

BREED ANCESTRY



GENETIC STATS

Predicted adult weight: **13 lbs**

TEST DETAILS

Kit number: EM-55473554

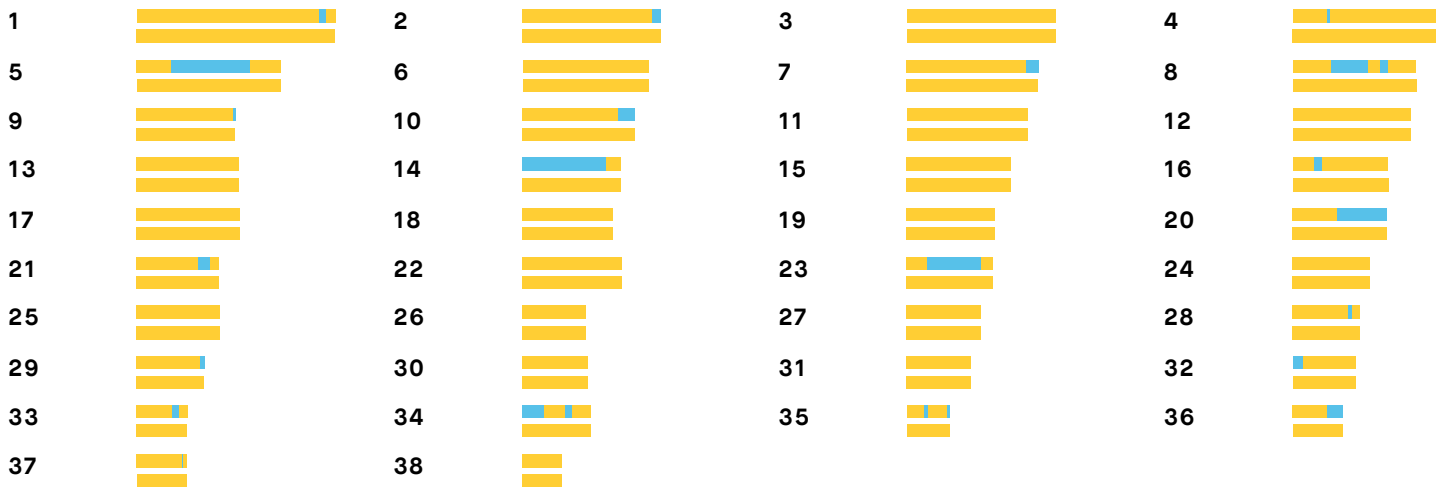
Swab number: 31220612405298

BREED ANCESTRY BY CHROMOSOME

Our advanced test identifies from where Ringo inherited every part of the chromosome pairs in his genome.

Breed colors:

Poodle (Small) **Poodle (Standard)**



POODLE (SMALL)



Miniature and toy poodles are varieties of the poodle breed which originated in Germany in the 15th century. Unlike the larger standard poodle (>15 inches tall), these small poodles were not developed for hunting---except for truffles!---and were generally used as lap dogs and companions. Small poodles are frequently used to create designer dogs like Schnoodles and Maltipoos with low-shedding, hypoallergenic coats. All poodles are highly intelligent and energetic, and need daily exercise and stimulation. They are overall healthy dogs, although heritable eye disease, epilepsy and allergies are relatively common, and toy poodles also have a heightened risk of accidents/trauma due to their small size.

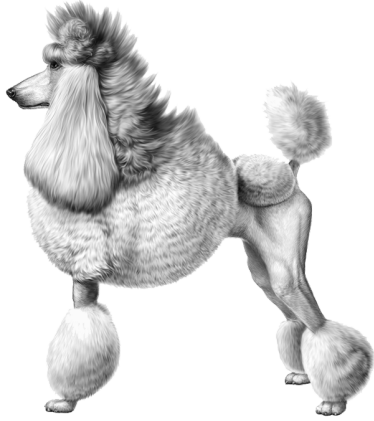
Alternative Names

Toy Poodle, Miniature Poodle

Fun Fact

Although Toy Poodles are the most popular dog breed in Japan, Poodles as a group are the eight most popular breed in the US, with miniature poodles being the most common variety.

POODLE (STANDARD)

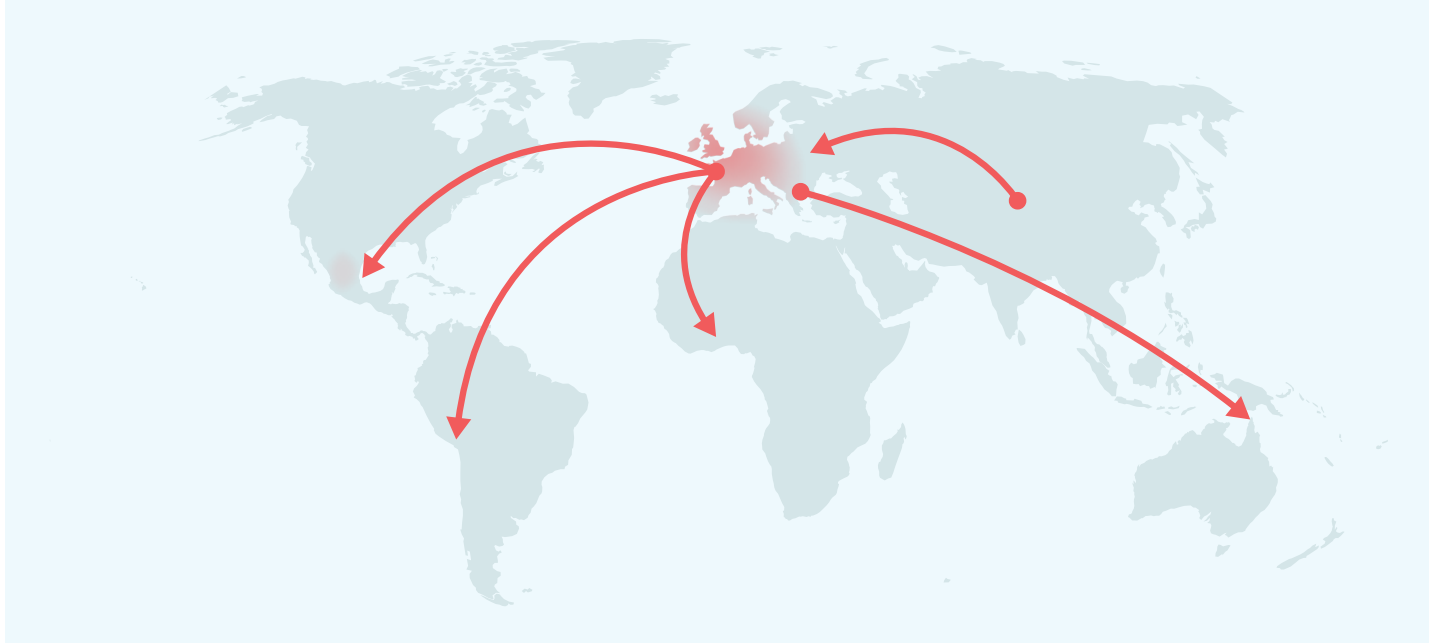


The Standard Poodle is a popular, water-loving dog used for centuries as a bird dog and popular pet. Poodles were established in Germany by the 15th century. Oddly enough, they are the national dog breed of France, and they were the most popular breed of dog in the United States throughout the 1960s and 70s. They're still quite popular today, owing to their intelligence, trainability, and non-shedding coats. Although well-known for their fancy fur, they're one of the most intelligent breeds of dog and require a lot of exercise and stimulation.

Fun Fact

From 1989 to 1991, John Suter raced a team of Poodles in the Iditarod. Although his teams placed in the back half of the pack, he managed to win \$2,000 in prize money before retiring his poodle team. The Iditarod has since changed its rules to specify that only northern dog breeds can compete.

MATERNAL LINE



Through Ringo's mitochondrial DNA we can trace his mother's ancestry back to where dogs and people first became friends. This map helps you visualize the routes that his ancestors took to your home. Their story is described below the map.

HAPLOGROUP: A1b

This female lineage was very likely one of the original lineages in the wolves that were first domesticated into dogs in Central Asia about 15,000 years ago. Since then, the lineage has been very successful and travelled the globe! Dogs from this group are found in ancient Bronze Age fossils in the Middle East and southern Europe. By the end of the Bronze Age, it became exceedingly common in Europe. These dogs later became many of the dogs that started some of today's most popular breeds, like German Shepherds, Pugs, Whippets, English Sheepdogs and Miniature Schnauzers. During the period of European colonization, the lineage became even more widespread as European dogs followed their owners to far-flung places like South America and Oceania. It's now found in many popular breeds as well as village dogs across the world!

HAPLOTYPE: A268

Part of the large A1b haplogroup, this uncommon haplotype occurs most frequently in Labrador Retrievers and has been spotted less often in Golden Retrievers, Poodles, and Chihuahuas.

PATERNAL LINE



Through Ringo's Y chromosome we can trace his father's ancestry back to where dogs and people first became friends. This map helps you visualize the routes that his ancestors took to your home. Their story is described below the map.

HAPLOGROUP: A1b

For most of dog history, this haplogroup was probably quite rare. However, a couple hundred years ago it seems to have found its way into a prized male guard dog in Europe who had many offspring, including the ancestors of many European guard breeds such as Doberman Pinchers, St. Bernards, and Great Danes. Despite being rare, many of the most imposing dogs on Earth have it; strangely, so do many Pomeranians! Perhaps this explains why some Poms are so tough, acting like they're ten times their actual size! This lineage is most commonly found in working dogs, in particular guard dogs. With origins in Europe, it spread widely across other regions as Europeans took their dogs across the world.

HAPLOTYPE: Ha.7

Part of the A1b haplogroup, this haplotype is found in village dogs from Lebanon and Indonesia. Among breeds, it is also found in Miniature Schnauzer and Toy Poodle.

TRAITS: COAT COLOR

TRAIT	RESULT
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E Locus (MC1R)

The E Locus determines if and where a dog can produce dark (black or brown) hair. Dogs with two copies of the recessive **e** allele do not produce dark hairs at all, and will be "red" over their entire body. The shade of red, which can range from a deep copper to yellow/gold to cream, is dependent on other genetic factors including the Intensity loci. In addition to determining if a dog can develop dark hairs at all, the E Locus can give a dog a black "mask" or "widow's peak," unless the dog has overriding coat color genetic factors. Dogs with one or two copies of the **Em** allele usually have a melanistic mask (dark facial hair as commonly seen in the German Shepherd and Pug). Dogs with no copies of **Em** but one or two copies of the **Eg** allele usually have a melanistic "widow's peak" (dark forehead hair as commonly seen in the Afghan Hound and Borzoi, where it is called either "grizzle" or "domino").

**No dark hairs
anywhere (ee)**

K Locus (CBD103)

The K Locus **K^B** allele "overrides" the A Locus, meaning that it prevents the A Locus genotype from affecting coat color. For this reason, the **K^B** allele is referred to as the "dominant black" allele. As a result, dogs with at least one **K^B** allele will usually have solid black or brown coats (or red/cream coats if they are **ee** at the E Locus) regardless of their genotype at the A Locus, although several other genes could impact the dog's coat and cause other patterns, such as white spotting. Dogs with the **k^Yk^Y** genotype will show a coat color pattern based on the genotype they have at the A Locus. Dogs who test as **K^Bk^Y** may be brindle rather than black or brown.

Not expressed (K^BK^B)

TRAITS: COAT COLOR (CONTINUED)

TRAIT	RESULT
<p>Intensity Loci LINKAGE</p> <p>Areas of a dog's coat where dark (black or brown) pigment is not expressed either contain red/yellow pigment, or no pigment at all. Five locations across five chromosomes explain approximately 70% of red pigmentation "intensity" variation across all dogs. Dogs with a result of Intense Red Pigmentation will likely have deep red hair like an Irish Setter or "apricot" hair like some Poodles, dogs with a result of Intermediate Red Pigmentation will likely have tan or yellow hair like a Soft-Coated Wheaten Terrier, and dogs with Dilute Red Pigmentation will likely have cream or white hair like a Samoyed. Because the mutations we test may not directly cause differences in red pigmentation intensity, we consider this to be a linkage test.</p>	<p>Any pigmented hair likely apricot or red (Intense Red Pigmentation)</p>
<p>A Locus (ASIP)</p> <p>The A Locus controls switching between black and red pigment in hair cells, but it will only be expressed in dogs that are not ee at the E Locus and are k^Yk^Y at the K Locus. Sable (also called "Fawn") dogs have a mostly or entirely red coat with some interspersed black hairs. Agouti (also called "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.</p>	<p>Not expressed (a^Ya^t)</p>
<p>D Locus (MLPH)</p> <p>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, will have all black pigment lightened ("diluted") to gray, or brown pigment lightened to lighter brown in their hair, skin, and sometimes eyes. There are many breed-specific names for these dilute colors, such as "blue", "charcoal", "fawn", "silver", and "Isabella". Note that in certain breeds, dilute dogs have a higher incidence of Color Dilution Alopecia. Dogs with one d allele will not be dilute, but can pass the d allele on to their puppies.</p>	<p>Not expressed (DD)</p>

TRAITS: COAT COLOR (CONTINUED)

TRAIT	RESULT
<p>Cocoa (HPS3)</p> <p>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 allele 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.</p>	<p>No co alleles, not expressed (NN)</p>
<p>B Locus (TYRP1)</p> <p>Dogs with two copies of the b allele produce brown pigment instead of black in both their hair and skin. Dogs with one copy of the b allele will produce black pigment, but can pass the b allele on to their puppies. E Locus ee dogs that carry two b alleles will have red or cream coats, but have brown noses, eye rims, and footpads (sometimes referred to as "Dudley Nose" in Labrador Retrievers). "Liver" or "chocolate" is the preferred color term for brown in most breeds; in the Doberman Pinscher it is referred to as "red".</p>	<p>Likely black colored nose/feet (BB)</p>
<p>Saddle Tan (RALY)</p> <p>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 ll 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.</p>	<p>Not expressed (NI)</p>
<p>S Locus (MITF)</p> <p>The S Locus determines white spotting and pigment distribution. MITF controls where pigment is produced, and an insertion in the MITF gene causes a loss of pigment in the coat and skin, resulting in white hair and/or pink skin. Dogs with two copies of this variant will likely have breed-dependent white patterning, with a nearly white, parti, or piebald coat. Dogs with one copy of this variant will have more limited white spotting and may be considered flash, parti or piebald. This MITF variant does not explain all white spotting patterns in dogs and other variants are currently being researched. Some dogs may have small amounts of white on the paws, chest, face, or tail regardless of their S Locus genotype.</p>	<p>Likely solid colored, but may have small amounts of white (Ssp)</p>

TRAITS: COAT COLOR (CONTINUED)

TRAIT	RESULT
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M Locus (PMEL)

Merle coat patterning is common to several dog breeds including the Australian Shepherd, Catahoula Leopard Dog, and Shetland Sheepdog, among many others. Merle arises from an unstable SINE insertion (which we term the "M*" allele) that disrupts activity of the pigmentary gene PMEL, leading to mottled or patchy coat color. Dogs with an **M*m** result are likely to be phenotypically 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 be phenotypically merle or double merle. Dogs with an **mm** result have no merle alleles and are unlikely to have a merle coat pattern.

No merle alleles (mm)

Note that Embark does not currently distinguish between the recently described cryptic, atypical, atypical+, classic, and harlequin merle alleles. Our merle test only detects the presence, but not the length of the SINE insertion. We do not recommend making breeding decisions on this result alone. Please pursue further testing for allelic distinction prior to breeding decisions.

R Locus (USH2A) LINKAGE

The R Locus regulates the presence or absence of the roan coat color pattern. Partial duplication of the USH2A gene is strongly associated with this coat pattern. Dogs with at least one **R** allele will likely have roaning on otherwise uniformly unpigmented white areas. Roan appears in white areas controlled by the S Locus but not in other white or cream areas created by other loci, such as the E Locus with **ee** along with Dilute Red Pigmentation by I Locus (for example, in Samoyeds). Mechanisms for controlling the extent of roaning are currently unknown, and roaning can appear in a uniform or non-uniform pattern. Further, non-uniform roaning may appear as ticked, and not obviously roan. The roan pattern can appear with or without ticking.

Likely no impact on coat pattern (rr)

H Locus (Harlequin)

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. This trait is thought to be homozygous lethal; a living dog with an **HH** genotype has never been found.

No harlequin alleles (hh)

TRAITS: OTHER COAT TRAITS

TRAIT	RESULT
Furnishings (RSPO2) LINKAGE	
Dogs with one or two copies of the F allele have "furnishings": the mustache, beard, and eyebrows characteristic of breeds like the Schnauzer, Scottish Terrier, and Wire Haired Dachshund. A dog with two I alleles will not have furnishings, which is sometimes called an "improper coat" in breeds where furnishings are part of the breed standard. The mutation is a genetic insertion which we measure indirectly using a linkage test highly correlated with the insertion.	Likely furnished (mustache, beard, and/or eyebrows) (FF)
Coat Length (FGF5)	
The FGF5 gene is known to affect hair length in many different species, including cats, dogs, mice, and humans. In dogs, the T allele confers a long, silky haircoat as observed in the Yorkshire Terrier and the Long Haired Whippet. The ancestral G allele causes a shorter coat as seen in the Boxer or the American Staffordshire Terrier. In certain breeds (such as Corgi), the long haircoat is described as "fluff."	Likely long coat (TT)
Shedding (MC5R)	
Dogs with at least one copy of the ancestral C allele, like many Labradors and German Shepherd Dogs, are heavy or seasonal shedders, while those with two copies of the T allele, including many Boxers, Shih Tzus and Chihuahuas, tend to be lighter shedders. Dogs with furnished/wire-haired coats caused by RSPO2 (the furnishings gene) tend to be low shedders regardless of their genotype at this gene.	Likely light shedding (CT)
Coat Texture (KRT71)	
Dogs with a long coat and at least one copy of the T allele have a wavy or curly coat characteristic of Poodles and Bichon Frises. Dogs with two copies of the ancestral C allele are likely to have a straight coat, but there are other factors that can cause a curly coat, for example if they at least one F allele for the Furnishings (RSPO2) gene then they are likely to have a curly coat. Dogs with short coats may carry one or two copies of the T allele but still have straight coats.	Likely curly coat (TT)

TRAITS: OTHER COAT TRAITS (CONTINUED)

TRAIT	RESULT
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Hairlessness (FOXI3) LINKAGE

A duplication in the FOXI3 gene causes hairlessness over most of the body as well as changes in tooth shape and number. This mutation occurs in Peruvian Inca Orchid, Xoloitzcuintli (Mexican Hairless), and Chinese Crested (other hairless breeds have different mutations). Dogs with the **NDup** genotype are likely to be hairless while dogs with the **NN** genotype are likely to have a normal coat. The **DupDup** genotype has never been observed, suggesting that dogs with that genotype cannot survive to birth. Please note that this is a linkage test, so it may not be as predictive as direct tests of the mutation in some lines.

Very unlikely to be hairless (NN)

Hairlessness (SGK3)

Hairlessness in the American Hairless Terrier arises from a mutation in the SGK3 gene. Dogs with the **DD** result are likely to be hairless. Dogs with the **ND** genotype will have a normal coat, but can pass the **D** variant on to their offspring.

Very unlikely to be hairless (NN)

Oculocutaneous Albinism Type 2 (SLC45A2) LINKAGE

Dogs with two copies **DD** of this deletion in the SLC45A2 gene have oculocutaneous albinism (OCA), also known as Doberman Z Factor Albinism, a recessive condition characterized by severely reduced or absent pigment in the eyes, skin, and hair. Affected dogs sometimes suffer from vision problems due to lack of eye pigment (which helps direct and absorb ambient light) and are prone to sunburn. Dogs with a single copy of the deletion **ND** will not be affected but can pass the mutation on to their offspring. This particular mutation can be traced back to a single white Doberman Pinscher born in 1976, and it has only been observed in dogs descended from this individual. Please note that this is a linkage test, so it may not be as predictive as direct tests of the mutation in some lines.

Likely not albino (NN)

TRAITS: OTHER BODY FEATURES

TRAIT	RESULT
<p>Muzzle Length (BMP3)</p> <p>Dogs in medium-length muzzle (mesocephalic) breeds like Staffordshire Terriers and Labradors, and long muzzle (dolichocephalic) breeds like Whippet and Collie have one, or more commonly two, copies of the ancestral C allele. Dogs in many short-length muzzle (brachycephalic) breeds such as the English Bulldog, Pug, and Pekingese have two copies of the derived A allele. At least five different genes affect muzzle length in dogs, with BMP3 being the only one with a known causal mutation. For example, the skull shape of some breeds, including the dolichocephalic Scottish Terrier or the brachycephalic Japanese Chin, appear to be caused by other genes. Thus, dogs may have short or long muzzles due to other genetic factors that are not yet known to science.</p>	<p>Likely medium or long muzzle (CC)</p>
<p>Tail Length (T)</p> <p>Whereas most dogs have two C alleles and a long tail, dogs with one G allele are likely to have a bobtail, which is an unusually short or absent tail. This mutation causes natural bobtail in many breeds including the Pembroke Welsh Corgi, the Australian Shepherd, and the Brittany Spaniel. Dogs with GG genotypes have not been observed, suggesting that dogs with the GG genotype do not survive to birth. Please note that this mutation does not explain every natural bobtail! While certain lineages of Boston Terrier, English Bulldog, Rottweiler, Miniature Schnauzer, Cavalier King Charles Spaniel, and Parson Russell Terrier, and Dobermans are born with a natural bobtail, these breeds do not have this mutation. This suggests that other unknown genetic mutations can also lead to a natural bobtail.</p>	<p>Likely normal-length tail (CC)</p>
<p>Hind Dewclaws (LMBR1)</p> <p>Common in certain breeds such as the Saint Bernard, hind dewclaws are extra, nonfunctional digits located midway between a dog's paw and hock. Dogs with at least one copy of the T allele have about a 50% chance of having hind dewclaws. Note that other (currently unknown to science) mutations can also cause hind dewclaws, so some CC or TC dogs will have hind dewclaws.</p>	<p>Unlikely to have hind dew claws (CC)</p>

TRAITS: OTHER BODY FEATURES (CONTINUED)

TRAIT	RESULT
<p>Chondrodysplasia (Chr. 18 FGF4 Retrogene)</p> <p>Dogs with one or two copies of the I allele will exhibit a short-legged trait known as chondrodysplasia (CDPA). CDPA is a breed-defining characteristic of many breeds exhibiting the "short-legged, long-bodied" appearance known as disproportionate dwarfism, including the corgi, dachshund and basset hound. The impact of the I allele on leg length is additive. Therefore, dogs with the II result display the largest reduction in leg length. Dogs with the NI genotype will have an intermediate leg length, while dogs with the NN result will not exhibit leg shortening due to this variant. Breeds that display disproportionate dwarfism also frequently inherit a genetic variant known as the chondrodystrophy (CDDY) variant. The CDDY variant also shortens legs (in a less significant amount than CDPA) but, secondarily, increases the risk of Type I Intervertebral Disc Disease (IVDD). Test results for CDDY are listed in this dog's health testing results under "Intervertebral Disc Disease (Type I)". In contrast, the CDPA variant has NOT been shown to increase the risk of IVDD.</p>	<p>Not indicative of chondrodysplasia (normal leg length) (NN)</p>
<p>Blue Eye Color (ALX4) LINKAGE</p> <p>Embark researchers discovered this large duplication associated with blue eyes in Arctic breeds like Siberian Husky as well as tri-colored (non-merle) Australian Shepherds. Dogs with at least one copy of the duplication (Dup) are more likely to have at least one blue eye. Some dogs with the duplication may have only one blue eye (complete heterochromia) or may not have blue eyes at all; nevertheless, they can still pass the duplication and the trait to their offspring. NN dogs do not carry this duplication, but may have blue eyes due to other factors, such as merle. Please note that this is a linkage test, so it may not be as predictive as direct tests of the mutation in some lines.</p>	<p>Less likely to have blue eyes (NN)</p>
<p>Back Muscling & Bulk, Large Breed (ACSL4)</p> <p>The T allele is associated with heavy muscling along the back and trunk in characteristically "bulky" large-breed dogs including the Saint Bernard, Bernese Mountain Dog, Greater Swiss Mountain Dog, and Rottweiler. The "bulky" T allele is absent from leaner shaped large breed dogs like the Great Dane, Irish Wolfhound, and Scottish Deerhound, which are fixed for the ancestral C allele. Note that this mutation does not seem to affect muscling in small or even mid-sized dog breeds with notable back muscling, including the American Staffordshire Terrier, Boston Terrier, and the English Bulldog.</p>	<p>Likely normal muscling (CC)</p>

TRAITS: BODY SIZE

TRAIT	RESULT
Body Size (IGF1) The I allele is associated with smaller body size.	Smaller (II)
Body Size (IGFR1) The A allele is associated with smaller body size.	Intermediate (GA)
Body Size (STC2) The A allele is associated with smaller body size.	Intermediate (TA)
Body Size (GHR - E191K) The A allele is associated with smaller body size.	Smaller (AA)
Body Size (GHR - P177L) The T allele is associated with smaller body size.	Larger (CC)

TRAITS: PERFORMANCE

TRAIT	RESULT
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Altitude Adaptation (EPAS1)

This mutation causes dogs to be especially tolerant of low oxygen environments (hypoxia), such as those found at high elevations. Dogs with at least one **A** allele are less susceptible to "altitude sickness." This mutation was originally identified in breeds from high altitude areas such as the Tibetan Mastiff.

Normal altitude tolerance (GG)**Appetite (POMC) LINKAGE**

This mutation in the POMC gene is found primarily in Labrador and Flat Coated Retrievers. Compared to dogs with no copies of the mutation (**NN**), dogs with one (**ND**) or two (**DD**) copies of the mutation are more likely to have high food motivation, which can cause them to eat excessively, have higher body fat percentage, and be more prone to obesity. Read more about the genetics of POMC, and learn how you can contribute to research, in our blog post (<https://embarkvet.com/resources/blog/pomc-dogs/>). We measure this result using a linkage test.

Normal food motivation (NN)

HEALTH REPORT

How to interpret Ringo's genetic health results:

If Ringo 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 Ringo 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.

Summary

Of the 256 genetic health risks we analyzed, we found 3 results that you should learn about.

Increased risk results (1)

Intervertebral Disc Disease (Type I)

Notable results (2)

ALT Activity

Dilated Cardiomyopathy, DCM1





Clear results

Breed-relevant (6)

Other (246)



















BREED-RELEVANT RESULTS

Research studies indicate that these results are more relevant to dogs like Ringo, and may influence his chances of developing certain health conditions.

 Intervertebral Disc Disease (Type I) (FGF4 retrogene - CFA12)	Increased risk
 Degenerative Myelopathy, DM (SOD1A)	Clear
 GM2 Gangliosidosis (HEXB, Poodle Variant)	Clear
 Neonatal Encephalopathy with Seizures, NEWS (ATF2)	Clear
 Osteochondrodysplasia (SLC13A1, Poodle Variant)	Clear
 Progressive Retinal Atrophy, prcd (PRCD Exon 1)	Clear
 Von Willebrand Disease Type I, Type I vWD (VWF)	Clear

OTHER RESULTS



















Research has not yet linked these conditions to dogs with similar breeds to Ringo. Review any increased risk or notable results to understand his potential risk and recommendations.

 ALT Activity (GPT)	Notable
 Dilated Cardiomyopathy, DCM1 (PDK4, Doberman Pinscher Variant 1)	Notable
 2-DHA Kidney & Bladder Stones (APRT)	Clear
 Acral Mutilation Syndrome (GDNF-AS, Spaniel and Pointer Variant)	Clear
 Alaskan Husky Encephalopathy (SLC19A3)	Clear
 Alaskan Malamute Polyneuropathy, AMPN (NDRG1 SNP)	Clear
 Alexander Disease (GFAP)	Clear
 Anhidrotic Ectodermal Dysplasia (EDA Intron 8)	Clear
 Autosomal Dominant Progressive Retinal Atrophy (RHO)	Clear
 Bald Thigh Syndrome (IGFBP5)	Clear
 Bernard-Soulier Syndrome, BSS (GP9, Cocker Spaniel Variant)	Clear
 Bully Whippet Syndrome (MSTN)	Clear
 Canine Elliptocytosis (SPTB Exon 30)	Clear
 Canine Fucosidosis (FUCA1)	Clear
 Canine Leukocyte Adhesion Deficiency Type I, CLAD I (ITGB2, Setter Variant)	Clear
 Canine Leukocyte Adhesion Deficiency Type III, CLAD III (FERMT3, German Shepherd Variant)	Clear
 Canine Multifocal Retinopathy, cmr1 (BEST1 Exon 2)	Clear
 Canine Multifocal Retinopathy, cmr2 (BEST1 Exon 5, Coton de Tulear Variant)	Clear



















OTHER RESULTS

- | | |
|--|-------|
| <input checked="" type="checkbox"/> Canine Multifocal Retinopathy, cmr3 (BEST1 Exon 10 Deletion, Finnish and Swedish Lapphund, Lapponian Herder Variant) | Clear |
| <input checked="" type="checkbox"/> Canine Multiple System Degeneration (SERAC1 Exon 4, Chinese Crested Variant) | Clear |
| <input checked="" type="checkbox"/> Canine Multiple System Degeneration (SERAC1 Exon 15, Kerry Blue Terrier Variant) | Clear |
| <input checked="" type="checkbox"/> Cardiomyopathy and Juvenile Mortality (YARS2) | Clear |
| <input checked="" type="checkbox"/> Centronuclear Myopathy, CNM (PTPLA) | Clear |
| <input checked="" type="checkbox"/> Cerebellar Hypoplasia (VLDLR, Eurasier Variant) | Clear |
| <input checked="" type="checkbox"/> Chondrodystrophy (ITGA10, Norwegian Elkhound and Karelian Bear Dog Variant) | Clear |
| <input checked="" type="checkbox"/> Cleft Lip and/or Cleft Palate (ADAMTS20, Nova Scotia Duck Tolling Retriever Variant) | Clear |
| <input checked="" type="checkbox"/> Cleft Palate, CP1 (DLX6 intron 2, Nova Scotia Duck Tolling Retriever Variant) | Clear |
| <input checked="" type="checkbox"/> Cobalamin Malabsorption (CUBN Exon 8, Beagle Variant) | Clear |
| <input checked="" type="checkbox"/> Cobalamin Malabsorption (CUBN Exon 53, Border Collie Variant) | Clear |
| <input checked="" type="checkbox"/> Collie Eye Anomaly (NHEJ1) | Clear |
| <input checked="" type="checkbox"/> Complement 3 Deficiency, C3 Deficiency (C3) | Clear |
| <input checked="" type="checkbox"/> Congenital Cornification Disorder (NSDHL, Chihuahua Variant) | Clear |
| <input checked="" type="checkbox"/> Congenital Hypothyroidism (TPO, Rat, Toy, Hairless Terrier Variant) | Clear |
| <input checked="" type="checkbox"/> Congenital Hypothyroidism (TPO, Tenterfield Terrier Variant) | Clear |
| <input checked="" type="checkbox"/> Congenital Hypothyroidism with Goiter (TPO Intron 13, French Bulldog Variant) | Clear |
| <input checked="" type="checkbox"/> Congenital Hypothyroidism with Goiter (SLC5A5, Shih Tzu Variant) | Clear |



















OTHER RESULTS

 Congenital Macrothrombocytopenia (TUBB1 Exon 1, Cairn and Norfolk Terrier Variant)	Clear
 Congenital Myasthenic Syndrome, CMS (COLQ, Labrador Retriever Variant)	Clear
 Congenital Myasthenic Syndrome, CMS (COLQ, Golden Retriever Variant)	Clear
 Congenital Myasthenic Syndrome, CMS (CHAT, Old Danish Pointing Dog Variant)	Clear
 Congenital Myasthenic Syndrome, CMS (CHRNE, Jack Russell Terrier Variant)	Clear
 Congenital Stationary Night Blindness (LRIT3, Beagle Variant)	Clear
 Congenital Stationary Night Blindness (RPE65, Briard Variant)	Clear
 Craniomandibular Osteopathy, CMO (SLC37A2)	Clear
 Craniomandibular Osteopathy, CMO (SLC37A2 Intron 16, Basset Hound Variant)	Clear
 Cystinuria Type I-A (SLC3A1, Newfoundland Variant)	Clear
 Cystinuria Type II-A (SLC3A1, Australian Cattle Dog Variant)	Clear
 Cystinuria Type II-B (SLC7A9, Miniature Pinscher Variant)	Clear
 Day Blindness (CNGB3 Deletion, Alaskan Malamute Variant)	Clear
 Day Blindness (CNGA3 Exon 7, German Shepherd Variant)	Clear
 Day Blindness (CNGA3 Exon 7, Labrador Retriever Variant)	Clear
 Day Blindness (CNGB3 Exon 6, German Shorthaired Pointer Variant)	Clear
 Deafness and Vestibular Syndrome of Dobermans, DVDob, DINGS (MYO7A)	Clear
 Demyelinating Polyneuropathy (SBF2/MTRM13)	Clear



















OTHER RESULTS

 Dental-Skeletal-Retinal Anomaly (MIA3, Cane Corso Variant)	Clear
 Diffuse Cystic Renal Dysplasia and Hepatic Fibrosis (INPP5E Intron 9, Norwich Terrier Variant)	Clear
 Dilated Cardiomyopathy, DCM (RBM20, Schnauzer Variant)	Clear
 Dilated Cardiomyopathy, DCM2 (TTN, Doberman Pinscher Variant 2)	Clear
 Disproportionate Dwarfism (PRKG2, Dogo Argentino Variant)	Clear
 Dry Eye Curly Coat Syndrome (FAM83H Exon 5)	Clear
 Dystrophic Epidermolysis Bullosa (COL7A1, Central Asian Shepherd Dog Variant)	Clear
 Dystrophic Epidermolysis Bullosa (COL7A1, Golden Retriever Variant)	Clear
 Early Bilateral Deafness (LOXHD1 Exon 38, Rottweiler Variant)	Clear
 Early Onset Adult Deafness, EOAD (EPS8L2 Deletion, Rhodesian Ridgeback Variant)	Clear
 Early Onset Cerebellar Ataxia (SEL1L, Finnish Hound Variant)	Clear
 Ehlers Danlos (ADAMTS2, Doberman Pinscher Variant)	Clear
 Enamel Hypoplasia (ENAM Deletion, Italian Greyhound Variant)	Clear
 Enamel Hypoplasia (ENAM SNP, Parson Russell Terrier Variant)	Clear
 Episodic Falling Syndrome (BCAN)	Clear
 Exercise-Induced Collapse, EIC (DNM1)	Clear
 Factor VII Deficiency (F7 Exon 5)	Clear
 Factor XI Deficiency (F11 Exon 7, Kerry Blue Terrier Variant)	Clear

OTHER RESULTS

 Familial Nephropathy (COL4A4 Exon 3, Cocker Spaniel Variant)	Clear
 Familial Nephropathy (COL4A4 Exon 30, English Springer Spaniel Variant)	Clear
 Fanconi Syndrome (FAN1, Basenji Variant)	Clear
 Fetal-Onset Neonatal Neuroaxonal Dystrophy (MFN2, Giant Schnauzer Variant)	Clear
 Glanzmann's Thrombasthenia Type I (ITGA2B Exon 13, Great Pyrenees Variant)	Clear
 Glanzmann's Thrombasthenia Type I (ITGA2B Exon 12, Otterhound Variant)	Clear
 Globoid Cell Leukodystrophy, Krabbe disease (GALC Exon 5, Terrier Variant)	Clear
 Glycogen Storage Disease Type IA, Von Gierke Disease, GSD IA (G6PC, Maltese Variant)	Clear
 Glycogen Storage Disease Type IIIA, GSD IIIA (AGL, Curly Coated Retriever Variant)	Clear
 Glycogen storage disease Type VII, Phosphofructokinase Deficiency, PFK Deficiency (PFKM, Whippet and English Springer Spaniel Variant)	Clear
 Glycogen storage disease Type VII, Phosphofructokinase Deficiency, PFK Deficiency (PFKM, Wachtelhund Variant)	Clear
 GM1 Gangliosidosis (GLB1 Exon 2, Portuguese Water Dog Variant)	Clear
 GM1 Gangliosidosis (GLB1 Exon 15, Shiba Inu Variant)	Clear
 GM1 Gangliosidosis (GLB1 Exon 15, Alaskan Husky Variant)	Clear
 GM2 Gangliosidosis (HEXA, Japanese Chin Variant)	Clear
 Golden Retriever Progressive Retinal Atrophy 1, GR-PRA1 (SLC4A3)	Clear
 Golden Retriever Progressive Retinal Atrophy 2, GR-PRA2 (TTC8)	Clear
 Goniodysgenesis and Glaucoma, Pectinate Ligament Dysplasia, PLD (OLFM3)	Clear



















OTHER RESULTS

 Hemophilia A (F8 Exon 11, German Shepherd Variant 1)	Clear
 Hemophilia A (F8 Exon 1, German Shepherd Variant 2)	Clear
 Hemophilia A (F8 Exon 10, Boxer Variant)	Clear
 Hemophilia B (F9 Exon 7, Terrier Variant)	Clear
 Hemophilia B (F9 Exon 7, Rhodesian Ridgeback Variant)	Clear
 Hereditary Ataxia, Cerebellar Degeneration (RAB24, Old English Sheepdog and Gordon Setter Variant)	Clear
 Hereditary Cataracts (HSF4 Exon 9, Australian Shepherd Variant)	Clear
 Hereditary Footpad Hyperkeratosis (FAM83G, Terrier and Kromfohrlander Variant)	Clear
 Hereditary Footpad Hyperkeratosis (DSG1, Rottweiler Variant)	Clear
 Hereditary Nasal Parakeratosis (SUV39H2 Intron 4, Greyhound Variant)	Clear
 Hereditary Nasal Parakeratosis, HNPk (SUV39H2)	Clear
 Hereditary Vitamin D-Resistant Rickets (VDR)	Clear
 Hypocatalasia, Acatlasemia (CAT)	Clear
 Hypomyelination and Tremors (FNIP2, Weimaraner Variant)	Clear
 Hypophosphatasia (ALPL Exon 9, Karelian Bear Dog Variant)	Clear
 Ichthyosis (NIPAL4, American Bulldog Variant)	Clear
 Ichthyosis (ASPRV1 Exon 2, German Shepherd Variant)	Clear
 Ichthyosis (SLC27A4, Great Dane Variant)	Clear



















OTHER RESULTS

<input checked="" type="checkbox"/> Ichthyosis, Epidermolytic Hyperkeratosis (KRT10, Terrier Variant)	Clear
<input checked="" type="checkbox"/> Ichthyosis, ICH1 (PNPLA1, Golden Retriever Variant)	Clear
<input checked="" type="checkbox"/> Inflammatory Myopathy (SLC25A12)	Clear
<input checked="" type="checkbox"/> Inherited Myopathy of Great Danes (BIN1)	Clear
<input checked="" type="checkbox"/> Inherited Selected Cobalamin Malabsorption with Proteinuria (CUBN, Komondor Variant)	Clear
<input checked="" type="checkbox"/> Intestinal Lipid Malabsorption (ACSL5, Australian Kelpie)	Clear
<input checked="" type="checkbox"/> Junctional Epidermolysis Bullosa (LAMA3 Exon 66, Australian Cattle Dog Variant)	Clear
<input checked="" type="checkbox"/> Junctional Epidermolysis Bullosa (LAMB3 Exon 11, Australian Shepherd Variant)	Clear
<input checked="" type="checkbox"/> Juvenile Epilepsy (LGI2)	Clear
<input checked="" type="checkbox"/> Juvenile Laryngeal Paralysis and Polyneuropathy (RAB3GAP1, Rottweiler Variant)	Clear
<input checked="" type="checkbox"/> Juvenile Myoclonic Epilepsy (DIRAS1)	Clear
<input checked="" type="checkbox"/> L-2-Hydroxyglutaricaciduria, L2HGA (L2HGDH, Staffordshire Bull Terrier Variant)	Clear
<input checked="" type="checkbox"/> Lagotto Storage Disease (ATG4D)	Clear
<input checked="" type="checkbox"/> Laryngeal Paralysis (RAPGEF6, Miniature Bull Terrier Variant)	Clear
<input checked="" type="checkbox"/> Late Onset Spinocerebellar Ataxia (CAPN1)	Clear
<input checked="" type="checkbox"/> Late-Onset Neuronal Ceroid Lipofuscinosis, NCL 12 (ATP13A2, Australian Cattle Dog Variant)	Clear
<input checked="" type="checkbox"/> Leonberger Polyneuropathy 1 (LPN1, ARHGEF10)	Clear
<input checked="" type="checkbox"/> Leonberger Polyneuropathy 2 (GJA9)	Clear



















OTHER RESULTS

 Lethal Acrodermatitis, LAD (MKLN1)	Clear
 Leukodystrophy (TSEN54 Exon 5, Standard Schnauzer Variant)	Clear
 Ligneous Membranitis, LM (PLG)	Clear
 Limb Girdle Muscular Dystrophy (SGCD, Boston Terrier Variant)	Clear
 Limb-Girdle Muscular Dystrophy 2D (SGCA Exon 3, Miniature Dachshund Variant)	Clear
 Long QT Syndrome (KCNQ1)	Clear
 Lunde hund Syndrome (LEPREL1)	Clear
 Macular Corneal Dystrophy, MCD (CHST6)	Clear
 Malignant Hyperthermia (RYR1)	Clear
 May-Hegglin Anomaly (MYH9)	Clear
 Methemoglobinemia (CYB5R3, Pit Bull Terrier Variant)	Clear
 Methemoglobinemia (CYB5R3)	Clear
 Microphthalmia (RBP4 Exon 2, Soft Coated Wheaten Terrier Variant)	Clear
 Mucopolysaccharidosis IIIB, Sanfilippo Syndrome Type B, MPS IIIB (NAGLU, Schipperke Variant)	Clear
 Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, Dachshund Variant)	Clear
 Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, New Zealand Huntaway Variant)	Clear
 Mucopolysaccharidosis Type VI, Maroteaux-Lamy Syndrome, MPS VI (ARSB Exon 5, Miniature Pinscher Variant)	Clear
 Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 3, German Shepherd Variant)	Clear



















OTHER RESULTS

 Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 5, Terrier Brasileiro Variant)	Clear
 Multiple Drug Sensitivity (ABCB1)	Clear
 Muscular Dystrophy (DMD, Cavalier King Charles Spaniel Variant 1)	Clear
 Muscular Dystrophy (DMD, Golden Retriever Variant)	Clear
 Musladin-Lueke Syndrome, MLS (ADAMTSL2)	Clear
 Myasthenia Gravis-Like Syndrome (CHRNE, Heideterrier Variant)	Clear
 Myotonia Congenita (CLCN1 Exon 23, Australian Cattle Dog Variant)	Clear
 Myotonia Congenita (CLCN1 Exon 7, Miniature Schnauzer Variant)	Clear
 Narcolepsy (HCRTR2 Exon 1, Dachshund Variant)	Clear
 Narcolepsy (HCRTR2 Intron 4, Doberman Pinscher Variant)	Clear
 Narcolepsy (HCRTR2 Intron 6, Labrador Retriever Variant)	Clear
 Nemaline Myopathy (NEB, American Bulldog Variant)	Clear
 Neonatal Cerebellar Cortical Degeneration (SPTBN2, Beagle Variant)	Clear
 Neonatal Interstitial Lung Disease (LAMP3)	Clear
 Neuroaxonal Dystrophy, NAD (VPS11, Rottweiler Variant)	Clear
 Neuroaxonal Dystrophy, NAD (TECPR2, Spanish Water Dog Variant)	Clear
 Neuronal Ceroid Lipofuscinosis 1, NCL 1 (PPT1 Exon 8, Dachshund Variant 1)	Clear
 Neuronal Ceroid Lipofuscinosis 10, NCL 10 (CTSD Exon 5, American Bulldog Variant)	Clear



















OTHER RESULTS

 Neuronal Ceroid Lipofuscinosis 2, NCL 2 (TPP1 Exon 4, Dachshund Variant 2)	Clear
 Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 SNP, Border Collie Variant)	Clear
 Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 Deletion, Golden Retriever Variant)	Clear
 Neuronal Ceroid Lipofuscinosis 6, NCL 6 (CLN6 Exon 7, Australian Shepherd Variant)	Clear
 Neuronal Ceroid Lipofuscinosis 7, NCL 7 (MFSD8, Chihuahua and Chinese Crested Variant)	Clear
 Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8, Australian Shepherd Variant)	Clear
 Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Exon 2, English Setter Variant)	Clear
 Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Insertion, Saluki Variant)	Clear
 Neuronal Ceroid Lipofuscinosis, Cerebellar Ataxia, NCL4A (ARSG Exon 2, American Staffordshire Terrier Variant)	Clear
 Oculocutaneous Albinism, OCA (SLC45A2 Exon 6, Bullmastiff Variant)	Clear
 Oculocutaneous Albinism, OCA (SLC45A2, Small Breed Variant)	Clear
 Oculoskeletal Dysplasia 2 (COL9A2, Samoyed Variant)	Clear
 Osteogenesis Imperfecta (COL1A2, Beagle Variant)	Clear
 Osteogenesis Imperfecta (SERPINH1, Dachshund Variant)	Clear
 Osteogenesis Imperfecta (COL1A1, Golden Retriever Variant)	Clear
 P2Y12 Receptor Platelet Disorder (P2Y12)	Clear
 Pachyonychia Congenita (KRT16, Dogue de Bordeaux Variant)	Clear
 Paroxysmal Dyskinesia, PxD (PIGN)	Clear



















OTHER RESULTS

 Persistent Mullerian Duct Syndrome, PMDS (AMHR2)	Clear
 Pituitary Dwarfism (POU1F1 Intron 4, Karelian Bear Dog Variant)	Clear
 Platelet Factor X Receptor Deficiency, Scott Syndrome (TMEM16F)	Clear
 Polycystic Kidney Disease, PKD (PKD1)	Clear
 Pompe's Disease (GAA, Finnish and Swedish Lapphund, Lapponian Herder Variant)	Clear
 Prekallikrein Deficiency (KLKB1 Exon 8)	Clear
 Primary Ciliary Dyskinesia, PCD (NME5, Alaskan Malamute Variant)	Clear
 Primary Ciliary Dyskinesia, PCD (CCDC39 Exon 3, Old English Sheepdog Variant)	Clear
 Primary Hyperoxaluria (AGXT)	Clear
 Primary Lens Luxation (ADAMTS17)	Clear
 Primary Open Angle Glaucoma (ADAMTS17 Exon 11, Basset Fauve de Bretagne Variant)	Clear
 Primary Open Angle Glaucoma (ADAMTS10 Exon 17, Beagle Variant)	Clear
 Primary Open Angle Glaucoma (ADAMTS10 Exon 9, Norwegian Elkhound Variant)	Clear
 Primary Open Angle Glaucoma and Primary Lens Luxation (ADAMTS17 Exon 2, Chinese Shar-Pei Variant)	Clear
 Progressive Retinal Atrophy (SAG)	Clear
 Progressive Retinal Atrophy (IFT122 Exon 26, Lapponian Herder Variant)	Clear
 Progressive Retinal Atrophy, Bardet-Biedl Syndrome (BBS2 Exon 11, Shetland Sheepdog Variant)	Clear
 Progressive Retinal Atrophy, CNGA (CNGA1 Exon 9)	Clear















OTHER RESULTS

 Progressive Retinal Atrophy, crd1 (PDE6B, American Staffordshire Terrier Variant)	Clear
 Progressive Retinal Atrophy, crd4/cord1 (RPGRIP1)	Clear
 Progressive Retinal Atrophy, PRA1 (CNGB1)	Clear
 Progressive Retinal Atrophy, PRA3 (FAM161A)	Clear
 Progressive Retinal Atrophy, rcd1 (PDE6B Exon 21, Irish Setter Variant)	Clear
 Progressive Retinal Atrophy, rcd3 (PDE6A)	Clear
 Proportionate Dwarfism (GH1 Exon 5, Chihuahua Variant)	Clear
 Protein Losing Nephropathy, PLN (NPHS1)	Clear
 Pyruvate Dehydrogenase Deficiency (PDP1, Spaniel Variant)	Clear
 Pyruvate Kinase Deficiency (PKLR Exon 5, Basenji Variant)	Clear
 Pyruvate Kinase Deficiency (PKLR Exon 7, Beagle Variant)	Clear
 Pyruvate Kinase Deficiency (PKLR Exon 10, Terrier Variant)	Clear
 Pyruvate Kinase Deficiency (PKLR Exon 7, Labrador Retriever Variant)	Clear
 Pyruvate Kinase Deficiency (PKLR Exon 7, Pug Variant)	Clear
 Raine Syndrome (FAM20C)	Clear
 Recurrent Inflammatory Pulmonary Disease, RIPD (AKNA, Rough Collie Variant)	Clear
 Renal Cystadenocarcinoma and Nodular Dermatofibrosis (FLCN Exon 7)	Clear
 Retina Dysplasia and/or Optic Nerve Hypoplasia (SIX6 Exon 1, Golden Retriever Variant)	Clear

OTHER RESULTS

 Sensory Neuropathy (FAM134B, Border Collie Variant)	Clear
 Severe Combined Immunodeficiency, SCID (PRKDC, Terrier Variant)	Clear
 Severe Combined Immunodeficiency, SCID (RAG1, Wetterhoun Variant)	Clear
 Shaking Puppy Syndrome (PLP1, English Springer Spaniel Variant)	Clear
 Shar-Pei Autoinflammatory Disease, SPAID, Shar-Pei Fever (MTBP)	Clear
 Skeletal Dysplasia 2, SD2 (COL11A2, Labrador Retriever Variant)	Clear
 Skin Fragility Syndrome (PKP1, Chesapeake Bay Retriever Variant)	Clear
 Spinocerebellar Ataxia (SCN8A, Alpine Dachsbracke Variant)	Clear
 Spinocerebellar Ataxia with Myokymia and/or Seizures (KCNJ10)	Clear
 Spongy Degeneration with Cerebellar Ataxia 1 (KCNJ10)	Clear
 Spongy Degeneration with Cerebellar Ataxia 2 (ATP1B2)	Clear
 Stargardt Disease (ABCA4 Exon 28, Labrador Retriever Variant)	Clear
 Succinic Semialdehyde Dehydrogenase Deficiency (ALDH5A1 Exon 7, Saluki Variant)	Clear
 Thrombopathia (RASGRP1 Exon 5, American Eskimo Dog Variant)	Clear
 Thrombopathia (RASGRP1 Exon 5, Basset Hound Variant)	Clear
 Thrombopathia (RASGRP1 Exon 8, Landseer Variant)	Clear
 Trapped Neutrophil Syndrome, TNS (VPS13B)	Clear
 Ullrich-like Congenital Muscular Dystrophy (COL6A3 Exon 10, Labrador Retriever Variant)	Clear

OTHER RESULTS

 Ullrich-like Congenital Muscular Dystrophy (COL6A1 Exon 3, Landseer Variant)	Clear
 Unilateral Deafness and Vestibular Syndrome (PTPRQ Exon 39, Doberman Pinscher)	Clear
 Urate Kidney & Bladder Stones (SLC2A9)	Clear
 Von Willebrand Disease Type II, Type II vWD (VWF, Pointer Variant)	Clear
 Von Willebrand Disease Type III, Type III vWD (VWF Exon 4, Terrier Variant)	Clear
 Von Willebrand Disease Type III, Type III vWD (VWF Intron 16, Nederlandse Kooikerhondje Variant)	Clear
 Von Willebrand Disease Type III, Type III vWD (VWF Exon 7, Shetland Sheepdog Variant)	Clear
 X-Linked Hereditary Nephropathy, XLHN (COL4A5 Exon 35, Samoyed Variant 2)	Clear
 X-Linked Myotubular Myopathy (MTM1, Labrador Retriever Variant)	Clear
 X-Linked Progressive Retinal Atrophy 1, XL-PRA1 (RPGR)	Clear
 X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG Exon 1, Basset Hound Variant)	Clear
 X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG, Corgi Variant)	Clear
 Xanthine Urolithiasis (XDH, Mixed Breed Variant)	Clear
 β -Mannosidosis (MANBA Exon 16, Mixed-Breed Variant)	Clear
Mast Cell Tumor	No result

HEALTH REPORT

Increased risk result

Intervertebral Disc Disease (Type I)

Ringo inherited both copies of the variant we tested for Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type I IVDD. Ringo is at increased risk for Type I IVDD.

How to interpret this result

Ringo has two copies of an FGF4 retrogene on chromosome 12. In some breeds such as Beagles, Cocker Spaniels, and Dachshunds (among others) this variant is found in nearly all dogs. While those breeds are known to have an elevated risk of IVDD, many dogs in those breeds never develop IVDD. For mixed breed dogs and purebreds of other breeds where this variant is not as common, risk for Type I IVDD is greater for individuals with this variant than for similar dogs.

What is Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type I IVDD?

Type I Intervertebral Disc Disease (IVDD) is a back/spine issue that refers to a health condition affecting the discs that act as cushions between vertebrae. With Type I IVDD, affected dogs can have a disc event where it ruptures or herniates towards the spinal cord. This pressure on the spinal cord causes neurologic signs which can range from a wobbly gait to impairment of movement. Chondrodystrophy (CDDY) refers to the relative proportion between a dog's legs and body, wherein the legs are shorter and the body longer. There are multiple different variants that can cause a markedly chondrodystrophic appearance as observed in Dachshunds and Corgis. However, this particular variant is the only one known to also increase the risk for IVDD.

When signs & symptoms develop in affected dogs

Signs of CDDY are recognized in puppies as it affects body shape. IVDD is usually first recognized in adult dogs, with breed specific differences in age of onset.

Signs & symptoms

Research indicates that dogs with one or two copies of this variant have a similar risk of developing IVDD. However, there are some breeds (e.g. Beagles and Cocker Spaniels, among others) where this variant has been passed down to nearly all dogs of the breed and most do not show overt clinical signs of the disorder. This suggests that there are other genetic and environmental factors (such as weight, mobility, and family history) that contribute to an individual dog's risk of developing clinical IVDD. Signs of IVDD include neck or back pain, a change in your dog's walking pattern (including dragging of the hind limbs), and paralysis. These signs can be mild to severe, and if your dog starts exhibiting these signs, you should schedule an appointment with your veterinarian for a diagnosis.

How vets diagnose this condition

For CDDY, dogs with one copy of this variant may have mild proportional differences in their leg length. Dogs with two copies of this variant will often have visually longer bodies and shorter legs. For IVDD, a neurological exam will be performed on any dog showing suspicious signs. Based on the result of this exam, radiographs to detect the presence of calcified discs or advanced imaging (MRI/CT) to detect a disc rupture may be recommended.

How this condition is treated

IVDD is treated differently based on the severity of the disease. Mild cases often respond to medical management which includes

HEALTH REPORT

Notable result

ALT Activity

Ringo inherited both copies of the variant we tested for Alanine Aminotransferase Activity

Why is this important to your vet?

Ringo has two copies of a variant in the GPT gene and is likely to have a lower than average baseline ALT activity. ALT is a commonly used measure of liver health on routine veterinary blood chemistry panels. As such, your veterinarian may want to watch for changes in Ringo's ALT activity above their current, healthy, ALT activity. As an increase above Ringo's baseline 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

Notable result

Dilated Cardiomyopathy, DCM1

Ringo inherited one copy of the variant we tested for Dilated Cardiomyopathy, DCM1

Ringo is not likely to be at increased risk for DCM1

What does this result mean?

Research indicates that this genetic variant is not likely to increase the risk that Ringo will develop this condition.

Scientific Basis

Dogs with Ringo's breed have been included in research studies or have had follow-up by our experts that indicate that this genetic variant is not likely to increase the risk of Ringo developing clinical disease.

Impact on Breeding

This genetic result should not be the primary factor in your breeding decisions.

What is Dilated Cardiomyopathy, DCM1?

DCM is the most common acquired heart disease of adult dogs. The heart has two heavily muscled ventricles that pump blood away from the heart. This disease causes progressive weakening of the ventricles by reducing the muscle mass, which causes the ventricles to dilate. Dilated ventricles do not contract and circulate oxygenated blood well, which eventually leads to heart failure.

When signs & symptoms develop in affected dogs

This disease can rarely be seen in puppies and young adults. It is typically seen in middle aged to older dogs.

How vets diagnose this condition

The earlier a diagnosis can be reached, the better the outcome. If you are concerned about your dog's heart, discuss it with your veterinarian who can run basic preliminary tests. They may recommend a visit to a veterinary cardiologist for a complete evaluation, including an ultrasound of the heart (echocardiogram).

How this condition is treated

Treatment is completely dependent on how advanced the disease is at the time of diagnosis. It can range from monitoring the patient periodically to intensive hospitalization at specialty veterinary practices.

Actions to take if your dog is affected

- The cause of this disease is multifactorial and not completely understood. Genetics, nutrition, infections and environmental exposures can all play a role in the development of DCM. In fact, DCM has recently been featured extensively in the news due to suspected nutritional deficiencies in some grain free diets.
- Annual echocardiograms by a board certified cardiologist and annual Holter monitoring are the best ways to diagnose DCM early.

INBREEDING AND DIVERSITY

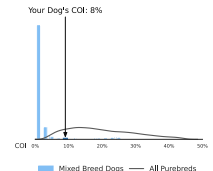
CATEGORY

RESULT

Coefficient Of Inbreeding

Our genetic COI measures the proportion of your dog's genome where the genes on the mother's side are identical by descent to those on the father's side.

8%

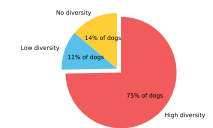


MHC Class II - DLA DRB1

A Dog Leukocyte Antigen (DLA) gene, DRB1 encodes a major histocompatibility complex (MHC) protein involved in the immune response. Some studies have shown associations between certain DRB1 haplotypes and autoimmune diseases such as Addison's disease (hypoadrenocorticism) in certain dog breeds, but these findings have yet to be scientifically validated.

High Diversity

How common is this amount of diversity in mixed breed dogs:



MHC Class II - DLA DQA1 and DQB1

DQA1 and DQB1 are two tightly linked DLA genes that code for MHC proteins involved in the immune response. A number of studies have shown correlations of DQA-DQB1 haplotypes and certain autoimmune diseases; however, these have not yet been scientifically validated.

High Diversity

How common is this amount of diversity in mixed breed dogs:

