

# Canadian Border Collie Association

# Société canadienne de Border Collie

Registration No. CBCA 17355

## Certificate of Registration

## Certificat d'enregistrement

Identification: 95600

Name - Nom: **Flj**

Born - Date de Naissance: 28-May-20

Sex - Sexe: F

Litter F/M - Porté F/M 3 / 1

Description:

Medium Coat, MSize, Black & white

Owner-Prop: Jamie Gardner

Box 1501, Shaunavon, SK, Canada

Breeder - Éleveur: Rose McLeod

Aldergrove, BC, Canada

Sire - Père: Sheepcreek Cole CBCA 14262

Owner-Prop: Rose McLeod

ALDERGROVE, BC CANADA

Breeder - Éleveur: Jamie Gardner

SHAUNAVON, SK

Dam-Mère: Kate CBCA 12017

Owner-Prop: Rose McLeod

ALDERGROVE, BC CANADA

Breeder - Éleveur: Rose McLeod

ALDERGROVE, BC

Registrar: Tara Dier/Carol Guy  
Box 817, Shillog, ON, K0K3E0  
registrar@canadabordercollies.org  
www.canadabordercollies.org

Date of Issue - Date d'Émission  
10-Aug-20

1 Canadian Champion

*Flj*

@Imp. Llanfarian Cap CBCA 1

Ken MacKenzie  
Drayton Valley, AB Canada

Glen ISDS 244489

S Van Der Zweep

Renkum Netherlands

Lucky ISDS 272440

S Van Der Zweep

Renkum Netherlands

#Spot ISDS 262341

Robert Dalziel

Scotland

Mist ISDS 284622

D. Henderson

Northumberland England

\*Imp. Nap ABC 292084

Alasdair & Patricia MacRae

Vichy, MO USA

JB Jill CBCA 5497

Steven Bonk

Wolsley, SK Canada

Keen-Eye Josh CBCA 4048

Bruce & Rose McLeod

Aldergrove, BC Canada

Jana CBCA 1952

Bruce Or Rose McLeod

Aldergrove, BC Canada

Skippy ISDS 233488

N. F. Deacon

Neil ISDS 225059

S. Heffernan

Joe ISDS 255447

Sean Donaghy

Lass ISDS 262619

S Van Der Zweep Renkum

Tam ISDS 227645

R. Robertson Ayrs Scotia

If ISDS 246859

Ms S. McGarty UK

Glen ISDS 248367

Andrew Dickman Benwick

Flame ISDS 252956

M. K. Northwood

Chip ISDS 224138

M. L. Evans Wales

Eryri Sian ISDS 24800

K. B. Williams UK

Nick ABC 288974

Date & Dawn Montgomerie

Dawn CBCA 4519

Jill Brodie Rumsey, AB C

Imp Keen CBCA 2826

Diana Gauthier Lumby, E

Bergen Bobbie CBCA

Diana Gauthier Lumby, E

Keen-Eye Craig CBC

Bruce Or Rose McLeod

BentHillKeeper CBCA

Bruce Or Rose McLeod



**Breed:** Border Collie  
**Microchip number:** 956000011401420  
**Birth date:** 2021-05-28  
**Owner:** Jamie Gardner

**Registry:** Canadian Border Collie Association  
**Registration number:** CBCA17355  
**Test date:** 2022-11-04  
**ID kit:** DBSHYVD

## Fly's Profile

### Pet information

**Registered name**  
Fly

**Sex**  
F

### Breed specific genetic health tests

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Early Adult Onset Deafness For Border Collies only (Linkage test)	Intergenic	Insertion	1	AR	Carrier
Collie Eye Anomaly (CEA)	NHEJ1	Deletion	0	AR	Clear
Dental Hypomineralization	FAM20C	C>T	0	AR	Clear
Hyperuricosuria	SLC2A9	G>T	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)	CUBN	Deletion	0	AR	Clear
MDR1 Medication Sensitivity	MDR1/ABCB1	Deletion	0	AD	Clear
Sensory Neuropathy	FAM134B	Insertion	0	AR	Clear
Trapped Neutrophil Syndrome	VPS13B	Deletion	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.

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## Fly's Profile

### Pet information

**Registered name**

Fly

**Sex**

F

**Owner reported breed**

Border Collie

**Date of birth**

2021-05-28

**Microchip number**

956000011401420

### Genetic Diversity

**Fly's Percentage of Heterozygosity**

34%

### Health summary

**At Risk** 0 conditions**Carrier** 1 condition

- Early Adult Onset Deafness For Border Collies only (Linkage test)

**Clear** 212 conditions

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## Genetic Diversity

### Heterozygosity

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#### Fly's Percentage of Heterozygosity

34%

Fly's genome analysis shows an average level of genetic heterozygosity when compared with other Border Collies.

#### Typical Range for Border Collies

32% - 39%

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## Health conditions known in the breed

### Early Adult Onset Deafness For Border Collies only (Linkage test)

Gene	Risk Variant	Copies	Inheritance	Result
Intergenic	Insertion	1	AR	Carrier

#### Information about the genetic condition

Gradual hearing loss affecting both ears is observed usually between the ages of 5 to 7 years. Please note that this test is specifically for the Border Collie breed and is a predictive linkage test rather than a test for the true causal variant. Not all dogs with two copies of the linked marker will go on to show signs of hearing loss.

#### 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 Deafness mutation can be safely bred with a clear dog with no copies of the Deafness mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Deafness mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. The carrier rate of the risk variant is up to 35% in the Border Collie population, highlighting the importance of keeping healthy carriers in the breeding program by breeding them to dogs tested "Clear" (zero copies) of the risk variant. Please note: It is possible that disease signs similar to the ones caused by the Deafness mutation could develop due to a different genetic or clinical cause.

### Collie Eye Anomaly (CEA)

Gene	Risk Variant	Copies	Inheritance	Result
NHEJ1	Deletion	0	AR	Clear

#### Information about the genetic condition

Collie Eye Anomaly is primarily characterized by choroidal hypoplasia, leading to an underdeveloped vascular supply to the retina, and is especially visible temporal to the optic nerve. CEA lesions may be present in both eyes or asymmetric in nature. CEA-associated choroidal hypoplasia is non-progressive and usually does not cause visual deficits on its own. However, CEA has a range of clinical expressions. Vision impairment is more likely in dogs with the "extended CEA phenotype," which may include optic nerve head colobomas, retinal detachment or intraocular hemorrhage secondary to coloboma(s) in severely affected dogs. Optic nerve head colobomas appear as excavations of the optic disc surface. Diagnosis of CEA lesions should be completed before 10 weeks of age, as retinal pigmentation can mask choroidal hypoplasia as the puppies grow, a phenomenon termed "go normal" by breeders. Research is ongoing to determine what additional genetic factors may be present that influence the range of severity seen in dogs with CEA.

#### 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 Collie Eye Anomaly variant can be safely bred with a clear dog with no copies of the Collie Eye Anomaly variant. About half of the puppies will have one copy (carriers) and half will have no copies of the variant. Furthermore, a dog with two copies of the CEA variant 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: Recent research has suggested that additional genetic risk factors likely exist in some breeds that resemble or contribute to CEA risk, especially the more severe disorder expression. It is possible that disorder signs similar to the ones associated with this CEA variant could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

### Dental Hypomineralization

Gene	Risk Variant	Copies	Inheritance	Result
FAM20C	C>T	0	AR	Clear

#### Information about the genetic condition

Clinical signs include brownish dental discoloration and abnormal wear of teeth. As the teeth wear, the biting surfaces of the teeth darkens, become dark brown in color; the enamel layer may also show a light brown discoloration and appear dull. The disorder causes severe tooth wear leading to pulp exposure, chronic inflammation of the pulp, and pulpal necrosis. Histologically, dentin of affected dogs has an abnormal structure and the enamel can be slightly hypoplastic.

#### 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 Dental Hypomineralization mutation can be safely bred with a clear dog with no copies of the Dental Hypomineralization mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Dental Hypomineralization mutation. A dog with two copies of the Dental Hypomineralization 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 Dental Hypomineralization 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

### Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)

Gene	Risk Variant	Copies	Inheritance	Result
CUBN	Deletion	0	AR	Clear

#### Information about the genetic condition

Initial signs of intestinal cobalamin malabsorption can be seen in puppies 6 to 12 weeks of age, when cobalamin store become depleted. Puppies with IGS suffer from weakness and loss of appetite and fail to grow normally. Bloodwork shows anemia, neutropenia, and low cobalamin concentrations. High levels of homocysteine and methylmalonic acid can also be observed in the blood. Proteinuria is typically present.

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

### MDR1 Medication Sensitivity

Gene	Risk Variant	Copies	Inheritance	Result
MDR1/ABCB1	Deletion	0	AD	Clear

#### Information about the genetic condition

Dogs with this variant are asymptomatic until exposed to a medication that uses the drug transport pump rendered defective by the mutation in the MDR1 (also called ABCB1) gene. Medications known to use this P-glycoprotein pump are macrocyclic lactones (antiparasitic drugs), loperamide (antidiarrheal), erythromycin (antibiotic), acepromazine (tranquilizer), butorphanol (opioid), certain drugs used in cancer treatment (vincristine, vinblastine, and doxorubicin), and others. When these medications are administered, they accumulate in the brain which results in adverse reactions. Typical symptoms include tremors, loss of balance, seizures, obtundation, excessive salivation, dilated pupils, and bradycardia. If untreated, the condition may lead to respiratory arrest, coma or death. Because dogs with 1 copy of the variant will have some P-glycoprotein function, the most severe cases tend to occur in dogs that have 2 copies of the variant and, therefore, lack any functional P-glycoprotein pumps. However, the disorder can still be very severe in dogs that have only one copy of the mutation.

#### Breeder recommendation

This disorder is autosomal dominant meaning that only one copy of the variant is needed for associated signs to occur. For some breeds where the MDR1 mutation frequency is particularly high, breeders may consider mating pairs using dogs that have one or two copies of the MDR1 variant to maintain genetic diversity within their breed. It is important that resulting puppies be tested for the MDR1 variant to ensure safe future medical treatment. If a dog with one copy of the MDR1 variant is bred with a clear dog with no copies of the MDR1 variant, about half of the puppies will have one copy and half will have no copies of the MDR1 variant. If a dog with two copies of the MDR1 variant is bred with a clear dog, the resulting puppies will all have one copy of the variant. Please note: It is possible that clinical signs similar to the ones caused by the MDR1 variant could develop due to a different genetic or clinical cause.



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## Health conditions known in the breed

### Sensory Neuropathy

Gene	Risk Variant	Copies	Inheritance	Result
FAM134B	Insertion	0	AR	<b>Clear</b>

#### Information about the genetic condition

Clinical signs are detectable in puppies from two to seven months of age. Clinical signs include incoordination of gait (ataxia), knuckling of the paws, hyperextension of the limbs, and self-mutilation of the limbs. The hind legs are usually most severely affected. Loss of sensation is progressive and affects all limbs. Urinary incontinence and regurgitation can occur in the later stages of the disorder.

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

### Trapped Neutrophil Syndrome

Gene	Risk Variant	Copies	Inheritance	Result
VPS13B	Deletion	0	AR	<b>Clear</b>

#### Information about the genetic condition

Clinical signs of TNS include an exceptional susceptibility to infections secondary to the low number of circulating neutrophils in the blood stream. Affected dogs also tend to suffer from chronic inflammatory conditions such as arthritis. Clinical signs are usually observed by 6 to 12 weeks of age and can include a smaller overall size as well as a ferret-like face due to abnormal craniofacial development leading to a narrowed, elongated skull shape. For some affected dogs, clinical signs can be mild and go unnoticed until adulthood. Nevertheless, TNS is a severe disease and affected dogs have a shorter life expectancy.

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

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## Traits

### Coat Color

	Gene	Variant	Copies	Result
<b>Fawn</b>	ASIP	ay	0	No effect
<b>Recessive Black</b>	ASIP	a	0	No effect
<b>Tan Points</b> Two copies, or occasionally one copy, of this variant may result in a black and tan coat color pattern.	ASIP	at	2	Tan points possible
<b>Dominant Black</b> 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>	1	Black or brindle possible
<b>Mask</b>	MC1R	E <sup>m</sup>	0	No effect
<b>Recessive Red (e1)</b>	MC1R	e <sup>1</sup>	0	No effect
<b>Recessive Red (e2)</b>	MC1R	e <sup>2</sup>	0	No effect
<b>Recessive Red (e3)</b>	MC1R	e <sup>3</sup>	0	No effect
<b>Widow's Peak (Discovered in Ancient dogs)</b>	MC1R	e <sup>A</sup>	0	No effect
<b>Widow's Peak (Discovered in the Afghan Hound and Saluki)</b>	MC1R	E <sup>G</sup>	0	No effect

### Color Modification

	Gene	Variant	Copies	Result
<b>Red Intensity</b>	MFSD12	i	0	No effect
<b>Dilution (d1) Linkage test</b>	MLPH	d <sup>1</sup>	0	No effect
<b>Dilution (d2)</b>	MLPH	d <sup>2</sup>	0	No effect
<b>Dilution (d3)</b>	MLPH	d <sup>3</sup>	0	No effect
<b>Chocolate (basd)</b>	TYRP1	b <sup>asd</sup>	0	No effect
<b>Chocolate (bc)</b>	TYRP1	b <sup>c</sup>	0	No effect

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## Color Modification

	Gene	Variant	Copies	Result
Chocolate (bd)	TYRP1	b <sup>d</sup>	0	No effect
Chocolate (bs)	TYRP1	b <sup>s</sup>	0	No effect

## Coat Patterns

	Gene	Variant	Copies	Result
Piebald	MITF	s <sup>p</sup>	0	No effect
Merle	PMEL	M	0	No effect
Harlequin	PSMB7	H	0	No effect
Saddle Tan	RALY	-	1	Saddle possible

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.

## 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 <sup>1</sup>	2	Long coat
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
Long Hair (lh5)	FGF5	lh <sup>5</sup>	0	No effect
Curly Coat	KRT71	C	0	No effect

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## 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	MC5R	sd	0	Seasonal 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

## 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



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## Ears

	Gene	Variant	Copies	Result
Floppy Ears	MSRB3	-	0	Pricked 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
Short Legs (Chondrodysplasia, CDPA)	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
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 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
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 Scott Syndrome	ANO6	G>A	0	AR	Clear
Centronuclear Myopathy (Discovered in the Great Dane)	BIN1	A>G	0	AR	Clear
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

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Test date: 2022-11-04  
ID kit: DBSHYVD

## Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Cleft Lip & Palate with Syndactyly	ADAMTS20	Deletion	0	AR	Clear
Cleft Palate	DLX6	C>A	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 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 Myasthenic Syndrome (Discovered in the Golden Retriever)	COLQ	G>A	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
Craniofacial Osteopathy (Discovered in Scottish Terrier breeds)	SLC37A2	C>T	0	AD	Clear
Cystic Renal Dysplasia and Hepatic Fibrosis	INPP5E	G>A	0	AR	Clear

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## Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Cystinuria Type I-A	SLC3A1	C>T	0	AR	Clear
Cystinuria Type II-A	SLC3A1	Deletion	0	AD	Clear
Deafness and Vestibular Dysfunction (DINGS1), (Discovered in Doberman Pinscher)	PTPRQ	Insertion	0	AR	Clear
Degenerative Myelopathy	SOD1	G>A	0	AR	Clear
Demyelinating Neuropathy	SBF2	G>T	0	AR	Clear
Dilated Cardiomyopathy (Discovered in the Schnauzer)	RBM20	Deletion	0	AR	Clear
Dominant Progressive Retinal Atrophy	RHO	C>G	0	AD	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 Retinal Degeneration (Discovered in the Norwegian Elkhound)	STK38L	Insertion	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
Epidermolytic Hyperkeratosis	KRT10	G>T	0	AR	Clear
Episodic Falling Syndrome	BCAN	Insertion	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
Fanconi Syndrome	FAN1	Deletion	0	AR	Clear
Fetal Onset Neuroaxonal Dystrophy	MFN2	G>C	0	AR	Clear



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## Other health conditions tested

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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 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
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

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Hemophilia B (Discovered in the Lhasa Apso)	FIX	Deletion	0	XR	Clear
Hereditary Ataxia (Discovered in the Norwegian Buhund)	KCNIP4	T>C	0	AR	Clear
Hereditary Elliptocytosis	SPTB	C>T	0	AD	Clear
Hereditary Footpad Hyperkeratosis	FAM83G	G>C	0	AR	Clear
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
Intestinal Cobalamin Malabsorption (Discovered in the Beagle)	CUBN	Deletion	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Komondor)	CUBN	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
Lagotto Storage Disease	ATG4D	G>A	0	AR	Clear

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## Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Lamellar Ichthyosis	TGM1	Insertion	0	AR	Clear
Lethal Acrodermatitis (Discovered in the Bull Terrier)	MKLN1	A>C	0	AR	Clear
Ligneous Membranitis	PLG	T>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
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
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

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## Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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)	HCRT2	G>A	0	AR	Clear
Narcolepsy (Discovered in the Labrador Retriever)	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
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
Osteochondrodysplasia	SLC13A1	Deletion	0	AR	Clear



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<b>Osteochondromatosis (Discovered in the American Staffordshire Terrier)</b>	EXT2	C>A	0	AR	Clear
<b>Osteogenesis Imperfecta (Discovered in the Beagle)</b>	COL1A2	C>T	0	AD	Clear
<b>Osteogenesis Imperfecta (Discovered in the Dachshund)</b>	SERPINH1	T>C	0	AR	Clear
<b>P2RY12-associated Bleeding Disorder</b>	P2RY12	Deletion	0	AR	Clear
<b>Paroxysmal Dyskinesia</b>	PIGN	C>T	0	AR	Clear
<b>Persistent Müllerian Duct Syndrome</b>	AMHR2	C>T	0	AR	Clear
<b>Phosphofructokinase Deficiency</b>	PFKM	G>A	0	AR	Clear
<b>Polycystic Kidney Disease</b>	PKD1	G>A	0	AD	Clear
<b>Prekallikrein Deficiency</b>	KLKB1	T>A	0	AR	Clear
<b>Primary Ciliary Dyskinesia</b>	CCDC39	C>T	0	AR	Clear
<b>Primary Ciliary Dyskinesia (Discovered in the Alaskan Malamute)</b>	NME5	Deletion	0	AR	Clear
<b>Primary Lens Luxation</b>	ADAMTS17	G>A	0	AR	Clear
<b>Primary Open Angle Glaucoma (Discovered in Basset Fauve de Bretagne)</b>	ADAMTS17	G>A	0	AR	Clear
<b>Primary Open Angle Glaucoma (Discovered in Petit Basset Griffon Vendéen)</b>	ADAMTS17	Insertion	0	AR	Clear
<b>Primary Open Angle Glaucoma and Lens Luxation (Discovered in Chinese Shar-Pei)</b>	ADAMTS17	Deletion	0	AR	Clear
<b>Progressive Early-Onset Cerebellar Ataxia</b>	SEL1L	T>C	0	AR	Clear
<b>Progressive Retinal Atrophy (Discovered in the Basenji)</b>	SAG	T>C	0	AR	Clear
<b>Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA1 variant)</b>	SLC4A3	Insertion	0	AR	Clear
<b>Progressive Retinal Atrophy (Discovered in the Lhasa Apso)</b>	IMPG2	Insertion	0	AR	Clear

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## Other health conditions tested

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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
Progressive Rod Cone Degeneration (prcd-PRA)	PRCD	G>A	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
Sensory Ataxic Neuropathy	tRNATyr	Deletion	0	MT	Clear
Severe Combined Immunodeficiency (Discovered in Frisian Water Dogs)	RAG1	G>T	0	AR	Clear

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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
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

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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 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.