

Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946

## Lilly's Profile

#### Pet information

Registered name

Date of birth

Lilly

2024-05-21

Sex

Spayed

No

### Top breeds

75% Poodle (Toy and Miniature) 24% Golden Retriever 1% Bichon Frise

### Predicted ideal adult weight

22-35 lbs

### Health summary

At Risk 1 condition

• Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD) Risk

Carrier O conditions

Clear 266 conditions



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## Breed ancestry

We detected 3 breeds in Lilly's DNA.



### Companion



 $75\,\%\,$  Poodle (Toy and Miniature)

1% Bichon Frise

### **Sporting**



24 % Golden Retriever

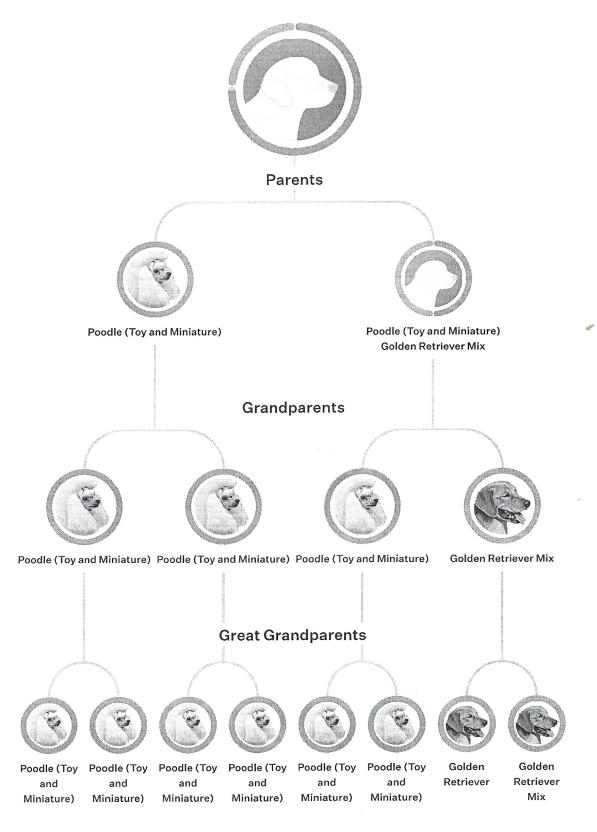
ROYAL CANIN

GENETIC HEALTH,

Analysis:

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### Family Tree





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### Lilly's predicted ideal adult weight

Based on our findings, we've calculated that Lilly's ideal adult weight should be 22-35 lbs.

Ideal Weight 22-35 lbs

Size Medium

We've factored everything we know about Lilly in predicting a healthy, adult weight. However environmental factors such as the nutrition of Lilly's mom during pregnancy and nursing, Lilly's nutrition during critical growth months, illness/parasites/ticks/fleas, and exercise levels can affect the actual weight of Lilly.

#### Calculating weight

Our weight-predictive algorithm uses a combination of the following to calculate Lilly's ideal, adult weight: The published weight ranges of more than 200 purebred dogs. The observed weights of purebred dogs, each with an ideal Body ConditionScore, from the Banfield® Pet Hospital database. Breeds the WISDOM PANEL™ test analysis has identified that reflect a dog's true heritage and genetic complexity. A genetic algorithm based on mixed-breed data that calculates the contribution of each set of chromosomal genetic markers.

### Environmental effects on weight

A dog's early life is very important in determining how they will grow and develop. They can fail to reach their ideal weight for a number of reasons, including the diet of their mother during pregnancy and nursing (as well as their own diet as puppies). Illness and disease can play a part too, as can having parasites like roundworms or fleas and ticks. For dogs who are adopted after they are fully-grown, it may be harder to find the historical background on these factors. Maintaining a healthy weight is a key factor in Lilly having a long and healthy life.



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## Genetic Diversity

### Heterozygosity

### Lilly's Percentage of Heterozygosity

45%

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

### Typical Range for Mixed-Breed Dogs

33% - 46%



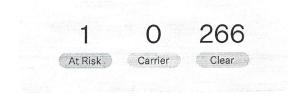
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### Summary of health conditions

#### **Key Findings**

We detected 1 genetic condition in Lilly's DNA.



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

#### What this means for Lilly



#### Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD) Risk

Lilly has one copy of the Chondrodystrophy and Intervertebral Disc Disease Risk variant and is at an elevated risk for being diagnosed with this condition. Lilly may show the first signs of this condition at birth. When compared to dogs without the variant, Lilly's legs will likely be slightly shorter. Lilly may also have early degeneration and calcification of her spinal discs which would predispose her to disc herniation (also known as having a "slipped" disc). The age for being diagnosed with disc herniation varies greatly, with averages ranging from 3 to 10 years. And, the risk for back surgery in dogs with one or two copies of this variant ranges from 5 to 15 times more likely. Although, additional considerations, like breed background and environmental factors, also impact overall risk level. One of the initial signs of disc herniation includes back pain, which may appear as generalized stiffness, shivering or shaking, muscle spasms, hesitation to move, or vocalization when being touched. Other clinical signs include lameness, limb weakness, incoordination when walking, or the inability to walk. Some affected dogs may also show fecal or urinary incontinence. If signs are observed, Lilly should be examined by a veterinarian as soon as possible to determine underlying cause and best course of treatment. Depending on severity, treatment may focus on medical intervention or surgical intervention, with both approaches including strict crate rest. Please note this variant is a risk factor and some dogs with one, or even two, copies of this variant may not go on to show signs of disc disease. Additionally, not all dogs affected by IVDD have this variant, indicating additional genetic or environmental causes for disc disease.



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### Health conditions tested

#### At-risk and carrier conditions (1)

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

#### 4 What is it

Chondrodystrophy (CDDY) is a skeletal disorder characterized by shortened limbs and abnormal early degeneration of the spinal discs, or intervertebral disc disease (IVDD), which predisposes to disc herniation.

#### What it means

Lilly has one copy of the Chondrodystrophy and Intervertebral Disc Disease Risk variant and is at an elevated risk for being diagnosed with this condition. Lilly may show the first signs of this condition at birth. When compared to dogs without the variant, Lilly's legs will likely be slightly shorter. Lilly may also have early degeneration and calcification of her spinal discs which would predispose her to disc herniation (also known as having a "slipped" disc). The age for being diagnosed with disc herniation varies greatly, with averages ranging from 3 to 10 years. And, the risk for back surgery in dogs with one or two copies of this variant ranges from 5 to 15 times more likely. Although, additional considerations, like breed background and environmental factors, also impact overall risk level.

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Please note this variant is a risk factor and some dogs with one, or even two, copies of this variant may not go on to show signs of disc disease. Additionally, not all dogs affected by IVDD have this variant, indicating additional genetic or environmental causes for disc disease.

#### ₩ What to do

#### Here's how to care for a dog with CDDY and IVDD risk

Partner with your veterinarian to make a plan regarding your dog's well-being, including any insights provided through genetic testing. If your pet is at risk or is showing signs of this disorder, then the first step is to speak with your veterinarian.



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



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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Centronuclear Myopathy (Discovered in the Labrador Retriever)	PTPLA	Insertion	0	AR	Clear
Cerebellar Ataxia	RAB24	A>C	0	AR	Clear
Cerebellar Cortical Degeneration	SNX14	C>T	0	AR	Clear
Cerebellar Hypoplasia	VLDLR	Deletion	0	AR	Clear
Cerebral Dysfunction	SLC6A3	G>A	0	AR	Clear
Chondrodysplasia (Discovered in Norwegian Elkhound and Karelian Bear Dog)	ITGA10	C>T	0	AR	Clear
Cleft Lip & Palate with Syndactyly	ADAMTS20	Deletion	0	AR	Clear
Cleft Palate	DLX6	C>A	0	AR	Clear
CNS Atrophy with Cerebellar Ataxia (Discovered in the Belgian Shepherd)	SEPP1	Deletion	0	AR	Clear
Coat Color Dilution and Neurological Defects (Discovered n the Miniature Dachshund)	MYO5A	Insertion	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
one-Rod Dystrophy 2	IQCB1	Insertion	0	AR	Clear
ongenital Cornification (Discovered in the Labrador etriever)	NSDHL	Deletion	0	SD	Clear
congenital Dyshormonogenic Hypothyroidism with Goiter Discovered in the Shih Tzu)	SLC5A5	G>A	0	AR	Clear



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Congenital Eye Malformations (Discovered in the Golden Retriever)	SIX6	C>T	0	AD	Clear
Congenital Hypothyroidism (Discovered in the Tenterfield Terrier)	TPO	C>T	0 .	AR	Clear
Congenital Hypothyroidism (Discovered in the Toy Fox and Rat Terrier)	TPO	C>T	0	AR	Clear
Congenital Muscular Dystrophy (Discovered in the Italian Greyhound)	LAMA2	G>A	0	AR	Clear
Congenital Muscular Dystrophy (Discovered in the Staffordshire Bull Terrier)	LAMA2	Deletion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Golden Retriever)	COLQ	G>A	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Heideterrier)	CHRNE	Insertion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Jack Russell Terrier)	CHRNE	Insertion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Labrador Retriever)	COLQ	T>C	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Old Danish Pointer)	CHAT	G>A	0	AR	Clear
Congenital Stationary Night Blindness (CSNB)	RPE65	A>T	0	AR	Clear
Craniomandibular Osteopathy (Discovered in Scottish Terrier breeds)	SLC37A2	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Australian Terrier)	COL1A1	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Basset Hound)	SLC37A2	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Weimaraner)	SLC35D1	Deletion	0	AD	Clear
Cystic Renal Dysplasia and Hepatic Fibrosis	INPP5E	G>A	0	AR	Clear
Cystinuria Type I-A	SLC3A1	C>T	0	AR	Clear
Cystinuria Type II-A	SLC3A1	Deletion	0	AD	Clear



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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Darier Disease (Discovered in the Irish Terrier)	ATP2A2	Insertion	0	AD	Clear
Deafness and Vestibular Dysfunction (DINGS1), (Discovered in Doberman Pinscher)	PTPRQ	Insertion	0	AR	Clear
Deafness and Vestibular Dysfunction (DINGS2), (Discovered in Doberman Pinscher)	МҮО7А	G>A	0	AR	Clear
Degenerative Myelopathy	SOD1	G>A	0	AR	Clear
Demyelinating Neuropathy	SBF2	G>T	0	AR	Clear
Dental Hypomineralization	FAM20C	C>T	0	AR	Clear
Dental-Skeletal-Retinal Anomaly (Discovered in the Cane Corso)	MIA3	I>S	0	AR	Clear
Dilated Cardiomyopathy (Discovered in the Schnauzer)	RBM20	Deletion	0	AR	Clear
Disproportionate Dwarfism (Discovered in the Dogo Argentino)	PRKG2	C>A	0	AR	Clear
Dominant Progressive Retinal Atrophy	RHO	C>G	0	AD	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Basset Hound)	COL7A1	Insertion	0	AR	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Central Asian Ovcharka)	COL7A1	C>T	0	AR	Clear
Dystrophic Epidermolysis Bullosa (Discovered in the Golden Retriever)	COL7A1	C>T	0	AR	Clear
Early Retinal Degeneration (Discovered in the Norwegian Elkhound)	STK38L	Insertion	0	AR	Clear
Early-Onset Adult Deafness (Discovered in the Rhodesian Ridgeback)	EPS8L2	Deletion	0	AR	Clear
Early-Onset Progressive Polyneuropathy (Discovered in the Alaskan Malamute)	NDRG1	G>T	0	AR	Clear
Early-Onset Progressive Polyneuropathy (Discovered in the Greyhound)	NDRG1	Deletion	0	AR	Clear
Early-Onset Progressive Retinal Atrophy (Discovered in the Portuguese Water Dog)	Confidential		0	AR	Clear



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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Early-Onset Progressive Retinal Atrophy, (Discovered in the Spanish Water Dog)	PDE6B	Deletion	0	AR	Clear
Ehlers-Danlos Syndrome (Discovered in mixed breed)	COL5A1	G>A	0	AD	Clear
Ehlers-Danlos Syndrome (Discovered in the Labrador Retriever)	COL5A1	Deletion	0	AD	Clear
Epidermolytic Hyperkeratosis	KRT10	G>T	0	AR	Clear
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
Familial Nephropathy (Discovered in the English Cocker Spaniel)	COL4A4	A>T	0	AR	Clear
Familial Nephropathy (Discovered in the English Springer Spaniel)	COL4A4	C>T	0	AR	Clear
Fanconi Syndrome	FAN1	Deletion	0	AR	Clear
Fetal Onset Neuroaxonal Dystrophy	MFN2	G>C	0	AR	Clear
Focal Non-Epidermolytic Palmoplantar Keratoderma	KRT16	G>C	0	AR	Clear
Generalized Progressive Retinal Atrophy (Discovered in he 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 preed dogs)	ITGA2B	C>T	0	AR	Clear
Globoid Cell Leukodystrophy (Discovered in Terriers)	GALC	A>C	0	AR	Clear
aloboid Cell Leukodystrophy (Discovered in the Irish setter)	GALC	A>T	0	AR	Clear
Glycogen Storage Disease Type Ia (Discovered in the German Pinscher)	G6PC	Insertion	0	AR	Clear



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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Resul
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	O	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	SR	Clear
Hemophilia A (Discovered in the Boxer)	FVIII	C>G	0	SR	Clear
Hemophilia A (Discovered in the German Shepherd Dog - Variant 1)	FVIII	G>A	0	SR	Clear
Hemophilia A (Discovered in the German Shepherd Dog - Variant 2)	FVIII	G>A	0	SR	Clear
Hemophilia A (Discovered in the Havanese)	FVIII	Insertion	0	SR	Clear
Hemophilia A (Discovered in the Labrador Retriever)	Confidential	-	0	SR	Clear
Hemophilia B	FIX	G>A	0	SR	Clear
Hemophilia B (Discovered in the Airedale Terrier)	FIX	Insertion	0	SR	Clear
Hemophilia B (Discovered in the Lhasa Apso)	FIX	Deletion	0	SR	Clear
Hereditary Ataxia (Discovered in the Belgian Malinois)	SLC12A6	Insertion	0	AR	Clear
Hereditary Ataxia (Discovered in the Norwegian Buhund)	KCNIP4	T>C	0	AR	Clear
Hereditary Calcium Oxalate Urolithiasis, Type 1	Confidential	=	0	AR	Clear
Hereditary Elliptocytosis	SPTB	C>T	0	AD	Clear
Hereditary Footpad Hyperkeratosis	FAM83G	G>C	0	AR	Clear
Hereditary Nasal Parakeratosis (Discovered in the Greyhound)	SUV39H2	Deletion	0	AR	Clear



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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Resul
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
Hyperuricosuria	SLC2A9	G>T	0	AR	Clear
Hypocatalasia	CAT	G>A	0	AR	Clear
Hypomyelination	FNIP2	Deletion	0	AR	Clear
- Hypophosphatasia	Confidential		0	AR	Clear
chthyosis (Discovered in the American Bulldog)	NIPAL4	Deletion	0	AR	Clear
chthyosis (Discovered in the Great Dane)	SLC27A4	G>A	0	AR	Clear
chthyosis Type 2 (Discovered in the Golden Retriever)	ABHD5	Deletion	0	AR	Clear
nflammatory Myopathy (Discovered in the Dutch Shepherd Dog)	SLC25A12	A>G	0	AR	Clear
nflammatory Pulmonary Disease (Discovered in the Rough Collie)	AKNA	Deletion	0	AR	Clear
ntestinal Cobalamin Malabsorption (Discovered in the leagle)	CUBN	Deletion	0	AR	Clear
ntestinal Cobalamin Malabsorption (Discovered in the corder Collie)	CUBN	Deletion	0	AR	Clear
ntestinal Cobalamin Malabsorption (Discovered in the comondor)	CUBN	G>A	0	AR	Clear
ntestinal Lipid Malabsorption (Discovered in the australian Kelpie)	ACSL5	Deletion	0	AR	Clear
unctional Epidermolysis Bullosa (Discovered in the ustralian Cattle Dog Mix)	LAMA3	T>A	0	AR	Clear
unctional Epidermolysis Bullosa (Discovered in the ustralian Shepherd)	LAMB3	A>G	0	AR	Clear
uvenile Cataract (Discovered in the Wirehaired Pointing riffon)	FYCO1	Deletion	0	AR	Clear
uvenile Dilated Cardiomyopathy (Discovered in the Toy lanchester Terrier)	Confidential	-	0	AR	Clear



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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Resu
Juvenile Encephalopathy (Discovered in the Parson Russell Terrier)	Confidential		0	AR	Clea
Juvenile Laryngeal Paralysis and Polyneuropathy	RAB3GAP1	Deletion	0	AR	Clea
Juvenile Myoclonic Epilepsy	DIRAS1	Deletion	0	AR	Clea
L-2-Hydroxyglutaric aciduria (Discovered in the Staffordshire Bull Terrier)	L2HGDH	T>C	0	AR	Clea
L-2-Hydroxyglutaric Aciduria (Discovered in the West Highland White Terrier)	Confidential	-	0	AR	Clea
Lagotto Storage Disease	ATG4D	G>A	0	AR	Clea
_amellar Ichthyosis	TGM1	Insertion	0	AR	Clea
aryngeal Paralysis (Discovered in the Bull Terrier and Miniature Bull Terrier)	RAPGEF6	Insertion	0	AR	Clea
eigh-like Subacute Necrotizing Encephalopathy Discovered in the Yorkshire Terrier)	SLC19A3	Insertion	0	AR	Clea
ethal Acrodermatitis (Discovered in the Bull Terrier)	MKLN1	A>C	0	AR	Clea
eukodystrophy (Discovered in the Standard Schnauzer)	TSEN54	C>T	0	AR	Clea
igneous Membranitis	PLG	T>A	0	AR	Clea
imb-girdle Muscular Dystrophy (Discovered in the Boston Terrier)	SGCD		0	AR	Clea
imb-girdle Muscular Dystrophy, Type L3 (Discovered in he Miniature Dachshund)	SGCA	G>A	0	AR	Clea
ung Developmental Disease (Discovered in the Airedale errier)	LAMP3	C>T	0	AR	Clea
Nacrothrombocytopenia (Discovered in Norfolk and Cairn Ferrier)	TUBB1	G>A	0	AR	Clea
lay-Hegglin Anomaly	МҮН9	G>A	0	AD	Clea
IDR1 Medication Sensitivity	MDR1/ABCB1	Deletion	0	AD	Clea
dicrophthalmia (Discovered in the Soft-Coated Wheaten errier)	RBP4	Deletion	0	AR	Clea



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Mucopolysaccharidosis Type IIIA (Discovered in the Dachshund)	SGSH	C>A	0	AR	Clear
Mucopolysaccharidosis Type IIIA (Discovered in the New Zealand Huntaway)	SGSH	Insertion	0	AR	Clear
Mucopolysaccharidosis Type VII (Discovered in the Brazilian Terrier)	GUSB	C>T	0	AR	Clear
Mucopolysaccharidosis Type VII (Discovered in the German Shepherd Dog)	GUSB	G>A	0	AR	Clear
Mucopolysaccharidosis VI (Discovered in the Miniature Pinscher)	ARSB	G>A	0	AR	Clear
Muscular Dystrophy (Discovered in the Cavalier King Charles Spaniel)	Dystrophin	G>T	0	SR	Clear
Muscular Dystrophy (Discovered in the Golden Retriever)	Dystrophin	A>G	0	SR	Clear
Muscular Dystrophy (Discovered in the Landseer)	COL6A1	G>T	0	AR	Clear
Muscular Dystrophy (Discovered in the Norfolk Terrier)	Dystrophin	Deletion	0	SR	Clear
Muscular Dystrophy-Dystroglycanopathy (Discovered in the Labrador Retriever)	LARGE	C>T	0	AR	Clear
Muscular Hypertrophy (Double Muscling)	MSTN	T>A	0	AR	Clear
Musladin-Lueke Syndrome	ADAMTSL2	C>T	0	AR	Clear
Myeloperoxidase Deficiency	MOP	C>T	0	AR	Clear
Myotonia Congenita (Discovered in Australian Cattle Dog)	CLCN1	Insertion	0	AR	Clear
Myotonia Congenita (Discovered in the Labrador Retriever)	CLCN1	T>A	0	AR	Clear
Myotonia Congenita (Discovered in the Miniature Schnauzer)	CLCN1	C>T	0	AR	Clear
Myotubular Myopathy	MTM1	A>C	0	SR	Clear
Narcolepsy (Discovered in the Dachshund)	HCRTR2	G>A	0	AR	Clear
Narcolepsy (Discovered in the Labrador Retriever)	HCRTR2	G>A	0	AR	Clear



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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
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 5 (Discovered in the Border Collie)	CLN5	C>T	0	AR	Clear
Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Golden Retriever)	CLN5	_	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
Osteochondromatosis (Discovered in the American Staffordshire Terrier)	EXT2	C>A	0	AR	Clear
Osteogenesis Imperfecta (Discovered in the Beagle)	COL1A2	C>T	0	AD	Clear



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Resu
Osteogenesis Imperfecta (Discovered in the Dachshund)	SERPINH1	T>C	0	AR	Clea
P2RY12-associated Bleeding Disorder	P2RY12	Deletion	0	AR	Clea
Palmoplantar Hyperkeratosis (Discovered in the Rottweiler)	DSG1	Deletion	0	AR	Clea
Paroxysmal Dyskinesia	PIGN	C>T	0	AR	Clea
Persistent Müllerian Duct Syndrome	AMHR2	C>T	0	AR	Clea
Phosphofructokinase Deficiency	PFKM	G>A	0	AR	Clea
Pituitary Dwarfism (Discovered in the Karelian Bear Dog)	POU1F1	C>A	0	AR	Clea
Polycystic Kidney Disease	PKD1	G>A	0	AD	Clea
Prekallikrein Deficiency	KLKB1	T>A	0	AR	Clea
Primary Ciliary Dyskinesia	CCDC39	C>T	0	AR	Clea
Primary Ciliary Dyskinesia (Discovered in the Alaskan Malamute)	NME5	Deletion	0	AR	Clea
Primary Lens Luxation	ADAMTS17	G>A	0	AR	Clea
Primary Open Angle Glaucoma (Discovered in Basset Fauve de Bretagne)	ADAMTS17	G>A	0	AR	Clea
Primary Open Angle Glaucoma (Discovered in Petit Basset Griffon Vendeen)	ADAMTS17	Insertion	0	AR	Clea
Primary Open Angle Glaucoma and Lens Luxation Discovered in Chinese Shar-Pei)	ADAMTS17	Deletion	0	AR	Clea
Progressive Early-Onset Cerebellar Ataxia	SEL1L	T>C	0	AR	Clea
Progressive Retinal Atrophy (Discovered in the Basenji)	SAG	T>C	0	AR	Clea
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA 2 variant)	TTC8	Deletion	0	AR	Clea
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA1 variant)	SLC4A3	Insertion	0	AR	Clea
Progressive Retinal Atrophy (Discovered in the Lapponian	IFT122	C>T	0	AR	Clea



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Progressive Retinal Atrophy (Discovered in the Lhasa Apso)	Confidential	-	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Papillon and Phalène)	CNGB1	Deletion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - BBS2 variant)	Confidential	-	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - CNGA1 variant)	CNGA1	Deletion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Swedish Vallhund)	MERTK	Insertion	0	AR	Clear
Progressive Retinal Atrophy 1 (Discovered in the Italian Greyhound)	Confidential	**************************************	0	AR	Clear
Progressive Retinal Atrophy Type III	FAM161A	Insertion	0	AR	Clear
Protein Losing Nephropathy	NPHS1	G>A	Ö	AR	Clear
Pyruvate Dehydrogenase Phosphatase 1 Deficiency	PDP1	C>T	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the Basenji)	PKLR	Deletion	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the Beagle)	PKLR	G>A	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the Pug)	PKLR	T>C	0	AR	Clear
Pyruvate Kinase Deficiency (Discovered in the West Highland White Terrier)	PKLR	Insertion	0	AR	Clear
QT Syndrome	KCNQ1	C>A	0	AD	Clear
Renal Cystadenocarcinoma and Nodular Dermatofibrosis	FLCN	A>G	0	AD	Clear
Rod-Cone Dysplasia 1	PDE6B	G>A	0	AR	Clear
Rod-Cone Dysplasia 1a	PDE6B	Insertion	0	AR	Clear
Rod-Cone Dysplasia 3	PDE6A	Deletion	0	AR	Clear
Sensorineural Deafness (Discovered in the Rottweiler)	LOXHD1	G>C	0	AR	Clear
Sensory Ataxic Neuropathy	tRNATyr	Deletion	0	MT	Clear



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Sensory Neuropathy	FAM134B	Insertion	0	AR	Clear
Severe Combined Immunodeficiency (Discovered in Frisian Water Dogs)	RAG1	G>T	0	AR	Clear
Severe Combined Immunodeficiency (Discovered in Russell Terriers)	PRKDC	G>T	0	AR	Clear
Shaking Puppy Syndrome (Discovered in the Border Terrier)	Confidential		0	AR	Clear
Skeletal Dysplasia 2	COL11A2	G>C	0	AR	Clear
Spinocerebellar Ataxia (Late-Onset Ataxia)	CAPN1	G>A	0	AR	Clear
Spinocerebellar Ataxia with Myokymia and/or Seizures	KCNJ10	C>G	0	AR	Clear
Spondylocostal Dysostosis	HES7	Deletion	0	AR	Clear
Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA1)	KCNJ10	T>C	0	AR	Clear
Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA2)	ATP1B2	Insertion	0.	AR	Clear
Stargardt Disease (Discovered in the Labrador Retriever)	ABCA4	Insertion	0	AR	Clear
Startle Disease (Discovered in Irish Wolfhounds)	SLC6A5	G>T	0	AR	Clear
Startle Disease (Discovered in the Miniature American Shepherd)	Confidential	_	0	AR	Clear
Succinic Semialdehyde Dehydrogenase Deficiency (Discovered in the Saluki)	ALDH5A1	G>A	0	AR	Clear
Thrombopathia (Discovered in the Basset Hound)	RASGRP1	Deletion	0	AR	Clear
Thrombopathia (Discovered in the Eskimo Spitz)	RASGRP1	<u> </u>	0	AR	Clear
Trapped Neutrophil Syndrome	VPS13B	Deletion	0	AR	Clear
√an den Ende-Gupta Syndrome	SCARF2	Deletion	0	AR	Clear
von Willebrand's Disease, type 1	VWF	G>A	0	AR	Clear
on Willebrand's Disease, type 2	VWF	T>G	0	AR	Clear



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



-			Diele Venient	Coning	Inheritance	Result
	Genetic Condition	Gene	Risk Variant	Copies	illieritance	Nesult
	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	SR	Clear
	X-Linked Hereditary Nephropathy (Discovered in the Navasota Dog)	COL4A5	Deletion	0	SR	Clear
	X-Linked Hereditary Nephropathy (Discovered in the Samoyed)	COL4A5	G>T	0	SR	Clear
	X-Linked Myotubular Myopathy	MTM1	C>A	0	SR	Clear
	X-Linked Progressive Retinal Atrophy 1	RPGR	Deletion	0	SR	Clear
	X-Linked Progressive Retinal Atrophy 2	RPGR	Deletion	0	SR	Clear
	X-Linked Severe Combined Immunodeficiency (Discovered in the Basset Hound)	IL2RG	Deletion	0	SR	Clear
	X-Linked Severe Combined Immunodeficiency (Discovered in the Cardigan Welsh Corgi)	IL2RG	Insertion	0	SR	Clear
	X-Linked Tremors	PLP1	A>C	0	SR	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



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



### **Traits**

### **Coat Color**

	Gene	Variant	Copies	Result
Fawn	ASIP	ау	0	No effect
Recessive Black	ASIP	а	0	No effect
Tan Points	ASIP	at	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	KΒ	1	Black or brindle 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.				
Mask	MC1R	Em	0	No effect
Recessive Red (e1)	MC1R	e <sup>1</sup>	2	Cream to red coat likely
To show a solid red coat, a dog must inherit two copies of a Recessive Red variant, one from each parent. This can either be two copies of a particular variant, such as this one (e1) or two of any combination of recessive red variants. Recessive red coats will appear white, cream, yellow or red, although there are other variants that can result in a similar appearance. The amount of red pigment in the coat, called the intensity, is governed by other genes.				
Recessive Red (e2)	MC1R	e²	0	No effect
Recessive Red (e3)	MC1R	e <sup>3</sup>	0	No effect
Sable (Discovered in the Cocker Spaniel)	MC1R	ен	0	No effect
Widow's Peak (Discovered in Ancient dogs)	MC1R	e <sup>A</sup>	0	No effect
Widow's Peak (Discovered in the Afghan Hound and Saluki)	MC1R	EG	0	No effect

### **Color Modification**

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



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



### **Color Modification**

	Gene	Variant	Copies	Result
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.				
Dilution (d1) Linkage test	MLPH	d¹	0	No effect
Dilution (d2)	MLPH	d²	0	No effect
Dilution (d3)	MLPH	d3	0	No effect
Chocolate (basd)	TYRP1	þasd	0	No effect
Chocolate (bc)  To show chocolate coloration a dog must inherit two chocolate variants, one from each parent. This can either be two copies of a particular variant, such as this one ("bc"), or two of any combination of chocolate variants.	TYRP1	рс	1	Black features likely, chocolate possible
Chocolate (bd)	TYRP1	b₫	0	No effect
Chocolate (be)	TYRP1	b <sub>e</sub>	0	No effect
Chocolate (bh)	TYRP1	þh	0	No effect
Chocolate (bs)	TYRP1	bs	0	No effect

### **Coat Patterns**

	Gene	Variant	Copies	Result
Piebald	MITF	Sp	0	No effect
Merle	PMEL	М	_	Inconclusive
Harlequin	PSMB7	Н	0	No effect
Saddle Tan	RALY	·-	0	No effect
Roan (Linkage test)	USH2A	Tr	0	No effect



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



### Coat Length and Curl

	Gene	Variant	Copies	Result
Long Hair (Ih1)	FGF5	lh1	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 (lh2)	FGF5	lh²	0	No effect
Long Hair (lh3)	FGF5	lh³	0	No effect
Long Hair (lh4)	FGF5	lh⁴	0	No effect
Long Hair (lh5)	FGF5	lh <sup>5</sup>	0	No effect
Curly Coat	KRT71	С	1	Soft curl or wave likely
One copy of this variant is likely to give a soft curl or wave whereas two copies are likely to give a tighter curl. A curly coat is less apparent in dogs with short hair than those with long. There is one other known Curl variant, and likely other unknown variants that exist.				

### Hairlessness

	Gene	Variant	Copies	Result
Hairlessness (Discovered in the Chinese Crested Dog) Linkage test	FOXI3	Hrcc	0	No effect
Hairlessness (Discovered in the American Hairless Terrier)	SGK3	hraht	0	No effect
Hairlessness (Discovered in the Scottish Deerhound)	SKG3	hrsd	0	No effect

### Shedding

	Gene	Variant	Copies	Result	
Reduced Shedding	MC5R	sd	2	Low shedder	
One or two copies of the Reduced Shedding variant is likely to reduce a dog's tendency to shed. Copies of the					
Furnishings variant, particularly two, also reduce the tendency of a dog to shed.		4,000			



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### **More Coat Traits**

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

### **Head Shape**

	Gene	Variant	Copies	Result
Short Snout (BMP3 variant)	вмР3	- -	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	- 1	0	No effect	

### Ears

	Gene	Variant	Copies	Result
Floppy Ears	MSRB3		1	Partially floppy ears more
Dogs with zero copies of this variant are more likely to have				likely
permanently upright or prick ears, and fully folded ears are				
more likely with two copies inherited. Please note however				
that many genetic variants influence ear carriage. Dogs with				
some cartilage stiffness to their ears can sometimes raise				
their ears upright when 'at alert' but will flop down when				
relaxed.				



Owner: Linda Bennett Veterinarian: Karen Degiorgis Vet address: PO BOX 5 KEENE VA 22946



### Extra Toes

Extra loes					
	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	

	Gene	Variant	Copies	Result
Back Muscle and Bulk	ACSL4		0	No effect
High Altitude Adaptation	EPAS1	-	0	No effect
Short Legs (Chondrodysplasia, CDPA)	FGF4		0	No effect
Short Legs (Chondrodystrophy, CDDY)  Dogs with one copy of the Short Legs (CDDY) variant typically have some shortening of their legs, whereas dogs with two copies can have more obvious shortening. Dogs that inherit both variants associated with short legs (CDDY and CDPA) tend to show a more drastic reduction in leg length.	FGF4	-	1	Slightly shortened legs likely



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#### Inheritance Mode Key

#### Autosomal Recessive (AR)

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 (AD)

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 (SR)

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.

#### X-linked Dominant (SD)

The trait is carried on a sex chromosome and the trait is expressed when one of the alleles (inherited from mother or father) is damaged (contains detrimental mutation). Only one single mutated allele already could cause the disease (unwanted trait). 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. Homozygotes in this case may be at higher risk or show a more severe form of the disease than heterozygotes. Males carry only one copy of a gene: they could be normal homozygote or affected homozygote.

#### Mitochondrial (MT)

Rather than genomic DNA, the trait is associated with mitochondrial DNA (mtDNA) of which there are thousands within each cell of the body. For disease (unwanted trait) to occur, a certain ratio of mtDNA, inherited only from mother, must contain the detrimental mutation compared to normal mtDNA.

#### Modifier (MO)

Genetic modifiers do not cause disease (unwanted trait) on their own. It is only when inherited in combination with specific detrimental mutations, the trait expression can be further influenced by the presence of a genetic modifier—either increasing likelihood of disease or the severity of a disease. It is dependent on the genetic modifier as to if heterozygotes or homozygotes will influence the trait expression.