



Preview



PREVIEW

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Wigglebutt Dio on Monument Mountain



DNA Test Report

Test Date: November 8th, 2021

embk.me/wigglebuttdioonmonumentmountain

Breed mix

Miniature/MAS-type Australian Shepherd : 54.2%
Australian Shepherd : 45.8%

Genetic Stats

Predicted adult weight: **47 lbs**

Genetic age: **35 human years**
Based on the date of birth you provided

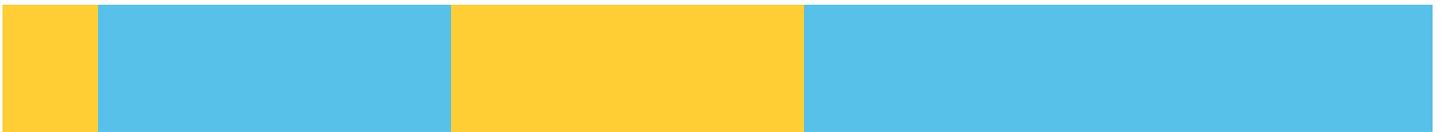
Test Details

Kit number: EM-26845058
Swab number: 31201153213861

Breed Mix by Chromosome

Our advanced test identifies from where Wigglebutt Dio on Monument Mountain inherited every part of the chromosome pairs in his genome.

Breed colors:
Miniature/MAS-type Australian Shepherd
Australian Shepherd
1

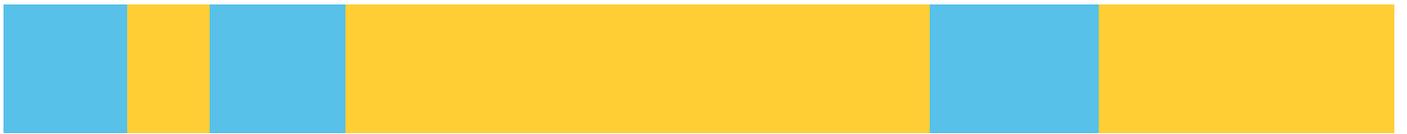




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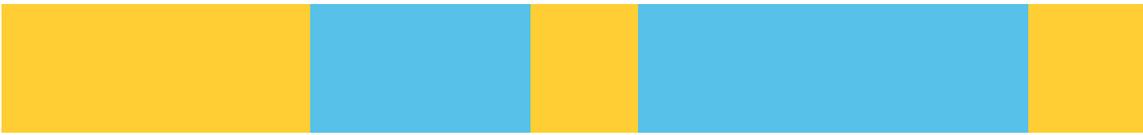
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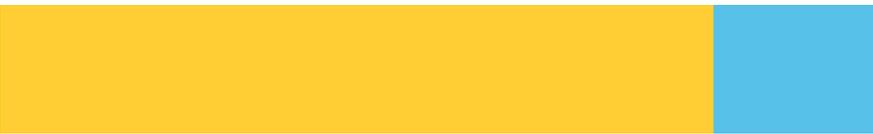
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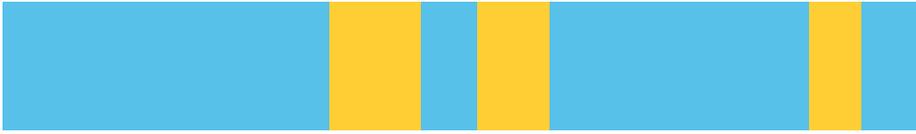
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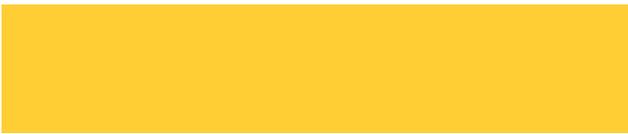
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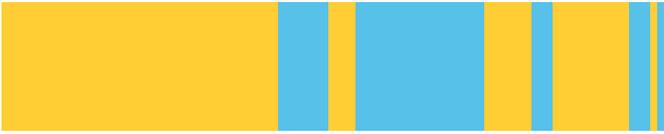
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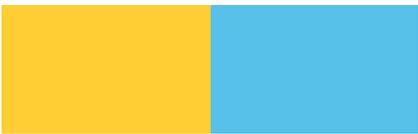
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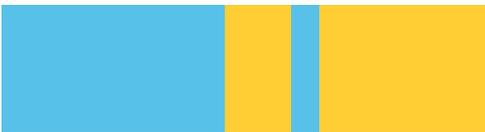
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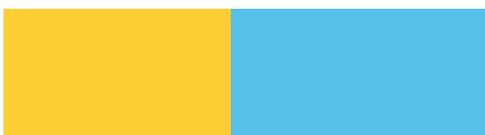
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Maternal Line



Through Wiggiebutt Dio on Monument Mountain'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: A1a

A1a is the most common maternal lineage among Western dogs. This lineage traveled from the site of dog domestication in Central Asia to Europe along with an early dog expansion perhaps 10,000 years ago. It hung around in European village dogs for many millennia. Then, about 300 years ago, some of the prized females in the line were chosen as the founding dogs for several dog breeds. That set in motion a huge expansion of this lineage. It's now the maternal lineage of the overwhelming majority of Mastiffs, Labrador Retrievers and Gordon Setters. About half of Boxers and less than half of Shar-Pei dogs descend from the A1a line. It is also common across the world among village dogs, a legacy of European colonialism.

HAPLOTYPE: A388

Part of the large A1a haplogroup, this haplotype occurs most frequently in Staffordshire Terriers, Labrador Retrievers, and English Bulldogs.

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Paternal Line



Through Wigglebutt Dio on Monument Mountain'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: A1a

Some of the wolves that became the original dogs in Central Asia around 15,000 years ago came from this long and distinguished line of male dogs. After domestication, they followed their humans from Asia to Europe and then didn't stop there. They took root in Europe, eventually becoming the dogs that founded the Vizsla breed 1,000 years ago. The Vizsla is a Central European hunting dog, and all male Vizslas descend from this line. During the Age of Exploration, like their owners, these pooches went by the philosophy, "Have sail, will travel!" From the windy plains of Patagonia to the snug and homey towns of the American Midwest, the beaches of a Pacific paradise, and the broad expanse of the Australian outback, these dogs followed their masters to the outposts of empires. Whether through good fortune or superior genetics, dogs from the A1a lineage traveled the globe and took root across the world. Now you find village dogs from this line frolicking on Polynesian beaches, hanging out in villages across the Americas, and scavenging throughout Old World settlements. You can also find this "prince of patrilineages" in breeds as different as German Shepherds, Golden Retrievers, Pugs, Border Collies, Scottish Terriers, and Irish Wolfhounds. No male wolf line has been as successful as the A1a line!

HAPLOTYPE: H1a.60

Part of the A1a haplogroup, this haplotype occurs most frequently in mixed-breed dogs.

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Traits: Coat Color

TRAIT

RESULT

TRAIT

RESULT

E Locus (MC1R)

<p>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”).</p>	<p>Can have a melanistic mask (E^mE)</p>
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K Locus (CBD103)

<p>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.</p>	<p>More likely to have a patterned haircoat (k^yk^y)</p>
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Traits: Coat Color (continued)

TRAIT

RESULT

Intensity Loci LINKAGE

<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 light hair likely yellow or tan (Intermediate Red Pigmentation)</p>
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A Locus (ASIP)

<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>Black/Brown and tan coat color pattern (a^a)</p>
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TRAIT

RESULT

D Locus (MLPH)

The D locus result that we report is determined by two different genetic variants that can work together to cause diluted pigmentation. These are the common **d** allele, also known as “**d1**”, and a less common allele known as “**d2**”. 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. To view your dog’s **d1** and **d2** test results, click the “SEE DETAILS” link in the upper right hand corner of the “Base Coat Color” section of the Traits page, and then click the “VIEW SUBLOCUS RESULTS” link at the bottom of the page.

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Traits: Coat Color (continued)

TRAIT

RESULT

Cocoa (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** 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 **Bbb** or **BB** genotypes at the B locus.

No co alleles, not expressed (NN)

B Locus (TYRP1)

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”.

Black or gray hair and skin (BB)

Saddle Tan (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.

Not saddle tan patterned (II)

S Locus (MITF)

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.

Likely to have little to no white in coat (SS)

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Traits: Coat Color (continued)

TRAIT	RESULT
<p>M Locus (PMEL)</p> <p>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 "phantom" merle, that is, they have a merle allele that does not affect coat color. 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.</p> <p>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.</p>	<p>Two merle alleles, likely to appear merle or double merle (M*M*)</p> <p>Note: This locus includes several alleles. At the time this dog was genotyped Embark we could not distinguish all of the possible alleles.</p>
<p>R Locus (USH2A) LINKAGE</p> <p>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.</p>	<p>Likely no impact on coat pattern (rr)</p>
<p>H Locus (Harlequin)</p> <p>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.</p>	<p>No harlequin alleles (hh)</p>

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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 unfurnished (no mustache, beard, and/or eyebrows) (II)
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 heavy/seasonal shedding (CC)
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 ND genotype are likely to be hairless while dogs with the NN genotype are likely to have a normal coat.	Very unlikely to be hairless (NN)

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Traits: Other Coat Traits (continued)

TRAIT	RESULT
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, (NN) 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)
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 straight coat (CC)

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Traits: Other Body Features

TRAIT

RESULT

TRAIT**RESULT****Muzzle Length (BMP3)**

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.

Likely medium or long muzzle (CC)

Tail Length (T)

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.

Likely normal-length tail (CC)

Hind Dewclaws (LMBR1)

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.

Unlikely to have hind dewclaws (CC)

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**Wigglebutt Dio on Monument Mountain****DNA Test Report**

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Traits: Other Body Features (continued)**TRAIT****RESULT****Blue Eye Color (ALX4) LINKAGE**

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.

Less likely to have blue eyes (NN)

Back Muscling & Bulk, Large Breed (ACSL4)

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.

Likely normal muscling (CC)



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Traits: Body Size

TRAIT	RESULT
Body Size (IGF1) The I allele is associated with smaller body size.	Intermediate (NI)
Body Size (IGFR1) The A allele is associated with smaller body size.	Larger (GG)
Body Size (STC2) The A allele is associated with smaller body size.	Larger (TT)
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)



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Traits: Performance

TRAIT	RESULT
<p>Altitude Adaptation (EPAS1)</p> <p>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.</p>	Normal altitude tolerance (GG)
<p>Appetite (POMC) LINKAGE</p> <p>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. We measure this result using a linkage test.</p>	Normal food motivation (NN)

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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)



Wigglebutt Dio on Monument Mountain's baseline ALT level may be Low Normal

Why is this important to your vet?

Wigglebutt Dio on Monument Mountain has one copy of a variant associated with reduced ALT activity as measured on veterinary blood chemistry panels. Please inform your veterinarian that Wigglebutt Dio on Monument Mountain has this genotype, as ALT is often used as an indicator of liver health and Wigglebutt Dio on Monument Mountain is likely to have a lower than average resting ALT activity. As such, an increase in Wigglebutt Dio on Monument Mountain'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.

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Health Report

How to interpret Wigglebutt Dio on Monument Mountain's genetic health results:

If Wigglebutt Dio on Monument Mountain 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 Wigglebutt Dio on Monument Mountain 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.



Wigglebutt Dio on Monument Mountain is at increased risk for one genetic health condition.

And inherited one variant that you should learn more about.

Hereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts



Hyperuricosuria and Hyperuricemia or Urolithiasis, HUU



Breed-Relevant Genetic Conditions 8 variants not detected



Additional Genetic Conditions 209 variants not detected



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Health Report

Hereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts (HSF4 Exon 9, Australian Shepherd Variant)



Wigglebutt Dio on Monument Mountain inherited one copy of the variant we tested

Wigglebutt Dio on Monument Mountain is at increased risk for Hereditary Cataracts

How to interpret this result

Wigglebutt Dio on Monument Mountain has one copy of a variant in the HSF4 gene that is thought to cause juvenile cataracts in the Australian Shepherd. variants in HSF4 have been reported in humans with juvenile cataracts; two variants have been identified in dogs. Please consult with your veterinarian regarding further diagnostics and a treatment plan for Wigglebutt Dio on Monument Mountain.

What is Hereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts?

Cataracts are the result of a progressive disease of the lens. The lens is normally a transparent structure of precisely organized fibers that lives in the pupil and focuses light. Cataracts cause the lens fibers to become disordered and turns the lens into a milky blue color. The lens is no longer transparent, light fails to reach the retina, and blindness is the end result. With this genetic mutation, dogs can develop cataracts at only a few weeks to months of age.

When signs & symptoms develop in affected dogs

While cataracts are typically a disease of the aged dog and can be associated with other eye diseases (these would be termed secondary cataracts), mutations in the HSF4 gene cause cataracts to form at an accelerated rate in comparatively young dogs (approximately 2-7 years of age).

Signs & symptoms

Affected dogs will first show a cloudy haze in their pupil that becomes progressively more milky blue to crystalline in appearance. Vision will become progressively worse, and dogs may start bumping into furniture, be more hesitant on steps, and run into walls or doorways.

How vets diagnose this condition

Veterinarians will examine your dog's eyes, and may use a light or lens to assist in the diagnosis. Please note that there are other ocular diseases that are commonly mistaken for cataracts so be sure to have your dog evaluated by a veterinarian.

How this condition is treated

Surgical correction by a veterinary ophthalmologist is currently the only treatment available to restore your dog's vision. The other alternative is careful monitoring and lifestyle changes to make your dog's blindness more manageable.

Actions to take if your dog is affected

- The best care you can provide your dog is seeking the expert opinion of your veterinarian for an accurate diagnosis and determining whether or not a specialty consult for surgery is required.

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Wigglebutt Dio on Monument Mountain



DNA Test Report

Test Date: November 8th, 2021

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Health Report

Hyperuricosuria and Hyperuricemia or Urolithiasis, HUU (SLC2A9)



Wigglebutt Dio on Monument Mountain inherited one copy of the variant we tested

What does this result mean?

This result should not impact Wigglebutt Dio on Monument Mountain's health but it could have consequences for siblings or other related dogs if they inherited two copies of the variant. We recommend discussing this result with their owners or breeders if you are in contact.

Impact on Breeding

Your dog carries this variant and will pass it on to ~50% of his offspring.

What is Hyperuricosuria and Hyperuricemia or Urolithiasis, HUU?

This condition causes kidney and bladder stones composed of urate. In most dogs, uric acid is converted to allantoin, an inert substance that is then excreted in the urine. Dogs with HUU have defects in the pathway that converts uric acid to allantoin. As such, uric acid builds up, crystallizes and forms urate stones in the kidney and bladder. Uric acid is an intermediate of purine metabolism. While hyperuricemia in other species (including humans) can lead to painful conditions such as gout, dogs do not develop systemic signs of hyperuricemia.

When signs & symptoms develop in affected dogs

Most dogs are adults before they show any signs of urinary issues (4-6 years on average).

How vets diagnose this condition

Urate stones are invisible on X-rays and must be diagnosed by a veterinarian via ultrasound or urine sediment analysis. If left undiagnosed, bladder stones can lead to urinary obstruction, which can be life-threatening.

How this condition is treated

If caught early, it is responsive to dietary management. Surgical intervention is often required when stones or an obstruction forms.

Actions to take if your dog is affected

- Closely monitoring your dog's urination habits, routine lab work, and following your veterinarian's nutritional advice are the best ways to keep your dog healthy.

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Breed-Relevant Conditions Tested



Wigglebutt Dio on Monument Mountain did not have the variants that we tested for, that are relevant to his breed:

- ✓ **MDR1 Drug Sensitivity (ABCB1)**
- ✓ **Progressive Retinal Atrophy, prcd (PRCD Exon 1)**
- ✓ **Collie Eye Anomaly, Choroidal Hypoplasia, CEA (NHEJ1)**
- ✓ **Day Blindness, Cone Degeneration, Achromatopsia (CNGB3 Deletion, Alaskan Malamute Variant)**
- ✓ **Canine Multifocal Retinopathy, cmr1 (BEST1 Exon 2)**
- ✓ **Neuronal Ceroid Lipofuscinosis 6, NCL 6 (CLN6 Exon 7, Australian Shepherd Variant)**
- ✓ **Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8, Australian Shepherd Variant)**
- ✓ **Craniomandibular Osteopathy, CMO (SLC37A2)**

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Additional Conditions Tested



Wigglebutt Dio on Monument Mountain did not have the variants that we tested for, in the following conditions that the potential effect on dogs with Wigglebutt Dio on Monument Mountain's breed may not yet be known.

- ✔ P2Y12 Receptor Platelet Disorder (P2Y12)
- ✔ Factor IX Deficiency, Hemophilia B (F9 Exon 7, Terrier Variant)
- ✔ Factor IX Deficiency, Hemophilia B (F9 Exon 7, Rhodesian Ridgeback Variant)
- ✔ Factor VII Deficiency (F7 Exon 5)
- ✔ Factor VIII Deficiency, Hemophilia A (F8 Exon 10, Boxer Variant)
- ✔ Factor VIII Deficiency, Hemophilia A (F8 Exon 11, German Shepherd Variant 1)
- ✔ Factor VIII Deficiency, Hemophilia A (F8 Exon 1, German Shepherd Variant 2)
- ✔ Thrombopathia (RASGRP1 Exon 5, Basset Hound Variant)
- ✔ Thrombopathia (RASGRP1 Exon 8, Landseer Variant)
- ✔ Thrombopathia (RASGRP1 Exon 5, American Eskimo Dog Variant)
- ✔ Von Willebrand Disease Type III, Type III vWD (VWF Exon 4, Terrier Variant)
- ✔ Von Willebrand Disease Type III, Type III vWD (VWF Exon 7, Shetland Sheepdog Variant)
- ✔ Von Willebrand Disease Type I, Type I vWD (VWF)
- ✔ Von Willebrand Disease Type II, Type II vWD (VWF, Pointer Variant)
- ✔ Canine Leukocyte Adhesion Deficiency Type I, CLAD I (ITGB2, Setter Variant)
- ✔ Canine Leukocyte Adhesion Deficiency Type III, CLAD III (FERMT3, German Shepherd Variant)
- ✔ Congenital Macrothrombocytopenia (TUBB1 Exon 1, Cairn and Norfolk Terrier Variant)
- ✔ Canine Elliptocytosis (SPTB Exon 30)
- ✔ Glanzmann's Thrombasthenia Type I (ITGA2B Exon 13, Great Pyrenees Variant)
- ✔ Glanzmann's Thrombasthenia Type I (ITGA2B Exon 12, Otterhound Variant)
- ✔ May-Hegglin Anomaly (MYH9)
- ✔ Prekallikrein Deficiency (KLKB1 Exon 8)
- ✔ Pyruvate Kinase Deficiency (PKLR Exon 5, Basenji Variant)
- ✔ Pyruvate Kinase Deficiency (PKLR Exon 7, Labrador Retriever Variant)
- ✔ Pyruvate Kinase Deficiency (PKLR Exon 7, Pug Variant)

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Additional Conditions Tested

- ✔ Pyruvate Kinase Deficiency (PKLR Exon 7, Beagle Variant)
- ✔ Pyruvate Kinase Deficiency (PKLR Exon 10, Terrier Variant)
- ✔ Trapped Neutrophil Syndrome, TNS (VPS13B)
- ✔ Ligneous Membranitis, LM (PLG)
- ✔ Platelet Factor X Receptor Deficiency, Scott Syndrome (TMEM16F)
- ✔ Methemoglobinemia (CYB5R3)
- ✔ Bernard-Soulier Syndrome, BSS (GP9, Cocker Spaniel Variant)
- ✔ Congenital Hypothyroidism (TPO, Tenterfield Terrier Variant)
- ✔ Congenital Hypothyroidism (TPO, Rat, Toy, Hairless Terrier Variant)
- ✔ Congenital Dysmorphogenic Hypothyroidism with Goiter (SLC5A5, Shih Tzu Variant)
- ✔ Complement 3 Deficiency, C3 Deficiency (C3)
- ✔ Severe Combined Immunodeficiency, SCID (PRKDC, Terrier Variant)
- ✔ Severe Combined Immunodeficiency, SCID (RAG1, Wetterhoun Variant)
- ✔ X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG Exon 1, Basset Hound Variant)
- ✔ X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG, Corgi Variant)
- ✔ Progressive Retinal Atrophy, rcd1 (PDE6B Exon 21, Irish Setter Variant)
- ✔ Progressive Retinal Atrophy, rcd3 (PDE6A)
- ✔ Progressive Retinal Atrophy, CNGA (CNGA1 Exon 9)
- ✔ Progressive Retinal Atrophy, PRA1 (CNGB1)
- ✔ Progressive Retinal Atrophy (SAG)
- ✔ Golden Retriever Progressive Retinal Atrophy 1, GR-PRA1 (SLC4A3)
- ✔ Golden Retriever Progressive Retinal Atrophy 2, GR-PRA2 (TTC8)
- ✔ Progressive Retinal Atrophy, crd1 (PDE6B, American Staffordshire Terrier Variant)

- ✔ Progressive Retinal Atrophy, crd4/cord1 (RPGRIP1)
- ✔ X-Linked Progressive Retinal Atrophy 1, XL-PRA1 (RPGR)

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Additional Conditions Tested

- ✔ Progressive Retinal Atrophy, PRA3 (FAM161A)
- ✔ Day Blindness, Cone Degeneration, Achromatopsia (CNGB3 Exon 6, German Shorthaired Pointer Variant)
- ✔ Achromatopsia (CNGA3 Exon 7, German Shepherd Variant)
- ✔ Achromatopsia (CNGA3 Exon 7, Labrador Retriever Variant)
- ✔ Autosomal Dominant Progressive Retinal Atrophy (RHO)
- ✔ Canine Multifocal Retinopathy, cmr2 (BEST1 Exon 5, Coton de Tulear Variant)
- ✔ Canine Multifocal Retinopathy, cmr3 (BEST1 Exon 10 Deletion, Finnish and Swedish Lapphund, Lapponian Herder Variant)
- ✔ Primary Open Angle Glaucoma (ADAMTS10 Exon 9, Norwegian Elkhound Variant)
- ✔ Primary Open Angle Glaucoma (ADAMTS10 Exon 17, Beagle Variant)
- ✔ Primary Open Angle Glaucoma (ADAMTS17 Exon 11, Basset Fauve de Bretagne Variant)
- ✔ Primary Open Angle Glaucoma and Primary Lens Luxation (ADAMTS17 Exon 2, Chinese Shar-Pei Variant)
- ✔ Goniodysgenesis and Glaucoma, Pectinate Ligament Dysplasia, PLD (OLFM3)
- ✔ Primary Lens Luxation (ADAMTS17)
- ✔ Congenital Stationary Night Blindness (RPE65, Briard Variant)
- ✔ Congenital Stationary Night Blindness (LRIT3, Beagle Variant)
- ✔ Macular Corneal Dystrophy, MCD (CHST6)
- ✔ 2,8-Dihydroxyadenine Urolithiasis, 2,8-DHA Urolithiasis (APRT)
- ✔ Cystinuria Type I-A (SLC3A1, Newfoundland Variant)
- ✔ Cystinuria Type II-A (SLC3A1, Australian Cattle Dog Variant)
- ✔ Cystinuria Type II-B (SLC7A9, Miniature Pinscher Variant)
- ✔ Polycystic Kidney Disease, PKD (PKD1)

- ✔ **Primary Hyperoxaluria (AGXT)**
- ✔ **Protein Losing Nephropathy, PLN (NPHS1)**
- ✔ **X-Linked Hereditary Nephropathy, XLHN (COL4A5 Exon 35, Samoyed Variant 2)**
- ✔ **Autosomal Recessive Hereditary Nephropathy, Familial Nephropathy, ARHN (COL4A4 Exon 30, English Springer Spaniel Variant)**

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Additional Conditions Tested

- ✔ **Autosomal Recessive Hereditary Nephropathy, Familial Nephropathy, ARHN (COL4A4 Exon 3, Cocker Spaniel Variant)**
- ✔ **Fanconi Syndrome (FAN1, Basenji Variant)**
- ✔ **Primary Ciliary Dyskinesia, PCD (CCDC39 Exon 3, Old English Sheepdog Variant)**
- ✔ **Primary Ciliary Dyskinesia, PCD (NME5, Alaskan Malamute Variant)**
- ✔ **Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatitis, Dry Eye Curly Coat Syndrome, CKCSID (FAM83H Exon 5)**
- ✔ **X-linked Ectodermal Dysplasia, Anhidrotic Ectodermal Dysplasia, XHED (EDA Intron 8)**
- ✔ **Renal Cystadenocarcinoma and Nodular Dermatofibrosis, RCND (FLCN Exon 7)**
- ✔ **Canine Fucosidosis (FUCA1)**
- ✔ **Glycogen Storage Disease Type II, Pompe's Disease, GSD II (GAA, Finnish and Swedish Lapphund, Lapponian Herder Variant)**
- ✔ **Glycogen Storage Disease Type IA, Von Gierke Disease, GSD IA (G6PC, Maltese Variant)**
- ✔ **Glycogen Storage Disease Type IIIA, GSD IIIA (AGL, Curly Coated Retriever Variant)**
- ✔ **Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, Dachshund Variant)**
- ✔ **Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, New Zealand Huntaway Variant)**
- ✔ **Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 5, Terrier Brasileiro Variant)**
- ✔ **Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 3, German Shepherd Variant)**
- ✔ **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)**
- ✔ **Lagotto Storage Disease (ATG4D)**
- ✔ **Neuronal Ceroid Lipofuscinosis 1, NCL 1 (PPT1 Exon 8, Dachshund Variant 1)**

- ✔ Neuronal Ceroid Lipofuscinosis 2, NCL 2 (TPP1 Exon 4, Dachshund Variant 2)
- ✔ Neuronal Ceroid Lipofuscinosis, Cerebellar Ataxia, NCL4A (ARSG Exon 2, American Staffordshire Terrier Variant)
- ✔ Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 SNP, Border Collie Variant)
- ✔ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Exon 2, English Setter Variant)
- ✔ Neuronal Ceroid Lipofuscinosis 7, NCL 7 (MFSD8, Chihuahua and Chinese Crested Variant)
- ✔ Neuronal Ceroid Lipofuscinosis 10, NCL 10 (CTSD Exon 5, American Bulldog Variant)

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Additional Conditions Tested

- ✔ Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 Deletion, Golden Retriever Variant)
- ✔ Adult-Onset Neuronal Ceroid Lipofuscinosis, NCL A, NCL 12 (ATP13A2, Tibetan Terrier Variant)
- ✔ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Insertion, Saluki Variant)
- ✔ Late-Onset Neuronal Ceroid Lipofuscinosis, NCL 12 (ATP13A2, Australian Cattle Dog Variant)
- ✔ GM1 Gangliosidosis (GLB1 Exon 15, Shiba Inu Variant)
- ✔ GM1 Gangliosidosis (GLB1 Exon 15, Alaskan Husky Variant)
- ✔ GM1 Gangliosidosis (GLB1 Exon 2, Portuguese Water Dog Variant)
- ✔ GM2 Gangliosidosis (HEXB, Poodle Variant)
- ✔ GM2 Gangliosidosis (HEXA, Japanese Chin Variant)
- ✔ Globoid Cell Leukodystrophy, Krabbe disease (GALC Exon 5, Terrier Variant)
- ✔ Autosomal Recessive Amelogenesis Imperfecta, Familial Enamel Hypoplasia (ENAM Deletion, Italian Greyhound Variant)
- ✔ Autosomal Recessive Amelogenesis Imperfecta, Familial Enamel Hypoplasia (ENAM SNP, Parson Russell Terrier Variant)
- ✔ Persistent Mullerian Duct Syndrome, PMDS (AMHR2)
- ✔ Deafness and Vestibular Syndrome of Dobermans, DVDob, DINGS (MYO7A)
- ✔ Shar-Pei Autoinflammatory Disease, SPAID, Shar-Pei Fever (MTBP)
- ✔ Neonatal Interstitial Lung Disease (LAMP3)
- ✔ Recurrent Inflammatory Pulmonary Disease, RIPD (AKNA, Rough Collie Variant)

- ✔ Alaskan Husky Encephalopathy, Subacute Necrotizing Encephalomyelopathy (SLC19A3)
- ✔ Alexander Disease (GFAP)
- ✔ Cerebellar Abiotrophy, Neonatal Cerebellar Cortical Degeneration, NCCD (SPTBN2, Beagle Variant)
- ✔ Cerebellar Ataxia, Progressive Early-Onset Cerebellar Ataxia (SEL1L, Finnish Hound Variant)
- ✔ Cerebellar Hypoplasia (VLDLR, Eurasier Variant)
- ✔ Spinocerebellar Ataxia, Late-Onset Ataxia, LoSCA (CAPN1)
- ✔ Spinocerebellar Ataxia with Myokymia and/or Seizures (KCNJ10)
- ✔ Hereditary Ataxia, Cerebellar Degeneration (RAB24, Old English Sheepdog and Gordon Setter Variant)

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Additional Conditions Tested

- ✔ Benign Familial Juvenile Epilepsy, Remitting Focal Epilepsy (LGI2)
- ✔ Degenerative Myelopathy, DM (SOD1A)
- ✔ Fetal-Onset Neonatal Neuroaxonal Dystrophy (MFN2, Giant Schnauzer Variant)
- ✔ Hypomyelination and Tremors (FNIP2, Weimaraner Variant)
- ✔ Shaking Puppy Syndrome, X-linked Generalized Tremor Syndrome (PLP1, English Springer Spaniel Variant)
- ✔ Neuroaxonal Dystrophy, NAD (TECPR2, Spanish Water Dog Variant)
- ✔ Neuroaxonal Dystrophy, NAD (VPS11, Rottweiler Variant)
- ✔ L-2-Hydroxyglutaricaciduria, L2HGA (L2HGDH, Staffordshire Bull Terrier Variant)
- ✔ Neonatal Encephalopathy with Seizures, NEWS (ATF2)
- ✔ Polyneuropathy, AMPN (NDRG1 SNP, Alaskan Malamute Variant)
- ✔ Narcolepsy (HCRTR2 Intron 4, Doberman Pinscher Variant)
- ✔ Narcolepsy (HCRTR2 Intron 6, Labrador Retriever Variant)
- ✔ Narcolepsy (HCRTR2 Exon 1, Dachshund Variant)
- ✔ Progressive Neuronal Abiotrophy, Canine Multiple System Degeneration, CMSD (SERAC1 Exon 15, Kerry Blue Terrier Variant)
- ✔ Progressive Neuronal Abiotrophy, Canine Multiple System Degeneration, CMSD (SERAC1 Exon 4, Chinese Crested Variant)

- ✔ Juvenile Laryngeal Paralysis and Polyneuropathy, Polyneuropathy with Ocular Abnormalities and Neuronal Vacuolation, POANV (RAB3GAP1, Rottweiler Variant)
- ✔ Hereditary Sensory Autonomic Neuropathy, Acral Mutilation Syndrome, AMS (GDNF-AS, Spaniel and Pointer Variant)
- ✔ Sensory Neuropathy (FAM134B, Border Collie Variant)
- ✔ Juvenile-Onset Polyneuropathy, Leonberger Polyneuropathy 1, LPN1 (LPN1, ARHGEF10)
- ✔ Juvenile Myoclonic Epilepsy (DIRAS1)
- ✔ Juvenile-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)
- ✔ Dilated Cardiomyopathy, DCM1 (PDK4, Doberman Pinscher Variant 1)
- ✔ Dilated Cardiomyopathy, DCM2 (TTN, Doberman Pinscher Variant 2)

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Additional Conditions Tested

- ✔ Long QT Syndrome (KCNQ1)
- ✔ Cardiomyopathy and Juvenile Mortality (YARS2)
- ✔ Muscular Dystrophy (DMD, Cavalier King Charles Spaniel Variant 1)
- ✔ Muscular Dystrophy (DMD, Golden Retriever Variant)
- ✔ Limb Girdle Muscular Dystrophy (SGCD, Boston Terrier Variant)
- ✔ Ulrich-like Congenital Muscular Dystrophy (COL6A3 Exon 10, Labrador Retriever Variant)
- ✔ Centronuclear Myopathy (PTPLA)
- ✔ Exercise-Induced Collapse (DNM1)
- ✔ Inherited Myopathy of Great Danes (BIN1)
- ✔ Myostatin Deficiency, Bully Whippet Syndrome (MSTN)
- ✔ Myotonia Congenita (CLCN1 Exon 7, Miniature Schnauzer Variant)
- ✔ Myotonia Congenita (CLCN1 Exon 23, Australian Cattle Dog Variant)
- ✔ Nemaline Myopathy (NEB, American Bulldog Variant)

- ✔ Myotubular Myopathy 1, X-linked Myotubular Myopathy, XL-MTM (MTM1, Labrador Retriever Variant)
- ✔ Inflammatory Myopathy (SLC25A12)
- ✔ Hypocatalasia, Acatalasemia (CAT)
- ✔ Pyruvate Dehydrogenase Deficiency (PDP1, Spaniel Variant)
- ✔ Malignant Hyperthermia (RYR1)
- ✔ Imerslund-Grasbeck Syndrome, Selective Cobalamin Malabsorption (CUBN Exon 53, Border Collie Variant)
- ✔ Imerslund-Grasbeck Syndrome, Selective Cobalamin Malabsorption (CUBN Exon 8, Beagle Variant)
- ✔ Inherited Selected Cobalamin Malabsorption with Proteinuria (CUBN, Komondor Variant)
- ✔ Lunde hund Syndrome (LEPREL1)
- ✔ Congenital Myasthenic Syndrome, CMS (CHAT, Old Danish Pointing Dog Variant)
- ✔ Congenital Myasthenic Syndrome, CMS (COLQ, Labrador Retriever Variant)
- ✔ Congenital Myasthenic Syndrome, CMS (CHRNE, Jack Russell Terrier Variant)

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Additional Conditions Tested

- ✔ Congenital Myasthenic Syndrome, CMS (COLQ, Golden Retriever Variant)
- ✔ Myasthenia Gravis Like Syndrome (CHRNE, Heideterrier Variant)
- ✔ Episodic Falling Syndrome (BCAN)
- ✔ Paroxysmal Dyskinesia, PxD (PGIN)
- ✔ Demyelinating Polyneuropathy (SBF2/MTRM13)
- ✔ Laryngeal Paralysis (RAPGEF6, Miniature Bull Terrier Variant)
- ✔ Dystrophic Epidermolysis Bullosa (COL7A1, Golden Retriever Variant)
- ✔ Dystrophic Epidermolysis Bullosa (COL7A1, Central Asian Shepherd Dog Variant)
- ✔ Ectodermal Dysplasia, Skin Fragility Syndrome (PKP1, Chesapeake Bay Retriever Variant)
- ✔ Ichthyosis, Epidermolytic Hyperkeratosis (KRT10, Terrier Variant)
- ✔ Ichthyosis, ICH1 (PNPLA1, Golden Retriever Variant)

- ✔ Ichthyosis (SLC27A4, Great Dane Variant)
- ✔ Ichthyosis (NIPAL4, American Bulldog Variant)
- ✔ Focal Non-Epidermolytic Palmoplantar Keratoderma, Pachyonychia Congenita (KRT16, Dogue de Bordeaux Variant)
- ✔ Hereditary Footpad Hyperkeratosis (FAM83G, Terrier and Kromfohrlander Variant)
- ✔ Hereditary Footpad Hyperkeratosis (DSG1, Rottweiler Variant)
- ✔ Hereditary Nasal Parakeratosis, HNPk (SUV39H2)
- ✔ Musladin-Lueke Syndrome, MLS (ADAMTSL2)
- ✔ Oculocutaneous Albinism, OCA (SLC45A2, Pekingese Variant)
- ✔ Bald Thigh Syndrome (IGFBP5)
- ✔ Lethal Acrodermatitis, LAD (MKLN1)
- ✔ Ehlers Danlos (ADAMTS2, Doberman Pinscher Variant)
- ✔ Cleft Lip and/or Cleft Palate (ADAMTS20, Nova Scotia Duck Tolling Retriever Variant)
- ✔ Hereditary Vitamin D-Resistant Rickets (VDR)
- ✔ Oculoskeletal Dysplasia 2, Dwarfism-Retinal Dysplasia 2, drd2, OSD2 (COL9A2, Samoyed Variant)

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Additional Conditions Tested

- ✔ Osteogenesis Imperfecta, Brittle Bone Disease (COL1A2, Beagle Variant)
- ✔ Osteogenesis Imperfecta, Brittle Bone Disease (SERPINH1, Dachshund Variant)
- ✔ Osteogenesis Imperfecta, Brittle Bone Disease (COL1A1, Golden Retriever Variant)
- ✔ Osteochondrodysplasia, Skeletal Dwarfism (SLC13A1, Poodle Variant)
- ✔ Skeletal Dysplasia 2, SD2 (COL11A2, Labrador Retriever Variant)
- ✔ Raine Syndrome, Canine Dental Hypomineralization Syndrome (FAM20C)
- ✔ Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type I IVDD (FGF4 retrogene - CFA12)
- ✔ Chondrodystrophy (ITGA10, Norwegian Elkhound and Karelian Bear Dog Variant)
- ✔ Mucopolysaccharidosis IIIB, Sanfilippo Syndrome Type B, MPS IIIB (NAGLU, Schipperke Variant)



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Inbreeding And Diversity

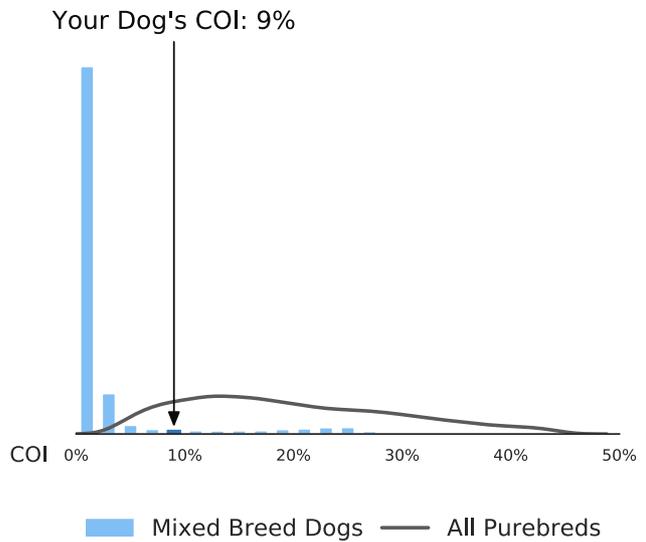
CATEGORY

RESULT

9%

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.



CATEGORY

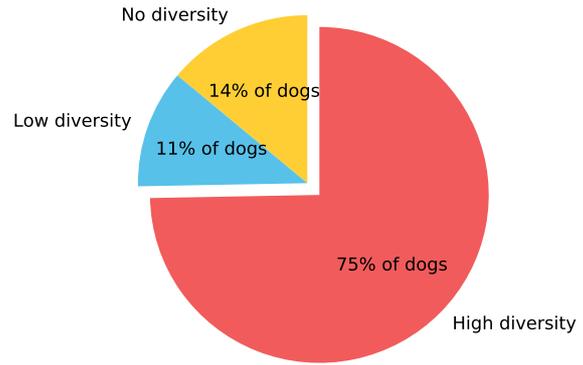
RESULT

High Diversity

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

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.

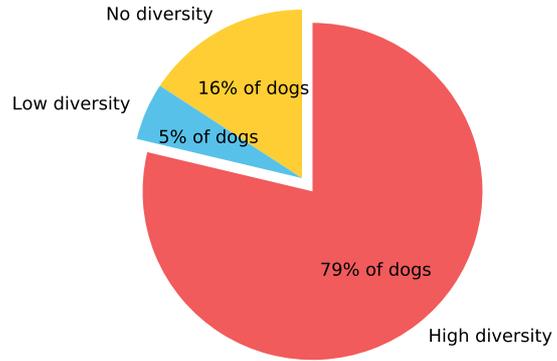


High Diversity

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.



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