

Test Date: 04/12/2024

Veterinarian: Rena Dunahoo

Ruby Mea’s Profile

Pet information

Registered name	Date of birth
Ruby Mea	05/06/2023
Sex	Spayed
F	No

Top breeds

100% Labrador Retriever

Predicted ideal adult weight

42-71 lbs

Health summary

- At Risk

1 condition
 - Stargardt Disease (Discovered in the Labrador Retriever)
- Carrier

0 conditions
- Clear

266 conditions

Ruby Mea

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Breed ancestry

Ruby Mea appears to be 100% Labrador Retriever.

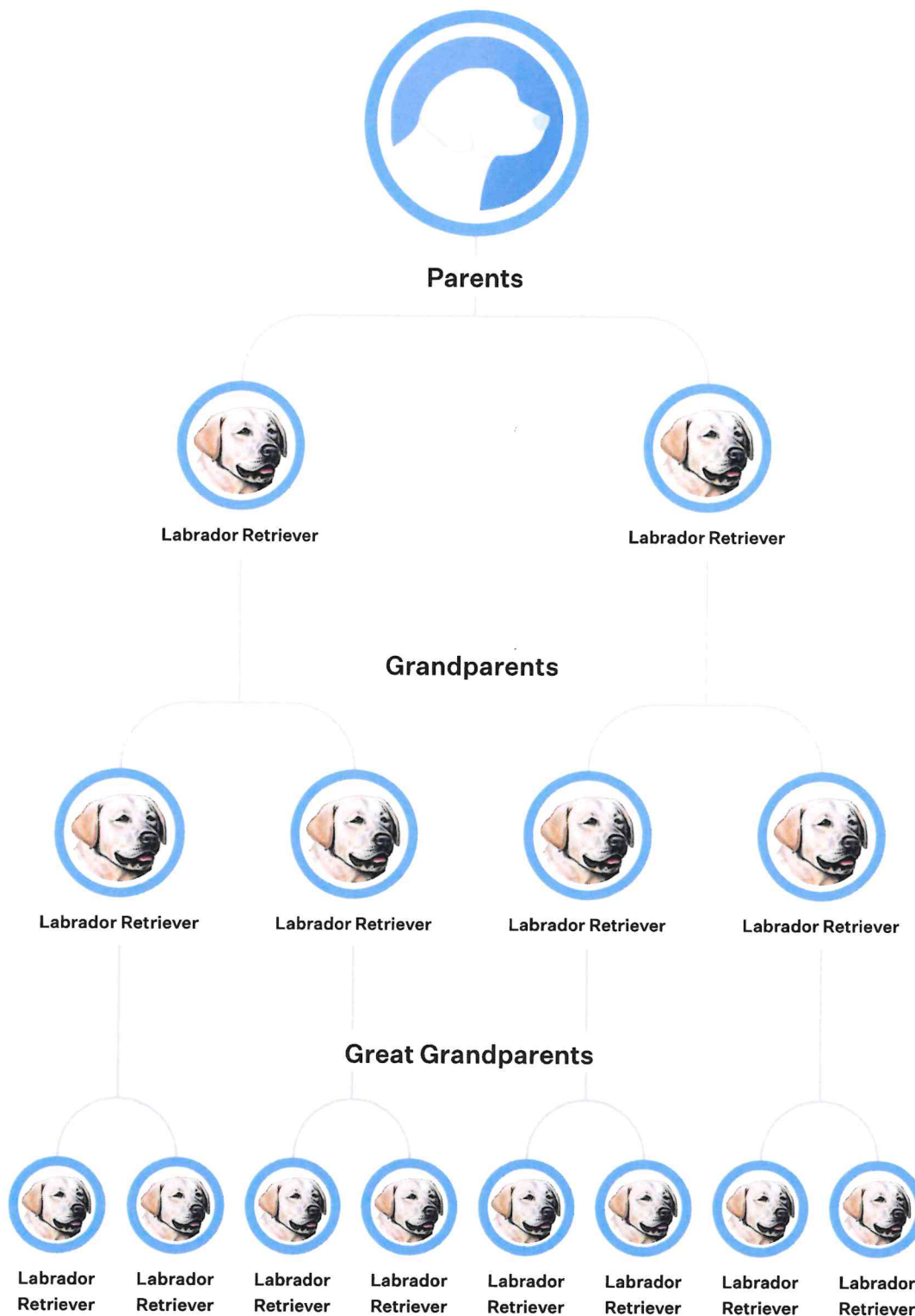


Sporting



100 % Labrador Retriever

Family Tree



Ruby Mea's predicted ideal adult weight

Based on our findings, we've calculated that Ruby Mea's ideal adult weight should be 42-71 lbs.

Ideal Weight
42-71 lbs

Size
Medium

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

Calculating weight

Our weight-predictive algorithm uses a combination of the following to calculate Ruby Mea'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 Condition Score, 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 Ruby Mea having a long and healthy life.

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Genetic Diversity

Heterozygosity

Ruby Mea’s Percentage of Heterozygosity

30%

This may make her more susceptible to genetic health complications when compared with other Labrador Retrievers.

Typical Range for Labrador Retrievers

31% - 40%

Summary of health conditions

Key Findings

We detected 1 genetic condition in Ruby Mea’s DNA.



Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Stargardt Disease (Discovered in the Labrador Retriever)	ABCA4	Insertion	2	AR	At Risk

What this means for Ruby Mea

At Risk **Stargardt Disease (Discovered in the Labrador Retriever)**

Two copies of the Stargardt Disease (Discovered in the Labrador Retriever) mutation are needed for this disease to occur so Ruby Mea is likely to show signs of this disorder. Stargardt Disease is a late onset disorder, so Ruby Mea may not start to show signs until their senior years. You may notice that Ruby Mea initially has difficulties navigating in the dark, but as the condition progresses Ruby Mea's level of vision will decrease and may lead to complete blindness. Affected dogs usually adjust well to their normal surroundings despite the loss of sight. As Ruby Mea's sight worsens, keeping the environment stable/familiar may help Ruby Mea to get around safely. However it is likely that Ruby Mea will need assistance in dark and unfamiliar places.

Health conditions tested

At-risk and carrier conditions (1)

Stargardt Disease (Discovered in the Labrador Retriever)	Gene	Risk Variant	Copies	Inheritance	Result
	ABCA4	Insertion	2	AR	At Risk

What is it

Stargardt Disease (STGD) is a form of late onset retinal degeneration, resulting in loss of the light detecting cells at the back of the eye and progressive vision loss.

What it means

Two copies of the Stargardt Disease (Discovered in the Labrador Retriever) mutation are needed for this disease to occur so Ruby Mea is likely to show signs of this disorder. Stargardt Disease is a late onset disorder, so Ruby Mea may not start to show signs until their senior years. You may notice that Ruby Mea initially has difficulties navigating in the dark, but as the condition progresses Ruby Mea's level of vision will decrease and may lead to complete blindness. Affected dogs usually adjust well to their normal surroundings despite the loss of sight. As Ruby Mea's sight worsens, keeping the environment stable/familiar may help Ruby Mea to get around safely. However it is likely that Ruby Mea will need assistance in dark and unfamiliar places.

For Veterinarians

Here's what a vet needs to know about STGD

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

Although this condition causes photoreceptor degeneration and loss of vision, many dogs adapt well to vision loss. Although there is no treatment, owners should be advised that the disease development is gradual and their dog may need assistance in unfamiliar surroundings as clinical signs progress. Owners may find that it is helpful to keep the dog's main environment as stable as possible (avoid moving furniture, etc.) to help them navigate as vision worsens.

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Other health conditions tested

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

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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
Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD) Risk	FGF4 retrogene	Insertion	0	AD	Clear
Cleft Lip & Palate with Syndactyly	ADAMTS20	Deletion	0	AR	Clear
Cleft Palate	DLX6	C>A	0	AR	Clear
CNS Atrophy with Cerebellar Ataxia (Discovered in the Belgian Shepherd)	SEPP1	Deletion	0	AR	Clear
Coat Color Dilution and Neurological Defects (Discovered in the Miniature Dachshund)	MYO5A	Insertion	0	AR	Clear
Complement 3 Deficiency	C3	Deletion	0	AR	Clear
Cone Degeneration (Discovered in the Alaskan Malamute)	CNGB3	Deletion	0	AR	Clear
Cone Degeneration (Discovered in the German Shepherd Dog)	CNGA3	C>T	0	AR	Clear
Cone Degeneration (Discovered in the German Shorthaired Pointer)	CNGB3	G>A	0	AR	Clear
Cone-Rod Dystrophy	NPHP4	Deletion	0	AR	Clear
Cone-Rod Dystrophy 1	PDE6B	Deletion	0	AR	Clear
Cone-Rod Dystrophy 2	IQCB1	Insertion	0	AR	Clear
Congenital Cornification (Discovered in the Labrador Retriever)	NSDHL	Deletion	0	SD	Clear

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Congenital Dyshormonogenic Hypothyroidism with Goiter (Discovered in the Shih Tzu)	SLC5A5	G>A	0	AR	Clear
Congenital Eye Malformations (Discovered in the Golden Retriever)	SIX6	C>T	0	AD	Clear
Congenital Hypothyroidism (Discovered in the Tenterfield Terrier)	TPO	C>T	0	AR	Clear
Congenital Hypothyroidism (Discovered in the Toy Fox and Rat Terrier)	TPO	C>T	0	AR	Clear
Congenital Muscular Dystrophy (Discovered in the Italian Greyhound)	LAMA2	G>A	0	AR	Clear
Congenital Muscular Dystrophy (Discovered in the Staffordshire Bull Terrier)	LAMA2	Deletion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Golden Retriever)	COLQ	G>A	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Heideterrier)	CHRNE	Insertion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Jack Russell Terrier)	CHRNE	Insertion	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Labrador Retriever)	COLQ	T>C	0	AR	Clear
Congenital Myasthenic Syndrome (Discovered in the Old Danish Pointer)	CHAT	G>A	0	AR	Clear
Congenital Stationary Night Blindness (CSNB)	RPE65	A>T	0	AR	Clear
Craniomandibular Osteopathy (Discovered in Scottish Terrier breeds)	SLC37A2	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Australian Terrier)	COL1A1	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Basset Hound)	SLC37A2	C>T	0	AD	Clear
Craniomandibular Osteopathy (Discovered in the Weimaraner)	SLC35D1	Deletion	0	AD	Clear
Cystic Renal Dysplasia and Hepatic Fibrosis	INPP5E	G>A	0	AR	Clear

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

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Early-Onset Progressive Retinal Atrophy (Discovered in the Portuguese Water Dog)	Confidential	-	0	AR	Clear
Early-Onset Progressive Retinal Atrophy, (Discovered in the Spanish Water Dog)	PDE6B	Deletion	0	AR	Clear
Ehlers-Danlos Syndrome (Discovered in mixed breed)	COL5A1	G>A	0	AD	Clear
Ehlers-Danlos Syndrome (Discovered in the Labrador Retriever)	COL5A1	Deletion	0	AD	Clear
Epidermolytic Hyperkeratosis	KRT10	G>T	0	AR	Clear
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 the Schapendoes)	CCDC66	Insertion	0	AR	Clear
Glanzmann Thrombasthenia Type I (Discovered in Great Pyrenees)	ITGA2B	C>G	0	AR	Clear
Glanzmann Thrombasthenia Type I (Discovered in mixed breed dogs)	ITGA2B	C>T	0	AR	Clear
Globoid Cell Leukodystrophy (Discovered in Terriers)	GALC	A>C	0	AR	Clear
Globoid Cell Leukodystrophy (Discovered in the Irish Setter)	GALC	A>T	0	AR	Clear

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Glycogen Storage Disease Type Ia (Discovered in the German Pinscher)	G6PC	Insertion	0	AR	Clear
Glycogen Storage Disease Type Ia (Discovered in the Maltese)	G6PC	G>C	0	AR	Clear
Glycogen Storage Disease Type IIIa, (GSD IIIa)	AGL	Deletion	0	AR	Clear
GM1 Gangliosidosis (Discovered in the Portuguese Water Dog)	GLB1	G>A	0	AR	Clear
GM1 Gangliosidosis (Discovered in the Shiba)	GLB1	Deletion	0	AR	Clear
GM2 Gangliosidosis (Discovered in the Japanese Chin)	HEXA	G>A	0	AR	Clear
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

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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
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
Ichthyosis (Discovered in the American Bulldog)	NIPAL4	Deletion	0	AR	Clear
Ichthyosis (Discovered in the Great Dane)	SLC27A4	G>A	0	AR	Clear
Ichthyosis Type 2 (Discovered in the Golden Retriever)	ABHD5	Deletion	0	AR	Clear
Inflammatory Myopathy (Discovered in the Dutch Shepherd Dog)	SLC25A12	A>G	0	AR	Clear
Inflammatory Pulmonary Disease (Discovered in the Rough Collie)	AKNA	Deletion	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Beagle)	CUBN	Deletion	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)	CUBN	Deletion	0	AR	Clear
Intestinal Cobalamin Malabsorption (Discovered in the Komondor)	CUBN	G>A	0	AR	Clear
Intestinal Lipid Malabsorption (Discovered in the Australian Kelpie)	ACSL5	Deletion	0	AR	Clear
Junctional Epidermolysis Bullosa (Discovered in the Australian Cattle Dog Mix)	LAMA3	T>A	0	AR	Clear
Junctional Epidermolysis Bullosa (Discovered in the Australian Shepherd)	LAMB3	A>G	0	AR	Clear

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Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Juvenile Cataract (Discovered in the Wirehaired Pointing Griffon)	FYCO1	Deletion	0	AR	Clear
Juvenile Dilated Cardiomyopathy (Discovered in the Toy Manchester Terrier)	Confidential	-	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
Lamellar Ichthyosis	TGM1	Insertion	0	AR	Clear
Laryngeal Paralysis (Discovered in the Bull Terrier and Miniature Bull Terrier)	RAPGEF6	Insertion	0	AR	Clear
Leigh-like Subacute Necrotizing Encephalopathy (Discovered in the Yorkshire Terrier)	SLC19A3	Insertion	0	AR	Clear
Lethal Acrodermatitis (Discovered in the Bull Terrier)	MKLN1	A>C	0	AR	Clear
Leukodystrophy (Discovered in the Standard Schnauzer)	TSEN54	C>T	0	AR	Clear
Ligneous Membranitis	PLG	T>A	0	AR	Clear
Limb-girdle Muscular Dystrophy (Discovered in the Boston Terrier)	SGCD	—	0	AR	Clear
Limb-girdle Muscular Dystrophy, Type L3 (Discovered in the Miniature Dachshund)	SGCA	G>A	0	AR	Clear
Lung Developmental Disease (Discovered in the Airedale Terrier)	LAMP3	C>T	0	AR	Clear
Macrothrombocytopenia (Discovered in Norfolk and Cairn Terrier)	TUBB1	G>A	0	AR	Clear
May-Hegglin Anomaly	MYH9	G>A	0	AD	Clear

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MDR1 Medication Sensitivity	MDR1/ABCB1	Deletion	0	AD	Clear
Microphthalmia (Discovered in the Soft-Coated Wheaten Terrier)	RBP4	Deletion	0	AR	Clear
Mucopolysaccharidosis Type IIIA (Discovered in the Dachshund)	SGSH	C>A	0	AR	Clear
Mucopolysaccharidosis Type IIIA (Discovered in the New Zealand Huntaway)	SGSH	Insertion	0	AR	Clear
Mucopolysaccharidosis Type VII (Discovered in the Brazilian Terrier)	GUSB	C>T	0	AR	Clear
Mucopolysaccharidosis Type VII (Discovered in the German Shepherd Dog)	GUSB	G>A	0	AR	Clear
Mucopolysaccharidosis VI (Discovered in the Miniature Pinscher)	ARSB	G>A	0	AR	Clear
Muscular Dystrophy (Discovered in the 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

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Myotubular Myopathy	MTM1	A>C	0	SR	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 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

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Osteochondromatosis (Discovered in the American Staffordshire Terrier)	EXT2	C>A	0	AR	Clear
Osteogenesis Imperfecta (Discovered in the Beagle)	COL1A2	C>T	0	AD	Clear
Osteogenesis Imperfecta (Discovered in the Dachshund)	SERPINH1	T>C	0	AR	Clear
P2RY12-associated Bleeding Disorder	P2RY12	Deletion	0	AR	Clear
Palmoplantar Hyperkeratosis (Discovered in the Rottweiler)	DSG1	Deletion	0	AR	Clear
Paroxysmal Dyskinesia	PIGN	C>T	0	AR	Clear
Persistent Müllerian Duct Syndrome	AMHR2	C>T	0	AR	Clear
Phosphofructokinase Deficiency	PFKM	G>A	0	AR	Clear
Pituitary Dwarfism (Discovered in the Karelian Bear Dog)	POU1F1	C>A	0	AR	Clear
Polycystic Kidney Disease	PKD1	G>A	0	AD	Clear
Prekallikrein Deficiency	KLKB1	T>A	0	AR	Clear
Primary Ciliary Dyskinesia	CCDC39	C>T	0	AR	Clear
Primary Ciliary Dyskinesia (Discovered in the Alaskan Malamute)	NME5	Deletion	0	AR	Clear
Primary Lens Luxation	ADAMTS17	G>A	0	AR	Clear
Primary Open Angle Glaucoma (Discovered in Basset Fauve de Bretagne)	ADAMTS17	G>A	0	AR	Clear
Primary Open Angle Glaucoma (Discovered in Petit Basset Griffon Vendeen)	ADAMTS17	Insertion	0	AR	Clear
Primary Open Angle Glaucoma and Lens Luxation (Discovered in Chinese Shar-Pei)	ADAMTS17	Deletion	0	AR	Clear
Progressive Early-Onset Cerebellar Ataxia	SEL1L	T>C	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Basenji)	SAG	T>C	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA 2 variant)	TTC8	Deletion	0	AR	Clear

Test Date: 04/12/2024

veterinarian: Renia Duranico

Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA1 variant)	SLC4A3	Insertion	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Lapponian Herder)	IFT122	C>T	0	AR	Clear
Progressive Retinal Atrophy (Discovered in the Lhasa Apso)	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	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

Test Date: 04/12/2024

Veterinarian: Rena Dunahoo

Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
Rod-Cone Dysplasia 3	PDE6A	Deletion	0	AR	Clear
Sensorineural Deafness (Discovered in the Rottweiler)	LOXHD1	G>C	0	AR	Clear
Sensory Ataxic Neuropathy	tRNATyr	Deletion	0	MT	Clear
Sensory Neuropathy	FAM134B	Insertion	0	AR	Clear
Severe Combined Immunodeficiency (Discovered in Frisian Water Dogs)	RAG1	G>T	0	AR	Clear
Severe Combined Immunodeficiency (Discovered in Russell Terriers)	PRKDC	G>T	0	AR	Clear
Shaking Puppy Syndrome (Discovered in the Border Terrier)	Confidential	-	0	AR	Clear
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
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	—	0	AR	Clear
Trapped Neutrophil Syndrome	VPS13B	Deletion	0	AR	Clear
Van den Ende-Gupta Syndrome	SCARF2	Deletion	0	AR	Clear

Test Date: 04/12/2024

Veterinarian: Rena Dunahoo

Other health conditions tested

Genetic Condition	Gene	Risk Variant	Copies	Inheritance	Result
von Willebrand's Disease, type 1	VWF	G>A	0	AR	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	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

Test Date: 04/12/2024

Veterinarian: Rena Dunahoo

Traits

Coat Color

	Gene	Variant	Copies	Result
Fawn	ASIP	a ^y	0	No effect
Recessive Black	ASIP	a	0	No effect
Tan Points Two copies, or occasionally one copy, of this variant may result in a black and tan coat color pattern.	ASIP	a ^t	2	Tan points possible
Dominant Black One or two copies of the dominant black will give a dog a black coat (depending on other variants), black eye rims, nose and pads. One copy may also give a tiger striped appearance, known as brindle patterning.	CBD103	K ^B	2	Black possible
Mask	MC1R	E ^m	0	No effect
Recessive Red (e1) To show a solid red coat, a dog must inherit two copies of a Recessive Red variant, one from each parent. This can either be two copies of a particular variant, such as this one (e1) or two of any combination of recessive red variants. Recessive red coats will appear white, cream, yellow or red, although there are other variants that can result in a similar appearance. The amount of red pigment in the coat, called the intensity, is governed by other genes.	MC1R	e ¹	2	Cream to red coat likely
Recessive Red (e2)	MC1R	e ²	0	No effect
Recessive Red (e3)	MC1R	e ³	0	No effect
Sable (Discovered in the Cocker Spaniel)	MC1R	e ^H	0	No effect
Widow's Peak (Discovered in Ancient dogs)	MC1R	e ^A	0	No effect
Widow's Peak (Discovered in the Afghan Hound and Saluki)	MC1R	E ^G	0	No effect

Color Modification

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

Test Date: 04/12/2024

Veterinarian: Rena Dunahoo

Color Modification

Red Intensity

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.

Gene	Variant	Copies	Result
MFSD12	i	1	No effect

Dilution (d1) Linkage test

MLPH	d ¹	0	No effect
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Dilution (d2)

MLPH	d ²	0	No effect
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Dilution (d3)

MLPH	d ³	0	No effect
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Chocolate (basd)

TYRP1	basd	0	No effect
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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	bc	1	Black features likely, chocolate possible
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Chocolate (bd)

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 ("bd"), or two of any combination of chocolate variants. This variant is unique in that it can occur on the same chromosome as another chocolate variant, where both variants are donated from one parent. If the other parent does not also donate a chocolate variant, the dog will still express black pigment, not chocolate.

TYRP1	bd	1	Black features likely, chocolate possible
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Chocolate (be)

TYRP1	be	0	No effect
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Chocolate (bh)

TYRP1	bh	0	No effect
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Chocolate (bs)

TYRP1	bs	0	No effect
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Coat Patterns

Piebald

Gene	Variant	Copies	Result
MITF	SP	0	No effect

Test Date: 04/12/2024

Veterinarian: Rena Dunanou

Coat Patterns

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

Coat Length and Curl

	Gene	Variant	Copies	Result
Long Hair (lh1)	FGF5	lh ¹	0	No effect
Long Hair (lh2)	FGF5	lh ²	0	No effect
Long Hair (lh3)	FGF5	lh ³	0	No effect
Long Hair (lh4)	FGF5	lh ⁴	0	No effect
Long Hair (lh5)	FGF5	lh ⁵	0	No effect
Curly Coat	KRT71	C	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	H ^{rcc}	0	No effect
Hairlessness (Discovered in the American Hairless Terrier)	SGK3	h ^{raht}	0	No effect
Hairlessness (Discovered in the Scottish Deerhound)	SKG3	h ^{rsd}	0	No effect

Test Date: 04/12/2024

Veterinarian: Rena Dunahoo

Shedding

Reduced Shedding

One or two copies of the Reduced Shedding variant is likely to reduce a dog's tendency to shed. Copies of the Furnishings variant, particularly two, also reduce the tendency of a dog to shed.

Gene	Variant	Copies	Result
MC5R	sd	1	Occasional shedder

More Coat Traits

Hair Ridge

Gene	Variant	Copies	Result
FGF3, FGF4, FGF19, ORAOV1	R	0	No effect

Furnishings

RSP02	F	0	No effect
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Albino

SLC45A2	cal	0	No effect
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Head Shape

Short Snout (BMP3 variant)

Gene	Variant	Copies	Result
BMP3	-	0	No effect

Short Snout (SMOC2 variant)

SMOC2	-	0	No effect
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Eye Color

Blue Eyes (Discovered in the Siberian Husky)

Gene	Variant	Copies	Result
ALX4	-	0	No effect

Test Date: 04/12/2024

Veterinarian: Kena Dunahoo

Ears

Floppy Ears

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

Gene	Variant	Copies	Result
MSRB3	-	1	Partially floppy ears more likely

Extra Toes

Hind Dewclaws (Discovered in Asian breeds)

Gene	Variant	Copies	Result
LMBR1	DC-1	0	No effect

Hind Dewclaws (Discovered in Western breeds)

LMBR1	DC-2	0	No effect
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More Body Features

Back Muscle and Bulk

Gene	Variant	Copies	Result
ACSL4	-	0	No effect

High Altitude Adaptation

EPAS1	-	0	No effect
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Short Legs (Chondrodysplasia, CDPA)

FGF4	-	0	No effect
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Short Legs (Chondrodystrophy, CDDY)

FGF4	-	0	No effect
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Short Tail

T-box	T	0	Full tail length 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.