HEARTS FULL OF PUPPIES

Poppy

Health Testing

Sex: Female

Breed: Cavapoo

Adult weight: Approx. 18 lb



Included documents

Genetics at a glance

OFA cardiac, eyes, patella

Complete DNA report

(28 pages)

DNA report name: Poppi

Original certificates and laboratory report follow.

from our hearts to yours forever.

Genetics at a glance

Poppy (DNA report: Poppi) | Wisdom Panel | July 28, 2025

Furnishings	F/f	1 RSPO2 furnishings copy
Curl	C/c	1 KRT71 curl copy
Coat length	lh1/lh1	2 FGF5 long-hair copies
Recessive red	e1/e1 (e/e)	2 MC1R recessive-red copies
Tan points	at/at	2 ASIP tan-point copies
Dominant black	1 KB copy	Second K allele not specified
Piebald / parti	1 sp copy	Carries tested piebald variant
Chocolate	0 copies	basd, bc, bd, be, bh, bs tested
Dilution	0 copies	d1, d2, d3 variants tested
Merle	0 M copies	PMEL merle variant tested
Reduced shedding	sd/sd	2 MC5R reduced-shedding copies
Red intensity	0 i copies	MFSD12 variant tested

Genetic health

CDDY / IVDD: 1 copy of the CDDY variant.

Other health results: 271 clear; 0 carrier results.

CDDY is widespread in Cavaliers and common in Toy and Miniature Poodles. One copy increases the chance of early spinal-disc degeneration and Type I IVDD, but does not mean Poppy has a herniated disc or will develop one. This is a genetic risk factor, not a clinical diagnosis.

Trait shorthand describes tested variants; K-locus second allele is unspecified.

Sources: Cornell - CDDY/IVDD | UC Davis VGL - Managing CDDY (click to read)

ORTHOPEDIC FOUNDATION FOR ANIMALS, INC.

POPPY
registered name

HYBRID
breed

25UVGU
film/test/lab #

tattoo/microchip/DNA profile

2648831
application number

08/11/2025
date of report

NOREG2648831
registration no.

F
sex

05/06/2024
date of birth

14
age at evaluation in months

A Not-For-Profit Organization

HY-BCA8520/14F/P-NOPI
O.F.A. NUMBER

This number issued with the right to correct or revoke by the Orthopedic Foundation for Animals.

RESULTS:

Normal cardiovascular examination via auscultation - No evidence of congenital or acquired heart disease was noted. Since acquired heart disease may develop later, these evaluation results remain valid for one year, and annual examinations are recommended to continue to monitor cardiac health.

NORMAL/CLEAR - PRACTITIONER

owner

BONNIE SMITH
ROBERT SMITH
65 EAST 200 NORTH STREET; P.O.
MONA UT 84645

OFA eCert

Verify QR scan

G.G. KELLER, DVM, MS, DACVR
CHIEF OF VETERINARY SERVICES

www.ofa.org

This electronic OFA certificate was generated on: 08/11/2025

This certification can be verified on the OFA website by entering the dog's registration number into the orange search box located at the top of the page or by scanning the QR code above.

If there are any errors on this certificate, please email CORRECTIONS@OFA.ORG to request a correction.

Orthopedic Foundation for Animals, Inc.
2300 E. Nifong Blvd.
Columbia, MO 65201-3806

OFA website: www.ofa.org
E-mail address: ofa@ofa.org
Phone number: 573-442-0418
Fax number: 573-875-5073

ORTHOPEDIC FOUNDATION FOR ANIMALS, INC.

POPPY
registered name

NOREG2648831
registration no.

HYBRID
breed

F
sex

26DRQ4
film/test/lab #

05/06/2024
date of birth

28
age at evaluation in months

A Not-For-Profit Organization

tattoo/microchip/DNA profile

2648831
application number

10/12/2026
date of report

HY-EYE13682/28F-NOPI
O.F.A. NUMBER

This number issued with the right to correct or revoke by the Orthopedic Foundation for Animals.

RESULTS:

Based upon the exam dated 10/05/2026, this animal has been found to be free of observable inherited eye disease and has been issued an Eye Certification Registry Number which is valid for one year from the time of the exam.

NORMAL

owner

ROBERT SMITH
BONNIE SMITH
65 EAST 200 NORTH STREET; P.O.
MONA UT 84645

OFA eCert

Verify QR scan

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POPPY
registered name

HYBRID
breed

25UVGU
film/test/lab #

tattoo/microchip/DNA profile

2648831
application number

08/11/2025
date of report

NOREG2648831
registration no.

F
sex

05/06/2024
date of birth

14
age at evaluation in months

A Not-For-Profit Organization

HY-PA13089/14F/P-NOPI
O.F.A. NUMBER

This number issued with the right to correct or revoke by the Orthopedic Foundation for Animals.

RESULTS:

The results of the examination submitted to OFA indicate that no evidence of patellar luxation was recognized.

NORMAL - PRACTITIONER

owner
BONNIE SMITH
ROBERT SMITH
65 EAST 200 NORTH STREET; P.O.
MONA UT 84645

OFA eCert

Verify QR scan

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Phone number: 573-442-0418
Fax number: 573-875-5073

Poppi

Optimal Selection™ powered by WISDOM PANEL™

Breed: Cavapoo
Birth date: 2024-01

Test date: 2025-07-28
ID kit: DSNBMVX

Poppi's Profile

Pet information

Registered name
Poppi

Sex
F

Owner reported breed
Cavapoo

Date of birth
2024-01

Genetic Diversity

Poppi's Percentage of Heterozygosity
42%

Health summary

At Risk 1 condition
- Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD) Risk

Carrier 0 conditions

Clear 271 conditions

Poppi

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Genetic Diversity

Heterozygosity

Poppi's Percentage of Heterozygosity

42%

Poppi's genome analysis shows an average level of genetic heterozygosity when compared with other mixed-breed dogs.

Typical Range for Mixed-Breed Dogs

33% - 46%

Poppi

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Breed: Cavapoo
Birth date: 2024-01

Test date: 2025-07-28
ID kit: DSNBMVX

Health conditions known in the breed

Degenerative Myelopathy

Gene	Risk Variant	Copies	Inheritance	Result
SOD1	G>A	0	AR	**Clear**

Information about the genetic condition

The onset of DM is usually in a dog's senior years. Affected dogs first begin by exhibiting muscle wasting, proprioceptive deficits, and knuckling of the hind feet. Though the condition is not painful, affected dogs will eventually require assistance walking. As the condition progresses, it moves up the spinal cord and the dog's neurologic deficits mirror the progress, losing fecal and urinary continence and eventually involving the front legs and the brainstem.

Breeder recommendation

Please note the DM mutation has only been associated with disease signs in some breeds, and this should be considered when making breeding decisions. 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 DM mutation can be safely bred with a clear dog with no copies of the DM mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the DM mutation. A dog with two copies of the DM mutation can be safely bred with a clear dog. The resulting puppies will be all carriers with one copy of the DM mutation. Puppies in litters which are 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 DM mutation could develop due to a different genetic or clinical cause.

Episodic Falling Syndrome

Gene	Risk Variant	Copies	Inheritance	Result
BCAN	Insertion	0	AR	**Clear**

Information about the genetic condition

First signs of EFS can be seen in dogs 3 to 7 months of age. Episodic collapses are often associated with exercise, excitement, or stressful events, usually last a few seconds to several minutes, and resolve on their own. Episodic collapses begin with increased muscle tone leading to temporary immobility of the dog. The affected dog usually falls down with its limbs in an extended position. During an episode, an affected dog may exhibit bunny hop-like movements, have its back arched, and vocalize. The dog experiencing an episode stays fully conscious throughout. The severity and number of episodes vary over the course of the dog's life. Affected dogs appear neurologically normal between episodes.

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 EFS mutation can be safely bred with a clear dog with no copies of the EFS mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the EFS mutation. A dog with two copies of the EFS 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 EFS mutation could develop due to a different genetic or clinical cause.

Poppi

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Test date: 2025-07-28
ID kit: DSNBMVX

Health conditions known in the breed

GM2 Gangliosidosis (Discovered in the Toy Poodle)

Gene	Risk Variant	Copies	Inheritance	Result
HEXB	Deletion	0	AR	**Clear**

Information about the genetic condition

First signs of GM2 gangliosidosis are usually observed at one year of age. Characteristic signs include ataxia (uncoordinated movements), intention tremors of the head, vision impairment, altered mental status, and feeding difficulties. GM2 gangliosidosis is a progressive condition, so affected dogs will have increasing difficultly with everyday tasks such as walking and going up stairs, and may need assistance. Affected dogs are usually euthanized on welfare grounds by two years of age.

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 GM2 Gangliosidosis mutation can be safely bred with a clear dog with no copies of the GM2 Gangliosidosis mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the GM2 Gangliosidosis 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 GM2 Gangliosidosis 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.

Poppi

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Birth date: 2024-01

Test date: 2025-07-28
ID kit: DSNBMVX

Health conditions known in the breed

Muscular Dystrophy (Discovered in the Cavalier King Charles Spaniel)

Gene	Risk Variant	Copies	Inheritance	Result
Dystrophin	G>T	0	XR	**Clear**

Information about the genetic condition

Duchenne muscular dystrophy results from dysfunction of the sarcolemma (cell membrane of muscle fiber cells) which causes degeneration and necrosis of muscle tissue. It is a progressive condition that eventually leads to muscle fibrosis (formation of excess fibrous connective tissue). First signs of disease, such as bunny-hopping gait, can be observed in eight to ten weeks old puppies. Affected puppies have a thick tongue base and are unable to open the mouth properly which causes eating difficulties and excess drooling. Duchenne muscular dystrophy is characterized by crouched posture caused by spinal curvature and bending of the back. Serum creatine kinase concentrations can be over 300 times higher than normal levels.

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. 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.

Neonatal Encephalopathy with Seizures

Gene	Risk Variant	Copies	Inheritance	Result
ATF2	T>G	0	AR	**Clear**

Information about the genetic condition

Puppies suffering from NEWS are smaller and weaker at birth than their littermates and they often have difficulties nursing. Most of the affected puppies won't make it through the first week of their life. Puppies that survive past the first week suffer from ataxia (uncoordinated movements), whole-body tremor and weakness progressing to severe generalized clonic-tonic seizures by 4 to 6 weeks of age. No affected puppies have survived past 7 weeks of age and they are often euthanized earlier.

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 NEWS mutation can be safely bred with a clear dog with no copies of the NEWS mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the NEWS 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 NEWS mutation could develop due to a different genetic or clinical cause.

Poppi

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Breed: Cavapoo
Birth date: 2024-01

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ID kit: DSNBMVX

Health conditions known in the breed

Osteochondrodysplasia	Gene	Risk Variant	Copies	Inheritance	Result
	SLC13A1	Deletion	0	AR	**Clear**

Information about the genetic condition

The signs of osteochondrodysplasia can typically be observed in puppies as young as 3 weeks of age. The clinical signs of osteochondrodysplasia in Miniature Poodles include a flattened ribcage, deformed paws, abducted hind limbs, enlarged joints, and an underbite. Long bones of the limbs are shortened and bent. Affected dogs are smaller in size compared to their unaffected littermates. Affected dogs can live for several years, but they often suffer from arthritis caused by misshapen limbs. Abnormal structure of the ribcage can cause breathing difficulties.

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 Osteochondrodysplasia mutation can be safely bred with a clear dog with no copies of the Osteochondrodysplasia mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Osteochondrodysplasia 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 Osteochondrodysplasia mutation could develop due to a different genetic or clinical cause.

Progressive Rod Cone Degeneration (prcd-PRA)	Gene	Risk Variant	Copies	Inheritance	Result
	PRCD	G>A	0	AR	**Clear**

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.

Poppi

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Breed: Cavapoo
Birth date: 2024-01

Test date: 2025-07-28
ID kit: DSNBMVX

Health conditions known in the breed

von Willebrand's Disease, type 1

Gene	Risk Variant	Copies	Inheritance	Result
VWF	G>A	0	AD	**Clear**

Information about the genetic condition

Von Willebrand's Disease Type 1 is the most common bleeding disorder in dogs and is considered the mildest form of vWD. Because clotting ability corresponds to the level of von Willebrand's factor present, severity of clinical signs in affected dogs vary widely. Dogs with this genetic variant may appear asymptomatic, display only mild signs, or show frequent and severe signs of abnormal clotting. Age of onset can vary with some affected dogs displaying signs later in life. Additionally, illness, estrus, or pregnancy may exacerbate clinical signs in affected dogs. Clinical signs may include bruising easily, bleeding after losing baby teeth or chewing on toys, excessive bleeding from trauma, injury or surgery, nosebleeds and other forms of spontaneous bleeding. Please note that subclinical cases can also be associated with increased bleeding after surgery or trauma. An affected dog will have a normal PT/aPTT but may demonstrate prolonged bleeding during functional testing, such as buccal mucosal bleed time (BMBT). However, performing von Willebrand's factor testing at a reference laboratory is considered necessary to confirm diagnosis and to determine the concentration of vW factor present within an individual

Breeder recommendation

This condition is considered autosomal dominant with incomplete penetrance, meaning not all dogs with one copy will show clinical signs and dogs with two copies are considered at highest risk for being diagnosed with vWD. Diagnosing vWD is performed through reference laboratory testing of the dog's blood levels of von Willebrand factor. A dog with the vWD Type 1 genetic variant with normal blood levels of von Willebrand factor as tested by a reference laboratory can be safely bred with a clear dog with no copies of the vWD Type 1 variant. Puppies in a litter expected to contain carriers should be screened before breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note that the estrus cycle and pregnancy can lead to further fluctuation in the von Willebrand factor in females which may exacerbate clinical signs. Additionally, it is possible that disease signs similar to the ones caused by the vWD Type 1 variant could develop due to a different genetic or clinical cause.

Xanthinuria (Discovered in the Cavalier King Charles Spaniel)

Gene	Risk Variant	Copies	Inheritance	Result
Confidential	-	0	AR	**Clear**

Information about the genetic condition

This disorder can cause formation of stones throughout the upper and lower urinary tracts, including the urethra, bladder, ureters, and kidneys. Clinical signs of urolithiasis in the lower urinary tract include pain while urinating, bloody urine, and blockage of the urinary tract. Patients with urinary stones are more susceptible to urinary tract infections. Blockage of the ureters and kidneys causes abdominal pain and kidney failure. A blocked urinary tract is a life-threatening condition that requires immediate veterinary care. The disorder can develop at any age, starting as early as when a pup is a few months of age. Males are more likely to form clinically relevant stones than females. Urine sampling and ultrasound examinations are used for diagnostics. These stones are radiolucent and thus not clearly visible on radiographs.

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 Xanthinuria mutation can be safely bred with a clear dog with no copies of the Xanthinuria mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Xanthinuria 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 Xanthinuria mutation could develop due to a different genetic or clinical cause.

Poppi

Optimal Selection™ powered by WISDOM PANEL™

Breed: Cavapoo
Birth date: 2024-01
Test date: 2025-07-28
ID kit: DSNBMVX

Traits

Coat Color

	Gene	Variant	Copies	Result
Fawn	ASIP	a^y	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^t	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^B	1	Black or brindle possible
Mask	MC1R	E^m	0	No effect
Recessive Red (e1) To show a solid red coat, a dog must inherit two copies of a Recessive Red variant, one from each parent. This can either be two copies of a particular variant, such as this one (e1) or two of any combination of recessive red variants. Recessive red coats will appear white, cream, yellow or red, although there are other variants that can result in a similar appearance. The amount of red pigment in the coat, called the intensity, is governed by other genes.	MC1R	e^1	2	Cream to red coat likely
Recessive Red (e2)	MC1R	e^2	0	No effect
Recessive Red (e3)	MC1R	e^3	0	No effect
Sable (Discovered in the Cocker Spaniel)	MC1R	e^H	0	No effect
Widow's Peak (Discovered in Ancient dogs)	MC1R	e^A	0	No effect
Widow's Peak (Discovered in the Afghan Hound and Saluki)	MC1R	e^G	0	No effect

Color Modification

	Gene	Variant	Copies	Result
Cocoa (Discovered in the French Bulldog)	HPS3	co	0	No effect

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Color Modification

	Gene	Variant	Copies	Result
Red Intensity	MFSD12	i	0	No effect
Dilution (d1) Linkage test	MLPH	d^1	0	No effect
Dilution (d2)	MLPH	d^2	0	No effect
Dilution (d3)	MLPH	d^3	0	No effect
Chocolate (basd)	TYRP1	b^{asd}	0	No effect
Chocolate (bc)	TYRP1	b^c	0	No effect
Chocolate (bd)	TYRP1	b^d	0	No effect
Chocolate (be)	TYRP1	b^e	0	No effect
Chocolate (bh)	TYRP1	b^h	0	No effect
Chocolate (bs)	TYRP1	b^s	0	No effect

Coat Patterns

	Gene	Variant	Copies	Result
Piebald Dog with copies of the Piebald variant are likely to show white spotting, patches and/or a white coat, with two copies having a greater effect than one, although the strength of this effect may be influenced by other genes.	MITF	s^p	1	White markings possible
Merle	PMEL	M	0	No effect
Harlequin	PSMB7	H	0	No effect
Saddle Tan One or two copies of the Saddle Tan variant are needed for the "saddle" to be seen. However the Tan Points variant must also be present. The Saddle Tan variant is actually considered to be the wild type, or default, variant.	RALY	-	1	Saddle possible
Roan Linkage Test	USH2A	TR^r	0	No effect

Poppi

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Breed: Cavapoo
Birth date: 2024-01

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Coat Length and Curl

	Gene	Variant	Copies	Result
Long Hair (lh1) To show a long coat, a dog must inherit two copies of a Long Hair variant, one from each parent. This can either be two copies of a particular variant, such as this one (lh1) or two of any combination of long hair variants. However, there are other variants suspected to influence coat length.	FGF5	lh^{1}	2	Long coat
Long Hair (lh2)	FGF5	lh^{2}	0	No effect
Long Hair (lh3)	FGF5	lh^{3}	0	No effect
Long Hair (lh4)	FGF5	lh^{4}	0	No effect
Long Hair (lh5)	FGF5	lh^{5}	0	No effect
Curly Coat One copy of this variant is likely to give a soft curl or wave whereas two copies are likely to give a tighter curl. A curly coat is less apparent in dogs with short hair than those with long. There is one other known Curl variant, and likely other unknown variants that exist.	KRT71	C	1	Soft curl or wave likely

Hairlessness

	Gene	Variant	Copies	Result
Hairlessness (Discovered in the Chinese Crested Dog) Linkage test	FOXI3	Hr^{cc}	0	No effect
Hairlessness (Discovered in the American Hairless Terrier)	SGK3	hr^{aht}	0	No effect
Hairlessness (Discovered in the Scottish Deerhound)	SKG3	hr^{sd}	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	2	Low shedder

Poppi

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More Coat Traits

	Gene	Variant	Copies	Result
Hair Ridge	FGF3, FGF4, FGF19, ORAOV1	R	0	No effect
Furnishings Dogs with one or two copies of the Furnishing variant are likely to display a fuzzy beard, moustache and eyebrows, but a long or curly coat will make this variant less apparent.	RSPO2	F	1	Furnishings likely
Albino	SLC45A2	c^{al}	0	No effect

Head Shape

	Gene	Variant	Copies	Result
Short Snout (BMP3 variant) Having two copies of this variant may have a slight shortening effect on snout length.	BMP3	-	1	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
Floppy Ears 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

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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
Short Legs (Chondrodysplasia, CDPA)	FGF4	-	0	No effect
Short Legs (Chondrodystrophy, CDDY) Dogs with one copy of the Short Legs (CDDY) variant typically have some shortening of their legs, whereas dogs with two copies can have more obvious shortening. Dogs that inherit both variants associated with short legs (CDDY and CDPA) tend to show a more drastic reduction in leg length.	FGF4	-	1	Slightly shortened legs likely
Short Tail	T-box	T	0	Full tail length likely

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Other health conditions tested

Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD) Risk	Gene	Risk Variant	Copies	Inheritance	Result
	FGF4 retrogene	Insertion	1	AD	**At Risk**

Information about the genetic condition

Chondrodystrophy (CDDY) is a form of skeletal dysplasia which affects the development of cartilage and bone growth in a number of dog breeds. The associated CDDY genetic variant is an FGF4-retrogene insertion on dog chromosome 12, discovered by researchers in the Bannasch Laboratory at the University of California, Davis (Brown et al. 2017), and should not be confused with the FGF4-retrogene insertion on dog chromosome 18 (Parker et al. 2017), associated with a short-legged phenotype known as chondrodysplasia (CDPA). In dogs with CDDY, disproportionate growth (short limbs, normal sized body and head) can be observed as early as one week of age. CDDY follows a semi-dominant mode of inheritance. This means dogs with one copy of the genetic variant typically have some shortening of their legs, whereas dogs with two copies will show a more obvious shortening. Although not necessarily directly associated with CDDY, valgus limb deformities may be observed during physical examination of some dogs. However, affected dogs are more likely to experience premature degeneration and calcification of the intervertebral discs, a process also known as intervertebral disc disease (IVDD). Dogs with IVDD secondary to this genetic variant have an increased risk of intervertebral disc herniation (IVDH), consistent with Hansen Type I. The risk of developing IVDH follows a dominant mode of inheritance, meaning only one copy of this variant is needed to consider a dog predisposed for disc herniation. Age of onset of disc herniation appears to vary considerably between breeds, with the median age of dogs presenting for surgery varying from 3 years to 10 years. However, please note this variant is a risk factor and some dogs with one, or even two copies, of this variant may not go on to show signs of disc disease. It is worth clarifying that if disc herniation does not occur dorsally, a dog may appear asymptomatic as the spinal cord is less likely to be compressed. Additionally, not all dogs affected by IVDD have the FGF4-retrogene insertion found on chromosome 12, indicating additional genetic causes remain to be discovered.

Breeder recommendation

This variant is considered a risk factor for Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD), and dogs with one or two copies of the variant are at increased risk. However not all dogs with one or two copies of this variant will show signs of disc disease. Use of dogs with one or two copies of the CDDY and IVDD variant should be critically considered, as there is a risk that the resulting litter will contain affected puppies. For example, if a dog with one copy of the CDDY and IVDD variant is bred with a clear dog with no copies of the CDDY and IVDD variant, about half of the puppies will have one copy and half will have no copies of the CDDY and IVDD variant. Some breeds carry the variant at such a high rate that breeding dogs with one copy of the disorder is unavoidable. In such cases, mate selection should be planned to slowly reduce the frequency of the variant within the breed over time if possible. In breeds where both FGF4 retrogenes are present and a short stature is desirable, breeders can select for dogs positive for the CDPA (chromosome 18) variant, and against dogs with the CDDY (chromosome 12) variant to maintain breed-specific leg length. Please note: It is possible that clinical signs similar to the ones associated with the CDDY and IVDD variant could develop due to a different genetic or clinical cause.

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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
Alexander Disease	GFAP	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

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Centronuclear Myopathy (Discovered in the Labrador Retriever)	PTPLA	Insertion	0	AR	Clear
Cerebellar Ataxia	RAB24	A>C	0	AR	Clear
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
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 Cornification (Discovered in the Labrador Retriever)	NSDHL	Deletion	0	XD	Clear

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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
Congenital Hypothyroidism (Discovered in the Tenterfield Terrier)	TPO	C>T	0	AR	Clear
Congenital Hypothyroidism (Discovered in the Toy Fox and Rat Terrier)	TPO	C>T	0	AR	Clear
Congenital Muscular Dystrophy (Discovered in the Italian Greyhound)	LAMA2	G>A	0	AR	Clear
Congenital Muscular Dystrophy (Discovered in the Staffordshire Bull Terrier)	LAMA2	Deletion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Golden Retriever)	COLQ	G>A	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Heideterrier)	CHRNE	Insertion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Jack Russell Terrier)	CHRNE	Insertion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Labrador Retriever)	COLQ	T>C	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Old Danish Pointer)	CHAT	G>A	0	AR	Clear
Congenital Stationary Night Blindness (CSNB)	RPE65	A>T	0	AR	Clear
Craniomandibular Osteopathy (Discovered in Scottish Terrier breeds)	SLC37A2	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Australian Terrier)	COL1A1	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Basset Hound)	SLC37A2	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Weimaraner)	SLC35D1	Deletion	0	AD	Clear
Cystic Renal Dysplasia and Hepatic Fibrosis	INPP5E	G>A	0	AR	Clear

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Cystinuria Type I-A	SLC3A1	C>T	0	AR	Clear
Cystinuria Type II-A	SLC3A1	Deletion	0	AD	Clear
Darier Disease (Discovered in the Irish Terrier)	ATP2A2	Insertion	0	AD	Clear
Deafness and Vestibular Dysfunction (DINGS1), (Discovered in Doberman Pinscher)	PTPRQ	Insertion	0	AR	Clear
Deafness and Vestibular Dysfunction (DINGS2), (Discovered in Doberman Pinscher)	MYO7A	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

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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
Ehlers-Danlos Syndrome (Discovered in mixed breed)	COL5A1	G>A	0	AD	Clear
Ehlers-Danlos Syndrome (Discovered in the Labrador Retriever)	COL5A1	Deletion	0	AD	Clear
Epidermolytic Hyperkeratosis	KRT10	G>T	0	AR	Clear
Exercise-Induced Collapse	DNM1	G>T	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

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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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
GM1 Gangliosidosis (Discovered in the Shiba)	GLB1	Deletion	0	AR	Clear
GM2 Gangliosidosis (Discovered in the Japanese Chin)	HEXA	G>A	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 A (Discovered in the Labrador Retriever)	Confidential	-	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 Elliptocytosis	SPTB	C>T	0	AD	Clear
Hereditary Footpad Hyperkeratosis	FAM83G	G>C	0	AR	Clear

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

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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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
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) Variant 1	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

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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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
Mucopolysaccharidosis VI (Discovered in the Miniature Pinscher)	ARSB	G>A	0	AR	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 Dystrophy-Dystroglycanopathy (Discovered in the Labrador Retriever)	LARGE	C>T	0	AR	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 Labrador Retriever)	CLCN1	T>A	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)	HCRTR2	G>A	0	AR	Clear

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Narcolepsy (Discovered in the Labrador Retriever)	HCRTR2	G>A	0	AR	Clear
Nemaline Myopathy	NEB	C>A	0	AR	Clear
Neonatal Cerebellar Cortical Degeneration	SPTBN2	Deletion	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
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
Obesity risk (POMC)	POMC	Deletion	0	AD	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

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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
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

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Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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
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

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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
Skeletal Dysplasia 2	COL11A2	G>C	0	AR	Clear
Spinocerebellar Ataxia (Late-Onset Ataxia)	CAPN1	G>A	0	AR	Clear
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
Stargardt Disease (Discovered in the Labrador Retriever)	ABCA4	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

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Other health conditions tested

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