reducing the frequency of the mutant gene within the breed population. Where possible, matings should be avoided that would result in litters that could contain dogs with two copies of the disease mutation, such as a mating between two dogs with two copies of the HUU mutation or between a dog with one copy and a dog with two copies of the HUU mutation. Please note: It is possible that disease signs similar to the ones caused by the HUU mutation could develop due to a different genetic or clinical cause.

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Health conditions known in the breed

Muscular Dystrophy-Dystroglycanopathy (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	LARGE	C>T	0	AR	Clear

Information about the genetic condition

Muscular Dystrophy-Dystroglycanopathy is a type of muscular dystrophy that is caused by defects in the modification of proteins by addition of sugars, also known as the cellular glycosylation process. Affected dogs will demonstrate signs of MDD as newborn puppies. Clinical signs can include difficulties feeding, poor weight gain, small stature, bow legged stance and weakness. Serum creatinine kinase (CK) values can be elevated in affected puppies. Histopathology can confirm the diagnosis of muscular dystrophy by showing the degenerative and regenerative changes consistent with a dystrophic phenotype. Due to poor suckling and difficulties eating, affected puppies experience a general failure to thrive and are unlikely to survive beyond a young age.

Breeder recommendation

This disorder is autosomal recessive, meaning two copies of the variant are needed for a dog to be at an elevated risk for being diagnosed with the condition. A carrier dog with one copy of the Muscular Dystrophy-Dystroglycanopathy (Discovered in the Labrador Retriever) variant can be safely bred with a clear dog with no copies of the Muscular Dystrophy-Dystroglycanopathy (Discovered in the Labrador Retriever) variant. About half of the puppies will have one copy (carriers) and half will have no copies of the variant. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disorder signs similar to the ones associated with this Muscular Dystrophy-Dystroglycanopathy variant could develop due to a different genetic or clinical cause.

Myotonia Congenita (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	CLCN1	T>A	0	AR	Clear

Information about the genetic condition

The clinical signs of Myotonia congenita can be seen in puppies only a few weeks old. Affected dogs suffer from muscle hypertrophy with stiff movements. There may be difficulties rising after rest or in attempting rapid changes in posture. Affected dog may also display a "bunny-hopping" gait. Additional clinical signs include difficulty swallowing, ptialism (excessive flow of saliva), and increased respiratory sounds during exercise. The tongue of affected dogs is enlarged and stiffens when touched.

Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to develop. A carrier dog with one copy of the Myotonia Congenita mutation can be safely bred with a clear dog with no copies of the Myotonia Congenita mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Myotonia Congenita mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the Myotonia Congenita mutation could develop due to a different genetic or clinical cause.



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## Health conditions known in the breed

Narcolepsy (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	HCRT2	G>A	0	AR	Clear

**Information about the genetic condition**

The first clinical signs of inherited narcolepsy are usually observed by 6 months of age. A typical sign of narcolepsy is excessive daytime drowsiness or decreased daytime activity compared to dogs of the same breed and age. The clinical signs also include cataplexic episodes characterized by sudden loss of muscle tone. Cataplexic episodes start with the dog's hind limbs bending and neck hanging down followed by a collapse which might result in the dog laying down for several seconds or minutes. An affected dog may try to resist the attack which can be seen as a wobbly gait and hind limb weakness. The dog usually stays conscious and alert especially in the beginning of the episode. However, if the attack lasts longer than a couple of minutes, the dog may fall asleep. In longer episodes, fast eye movement characteristic for REM sleep can be observed. Muscle twitches and slow repetitive muscle movements are also possible. Unlike in epileptic seizures, muscles are relaxed during cataplexic episodes and no drooling, urinating, or defecation is observed. Feeding and playing with the dog can provoke cataplexic episodes.

**Breeder recommendation**

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the Narcolepsy mutation can be safely bred with a clear dog with no copies of the Narcolepsy mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Narcolepsy mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the Narcolepsy mutation could develop due to a different genetic or clinical cause.

Obesity risk (POMC)	Gene	Risk Variant	Copies	Inheritance	Result
	POMC	Deletion	0	AD	Clear

**Information about the genetic condition**

Key signs include increased appetite compared to dogs without the mutation, hunger even after being fed, and obesity. The effect is more notable in dogs with two copies of the POMC mutation.

**Breeder recommendation**

This disease is autosomal dominant meaning that one copy of the mutation is needed for disease signs to develop. As a healthy weight can be maintained for dogs with this mutation through appropriate diet and exercise, dogs with the POMC mutation can be considered for breeding purposes. This will also help to maintain diversity in breeds such as the Labrador Retriever and Flat-Coated Retriever where the mutation is very common.

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Health conditions known in the breed

Progressive Rod Cone Degeneration (prcd-PRA)	Gene PRCD	Risk Variant G>A	Copies 0	Inheritance AR	Result Clear
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Information about the genetic condition

Clinical signs of PRCD are related to progressive loss of function of rod photoreceptors, followed by loss of function of cone photoreceptors. Typical signs of disease include hyper-reflective tapetum and attenuated blood vessels. Age of onset for this form of PRA is generally early adulthood, although exact age of onset may vary significantly among different breeds. The disorder is progressive, causing increasing levels of vision loss and eventual blindness.

Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the prcd-PRA mutation can be safely bred with a clear dog with no copies of the prcd-PRA mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the prcd-PRA mutation. A dog with two copies of the prcd-PRA mutation can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the prcd-PRA mutation could develop due to a different genetic or clinical cause.

Skeletal Dysplasia 2	Gene COL11A2	Risk Variant G>C	Copies 0	Inheritance AR	Result Clear
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Information about the genetic condition

Affected dogs have shorter limbs, but otherwise normal build. Forelegs are usually slightly more affected than hind legs. Shoulder height is lower (<50 cm) compared to the international breed standard (54-57 cm), which can be observed after the dog's growth period is finished. In contrast to other skeletal dysplasias, no auditory problems, deafness, or secondary joint problems are associated with SD2. Because the mutation is superimposed on the normal variation seen in the breed, it can be difficult to identify the trait in some individuals.

Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the SD2 mutation can be safely bred with a clear dog with no copies of the SD2 mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the SD2 mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Please note: It is possible that disease signs similar to the ones caused by the SD2 mutation could develop due to a different genetic or clinical cause.



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## Health conditions known in the breed

Stargardt Disease (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	ABCA4	Insertion	0	AR	Clear

### Information about the genetic condition

Clinical signs include variable reflectivity of the tapetum and attenuated blood vessels. Age of onset for this form of PRA is typically late, although onset age can vary significantly. The disorder is progressive, causing increasing levels of vision loss and may eventually lead to blindness.

### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the STGD mutation can be safely bred with a clear dog with no copies of the STGD mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the STGD mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the STGD mutation could develop due to a different genetic or clinical cause.

X-Linked Myotubular Myopathy	Gene	Risk Variant	Copies	Inheritance	Result
	MTM1	C>A	0	XR	Clear

### Information about the genetic condition

The clinical signs of X-linked myotubular myopathy can be seen in puppies as young as 10 to 19 weeks of age with early signs of muscle atrophy being apparent earlier in some cases. Pelvic limb weakness is typically observed as one of the first signs. Affected dogs also lack patellar reflexes. X-linked myotubular myopathy is characterized by rapidly progressing muscle weakness and muscle atrophy. Affected dog won't be able to rise and move unassisted within a few weeks of the onset of clinical signs and may also have difficulties chewing and swallowing. There is no cure for the condition.

### Breeder recommendation

This disorder is X-linked recessive, meaning the genetic variant is found on the X chromosome. Given males only have one X chromosome, a single affected copy will increase the risk of being diagnosed with the disorder. Females typically require two copies to be at an elevated risk for full clinical signs but are not generally seen due to the poor prognosis for affected males. Use of dogs with one or two copies of the variant is not recommended for breeding as there is a risk that the resulting litter will contain affected puppies. Please note: It is possible that clinical signs similar to the ones caused by this variant could develop due to a different genetic or clinical cause.

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## Traits

### Coat Color

	Gene	Variant	Copies	Result
Fawn	ASIP	a <sup>y</sup>	0	No effect
Recessive Black	ASIP	a	0	No effect
Tan Points Two copies, or occasionally one copy, of this variant may result in a black and tan coat color pattern.	ASIP	a <sup>t</sup>	2	Tan points possible
Dominant Black One or two copies of the dominant black will give a dog a black coat (depending on other variants), black eye rims, nose and pads. One copy may also give a tiger striped appearance, known as brindle patterning.	CBD103	K <sup>B</sup>	2	Black possible
Mask	MC1R	E <sup>m</sup>	0	No effect
Recessive Red (e1)	MC1R	e <sup>1</sup>	0	No effect
Recessive Red (e2)	MC1R	e <sup>2</sup>	0	No effect
Recessive Red (e3)	MC1R	e <sup>3</sup>	0	No effect
Sable (Discovered in the Cocker Spaniel)	MC1R	e <sup>H</sup>	0	No effect
Widow's Peak (Discovered in Ancient dogs)	MC1R	e <sup>A</sup>	0	No effect
Widow's Peak (Discovered in the Afghan Hound and Saluki)	MC1R	E <sup>G</sup>	0	No effect

### Color Modification

	Gene	Variant	Copies	Result
Cocoa (Discovered in the French Bulldog)	HPS3	co	0	No effect
Red Intensity	MFS12	i	0	No effect
Dilution (d1) Linkage test	MLPH	d <sup>1</sup>	0	No effect
Dilution (d2)	MLPH	d <sup>2</sup>	0	No effect
Dilution (d3)	MLPH	d <sup>3</sup>	0	No effect

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## Color Modification

	Gene	Variant	Copies	Result
Chocolate (basd)	TYRP1	basd	0	No effect
Chocolate (bc)	TYRP1	bc	0	No effect
Chocolate (bd)	TYRP1	bd	0	No effect
Chocolate (be)	TYRP1	be	0	No effect
Chocolate (bh)	TYRP1	bh	0	No effect
Chocolate (bs) To show chocolate coloration a dog must inherit two chocolate variants, one from each parent. This can either be two copies of a particular variant, such as this one ("bs"), or two of any combination of chocolate variants.	TYRP1	bs	1	Black features likely, chocolate possible

## Coat Patterns

	Gene	Variant	Copies	Result
Piebald	MITF	sp	0	No effect
Merle	PMEL	M	0	No effect
Harlequin	PSMB7	H	0	No effect
Saddle Tan	RALY	-	0	No effect
Roan (Linkage test)	USH2A	Tr	0	No effect

## Coat Length and Curl

	Gene	Variant	Copies	Result
Long Hair (lh1)	FGF5	lh <sup>1</sup>	0	No effect
Long Hair (lh2)	FGF5	lh <sup>2</sup>	0	No effect
Long Hair (lh3)	FGF5	lh <sup>3</sup>	0	No effect
Long Hair (lh4)	FGF5	lh <sup>4</sup>	0	No effect

# Poker

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## Coat Length and Curl

	Gene	Variant	Copies	Result
Long Hair (lh5)	FGF5	lh <sup>5</sup>	0	No effect
Curly Coat	KRT71	C	0	No effect

## Hairlessness

	Gene	Variant	Copies	Result
Hairlessness (Discovered in the Chinese Crested Dog) Linkage test	FOXI3	H <sup>rcc</sup>	0	No effect
Hairlessness (Discovered in the American Hairless Terrier)	SGK3	h <sup>raht</sup>	0	No effect
Hairlessness (Discovered in the Scottish Deerhound)	SKG3	h <sup>rsd</sup>	0	No effect

## Shedding

	Gene	Variant	Copies	Result
Reduced Shedding  One or two copies of the Reduced Shedding variant is likely to reduce a dog's tendency to shed. Copies of the Furnishings variant, particularly two, also reduce the tendency of a dog to shed.	MC5R	sd	1	Occasional shedder

## More Coat Traits

	Gene	Variant	Copies	Result
Hair Ridge	FGF3, FGF4, FGF19, ORAOV1	R	0	No effect
Furnishings	RSPO2	F	0	No effect
Albino	SLC45A2	c <sup>al</sup>	0	No effect

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## Head Shape

	Gene	Variant	Copies	Result
Short Snout (BMP3 variant)	BMP3	-	0	No effect
Short Snout (SMOC2 variant)	SMOC2	-	0	No effect

## Eye Color

	Gene	Variant	Copies	Result
Blue Eyes (Discovered in the Siberian Husky)	ALX4	-	0	No effect

## Ears

	Gene	Variant	Copies	Result
<b>Floppy Ears</b> Dogs with zero copies of this variant are more likely to have permanently upright or prick ears, and fully folded ears are more likely with two copies inherited. Please note however that many genetic variants influence ear carriage. Dogs with some cartilage stiffness to their ears can sometimes raise their ears upright when 'at alert' but will flop down when relaxed.	MSRB3	-	2	Floppy ears more likely

## Extra Toes

	Gene	Variant	Copies	Result
Hind Dewclaws (Discovered in Asian breeds)	LMBR1	DC-1	0	No effect
Hind Dewclaws (Discovered in Western breeds)	LMBR1	DC-2	0	No effect

## More Body Features

	Gene	Variant	Copies	Result
Back Muscle and Bulk	ACSL4	-	0	No effect
High Altitude Adaptation	EPAS1	-	0	No effect

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## More Body Features

	Gene	Variant	Copies	Result
Short Legs (Chondrodysplasia, CDPA)	FGF4	-	0	No effect
Short Legs (Chondrodystrophy, CDDY)	FGF4	-	0	No effect
Short Tail	T-box	T	0	Full tail length likely



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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
2,8-dihydroxyadenine (DHA) Urolithiasis	APRT	G>A	0	AR	Clear
Acral Mutilation Syndrome	GDNF	C>T	0	AR	Clear
Acute Respiratory Distress Syndrome	ANLN	C>T	0	AR	Clear
Alaskan Husky Encephalopathy	SLC19A3	G>A	0	AR	Clear
Amelogenesis Imperfecta (Discovered in the Italian Greyhound)	ENAM	Deletion	0	AR	Clear
Amelogenesis Imperfecta (Discovered in the Lancashire Heeler)	Confidential	-	0	AR	Clear
Amelogenesis Imperfecta (Discovered in the Parson Russell Terrier)	ENAM	C>T	0	AR	Clear
Bandera's Neonatal Ataxia	GRM1	Insertion	0	AR	Clear
Benign Familial Juvenile Epilepsy	LGI2	A>T	0	AR	Clear
Bernard-Soulier Syndrome (Discovered in the Cocker Spaniel)	GP9	Deletion	0	AR	Clear
Canine Congenital Stationary Night Blindness (Discovered in the Beagle)	LRIT3	Deletion	0	AR	Clear
Canine Leukocyte Adhesion Deficiency (CLAD), type III	FERMT3	Insertion	0	AR	Clear
Canine Multifocal Retinopathy 1	BEST1	C>T	0	AR	Clear
Canine Multifocal Retinopathy 2	BEST1	G>A	0	AR	Clear
Canine Multifocal Retinopathy 3	BEST1	Deletion	0	AR	Clear
Canine Multiple Systems Degeneration (Discovered in the Chinese Crested Dog)	SERAC1	Deletion	0	AR	Clear
Canine Scott Syndrome	ANO6	G>A	0	AR	Clear
Cardiomyopathy and Juvenile Mortality (Discovered in the Belgian Shepherd)	YARS2	G>A	0	AR	Clear
Centronuclear Myopathy (Discovered in the Great Dane)	BIN1	A>G	0	AR	Clear
Cerebellar Ataxia	RAB24	A>C	0	AR	Clear

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Cerebellar Cortical Degeneration	SNX14	C>T	0	AR	Clear
Cerebellar Hypoplasia	VLDLR	Deletion	0	AR	Clear
Cerebral Dysfunction	SLC6A3	G>A	0	AR	Clear
Chondrodysplasia (Discovered in Norwegian Elkhound and Karelian Bear Dog)	ITGA10	C>T	0	AR	Clear
Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD) Risk	FGF4 retrogene	Insertion	0	AD	Clear
Cleft Lip & Palate with Syndactyly	ADAMTS20	Deletion	0	AR	Clear
Cleft Palate	DLX6	C>A	0	AR	Clear
CNS Atrophy with Cerebellar Ataxia (Discovered in the Belgian Shepherd)	SEPP1	Deletion	0	AR	Clear
Coat Color Dilution and Neurological Defects (Discovered in the Miniature Dachshund)	MYO5A	Insertion	0	AR	Clear
Collie Eye Anomaly (CEA)	NHEJ1	Deletion	0	AR	Clear
Complement 3 Deficiency	C3	Deletion	0	AR	Clear
Cone Degeneration (Discovered in the Alaskan Malamute)	CNGB3	Deletion	0	AR	Clear
Cone Degeneration (Discovered in the German Shepherd Dog)	CNGA3	C>T	0	AR	Clear
Cone Degeneration (Discovered in the German Shorthaired Pointer)	CNGB3	G>A	0	AR	Clear
Cone-Rod Dystrophy	NPHP4	Deletion	0	AR	Clear
Cone-Rod Dystrophy 1	PDE6B	Deletion	0	AR	Clear
Cone-Rod Dystrophy 2	IQCB1	Insertion	0	AR	Clear
Congenital Dyshormonogenic Hypothyroidism with Goiter (Discovered in the Shih Tzu)	SLC5A5	G>A	0	AR	Clear
Congenital Eye Malformations (Discovered in the Golden Retriever)	SIX6	C>T	0	AD	Clear

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<b>Congenital Hypothyroidism (Discovered in the Tenterfield Terrier)</b>	TPO	C>T	0	AR	Clear
<b>Congenital Hypothyroidism (Discovered in the Toy Fox and Rat Terrier)</b>	TPO	C>T	0	AR	Clear
<b>Congenital Muscular Dystrophy (Discovered in the Italian Greyhound)</b>	LAMA2	G>A	0	AR	Clear
<b>Congenital Muscular Dystrophy (Discovered in the Staffordshire Bull Terrier)</b>	LAMA2	Deletion	0	AR	Clear
<b>Congenital Myasthenic Syndrome (Discovered in the Golden Retriever)</b>	COLQ	G>A	0	AR	Clear
<b>Congenital Myasthenic Syndrome (Discovered in the Heideterrier)</b>	CHRNE	Insertion	0	AR	Clear
<b>Congenital Myasthenic Syndrome (Discovered in the Jack Russell Terrier)</b>	CHRNE	Insertion	0	AR	Clear
<b>Congenital Myasthenic Syndrome (Discovered in the Old Danish Pointer)</b>	CHAT	G>A	0	AR	Clear
<b>Congenital Stationary Night Blindness (CSNB)</b>	RPE65	A>T	0	AR	Clear
<b>Craniomandibular Osteopathy (Discovered in Scottish Terrier breeds)</b>	SLC37A2	C>T	0	AD	Clear
<b>Craniomandibular Osteopathy (Discovered in the Australian Terrier)</b>	COL1A1	C>T	0	AD	Clear
<b>Craniomandibular Osteopathy (Discovered in the Basset Hound)</b>	SLC37A2	C>T	0	AD	Clear
<b>Craniomandibular Osteopathy (Discovered in the Weimaraner)</b>	SLC35D1	Deletion	0	AD	Clear
<b>Cystic Renal Dysplasia and Hepatic Fibrosis</b>	INPP5E	G>A	0	AR	Clear
<b>Cystinuria Type I-A</b>	SLC3A1	C>T	0	AR	Clear
<b>Cystinuria Type II-A</b>	SLC3A1	Deletion	0	AD	Clear
<b>Darier Disease (Discovered in the Irish Terrier)</b>	ATP2A2	Insertion	0	AD	Clear
<b>Deafness and Vestibular Dysfunction (DINGS1), (Discovered in Doberman Pinscher)</b>	PTPRQ	Insertion	0	AR	Clear

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Deafness and Vestibular Dysfunction (DINGS2), (Discovered in Doberman Pinscher)	MYO7A	G>A	0	AR	Clear
Degenerative Myelopathy	SOD1	G>A	0	AR	Clear
Demyelinating Neuropathy	SBF2	G>T	0	AR	Clear
Dental Hypomineralization	FAM20C	C>T	0	AR	Clear
Dental-Skeletal-Retinal Anomaly (Discovered in the Cane Corso)	MIA3	Deletion	0	AR	Clear
Dilated Cardiomyopathy (Discovered in the Schnauzer)	RBM20	Deletion	0	AR	Clear
Disproportionate Dwarfism (Discovered in the Dogo Argentino)	PRKG2	C>A	0	AR	Clear
Dominant Progressive Retinal Atrophy	RHO	C>G	0	AD	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Basset Hound)	COL7A1	Insertion	0	AR	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Central Asian Ovcharka)	COL7A1	C>T	0	AR	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Golden Retriever)	COL7A1	C>T	0	AR	Clear
Early Adult Onset Deafness For Border Collies only (Linkage test)	Intergenic	Insertion	0	AR	Clear
Early Retinal Degeneration (Discovered in the Norwegian Elkhound)	STK38L	Insertion	0	AR	Clear
Early-Onset Adult Deafness (Discovered in the Rhodesian Ridgeback)	EPS8L2	Deletion	0	AR	Clear
Early-Onset Progressive Polyneuropathy (Discovered in the Alaskan Malamute)	NDRG1	G>T	0	AR	Clear
Early-Onset Progressive Polyneuropathy (Discovered in the Greyhound)	NDRG1	Deletion	0	AR	Clear
Early-Onset Progressive Retinal Atrophy (Discovered in the Portuguese Water Dog)	CCDC66	Insertion	0	AR	Clear
Early-Onset Progressive Retinal Atrophy, (Discovered in the Spanish Water Dog)	PDE6B	Deletion	0	AR	Clear

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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Ehlers-Danlos Syndrome (Discovered in mixed breed)	COL5A1	G>A	0	AD	Clear
Epidermolytic Hyperkeratosis	KRT10	G>T	0	AR	Clear
Episodic Falling Syndrome	BCAN	Insertion	0	AR	Clear
Factor VII Deficiency	F7	G>A	0	AR	Clear
Factor XI Deficiency	FXI	Insertion	0	AD	Clear
Familial Nephropathy (Discovered in the English Cocker Spaniel)	COL4A4	A>T	0	AR	Clear
Familial Nephropathy (Discovered in the English Springer Spaniel)	COL4A4	C>T	0	AR	Clear
Fanconi Syndrome	FAN1	Deletion	0	AR	Clear
Fetal Onset Neuroaxonal Dystrophy	MFN2	G>C	0	AR	Clear
Focal Non-Epidermolytic Palmoplantar Keratoderma	KRT16	G>C	0	AR	Clear
Generalized Progressive Retinal Atrophy (Discovered in the Schapendoes)	CCDC66	Insertion	0	AR	Clear
Glanzmann Thrombasthenia Type I (Discovered in Great Pyrenees)	ITGA2B	C>G	0	AR	Clear
Glanzmann Thrombasthenia Type I (Discovered in mixed breed dogs)	ITGA2B	C>T	0	AR	Clear
Globoid Cell Leukodystrophy (Discovered in Terriers)	GALC	A>C	0	AR	Clear
Globoid Cell Leukodystrophy (Discovered in the Irish Setter)	GALC	A>T	0	AR	Clear
Glycogen Storage Disease Type Ia (Discovered in the German Pinscher)	G6PC	Insertion	0	AR	Clear
Glycogen Storage Disease Type Ia (Discovered in the Maltese)	G6PC	G>C	0	AR	Clear
Glycogen Storage Disease Type IIIa, (GSD IIIa)	AGL	Deletion	0	AR	Clear
GM1 Gangliosidosis (Discovered in the Portuguese Water Dog)	GLB1	G>A	0	AR	Clear

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## Other health conditions tested

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GM1 Gangliosidosis (Discovered in the Shiba)	GLB1	Deletion	0	AR	Clear
GM2 Gangliosidosis (Discovered in the Japanese Chin)	HEXA	G>A	0	AR	Clear
GM2 Gangliosidosis (Discovered in the Toy Poodle)	HEXB	Deletion	0	AR	Clear
Hemophilia A (Discovered in Old English Sheepdog)	FVIII	C>T	0	XR	Clear
Hemophilia A (Discovered in the Boxer)	FVIII	C>G	0	XR	Clear
Hemophilia A (Discovered in the German Shepherd Dog - Variant 1)	FVIII	G>A	0	XR	Clear
Hemophilia A (Discovered in the German Shepherd Dog - Variant 2)	FVIII	G>A	0	XR	Clear
Hemophilia A (Discovered in the Havanese)	FVIII	Insertion	0	XR	Clear
Hemophilia B	FIX	G>A	0	XR	Clear
Hemophilia B (Discovered in the Airedale Terrier)	FIX	Insertion	0	XR	Clear
Hemophilia B (Discovered in the Lhasa Apso)	FIX	Deletion	0	XR	Clear
Hereditary Ataxia (Discovered in the Belgian Malinois)	SLC12A6	Insertion	0	AR	Clear
Hereditary Ataxia (Discovered in the Norwegian Buhund)	KCNIP4	T>C	0	AR	Clear
Hereditary Calcium Oxalate Urolithiasis, Type 1	Confidential	-	0	AR	Clear
Hereditary Footpad Hyperkeratosis	FAM83G	G>C	0	AR	Clear
Hereditary Nasal Parakeratosis (Discovered in the Greyhound)	SUV39H2	Deletion	0	AR	Clear
Hereditary Vitamin D-Resistant Rickets Type II	VDR	Deletion	0	AR	Clear
Hypocatalasia	CAT	G>A	0	AR	Clear
Hypomyelination	FNIP2	Deletion	0	AR	Clear
Hypophosphatasia	Confidential	-	0	AR	Clear
Ichthyosis (Discovered in the American Bulldog)	NIPAL4	Deletion	0	AR	Clear

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Ichthyosis (Discovered in the Great Dane)	SLC27A4	G>A	0	AR	Clear
Ichthyosis Type 2 (Discovered in the Golden Retriever)	ABHD5	Deletion	0	AR	Clear
Inflammatory Myopathy (Discovered in the Dutch Shepherd Dog)	SLC25A12	A>G	0	AR	Clear
Inflammatory Pulmonary Disease (Discovered in the Rough Collie)	AKNA	Deletion	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Beagle)	CUBN	Deletion	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)	CUBN	Deletion	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Komondor)	CUBN	G>A	0	AR	Clear
Intestinal Lipid Malabsorption (Discovered in the Australian Kelpie)	ACSL5	Deletion	0	AR	Clear
Junctional Epidermolysis Bullosa (Discovered in the Australian Cattle Dog Mix)	LAMA3	T>A	0	AR	Clear
Junctional Epidermolysis Bullosa (Discovered in the Australian Shepherd)	LAMB3	A>G	0	AR	Clear
Juvenile Cataract (Discovered in the Wirehaired Pointing Griffon)	FYCO1	Deletion	0	AR	Clear
Juvenile Dilated Cardiomyopathy (Discovered in the Toy Manchester Terrier)	ABCC9	G>A	0	AR	Clear
Juvenile Encephalopathy (Discovered in the Parson Russell Terrier)	Confidential	-	0	AR	Clear
Juvenile Laryngeal Paralysis and Polyneuropathy	RAB3GAP1	Deletion	0	AR	Clear
Juvenile Myoclonic Epilepsy	DIRAS1	Deletion	0	AR	Clear
L-2-Hydroxyglutaric aciduria (Discovered in the Staffordshire Bull Terrier)	L2HGDH	T>C	0	AR	Clear
L-2-Hydroxyglutaric Aciduria (Discovered in the West Highland White Terrier)	Confidential	-	0	AR	Clear
Lafora Disease (Linkage test)	NHLRC1	Insertion	0	AR	Clear

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Lagotto Storage Disease	ATG4D	G>A	0	AR	Clear
Lamellar Ichthyosis	TGM1	Insertion	0	AR	Clear
Laryngeal Paralysis (Discovered in the Bull Terrier and Miniature Bull Terrier)	RAPGEF6	Insertion	0	AR	Clear
Leigh-like Subacute Necrotizing Encephalopathy (Discovered in the Yorkshire Terrier)	SLC19A3	Insertion	0	AR	Clear
Lethal Acrodermatitis (Discovered in the Bull Terrier)	MKLN1	A>C	0	AR	Clear
Leukodystrophy (Discovered in the Standard Schnauzer)	TSEN54	C>T	0	AR	Clear
Ligneous Membranitis	PLG	T>A	0	AR	Clear
Limb-girdle Muscular Dystrophy (Discovered in the Boston Terrier)	SGCD	Deletion	0	AR	Clear
Limb-girdle Muscular Dystrophy, Type L3 (Discovered in the Miniature Dachshund)	SGCA	G>A	0	AR	Clear
Lung Developmental Disease (Discovered in the Airedale Terrier)	LAMP3	C>T	0	AR	Clear
Macrothrombocytopenia (Discovered in Norfolk and Cairn Terrier)	TUBB1	G>A	0	AR	Clear
May-Hegglin Anomaly	MYH9	G>A	0	AD	Clear
MDR1 Medication Sensitivity	MDR1/ABCB1	Deletion	0	AD	Clear
Microphthalmia (Discovered in the Soft-Coated Wheaten Terrier)	RBP4	Deletion	0	AR	Clear
Mucopolysaccharidosis Type IIIA (Discovered in the Dachshund)	SGSH	C>A	0	AR	Clear
Mucopolysaccharidosis Type IIIA (Discovered in the New Zealand Huntaway)	SGSH	Insertion	0	AR	Clear
Mucopolysaccharidosis Type VII (Discovered in the Brazilian Terrier)	GUSB	C>T	0	AR	Clear
Mucopolysaccharidosis Type VII (Discovered in the German Shepherd Dog)	GUSB	G>A	0	AR	Clear



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Mucopolysaccharidosis VI (Discovered in the Miniature Pinscher)	ARSB	G>A	0	AR	Clear
Muscular Dystrophy (Discovered in the Cavalier King Charles Spaniel)	Dystrophin	G>T	0	XR	Clear
Muscular Dystrophy (Discovered in the Golden Retriever)	Dystrophin	A>G	0	XR	Clear
Muscular Dystrophy (Discovered in the Landseer)	COL6A1	G>T	0	AR	Clear
Muscular Dystrophy (Discovered in the Norfolk Terrier)	Dystrophin	Deletion	0	XR	Clear
Muscular Hypertrophy (Double Muscling)	MSTN	T>A	0	AR	Clear
Musladin-Lueke Syndrome	ADAMTSL2	C>T	0	AR	Clear
Myeloperoxidase Deficiency	MOP	C>T	0	AR	Clear
Myotonia Congenita (Discovered in Australian Cattle Dog)	CLCN1	Insertion	0	AR	Clear
Myotonia Congenita (Discovered in the Miniature Schnauzer)	CLCN1	C>T	0	AR	Clear
Myotubular Myopathy	MTM1	A>C	0	XR	Clear
Narcolepsy (Discovered in the Dachshund)	HCRT2	G>A	0	AR	Clear
Nemaline Myopathy	NEB	C>A	0	AR	Clear
Neonatal Cerebellar Cortical Degeneration	SPTBN2	Deletion	0	AR	Clear
Neonatal Encephalopathy with Seizures	ATF2	T>G	0	AR	Clear
Neuroaxonal Dystrophy (Discovered in Spanish Water Dog)	TECPR2	C>T	0	AR	Clear
Neuroaxonal Dystrophy (Discovered in the Papillon)	PLA2G6	G>A	0	AR	Clear
Neuroaxonal Dystrophy (Discovered in the Rottweiler)	VPS11	A>G	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 1	PPT1	Insertion	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 12 (Discovered in the Australian Cattle Dog)	ATP13A2	C>T	0	AR	Clear

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Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie)	CLN5	C>T	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Golden Retriever)	CLN5	Deletion	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 7	MFSD8	Deletion	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Alpine Dachsbracke)	CLN8	Deletion	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Australian Shepherd)	CLN8	G>A	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the English Setter)	CLN8	T>C	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Saluki)	CLN8	Insertion	0	AR	Clear
Osteochondrodysplasia	SLC13A1	Deletion	0	AR	Clear
Osteochondromatosis (Discovered in the American Staffordshire Terrier)	EXT2	C>A	0	AR	Clear
Osteogenesis Imperfecta (Discovered in the Beagle)	COL1A2	C>T	0	AD	Clear
Osteogenesis Imperfecta (Discovered in the Dachshund)	SERPINH1	T>C	0	AR	Clear
P2RY12-associated Bleeding Disorder	P2RY12	Deletion	0	AR	Clear
Palmoplantar Hyperkeratosis (Discovered in the Rottweiler)	DSG1	Deletion	0	AR	Clear
Paroxysmal Dyskinesia	PIGN	C>T	0	AR	Clear
Persistent Müllerian Duct Syndrome	AMHR2	C>T	0	AR	Clear
Phosphofructokinase Deficiency	PFKM	G>A	0	AR	Clear
Pituitary Dwarfism (Discovered in the Karelian Bear Dog)	POU1F1	C>A	0	AR	Clear
Polycystic Kidney Disease	PKD1	G>A	0	AD	Clear
Prekallikrein Deficiency	KLKB1	T>A	0	AR	Clear

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Primary Ciliary Dyskinesia	CCDC39	C>T	0	AR	Clear
Primary Ciliary Dyskinesia (Discovered in the Alaskan Malamute)	NME5	Deletion	0	AR	Clear
Primary Lens Luxation	ADAMTS17	G>A	0	AR	Clear
Primary Open Angle Glaucoma (Discovered in Basset Fauve de Bretagne)	ADAMTS17	G>A	0	AR	Clear
Primary Open Angle Glaucoma (Discovered in Petit Basset Griffon Vendeen)	ADAMTS17	Insertion	0	AR	Clear
Primary Open Angle Glaucoma and Lens Luxation (Discovered in Chinese Shar-Pei)	ADAMTS17	Deletion	0	AR	Clear
Progressive Early-Onset Cerebellar Ataxia	SEL1L	T>C	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Basenji)	SAG	T>C	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA 2 variant)	TTC8	Deletion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA1 variant)	SLC4A3	Insertion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Lapponian Herder)	IFT122	C>T	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Lhasa Apso)	IMPG2	Insertion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Miniature Long Haired Dachshund)	RPGRIP1	Insertion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Papillon and Phalène)	CNGB1	Deletion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - BBS2 variant)	Confidential	-	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - CNGA1 variant)	CNGA1	Deletion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Swedish Vallhund)	MERTK	Insertion	0	AR	Clear

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Progressive Retinal Atrophy 1 (Discovered in the Italian Greyhound)	Confidential	-	0	AR	Clear
Progressive Retinal Atrophy Type III	FAM161A	Insertion	0	AR	Clear
Protein Losing Nephropathy	NPHS1	G>A	0	AR	Clear
Pyruvate Dehydrogenase Phosphatase 1 Deficiency	PDP1	C>T	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the Basenji)	PKLR	Deletion	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the Beagle)	PKLR	G>A	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the Pug)	PKLR	T>C	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the West Highland White Terrier)	PKLR	Insertion	0	AR	Clear
QT Syndrome	KCNQ1	C>A	0	AD	Clear
Renal Cystadenocarcinoma and Nodular Dermatofibrosis	FLCN	A>G	0	AD	Clear
Rod-Cone Dysplasia 1	PDE6B	G>A	0	AR	Clear
Rod-Cone Dysplasia 1a	PDE6B	Insertion	0	AR	Clear
Rod-Cone Dysplasia 3	PDE6A	Deletion	0	AR	Clear
Sensorineural Deafness (Discovered in the Rottweiler)	LOXHD1	G>C	0	AR	Clear
Sensory Ataxic Neuropathy	tRNATyr	Deletion	0	MT	Clear
Sensory Neuropathy	FAM134B	Insertion	0	AR	Clear
Severe Combined Immunodeficiency (Discovered in Frisian Water Dogs)	RAG1	G>T	0	AR	Clear
Severe Combined Immunodeficiency (Discovered in Russell Terriers)	PRKDC	G>T	0	AR	Clear
Shaking Puppy Syndrome (Discovered in the Border Terrier)	Confidential	-	0	AR	Clear
Spinocerebellar Ataxia (Late-Onset Ataxia)	CAPN1	G>A	0	AR	Clear

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Spinocerebellar Ataxia with Myokymia and/or Seizures	KCNJ10	C>G	0	AR	Clear
Spondylocostal Dysostosis	HES7	Deletion	0	AR	Clear
Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA1)	KCNJ10	T>C	0	AR	Clear
Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA2)	ATP1B2	Insertion	0	AR	Clear
Startle Disease (Discovered in Irish Wolfhounds)	SLC6A5	G>T	0	AR	Clear
Startle Disease (Discovered in the Miniature American Shepherd)	Confidential	-	0	AR	Clear
Succinic Semialdehyde Dehydrogenase Deficiency (Discovered in the Saluki)	ALDH5A1	G>A	0	AR	Clear
Thrombopathia (Discovered in the Basset Hound)	RASGRP1	Deletion	0	AR	Clear
Thrombopathia (Discovered in the Eskimo Spitz)	RASGRP1	Insertion	0	AR	Clear
Trapped Neutrophil Syndrome	VPS13B	Deletion	0	AR	Clear
Van den Ende-Gupta Syndrome	SCARF2	Deletion	0	AR	Clear
von Willebrand's Disease, type 1	VWF	G>A	0	AD	Clear
von Willebrand's Disease, type 2	VWF	T>G	0	AR	Clear
von Willebrand's Disease, type 3 (Discovered in the Kooiker Hound)	VWF	G>A	0	AR	Clear
von Willebrand's Disease, type 3 (Discovered in the Scottish Terrier)	VWF	Deletion	0	AR	Clear
von Willebrand's Disease, type 3 (Discovered in the Shetland Sheepdog)	VWF	Deletion	0	AR	Clear
X-Linked Ectodermal Dysplasia	EDA	G>A	0	XR	Clear
X-Linked Hereditary Nephropathy (Discovered in the Navasota Dog)	COL4A5	Deletion	0	XR	Clear
X-Linked Hereditary Nephropathy (Discovered in the Samoyed)	COL4A5	G>T	0	XR	Clear

# Poker

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Registration number: X  
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## Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
X-Linked Progressive Retinal Atrophy 1	RPGR	Deletion	0	XR	Clear
X-Linked Progressive Retinal Atrophy 2	RPGR	Deletion	0	XR	Clear
X-Linked Severe Combined Immunodeficiency (Discovered in the Basset Hound)	IL2RG	Deletion	0	XR	Clear
X-Linked Severe Combined Immunodeficiency (Discovered in the Cardigan Welsh Corgi)	IL2RG	Insertion	0	XR	Clear
X-Linked Tremors	PLP1	A>C	0	XR	Clear
Xanthinuria (Discovered in a mixed breed dog)	Confidential	-	0	AR	Clear
Xanthinuria (Discovered in the Cavalier King Charles Spaniel)	Confidential	-	0	AR	Clear
Xanthinuria (Discovered in the Toy Manchester Terrier)	Confidential	-	0	AR	Clear

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## Glossary of genetic terms

### Test result definitions

**At Risk:** Based on the disorder's mode of inheritance, the dog inherited a number of genetic variant(s) which increases the dog's risk of being diagnosed with the associated disorder.

**Carrier:** The dog inherited one copy of a genetic variant when two copies are usually necessary to increase the dog's risk of being diagnosed with the associated disorder. While carriers are usually not at risk of clinical expression of the disorder, carriers of some complex variants may be associated with a low risk of developing the disorder.

**Clear:** The dog did not inherit the genetic variant(s) associated with the disorder and will not be at elevated risk of being diagnosed with the disorder due to this genotype. However, similar clinical signs could develop from different genetic or clinical causes.

**Inconclusive:** An inconclusive result indicates a confident call could not be made based on the data for that genetic variant. Health testing is performed in replicates, and on occasion the outcomes do not agree. This may occur due to an unusual sequence of DNA in the region tested, multiple cell genotypes present due to chimerism or acquired mutations, or due to quality of the DNA sample.

### Inheritance mode definitions

**Autosomal Recessive (AR):** For autosomal recessive disorders, dogs with two copies of the genetic variant are at risk of developing the associated disorder. Dogs with one copy of the variant are considered carriers and are usually not at risk of developing the disorder. However, carriers of some complex variants grouped in this category may be associated with a low risk of developing the disorder. Dogs with one or two copies may pass the disorder-associated variant to their puppies if bred.

**Autosomal Dominant (AD):** For autosomal dominant disorders, dogs with one or two copies of the genetic variant are at risk of developing the associated disorder. Inheriting two copies of the variant may increase the risk of development of the disorder or cause the condition to be more severe. These dogs may pass the disorder-associated variant to their puppies if bred.

**X-linked Recessive (XR):** For X-linked recessive disorders, the genetic variant is found on the X chromosome. Female dogs must inherit two copies of the variant to be at risk of developing the condition, whereas male dogs only need one copy to be at risk. Males and females with any copies of the variant may pass the disorder-associated variant to their puppies if bred.

**X-linked Dominant (XD):** For X-linked dominant disorders, the genetic variant is found on the X chromosome. Both male and female dogs with one copy of the variant are at risk of developing the disorder. Females inheriting two copies of the variant may be at higher risk or show a more severe form of the disorder than with one copy. Males and females with any copies of the variant may pass the disorder-associated variant to their puppies if bred.

**Mitochondrial (MT):** Unlike the two copies of genomic DNA held in the nucleus, there are thousands of mitochondria in each cell of the body, and each holds its own mitochondrial DNA (mtDNA). Mitochondria are called the "powerhouses" of the cell. For a dog to be at risk for a mitochondrial disorder, it must inherit a certain ratio of mtDNA with the associated variant compared to normal mtDNA. mtDNA is inherited only from the mother.