

GRETA THUMBERG - NIKE
Record: 12057
Razza: BORDER COLLIE



ID sample: DFLTVBD
Date test:04/11/2025
MyDogDNA - Vetogene
ENCI SERVIZI

DNA Test Report

Info proprietario

Nome proprietario
ZANE CHIARA-FEDERICA

Info Animale

Nome
GRETA THUMBERG - NIKE

Data di nascita
15/03/2023

Sesso
F

ID campione
DFLTVBD

Registrazione
LO2379040

Microchip
380260102392538

Razza
BORDER COLLIE

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Variabilità Genetica - Eterozigosità

Cooper's Percentage of Heterozygosity: 0.33%

Cooper's genome analysis shows an average level of genetic heterozygosity when compared with other BORDER COLLIE
Typical Range for BORDER COLLIE 0.3129 - 0.4012%

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Health Conditions Known in This Breed

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Hereditary Calcium Oxalate Urolithiasis, Type 1	Confidential	->	R	0	Clear

Informazioni sulla patologia genetica

Hereditary Calcium Oxalate Urolithiasis, Type 1 is a disorder that is associated with increased risk of urinary calcium oxalate stone formation. Affected dogs will demonstrate clinical signs consistent with urolithiasis. This may range from being asymptomatic to hematuria (bloody urine), dysuria (painful urination), stranguria (straining to pass urine) and pollakiuria (frequent urination). Dogs with urinary stones are also more susceptible to urinary tract infections. And, due to the presence of the stones, affected dogs are at risk of urinary obstruction occurring at the renal pelvis, ureters, or urethra. Blockage of the urinary tract is a life-threatening condition that requires immediate intervention. While the average age of diagnosis is 3 years old, dogs affected by CaOx1 have the potential to develop urinary stones as puppies. And recurrent stone formation is common for affected dogs. There is evidence to suggest the clinical signs are more common in males than in females.

Indicazioni per la riproduzione

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 Hereditary Calcium Oxalate Urolithiasis, Type 1 variant can be safely bred with a clear dog with no copies of the Hereditary Calcium Oxalate Urolithiasis, Type 1 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 Hereditary Calcium Oxalate Urolithiasis, Type 1 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: It is possible that disorder signs similar to the ones associated with this CaOx1 variant could develop due to a different genetic or clinical cause.

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)	CUBN	I>D	R	0	Clear

Informazioni sulla patologia genetica

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.

Indicazioni per la riproduzione

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.

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Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
MDR1 Medication Sensitivity	MDR1/ABCB1	I>D	D	0	Clear

Informazioni sulla patologia genetica

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.

Indicazioni per la riproduzione

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.

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Collie Eye Anomaly (CEA)	NHEJ1	I>D	R	0	Clear

Informazioni sulla patologia genetica

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.

Indicazioni per la riproduzione

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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Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Sensory Neuropathy	FAM134B	D>I	R	0	Clear

Informazioni sulla patologia genetica

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.

Indicazioni per la riproduzione

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.

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Dental Hypomineralization	FAM20C	C>T	R	0	Clear

Informazioni sulla patologia genetica

Clinical signs include brownish dental discoloration and abnormal wear of teeth. As the teeth wear, the biting surfaces of the teeth darken, 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.

Indicazioni per la riproduzione

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.

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Trapped Neutrophil Syndrome	VPS13B	I>D	R	0	Clear

Informazioni sulla patologia genetica

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

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expectancy.

Indicazioni per la riproduzione

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.

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Goniodysgenesis and Glaucoma (Discovered in the Border Collie)	OLFML3	G>A	R	0	Clear

Informazioni sulla patologia genetica

Glaucoma is characterized by reduced outflow of intraocular fluid and elevated intraocular pressure. The first clinical signs are usually early onset for this condition. Goniodysgenesis, narrowing or closure of the iridocorneal angle through which the aqueous humor drains, may be detectable through gonioscopy. Goniodysgenesis may lead to glaucoma, however some dogs will go through their lives without any increase in eye pressure despite severe goniodysgenesis. Untreated glaucoma may lead to damage to the retina, resulting in vision loss and blindness.

Indicazioni per la riproduzione

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

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie)	CLN5	C>T	R	0	Clear

Informazioni sulla patologia genetica

Neuronal ceroid lipofuscinoses (NCLs) are a group of inherited progressive neurodegenerative lysosomal storage disorders. NCLs are characterized by excessive accumulation of lipofuscin and ceroid lipopigments in the central nervous system and other tissues. The age of onset for dogs affected with Neuronal Ceroid Lipofuscinosis 5 (NCL5) can vary significantly, with some showing initial signs at 1 to 2 years of age while others show later in life. Similarly, severity of clinical signs can vary between affected individuals. Typical signs of NCL5 include vision impairment, epileptic seizures, ataxia (uncoordinated movements), and behavioral changes, such as hyperactivity and aggression. Some affected dogs can show air biting, likely secondary to hallucinations. Due to the progressive nature of NCL5, the average prognosis is considered poor for affected dogs. And the average life expectancy is less than 2.5 years.

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Indicazioni per la riproduzione

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 Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie) variant can be safely bred with a clear dog with no copies of the Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie) variant. About half of the puppies will have one copy (carriers) and half will have no copies of the variant. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disorder signs similar to the ones associated with this NCL5 variant could develop due to a different genetic or clinical cause.

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Hyperuricosuria	SLC2A9	G>T	R	0	Clear

Informazioni sulla patologia genetica

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.

Indicazioni per la riproduzione

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.

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Early Adult Onset Deafness For Border Collies only (Linkage test)	Intergenic	D>I	R	0	Clear

Informazioni sulla patologia genetica

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.

Indicazioni per la riproduzione

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

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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.					

Legenda for Inheritance mode:

R - Recessive

D - Dominant

S - X-linked

Autosomal Recessive

The trait is only expressed when both alleles (inherited from mother and father) contain the detrimental mutation. Regarding to the presence of mutations dogs are classified into three groups:

- Affected (mut/mut)- both alleles carry mutation, disease could be clinically expressed
- Carrier (mut/normal)- one of two alleles carry mutation (heterozygotes), disease is not clinically expressed
- Clear (normal/normal)- mutation is not detected, normal genotype, healthy animal for the trait

Heterozygotes in this case are the carriers of mutation since they do not express the disease (unwanted trait). It is especially important to test such animals for mutations, since mutated alleles are "silently" (without seeing unwanted phenotype) carried through the population.

Autosomal Dominant

The trait is expressed when one of the alleles (inherited either from mother or father) is damaged (contains detrimental mutation). Only one single mutated allele already could cause the disease. The importance for genetic testing of such animals is primarily in early diagnostics of the disease and identification of animals before they mate because most of diseases with autosomal dominant mode of inheritance have an onset later in animals life.

X-linked Recessive

The trait is carried on a sex chromosome and that a trait is expressed only when both alleles (inherited from mother and father) are damaged (contain detrimental mutation). Males carry only a single copy of the gene, inherited from mother, since male sex chromosome Y does not contain full DNA sequence as female X chromosome does. Females on the other hand contain two X chromosomes. Heterozygotes in this case are the carriers of mutation since they do not express the disease (unwanted trait). Males carry only one copy of a gene: they could be normal homozygote or affected homozygote.

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Trait Test

Coat Color

Tratto genetico	Gene	Variante	Copie	Risultato
Widow's Peak (Discovered in Ancient dogs)	MC1R	e ^A	0	No effect
Sable (Discovered in the Cocker Spaniel)	MC1R	e ^H	0	No effect
Recessive Red (Variant 1)	MC1R	e ¹	0	No effect
Recessive Red (Variant 3)	MC1R	e ³	0	No effect
Tan Points	ASIP	a ^t	2	Tan points possible
Two copies, or occasionally one copy, of this variant may result in a black and tan coat color pattern.				
Dominant Black	CBD103	K ^B	2	Black possible
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.				
Widow's Peak (Discovered in the Afghan Hound and Saluki)	MC1R	e ^G	0	No effect
Recessive Red (Variant 2)	MC1R	e ²	0	No effect
Fawn	ASIP	a ^y	0	No effect
Recessive Black	ASIP	a	0	No effect
Mask	MC1R	E ^m	0	No effect

Coat Length and Curl

Tratto genetico	Gene	Variante	Copie	Risultato
Long Hair (Variant 1)	FGF5	lh ¹	2	Long coat
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.				
Long Hair (Variant 2)	FGF5	lh ²	0	No effect
Curly Coat	KRT71	C	0	No effect
Long Hair (Variant 4)	FGF5	lh ⁴	0	No effect
Long Hair (Variant 3)	FGF5	lh ³	0	No effect

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Tratto genetico	Gene	Variante	Copie	Risultato
Long Hair (Variant 5)	FGF5	lh ⁵	0	No effect

Coat Patterns

Tratto genetico	Gene	Variante	Copie	Risultato
Harlequin	PSMB7	H	0	No effect
Merle	PMEL	M	0	No effect
Piebald	MITF	s ^p	0	No effect
Roan (Linkage test)	USH2A	T ^r	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.

Color Modification

Tratto genetico	Gene	Variante	Copie	Risultato
Dilution (Variant 1) Linkage test	MLPH	d ¹	0	No effect
Chocolate (bc)	TYRP1	b ^c	0	No effect
Cocoa (Discovered in the French Bulldog)	HPS3	co	0	No effect
Chocolate (bs)	TYRP1	b ^s	0	No effect
Chocolate (be)	TYRP1	b ^e	0	No effect
Dilution (Variant 3)	MLPH	d ³	0	No effect
Chocolate (bd)	TYRP1	b ^d	0	No effect
Red Intensity	MFSD12	i	1	No effect

Dogs with two copies of the Red Intensity variant are more likely to show yellow, cream or white coat shades instead of deeper red shades. If the dog does not display solid red or red coat patterns, there will be no visible effect. Other genes, notably variants in the KITLG gene, are also thought to contribute to red pigment intensity variation, so some dogs may have yellow or buff colored coats.

Chocolate (bh)	TYRP1	b ^h	0	No effect
Dilution (Variant 2)	MLPH	d ²	0	No effect
Chocolate (basd)	TYRP1	b ^{asd}	0	No effect

Ears

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Tratto genetico	Gene	Variante	Copie	Risultato
Floppy Ears	MSRB3	-	0	Pricked ears more likely

Eye Color

Tratto genetico	Gene	Variante	Copie	Risultato
Blue Eyes	ALX4	-	0	No effect

Furnishings

Tratto genetico	Gene	Variante	Copie	Risultato
Furnishings	RSPO2	F	0	No effect

Hair Ridge

Tratto genetico	Gene	Variante	Copie	Risultato
Hair Ridge	FGF3, FGF4, FGF19, ORAOV1	R	0	No effect

Hairlessness

Tratto genetico	Gene	Variante	Copie	Risultato
Hairlessness (Discovered in the American Hairless Terrier)	SGK3	hr ^{ah} t	0	No effect
Hairlessness (Discovered in the Scottish Deerhound)	SKG3	hr ^{sd}	0	No effect
Hairlessness (Discovered in the Chinese Crested Dog) Linkage test	FOXI3	Hr ^{cc}	0	No effect

Head Shape

Tratto genetico	Gene	Variante	Copie	Risultato
Short Snout (Variant 1)	SMOC2	-	0	No effect
Short Snout (Variant 2)	BMP3	-	0	No effect

Hind Dewclaws

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Hind Dewclaws (Discovered in Asian breeds)	LMBR1	DC-1	0	No effect
Hind Dewclaws (Discovered in Western breeds)	LMBR1	DC-2	1	Hind dewclaws possible

One or two copies of this Hind Dewclaws variant may result in your dog having hind dewclaws. Around half of the dogs with one copy of this variant will have hind dewclaws, and it is possible for the dewclaws to be just on one leg. With two copies the trait is more likely to be expressed and could be more pronounced.

Leg Length

Tratto genetico	Gene	Variante	Copie	Risultato
Short Legs	FGF4	-	0	No effect

Muscular Body

Tratto genetico	Gene	Variante	Copie	Risultato
Back Muscle and Bulk	ACSL4	-	0	No effect

Pigment Absence

Tratto genetico	Gene	Variante	Copie	Risultato
Albino	SLC45A2	c ^{al}	0	No effect

Shedding

Tratto genetico	Gene	Variante	Copie	Risultato
Reduced Shedding	MC5R	sd	0	Seasonal shedder

Special Adaptations

Tratto genetico	Gene	Variante	Copie	Risultato
High Altitude Adaptation	EPAS1	-	0	No effect

Tail Length

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Short Tail	T-box	T	0	Full tail length likely

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Altre condizioni testate

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
Rod-Cone Dysplasia 3	PDE6A	I>D	R	0	Clear
X-Linked Hereditary Nephropathy (Discovered in the Samoyed)	COL4A5	G>T	S	0	Clear
Paroxysmal Dyskinesia	PIGN	C>T	R	0	Clear
Cerebellar Cortical Degeneration	SNX14	C>T	R	0	Clear
Early-onset PRA (Discovered in the Portuguese Water Dog)	CCDC66	D>I	R	0	Clear
Hemophilia A (Discovered in Old English Sheepdog)	FVIII	C>T	S	0	Clear
Neuroaxonal Dystrophy (Discovered in the Rottweiler)	VPS11	A>G	R	0	Clear
Congenital Hypothyroidism (Discovered in the Toy Fox and Rat Terrier)	TPO	C>T	R	0	Clear
Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA1)	KCNJ10	T>C	R	0	Clear
mpsiib-shipperke	NAGLU	D>I	R	0	Clear
Canine Multifocal Retinopathy 1	BEST1	C>T	R	0	Clear
Neuronal Ceroid Lipofuscinosis 12 (Discovered in the Australian Cattle Dog)	ATP13A2	C>T	R	0	Clear
Early-Onset Progressive Retinal Atrophy, (Discovered in the Spanish Water Dog)	PDE6B	I>D	R	0	Clear
Amelogenesis Imperfecta	ENAM	I>D	R	0	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Golden Retriever)	COL7A1	C>T	R	0	Clear
Inflammatory Myopathy (Discovered in the Dutch Shepherd Dog)	SLC25A12	A>G	R	0	Clear
Laryngeal Paralysis (Discovered in the Bull Terrier and Miniature Bull Terrier)	RAPGEF6	D>I	R	0	Clear
Limb-girdle Muscular Dystrophy	SGCD	I>D	R	0	Clear

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(Discovered in the Boston Terrier)	SGCD	I>D	R	0	Clear
Bandera's Neonatal Ataxia	GRM1	D>I	R	0	Clear
Neuronal Ceroid Lipofuscinosis 7	MFSD8	I>D	R	0	Clear
Neonatal Encephalopathy with Seizures	ATF2	T>G	R	0	Clear
Dilated Cardiomyopathy (Discovered in the Schnauzer)	RBM20	I>D	R	0	Clear
Congenital Muscular Dystrophy (Discovered in the Staffordshire Bull Terrier)	LAMA2	I>D	R	0	Clear
Palmoplantar Hyperkeratosis (Discovered in the Rottweiler)	DSG1	I>D	R	0	Clear
Von Willebrand's Disease, type 3 (Discovered in the Kooiker Hound)	VWF	G>A	R	0	Clear
Mucopolysaccharidosis Type IIIA (Discovered in the New Zealand Huntaway)	SGSH	D>I	R	0	Clear
X-Linked Progressive Retinal Atrophy 2	RPGR	I>D	S	0	Clear
Darier Disease (Discovered in the Irish Terrier)	ATP2A2	D>I	D	0	Clear
Hypocatalasia	CAT	G>A	R	0	Clear
Hereditary Nasal Parakeratosis (Discovered in the Labrador Retriever)	SUV39H2	A>C	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Lapponian Herder)	IFT122	C>T	R	0	Clear
Neonatal Cerebellar Cortical Degeneration	SPTBN2	I>D	R	0	Clear
limb-girdle-boston2	SGCD	CAT>GG	R	0	Clear
Inflammatory Pulmonary Disease (Discovered in the Rough Collie)	AKNA	I>D	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA 2 variant)	TTC8	I>D	R	0	Clear
Primary Open Angle Glaucoma	ADAMTS17	D>I	R	0	Clear

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(Discovered in Petit Basset Griffon Vendéen)	ADAMTS17	D>I	R	0	Clear
Sensorineural Deafness (Discovered in the Rottweiler)	LOXHD1	G>C	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - BBS2 variant)	Confidential	G>C	R	0	Clear
Pyruvate Kinase Deficiency (Discovered in the Pug)	PKLR	T>C	R	0	Clear
Congenital Dysmorphogenic Hypothyroidism with Goiter (Discovered in the Shih Tzu)	SLC5A5	G>A	R	0	Clear
Factor XI Deficiency	FXI	D>I	D	0	Clear
Spondylocostal Dysostosis	HES7	I>D	R	0	Clear
Lafora Disease (Linkage test)	NHLRC1	D>I	R	0	Clear
Congenital Myasthenic Syndrome (Discovered in the Heideterrier)	CHRNE	D>I	R	0	Clear
Congenital Myasthenic Syndrome (Discovered in the Labrador Retriever)	COLQ	T>C	R	0	Clear
Early Retinal Degeneration (Discovered in the Norwegian Elkhound)	STK38L	D>I	R	0	Clear
Dominant Progressive Retinal Atrophy	RHO	C>G	D	0	Clear
X-Linked Severe Combined Immunodeficiency (Discovered in the Basset Hound)	IL2RG	I>D	S	0	Clear
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA1 variant)	SLC4A3	D>I	R	0	Clear
Ichthyosis (Discovered in the American Bulldog)	NIPAL4	I>D	R	0	Clear
Craniomandibular Osteopathy (Discovered in the Australian Terrier)	COL1A1	C>T	D	0	Clear
Canine Multifocal Retinopathy 3	BEST1	I>D	R	0	Clear

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X-Linked Hereditary Nephropathy (Discovered in the Navasota Dog)	COL4A5	I>D	S	0	Clear
Congenital Myasthenic Syndrome (Discovered in the Jack Russell Terrier)	CHRNE	D>I	R	0	Clear
Amelogenesis Imperfecta (Discovered in the Lancashire Heeler)	Confidential	->-	R	0	Clear
Cystic Renal Dysplasia and Hepatic Fibrosis	INPP5E	G>A	R	0	Clear
Cone-Rod Dystrophy	NPHP4	I>D	R	0	Clear
Hemophilia A (Discovered in the Labrador Retriever)	Confidential	->-	S	0	Clear
Glycogen Storage Disease Type Ia	G6PC	G>C	R	0	Clear
Von Willebrand's Disease, type 2	VWF	T>G	R	0	Clear
Progressive Retinal Atrophy 1 (Discovered in the Italian Greyhound)	Confidential	G>A	R	0	Clear
Cone Degeneration (Discovered in the Alaskan Malamute)	CNGB3	I>D	R	0	Clear
Primary Ciliary Dyskinesia (Discovered in the Alaskan Malamute)	NME5	I>D	R	0	Clear
Stargardt Disease (Discovered in the Labrador Retriever)	ABCA4	D>I	R	0	Clear
Congenital Myasthenic Syndrome (Discovered in the Old Danish Pointer)	CHAT	G>A	R	0	Clear
Ehlers-Danlos syndrome (Discovered in mixed breed)	COL5A1	G>A	D	0	Clear
Canine Scott Syndrome	ANO6	G>A	R	0	Clear
L-2-Hydroxyglutaric Aciduria (Discovered in the Westie)	Confidential	D>I	R	0	Clear
muscular-dystrophy-lab-col6a3-r1576x	COL6A3	G>A	R	0	Clear
Congenital Myasthenic Syndrome (Discovered in the Golden Retriever)	COLQ	G>A	R	0	Clear

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Osteochondromatosis (Discovered in the American Staffordshire Terrier)	EXT2	C>A	R	0	Clear
Centronuclear Myopathy (Discovered in the Great Dane)	BIN1	A>G	R	0	Clear
Pyruvate Kinase Deficiency (Discovered in the West Highland White Terrier)	PKLR	D>I	R	0	Clear
Van den Ende-Gupta Syndrome	SCARF2	I>D	R	0	Clear
Hemophilia B	FIX	G>A	S	0	Clear
Xanthinuria (Discovered in the Toy Manchester Terrier)	Confidential	G>T	R	0	Clear
Mucopolysaccharidosis Type IIIA (Discovered in the Dachshund)	SGSH	C>A	R	0	Clear
Hemophilia A (Discovered in the Havanese)	FVIII	D>I	S	0	Clear
sca-dachsbracke	SCN8A	G>T	R	0	Clear
Muscular Dystrophy (Discovered in the Golden Retriever)	Dystrophin	A>G	S	0	Clear
Hereditary Footpad Hyperkeratosis	FAM83G	G>C	R	0	Clear
Congenital Hypothyroidism (Discovered in the Tenterfield Terrier)	TPO	C>T	R	0	Clear
Juvenile Dilated Cardiomyopathy (Discovered in the Toy Manchester Terrier)	ABCC9	G>A	R	0	Clear
Spinocerebellar Ataxia (Late-Onset Ataxia)	CAPN1	G>A	R	0	Clear
Cerebellar Ataxia	RAB24	A>C	R	0	Clear
Cleft Palate	DLX6	C>A	R	0	Clear
GM1 Gangliosidosis (Discovered in the Portuguese Water Dog)	GLB1	G>A	R	0	Clear
Cleft Lip & Palate with Syndactyly	ADAMTS20	I>D	R	0	Clear
Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA2)	ATP1B2	D>I	R	0	Clear

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Cystinuria Type I-A	SLC3A1	C>T	R	0	Clear
Disproportionate Dwarfism (Discovered in the Dogo Argentino)	PRKG2	C>A	R	0	Clear
Hypophosphatasia	Confidential	T>G	R	0	Clear
Obesity risk (POMC)	POMC	I>D	D	0	Clear
Rod-Cone Dysplasia 1	PDE6B	G>A	R	0	Clear
Degenerative Myelopathy	SOD1	G>A	R	0	Clear
Canine Congenital Stationary Night Blindness (Discovered in the Beagle)	LRIT3	I>D	R	0	Clear
Coat Color Dilution and Neurological Defects (Discovered in the Miniature Dachshund)	LAMA2	G>A	R	0	Clear
Hemophilia B (Discovered in the Airedale Terrier)	FIX	D>I	S	0	Clear
Juvenile Myoclonic Epilepsy	DIRAS1	I>D	R	0	Clear
Thrombopathia (Discovered in the Basset Hound)	RASGRP1	I>D	R	0	Clear
Fanconi Syndrome	FAN1	I>D	R	0	Clear
Early-Onset Adult Deafness (Discovered in the Rhodesian Ridgeback)	EPS8L2	I>D	R	0	Clear
Congenital Stationary Night Blindness (CSNB)	RPE65	A>T	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Papillon and Phalène)	CNGB1	I>D	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Miniature Long Haired Dachshund)	RPGRIP1	D>I	R	0	Clear
Startle Disease (Discovered in the Miniature American Shepherd)	Confidential	->-	R	0	Clear
Von Willebrand's Disease, type 3 (Discovered in the Scottish Terrier)	VWF	I>D	R	0	Clear
Leukodystrophy (Discovered in the	TSEN54	C>T	R	0	Clear

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Standard Schnauzer)	TSEN54	C>T	R	0	Clear
CNS Atrophy with Cerebellar Ataxia (Discovered in the Belgian Shepherd)	SEPP1	I>D	R	0	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Beagle)	CUBN	I>D	R	0	Clear
Hereditary Ataxia (Discovered in the Norwegian Buhund)	KCNIP4	T>C	R	0	Clear
Myeloperoxidase Deficiency	MOP	C>T	R	0	Clear
X-Linked Severe Combined Immunodeficiency (Discovered in the Cardigan Welsh Corgi)	IL2RG	D>I	S	0	Clear
Lagotto Storage Disease	ATG4D	G>A	R	0	Clear
Narcolepsy (Discovered in the Dachshund)	HCRTR2	G>A	R	0	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Saluki)	CLN8	D>I	R	0	Clear
Progressive Rod Cone Degeneration (prcd-PRA)	PRCD	G>A	R	0	Clear
Narcolepsy (Discovered in the Labrador Retriever)	HCRTR2	G>A	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Swedish Vallhund)	MERTK	D>I	R	0	Clear
Xanthinuria (Discovered in the Cavalier King Charles Spaniel)	Confidential	I>D	R	0	Clear
GM1 Gangliosidosis (Discovered in the Shiba)	GLB1	I>D	R	0	Clear
Canine Multifocal Retinopathy 2	BEST1	G>A	R	0	Clear
Xanthinuria (Discovered in a mixed breed dog)	Confidential	G>A	R	0	Clear
Acute Respiratory Distress Syndrome	ANLN	C>T	R	0	Clear
Succinic Semialdehyde Dehydrogenase Deficiency (Discovered in the Saluki)	ALDH5A1	G>A	R	0	Clear

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Primary Open Angle Glaucoma (Discovered in Basset Fauve de Bretagne)	ADAMTS17	G>A	R	0	Clear
Enamel Hypoplasia (Discovered in the Parson Russell Terrier)	ENAM	C>T	R	0	Clear
Protein Losing Nephropathy	NPHS1	G>A	R	0	Clear
Lamellar Ichthyosis	TGM1	D>I	R	0	Clear
Muscular Hypertrophy (Double Muscling)	MSTN	T>A	R	0	Clear
May-Hegglin Anomaly	MYH9	G>A	D	0	Clear
Progressive Retinal Atrophy (Discovered in the Basenji)	SAG	T>C	R	0	Clear
Congenital Cornification (Discovered in the Labrador Retriever)	NSDHL	I>D	X-linked dominant	0	Clear
Centronuclear Myopathy (Discovered in the Labrador Retriever)	PTPLA	D>I	R	0	Clear
Primary Ciliary Dyskinesia	CCDC39	C>T	R	0	Clear
Progressive Early-Onset Cerebellar Ataxia	SEL1L	T>C	R	0	Clear
Ichthyosis (Discovered in the Great Dane)	SLC27A4	G>A	R	0	Clear
Glanzmann Thrombasthenia Type I (Discovered in Great Pyrenees)	ITGA2B	C>G	R	0	Clear
Alaskan Husky Encephalopathy	SLC19A3	G>A	R	0	Clear
Osteogenesis Imperfecta (Discovered in the Dachshund)	SERPINH1	T>C	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Lhasa Apso)	IMPG2	D>I	R	0	Clear
Globoid Cell Leukodystrophy (Discovered in Terriers)	GALC	A>C	R	0	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Australian Shepherd)	CLN8	G>A	R	0	Clear
Globoid Cell Leukodystrophy (Discovered in the Irish Setter)	GALC	A>T	R	0	Clear

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Lung Developmental Disease (Discovered in the Airedale Terrier)	LAMP3	C>T	R	0	Clear
Glycogen Storage Disease Type Ia (Discovered in the German Pinscher)	G6PC	D>I	R	0	Clear
Chondrodysplasia	ITGA10	C>T	R	0	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the English Setter)	CLN8	T>C	R	0	Clear
Bernard-Soulier Syndrome (Discovered in the Cocker Spaniel)	GP9	I>D	R	0	Clear
Microphthalmia (Discovered in the Soft-Coated Wheaten Terrier)	RBP4	I>D	R	0	Clear
Hemophilia B (Discovered in the Lhasa Apso)	FIX	I>D	S	0	Clear
Hereditary Nasal Parakeratosis (Discovered in the Greyhound)	SUV39H2	I>D	R	0	Clear
Nemaline Myopathy	NEB	C>A	R	0	Clear
L-2-Hydroxyglutaric Aciduria	L2HGDH	T>C	R	0	Clear
Phosphofructokinase Deficiency	PFKM	G>A	R	0	Clear
GM2 Gangliosidosis (Discovered in the Japanese Chin)	HEXA	G>A	R	0	Clear
2,8-dihydroxyadenine (DHA) Urolithiasis	APRT	G>A	R	0	Clear
Osteochondrodysplasia	SLC13A1	I>D	R	0	Clear
Muscular Dystrophy (Discovered in the Norfolk Terrier)	Dystrophin	I>D	S	0	Clear
Dental-Skeletal-Retinal Anomaly (Discovered in the Cane Corso)	MIA3	I>D	R	0	Clear
Cone Degeneration (Discovered in the German Shorthaired Pointer)	CNGB3	G>A	R	0	Clear
Deafness and Vestibular Dysfunction (Discovered in Doberman Pinscher)	PTPRQ	D>I	R	0	Clear
Cardiomyopathy and Juvenile Mortality	YARS2	G>A	R	0	Clear

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(Discovered in the Belgian Shepherd)	YARS2	G>A	R	0	Clear
QT Syndrome	KCNQ1	C>A	D	0	Clear
Renal Cystadenocarcinoma and Nodular Dermatofibrosis	FLCN	A>G	D	0	Clear
Fetal Onset Neuroaxonal Dystrophy	MFN2	G>C	R	0	Clear
Complement 3 Deficiency	C3	I>D	R	0	Clear
Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Alpine Dachsbracke)	CLN8	I>D	R	0	Clear
Canine Multiple Systems Degeneration (Discovered in the Chinese Crested Dog)	SERAC1	I>D	R	0	Clear
Thrombopathia (Discovered in the Eskimo Spitz)	RASGRP1	D>I	R	0	Clear
Muscular Dystrophy (Discovered in the Landseer)	COL6A1	G>T	R	0	Clear
Severe Combined Immunodeficiency (Discovered in Frisian Water Dogs)	RAG1	G>T	R	0	Clear
Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - CNGA1 variant)	CNGA1	I>D	R	0	Clear
Congenital Eye Malformations (Discovered in the Golden Retriever)	SIX6	C>T	D	0	Clear
Canine Leukocyte Adhesion Deficiency (CLAD), type III	FERMT3	D>I	R	0	Clear
Primary Open Angle Glaucoma and Lens Luxation (Discovered in Chinese Shar-Pei)	ADAMTS17	I>D	R	0	Clear
Leigh-like Subacute Necrotizing Encephalopathy (Discovered in the Yorkshire Terrier)	SLC19A3	D>I	R	0	Clear
Exercise-Induced Collapse	DNM1	G>T	R	0	Clear
Hemophilia A (Discovered in the German Shepherd Dog - Variant 2)	FVIII	G>A	S	0	Clear
Juvenile Laryngeal Paralysis and	RAB3GAP1	I>D	R	0	Clear

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Polyneuropathy	RAB3GAP1	I>D	R	0	Clear
Early-Onset Progressive Polyneuropathy (Discovered in the Alaskan Malamute)	NDRG1	G>T	R	0	Clear
Cerebral Dysfunction	SLC6A3	G>A	R	0	Clear
Von Willebrand's Disease, type 1	VWF	G>A	D	0	Clear
Von Willebrand's Disease, type 3 (Discovered in the Shetland Sheepdog)	VWF	I>D	R	0	Clear
Episodic Falling Syndrome	BCAN	D>I	R	0	Clear
gm2-gangliosidosis-poodle	HEXB	I>D	R	0	Clear
Alexander Disease	GFAP	G>A	R	0	Clear
Severe Combined Immunodeficiency	PRKDC	G>T	R	0	Clear
Benign Familial Juvenile Epilepsy	LGI2	A>T	R	0	Clear
Myotonia Congenita (Discovered in the Miniature Schnauzer)	CLCN1	C>T	R	0	Clear
Mucopolysaccharidosis Type VII (Discovered in the German Shepherd Dog)	GUSB	G>A	R	0	Clear
Spinocerebellar Ataxia with Myokymia and/or Seizures	KCNJ10	C>G	R	0	Clear
Junctional Epidermolysis Bullosa (Discovered in the Australian Cattle Dog Mix)	LAMA3	T>A	R	0	Clear
Lethal Acrodermatitis (Discovered in the Bull Terrier)	MKLN1	A>C	R	0	Clear
Neuroaxonal Dystrophy	TECPR2	C>T	R	0	Clear
Cystinuria Type II-A	SLC3A1	I>D	D	0	Clear
Limb-girdle Muscular Dystrophy, Type L3 (Discovered in the Miniature Dachshund)	SGCA	G>A	R	0	Clear
Cone-Rod Dystrophy 1	PDE6B	I>D	R	0	Clear
Pyruvate Kinase Deficiency (Discovered in the Basenji)	PKLR	I>D	R	0	Clear

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Polycystic Kidney Disease	PKD1	G>A	D	0	Clear
Hyperekplexia or Startle Disease	SLC6A5	G>T	R	0	Clear
Myotubular Myopathy	MTM1	A>C	S	0	Clear
Demyelinating Neuropathy	SBF2	G>T	R	0	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Komondor)	CUBN	G>A	R	0	Clear
Craniomandibular Osteopathy (Discovered in the Basset Hound)	SLC37A2	C>T	D	0	Clear
Glanzmann Thrombasthenia Type I	ITGA2B	C>T	R	0	Clear
Primary Lens Luxation	ADAMTS17	G>A	R	0	Clear
Musladin-Lueke Syndrome	ADAMTSL2	C>T	R	0	Clear
Shaking Puppy Syndrome (Discovered in the Border Terrier)	Confidential	G>A	R	0	Clear
Hereditary Elliptocytosis	SPTB	C>T	D	0	Clear
Juvenile Encephalopathy (Discovered in the Parson Russell Terrier)	Confidential	I>D	R	0	Clear
Osteogenesis Imperfecta (Discovered in the Beagle)	COL1A2	C>T	D	0	Clear
Junctional Epidermolysis Bullosa (Discovered in the Australian Shepherd)	LAMB3	A>G	R	0	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Central Asian Ovcharka)	COL7A1	C>T	R	0	Clear
X-Linked Tremors	PLP1	A>C	S	0	Clear
Juvenile Cataract (Discovered in the Wirehaired Pointing Griffon)	FYCO1	I>D	R	0	Clear
Muscular Dystrophy (Discovered in the Cavalier King Charles Spaniel)	Dystrophin	G>T	S	0	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Basset Hound)	COL7A1	D>I	R	0	Clear
Generalized Progressive Retinal Atrophy	CCDC66	D>I	R	0	Clear

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(Discovered in the Schapendoes)	CCDC66	D>I	R	0	Clear
Myotonia Congenita	CLCN1	D>I	R	0	Clear
griscelli	MYO5A	D>I	R	0	Clear
Neuroaxonal Dystrophy (Discovered in the Papillon)	PLA2G6	G>A	R	0	Clear
Hemophilia A (Discovered in the German Shepherd Dog - Variant 1)	FVIII	G>A	S	0	Clear
Familial Nephropathy, (Discovered in the English Springer Spaniel)	COL4A4	C>T	R	0	Clear
Pyruvate Dehydrogenase Phosphatase 1 Deficiency	PDP1	C>T	R	0	Clear
Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Golden Retriever)	CLN5	I>D	R	0	Clear
Muscular Dystrophy-Dystroglycanopathy (Discovered in the Labrador Retriever)	LARGE	C>T	R	0	Clear
X-Linked Ectodermal Dysplasia	EDA	G>A	S	0	Clear
Myotonia Congenita (Discovered in the Labrador Retriever)	CLCN1	T>A	R	0	Clear
Ligneous Membranitis	PLG	T>A	R	0	Clear
Pyruvate Kinase Deficiency (Discovered in the Beagle)	PKLR	G>A	R	0	Clear
Acral Mutilation Syndrome	GDNF	C>T	R	0	Clear
Skeletal Dysplasia 2	COL11A2	G>C	R	0	Clear
Prekallikrein Deficiency	KLKB1	T>A	R	0	Clear
Mucopolysaccharidosis VI (Discovered in the Miniature Pinscher)	ARSB	G>A	R	0	Clear
Glycogen Storage Disease Type IIIa, (GSD IIIa)	AGL	I>D	R	0	Clear
Rod-Cone Dysplasia 1a	PDE6B	D>I	R	0	Clear
Craniomandibular Osteopathy (Discovered	SLC35D1	I>D	D	0	Clear

GRETA THUMBERG - NIKE
Record: 12057
Razza: BORDER COLLIE



ID sample: DFLTVBD
Date test:04/11/2025
MyDogDNA - Vetogene
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DNA Test Report

Condizione genetica	Gene	Variante rischio	Ereditario	Copie	Risultato
in the Weimaraner)	SLC35D1	I>D	D	0	Clear
Hypomyelination	FNIP2	I>D	R	0	Clear
Persistent Müllerian Duct Syndrome	AMHR2	C>T	R	0	Clear
Deafness and Vestibular Dysfunction (DINGS2), (Discovered in Doberman Pinscher)	MYO7A	G>A	R	0	Clear
Pituitary Dwarfism (Discovered in the Karelian Bear Dog)	POU1F1	C>A	R	0	Clear
Ehlers-Danlos syndrome (Discovered in the Labrador Retriever)	COL5A1	I>D	D	0	Clear
Familial Nephropathy (Discovered in the English Cocker Spaniel)	COL4A4	A>T	R	0	Clear
Hereditary Ataxia (Discovered in the Belgian Malinois)	SLC12A6	D>I	R	0	Clear
Mucopolysaccharidosis Type VII (Discovered in the Brazilian Terrier)	GUSB	C>T	R	0	Clear
Macrothrombocytopenia	TUBB1	G>A	R	0	Clear
Cerebellar Hypoplasia	VLDLR	I>D	R	0	Clear
Cranio-mandibular Osteopathy	SLC37A2	C>T	D	0	Clear
Hereditary Vitamin D-Resistant Rickets Type II	VDR	I>D	R	0	Clear
Cone Degeneration (Discovered in the German Shepherd Dog)	CNGA3	C>T	R	0	Clear
Epidermolytic Hyperkeratosis	KRT10	G>T	R	0	Clear
P2RY12-associated Bleeding Disorder	P2RY12	I>D	R	0	Clear
Neuronal Ceroid Lipofuscinosis 1	PPT1	D>I	R	0	Clear
Hemophilia A (Discovered in the Boxer)	FVIII	C>G	S	0	Clear
X-Linked Progressive Retinal Atrophy 1	RPGR	I>D	S	0	Clear
Focal Non-Epidermolytic Palmoplantar Keratoderma	KRT16	G>C	R	0	Clear

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X-Linked Myotubular Myopathy	MTM1	C>A	S	0	Clear
Cone-Rod Dystrophy 2	IQCB1	D>I	R	0	Clear
Ichthyosis Type 2 (Discovered in the Golden Retriever)	ABHD5	I>D	R	0	Clear
Factor VII Deficiency	F7	G>A	R	0	Clear
Intestinal Lipid Malabsorption (Discovered in the Australian Kelpie)	ACSL5	I>D	R	0	Clear

Le analisi vengono svolte dal Laboratorio "Neogen Europe LTD" (Laboratorio certificato UKAS ISO/IEC 17025). Il profilo genetico riportato è standardizzato secondo le specifiche della Società Internazionale di Genetica Animale (ISAG)

L'/Gli esame/i è/sono stato/i condotto/i secondo l'attuale conoscenza scientifica generale sulle mutazioni genetiche testate.

Il presente referto riguarda solo il campione sottoposto a prova. I risultati si riferiscono al campione così come ricevuto ed il laboratorio declina ogni responsabilità sui dati dell'oggetto sottoposto ad analisi forniti dal committente. Il referto non può essere riprodotto parzialmente senza l'approvazione preventiva, in forma scritta, da parte del Laboratorio Vetogene - ENCI Servizi.

Il Medico Veterinario che ha firmato il modulo di certificazione allegato al campione, conferma l'identità dell'animale e di aver prelevato il campione dal soggetto identificato.