



"IDGIE"

"AMY'S CANDI DOTS"



DNA Test Report

Test Date: March 14th, 2025

embk.me/idgie81

BREED ANCESTRY

 **Dachshund : 100.0%**

GENETIC STATS

Predicted adult weight: **15 lbs**

TEST DETAILS

Kit number: EM-19094678

Swab number: 31240412700481

Registration: American Kennel Club

(AKC) HP70221401





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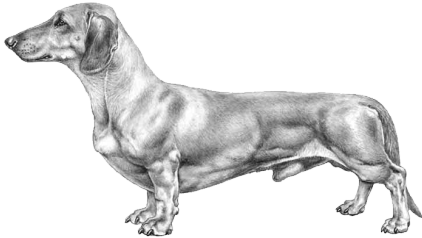


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DACHSHUND



The Dachshund was bred originally in Germany to flush out Badgers and other den animals in the 15th century. The breed, originally known as the Teckel, was refined by German Foresters to have the elongated shape that is advantageous for fitting into tight animal burrows. Dachshunds are often viewed as a symbol for Germany. For example, a Dachshund named Waldi was the first official mascot of the 1972 Summer Olympics held in Munich. Dachshunds are one of the most popular breeds in the United States, ranking 13th in AKC's most popular breeds. The Dachshund's personality is described as energetic, clever, and persistent to the point of stubbornness.

Alternative Names

Dachshund (Miniature), Dachshund (Standard)

Fun Fact

The name Dachshund is derived from "Dachs Krieger" meaning "Badger Warrior", who knew your Dachshund has such a fearsome name!

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MATERNAL LINE



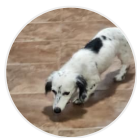
Through Idgie's mitochondrial DNA we can trace her mother's ancestry back to where dogs and people first became friends. This map helps you visualize the routes that her ancestors took to your home. Their story is described below the map.

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/A522

Part of the A1d haplogroup, the A247/A522 haplotype occurs most frequently in Pomeranians, Dachshunds, and Australian Shepherds.



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TRAITS: COAT COLOR

TRAIT

RESULT

E Locus (MC1R)

The E Locus determines if and where a dog can produce dark (black or brown) hair. Dogs with two copies of the recessive **e** variant do not produce dark hairs and will express a red pigment called pheomelanin over their entire body. The shade of red, which can range from a deep copper to white, depends on other genetic factors, including the Intensity loci. In addition to determining if a dog can develop dark hairs, 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 **E^m** variant may have a melanistic mask (dark facial hair as commonly seen in the German Shepherd Dog and Pug). In the absence of **E^m**, dogs with the **E^g** variant can have a "grizzle" phenotype (darker color on the head and top with a melanistic "widow's peak" and a lighter underside, commonly seen in the Afghan Hound and Borzoi and also referred to as "domino"). In the absence of both **E^m** and **E** variants, dogs with the **E^a** or **E^h** variants can express the grizzle phenotype. Additionally, a dog with any combination of two of the **E^g**, **E^a**, or **E^h** variants (example: **E^gE^a**) is also expected to express the grizzle phenotype.

No dark mask or
grizzle (**EE**)

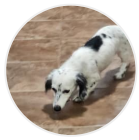
K Locus (CBD103)

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

More likely to have a
patterned haircoat
(**k^yk^y**)

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TRAITS: COAT COLOR (CONTINUED)

TRAIT

RESULT

Intensity Loci

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.

Any light hair likely yellow or tan (Intermediate Red Pigmentation)

A Locus (ASIP)

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.

Black/Brown and tan coat color pattern (a⁺a⁺)

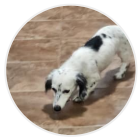
D Locus (MLPH)

The D locus result that we report is determined by three different genetic variants that can work together to cause diluted pigmentation. These are the common **d** allele, also known as "**d1**", and the less common alleles known as "**d2**" and "**d3**". Dogs with two **d** alleles, regardless of which variant, will have all black pigment lightened ("diluted") to gray, or brown pigment lightened to lighter brown in their hair, skin, and sometimes eyes. There are many breed-specific names for these dilute colors, such as "blue", "charcoal", "fawn", "silver", and "Isabella". Note that in certain breeds, dilute dogs have a higher incidence of Color Dilution Alopecia. Dogs with one **d** allele will not be dilute, but can pass the **d** allele on to their puppies.

Dark areas of hair and skin are not lightened (DD)

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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 **Bb** 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)

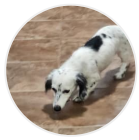
S Locus (MITF)

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 flash, parti, piebald, or extreme white (spsp)

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TRAITS: COAT COLOR (CONTINUED)

TRAIT

RESULT

M Locus (PMEL)

Merle coat patterning is common to several dog breeds including the Australian Shepherd, Catahoula Leopard Dog, and Shetland Sheepdog, among many others. Merle arises from an unstable SINE insertion (which we term the "M*" allele) that disrupts activity of the pigmentary gene PMEL, leading to mottled or patchy coat color. Dogs with an **M*m** result are likely to be phenotypically merle or could be "non-expressing" merle, meaning that the merle pattern is very subtle or not at all evident in their coat. Dogs with an **M*M*** result are likely to be phenotypically merle or double merle. Dogs with an **mm** result have no merle alleles and are unlikely to have a merle coat pattern.

No merle alleles (mm)

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

R Locus (USH2A)

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

Likely no impact on coat pattern (rr)

H Locus (Harlequin)

This pattern is recognized in Great Danes and causes dogs to have a white coat with patches of darker pigment. A dog with an **Hh** result will be harlequin if they are also **M*m** or **M*M*** at the M Locus and are not **ee** at the E locus. Dogs with a result of **hh** will not be harlequin. This trait is thought to be homozygous lethal; a living dog with an **HH** genotype has never been found.

No harlequin alleles (hh)

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TRAITS: COAT COLOR (CONTINUED)

TRAIT

RESULT

Panda White Spotting

Panda White Spotting originated in a line of German Shepherd Dogs and causes a mostly symmetrical white spotting of the head and/or body. This is a dominant variant of the KIT gene, which has a role in pigmentation.

Dogs with one copy of the **I** allele will exhibit this white spotting. Dogs with two copies of the **I** allele have never been observed, as two copies of the variant is suspected to be lethal to the developing embryo. Dogs with the **NN** result will not exhibit white spotting due to this variant.

Not expected to display Panda pattern (NN)

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TRAITS: OTHER COAT TRAITS

TRAIT

RESULT

Furnishings (RSP02)

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)

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TRAITS: OTHER COAT TRAITS (CONTINUED)

TRAIT

RESULT

Coat Length (FGF5)

The FGF5 gene affects hair length in many species, including cats, dogs, mice, and humans. In dogs, an **Lh** allele confers a long, silky hair coat across many breeds, including Yorkshire Terriers, Cocker Spaniels, and Golden Retrievers, while the **Sh** allele causes a shorter coat, as seen in the Boxer or the American Staffordshire Terrier. In certain breeds, such as the Pembroke Welsh Corgi and French Bulldog, the long haircoat is described as "fluffy". The coat length determined by FGF5, as reported by us, is influenced by four genetic variants that work together to promote long hair.

The most common of these is the **Lh1** variant (G/T, CanFam3.1, chr32, g.4509367) and the less common ones are **Lh2** (C/T, CanFam3.1, chr32, g.4528639), **Lh3** (16bp deletion, CanFam3.1, chr32, g.4528616), and **Lh4** (GG insertion, CanFam3.1, chr32, g.4528621). The FGF5_Lh1 variant is found across many dog breeds. The less common alleles, FGF5_Lh2, have been found in the Akita, Samoyed, and Siberian Husky, FGF5_Lh3 have been found in the Eurasier, and FGF5_Lh4 have been found in the Afghan Hound, Eurasier, and French Bulldog.

Likely long coat (LhLh)

The **Lh** alleles have a recessive mode of inheritance, meaning that two copies of the **Lh** alleles are required to have long hair. The presence of two Lh alleles at any of these FGF5 loci is expected to result in long hair. One copy each of **Lh1** and **Lh2** have been found in Samoyeds, one copy each of **Lh1** and **Lh3** have been found in Eurasiers, and one copy each of **Lh1** and **Lh4** have been found in the Afghan Hounds and Eurasiers.

Interestingly, the Lh3 variant, a 16 base pair deletion, encompasses the Lh4 variant (GG insertion). The presence of one or two copies of Lh3 influences the outcome at the Lh4 locus. When two copies of Lh3 are present, there will be no reportable result for the FGF5_Lh4 locus. With one copy of Lh3, Lh4 can have either one copy of the variant allele or the normal allele. The overall FGF5 result remains unaffected by this.

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TRAITS: OTHER COAT TRAITS (CONTINUED)

TRAIT

RESULT

Shedding (MC5R)

Dogs with at least one copy of the ancestral **C** allele, like many Labradors and German Shepherd Dogs, are heavy or seasonal shedders, while those with two copies of the **T** allele, including many Boxers, Shih Tzus and Chihuahuas, tend to be lighter shedders. Dogs with furnished/wire-haired coats caused by RSPO2 (the furnishings gene) tend to be low shedders regardless of their genotype at this gene.

Likely light shedding (TT)

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)

Hairlessness (FOXI3)

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

Very unlikely to be hairless (NN)

Hairlessness (SGK3)

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

Very unlikely to be hairless (NN)

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TRAIT

RESULT

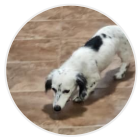
Oculocutaneous Albinism Type 2 (SLC45A2)

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

Likely not albino (NN)

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TRAITS: OTHER BODY FEATURES

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)

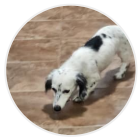
Hind Dewclaws (LMBR1)

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.

Likely to have hind dew claws (CT)

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TRAITS: OTHER BODY FEATURES (CONTINUED)

TRAIT

RESULT

Chondrodysplasia (Chr. 18 FGF4 Retrogene)

Dogs with one or two copies of the **I** allele will exhibit a short-legged trait known as chondrodysplasia (CDPA). CDPA is a breed-defining characteristic of many breeds exhibiting the "short-legged, long-bodied" appearance known as disproportionate dwarfism, including the corgi, dachshund and basset hound. The impact of the **I** allele on leg length is additive. Therefore, dogs with the **II** result display the largest reduction in leg length. Dogs with the **NI** genotype will have an intermediate leg length, while dogs with the **NN** result will not exhibit leg shortening due to this variant. Breeds that display disproportionate dwarfism also frequently inherit a genetic variant known as the chondrodystrophy (CDDY) variant. The CDDY variant also shortens legs (in a less significant amount than CDPA) but, secondarily, increases the risk of Type I Intervertebral Disc Disease (IVDD). Test results for CDDY are listed in this dog's health testing results under "Intervertebral Disc Disease (Type I)". In contrast, the CDPA variant has NOT been shown to increase the risk of IVDD.

Likely to have chondrodysplasia (short legs) (II)

Blue Eye Color (ALX4)

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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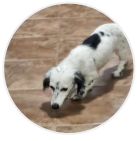
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TRAITS: BODY SIZE

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

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TRAITS: PERFORMANCE

TRAIT

RESULT

Altitude Adaptation (EPAS1)

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

**Normal altitude
tolerance (GG)**

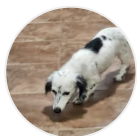
Appetite (POMC)

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

**Normal food
motivation (NN)**

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HEALTH REPORT

How to interpret Idgie's genetic health results:

If Idgie 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 Idgie for that we did not detect the risk variant for.

A genetic test is not a diagnosis

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

Summary

Of the 274 genetic health risks we analyzed, we found 5 results that you should learn about.

⚠ Increased risk results (1)

Intervertebral Disc Disease (Type I)

ⓘ Notable results (4)

ALT Activity

Copper Toxicosis (Accumulating)

Progressive Retinal Atrophy 5, PRA5

Progressive Retinal Atrophy, crd4/crd1

✅ Clear results

Breed-relevant (6)

Other (262)



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BREED-RELEVANT RESULTS

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

⚠️	Intervertebral Disc Disease (Type I) (FGF4 retrogene - CFA12)	Increased risk
⚠️	Progressive Retinal Atrophy 5, PRA5 (NECAP1 Exon 6, Giant Schnauzer Variant)	Notable
⚠️	Progressive Retinal Atrophy, crd4/cord1 (RPGRIP1)	Notable
✅	Limb-Girdle Muscular Dystrophy 2D (SGCA Exon 3, Miniature Dachshund Variant)	Clear
✅	Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, Dachshund Variant)	Clear
✅	Narcolepsy (HCRTR2 Exon 1, Dachshund Variant)	Clear
✅	Neuronal Ceroid Lipofuscinosis 1, NCL 1 (PPT1 Exon 8, Dachshund Variant 1)	Clear
✅	Neuronal Ceroid Lipofuscinosis 2, NCL 2 (TPP1 Exon 4, Dachshund Variant 2)	Clear
✅	Osteogenesis Imperfecta (SERPINH1, Dachshund Variant)	Clear

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OTHER RESULTS

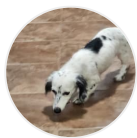
Research has not yet linked these conditions to dogs with similar breeds to Idgie. Review any increased risk or notable results to understand her potential risk and recommendations.

⊖ ALT Activity (GPT)	Notable
⊖ Copper Toxicosis (Accumulating) (ATP7B)	Notable
✓ 2-DHA Kidney & Bladder Stones (APRT)	Clear
✓ Acral Mutilation Syndrome (GDNF-AS, Spaniel and Pointer Variant)	Clear
✓ Alaskan Husky Encephalopathy (SLC19A3)	Clear
✓ Alaskan Malamute Polyneuropathy, AMPN (NDRG1 SNP)	Clear
✓ Alexander Disease (GFAP)	Clear
✓ Anhidrotic Ectodermal Dysplasia (EDA Intron 8)	Clear
✓ Autosomal Dominant Progressive Retinal Atrophy (RHO)	Clear
✓ Bald Thigh Syndrome (IGFBP5)	Clear
✓ Bernard-Soulier Syndrome, BSS (GP9, Cocker Spaniel Variant)	Clear
✓ Bully Whippet Syndrome (MSTN)	Clear
✓ Canine Elliptocytosis (SPTB Exon 30)	Clear
✓ Canine Fucosidosis (FUCA1)	Clear
✓ Canine Leukocyte Adhesion Deficiency Type I, CLAD I (ITGB2, Setter Variant)	Clear
✓ Canine Leukocyte Adhesion Deficiency Type III, CLAD III (FERMT3, German Shepherd Variant)	Clear
✓ Canine Multifocal Retinopathy, cmr1 (BEST1 Exon 2)	Clear
✓ Canine Multifocal Retinopathy, cmr2 (BEST1 Exon 5, Coton de Tulear Variant)	Clear

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✓ Canine Multifocal Retinopathy, cmr3 (BEST1 Exon 10 Deletion, Finnish and Swedish Lapphund, Lapponian Herder Variant)	Clear
✓ Canine Multiple System Degeneration (SERAC1 Exon 4, Chinese Crested Variant)	Clear
✓ Canine Multiple System Degeneration (SERAC1 Exon 15, Kerry Blue Terrier Variant)	Clear
✓ Cardiomyopathy and Juvenile Mortality (YARS2)	Clear
✓ Centronuclear Myopathy, CNM (PTPLA)	Clear
✓ Cerebellar Hypoplasia (VLDLR, Eurasier Variant)	Clear
✓ Chondrodysplasia (ITGA10, Norwegian Elkhound and Karelian Bear Dog Variant)	Clear
✓ Cleft Lip and/or Cleft Palate (ADAMTS20, Nova Scotia Duck Tolling Retriever Variant)	Clear
✓ Cleft Palate, CP1 (DLX6 intron 2, Nova Scotia Duck Tolling Retriever Variant)	Clear
✓ Cobalamin Malabsorption (CUBN Exon 8, Beagle Variant)	Clear
✓ Cobalamin Malabsorption (CUBN Exon 53, Border Collie Variant)	Clear
✓ Collie Eye Anomaly (NHEJ1)	Clear
✓ Complement 3 Deficiency, C3 Deficiency (C3)	Clear
✓ Congenital Cornification Disorder (NSDHL, Chihuahua Variant)	Clear
✓ Congenital Dyserythropoietic Anemia and Polymyopathy (EHPB1L1, Labrador Retriever Variant)	Clear
✓ Congenital Hypothyroidism (TPO, Rat, Toy, Hairless Terrier Variant)	Clear
✓ Congenital Hypothyroidism (TPO, Tenterfield Terrier Variant)	Clear
✓ Congenital Hypothyroidism with Goiter (TPO Intron 13, French Bulldog Variant)	Clear

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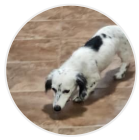
OTHER RESULTS

✓ Congenital Hypothyroidism with Goiter (SLC5A5, Shih Tzu Variant)	Clear
✓ Congenital Macrothrombocytopenia (TUBB1 Exon 1, Cairn and Norfolk Terrier Variant)	Clear
✓ Congenital Muscular Dystrophy (LAMA2, Italian Greyhound)	Clear
✓ Congenital Myasthenic Syndrome, CMS (COLQ, Labrador Retriever Variant)	Clear
✓ Congenital Myasthenic Syndrome, CMS (COLQ, Golden Retriever Variant)	Clear
✓ Congenital Myasthenic Syndrome, CMS (CHAT, Old Danish Pointing Dog Variant)	Clear
✓ Congenital Myasthenic Syndrome, CMS (CHRNE, Jack Russell Terrier Variant)	Clear
✓ Congenital Stationary Night Blindness (LRIT3, Beagle Variant)	Clear
✓ Congenital Stationary Night Blindness (RPE65, Briard Variant)	Clear
✓ Copper Toxicosis (Attenuating) (ATP7A, Labrador Retriever)	Clear
✓ Copper Toxicosis (Attenuating) (RETN, Labrador Retriever)	Clear
✓ Craniomandibular Osteopathy, CMO (SLC37A2)	Clear
✓ Craniomandibular Osteopathy, CMO (SLC37A2 Intron 16, Basset Hound Variant)	Clear
✓ Cystinuria Type I-A (SLC3A1, Newfoundland Variant)	Clear
✓ Cystinuria Type II-A (SLC3A1, Australian Cattle Dog Variant)	Clear
✓ Cystinuria Type II-B (SLC7A9, Miniature Pinscher Variant)	Clear
✓ Darier Disease (ATP2A2, Irish Terrier Variant)	Clear
✓ Day Blindness (CNGB3 Deletion, Alaskan Malamute Variant)	Clear

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OTHER RESULTS

✓ Day Blindness (CNGA3 Exon 7, German Shepherd Variant)	Clear
✓ Day Blindness (CNGA3 Exon 7, Labrador Retriever Variant)	Clear
✓ Day Blindness (CNGB3 Exon 6, German Shorthaired Pointer Variant)	Clear
✓ Deafness and Vestibular Syndrome of Dobermans, DVDob, DINGS (MYO7A)	Clear
✓ Degenerative Myelopathy, DM (SOD1A)	Clear
✓ Demyelinating Polyneuropathy (SBF2/MTRM13)	Clear
✓ Dental-Skeletal-Retinal Anomaly (MIA3, Cane Corso Variant)	Clear
✓ Diffuse Cystic Renal Dysplasia and Hepatic Fibrosis (INPP5E Intron 9, Norwich Terrier Variant)	Clear
✓ Dilated Cardiomyopathy, DCM (RBM20, Schnauzer Variant)	Clear
✓ Dilated Cardiomyopathy, DCM1 (PDK4, Doberman Pinscher Variant 1)	Clear
✓ Dilated Cardiomyopathy, DCM2 (TTN, Doberman Pinscher Variant 2)	Clear
✓ Disproportionate Dwarfism (PRKG2, Dogo Argentino Variant)	Clear
✓ Dry Eye Curly Coat Syndrome (FAM83H Exon 5)	Clear
✓ Dystrophic Epidermolysis Bullosa (COL7A1, Central Asian Shepherd Dog Variant)	Clear
✓ Dystrophic Epidermolysis Bullosa (COL7A1, Golden Retriever Variant)	Clear
✓ Early Bilateral Deafness (LOXHD1 Exon 38, Rottweiler Variant)	Clear
✓ Early Onset Adult Deafness, EOAD (EPS8L2 Deletion, Rhodesian Ridgeback Variant)	Clear
✓ Early Onset Cerebellar Ataxia (SEL1L, Finnish Hound Variant)	Clear

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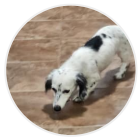
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OTHER RESULTS

✓ Ehlers Danlos (ADAMTS2, Doberman Pinscher Variant)	Clear
✓ Ehlers-Danlos Syndrome (EDS) (COL5A1, Labrador Retriever Variant)	Clear
✓ Enamel Hypoplasia (ENAM Deletion, Italian Greyhound Variant)	Clear
✓ Enamel Hypoplasia (ENAM SNP, Parson Russell Terrier Variant)	Clear
✓ Episodic Falling Syndrome (BCAN)	Clear
✓ Exercise-Induced Collapse, EIC (DNM1)	Clear
✓ Factor VII Deficiency (F7 Exon 5)	Clear
✓ Factor XI Deficiency (F11 Exon 7, Kerry Blue Terrier Variant)	Clear
✓ Familial Nephropathy (COL4A4 Exon 3, Cocker Spaniel Variant)	Clear
✓ Familial Nephropathy (COL4A4 Exon 30, English Springer Spaniel Variant)	Clear
✓ Fanconi Syndrome (FAN1, Basenji Variant)	Clear
✓ Fetal-Onset Neonatal Neuroaxonal Dystrophy (MFN2, Giant Schnauzer Variant)	Clear
✓ Glanzmann's Thrombasthenia Type I (ITGA2B Exon 13, Great Pyrenees Variant)	Clear
✓ Glanzmann's Thrombasthenia Type I (ITGA2B Exon 12, Otterhound Variant)	Clear
✓ Globoid Cell Leukodystrophy, Krabbe disease (GALC Exon 5, Terrier Variant)	Clear
✓ Glycogen Storage Disease Type IA, Von Gierke Disease, GSD IA (G6PC1, German Pinscher Variant)	Clear
✓ Glycogen Storage Disease Type IA, Von Gierke Disease, GSD IA (G6PC, Maltese Variant)	Clear
✓ Glycogen Storage Disease Type IIIA, GSD IIIA (AGL, Curly Coated Retriever Variant)	Clear

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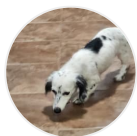
OTHER RESULTS

✓ Glycogen storage disease Type VII, Phosphofructokinase Deficiency, PFK Deficiency (PFKM, Whippet and English Springer Spaniel Variant)	Clear
✓ Glycogen storage disease Type VII, Phosphofructokinase Deficiency, PFK Deficiency (PFKM, Wachtelhund Variant)	Clear
✓ GM1 Gangliosidosis (GLB1 Exon 2, Portuguese Water Dog Variant)	Clear
✓ GM1 Gangliosidosis (GLB1 Exon 15, Shiba Inu Variant)	Clear
✓ GM1 Gangliosidosis (GLB1 Exon 15, Alaskan Husky Variant)	Clear
✓ GM2 Gangliosidosis (HEXA, Japanese Chin Variant)	Clear
✓ GM2 Gangliosidosis (HEXB, Poodle Variant)	Clear
✓ Golden Retriever Progressive Retinal Atrophy 1, GR-PRA1 (SLC4A3)	Clear
✓ Golden Retriever Progressive Retinal Atrophy 2, GR-PRA2 (TTC8)	Clear
✓ Goniodysgenesis and Glaucoma, Pectinate Ligament Dysplasia, PLD (OLFM3)	Clear
✓ Hemophilia A (F8 Exon 11, German Shepherd Variant 1)	Clear
✓ Hemophilia A (F8 Exon 1, German Shepherd Variant 2)	Clear
✓ Hemophilia A (F8 Exon 10, Boxer Variant)	Clear
✓ Hemophilia B (F9 Exon 7, Terrier Variant)	Clear
✓ Hemophilia B (F9 Exon 7, Rhodesian Ridgeback Variant)	Clear
✓ Hereditary Ataxia (PNPLA8, Australian Shepherd Variant)	Clear
✓ Hereditary Ataxia, Cerebellar Degeneration (RAB24, Old English Sheepdog and Gordon Setter Variant)	Clear
✓ Hereditary Cataracts (HSF4 Exon 9, Australian Shepherd Variant)	Clear

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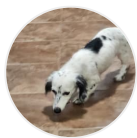
OTHER RESULTS

✓ Hereditary Cataracts (FYCO1, Wirehaired Pointing Griffon Variant)	Clear
✓ Hereditary Cerebellar Ataxia (SELENOP, Belgian Shepherd Variant)	Clear
✓ Hereditary Footpad Hyperkeratosis (FAM83G, Terrier and Kromfohrlander Variant)	Clear
✓ Hereditary Footpad Hyperkeratosis (DSG1, Rottweiler Variant)	Clear
✓ Hereditary Nasal Parakeratosis (SUV39H2 Intron 4, Greyhound Variant)	Clear
✓ Hereditary Nasal Parakeratosis, HNPk (SUV39H2)	Clear
✓ Hereditary Vitamin D-Resistant Rickets (VDR)	Clear
✓ Hypocatalasia, Acatlasemia (CAT)	Clear
✓ Hypomyelination and Tremors (FNIP2, Weimaraner Variant)	Clear
✓ Hypophosphatasia (ALPL Exon 9, Karelian Bear Dog Variant)	Clear
✓ Ichthyosis (NIPAL4, American Bulldog Variant)	Clear
✓ Ichthyosis (ASPRV1 Exon 2, German Shepherd Variant)	Clear
✓ Ichthyosis (SLC27A4, Great Dane Variant)	Clear
✓ Ichthyosis, Epidermolytic Hyperkeratosis (KRT10, Terrier Variant)	Clear
✓ Ichthyosis, ICH1 (PNPLA1, Golden Retriever Variant)	Clear
✓ Ichthyosis, ICH2 (ABHD5, Golden Retriever Variant)	Clear
✓ Inflammatory Myopathy (SLC25A12)	Clear
✓ Inherited Myopathy of Great Danes (BIN1)	Clear

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OTHER RESULTS

✓ Inherited Selected Cobalamin Malabsorption with Proteinuria (CUBN, Komondor Variant)	Clear
✓ Intestinal Lipid Malabsorption (ACSL5, Australian Kelpie)	Clear
✓ Junctional Epidermolysis Bullosa (LAMA3 Exon 66, Australian Cattle Dog Variant)	Clear
✓ Junctional Epidermolysis Bullosa (LAMB3 Exon 11, Australian Shepherd Variant)	Clear
✓ Juvenile Epilepsy (LGI2)	Clear
✓ Juvenile Laryngeal Paralysis and Polyneuropathy (RAB3GAP1, Rottweiler Variant)	Clear
✓ Juvenile Myoclonic Epilepsy (DIRAS1)	Clear
✓ L-2-Hydroxyglutaricaciduria, L2HGA (L2HGDH, Staffordshire Bull Terrier Variant)	Clear
✓ Lagotto Storage Disease (ATG4D)	Clear
✓ Laryngeal Paralysis (RAPGEF6, Miniature Bull Terrier Variant)	Clear
✓ Laryngeal Paralysis and Polyneuropathy (CNTNAP1, Leonberger, Saint Bernard, and Labrador Retriever variant)	Clear
✓ Late Onset Spinocerebellar Ataxia (CAPN1)	Clear
✓ Late-Onset Neuronal Ceroid Lipofuscinosis, NCL 12 (ATP13A2, Australian Cattle Dog Variant)	Clear
✓ Leonberger Polyneuropathy 1 (LPN1, ARHGEF10)	Clear
✓ Leonberger Polyneuropathy 2 (GJA9)	Clear
✓ Lethal Acrodermatitis, LAD (MKLN1)	Clear
✓ Leukodystrophy (TSEN54 Exon 5, Standard Schnauzer Variant)	Clear
✓ Lignous Membranitis, LM (PLG)	Clear

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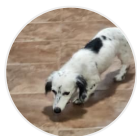
OTHER RESULTS

✓ Limb Girdle Muscular Dystrophy (SGCD, Boston Terrier Variant)	Clear
✓ Long QT Syndrome (KCNQ1)	Clear
✓ Lundehund Syndrome (LEPREL1)	Clear
✓ Macular Corneal Dystrophy, MCD (CHST6)	Clear
✓ Malignant Hyperthermia (RYR1)	Clear
✓ May-Hegglin Anomaly (MYH9)	Clear
✓ MDR1 Drug Sensitivity (ABCB1)	Clear
✓ Medium-Chain Acyl-CoA Dehydrogenase Deficiency, MCADD (ACADM, Cavalier King Charles Spaniel Variant)	Clear
✓ Methemoglobinemia (CYB5R3, Pit Bull Terrier Variant)	Clear
✓ Methemoglobinemia (CYB5R3)	Clear
✓ Microphthalmia (RBP4 Exon 2, Soft Coated Wheaten Terrier Variant)	Clear
✓ Mucopolysaccharidosis IIIB, Sanfilippo Syndrome Type B, MPS IIIB (NAGLU, Schipperke Variant)	Clear
✓ Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, New Zealand Huntaway Variant)	Clear
✓ Mucopolysaccharidosis Type VI, Maroteaux-Lamy Syndrome, MPS VI (ARSB Exon 5, Miniature Pinscher Variant)	Clear
✓ Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 3, German Shepherd Variant)	Clear
✓ Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 5, Terrier Brasileiro Variant)	Clear
✓ Muscular Dystrophy (DMD, Cavalier King Charles Spaniel Variant 1)	Clear
✓ Muscular Dystrophy (DMD, Golden Retriever Variant)	Clear

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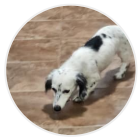
OTHER RESULTS

✓ Muscular Dystrophy-Dystroglycanopathy (LARGE1, Labrador Retriever Variant)	Clear
✓ Musladin-Lueke Syndrome, MLS (ADAMTSL2)	Clear
✓ Myasthenia Gravis-Like Syndrome (CHRNE, Heideterrrier Variant)	Clear
✓ Myotonia Congenita (CLCN1 Exon 23, Australian Cattle Dog Variant)	Clear
✓ Myotonia Congenita (CLCN1 Exon 19, Labrador Retriever Variant)	Clear
✓ Myotonia Congenita (CLCN1 Exon 7, Miniature Schnauzer Variant)	Clear
✓ Narcolepsy (HCRT2 Intron 4, Doberman Pinscher Variant)	Clear
✓ Narcolepsy (HCRT2 Intron 6, Labrador Retriever Variant)	Clear
✓ Nemaline Myopathy (NEB, American Bulldog Variant)	Clear
✓ Neonatal Cerebellar Cortical Degeneration (SPTBN2, Beagle Variant)	Clear
✓ Neonatal Encephalopathy with Seizures, NEWS (ATF2)	Clear
✓ Neonatal Interstitial Lung Disease (LAMP3)	Clear
✓ Neuroaxonal Dystrophy, NAD (VPS11, Rottweiler Variant)	Clear
✓ Neuroaxonal Dystrophy, NAD (TECPR2, Spanish Water Dog Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis 10, NCL 10 (CTSD Exon 5, American Bulldog Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 SNP, Border Collie Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 Deletion, Golden Retriever Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis 6, NCL 6 (CLN6 Exon 7, Australian Shepherd Variant)	Clear

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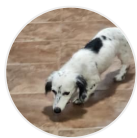
OTHER RESULTS

✓ Neuronal Ceroid Lipofuscinosis 7, NCL 7 (MFSD8, Chihuahua and Chinese Crested Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8, Australian Shepherd Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Exon 2, English Setter Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Insertion, Saluki Variant)	Clear
✓ Neuronal Ceroid Lipofuscinosis, Cerebellar Ataxia, NCL4A (ARSG Exon 2, American Staffordshire Terrier Variant)	Clear
✓ Oculocutaneous Albinism, OCA (SLC45A2 Exon 6, Bullmastiff Variant)	Clear
✓ Oculocutaneous Albinism, OCA (SLC45A2, Small Breed Variant)	Clear
✓ Oculoskeletal Dysplasia 2 (COL9A2, Samoyed Variant)	Clear
✓ Osteochondrodysplasia (SLC13A1, Poodle Variant)	Clear
✓ Osteogenesis Imperfecta (COL1A2, Beagle Variant)	Clear
✓ Osteogenesis Imperfecta (COL1A1, Golden Retriever Variant)	Clear
✓ P2Y12 Receptor Platelet Disorder (P2Y12)	Clear
✓ Pachyonychia Congenita (KRT16, Dogue de Bordeaux Variant)	Clear
✓ Paroxysmal Dyskinesia, PxD (PIGN)	Clear
✓ Persistent Mullerian Duct Syndrome, PMDS (AMHR2)	Clear
✓ Pituitary Dwarfism (POU1F1 Intron 4, Karelian Bear Dog Variant)	Clear
✓ Platelet Factor X Receptor Deficiency, Scott Syndrome (TMEM16F)	Clear
✓ Polycystic Kidney Disease, PKD (PKD1)	Clear

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✓ Pompe's Disease (GAA, Finnish and Swedish Lapphund, Lapponian Herder Variant)	Clear
✓ Prekallikrein Deficiency (KLKB1 Exon 8)	Clear
✓ Primary Ciliary Dyskinesia, PCD (NME5, Alaskan Malamute Variant)	Clear
✓ Primary Ciliary Dyskinesia, PCD (STK36, Australian Shepherd Variant)	Clear
✓ Primary Ciliary Dyskinesia, PCD (CCDC39 Exon 3, Old English Sheepdog Variant)	Clear
✓ Primary Hyperoxaluria (AGXT)	Clear
✓ Primary Lens Luxation (ADAMTS17)	Clear
✓ Primary Open Angle Glaucoma (ADAMTS17 Exon 11, Basset Fauve de Bretagne Variant)	Clear
✓ Primary Open Angle Glaucoma (ADAMTS10 Exon 17, Beagle Variant)	Clear
✓ Primary Open Angle Glaucoma (ADAMTS10 Exon 9, Norwegian Elkhound Variant)	Clear
✓ Primary Open Angle Glaucoma and Primary Lens Luxation (ADAMTS17 Exon 2, Chinese Shar-Pei Variant)	Clear
✓ Progressive Retinal Atrophy (SAG)	Clear
✓ Progressive Retinal Atrophy (IFT122 Exon 26, Lapponian Herder Variant)	Clear
✓ Progressive Retinal Atrophy, Bardet-Biedl Syndrome (BBS2 Exon 11, Shetland Sheepdog Variant)	Clear
✓ Progressive Retinal Atrophy, CNGA (CNGA1 Exon 9)	Clear
✓ Progressive Retinal Atrophy, crd1 (PDE6B, American Staffordshire Terrier Variant)	Clear
✓ Progressive Retinal Atrophy, PRA1 (CNGB1)	Clear
✓ Progressive Retinal Atrophy, PRA3 (FAM161A)	Clear

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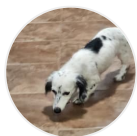
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OTHER RESULTS

✓ Progressive Retinal Atrophy, prcd (PRCD Exon 1)	Clear
✓ Progressive Retinal Atrophy, rcd1 (PDE6B Exon 21, Irish Setter Variant)	Clear
✓ Progressive Retinal Atrophy, rcd3 (PDE6A)	Clear
✓ Proportionate Dwarfism (GH1 Exon 5, Chihuahua Variant)	Clear
✓ Protein Losing Nephropathy, PLN (NPHS1)	Clear
✓ Pyruvate Dehydrogenase Deficiency (PDP1, Spaniel Variant)	Clear
✓ Pyruvate Kinase Deficiency (PKLR Exon 5, Basenji Variant)	Clear
✓ Pyruvate Kinase Deficiency (PKLR Exon 7, Beagle Variant)	Clear
✓ Pyruvate Kinase Deficiency (PKLR Exon 10, Terrier Variant)	Clear
✓ Pyruvate Kinase Deficiency (PKLR Exon 7, Labrador Retriever Variant)	Clear
✓ Pyruvate Kinase Deficiency (PKLR Exon 7, Pug Variant)	Clear
✓ Raine Syndrome (FAM20C)	Clear
✓ Recurrent Inflammatory Pulmonary Disease, RIPD (AKNA, Rough Collie Variant)	Clear
✓ Renal Cystadenocarcinoma and Nodular Dermatofibrosis (FLCN Exon 7)	Clear
✓ Retina Dysplasia and/or Optic Nerve Hypoplasia (SIX6 Exon 1, Golden Retriever Variant)	Clear
✓ Sensory Neuropathy (FAM134B, Border Collie Variant)	Clear
✓ Severe Combined Immunodeficiency, SCID (PRKDC, Terrier Variant)	Clear
✓ Severe Combined Immunodeficiency, SCID (RAG1, Wetterhoun Variant)	Clear

Registration: American Kennel Club (AKC)
HP70221401





"IDGIE"

"AMY'S CANDI DOTS"



DNA Test Report

Test Date: March 14th, 2025

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OTHER RESULTS

✓ Shaking Puppy Syndrome (PLP1, English Springer Spaniel Variant)	Clear
✓ Shar-Pei Autoinflammatory Disease, SPAID, Shar-Pei Fever (MTBP)	Clear
✓ Skeletal Dysplasia 2, SD2 (COL11A2, Labrador Retriever Variant)	Clear
✓ Skin Fragility Syndrome (PKP1, Chesapeake Bay Retriever Variant)	Clear
✓ Spinocerebellar Ataxia (SCN8A, Alpine Dachsbracke Variant)	Clear
✓ Spinocerebellar Ataxia with Myokymia and/or Seizures (KCNJ10)	Clear
✓ Spongy Degeneration with Cerebellar Ataxia 1 (KCNJ10)	Clear
✓ Spongy Degeneration with Cerebellar Ataxia 2 (ATP1B2)	Clear
✓ Stargardt Disease (ABCA4 Exon 28, Labrador Retriever Variant)	Clear
✓ Succinic Semialdehyde Dehydrogenase Deficiency (ALDH5A1 Exon 7, Saluki Variant)	Clear
✓ Thrombopathia (RASGRP1 Exon 5, American Eskimo Dog Variant)	Clear
✓ Thrombopathia (RASGRP1 Exon 5, Basset Hound Variant)	Clear
✓ Thrombopathia (RASGRP1 Exon 8, Landseer Variant)	Clear
✓ Trapped Neutrophil Syndrome, TNS (VPS13B)	Clear
✓ Ullrich-like Congenital Muscular Dystrophy (COL6A3 Exon 10, Labrador Retriever Variant)	Clear
✓ Ullrich-like Congenital Muscular Dystrophy (COL6A1 Exon 3, Landseer Variant)	Clear
✓ Unilateral Deafness and Vestibular Syndrome (PTPRQ Exon 39, Doberman Pinscher)	Clear
✓ Urate Kidney & Bladder Stones (SLC2A9)	Clear

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OTHER RESULTS

✓ Von Willebrand Disease Type I, Type I vWD (VWF)	Clear
✓ Von Willebrand Disease Type II, Type II vWD (VWF, Pointer Variant)	Clear
✓ Von Willebrand Disease Type III, Type III vWD (VWF Exon 4, Terrier Variant)	Clear
✓ Von Willebrand Disease Type III, Type III vWD (VWF Intron 16, Nederlandse Kooikerhondje Variant)	Clear
✓ Von Willebrand Disease Type III, Type III vWD (VWF Exon 7, Shetland Sheepdog Variant)	Clear
✓ X-Linked Hereditary Nephropathy, XLHN (COL4A5 Exon 35, Samoyed Variant 2)	Clear
✓ X-Linked Myotubular Myopathy (MTM1, Labrador Retriever Variant)	Clear
✓ X-Linked Progressive Retinal Atrophy 1, XL-PRA1 (RPGR)	Clear
✓ X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG Exon 1, Basset Hound Variant)	Clear
✓ X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG, Corgi Variant)	Clear
✓ Xanthine Urolithiasis (XDH, Mixed Breed Variant)	Clear
✓ β -Mannosidosis (MANBA Exon 16, Mixed-Breed Variant)	Clear
Mast Cell Tumor	No result

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HEALTH REPORT

Increased risk result

Intervertebral Disc Disease (Type I)

"Amy's Candi Dots" inherited one copy of the variant we tested for Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type I IVDD

Idgie is at increased risk for Type I IVDD

How to interpret this result

Idgie 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.

Actions to take if your dog is affected

- Talk to your vet about your dog's chondrodystrophy and intervertebral disc disease result so you can discuss how it may influence their daily activities and lifestyle.
- This variant is very common in certain breeds, and many dogs with this result will not need any special accommodations because they are unlikely to develop symptoms. However, some breeds are at greater risk, and precautions may help reduce strain on the back and neck.
- Keep your dog fit with regular, low-impact exercise and maintain a healthy weight to support spinal health.
- Consider using ramps to access furniture, avoiding long flights of stairs, and choosing a harness instead of a collar to minimize stress on the spine.

Registration:





"IDGIE"

"AMY'S CANDI DOTS"



DNA Test Report

Test Date: March 14th, 2025

embk.me/idgie81

HEALTH REPORT

Notable result

ALT Activity

"Amy's Candi Dots" inherited one copy of the variant we tested for Alanine Aminotransferase Activity

Why is this important to your vet?

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

Actions to take if your dog is affected

- Talk to your vet about your dog's ALT result, as it may help them better interpret your dog's blood work.
- Dogs with this result do not exhibit symptoms or develop health issues associated with this variant.



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HEALTH REPORT

⊖ Notable result

Copper Toxicosis (Accumulating)

"Amy's Candi Dots" inherited one copy of the variant we tested for Copper Toxicosis (Accumulating)

Idgie is not known to be at increased risk for Copper Toxicosis (Accumulating)

What does this result mean?

We do not know whether this increases the risk that Idgie will develop Copper Toxicosis (Accumulating).

Scientific Basis

Research studies for this variant have been based on dogs of other breeds. Not enough dogs with Idgie's breed have been studied to know whether or not this variant will increase Idgie's risk of developing this disease.

Impact on Breeding

Research into the clinical impact of this variant is ongoing. We recommend tracking this genetic result and incidence of Copper Toxicosis (Accumulating) in your breeding program and related dogs.

What is Copper Toxicosis (Accumulating)?

Copper toxicosis is a condition in which affected dogs have difficulty excreting excess copper from their liver. The liver accumulates more copper until it eventually begins failing. Multiple genetic and environmental factors contribute to the development of this condition.

When signs & symptoms develop in affected dogs

Signs typically develop in adults.

How vets diagnose this condition

Genetic testing, blood work, abdominal ultrasound, and surgical biopsy are all used to diagnose this condition.

How this condition is treated

Treatment includes a low copper diet and medical management to help bind excess copper. Antioxidant supplements may also be considered.

Actions to take if your dog is affected

- Talk to your vet about your dog's copper toxicosis result so you can discuss if dietary management or monitoring is indicated.
- Copper is an essential nutrient, but amounts can vary widely among commercial diets, so your vet may recommend a specific food or periodic testing to maintain safe levels.
- Many dogs with this result never develop clinical disease. Watch for signs that may indicate high copper levels, such as decreased appetite, vomiting, lethargy, or jaundice.
- Learn more about how the three variants for Copper Toxicosis are inherited and, if applicable, how results can be used in a breeding program here (<https://embarkvet.com/resources/embark-adds-copper-toxicosis-dna-test/>).

Registration:





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HEALTH REPORT

⊖ Notable result

Progressive Retinal Atrophy 5, PRA5

"Amy's Candi Dots" inherited one copy of the variant we tested for Progressive Retinal Atrophy 5, PRA5

What does this result mean?

This variant should not impact Idgie's health. This variant is inherited in an autosomal recessive manner, meaning a dog needs two copies of the variant to show signs of this condition. Idgie is unlikely to develop this condition due to this variant because she only has one copy of the variant.

Impact on Breeding

Your dog carries this variant and will pass it on to ~50% of her offspring. You can email breeders@embarkvet.com to discuss with a genetic counselor how the genotype results should be applied to a breeding program.

What is Progressive Retinal Atrophy 5, PRA5?

Progressive retinal atrophy (PRA) describes a group of non-painful inherited disorders of the retina's photoreceptor cells that result in vision loss in dogs. PRA caused by this NECAP1 variant is a late-onset (later in life) form of PRA, affecting dogs whose eyes have completed their normal development.

When signs & symptoms develop in affected dogs

This form of PRA is typically diagnosed at around four years old.

How vets diagnose this condition

PRA is diagnosed by examination of the back of the eye (the fundus). In the early stages, observing changes to the retina may be difficult. Still, as the disease progresses, a veterinarian will notice increased reflectivity and thinning of the retinal blood vessels. Changes affect both eyes equally, helping to distinguish PRA from other retinal diseases. A veterinary ophthalmologist can perform an ERG, which measures the electrical activity, and thus the function, of the retinas.

How this condition is treated

Currently, there is no widespread treatment for progressive retinal atrophy. However, gene therapy is an evolving field.

Actions to take if your dog is affected

- Talk to your vet about your dog's PRA result so you can work together to plan their ongoing care and monitoring.
- Schedule regular eye exams with your vet or a veterinary ophthalmologist to monitor for changes or complications such as cataracts.
- If your dog's vision changes, help them adjust by keeping furniture and routines consistent, using verbal cues, and keeping them on a leash in unfamiliar areas.
- Products such as protective halos can also help dogs navigate safely if their vision declines.

Registration:





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HEALTH REPORT

Notable result

Progressive Retinal Atrophy, crd4/cord1

"Amy's Candi Dots" inherited one copy of the variant we tested for Progressive Retinal Atrophy, crd4/cord1

What does this result mean?

This variant should not impact Idgie's health. This variant is inherited in an autosomal recessive manner, meaning that a dog needs two copies of the variant to show signs of this condition. Idgie is unlikely to develop this condition due to this variant because she only has one copy of the variant.

Impact on Breeding

Your dog carries this variant and will pass it on to ~50% of her offspring. You can email breeders@embarkvet.com to discuss with a genetic counselor how the genotype results should be applied to a breeding program.

What is Progressive Retinal Atrophy, crd4/cord1?

PRA-CRD4/cord1 is a retinal disease that causes progressive, non-painful vision loss over a 1-2 year period. 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 cone cells, causing day blindness before night blindness.

When signs & symptoms develop in affected dogs

The earliest ophthalmic signs are typically present by 6 months of age. There is a wide range in the age of when dogs become clinically affected, although the average age is approximately 5 years. Dogs as young as 6 months may be blind, while dogs as old as 10 may still have vision.

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

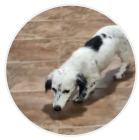
Currently, there is no definitive treatment for PRA. Supplements, including antioxidants, have been proposed for management of the disease, but have not been scientifically proven effective.

Actions to take if your dog is affected

- Talk to your vet about your dog's PRA result so you can work together to plan their ongoing care and monitoring.
- Schedule regular eye exams with your vet or a veterinary ophthalmologist to monitor for changes or complications such as cataracts.
- If your dog's vision changes, help them adjust by keeping furniture and routines consistent, using verbal cues, and keeping them on a leash in unfamiliar areas.
- Products such as protective halos can also help dogs navigate safely if their vision declines.

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INBREEDING AND DIVERSITY

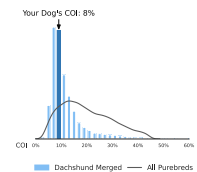
CATEGORY

RESULT

Coefficient Of Inbreeding

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

8%

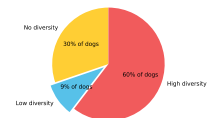


MHC Class II - DLA DRB1

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

Low Diversity

How common is this amount of diversity in purebreds:



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.

Low Diversity

How common is this amount of diversity in purebreds:

