



"KAÏN"

Q'STEEL DE SOL CASA



DNA Test Report

Test Date: July 25th, 2024

embk.me/qsteeldesolcasa

BREED ANCESTRY

■ Belgian Malinois : 100.0%

GENETIC STATS

Predicted adult weight: **65 lbs**

Life stage: **Mature adult**

Based on your dog's date of birth provided.

TEST DETAILS

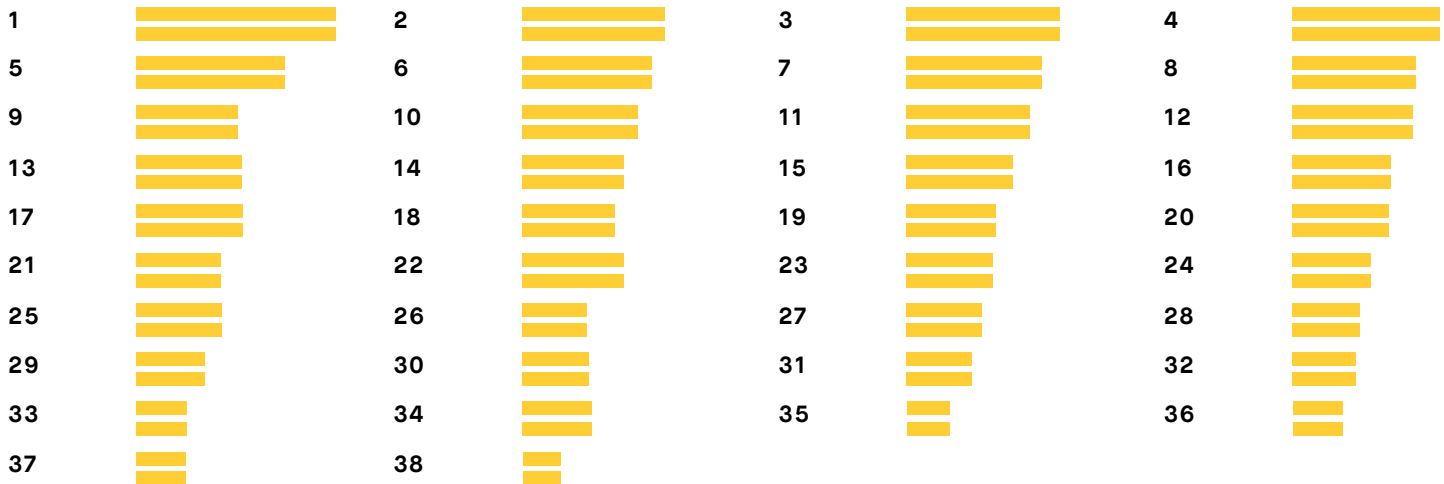
Kit number: EM-41336210

Swab number: 31220910912011

BREED ANCESTRY BY CHROMOSOME

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

Breed colors:
Belgian Malinois





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BELGIAN MALINOIS



The Belgian Malinois is the ultimate working dog. They were developed during the late 1800's in Belgium with the intent of creating a highly intelligent dog with a very strong work ethic. The breed has made a statement through its work in the police force and the military. Malinois were Belgium's first police dogs. American servicemen brought these dogs back to the United States after World War I with the desire to incorporate them into the military. This is a very smart breed that requires a strong owner in order to tackle their intelligence. Malinois have boundless energy, but can thrive anywhere so long as they are thoroughly exercised on a daily basis. Their double coat, unfortunately, can result in quite a bit of shedding. These are people oriented dogs that want to be included in all family happenings.

Fun Fact

The Belgian Malinois gets its name from the Belgian city of Maline, from where the breed originated.

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MATERNAL LINE



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

HAPLOGROUP: B1

B1 is the second most common maternal lineage in breeds of European or American origin. It is the female line of the majority of Golden Retrievers, Basset Hounds, and Shih Tzus, and about half of Beagles, Pekingese and Toy Poodles. This lineage is also somewhat common among village dogs that carry distinct ancestry from these breeds. We know this is a result of B1 dogs being common amongst the European dogs that their conquering owners brought around the world, because nowhere on earth is it a very common lineage in village dogs. It even enables us to trace the path of (human) colonization: Because most Bichons are B1 and Bichons are popular in Spanish culture, B1 is now fairly common among village dogs in Latin America.

HAPLOTYPE: B57

Part of the large B1 haplogroup, we have detected this haplotype in Belgian Tervurens, Belgian Malinois, Schipperkes, and village dogs in the Democratic Republic of the Congo.



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PATERNAL LINE



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

HAPLOGROUP: A1a

Some of the wolves that became the original dogs in Central Asia around 15,000 years ago came from this long and distinguished line of male dogs. After domestication, they followed their humans from Asia to Europe and then didn't stop there. They took root in Europe, eventually becoming the dogs that founded the Vizsla breed 1,000 years ago. The Vizsla is a Central European hunting dog, and all male Vizslas descend from this line. During the Age of Exploration, like their owners, these pooches went by the philosophy, "Have sail, will travel!" From the windy plains of Patagonia to the snug and homey towns of the American Midwest, the beaches of a Pacific paradise, and the broad expanse of the Australian outback, these dogs followed their masters to the outposts of empires. Whether through good fortune or superior genetics, dogs from the A1a lineage traveled the globe and took root across the world. Now you find village dogs from this line frolicking on Polynesian beaches, hanging out in villages across the

HAPLOTYPE: H1a.56

Part of the A1a haplogroup, the H1a.56 haplotype occurs most commonly in Belgian Malinois. It's a rare find!

Registration: Fédération Cynologique
Internationale (FCI)





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

Can have a melanistic mask (E^mE^m)

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.

Fawn Sable coat color pattern (a^Ya^Y)

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
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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 **ll** genotype at this locus are more likely to be mostly black with tan points on the eyebrows, muzzle, and legs as commonly seen in the Doberman Pinscher and the Rottweiler. This gene modifies the A Locus **a^t** allele, so dogs that do not express **a^t** are not influenced by this gene.

Not expressed (NN)

S Locus (MITF)

The S Locus determines white spotting and pigment distribution. MITF controls where pigment is produced, and an insertion in the MITF gene causes a loss of pigment in the coat and skin, resulting in white hair and/or pink skin. Dogs with two copies of this variant will likely have breed-dependent white patterning, with a nearly white, parti, or piebald coat. Dogs with one copy of this variant will have more limited white spotting and may be considered flash, parti or piebald. This MITF variant does not explain all white spotting patterns in dogs and other variants are currently being researched. Some dogs may have small amounts of white on the paws, chest, face, or tail regardless of their S Locus genotype.

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

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TRAITS: COAT COLOR (CONTINUED)

TRAIT **RESULT**

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
<p data-bbox="86 516 329 548">Panda White Spotting</p> <p data-bbox="86 583 1179 688">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.</p> <p data-bbox="86 724 1218 829">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.</p>	<p data-bbox="1276 625 1515 730">Not expected to display Panda pattern (NN)</p>



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TRAITS: OTHER COAT TRAITS

TRAIT	RESULT
Furnishings (RSPO2)	
<p>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.</p>	Likely unfurnished (no mustache, beard, and/or eyebrows) (II)

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TRAITS: OTHER COAT TRAITS (CONTINUED)

TRAIT	RESULT
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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 short or mid-length coat (ShLh)

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 heavy/seasonal shedding (CT)

Coat Texture (KRT71)

Dogs with a long coat and at least one copy of the **T** allele have a wavy or curly coat characteristic of Poodles and Bichon Frises. Dogs with two copies of the ancestral **C** allele are likely to have a straight coat, but there are other factors that can cause a curly coat, for example if they at least one **F** allele for the Furnishings (RSPO2) gene then they are likely to have a curly coat. Dogs with short coats may carry one or two copies of the **T** allele but still have straight coats.

Likely 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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TRAITS: OTHER COAT TRAITS (CONTINUED)

TRAIT	RESULT
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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.

Not indicative of chondrodysplasia (normal leg length) (NN)

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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TRAITS: BODY SIZE

TRAIT	RESULT
Body Size (IGF1) The I allele is associated with smaller body size.	Larger (NN)
Body Size (IGFR1) The A allele is associated with smaller body size.	Larger (GG)
Body Size (STC2) The A allele is associated with smaller body size.	Larger (TT)
Body Size (GHR - E191K) The A allele is associated with smaller body size.	Larger (GG)
Body Size (GHR - P177L) The T allele is associated with smaller body size.	Larger (CC)

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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 Kaïn's genetic health results:

If Kaïn 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 Kaïn 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 3 results that you should learn about.

Notable results (3)

ALT Activity

Copper Toxicosis (Accumulating)

Urate Kidney & Bladder Stones

Clear results

Breed-relevant (5)

Other (265)



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BREED-RELEVANT RESULTS

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

<input checked="" type="checkbox"/> Cardiomyopathy and Juvenile Mortality (YARS2)	Clear
<input checked="" type="checkbox"/> Hereditary Cerebellar Ataxia (SELENOP, Belgian Shepherd Variant)	Clear
<input checked="" type="checkbox"/> Mucopolysaccharidosis Type VII, Sly Syndrome, MPS VII (GUSB Exon 3, German Shepherd Variant)	Clear
<input checked="" type="checkbox"/> Spongy Degeneration with Cerebellar Ataxia 1 (KCNJ10)	Clear
<input checked="" type="checkbox"/> Spongy Degeneration with Cerebellar Ataxia 2 (ATP1B2)	Clear

Registration: Fédération Cynologique Internationale
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"KAÏN"

Q'STEEL DE SOL CASA



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OTHER RESULTS

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

⊖ ALT Activity (GPT)	Notable
⊖ Copper Toxicosis (Accumulating) (ATP7B)	Notable
⊖ Urate Kidney & Bladder Stones (SLC2A9)	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



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✔ Canine Multifocal Retinopathy, cmr2 (BEST1 Exon 5, Coton de Tulear Variant)	Clear
✔ 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
✔ Centronuclear Myopathy, CNM (PTPLA)	Clear
✔ Cerebellar Hypoplasia (VLDLR, Eurasier Variant)	Clear
✔ Chondrodystrophy (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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<input checked="" type="checkbox"/> Ehlers Danlos (ADAMTS2, Doberman Pinscher Variant)	Clear
<input checked="" type="checkbox"/> Ehlers-Danlos Syndrome (EDS) (COL5A1, Labrador Retriever Variant)	Clear
<input checked="" type="checkbox"/> Enamel Hypoplasia (ENAM Deletion, Italian Greyhound Variant)	Clear
<input checked="" type="checkbox"/> Enamel Hypoplasia (ENAM SNP, Parson Russell Terrier Variant)	Clear
<input checked="" type="checkbox"/> Episodic Falling Syndrome (BCAN)	Clear
<input checked="" type="checkbox"/> Exercise-Induced Collapse, EIC (DNM1)	Clear
<input checked="" type="checkbox"/> Factor VII Deficiency (F7 Exon 5)	Clear
<input checked="" type="checkbox"/> Factor XI Deficiency (F11 Exon 7, Kerry Blue Terrier Variant)	Clear
<input checked="" type="checkbox"/> Familial Nephropathy (COL4A4 Exon 3, Cocker Spaniel Variant)	Clear
<input checked="" type="checkbox"/> Familial Nephropathy (COL4A4 Exon 30, English Springer Spaniel Variant)	Clear
<input checked="" type="checkbox"/> Fanconi Syndrome (FAN1, Basenji Variant)	Clear
<input checked="" type="checkbox"/> Fetal-Onset Neonatal Neuroaxonal Dystrophy (MFN2, Giant Schnauzer Variant)	Clear
<input checked="" type="checkbox"/> Glanzmann's Thrombasthenia Type I (ITGA2B Exon 13, Great Pyrenees Variant)	Clear
<input checked="" type="checkbox"/> Glanzmann's Thrombasthenia Type I (ITGA2B Exon 12, Otterhound Variant)	Clear
<input checked="" type="checkbox"/> Globoid Cell Leukodystrophy, Krabbe disease (GALC Exon 5, Terrier Variant)	Clear
<input checked="" type="checkbox"/> Glycogen Storage Disease Type IA, Von Gierke Disease, GSD IA (G6PC1, German Pinscher Variant)	Clear
<input checked="" type="checkbox"/> Glycogen Storage Disease Type IA, Von Gierke Disease, GSD IA (G6PC, Maltese Variant)	Clear
<input checked="" type="checkbox"/> Glycogen Storage Disease Type IIIA, GSD IIIA (AGL, Curly Coated Retriever Variant)	Clear

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OTHER RESULTS

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

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OTHER RESULTS

<input checked="" type="checkbox"/> Hereditary Cataracts (FYCO1, Wirehaired Pointing Griffon Variant)	Clear
<input checked="" type="checkbox"/> Hereditary Footpad Hyperkeratosis (FAM83G, Terrier and Kromfohrlander Variant)	Clear
<input checked="" type="checkbox"/> Hereditary Footpad Hyperkeratosis (DSG1, Rottweiler Variant)	Clear
<input checked="" type="checkbox"/> Hereditary Nasal Parakeratosis (SUV39H2 Intron 4, Greyhound Variant)	Clear
<input checked="" type="checkbox"/> Hereditary Nasal Parakeratosis, HNPk (SUV39H2)	Clear
<input checked="" type="checkbox"/> Hereditary Vitamin D-Resistant Rickets (VDR)	Clear
<input checked="" type="checkbox"/> Hypocatalasia, Acatlasemia (CAT)	Clear
<input checked="" type="checkbox"/> Hypomyelination and Tremors (FNIP2, Weimaraner Variant)	Clear
<input checked="" type="checkbox"/> Hypophosphatasia (ALPL Exon 9, Karelian Bear Dog Variant)	Clear
<input checked="" type="checkbox"/> Ichthyosis (NIPAL4, American Bulldog Variant)	Clear
<input checked="" type="checkbox"/> Ichthyosis (ASPRV1 Exon 2, German Shepherd Variant)	Clear
<input checked="" type="checkbox"/> Ichthyosis (SLC27A4, Great Dane Variant)	Clear
<input checked="" type="checkbox"/> Ichthyosis, Epidermolytic Hyperkeratosis (KRT10, Terrier Variant)	Clear
<input checked="" type="checkbox"/> Ichthyosis, ICH1 (PNPLA1, Golden Retriever Variant)	Clear
<input checked="" type="checkbox"/> Ichthyosis, ICH2 (ABHD5, Golden Retriever Variant)	Clear
<input checked="" type="checkbox"/> Inflammatory Myopathy (SLC25A12)	Clear
<input checked="" type="checkbox"/> Inherited Myopathy of Great Danes (BIN1)	Clear
<input checked="" type="checkbox"/> Inherited Selected Cobalamin Malabsorption with Proteinuria (CUBN, Komondor Variant)	Clear

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OTHER RESULTS

✓ Intervertebral Disc Disease (Type I) (FGF4 retrogene - CFA12)	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 (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

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OTHER RESULTS

✓ 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
✓ Macular Corneal Dystrophy, MCD (CHST6)	Clear
✓ Malignant Hyperthermia (RYR1)	Clear
✓ May-Hegglin Anomaly (MYH9)	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, 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 5, Terrier Brasileiro Variant)	Clear
✓ Multiple Drug Sensitivity (ABCB1)	Clear
✓ Muscular Dystrophy (DMD, Cavalier King Charles Spaniel Variant 1)	Clear



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OTHER RESULTS

 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
 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 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 1, NCL 1 (PPT1 Exon 8, Dachshund Variant 1)	Clear
 Neuronal Ceroid Lipofuscinosis 10, NCL 10 (CTSD Exon 5, American Bulldog Variant)	Clear

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✔ Neuronal Ceroid Lipofuscinosis 2, NCL 2 (TPP1 Exon 4, Dachshund Variant 2)	Clear
✔ Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 SNP, Border Collie Variant)	Clear
✔ Neuronal Ceroid Lipofuscinosis 5, NCL 5 (CLN5 Exon 4 Deletion, Golden Retriever Variant)	Clear
✔ Neuronal Ceroid Lipofuscinosis 6, NCL 6 (CLN6 Exon 7, Australian Shepherd Variant)	Clear
✔ Neuronal Ceroid Lipofuscinosis 7, NCL 7 (MFSD8, Chihuahua and Chinese Crested Variant)	Clear
✔ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8, Australian Shepherd Variant)	Clear
✔ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Exon 2, English Setter Variant)	Clear
✔ Neuronal Ceroid Lipofuscinosis 8, NCL 8 (CLN8 Insertion, Saluki Variant)	Clear
✔ Neuronal Ceroid Lipofuscinosis, Cerebellar Ataxia, NCL4A (ARSG Exon 2, American Staffordshire Terrier Variant)	Clear
✔ Oculocutaneous Albinism, OCA (SLC45A2 Exon 6, Bullmastiff Variant)	Clear
✔ Oculocutaneous Albinism, OCA (SLC45A2, Small Breed Variant)	Clear
✔ Oculoskeletal Dysplasia 2 (COL9A2, Samoyed Variant)	Clear
✔ Osteochondrodysplasia (SLC13A1, Poodle 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

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✔ 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
✔ 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

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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
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

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OTHER RESULTS

✓ 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
✓ 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
✓ 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

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OTHER RESULTS

<input checked="" type="checkbox"/> Trapped Neutrophil Syndrome, TNS (VPS13B)	Clear
<input checked="" type="checkbox"/> Ullrich-like Congenital Muscular Dystrophy (COL6A3 Exon 10, Labrador Retriever Variant)	Clear
<input checked="" type="checkbox"/> Ullrich-like Congenital Muscular Dystrophy (COL6A1 Exon 3, Landseer Variant)	Clear
<input checked="" type="checkbox"/> Unilateral Deafness and Vestibular Syndrome (PTPRQ Exon 39, Doberman Pinscher)	Clear
<input checked="" type="checkbox"/> Von Willebrand Disease Type I, Type I vWD (VWF)	Clear
<input checked="" type="checkbox"/> Von Willebrand Disease Type II, Type II vWD (VWF, Pointer Variant)	Clear
<input checked="" type="checkbox"/> Von Willebrand Disease Type III, Type III vWD (VWF Exon 4, Terrier Variant)	Clear
<input checked="" type="checkbox"/> Von Willebrand Disease Type III, Type III vWD (VWF Intron 16, Nederlandse Kooikerhondje Variant)	Clear
<input checked="" type="checkbox"/> Von Willebrand Disease Type III, Type III vWD (VWF Exon 7, Shetland Sheepdog Variant)	Clear
<input checked="" type="checkbox"/> X-Linked Hereditary Nephropathy, XLHN (COL4A5 Exon 35, Samoyed Variant 2)	Clear
<input checked="" type="checkbox"/> X-Linked Myotubular Myopathy (MTM1, Labrador Retriever Variant)	Clear
<input checked="" type="checkbox"/> X-Linked Progressive Retinal Atrophy 1, XL-PRA1 (RPGR)	Clear
<input checked="" type="checkbox"/> X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG Exon 1, Basset Hound Variant)	Clear
<input checked="" type="checkbox"/> X-linked Severe Combined Immunodeficiency, X-SCID (IL2RG, Corgi Variant)	Clear
<input checked="" type="checkbox"/> Xanthine Urolithiasis (XDH, Mixed Breed Variant)	Clear
<input checked="" type="checkbox"/> β -Mannosidosis (MANBA Exon 16, Mixed-Breed Variant)	Clear
Mast Cell Tumor	No result

Registration: Fédération Cynologique Internationale
(FCI)





"KAÏN"

Q'STEEL DE SOL CASA



DNA Test Report

Test Date: July 25th, 2024

embk.me/qsteeldesolcasa

HEALTH REPORT

Notable result

ALT Activity

Q'Steel de Sol Casa inherited one copy of the variant we tested for Alanine Aminotransferase Activity

Why is this important to your vet?

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



"KAÏN"

Q'STEEL DE SOL CASA



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HEALTH REPORT

⊖ Notable result

Copper Toxicosis (Accumulating)

Q'Steel de Sol Casa inherited one copy of the variant we tested for Copper Toxicosis (Accumulating)
Kaïn 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 Kaïn will develop Copper Toxicosis (Accumulating).

Scientific Basis

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

- Please consult your veterinarian for dietary advice and follow their recommendations for monitoring.
- 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/>)



“KAÏN”

Q'STEEL DE SOL CASA



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HEALTH REPORT

⊖ Notable result

Urate Kidney & Bladder Stones

Q'Steel de Sol Casa inherited one copy of the variant we tested for Hyperuricosuria and Hyperuricemia or Urolithiasis, HUU

What does this result mean?

This variant should not impact Kaïn'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. Kaïn is unlikely to develop this condition due to this variant because he only has one copy of the variant.

Impact on Breeding

Your dog carries this variant and will pass it on to ~50% of his 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 Hyperuricosuria and Hyperuricemia or Urolithiasis, HUU?

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

When signs & symptoms develop in affected dogs

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

How vets diagnose this condition

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

How this condition is treated

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

Actions to take if your dog is affected

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



"KAÏN"

Q'STEEL DE SOL CASA



DNA Test Report

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INBREEDING AND DIVERSITY

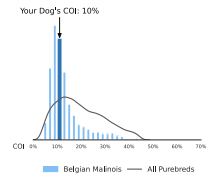
CATEGORY

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.

10%

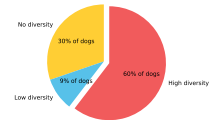


MHC Class II - DLA DRB1

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

High Diversity

How common is this amount of diversity in 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.

High Diversity

How common is this amount of diversity in purebreds:

