# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## BREED ANCESTRY

Poodle (Small) : 50.0%

Cavalier King Charles Spaniel : 50.0%

## GENETIC STATS

Predicted adult weight: **12 lbs**

## TEST DETAILS

Kit number: EM-28724503

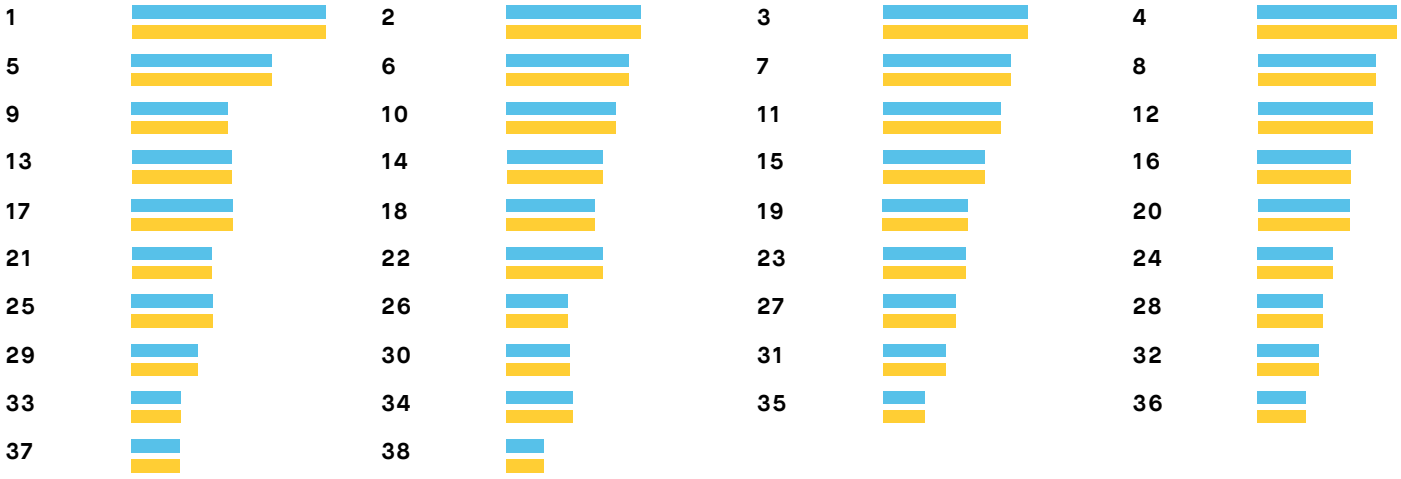
Swab number: 31241260208389

## BREED ANCESTRY BY CHROMOSOME

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

Breed colors:

Poodle (Small) Cavalier King Charles Spaniel



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## POODLE (SMALL)

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

**Alternative Names**
Toy Poodle, Miniature Poodle

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

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## CAVALIER KING CHARLES SPANIEL

The Cavalier King Charles Spaniel is one of the most popular dog breeds in the United States, and with good reason. Their affectionate personalities combined with their need to be close to their humans make them a lovely breed of choice for families. They tend to get along well with children and peaceably with other dogs and animals in the home (though as the breed used to be used for hunting, caution around small animals should be exercised). The Cavalier has an interesting history -- their ancestors were dogs of the British monarchy, but over time, the breed began to die out as dogs with shorter muzzles were favored in the 1800s. They were crossed with Pugs and some other breeds to change their appearance. However, Roswell Eldridge sought out King Charles Spaniels that had longer muzzles, and recreated the Cavalier as it used to be from those dogs.

**Fun Fact**

The breed experienced two large bursts in popularity. The first is when Queen Victoria revived the dying breed. The second was when Charlotte, a popular character from the popular show Sex and the City adopted one on TV.

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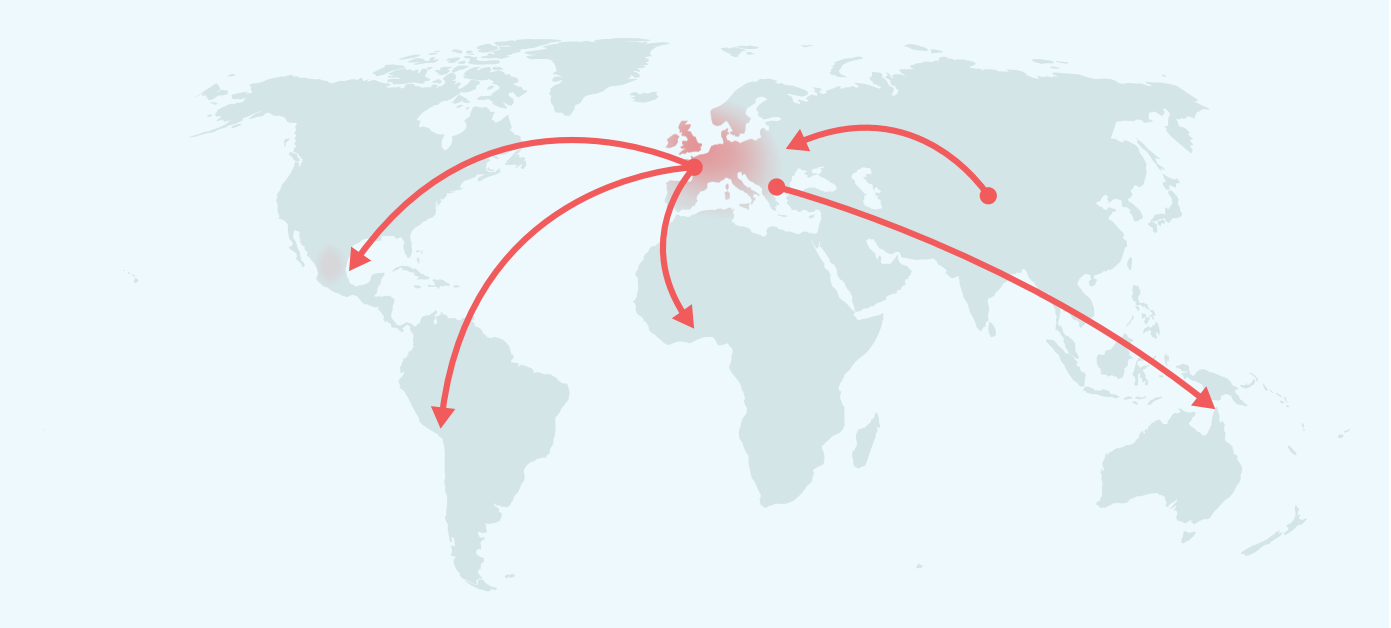
## MATERNAL LINE



Through Peaches’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: A1b**

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

**HAPLOTYPE: A413**

Part of the A1b haplogroup, the A413 haplotype occurs most commonly in Cavalier King Charles Spaniels. It's a rare find!

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## TRAITS: COAT COLOR

| TRAIT | RESULT |
|---|---|
| **E Locus (MC1R)**<br><br>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.<br><br>Dogs with one or two copies of the $\mathbf{E^m}$ variant may have a melanistic mask (dark facial hair as commonly seen in the German Shepherd Dog and Pug). In the absence of $\mathbf{E^m}$, dogs with the $\mathbf{E^g}$ 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 $\mathbf{E^m}$ and **E** variants, dogs with the $\mathbf{E^a}$ or $\mathbf{E^h}$ variants can express the grizzle phenotype. Additionally, a dog with any combination of two of the $\mathbf{E^g}$, $\mathbf{E^a}$, or $\mathbf{E^h}$ variants (example: $\mathbf{E^gE^a}$) is also expected to express the grizzle phenotype. | **No dark hairs anywhere (ee)** |
| **K Locus (CBD103)**<br><br>The K Locus $\mathbf{K^B}$ allele "overrides” the A Locus, meaning that it prevents the A Locus genotype from affecting coat color. For this reason, the $\mathbf{K^B}$ allele is referred to as the "dominant black” allele. As a result, dogs with at least one $\mathbf{K^B}$ 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 $\mathbf{k^yk^y}$ genotype will show a coat color pattern based on the genotype they have at the A Locus. Dogs who test as $\mathbf{K^Bk^y}$ may be brindle rather than black or brown. | **Not expressed ($k^yk^y$)** |

# “PEACHES”

## LINDSAY'S PEACHES



## TRAITS: COAT COLOR (CONTINUED)

| TRAIT | RESULT |
|---|---|
| **Intensity Loci**<br><br>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 pigmented hair likely apricot or red (Intense Red Pigmentation)** |
| **A Locus (ASIP)**<br><br>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 $\mathbf{k^y k^y}$ 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. | **Not expressed ($a^y a^t$)** |
| **D Locus (MLPH)**<br><br>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. | **Not expressed (DD)** |

## TRAITS: COAT COLOR (CONTINUED)

| TRAIT | RESULT |
|---|---|
| **Cocoa (HPS3)**<br><br>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)**<br><br>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". | **Likely black colored nose/feet (BB)** |
| **Saddle Tan (RALY)**<br><br>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 $\mathbf{a^t}$ allele, so dogs that do not express $\mathbf{a^t}$ are not influenced by this gene. | **Not expressed (NI)** |
| **S Locus (MITF)**<br><br>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 solid colored, but may have small amounts of white (Ssp)** |

# “PEACHES”

## LINDSAY'S PEACHES



## TRAITS: COAT COLOR (CONTINUED)

| TRAIT | RESULT |
| --- | --- |
| **M Locus (PMEL)**<br><br>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.<br><br>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. | **No merle alleles (mm)** |
| **R Locus (USH2A)**<br><br>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)**<br><br>This pattern is recognized in Great Danes and causes dogs to have a white coat with patches of darker pigment. A dog with an **Hh** result will be harlequin if they are also **M*m** or **M*M*** at the M Locus and are not **ee** at the E locus. Dogs with a result of **hh** will not be harlequin. This trait is thought to be homozygous lethal; a living dog with an **HH** genotype has never been found. | **No harlequin alleles (hh)** |

## TRAITS: COAT COLOR (CONTINUED)

| TRAIT | RESULT |
| --- | --- |
| **Panda White Spotting**<br><br>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.<br><br>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)** |

# “PEACHES”

## LINDSAY'S PEACHES



## TRAITS: OTHER COAT TRAITS

| TRAIT | RESULT |
| --- | --- |
| **Furnishings (RSPO2)**<br><br>Dogs with one or two copies of the **F** allele have "furnishings”: the mustache, beard, and eyebrows characteristic of breeds like the Schnauzer, Scottish Terrier, and Wire Haired Dachshund. A dog with two **I** alleles will not have furnishings, which is sometimes called an "improper coat” in breeds where furnishings are part of the breed standard. The mutation is a genetic insertion which we measure indirectly using a linkage test highly correlated with the insertion. | **Likely furnished (mustache, beard, and/or eyebrows) (FI)** |

## TRAITS: OTHER COAT TRAITS (CONTINUED)

| TRAIT | RESULT |
| --- | --- |
| **Coat Length (FGF5)**<br><br>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.<br><br>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.<br><br>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.<br><br>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. | **Likely long coat (LhLh)** |

## TRAITS: OTHER COAT TRAITS (CONTINUED)

| TRAIT | RESULT |
|---|---|
| **Shedding (MC5R)**<br><br>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)**<br><br>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 wavy coat (CC)** |
| **Hairlessness (FOXI3)**<br><br>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)**<br><br>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)** |

## TRAITS: OTHER COAT TRAITS (CONTINUED)

| TRAIT | RESULT |
| --- | --- |
| **Oculocutaneous Albinism Type 2 (SLC45A2)**<br><br>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)**<br><br>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)**<br><br>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.<br>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)**<br><br>Common in certain breeds such as the Saint Bernard, hind dewclaws are extra, nonfunctional digits located midway between a dog's paw and hock. Dogs with at least one copy of the **T** allele have about a 50% chance of having hind dewclaws. Note that other (currently unknown to science) mutations can also cause hind dewclaws, so some **CC** or **TC** dogs will have hind dewclaws. | **Unlikely to have hind dew claws (CC)** |

## TRAITS: OTHER BODY FEATURES (CONTINUED)

| TRAIT | RESULT |
|---|---|
| **Chondrodysplasia (Chr. 18 FGF4 Retrogene)**<br><br>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. | **Not indicative of chondrodysplasia (normal leg length) (NN)** |
| **Blue Eye Color (ALX4)**<br><br>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)**<br><br>The **T** allele is associated with heavy muscling along the back and trunk in characteristically "bulky" large-breed dogs including the Saint Bernard, Bernese Mountain Dog, Greater Swiss Mountain Dog, and Rottweiler. The "bulky” **T** allele is absent from leaner shaped large breed dogs like the Great Dane, Irish Wolfhound, and Scottish Deerhound, which are fixed for the ancestral **C** allele. Note that this mutation does not seem to affect muscling in small or even mid-sized dog breeds with notable back muscling, including the American Staffordshire Terrier, Boston Terrier, and the English Bulldog. | **Likely normal muscling (CC)** |

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## TRAITS: BODY SIZE

| TRAIT | RESULT |
| --- | --- |
| **Body Size (IGF1)**<br>The **I** allele is associated with smaller body size. | **Smaller (II)** |
| **Body Size (IGFR1)**<br>The **A** allele is associated with smaller body size. | **Intermediate (GA)** |
| **Body Size (STC2)**<br>The **A** allele is associated with smaller body size. | **Intermediate (TA)** |
| **Body Size (GHR - E191K)**<br>The **A** allele is associated with smaller body size. | **Smaller (AA)** |
| **Body Size (GHR - P177L)**<br>The **T** allele is associated with smaller body size. | **Intermediate (CT)** |

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## TRAITS: PERFORMANCE

| TRAIT | RESULT |
| --- | --- |
| **Altitude Adaptation (EPAS1)**<br>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)**<br>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 Peaches’s genetic health results:**

If Peaches 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 Peaches 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 6 results that you should learn about.

**Increased risk results** (1)

**Intervertebral Disc Disease (Type I)**

**Notable results** (5)

**ALT Activity**

**Copper Toxicosis (Accumulating)**

**Degenerative Myelopathy, DM**

**Dry Eye Curly Coat Syndrome**

**Medium-Chain Acyl-CoA Dehydrogenase Deficiency, MCADD**

**Clear results**

**Breed-relevant** (7)

**Other** (260)

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## BREED-RELEVANT RESULTS

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

| | |
|---|---|
| Intervertebral Disc Disease (Type I) (FGF4 retrogene - CFA12) | Increased risk |
| Degenerative Myelopathy, DM (SOD1A) | Notable |
| Dry Eye Curly Coat Syndrome (FAM83H Exon 5) | Notable |
| Medium-Chain Acyl-CoA Dehydrogenase Deficiency, MCADD (ACADM, Cavalier King Charles Spaniel Variant) | Notable |
| Episodic Falling Syndrome (BCAN) | Clear |
| GM2 Gangliosidosis (HEXB, Poodle Variant) | Clear |
| Muscular Dystrophy (DMD, Cavalier King Charles Spaniel Variant 1) | Clear |
| Neonatal Encephalopathy with Seizures, NEWS (ATF2) | Clear |
| Osteochondrodysplasia (SLC13A1, Poodle Variant) | Clear |
| Progressive Retinal Atrophy, prcd (PRCD Exon 1) | Clear |
| Von Willebrand Disease Type I, Type I vWD (VWF) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

Research has not yet linked these conditions to dogs with similar breeds to Peaches. 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 |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| 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 |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## 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 |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## 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 |
| 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 |
| Dystrophic Epidermolysis Bullosa (COL7A1, Central Asian Shepherd Dog Variant) | Clear |
| Dystrophic Epidermolysis Bullosa (COL7A1, Golden Retriever Variant) | Clear |
| Early Bilateral Deafness (LOXHD1 Exon 38, Rottweiler Variant) | Clear |
| Early Onset Adult Deafness, EOAD (EPS8L2 Deletion, Rhodesian Ridgeback Variant) | Clear |
| Early Onset Cerebellar Ataxia (SEL1L, Finnish Hound Variant) | Clear |
| Ehlers Danlos (ADAMTS2, Doberman Pinscher Variant) | Clear |
| Ehlers-Danlos Syndrome (EDS) (COL5A1, Labrador Retriever Variant) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| Enamel Hypoplasia (ENAM Deletion, Italian Greyhound Variant) | Clear |
| Enamel Hypoplasia (ENAM SNP, Parson Russell Terrier Variant) | 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 |
| 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 |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| GM1 Gangliosidosis (GLB1 Exon 15, Shiba Inu Variant) | Clear |
| GM1 Gangliosidosis (GLB1 Exon 15, Alaskan Husky Variant) | Clear |
| GM2 Gangliosidosis (HEXA, Japanese Chin Variant) | Clear |
| Golden Retriever Progressive Retinal Atrophy 1, GR-PRA1 (SLC4A3) | Clear |
| Golden Retriever Progressive Retinal Atrophy 2, GR-PRA2 (TTC8) | Clear |
| Goniodysgenesis and Glaucoma, Pectinate Ligament Dysplasia, PLD (OLFM3) | Clear |
| 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 |
| 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 |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| Hereditary Nasal Parakeratosis (SUV39H2 Intron 4, Greyhound Variant) | Clear |
| Hereditary Nasal Parakeratosis, HNPK (SUV39H2) | Clear |
| Hereditary Vitamin D-Resistant Rickets (VDR) | Clear |
| Hypocatalasia, Acatalasemia (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 |
| 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 |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| 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 |
| Ligneous Membranitis, LM (PLG) | Clear |
| Limb Girdle Muscular Dystrophy (SGCD, Boston Terrier Variant) | Clear |
| Limb-Girdle Muscular Dystrophy 2D (SGCA Exon 3, Miniature Dachshund Variant) | Clear |
| Long QT Syndrome (KCNQ1) | Clear |
| Lundehund Syndrome (LEPREL1) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| Macular Corneal Dystrophy, MCD (CHST6) | Clear |
| Malignant Hyperthermia (RYR1) | Clear |
| May-Hegglin Anomaly (MYH9) | Clear |
| MDR1 Drug Sensitivity (ABCB1) | Clear |
| Methemoglobinemia (CYB5R3, Pit Bull Terrier Variant) | Clear |
| Methemoglobinemia (CYB5R3) | Clear |
| Microphthalmia (RBP4 Exon 2, Soft Coated Wheaten Terrier Variant) | Clear |
| Mucopolysaccharidosis IIIB, Sanfilippo Syndrome Type B, MPS IIIB (NAGLU, Schipperke Variant) | Clear |
| Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, Dachshund Variant) | Clear |
| Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A, MPS IIIA (SGSH Exon 6, New Zealand Huntaway Variant) | Clear |
| Mucopolysaccharidosis Type VI, Maroteaux-Lamy Syndrome, MPS VI (ARSB Exon 5, Miniature Pinscher Variant) | Clear |
| Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 3, German Shepherd Variant) | Clear |
| Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 5, Terrier Brasileiro Variant) | Clear |
| Muscular Dystrophy (DMD, Golden Retriever Variant) | Clear |
| Muscular Dystrophy-Dystroglycanopathy (LARGE1, Labrador Retriever Variant) | Clear |
| Musladin-Lueke Syndrome, MLS (ADAMTSL2) | Clear |
| Myasthenia Gravis-Like Syndrome (CHRNE, Heideterrier Variant) | Clear |
| Myotonia Congenita (CLCN1 Exon 23, Australian Cattle Dog Variant) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| Myotonia Congenita (CLCN1 Exon 19, Labrador Retriever Variant) | Clear |
| Myotonia Congenita (CLCN1 Exon 7, Miniature Schnauzer Variant) | Clear |
| Narcolepsy (HCRTR2 Exon 1, Dachshund Variant) | Clear |
| Narcolepsy (HCRTR2 Intron 4, Doberman Pinscher Variant) | Clear |
| Narcolepsy (HCRTR2 Intron 6, Labrador Retriever Variant) | Clear |
| Nemaline Myopathy (NEB, American Bulldog Variant) | Clear |
| Neonatal Cerebellar Cortical Degeneration (SPTBN2, Beagle Variant) | Clear |
| Neonatal Interstitial Lung Disease (LAMP3) | Clear |
| Neuroaxonal Dystrophy, NAD (VPS11, Rottweiler Variant) | Clear |
| Neuroaxonal Dystrophy, NAD (TECPR2, Spanish Water Dog Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis 1, NCL 1 (PPT1 Exon 8, Dachshund Variant 1) | Clear |
| Neuronal Ceroid Lipofuscinosis 10, NCL 10 (CTSD Exon 5, American Bulldog Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis 2, NCL 2 (TPP1 Exon 4, Dachshund Variant 2) | Clear |
| Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 SNP, Border Collie Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 Deletion, Golden Retriever Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis 6, NCL 6 (CLN6 Exon 7, Australian Shepherd Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis 7, NCL 7 (MFSD8, Chihuahua and Chinese Crested Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8, Australian Shepherd Variant) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Exon 2, English Setter Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Insertion, Saluki Variant) | Clear |
| Neuronal Ceroid Lipofuscinosis, Cerebellar Ataxia, NCL4A (ARSG Exon 2, American Staffordshire Terrier Variant) | Clear |
| Oculocutaneous Albinism, OCA (SLC45A2 Exon 6, Bullmastiff Variant) | Clear |
| Oculocutaneous Albinism, OCA (SLC45A2, Small Breed Variant) | Clear |
| Oculoskeletal Dysplasia 2 (COL9A2, Samoyed Variant) | Clear |
| Osteogenesis Imperfecta (COL1A2, Beagle Variant) | Clear |
| Osteogenesis Imperfecta (SERPINH1, Dachshund Variant) | Clear |
| Osteogenesis Imperfecta (COL1A1, Golden Retriever Variant) | Clear |
| P2Y12 Receptor Platelet Disorder (P2Y12) | Clear |
| Pachyonychia Congenita (KRT16, Dogue de Bordeaux Variant) | Clear |
| Paroxysmal Dyskinesia, PxD (PIGN) | Clear |
| Persistent Mullerian Duct Syndrome, PMDS (AMHR2) | Clear |
| Pituitary Dwarfism (POU1F1 Intron 4, Karelian Bear Dog Variant) | Clear |
| Platelet Factor X Receptor Deficiency, Scott Syndrome (TMEM16F) | Clear |
| Polycystic Kidney Disease, PKD (PKD1) | Clear |
| Pompe's Disease (GAA, Finnish and Swedish Lapphund, Lapponian Herder Variant) | Clear |
| Prekallikrein Deficiency (KLKB1 Exon 8) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| 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 5, PRA5 (NECAP1 Exon 6, Giant Schnauzer 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, crd4/cord1 (RPGRIP1) | Clear |
| Progressive Retinal Atrophy, PRA1 (CNGB1) | Clear |
| Progressive Retinal Atrophy, PRA3 (FAM161A) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| 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 |
| Shaking Puppy Syndrome (PLP1, English Springer Spaniel Variant) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| 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 |
| Von Willebrand Disease Type II, Type II vWD (VWF, Pointer Variant) | Clear |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## OTHER RESULTS

| | |
|---|---|
| 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 |

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## HEALTH REPORT

**Increased risk result**

**Intervertebral Disc Disease (Type I)**

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

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

**What is Chondrodystrophy and Intervertebral Disc Disease, CDDY/IVDD, Type I IVDD?**
This condition is associated with differences in body proportions, such as a longer back and shorter legs, and may increase the risk of spinal disc problems. Disc disease can vary in severity, from mild discomfort to more serious movement changes.

**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.

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## HEALTH REPORT

**Notable result**

**ALT Activity**

Lindsay's Peaches inherited one copy of the variant we tested for Alanine Aminotransferase Activity

**Why is this important to your vet?**
Peaches has one copy of a variant associated with reduced ALT activity as measured on veterinary blood chemistry panels. Please inform your veterinarian that Peaches has this genotype, as ALT is often used as an indicator of liver health and Peaches is likely to have a lower than average resting ALT activity. As such, an increase in Peaches’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?**
ALT is a liver enzyme that vets measure to monitor liver health. With this result, your dog may naturally have a lower ALT baseline. Knowing this helps your veterinarian interpret future bloodwork results more accurately.

**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.

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## HEALTH REPORT

**Notable result**

**Copper Toxicosis (Accumulating)**

Lindsay's Peaches inherited one copy of the variant we tested for Copper Toxicosis (Accumulating)
Peaches 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 Peaches will develop Copper Toxicosis (Accumulating).

**Scientific Basis**
Research studies for this variant have been based on dogs of other breeds. Not enough dogs with Peaches's breed have been studied to know whether or not this variant will increase Peaches'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)?**
This condition affects the liver’s ability to remove excess copper. Over time, copper can build up in the liver and damage liver cells. Both genetic and environmental factors play a role in how the condition develops.

**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/).

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## HEALTH REPORT

**Notable result**

**Degenerative Myelopathy, DM**

Lindsay's Peaches inherited one copy of the variant we tested for Degenerative Myelopathy, DM

**What does this result mean?**
This variant should not impact Peaches’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. Peaches 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 Degenerative Myelopathy, DM?**
This condition affects the spinal cord nerves involved in movement, most noticeably in the hind limbs. It is progressive, meaning symptoms worsen over time, including weakness, muscle loss, and changes in walking.

**When signs & symptoms develop in affected dogs**
Affected dogs do not usually show signs of DM until they are at least 8 years old.

**How vets diagnose this condition**
Definitive diagnosis requires microscopic analysis of the spinal cord after death. However, veterinarians use clues such as genetic testing, breed, age, and other diagnostics to determine if DM is the most likely cause of your dog’s clinical signs.

**How this condition is treated**
As dogs are seniors at the time of onset, the treatment for DM is aimed towards increasing their comfort through a combination of lifestyle changes, medication, and physical therapy.

**Actions to take if your dog is affected**

- Talk to your vet about your dog’s degenerative myelopathy result, as it may influence how they monitor your dog’s mobility and overall health, especially in their senior years.
- Keep your dog active with regular, low-impact exercise to help them maintain a healthy weight and support their mobility.
- Watch for changes in movement, such as wobbling, reluctance to jump, or dragging their back paws, and consult your vet if you notice any of these signs.
- Provide good traction in your home with rugs or mats to help prevent slipping as your dog ages. If mobility becomes difficult, ask your vet about supportive devices such as harnesses or wheelchairs.

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## HEALTH REPORT

**Notable result**

**Dry Eye Curly Coat Syndrome**

Lindsay's Peaches inherited one copy of the variant we tested for Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatosis, Dry Eye Curly Coat Syndrome, CKCSID

**What does this result mean?**
This variant should not impact Peaches’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. Peaches 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 Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatosis, Dry Eye Curly Coat Syndrome, CKCSID?**
This condition affects the development of the eyes, skin, hair, and nails. Affected dogs may have dry eyes, corneal ulcers, itchy skin, and thick or cracked paw pads.

**When signs & symptoms develop in affected dogs**
Affected dogs are often first noted at birth, although signs continue to develop into adulthood.

**How vets diagnose this condition**
Genetic and laboratory testing are used to diagnosis this disorder.

**How this condition is treated**
The symptoms of CKCSICD can be managed with artificial tears and antibiotics as needed for infected corneal ulcers, soothing baths for itchy skin, and conditioning creams for thick paw pads. Regular dental check ups are recommended for all adult dogs.

**Actions to take if your dog is affected**

- Talk to your vet about your dog’s CKCSID result so you can work together to plan ongoing care and monitoring.
- Follow your vet’s treatment plan carefully, including any prescribed eye medications or skin care routines.
- Gently clean your dog’s eyes and skin with vet-approved wipes or a soft, damp cloth to help remove buildup and keep them comfortable.
- Use a humidifier or keep your home environment from getting too dry, which can help ease eye irritation and support skin moisture.

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## HEALTH REPORT

**Notable result**

**Medium-Chain Acyl-CoA Dehydrogenase Deficiency, MCADD**

Lindsay's Peaches inherited one copy of the variant we tested for Medium-Chain Acyl-CoA Dehydrogenase Deficiency, MCADD

**What does this result mean?**
This variant should not impact Peaches’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. Peaches 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 Medium-Chain Acyl-CoA Dehydrogenase Deficiency, MCADD?**
This condition affects how the body uses certain fats for energy. When energy demands are high, affected dogs may develop low energy levels and, in some cases, seizures.

**When signs & symptoms develop in affected dogs**
The age of diagnosis can vary, but signs typically first appear in young dogs.

**How vets diagnose this condition**
A combination of physical examination, blood tests, and genetic testing can be used to diagnose MCADD.

**How this condition is treated**
Treatment consists of frequent feeding of low-fat diets. Anti-convulsant therapy may be indicated in some cases.

**Actions to take if your dog is affected**

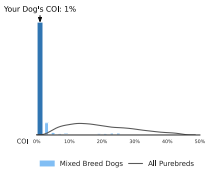
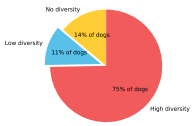
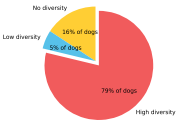
- Talk to your vet about your dog’s MCADD result so you can discuss a feeding schedule and diet that help prevent low blood sugar.
- Provide frequent, regular meals and avoid prolonged fasting, as affected dogs have trouble using fat for energy between meals.
- Keep a small, easily digestible snack on hand for times when meals may be delayed or if your dog seems weak or lethargic.
- If your dog shows signs of low energy, wobbliness, or collapse, offer a small meal or a bit of a sugar source like honey on the gums and contact your vet.

# “PEACHES”

## LINDSAY'S PEACHES

DNA Test Report | Test Date: August 12th, 2025 | embk.me/peaches1653

## INBREEDING AND DIVERSITY

| CATEGORY | RESULT |
| --- | --- |
| **Coefficient Of Inbreeding**<br>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. | **1%**<br>  |
| **MHC Class II - DLA DRB1**<br>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**<br>How common is this amount of diversity in mixed breed dogs:<br>  |
| **MHC Class II - DLA DQA1 and DQB1**<br>DQA1 and DQB1 are two tightly linked DLA genes that code for MHC proteins involved in the immune response. A number of studies have shown correlations of DQA-DQB1 haplotypes and certain autoimmune diseases; however, these have not yet been scientifically validated. | **High Diversity**<br>How common is this amount of diversity in mixed breed dogs:<br>  |