

Dark or Light Fur E (Extension) Locus

Genetic Result: Ee

Gene: Melanocortin Receptor 1 (MC1R)

This gene helps determine whether a dog can produce dark (black or brown) hairs or lighter yellow or red hairs. Any result except for **ee** means that the dog can produce dark hairs. An **ee** result means that the dog does not produce dark hairs and will have lighter yellow or red hairs all over its entire body.

The overall MC1R genetic result is influenced by more subloci than those presented in this section. Additional MC1R subloci results can be found under the **Coat Color Modifiers** > **Facial Fur Pattern** section below.

Did You Know?

If a dog has an **ee** result, then the fur's actual shade can range from a deep copper to white - the exact color cannot be predicted solely from this result and will depend on other genetic factors, including the red pigment intensity test.

Citations

Anderson et al 2020 , Dreger and Schmutz 2010 , Schmutz et al 2003 , Honkanen et al 2024 , Durig N et al 2018 (https://pubmed.ncbi.nlm.nih.gov/29932470/)

Can have dark fur

More information: http://www.doggenetics.co.uk/masks.html (http://www.doggenetics.co.uk/masks.html)

Dark brown pigment

Сосоа

No impact on fur and skin color

Genetic Result: NN

Gene: HPS3

Dogs with the **coco** genotype will produce dark brown pigment instead of black in both their hair and skin. Dogs with the **Nco** genotype will produce black pigment, but can pass the **co** variant on to their puppies. Dogs that have the **coco** genotype as well as the **bb** genotype at the B locus are generally a lighter brown than dogs that have the **Bb** or **BB** genotypes at the B locus.

Did You Know?

The **co** variant and the dark brown "cocoa" coat color have only been documented in French Bulldogs. Dogs with the cocoa coat color are sometimes born with light brown coats that darken as they reach maturity.

Citations

Kiener et al 2020 (https://pubmed.ncbi.nlm.nih.gov/32526956/)

More information: http://www.doggenetics.co.uk/liver.html#cocoa (http://www.doggenetics.co.uk/liver.html#cocoa)

Red Pigment Intensity

No impact on coat pattern

I (Intensity) Loci

Genetic Result: Dilute Red Pigmentation

Intensity refers to the concentration of red pigment in the coat. Dogs with more densely concentrated (intense) pigment will be a deeper red, while dogs with less concentrated (dilute) pigment will be tan, yellow, cream, or white. Five locations in the dog genome explain approximately 70% of red pigmentation intensity variation across all dogs. Because the locations we test may not directly cause differences in red pigmentation intensity, we consider this to be a linkage test.

Did You Know?

One of the genes that influences pigment intensity in dogs, TYR, is also responsible for intensity variation in domestic mice, cats, cattle, rabbits, and llamas. In dogs and humans, more genes are involved.

Citations

Slavney et al 2021, Weich et al 2020, Hedan et al 2019 (https://pubmed.ncbi.nlm.nih.gov/31117290/)

Brown or Black Pigment *B (Brown) Locus* Black or gray fur and skin

Genetic Result: BB

Gene: Tyrosinase Related Protein 1 (TYRP1)

This gene helps determine whether a dog produces brown or black pigments. Dogs with a **bb** result produce brown pigment instead of black in both their hair and skin, while dogs with a **Bb** or **BB** result produce black pigment. Dogs that have **ee** at the E (Extension) Locus and **bb** at this B (Brown) Locus are likely to have red or cream coats and brown noses, eye rims, and footpads, which is sometimes referred to as "Dudley Nose" in Labrador Retrievers.

Did You Know?

"Liver" or "chocolate" is the preferred color term for brown in most breeds; in the Doberman Pinscher it is referred to as "red".

Citations

Schmutz et al 2002, Hrckova Turnova et al 2017, Jancuskova et al 2018 (https://pubmed.ncbi.nlm.nih.gov/30109695/)

More information: http://www.doggenetics.co.uk/liver.html (http://www.doggenetics.co.uk/liver.html)

Color Dilution

D (Dilute) Locus

Dark (non-dilute) fur and skin

Genetic Result: DD

Gene: Melanophilin (MLPH)

This gene helps determine whether a dog has lighter "diluted" pigment. A dog with a **Dd** or **DD** result will not be dilute. A dog with a **dd** result will have all their black or brown pigment lightened ("diluted") to gray or light brown, and may lighten red pigment to cream. This affects their fur, skin, and sometimes eye color. The D locus result that we report is determined by three different genetic variants that can work together to cause diluted pigmentation. These are the common **d** allele, also known as "**d1**", and the less common alleles known as "**d2**" and "**d3**". Dogs with two **d** alleles, regardless of which variant, are typically dilute.

Did You Know?

There are many breed-specific names for these dilute colors, such as "blue", "charcoal", "fawn", "silver", and "Isabella". Dilute dogs, especially in certain breeds, have a higher incidence of Color Dilution Alopecia which causes hair loss in some patches.

Citations

Drogemuller et al 2007, Bauer et al 2018, Van Buren et al 2020 (https://pubmed.ncbi.nlm.nih.gov/32531980/)

More information: http://www.doggenetics.co.uk/dilutes.html (http://www.doggenetics.co.uk/dilutes.html)



Coat Color Modifiers

Hidden Patterning K (Dominant Black) Locus

More likely to have patterned fur

Genetic Result: k^yk^y

Gene: Canine Beta-Defensin 103 (CBD103)

This gene helps determine whether the dog has a black coat. Dogs with a $k^y k^y$ result will show a coat color pattern based on the result they have at the A (Agouti) Locus. A $K^B K^B$ or $K^B k^y$ result means the dog is dominant black, which overrides the fur pattern that would otherwise be determined by the A (Agouti) Locus. These dogs will usually have solid black or brown coats, or if they have **ee** at the E (Extension) Locus then red/cream coats, regardless of their result at the A (Agouti) Locus. Dogs who test as $K^B k^y$ may be brindle rather than black or brown.

Did You Know?

Even if a dog is "dominant black" several other genes could still impact the dog's fur and cause other patterns, such as white spotting.

Citations

Candille et al 2007 (http://www.ncbi.nlm.nih.gov/pubmed/17947548)

More information: http://www.doggenetics.co.uk/black.htm (http://www.doggenetics.co.uk/black.htm)

Body Pattern A (Agouti) Locus **Recessive Black/Brown**

Genetic Result: aa

Gene: Agouti Signalling Protein (ASIP)

This gene is responsible for causing different coat patterns. It only affects the fur of dogs that do not have **ee** at the E (Extension) Locus and do have **k**^y**k**^y at the K (Dominant Black) Locus. It controls switching between black and red pigment in hair cells, which means that it can cause a dog to have hairs that have sections of black and sections of red/cream, or hairs with different colors on different parts of the dog's body. Sable or Fawn dogs have a mostly or entirely red coat with some interspersed black hairs. Agouti or Wolf Sable dogs have red hairs with black tips, mostly on their head and back. Black and tan dogs are mostly black or brown with lighter patches on their cheeks, eyebrows, chest, and legs. Recessive black dogs have solid-colored black or brown coats.

Did You Know?

The ASIP gene causes interesting coat patterns in many other species of animals as well as dogs.

Citations

Dreger and Schmutz 2011, Berryere et al 2005 (http://www.ncbi.nlm.nih.gov/pubmed/15965787)

More information: http://www.doggenetics.co.uk/tan.html (http://www.doggenetics.co.uk/tan.html)

Facial Fur Pattern

No dark mask or grizzle facial fur patterns

E (Extension) Locus

Genetic Result: Ee

Gene: Melanocortin Receptor 1 (MC1R)

This gene determines whether a dog can have dark hair and can give it a black "mask" or "widow's peak," unless the dog has overriding coat color genetic factors. Dogs with one or two copies of **E**^m in their result may have a mask, which is dark facial fur as seen in the German Shepherd Dog and Pug. Dogs with no **E**^m in their result but one or two copies of the **E**^g, **E**^a, or **E**^h variants can instead have a "widow's peak", which is dark forehead fur.

Did You Know?

The "widow's peak" is seen in the Afghan Hound and Borzoi, and is called either "grizzle" or "domino."

In the absence of E^m , dogs with the E^g variant can have a "widow's peak" phenotype. In the absence of both E^m and E variants, dogs with the E^a or E^h variants can express the "widow's peak" phenotype. Additionally, a dog with any combination of two of the E^g , E^a , or E^h variants (example: E^gE^a) is also expected to express the grizzle phenotype.

Citations

Anderson et al 2020, Dreger and Schmutz 2010, Schmutz et al 2003, Honkanen et al 2024, Durig N et al 2018 (https://pubmed.ncbi.nlm.nih.gov/29932470/)

More information: http://www.doggenetics.co.uk/masks.html (http://www.doggenetics.co.uk/masks.html)

Saddle Tan

No impact on coat pattern

Genetic Result: NI

Gene: RALY

The "Saddle Tan" pattern causes the black hairs to recede into a "saddle" shape on the back, leaving a tan face, legs, and belly, as a dog ages. The Saddle Tan pattern is characteristic of breeds like the Corgi, Beagle, and German Shepherd. Dogs that have the **II** genotype at this locus are more likely to be mostly black with tan points on the eyebrows, muzzle, and legs as commonly seen in the Doberman Pinscher and the Rottweiler. This gene modifies the A Locus **a**^t allele, so dogs that do not express **a**^t are not influenced by this gene.

Did You Know?

The Saddle Tan pattern is characteristic of breeds like the Corgi, Beagle, and German Shepherd.

Citations

Dreger et al 2013 (https://www.ncbi.nlm.nih.gov/pubmed/23519866)

White Spotting

S (White Spotting) Locus

Likely to have little to no white in coat

Genetic Result: SS

Gene: MITF

This gene is responsible for most of the white spotting observed in dogs. Dogs with a result of **spsp** will have a nearly white coat or large patches of white in their coat. Dogs with a result of **Ssp** will have more limited white spotting that is breed-dependent. A result of **SS** means that a dog likely has no white or minimal white in their coat. The S Locus does not explain all white spotting patterns in dogs and other causes are currently being researched. Some dogs may have small amounts of white on the paws, chest, face, or tail regardless of their result at this gene.

Did You Know?

Any dog can have white spotting regardless of coat color. The colored sections of the coat will reflect the dog's other genetic coat color results.

Citations

Karlsson et al 2007 (http://www.ncbi.nlm.nih.gov/pubmed/17906626)

More information: http://www.doggenetics.co.uk/white.htm (http://www.doggenetics.co.uk/white.htm)

Roan

R (Roan) Locus

Likely no impact on coat pattern

Genetic Result: rr

Gene: USH2A

This gene, along with the S Locus, regulates whether a dog will have roaning. Dogs with at least one copy of **R** will likely have roaning on otherwise uniformly unpigmented white areas created by the S Locus. Roan may not be visible if white spotting is limited to small areas, such as the paws, chest, face, or tail. The extent of roaning varies from uniform roaning to non-uniform roaning, and patchy, non-uniform roaning may look similar to ticking. Roan does not appear in white areas created by other genes, such as a combination of the E Locus and I Locus (for example, Samoyeds). The roan pattern can appear with or without ticking.

Did You Know?

Roan, tick, and Dalmatians' spots become visible a few weeks after birth. The R Locus is probably involved in the development of Dalmatians' spots.

Citations

Brancalion et al 2021, Kawakami et al 2021 (https://pubmed.ncbi.nlm.nih.gov/33755696/)

More information: http://www.doggenetics.co.uk/ticking.html (http://www.doggenetics.co.uk/ticking.html)

Merle

M (Merle) Locus

Unlikely to have merle pattern

Genetic Result: mm

Gene: PMEL

This gene is responsible for mottled or patchy coat color in some dogs. Dogs with an **M*m** result are likely to appear merle or could be "non-expressing" merle, meaning that the merle pattern is very subtle or not at all evident in their coat. Dogs with an **M*M*** result are likely to have merle or double merle coat patterning. Dogs with an **mm** result are unlikely to have a merle coat pattern.

Did You Know?

Merle coat patterning is common to several dog breeds including the Australian Shepherd, Catahoula Leopard Dog, and Shetland Sheepdog.

Citations

Clark et al 2006 (https://www.pnas.org/content/103/5/1376)

More information: http://www.doggenetics.co.uk/merle.html (http://www.doggenetics.co.uk/merle.html)

Harlequin

No impact on coat pattern

Genetic Result: hh

Gene: PSMB

This gene, along with the M Locus, determines whether a dog will have harlequin patterning. This pattern is recognized in Great Danes and causes dogs to have a white coat with patches of darker pigment. A dog with an **Hh** result will be harlequin if they are also **M*m** or **M*M*** at the M Locus and are not **ee** at the E locus. Dogs with a result of **hh** will not be harlequin.

Did You Know?

While many harlequin dogs are white with black patches, some dogs have grey, sable, or brindle patches of color, depending on their genotypes at other coat color genes.

Citations

Clark et al 2011 (http://www.ncbi.nlm.nih.gov/pubmed/21256207)

More information: http://www.doggenetics.co.uk/harlequin.html (http://www.doggenetics.co.uk/harlequin.html)

Other Coat Traits

Furnishings	Likely unfurnished (no mustache, beard, and/or eyebrows)
Coat Length	Likely short or mid-length coat
Shedding	Likely heavy/seasonal shedding
Coat Texture	Likely straight coat
Hairlessness (Xolo type)	Very unlikely to be hairless
Hairlessness (Terrier type)	Very unlikely to be hairless
Oculocutaneous Albinism Type 2	Likely not albino

Cther Body Features	
Muzzle Length	Likely medium or long muzzle
Tail Length	Likely normal-length tail
Hind Dew Claws	Unlikely to have hind dew claws
Back Muscling & Bulk (Large Breed)	Likely normal muscling
Eye Color	Less likely to have blue eyes
Body Size	

Body Size 1	Larger
Body Size 2	Larger
Body Size 3	Intermediate
Body Size 4	Larger
Body Size 5	Larger



Altitude Adaptation

Appetite

Normal altitude tolerance

Normal food motivation

Dig into your dog's mix

From energy to appetite, from herding to health it's amazing how much you can learn about your dog with just a cheek swab. What will you find out?

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Maternal Haplotype Paternal Haplotype >

QUINN

Veterinary Report by Embark

embarkvet.com

Test Date: November 16th, 2020

Customer-supplied information

Owner Name: Sonja Needs Dog Name: Quinn Sex: Male (intact) Date of birth: 12/19/17 Breed type: mixed Breed: n/a Breed registration: n/a Microchip: n/a

Clinical Tools

These clinical genetic tools can inform clinical decisions and diagnoses. These tools do not predict increased risk for disease.

Alanine Aminotransferase Activity (GPT)

O Quinn's baseline ALT level is Low Normal

Why is this important to your vet?

Quinn has one copy of a variant associated with reduced ALT activity as measured on veterinary blood chemistry panels. Please inform your veterinarian that Quinn has this genotype, as ALT is often used as an indicator of liver health and Quinn is likely to have a lower than average resting ALT activity. As such, an increase in Quinn's ALT activity could be evidence of liver damage, even if it is within normal limits by standard ALT reference ranges.

What is Alanine Aminotransferase Activity?

Alanine aminotransferase (ALT) is a clinical tool that can be used by veterinarians to better monitor liver health. This result is not associated with liver disease. ALT is one of several values veterinarians measure on routine blood work to evaluate the liver. It is a naturally occurring enzyme located in liver cells that helps break down protein. When the liver is damaged or inflamed, ALT is released into the bloodstream.

How vets diagnose this condition

Genetic testing is the only way to provide your veterinarian with this clinical tool.

How this condition is treated

Veterinarians may recommend blood work to establish a baseline ALT value for healthy dogs with one or two copies of this variant.

Health Report

How to interpret Quinn's genetic health results:

If Quinn inherited any of the variants that we tested, they will be listed at the top of the Health Report section, along with a description of how to interpret this result. We also include all of the variants that we tested Quinn for that we did not detect the risk variant for.

A genetic test is not a diagnosis

This genetic test does not diagnose a disease. Please talk to your vet about your dog's genetic results, or if you think that your pet may have a health condition or disease.



Good news!

Quinn is not at increased risk for the genetic health conditions that Embark tests.

Breed-Relevant Genetic Conditions	17 variants not detected	V
Additional Genetic Conditions	189 variants not detected	<



Quinn did not have the variants that we tested for, that are relevant to his breeds:

- Sensitivity (MDR1)
- Sactor VII Deficiency (F7 Exon 5)
- Sector VIII Deficiency, Hemophilia A (F8 Exon 11, Shepherd Variant 1)
- Sector VIII Deficiency, Hemophilia A (F8 Exon 1, Shepherd Variant 2)
- Canine Leukocyte Adhesion Deficiency Type III, CLADIII (FERMT3)
- Scott Syndrome (TMEM16F)
- S X-Linked Progressive Retinal Atrophy 1, XL-PRA1 (RPGR)
- S Achromatopsia (CNGA3 Exon 7 German Shepherd Variant)
- S Hyperuricosuria and Hyperuricemia or Urolithiasis, HUU (SLC2A9)
- Samoyed Variant 2) X-Linked Hereditary Nephropathy, XLHN (COL4A5 Exon 35, Samoyed Variant 2)
- Seriary Ciliary Dyskinesia, PCD (NME5)
- S X-linked Ectodermal Dysplasia, Anhidrotic Ectodermal Dysplasia (EDA Intron 8)
- Senal Cystadenocarcinoma and Nodular Dermatofibrosis, RCND (FLCN Exon 7)
- Syndrome, MPS VII (GUSB Exon 3)
- 📀 GM1 Gangliosidosis (GLB1 Exon 15 Alaskan Husky Variant)
- SOD1A)
- Section 2017 Polyneuropathy, NDRG1 Malamute Variant (NDRG1 Exon 4)



Quinn did not have the variants that we tested for, in the following conditions that the potential effect on dogs with Quinn's breeds may not yet be known.

- P2Y12 Receptor Platelet Disorder (P2Y12)
- Sector IX Deficiency, Hemophilia B (F9 Exon 7, Terrier Variant)
- Sector IX Deficiency, Hemophilia B (F9 Exon 7, Rhodesian Ridgeback Variant)
- 📀 Factor VIII Deficiency, Hemophilia A (F8 Exon 10, Boxer Variant)
- S Thrombopathia (RASGRP1 Exon 5, Basset Hound Variant)
- 🍼 Thrombopathia (RASGRP1 Exon 8)
- S Thrombopathia (RASGRP1 Exon 5, American Eskimo Dog Variant)
- Son Willebrand Disease Type III, Type III vWD (VWF Exon 4)
- Son Willebrand Disease Type III, Type III vWD (VWF Exon 7)
- Von Willebrand Disease Type I (VWF)
- 📀 Von Willebrand Disease Type II, Type II vWD (VWF)
- Canine Leukocyte Adhesion Deficiency Type I, CLADI (ITGB2)
- Congenital Macrothrombocytopenia (TUBB1 Exon 1, Cairn and Norfolk Terrier Variant)
- Canine Elliptocytosis (SPTB Exon 30)
- Slanzmann's Thrombasthenia Type I (ITGA2B Exon 12)
- S May-Hegglin Anomaly (MYH9)
- Service Stress Prekallikrein Deficiency (KLKB1 Exon 8)
- S Pyruvate Kinase Deficiency (PKLR Exon 5)
- Service Anticipation of the service of the service
- Pyruvate Kinase Deficiency (PKLR Exon 7 Pug Variant)
- Search Pyruvate Kinase Deficiency (PKLR Exon 7 Beagle Variant)
- Service Analysis Pyruvate Kinase Deficiency (PKLR Exon 10)
- Strapped Neutrophil Syndrome (VPS13B)
- 📀 Ligneous Membranitis, LM (PLG)

- C Methemoglobinemia CYB5R3
- 📀 Congenital Hypothyroidism (TPO, Tenterfield Terrier Variant)
- Congenital Hypothyroidism (TPO, Rat, Toy, Hairless Terrier Variant)
- Complement 3 Deficiency, C3 Deficiency (C3)
- 📀 Severe Combined Immunodeficiency (PRKDC)
- 📀 Severe Combined Immunodeficiency (RAG1)
- X-linked Severe Combined Immunodeficiency (IL2RG Variant1)
- X-linked Severe Combined Immunodeficiency (IL2RG Variant 2)
- Setter Variant) Progressive Retinal Atrophy, rcd1 (PDE6B Exon 21 Irish Setter Variant)
- Progressive Retinal Atrophy, rcd3 (PDE6A)
- Section 2017 Progressive Retinal Atrophy, CNGA (CNGA1 Exon9)
- Progressive Retinal Atrophy, prcd (PRCD Exon 1)
- Progressive Retinal Atrophy (CNGB1)
- Progressive Retinal Atrophy (SAG)
- Solden Retriever Progressive Retinal Atrophy 1, GR-PRA1 (SLC4A3)
- Solden Retriever Progressive Retinal Atrophy 2, GR-PRA2 (TTC8)
- Second Se
- Second Se
- Section 2012 Progressive Retinal Atrophy, PRA3 (FAM161A)
- Collie Eye Anomaly, Choroidal Hypoplasia, CEA (NHEJ1)
- Solution Contemporation Contemporation (CNGB3Exon6)
- S Achromatopsia (CNGA3 Exon 7 Labrador Retriever Variant)
- S Autosomal Dominant Progressive Retinal Atrophy (RHO)
- Canine Multifocal Retinopathy (BEST1 Exon 2)

- 📀 Canine Multifocal Retinopathy (BEST1 Exon 5)
- 📀 Canine Multifocal Retinopathy (BEST1 Exon 10 Deletion)
- Canine Multifocal Retinopathy (BEST1 Exon 10 SNP)
- 📀 Glaucoma (ADAMTS10 Exon 9)
- 📀 Glaucoma (ADAMTS10 Exon 17)
- 📀 Glaucoma (ADAMTS17 Exon 11)
- 📀 Glaucoma (ADAMTS17 Exon 2)
- 📀 Goniodysgenesis and Glaucoma (OLFM3)
- Wereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts (HSF4 Exon 9 Shepherd Variant)
- Serimary Lens Luxation (ADAMTS17)
- Congenital Stationary Night Blindness (RPE65)
- Congenital Stationary Night Blindness (LRIT3)
- S Macular Corneal Dystrophy, MCD (CHST6)
- C 2,8-Dihydroxyadenine Urolithiasis, 2,8-DHA Urolithiasis (APRT)
- Cystinuria Type I-A (SLC3A1)
- CystinuriaTypeII-A(SLC3A1)
- 📀 Cystinuria Type II-B (SLC7A9)
- Selvent State State (PKD1) Polycystic Kidney Disease, PKD (PKD1)
- 📀 Primary Hyperoxaluria (AGXT)
- 📀 Protein Losing Nephropathy, PLN (NPHS1)
- S Autosomal Recessive Hereditary Nephropathy, Familial Nephropathy, ARHN (COL4A4 Exon 3)
- Primary Ciliary Dyskinesia, PCD (CCDC39 Exon 3)
- Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatosis, Dry Eye Curly Coat Syndrome, CKCSID (FAM83H Exon 5)
- 🔮 Canine Fucosidosis (FUCA1)

- Slycogen Storage Disease Type II, Pompe's Disease, GSD II (GAA)
- 📀 Glycogen Storage Disease Type IA, Von Gierke Disease, GSD IA (G6PC)
- 📀 Glycogen Storage Disease Type IIIA, GSD IIIA (AGL)
- Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6 Variant 1)
- Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6 Variant 2)
- S Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 5)
- Glycogen storage disease Type VII, Phosphofructokinase Deficiency, PFK Deficiency (PFKM Whippet and English Springer Spaniel Variant)
- Glycogen storage disease Type VII, Phosphofructokinase Deficiency, PFK Deficiency (PFKM Wachtelhund Variant)
- C Lagotto Storage Disease (ATG4D)
- Solution Neuronal Ceroid Lipofuscinosis 1, NCL 1 (PPT1 Exon 8)
- Neuronal Ceroid Lipofuscinosis 2, NCL 2 (TPP1 Exon 4)
- Solution Neuronal Ceroid Lipofuscinosis 1, Cerebellar Ataxia, NCL4A (ARSG Exon 2)
- Neuronal Ceroid Lipofuscinosis 1, NCL5 (CLN5 Border Collie Variant)
- Neuronal Ceroid Lipofuscinosis 6, NCL 6 (CLN6 Exon 7)
- Seuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 English Setter Variant)
- S Neuronal Ceroid Lipofuscinosis (MFSD8)
- S Neuronal Ceroid Lipofuscinosis (CLN8 Australian Shepherd Variant)
- Veuronal Ceroid Lipofuscinosis 10, NCL 10 (CTSD Exon 5)
- Solution Neuronal Ceroid Lipofuscinosis (CLN5 Golden Retriever Variant)
- S Adult-Onset Neuronal Ceroid Lipofuscinosis (ATP13A2, Tibetan Terrier Variant)
- **Solution** Caroid Lipofuscinosis (ATP13A2, Australian Cattle Dog Variant)
- 📀 GM1 Gangliosidosis (GLB1 Exon 15 Shiba Inu Variant)
- SM1 Gangliosidosis (GLB1 Exon 2)
- 📀 GM2 Gangliosidosis (HEXB, Poodle Variant)

- SM2 Gangliosidosis (HEXA)
- 🛇 Globoid Cell Leukodystrophy, Krabbe disease (GALC Exon 5)
- Autosomal Recessive Amelogenesis Imperfecta, Familial Enamel Hypoplasia (Italian Greyhound Variant)
- Autosomal Recessive Amelogenesis Imperfecta, Familial Enamel Hypoplasia (Parson Russell Terrier Variant)
- Service American Duct Syndrome, PMDS (AMHR2)
- S Deafness and Vestibular Syndrome of Dobermans, DVDob, DINGS (MYO7A)
- Shar-Pei Autoinflammatory Disease, SPAID, Shar-Pei Fever (MTBP)
- 📀 Neonatal Interstitial Lung Disease (LAMP3)
- S Alaskan Husky Encephalopathy, Subacute Necrotizing Encephalomyelopathy (SLC19A3)
- Alexander Disease (GFAP)
- Cerebellar Abiotrophy, Neonatal Cerebellar Cortical Degeneration, NCCD (SPTBN2)
- 🛇 Cerebellar Ataxia, Progressive Early-Onset Cerebellar Ataxia (SEL1L)
- 📀 Cerebellar Hypoplasia (VLDLR)
- Spinocerebellar Ataxia, Late-Onset Ataxia, LoSCA (CAPN1)
- Spinocerebellar Ataxia with Myokymia and/or Seizures (KCNJ10)
- Vereditary Ataxia (RAB24)
- Senign Familial Juvenile Epilepsy, Remitting Focal Epilepsy (LGI2)
- Setal-Onset Neonatal Neuroaxonal Dystrophy (MFN2)
- S Hypomyelination and Tremors (FNIP2)
- Shaking Puppy Syndrome, X-linked Generalized Tremor Syndrome (PLP)
- Neuroaxonal Dystrophy, NAD (Spanish Water Dog Variant)
- 📀 Neuroaxonal Dystrophy, NAD (Rottweiler Variant)
- 🍼 L-2-Hydroxyglutaricaciduria, L2HGA (L2HGDH)
- 📀 Neonatal Encephalopathy with Seizures, NEWS (ATF2)

- 🔇 Narcolepsy (HCRTR2Intron 6)
- 🔇 Narcolepsy (HCRTR2 Exon 1)
- Progressive Neuronal Abiotrophy, Canine Multiple System Degeneration, CMSD (SERAC1 Exon 15)
- Progressive Neuronal Abiotrophy, Canine Multiple System Degeneration, CMSD (SERAC1 Exon
 4)
- Juvenile Laryngeal Paralysis and Polyneuropathy, Polyneuropathy with Ocular Abnormalities and Neuronal Vacuolation, POANV (RAB3GAP1, Rottweiler Variant)
- S Hereditary Sensory Autonomic Neuropathy, Acral Mutilation Syndrome, AMS (GDNF-AS)
- S Juvenile-Onset Polyneuropathy, Leonberger Polyneuropathy 1, LPN1 (LPN1, ARHGEF10)
- 🗸 Juvenile Myoclonic Epilepsy (DIRAS1)
- Suvenile-Onset Polyneuropathy, Leonberger Polyneuropathy 2, LPN2 (GJA9)
- Spongy Degeneration with Cerebellar Ataxia 1, SDCA1, SeSAME/EAST Syndrome (KCNJ10)
- Spongy Degeneration with Cerebellar Ataxia 2, SDCA2 (ATP1B2)
- Oilated Cardiomyopathy, DCM1 (PDK4)
- Oilated Cardiomyopathy, DCM2 (TTN)
- 📀 Long QT Syndrome (KCNQ1)
- Cardiomyopathy and Juvenile Mortality (YARS2)
- Spaniel Variant 1) Muscular Dystrophy (DMD, Cavalier King Charles Spaniel Variant 1)
- Source of the second se
- S Muscular Dystrophy (DMD Golden Retriever Variant)
- **Simb Girdle Muscular Dystrophy (SGCD, Boston Terrier Variant)**
- Sullrich-like Congenital Muscular Dystrophy (COL6A3, Labrador Variant)
- Centronuclear Myopathy (PTPLA)
- C Exercise-Induced Collapse (DNM1)
- S Inherited Myopathy of Great Danes (BIN1)
- 🛇 Myostatin Deficiency, Bully Whippet Syndrome (MSTN)

- S Myotonia Congenita (CLCN1 Exon 7)
- 📀 Myotonia Congenita (CLCN1 Exon 23)
- 📀 Myotubular Myopathy 1, X-linked Myotubular Myopathy, XL-MTM (MTM1, Labrador Variant)
- 🔇 Inflammatory Myopathy (SLC25A12)
- 🔇 Hypocatalasia, Acatalasemia (CAT)
- S Pyruvate Dehydrogenase Deficiency (PDP1)
- 📀 Malignant Hyperthermia (RYR1)
- S Imerslund-Grasbeck Syndrome, Selective Cobalamin Malabsorption (CUBN Exon 53)
- S Imerslund-Grasbeck Syndrome, Selective Cobalamin Malabsorption (CUBN Exon 8)
- S Inherited Selected Cobalamin Malabsorption with Proteinuria (CUBN)
- C Lundehund Syndrome (LEPREL1)
- Congenital Myasthenic Syndrome (CHAT)
- Congenital Myasthenic Syndrome (COLQ)
- Congenital Myasthenic Syndrome (CHRNE)
- Congenital Myasthenic Syndrome (COLQ)
- S Myasthenia Gravis Like Syndrome (CHRNE)
- C Episodic Falling Syndrome (BCAN)
- 📀 Paroxysmal Dyskinesia, PxD (PGIN)
- **Orgonal States and St**
- S Dystrophic Epidermolysis Bullosa (COL7A1)
- Sullosa (COL7A1)
- 交 Ectodermal Dysplasia, Skin Fragility Syndrome (PKP1)
- 🗸 Ichthyosis, Epidermolytic Hyperkeratosis (KRT10)
- 📀 Ichthyosis (PNPLA1)

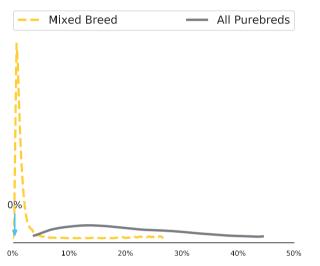
- 📀 Ichthyosis (SLC27A4)
- 📀 Ichthyosis (NIPAL4)
- S Hereditary Footpad Hyperkeratosis (FAM83G)
- S Hereditary Footpad Hyperkeratosis (DSG1)
- 📀 Hereditary Nasal Parakeratosis (SUV39H2)
- S Musladin-Lueke Syndrome (ADAMTSL2)
- 📀 Oculocutaneous Albinism, OCA (Pekingese Type)
- 🍼 Bald Thigh Syndrome (IGFBP5)
- 🔇 Lethal Acrodermatitis (MKLN1)
- Sehlers Danlos (Doberman) (ADAMTS2)
- Cleft Lip and/or Cleft Palate (ADAMTS20)
- Section 2017 President All Contract Contract Contract (VDR)
- Steogenesis Imperfecta, Brittle Bone Disease (COL1A2)
- SERPINH1) Osteogenesis Imperfecta, Brittle Bone Disease (SERPINH1)
- Solution Contemporation Strength Streng
- 📀 Osteochondrodysplasia, Skeletal Dwarfism (SLC13A1)
- Skeletal Dysplasia 2, SD2 (COL11A2)
- Craniomandibular Osteopathy, CMO (SLC37A2)
- Saine Syndrome, Canine Dental Hypomineralization Syndrome (FAM20C)
- Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type I IVDD (FGF4 retrogene -CFA12)
- Chondrodystrophy, Norwegian Elkhound and Karelian Bear Dog Variant (ITGA10)

Coefficient of Inbreeding (COI)

Genetic Result: 0%

Our genetic COI measures the proportion of your dog's genome (her genes) where the genes on the mother's side are identical by descent to those on the father's side. The higher your dog's coefficient of inbreeding (the percentage), the more inbred your dog is.

Your Dog's COI



This graph represents where your dog's inbreeding levels fall on a scale compared to both dogs with a similar breed makeup to her (the yellow dotted line) and all purebred dogs (the grey line).

More on the Science

Embark scientists, along with our research partners at Cornell University, have shown the impact of inbreeding on longevity and fertility and developed a state-of-the-art, peer-reviewed method for accurately measuring COI and predicting average COI in litters.

Citations

Sams & Boyko 2019 "Fine-Scale Resolution of Runs of Homozygosity Reveal Patterns of Inbreeding and Substantial Overlap with Recessive Disease Genotypes in Domestic Dogs" (https://www.ncbi.nlm.nih.gov/pubmed/30429214)

Chu et al 2019 "Inbreeding depression causes reduced fecundity in Golden Retrievers" (https://link.springer.com/article/10.1007/s00335-019-09805-4)

Yordy et al 2019 "Body size, inbreeding, and lifespan in domestic dogs" (https://www.semanticscholar.org/paper/Body-size%2C-inbreeding%2C-and-lifespan-indomestic-Yordy-Kraus/61d0fa7a71afb26f547f0fb7ff71e23a14d19d2c)

About Embark

Embark Veterinary is a canine genetics company offering research-grade genetic tests to pet owners and breeders. Every Embark test examines over 200,000 genetic markers, and provides results for over 200 genetic health conditions, breed identification, clinical tools, and more.

Embark is a research partner of the Cornell University College of Veterinary Medicine and collaborates with scientists and registries to accelerate genetic research in canine health. We make it easy for customers and vets to understand, share and make use of their dog's unique genetic profile to improve canine health and happiness.

Learn more at embarkvet.com

Veterinarians and hospitals can send inquiries to veterinarians@embarkvet.com.