



BREED MIX

Poodle (Small) : 100.0%

GENETIC STATS

Wolfiness: 0.9 % MEDIUM Predicted adult weight: 8 lbs Genetic age: 28 human years

Based on the date of birth you provided

TEST DETAILS

Kit number: EM-4182929

Swab number: 31019090907826





FAMILY TREE



Our algorithms predict this is the most likely family tree to explain Mister Murphy's breed mix, but this family tree may not be the only possible one.







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 NamesToy 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.

RELATED BREEDS



Poodle (Standard) Sibling breed



MalteseCousin breed



HavaneseCousin breed



Bichon Frise Cousin breed







MATERNAL LINE



Through Mister Murphy'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: A1d

This female lineage can be traced back about 15,000 years to some of the original Central Asian wolves that were domesticated into modern dogs. The early females that represent this lineage were likely taken into Eurasia, where they spread rapidly. As a result, many modern breed and village dogs from the Americas, Africa, through Asia and down into Oceania belong to this group! This widespread lineage is not limited to a select few breeds, but the majority of Rottweilers, Afghan Hounds and Wirehaired Pointing Griffons belong to it. It is also the most common female lineage among Papillons, Samoyeds and Jack Russell Terriers. Considering its occurrence in breeds as diverse as Afghan Hounds and Samoyeds, some of this is likely ancient variation. But because of its presence in many modern European breeds, much of its diversity likely can be attributed to much more recent breeding.

HAPLOTYPE: A247

Part of the large A1d haplogroup, this common haplotype occurs in village dogs all over the world. Among the 32 breeds we have sampled it in, the most common occurrences include Boxers, Labrador Retrievers, and Papillons.







PATERNAL LINE



Through Mister Murphy'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

HAPLOTYPE: H1a.59

Part of the A1a haplogroup, this haplotype occurs most frequently in European village dogs.







TRAITS: BASE COAT COLOR

TRAIT RESULT

Dark or Light Fur | E (Extension) Locus | Gene: Melanocortin Receptor 1 (MC1R) | Genetic Result: ee

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

Light colored fur (cream to red)

Did You Know? If a dog has a **ee** result then the fur's actual shade can range from a deep copper to yellow/gold to cream - the exact color cannot be predicted solely from this result, and will depend on other genetic factors.

Red Pigment Intensity LINKAGE | I (Intensity) Loci | Genetic Result: Intense Red Pigmentation

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

Any pigmented fur likely apricot or red

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

Brown or Black Pigment | B (Brown) Locus | Gene: Tyrosinase Related Protein 1 (TYRP1) | Genetic Result: BB

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

Likely black colored nose/feet

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







TRAITS: BASE COAT COLOR (CONTINUED)

TRAIT RESULT

Color Dilution | D (Dilute) Locus | Gene: Melanophilin (MLPH) | Genetic Result: DD

This gene helps determine whether a dog has lighter "diluted" pigment. A dog with a **Dd** or **DD** result will not be dilute. A dog with a **dd** result will have all their black or brown pigment lightened ("diluted") to gray or light brown, and may lighten red pigment to cream. This affects their fur, skin, and sometimes eye color. The D locus result that we report is determined by 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 one **d1** allele and one **d2** allele are typically dilute. 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.

Dark (non-dilute) skin

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







TRAITS: COAT COLOR MODIFIERS

TRAIT RESULT

Hidden Patterning | K (Dominant Black) Locus | Gene: Canine Beta-Defensin 103 (CBD103) | Genetic Result: K^Bk^y

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

No impact on coat color

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

Body Pattern | A (Agouti) Locus | Gene: Agouti Signalling Protein (ASIP) | Genetic Result: atat

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

No impact on coat pattern

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

Facial Fur Pattern | E (Extension) Locus | Gene: Melanocortin Receptor 1 (MC1R) | Genetic Result: ee

In addition to determining if a dog can develop dark fur at all, this gene 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 $\mathbf{E}^{\mathbf{m}}$ in their result will have a mask, which is dark facial fur as seen in the German Shepherd and Pug. Dogs with no $\mathbf{E}^{\mathbf{m}}$ in their result but one or two copies of $\mathbf{E}^{\mathbf{g}}$ will instead have a "widow's peak", which is dark forehead fur.

No dark fur anywhere

Did You Know? The widow's peak is seen in the Afghan Hound and Borzoi, where it is called either "grizzle" or "domino".







TRAITS: COAT COLOR MODIFIERS (CONTINUED)

TRAIT RESULT

Saddle Tan | Gene: RALY | Genetic Result: II

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 at allele, so dogs that do not express at are not influenced by this gene.

No impact on coat pattern

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

White Spotting | S (White Spotting) Locus | Gene: MITF | Genetic Result: SS

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

Likely to have little to no white in coat

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

Merle | M (Merle) Locus | Gene: PMEL | Genetic Result: mm

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

No impact on coat color

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







TRAITS: COAT COLOR MODIFIERS (CONTINUED)

TRAIT RESULT

Harlequin | Gene: PSMB | Genetic Result: hh

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

No impact on coat pattern

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







TRAITS: OTHER COAT TRAITS

TRAIT RESULT

Furnishings LINKAGE | Gene: RSP02 | Genetic Result: FF

This gene is responsible for "furnishings", which is another name for the mustache, beard, and eyebrows that are characteristic of breeds like the Schnauzer, Scottish Terrier, and Wire Haired Dachshund. A dog with an **FF** or **FI** result is likely to have furnishings. A dog with an **II** result will not have furnishings. We measure this result using a linkage test.

Likely furnished (mustache, beard, and/or eyebrows)

Did You Know? In breeds that are expected to have furnishings, dogs without furnishings are the exception - this is sometimes called an "improper coat".

Coat Length | Gene: FGF5 | Genetic Result: TT

This gene is known to affect hair/fur length in many different species, including cats, dogs, mice, and humans. In dogs, a **TT** result means the dog is likely to have a long, silky coat as seen in the Yorkshire Terrier and the Long Haired Whippet. A **GG** or **GT** result is likely to mean a shorter coat, like in the Boxer or the American Staffordshire Terrier.

Likely long coat

Did You Know? In certain breeds, such as Corgi, the long coat is described as "fluff."

Shedding | Gene: MC5R | Genetic Result: CT

This gene affects how much a dog sheds. Dogs with furnishings or wire-haired coats tend to be low shedders regardless of their result for this gene. In other dogs, a **CC** or **CT** result indicates heavy or seasonal shedding, like many Labradors and German Shepherd Dogs. Dogs with a **TT** result tend to be lighter shedders, like Boxers, Shih Tzus and Chihuahuas.

Likely light shedding

Coat Texture | Gene: KRT71 | Genetic Result: TT

For dogs with long fur, dogs with a **TT** or **CT** result will likely have a wavy or curly coat like the coat of Poodles and Bichon Frises. Dogs with a **CC** result will likely have a straight coat—unless the dog has a "Likely Furnished" result for the Furnishings trait, since this can also make the coat more curly.

Likely curly coat

Did You Know? Dogs with short coats may have straight coats, whatever result they have for this gene.







TRAITS: OTHER COAT TRAITS (CONTINUED)

TRAIT RESULT

Hairlessness (Terrier type) | Gene: SGK3 | Genetic Result: NN

This gene is responsible for Hairlessness in the American Hairless Terrier. 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

Oculocutaneous Albinism Type 2 LINKAGE | Gene: SLC45A2 | Genetic Result: NN

This gene causes oculocutaneous albinism (OCA), also known as Doberman Z Factor Albinism. Dogs with a **DD** result will have OCA. Effects include severely reduced or absent pigment in the eyes, skin, and hair, and sometimes vision problems due to lack of eye pigment (which helps direct and absorb ambient light) and are prone to sunburn. Dogs with a **ND** result will not be affected, but can pass the mutation on to their offspring. We measure this result using a linkage test.

Likely not albino

Did You Know? 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.







TRAITS: OTHER BODY FEATURES

TRAIT RESULT

Muzzle Length | Gene: BMP3 | Genetic Result: CC

This gene affects muzzle length. A dog with a **AC** or **CC** result is likely to have a medium-length muzzle like a Staffordshire Terrier or Labrador, or a long muzzle like a Whippet or Collie. A dog with a **AA** result is likely to have a short muzzle, like an English Bulldog, Pug, or Pekingese.

Did You Know? At least five different genes affect snout length in dogs, with BMP3 being the only one with a known causal mutation. For example, the muzzle length of some breeds, including the long-snouted Scottish Terrier or the short-snouted Japanese Chin, appear to be caused by other genes. This means your dog may have a long or short snout due to other genetic factors. Embark is working to figure out what these might be.

Likely medium or long muzzle

Tail Length | Gene: T | Genetic Result: CC

This is one of the genes that can cause a short bobtail. Most dogs have a **CC** result and a long tail. Dogs with a **CG** result are likely to have a bobtail, which is an unusually short or absent tail. This can be seen in many "natural bobtail" 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 such a result do not survive to birth.

Likely normal-length

Did You Know? 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, it is not always caused by this gene. This suggests that other unknown genetic effects can also lead to a natural bobtail.

Hind Dew Claws | Gene: LMBR1 | Genetic Result: CC

This is one of the genes that can cause hind dew claws, which are extra, nonfunctional digits located midway between a dog's paw and hock. Dogs with a **CT** or **TT** result have about a 50% chance of having hind dewclaws. Hind dew claws can also be caused by other, still unknown, genes. Embark is working to figure those out.

Unlikely to have hind dew claws

Did You Know? Hind dew claws are commonly found in certain breeds such as the Saint Bernard.







TRAITS: OTHER BODY FEATURES (CONTINUED)

TRAIT RESULT

Back Muscling & Bulk (Large Breed) | Gene: ACSL4 | Genetic Result: CC

This gene can cause 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. A dog with the **TT** result is likely to have heavy muscling. Leaner-shaped large breed dogs like the Great Dane, Irish Wolfhound, and Scottish Deerhound generally have a **CC** result. The **TC** result also indicates likely normal muscling.

Likely normal muscling

Did You Know? This gene does not seem to affect muscling in small or even mid-sized dog breeds with lots of back muscling, including the American Staffordshire Terrier, Boston Terrier, and the English Bulldog.

Eye Color LINKAGE | Gene: ALX4 | Genetic Result: NN

This gene is associated with blue eyes in Arctic breeds like Siberian Husky as well as tri-colored (non-merle) Australian Shepherds. Dogs with a **DupDup** or **NDup** result are more likely to have blue eyes, although some dogs may have only one blue eye or may not have blue eyes at all; nevertheless, they can still pass blue eyes to their offspring. Dogs with a **NN** result may have blue eyes due to other factors, such as merle or white spotting. We measure this result using a linkage test.

Less likely to have blue eyes

Did You Know? Embark researchers discovered this gene by studying data from dogs like yours. Who knows what we will be able to discover next? Answer the questions on our research surveys to contribute to future discoveries!







TRAITS: BODY SIZE

TRAIT	RESULT
Body Size 1 Gene: IGF1 Genetic Result: II This is one of several genes that influence the size of a dog. A result of II for this gene is associated with smaller body size. A result of NN is associated with larger body size.	
Body Size 2 Gene: IGFR1 Genetic Result: GA This is one of several genes that influence the size of a dog. A result of AA for this gene is associated with smaller body size. A result of GG is associated with larger body size.	e
Body Size 3 Gene: STC2 Genetic Result: TA This is one of several genes that influence the size of a dog. A result of AA for this gene is associated with smaller body size. A result of TT is associated with larger body size.	е
Body Size 4 Gene: GHR - E191K Genetic Result: AA This is one of several genes that influence the size of a dog. A result of AA for this gene is associated with smaller body size. A result of GG is associated with larger body size.	
Body Size 5 Gene: GHR - P177L Genetic Result: CT This is one of several genes that influence the size of a dog. A result of TT for this gene is associated with smaller body size. A result of CC is associated with larger body size.	e





TRAITS: PERFORMANCE

TRAIT RESULT

Altitude Adaptation | Gene: EPAS1 | Genetic Result: GG

This gene causes dogs to be especially tolerant of low oxygen environments, such as those found at high elevations. Dogs with a AA or GA result will be less susceptible to "altitude sickness."

Normal altitude tolerance

Did You Know? This gene was originally identified in breeds from high altitude areas such as the Tibetan Mastiff.

Appetite LINKAGE | Gene: POMC | Genetic Result: NN

This gene influences eating behavior. An **ND** or **DD** result would predict higher food motivation compared to **NN** result, increasing the likelihood 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

Did You Know? POMC is actually short for "proopiomelanocortin," and is a large protein that is broken up into several smaller proteins that have biological activity. The smaller proteins generated from POMC control, among other things, distribution of pigment to the hair and skin cells, appetite, and energy expenditure.





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)



Mister Murphy's baseline ALT level may be Low Normal

Why is this important to your vet?

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

What is Alanine Aminotransferase Activity?

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

How vets diagnose this condition

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

How this condition is treated

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





HEALTH REPORT

How to interpret Mister Murphy's genetic health results:

If Mister Murphy 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 Mister Murphy 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.



Mister Murphy is at increased risk for one genetic health condition.

And inherited two variants that you should learn more about.

Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type	e I IVDD	•
Progressive Retinal Atrophy, prcd		0
Dilated Cardiomyopathy, DCM1		0
Breed-Relevant Genetic Conditions	4 variants not detected	•
Additional Genetic Conditions	188 variants not detected	©







HEALTH REPORT

Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type I IVDD (FGF4 retrogene - CFA12)



Mister Murphy inherited one copy of the variant we tested

Mister Murphy is at increased risk for Type I IVDD

How to interpret this result

Mister Murphy has one copy 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 cage rest and pain management, while severe cases are often treated with surgical intervention. Both conservative and surgical treatment should be followed up with rehabilitation and physical therapy.







HEALTH REPORT

Progressive Retinal Atrophy, prcd (PRCD Exon 1)



Mister Murphy inherited one copy of the variant we tested

What does this result mean?

This result does not impact your dog's health. It could have consequences for siblings or other family members, and you should let them know if you are in contact with them. This result is also important if you decide to breed this dog - to produce the healthiest puppies we recommend genetic testing any potential mates for this condition.

What is Progressive Retinal Atrophy, prcd?

PRA-prod is a retinal disease that causes progressive, non-painful vision loss. The retina contains cells, called photoreceptors, that collect information about light and send signals to the brain. There are two types of photoreceptors: rods, for night vision and movement, and cones, for day vision and color. This type of PRA leads to early loss of rod cells, leading to night blindness before day blindness.

When signs & symptoms develop in affected dogs

The age affected dogs will first show signs of visual impairment varies by breed. However, most begin showing clinical signs in early adulthood.

How vets diagnose this condition

Veterinarians use a focused light to examine the pupils. In affected dogs, the pupils will appear more dilated and slower to contract. Your vet may also use a lens to visualize the retina at the back of the eye to look for changes in the optic nerve or blood vessels. You may be referred to a veterinary ophthalmologist for a definitive diagnosis.

How this condition is treated

Currently, there is no definitive treatment for PRA. Supplements, including antioxidants, have been proposed for management of the disease, but have not been scientifically proven effective.

Actions to take if your dog is affected

- Careful monitoring by your veterinarian will be required for the rest of your affected dog's life as secondary complications, including cataracts, can develop.
- With blind dogs, keeping furniture in the same location, making sure they are on a leash in unfamiliar territory, and training them to understand verbal commands are some of the ways to help them at home.





HEALTH REPORT

Dilated Cardiomyopathy, DCM1 (PDK4, Doberman Pinscher Variant 1)

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Mister Murphy inherited one copy of the variant we tested

Mister Murphy is not likely to be at increased risk for DCM1

What does this result mean?

Our research indicates that this genetic variant is not likely to increase the risk that Mister Murphy will develop this disease.

Scientific Basis

Dogs with similar breeds to Mister Murphy are not likely to have increased risk of developing the disease. Research has indicated increased risk in other breeds that are not found in Mister Murphy.

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.





BREED-RELEVANT CONDITIONS TESTED



Mister Murphy did not have the variants that we tested for, that are relevant to his breed:

- Von Willebrand Disease Type I, Type I vWD (VWF)
- GM2 Gangliosidosis (HEXB, Poodle Variant)
- Neonatal Encephalopathy with Seizures, NEWS (ATF2)
- Osteochondrodysplasia, Skeletal Dwarfism (SLC13A1, Poodle Variant)





ADDITIONAL CONDITIONS TESTED



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

- MDR1 Drug Sensitivity (ABCB1)
- 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 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, 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)
- Congenital Hypothyroidism (TPO, Tenterfield Terrier Variant)
- Congenital Hypothyroidism (TPO, Rat, Toy, Hairless Terrier 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)
- Progressive Retinal Atrophy, PRA3 (FAM161A)
- Collie Eye Anomaly, Choroidal Hypoplasia, CEA (NHEJ1)







- 🚺 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, cmr1 (BEST1 Exon 2)
- 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)
- Hereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts (HSF4 Exon 9, Australian Shepherd Variant)
- Primary Lens Luxation (ADAMTS17)
- Congenital Stationary Night Blindness (RPE65, Briard 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)
- Hyperuricosuria and Hyperuricemia or Urolithiasis, HUU (SLC2A9)
- 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 3, Cocker Spaniel Variant)
- Primary Ciliary Dyskinesia, PCD (CCDC39 Exon 3, Old English Sheepdog Variant)
- Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatosis, 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 I, MPS I (IDUA, Plott Hound 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 6, NCL 6 (CLN6 Exon 7, Australian Shepherd 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 8, NCL 8 (CLN8, Australian Shepherd Variant)





- Neuronal Ceroid Lipofuscinosis 10, NCL 10 (CTSD Exon 5, American Bulldog Variant)
- 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)
- 📞 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 (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)
- 🚺 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)
- 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)
- Polyneuropathy (NDRG1 Deletion, Greyhound Variant)
- Alaskan Malamute Polyneuropathy, AMPN (NDRG1 SNP)
- 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)
- 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, DCM2 (TTN, Doberman Pinscher Variant 2)
- Long QT Syndrome (KCNQ1)
- Muscular Dystrophy (DMD, Cavalier King Charles Spaniel Variant 1)
- Muscular Dystrophy (DMD, Golden Retriever Variant)
- Limb Girdle Muscular Dystrophy (SGCD, Boston Terrier Variant)
- Centronuclear Myopathy, CNM (PTPLA)
- Exercise-Induced Collapse, EIC (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)
- 🚫 Myotubular Myopathy 1, X-linked Myotubular Myopathy, XL-MTM (MTM1, Labrador Retriever Variant)
- Hypocatalasia, Acatalasemia (CAT)
- Pyruvate Dehydrogenase Deficiency (PDP1, Spaniel Variant)
- Malignant Hyperthermia (RYR1)
- Merslund-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)
- Lundehund 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)
- Episodic Falling Syndrome (BCAN)
- Paroxysmal Dyskinesia, PxD (PIGN)
- Dystrophic Epidermolysis Bullosa (COL7A1, Golden Retriever Variant)
- Dystrophic Epidermolysis Bullosa (COL7A1, Central Asian Shepherd Dog Variant)
- C 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 Nasal Parakeratosis, HNPK (SUV39H2)
- Musladin-Lueke Syndrome, MLS (ADAMTSL2)
- Oculocutaneous Albinism, OCA (SLC45A2, Small Breed Variant)
- Bald Thigh Syndrome (IGFBP5)
- Cleft Lip and/or Cleft Palate (ADAMTS20, Nova Scotia Duck Tolling Retriever Variant)
- Hereditary Vitamin D-Resistant Rickets (VDR)
- 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)
- Skeletal Dysplasia 2, SD2 (COL11A2, Labrador Retriever Variant)
- Craniomandibular Osteopathy, CMO (SLC37A2)
- Chondrodystrophy (ITGA10, Norwegian Elkhound and Karelian Bear Dog Variant)



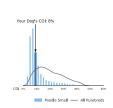


INBREEDING AND DIVERSITY

CATEGORY RESULT

Inbreeding | Gene: n/a | Genetic Result: 8%

Inbreeding is a measure of how closely related this dog's parents were. The higher the number, the more closely related the parents. In general, greater inbreeding is associated with increased incidence of genetically inherited conditions.



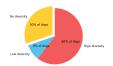
Immune Response 1 | Gene: DRB1 | Genetic Result: No Diversity

Diversity in the Major Histocompatibility Complex (MHC) region of the genome has been found in some studies to be associated with the incidence of certain autoimmune diseases. Dogs that have less diversity in the MHC region—i.e. the Dog Leukocyte Antigen (DLA) inherited from the mother is similar to the DLA inherited from the father—are considered less immunologically diverse. A High Diversity result means the dog has two highly dissimilar haplotypes. A Low Diversity result means the dog has two similar but not identical haplotypes. A No Diversity result means the dog has inherited identical haplotypes from both parents. Some studies have shown associations between certain DRB1 haplotypes and autoimmune diseases such as Cushing's disease, but these findings have yet to be scientifically validated.

No Diversity

8%

How common is this amount of diversity in purebreds:



Immune Response 2 | Gene: DQA1 and DQB1 | Genetic Result: No Diversity

Diversity in the Major Histocompatibility Complex (MHC) region of the genome has been found in some studies to be associated with the incidence of certain autoimmune diseases. Dogs that have less diversity in the MHC region—i.e. the Dog Leukocyte Antigen (DLA) inherited from the mother is similar to the DLA inherited from the father—are considered less immunologically diverse. A High Diversity result means the dog has two highly dissimilar haplotypes. A Low Diversity result means the dog has two similar but not identical haplotypes. A No Diversity result means the dog has inherited identical haplotypes from both parents. A number of studies have shown correlations of DQA-DQB1 haplotypes and certain autoimmune diseases; however, these have not yet been scientifically validated.

No Diversity

How common is this amount of diversity in purebreds:

