# American Border Collie Association, Inc.

## Certificate of Registration

ABC No: **563758**

Name PARIS | Sex Female | Date of Birth 8/6/2025 | Tran. No. | Litter Females 3 | Litter Males 4

Owner JAMIE GARDNER
BOX 1501
SHAUNAVON, SK
S0N 2M0

Breeder: TROY MILLER
30001 PET LN
PARMA, ID
83660

Color and Markings: BLACK W/WHITE, FRECKLES; Smooth ; Medium

Sire IMP.GELLI YOGI ABC 537293
Owner: BARRY BREEMERSCH
DELORAINE, MB
Breeder: D.M. HOWELLS
GLAM WALES,

PRESELI JOCK ISDS 346215
L. HARRIES
PEMBROKESHIRE WALES,
L. HARRIES
PEMBROKESHIRE WALES,

ROSE ISDS 356501
D.M. HOWELLS
GLAM WALES,
L.M. HOWELLS
W GLAMORGAN WALES,

Dam FLEA ABC 544745
Owner: TROY MILLER
PARMA, ID
Breeder: TANYA W. GIFFORD
HARRISBURG, NE

IMP. RANGER ABC 537292
BARRY BREEMERSCH
DELORAINE, MB
I.E. PEARCE MORGAN
SWANSEA WALES,

CNM WYN ABC 502982
NANCY A. PENLEY
LOVELAND, CO
TREY HUNTER
GOODLETTSVILLE, TN

##JOCK ISDS 327212
R. J. HUTCHINSON
LANCASHIRE ENG,

PRESELI FLY ISDS 331682
L. HARRIES
PEMBROKESHIRE, WALES,

DICK ISDS 345986
D.M. HOWELLS
GLAM WALES,

TYTALWYN LYN ISDS 311960
L.M. HOWELLS
W GLAMORGAN WALES,

BOSS ISDS 343213
L.M. HOWELLS
W GLAMORGAN WALES,

FLOSS ISDS 372106
I.E. PEARCE MORGAN
SWANSEA WALES,

KEN ISDS 313465
K. DAVIES
GWYNEDD WALES,

IMP.DENWYN TESS ABC 502286
TREY HUNTER
GOODLETTSVILLE, TN

#SWEEP ISDS 293085
R. J. HUTCHINSON, LANCASHIRE EN
##MO ISDS 295102
I.M. BROWNLIE, DUNBAR SCOT,
ROY ISDS 309624
R. GAMES, MERTHYR TYDFIL WALI
GROESFAEN JET ISDS 314581
L. HARRIES, PEMBROKESHIRE, WAI
NIP ISDS 325496
D.M. HOWELLS, GLAM WALES,
DEWI GWEN ISDS 335471
A. M. DRISCOLL, CARM DYFED WAI
TWM ISDS 256553
D.R. THOMAS, MID GLAMORGAN W
CAMMEN FLY ISDS 296247
D.R. THOMAS, MID GLAMORGAN WA
RICK ISDS 329735
D.F. CONNICK, POWYS WALES,
MEG ISDS 328325
D.F. CONNICK, POWYS WALES,
NIP ISDS 325496
D.M. HOWELLS, GLAM WALES,
FLOSS ISDS 358199
R. WATKINS, POWYS UK,
RHAIADR BILL ISDS 266251
K.W. OWEN, MERIONETH WALES,
JET ISDS 263006
K.W. OWEN, MERIONETH WALES,
##LLANGWM CAP ISDS 315270
A. OWEN, CLWYD WALES,
DENWYN SEREN ISDS 320737
MRS.J. REES-DENMAN, NR HOLYWELL

**Transfer of Ownership To:**

Name: _____

Address: _____

This the _____ day of _____, _____

_____
SIGNATURE OF PREVIOUS OWNER(S)

This is to certify that the above named and described Border Collie has been registered in the Stud Book of the American Border Collie Association. This c
on a written application submitted and attested by the owner at time of whelping and upon the express condition that the Association has the right to corre
certificate for cause under its rules and regulations.
@ Hips-Veterinary Radiologist Approved ## G.B. International Champion/USBCHA Finals Champion
(OFA or Canadian Equivalent) # G.B. National Champion ¢ CEA Normal

Note: If this dog is named a conformation champion by any registry after January 1, 2004, its ABCA registration will be rescinded and its offspring will no
with the American Border Collie Association

This the 6 day of December, 2025
AMERICAN BORDER COLLIE ASSSOCIATION

Signature Deborah N Bailey

ABCA, PO Box 535, Pine Mountain, GA 31822
abca@americanbordercollie.org (706) 663-5999

# DNA Profile Certificate:
# ISAG 2020 Canine SNP Profile

# Paris

| Call name | Date of birth | Kennel Club or Registry | Breed |
|---|---|---|---|
| Paris | 2025-08-05 | American Border Collie Association | Border Collie |

| Genetic sex | Sample ID |
|---|---|
| Female | DMPJDYH |


Rebecca Foran, PhD
**Head of R&D**

The genetic testing conducted by Wisdom Panel™ was performed on a sample represented by the submitter as the dog/cat listed on this certificate. The results presented in this report are applicable solely to the items tested using the sample provided. These tests have been developed, and their accuracy and precision have been established and verified by Wisdom Panel, with a sensitivity and specificity exceeding 99.9%. It's important to note that this test is not intended for breed identification purposes. Due to the DNA-based nature of this method, rare genomic variations may occur, potentially leading to false results.

Should you believe that the results provided are in error, please promptly contact breeder@wisdompanel.com for further evaluation. In the event of a valid dispute concerning the results, Wisdom Panel will do its best to resolve such a claim to the customer's satisfaction. If, following an investigation conducted by Wisdom Panel in cooperation with the customer, no resolution can be reached, the customer's sole remedy will be a refund of the testing fee. Wisdom Panel shall not be held liable for any indirect, consequential, or incidental damages of any nature. Any claims must be submitted within 60 days of the report of the test results.

# Paris

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Border Collie
**Birth date:** 2025-08-05
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

## Paris's Profile

### Pet information

**Registered name**
Paris

**Sex**
F

**Owner reported breed**
Border Collie

**Date of birth**
2025-08-05

### Genetic Diversity

**Paris's Percentage of Heterozygosity**
36%

### Health summary

At Risk 0 conditions

Carrier 2 conditions

- Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)
- Trapped Neutrophil Syndrome

Clear 270 conditions

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## Genetic Diversity

### Heterozygosity

#### Paris's Percentage of Heterozygosity

36%

Paris's genome analysis shows an average level of genetic heterozygosity when compared with other Border Collies.

#### Typical Range for Border Collies

32% - 39%

# Paris

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**ID kit:** DMPJDYH

## Health conditions known in the breed

### Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| CUBN | Deletion | 1 | AR | Carrier |

#### Information about the genetic condition

Initial signs of intestinal cobalamin malabsorption can be seen in puppies 6 to 12 weeks of age, when cobalamin store become depleted. Puppies with IGS suffer from weakness and loss of appetite and fail to grow normally Bloodwork shows anemia, neutropenia, and low cobalamin concentrations. High levels of homocysteine and methylmalonic acid can also be observed in the blood. Proteinuria is typically present.

#### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the ICM mutation can be safely bred with a clear dog with no copies of the ICM mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the ICM mutation. A dog with two copies of the ICM mutation can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the ICM mutation could develop due to a different genetic or clinical cause.

### Trapped Neutrophil Syndrome

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| VPS13B | Deletion | 1 | AR | Carrier |

#### Information about the genetic condition

Clinical signs of TNS include an exceptional susceptibility to infections secondary to the low number of circulating neutrophils in the blood stream. Affected dogs also tend to suffer from chronic inflammatory conditions such as arthritis. Clinical signs are usually observed by 6 to 12 weeks of age and can include a smaller overall size as well as a ferret-like face due to abnormal craniofacial development leading to a narrowed, elongated skull shape. For some affected dogs, clinical signs can be mild and go unnoticed until adulthood. Nevertheless, TNS is a severe disease and affected dogs have a shorter life expectancy.

#### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the TNS mutation can be safely bred with a clear dog with no copies of the TNS mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the TNS mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the TNS mutation could develop due to a different genetic or clinical cause.

**Breed:** Border Collie
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# Health conditions known in the breed

## Collie Eye Anomaly (CEA)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| NHEJ1 | Deletion | 0 | AR | **Clear** |

### Information about the genetic condition

Collie Eye Anomaly is primarily characterized by choroidal hypoplasia, leading to an underdeveloped vascular supply to the retina, and is especially visible temporal to the optic nerve. CEA lesions may be present in both eyes or asymmetric in nature. CEA-associated choroidal hypoplasia is non-progressive and usually does not cause visual deficits on its own. However, CEA has a range of clinical expressions. Vision impairment is more likely in dogs with the “extended CEA phenotype,” which may include optic nerve head colobomas, retinal detachment or intraocular hemorrhage secondary to coloboma(s) in severely affected dogs. Optic nerve head colobomas appear as excavations of the optic disc surface. Diagnosis of CEA lesions should be completed before 10 weeks of age, as retinal pigmentation can mask choroidal hypoplasia as the puppies grow, a phenomenon termed “go normal” by breeders. Research is ongoing to determine what additional genetic factors may be present that influence the range of severity seen in dogs with CEA.

### Breeder recommendation

This disorder is autosomal recessive, meaning two copies of the variant are needed for a dog to be at an elevated risk for being diagnosed with the condition. A carrier dog with one copy of the Collie Eye Anomaly variant can be safely bred with a clear dog with no copies of the Collie Eye Anomaly variant. About half of the puppies will have one copy (carriers) and half will have no copies of the variant. Furthermore, a dog with two copies of the CEA variant can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: Recent research has suggested that additional genetic risk factors likely exist in some breeds that resemble or contribute to CEA risk, especially the more severe disorder expression. It is possible that disorder signs similar to the ones associated with this CEA variant could develop due to a different genetic or clinical cause.

## Dental Hypomineralization

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| FAM20C | C>T | 0 | AR | **Clear** |

### Information about the genetic condition

Clinical signs include brownish dental discoloration and abnormal wear of teeth. As the teeth wear, the biting surfaces of the teeth darkens, become dark brown in color; the enamel layer may also show a light brown discoloration and appear dull. The disorder causes severe tooth wear leading to pulp exposure, chronic inflammation of the pulp, and pulpal necrosis. Histologically, dentin of affected dogs has an abnormal structure and the enamel can be slightly hypoplastic.

### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to be shown. A carrier dog with one copy of the Dental Hypomineralization mutation can be safely bred with a clear dog with no copies of the Dental Hypomineralization mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Dental Hypomineralization mutation. A dog with two copies of the Dental Hypomineralization mutation can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the Dental Hypomineralization mutation could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

| **Early Adult Onset Deafness For Border Collies only (Linkage test)** | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | Intergenic | Insertion | 0 | AR | **Clear** |

### Information about the genetic condition

Gradual hearing loss affecting both ears is observed usually between the ages of 5 to 7 years. Please note that this test is specifically for the Border Collie breed and is a predictive linkage test rather than a test for the true causal variant. Not all dogs with two copies of the linked marker will go on to show signs of hearing loss.

### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to develop. A carrier dog with one copy of the Deafness mutation can be safely bred with a clear dog with no copies of the Deafness mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Deafness mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. The carrier rate of the risk variant is up to 35% in the Border Collie population, highlighting the importance of keeping healthy carriers in the breeding program by breeding them to dogs tested “Clear” (zero copies) of the risk variant. Please note: It is possible that disease signs similar to the ones caused by the Deafness mutation could develop due to a different genetic or clinical cause.

| **Hereditary Calcium Oxalate Urolithiasis, Type 1** | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| | Confidential | - | 0 | AR | **Clear** |

### Information about the genetic condition

Hereditary Calcium Oxalate Urolithiasis, Type 1 is a disorder that is associated with increased risk of urinary calcium oxalate stone formation. Affected dogs will demonstrate clinical signs consistent with urolithiasis. This may range from being asymptomatic to hematuria (bloody urine), dysuria (painful urination), stranguria (straining to pass urine) and pollakiuria (frequent urination). Dogs with urinary stones are also more susceptible to urinary tract infections. And, due to the presence of the stones, affected dogs are at risk of urinary obstruction occurring at the renal pelvis, ureters, or urethra. Blockage of the urinary tract is a life-threatening condition that requires immediate intervention. While the average age of diagnosis is 3 years old, dogs affected by CaOx1 have the potential to develop urinary stones as puppies. And recurrent stone formation is common for affected dogs. There is evidence to suggest the clinical signs are more common in males than in females.

### Breeder recommendation

This disorder is autosomal recessive, meaning two copies of the variant are needed for a dog to be at an elevated risk for being diagnosed with the condition. A carrier dog with one copy of the Hereditary Calcium Oxalate Urolithiasis, Type 1 variant can be safely bred with a clear dog with no copies of the Hereditary Calcium Oxalate Urolithiasis, Type 1 variant. About half of the puppies will have one copy (carriers) and half will have no copies of the variant. Furthermore, a dog with two copies of the Hereditary Calcium Oxalate Urolithiasis, Type 1 variant can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disorder signs similar to the ones associated with this CaOx1 variant could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

### Hyperuricosuria

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| SLC2A9 | G>T | 0 | AR | **Clear** |

#### Information about the genetic condition

HUU predisposes affected dogs to the formation of urate stones. Clinical signs of urolithiasis include hematuria, pain while urinating, and blockage of the urinary tract. Patients with urinary stones are more susceptible to urinary tract infections. Blockage of the urinary tract is a life-threatening condition that requires immediate veterinary care. In Dalmatians, the clinical signs are more common in males than in females. As many as 34% of all male Dalmatians are diagnosed with urate stones.

#### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to occur. A carrier dog with one copy of the HUU mutation can be safely bred with a clear dog with no copies of the HUU mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the HUU mutation. A dog with two copies of the HUU mutation can be safely bred with a clear dog. The resulting puppies will all be carriers. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. In some breeds, such as the Dalmatian, the frequency of the disease mutation is very high. Carriers and dogs with two copies of the disease mutation (genetically affected dogs) should be used for breeding purposes, with the aim of gradually reducing the frequency of the mutant gene within the breed population. Where possible, matings should be avoided that would result in litters that could contain dogs with two copies of the disease mutation, such as a mating between two dogs with two copies of the HUU mutation or between a dog with one copy and a dog with two copies of the HUU mutation. Please note: It is possible that disease signs similar to the ones caused by the HUU mutation could develop due to a different genetic or clinical cause.

### MDR1 Medication Sensitivity

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| MDR1/ABCB1 | Deletion | 0 | AD | **Clear** |

#### Information about the genetic condition

Dogs with this variant are asymptomatic until exposed to a medication that uses the drug transport pump rendered defective by the mutation in the MDR1 (also called ABCB1) gene. Medications known to use this P-glycoprotein pump are macrocyclic lactones (antiparasitic drugs), loperamide (antidiarrheal), erythromycin (antibiotic), acepromazine (tranquilizer), butorphanol (opioid), certain drugs used in cancer treatment (vincristine, vinblastine, and doxorubicin), and others. When these medications are administered, they accumulate in the brain which results in adverse reactions. Typical symptoms include tremors, loss of balance, seizures, obtundation, excessive salivation, dilated pupils, and bradycardia. If untreated, the condition may lead to respiratory arrest, coma or death. Because dogs with 1 copy of the variant will have some P-glycoprotein function, the most severe cases tend to occur in dogs that have 2 copies of the variant and, therefore, lack any functional P-glycoprotein pumps. However, the disorder can still be very severe in dogs that have only one copy of the mutation.

#### Breeder recommendation

This disorder is autosomal dominant meaning that only one copy of the variant is needed for associated signs to occur. For some breeds where the MDR1 mutation frequency is particularly high, breeders may consider mating pairs using dogs that have one or two copies of the MDR1 variant to maintain genetic diversity within their breed. It is important that resulting puppies be tested for the MDR1 variant to ensure safe future medical treatment. If a dog with one copy of the MDR1 variant is bred with a clear dog with no copies of the MDR1 variant, about half of the puppies will have one copy and half will have no copies of the MDR1 variant. If a dog with two copies of the MDR1 variant is bred with a clear dog, the resulting puppies will all have one copy of the variant. Please note: It is possible that clinical signs similar to the ones caused by the MDR1 variant could develop due to a different genetic or clinical cause.

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## Health conditions known in the breed

### Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie)

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| CLN5 | C>T | 0 | AR | **Clear** |

#### Information about the genetic condition

Neuronal ceroid lipofuscinoses (NCLs) are a group of inherited progressive neurodegenerative lysosomal storage disorders. NCLs are characterized by excessive accumulation of lipofuscin and ceroid lipopigments in the central nervous system and other tissues. The age of onset for dogs affected with Neuronal Ceroid Lipofuscinosis 5 (NCL5) can vary significantly, with some showing initial signs at 1 to 2 years of age while others show later in life. Similarly, severity of clinical signs can vary between affected individuals. Typical signs of NCL5 include vision impairment, epileptic seizures, ataxia (uncoordinated movements), and behavioral changes, such as hyperactivity and aggression. Some affected dogs can show air biting, likely secondary to hallucinations. Due to the progressive nature of NCL5, the average prognosis is considered poor for affected dogs. And the average life expectancy is less than 2.5 years.

#### Breeder recommendation

This disorder is autosomal recessive, meaning two copies of the variant are needed for a dog to be at an elevated risk for being diagnosed with the condition. A carrier dog with one copy of the Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie) variant can be safely bred with a clear dog with no copies of the Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie) variant. About half of the puppies will have one copy (carriers) and half will have no copies of the variant. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disorder signs similar to the ones associated with this NCL5 variant could develop due to a different genetic or clinical cause.

### Sensory Neuropathy

| Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|
| FAM134B | Insertion | 0 | AR | **Clear** |

#### Information about the genetic condition

Clinical signs are detectable in puppies from two to seven months of age. Clinical signs include incoordination of gait (ataxia), knuckling of the paws, hyperextension of the limbs, and self-mutilation of the limbs. The hind legs are usually most severely affected. Loss of sensation is progressive and affects all limbs. Urinary incontinence and regurgitation can occur in the later stages of the disorder.

#### Breeder recommendation

This disease is autosomal recessive meaning that two copies of the mutation are needed for disease signs to develop. A carrier dog with one copy of the Sensory Neuropathy mutation can be safely bred with a clear dog with no copies of the Sensory Neuropathy mutation. About half of the puppies will have one copy (carriers) and half will have no copies of the Sensory Neuropathy mutation. Puppies in a litter which is expected to contain carriers should be tested prior to breeding. Carrier to carrier matings are not advised as the resulting litter may contain affected puppies. Please note: It is possible that disease signs similar to the ones caused by the Sensory Neuropathy mutation could develop due to a different genetic or clinical cause.

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# Traits

## Coat Color

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Fawn** | ASIP | $a^y$ | 0 | No effect |
| **Recessive Black** | ASIP | a | 0 | No effect |
| **Tan Points**<br>Two copies, or occasionally one copy, of this variant may result in a black and tan coat color pattern. | ASIP | $a^t$ | 2 | Tan points possible |
| **Dominant Black**<br>One or two copies of the dominant black will give a dog a black coat (depending on other variants), black eye rims, nose and pads. One copy may also give a tiger striped appearance, known as brindle patterning. | CBD103 | $K^B$ | 2 | Black possible |
| **Mask**<br>One or two copies of the Mask mutation will result in the presence of a dark facial mask covering the muzzle. This mask can cover only the very front of the muzzle, or can extend down to the chest and front legs. Mask can be hidden by other trait variants. | MC1R | $E^m$ | 1 | Dark Muzzle possible |
| **Recessive Red (e1)** | MC1R | $e^1$ | 0 | No effect |
| **Recessive Red (e2)** | MC1R | $e^2$ | 0 | No effect |
| **Recessive Red (e3)** | MC1R | $e^3$ | 0 | No effect |
| **Sable (Discovered in the Cocker Spaniel)** | MC1R | $e^H$ | 0 | No effect |
| **Widow's Peak (Discovered in Ancient dogs)** | MC1R | $e^A$ | 0 | No effect |
| **Widow's Peak (Discovered in the Afghan Hound and Saluki)** | MC1R | $e^G$ | 0 | No effect |

## Color Modification

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Cocoa (Discovered in the French Bulldog)** | HPS3 | co | 0 | No effect |
| **Red Intensity** | MFSD12 | i | 0 | No effect |

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## Color Modification

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Dilution (d1) Linkage test** | MLPH | $d^1$ | 0 | No effect |
| **Dilution (d2)** | MLPH | $d^2$ | 0 | No effect |
| **Dilution (d3)** | MLPH | $d^3$ | 0 | No effect |
| **Chocolate (basd)** | TYRP1 | $b^{asd}$ | 0 | No effect |
| **Chocolate (bc)** | TYRP1 | $b^c$ | 0 | No effect |
| **Chocolate (bd)** | TYRP1 | $b^d$ | 0 | No effect |
| **Chocolate (be)** | TYRP1 | $b^e$ | 0 | No effect |
| **Chocolate (bh)** | TYRP1 | $b^h$ | 0 | No effect |
| **Chocolate (bs)** | TYRP1 | $b^s$ | 0 | No effect |

## Coat Patterns

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Piebald** | MITF | $s^p$ | 0 | No effect |
| **Merle** | PMEL | M | 0 | No effect |
| **Harlequin** | PSMB7 | H | 0 | No effect |
| **Saddle Tan**<br>One or two copies of the Saddle Tan variant are needed for the "saddle" to be seen. However the Tan Points variant must also be present. The Saddle Tan variant is actually considered to be the wild type, or default, variant. | RALY | - | 1 | Saddle possible |
| **Roan Linkage Test**<br>To show roan patterning, a dog must inherit one or two copies of the roan variant and also express Piebald or another variant associated with white markings. Roan is only visible on the white areas of a dog's coat. | USH2A | $TR^r$ | 1 | Roan possible |

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## Coat Length and Curl

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Long Hair (lh1)**<br>To show a long coat, a dog must inherit two copies of a Long Hair variant, one from each parent. This can either be two copies of a particular variant, such as this one (lh1) or two of any combination of long hair variants. However, there are other variants suspected to influence coat length. | FGF5 | $lh^{1}$ | 1 | Short coat likely, long coat possible |
| **Long Hair (lh2)** | FGF5 | $lh^{2}$ | 0 | No effect |
| **Long Hair (lh3)** | FGF5 | $lh^{3}$ | 0 | No effect |
| **Long Hair (lh4)** | FGF5 | $lh^{4}$ | 0 | No effect |
| **Long Hair (lh5)** | FGF5 | $lh^{5}$ | 0 | No effect |
| **Curly Coat** | KRT71 | C | 0 | No effect |

## Hairlessness

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Hairlessness (Discovered in the Chinese Crested Dog) Linkage test** | FOXI3 | $Hr^{cc}$ | 0 | No effect |
| **Hairlessness (Discovered in the American Hairless Terrier)** | SGK3 | $hr^{aht}$ | 0 | No effect |
| **Hairlessness (Discovered in the Scottish Deerhound)** | SKG3 | $hr^{sd}$ | 0 | No effect |

## Shedding

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Reduced Shedding** | MC5R | sd | 0 | Seasonal shedder |

# Paris

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Border Collie
**Birth date:** 2025-08-05
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

## More Coat Traits

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Hair Ridge** | FGF3, FGF4, FGF19, ORAOV1 | R | 0 | No effect |
| **Furnishings** | RSPO2 | F | 0 | No effect |
| **Albino** | SLC45A2 | $c^{al}$ | 0 | No effect |

## Head Shape

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Short Snout (BMP3 variant)** | BMP3 | - | 0 | No effect |
| **Short Snout (SMOC2 variant)** | SMOC2 | - | 0 | No effect |

## Eye Color

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Blue Eyes (Discovered in the Siberian Husky)** | ALX4 | - | 0 | No effect |

## Ears

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Floppy Ears** | MSRB3 | - | 0 | Pricked ears more likely |

## Extra Toes

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Hind Dewclaws (Discovered in Asian breeds)** | LMBR1 | DC-1 | 0 | No effect |

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## Extra Toes

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Hind Dewclaws (Discovered in Western breeds)**<br>One or two copies of this Hind Dewclaws variant may result in your dog having hind dewclaws. Around half of the dogs with one copy of this variant will have hind dewclaws, and it is possible for the dewclaws to be just on one leg. With two copies the trait is more likely to be expressed and could be more pronounced. | LMBR1 | DC-2 | 1 | Hind dewclaws possible |

## More Body Features

| | Gene | Variant | Copies | Result |
|---|---|---|---|---|
| **Back Muscle and Bulk** | ACSL4 | - | 0 | No effect |
| **High Altitude Adaptation** | EPAS1 | - | 0 | No effect |
| **Short Legs (Chondrodysplasia, CDPA)** | FGF4 | - | 0 | No effect |
| **Short Legs (Chondrodystrophy, CDDY)** | FGF4 | - | 0 | No effect |
| **Short Tail** | T-box | T | 0 | Full tail length likely |

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## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **2,8-dihydroxyadenine (DHA) Urolithiasis** | APRT | G>A | 0 | AR | Clear |
| **Acral Mutilation Syndrome** | GDNF | C>T | 0 | AR | Clear |
| **Acute Respiratory Distress Syndrome** | ANLN | C>T | 0 | AR | Clear |
| **Alaskan Husky Encephalopathy** | SLC19A3 | G>A | 0 | AR | Clear |
| **Alexander Disease** | GFAP | G>A | 0 | AR | Clear |
| **Amelogenesis Imperfecta (Discovered in the Italian Greyhound)** | ENAM | Deletion | 0 | AR | Clear |
| **Amelogenesis Imperfecta (Discovered in the Lancashire Heeler)** | Confidential | - | 0 | AR | Clear |
| **Amelogenesis Imperfecta (Discovered in the Parson Russell Terrier)** | ENAM | C>T | 0 | AR | Clear |
| **Bandera's Neonatal Ataxia** | GRM1 | Insertion | 0 | AR | Clear |
| **Benign Familial Juvenile Epilepsy** | LGI2 | A>T | 0 | AR | Clear |
| **Bernard-Soulier Syndrome (Discovered in the Cocker Spaniel)** | GP9 | Deletion | 0 | AR | Clear |
| **Canine Congenital Stationary Night Blindness (Discovered in the Beagle)** | LRIT3 | Deletion | 0 | AR | Clear |
| **Canine Leukocyte Adhesion Deficiency (CLAD), type III** | FERMT3 | Insertion | 0 | AR | Clear |
| **Canine Multifocal Retinopathy 1** | BEST1 | C>T | 0 | AR | Clear |
| **Canine Multifocal Retinopathy 2** | BEST1 | G>A | 0 | AR | Clear |
| **Canine Multifocal Retinopathy 3** | BEST1 | Deletion | 0 | AR | Clear |
| **Canine Multiple Systems Degeneration (Discovered in the Chinese Crested Dog)** | SERAC1 | Deletion | 0 | AR | Clear |
| **Canine Scott Syndrome** | ANO6 | G>A | 0 | AR | Clear |
| **Cardiomyopathy and Juvenile Mortality (Discovered in the Belgian Shepherd)** | YARS2 | G>A | 0 | AR | Clear |
| **Centronuclear Myopathy (Discovered in the Great Dane)** | BIN1 | A>G | 0 | AR | Clear |

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## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Centronuclear Myopathy (Discovered in the Labrador Retriever)** | PTPLA | Insertion | 0 | AR | Clear |
| **Cerebellar Ataxia** | RAB24 | A>C | 0 | AR | Clear |
| **Cerebellar Cortical Degeneration** | SNX14 | C>T | 0 | AR | Clear |
| **Cerebellar Hypoplasia** | VLDLR | Deletion | 0 | AR | Clear |
| **Cerebral Dysfunction** | SLC6A3 | G>A | 0 | AR | Clear |
| **Chondrodysplasia (Discovered in Norwegian Elkhound and Karelian Bear Dog)** | ITGA10 | C>T | 0 | AR | Clear |
| **Chondrodystrophy (CDDY) and Intervertebral Disc Disease (IVDD) Risk** | FGF4 retrogene | Insertion | 0 | AD | Clear |
| **Cleft Lip & Palate with Syndactyly** | ADAMTS20 | Deletion | 0 | AR | Clear |
| **Cleft Palate** | DLX6 | C>A | 0 | AR | Clear |
| **CNS Atrophy with Cerebellar Ataxia (Discovered in the Belgian Shepherd)** | SEPP1 | Deletion | 0 | AR | Clear |
| **Coat Color Dilution and Neurological Defects (Discovered in the Miniature Dachshund)** | MYO5A | Insertion | 0 | AR | Clear |
| **Complement 3 Deficiency** | C3 | Deletion | 0 | AR | Clear |
| **Cone Degeneration (Discovered in the Alaskan Malamute)** | CNGB3 | Deletion | 0 | AR | Clear |
| **Cone Degeneration (Discovered in the German Shepherd Dog)** | CNGA3 | C>T | 0 | AR | Clear |
| **Cone Degeneration (Discovered in the German Shorthaired Pointer)** | CNGB3 | G>A | 0 | AR | Clear |
| **Cone-Rod Dystrophy** | NPHP4 | Deletion | 0 | AR | Clear |
| **Cone-Rod Dystrophy 1** | PDE6B | Deletion | 0 | AR | Clear |
| **Cone-Rod Dystrophy 2** | IQCB1 | Insertion | 0 | AR | Clear |
| **Congenital Cornification (Discovered in the Labrador Retriever)** | NSDHL | Deletion | 0 | XD | Clear |

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## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Congenital Dyshormonogenic Hypothyroidism with Goiter (Discovered in the Shih Tzu)** | SLC5A5 | G>A | 0 | AR | Clear |
| **Congenital Eye Malformations (Discovered in the Golden Retriever)** | SIX6 | C>T | 0 | AD | Clear |
| **Congenital Hypothyroidism (Discovered in the Tenterfield Terrier)** | TPO | C>T | 0 | AR | Clear |
| **Congenital Hypothyroidism (Discovered in the Toy Fox and Rat Terrier)** | TPO | C>T | 0 | AR | Clear |
| **Congenital Muscular Dystrophy (Discovered in the Italian Greyhound)** | LAMA2 | G>A | 0 | AR | Clear |
| **Congenital Muscular Dystrophy (Discovered in the Staffordshire Bull Terrier)** | LAMA2 | Deletion | 0 | AR | Clear |
| **Congenital Myasthenic Syndrome (Discovered in the Golden Retriever)** | COLQ | G>A | 0 | AR | Clear |
| **Congenital Myasthenic Syndrome (Discovered in the Heideterrier)** | CHRNE | Insertion | 0 | AR | Clear |
| **Congenital Myasthenic Syndrome (Discovered in the Jack Russell Terrier)** | CHRNE | Insertion | 0 | AR | Clear |
| **Congenital Myasthenic Syndrome (Discovered in the Labrador Retriever)** | COLQ | T>C | 0 | AR | Clear |
| **Congenital Myasthenic Syndrome (Discovered in the Old Danish Pointer)** | CHAT | G>A | 0 | AR | Clear |
| **Congenital Stationary Night Blindness (CSNB)** | RPE65 | A>T | 0 | AR | Clear |
| **Craniomandibular Osteopathy (Discovered in Scottish Terrier breeds)** | SLC37A2 | C>T | 0 | AD | Clear |
| **Craniomandibular Osteopathy (Discovered in the Australian Terrier)** | COL1A1 | C>T | 0 | AD | Clear |
| **Craniomandibular Osteopathy (Discovered in the Basset Hound)** | SLC37A2 | C>T | 0 | AD | Clear |
| **Craniomandibular Osteopathy (Discovered in the Weimaraner)** | SLC35D1 | Deletion | 0 | AD | Clear |
| **Cystic Renal Dysplasia and Hepatic Fibrosis** | INPP5E | G>A | 0 | AR | Clear |

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## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Cystinuria Type I-A** | SLC3A1 | C>T | 0 | AR | Clear |
| **Cystinuria Type II-A** | SLC3A1 | Deletion | 0 | AD | Clear |
| **Darier Disease (Discovered in the Irish Terrier)** | ATP2A2 | Insertion | 0 | AD | Clear |
| **Deafness and Vestibular Dysfunction (DINGS1), (Discovered in Doberman Pinscher)** | PTPRQ | Insertion | 0 | AR | Clear |
| **Deafness and Vestibular Dysfunction (DINGS2), (Discovered in Doberman Pinscher)** | MYO7A | G>A | 0 | AR | Clear |
| **Degenerative Myelopathy** | SOD1 | G>A | 0 | AR | Clear |
| **Demyelinating Neuropathy** | SBF2 | G>T | 0 | AR | Clear |
| **Dental-Skeletal-Retinal Anomaly (Discovered in the Cane Corso)** | MIA3 | Deletion | 0 | AR | Clear |
| **Dilated Cardiomyopathy (Discovered in the Schnauzer)** | RBM20 | Deletion | 0 | AR | Clear |
| **Disproportionate Dwarfism (Discovered in the Dogo Argentino)** | PRKG2 | C>A | 0 | AR | Clear |
| **Dominant Progressive Retinal Atrophy** | RHO | C>G | 0 | AD | Clear |
| **Dystrophic Epidermolysis Bullosa (Discovered in the Basset Hound)** | COL7A1 | Insertion | 0 | AR | Clear |
| **Dystrophic Epidermolysis Bullosa (Discovered in the Central Asian Ovcharka)** | COL7A1 | C>T | 0 | AR | Clear |
| **Dystrophic Epidermolysis Bullosa (Discovered in the Golden Retriever)** | COL7A1 | C>T | 0 | AR | Clear |
| **Early Retinal Degeneration (Discovered in the Norwegian Elkhound)** | STK38L | Insertion | 0 | AR | Clear |
| **Early-Onset Adult Deafness (Discovered in the Rhodesian Ridgeback)** | EPS8L2 | Deletion | 0 | AR | Clear |
| **Early-Onset Progressive Polyneuropathy (Discovered in the Alaskan Malamute)** | NDRG1 | G>T | 0 | AR | Clear |
| **Early-Onset Progressive Polyneuropathy (Discovered in the Greyhound)** | NDRG1 | Deletion | 0 | AR | Clear |

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## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Early-Onset Progressive Retinal Atrophy (Discovered in the Portuguese Water Dog)** | CCDC66 | Insertion | 0 | AR | Clear |
| **Early-Onset Progressive Retinal Atrophy, (Discovered in the Spanish Water Dog)** | PDE6B | Deletion | 0 | AR | Clear |
| **Ehlers-Danlos Syndrome (Discovered in mixed breed)** | COL5A1 | G>A | 0 | AD | Clear |
| **Ehlers-Danlos Syndrome (Discovered in the Labrador Retriever)** | COL5A1 | Deletion | 0 | AD | Clear |
| **Epidermolytic Hyperkeratosis** | KRT10 | G>T | 0 | AR | Clear |
| **Episodic Falling Syndrome** | BCAN | Insertion | 0 | AR | Clear |
| **Exercise-Induced Collapse** | DNM1 | G>T | 0 | AR | Clear |
| **Factor VII Deficiency** | F7 | G>A | 0 | AR | Clear |
| **Factor XI Deficiency** | FXI | Insertion | 0 | AD | Clear |
| **Familial Nephropathy (Discovered in the English Cocker Spaniel)** | COL4A4 | A>T | 0 | AR | Clear |
| **Familial Nephropathy (Discovered in the English Springer Spaniel)** | COL4A4 | C>T | 0 | AR | Clear |
| **Fanconi Syndrome** | FAN1 | Deletion | 0 | AR | Clear |
| **Fetal Onset Neuroaxonal Dystrophy** | MFN2 | G>C | 0 | AR | Clear |
| **Focal Non-Epidermolytic Palmoplantar Keratoderma** | KRT16 | G>C | 0 | AR | Clear |
| **Generalized Progressive Retinal Atrophy (Discovered in the Schapendoes)** | CCDC66 | Insertion | 0 | AR | Clear |
| **Glanzmann Thrombasthenia Type I (Discovered in Great Pyrenees)** | ITGA2B | C>G | 0 | AR | Clear |
| **Glanzmann Thrombasthenia Type I (Discovered in mixed breed dogs)** | ITGA2B | C>T | 0 | AR | Clear |
| **Globoid Cell Leukodystrophy (Discovered in Terriers)** | GALC | A>C | 0 | AR | Clear |
| **Globoid Cell Leukodystrophy (Discovered in the Irish Setter)** | GALC | A>T | 0 | AR | Clear |

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| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Glycogen Storage Disease Type Ia (Discovered in the German Pinscher)** | G6PC | Insertion | 0 | AR | Clear |
| **Glycogen Storage Disease Type Ia (Discovered in the Maltese)** | G6PC | G>C | 0 | AR | Clear |
| **Glycogen Storage Disease Type IIIa, (GSD IIIa)** | AGL | Deletion | 0 | AR | Clear |
| **GM1 Gangliosidosis (Discovered in the Portuguese Water Dog)** | GLB1 | G>A | 0 | AR | Clear |
| **GM1 Gangliosidosis (Discovered in the Shiba)** | GLB1 | Deletion | 0 | AR | Clear |
| **GM2 Gangliosidosis (Discovered in the Japanese Chin)** | HEXA | G>A | 0 | AR | Clear |
| **GM2 Gangliosidosis (Discovered in the Toy Poodle)** | HEXB | Deletion | 0 | AR | Clear |
| **Hemophilia A (Discovered in Old English Sheepdog)** | FVIII | C>T | 0 | XR | Clear |
| **Hemophilia A (Discovered in the Boxer)** | FVIII | C>G | 0 | XR | Clear |
| **Hemophilia A (Discovered in the German Shepherd Dog - Variant 1)** | FVIII | G>A | 0 | XR | Clear |
| **Hemophilia A (Discovered in the German Shepherd Dog - Variant 2)** | FVIII | G>A | 0 | XR | Clear |
| **Hemophilia A (Discovered in the Havanese)** | FVIII | Insertion | 0 | XR | Clear |
| **Hemophilia A (Discovered in the Labrador Retriever)** | Confidential | - | 0 | XR | Clear |
| **Hemophilia B** | FIX | G>A | 0 | XR | Clear |
| **Hemophilia B (Discovered in the Airedale Terrier)** | FIX | Insertion | 0 | XR | Clear |
| **Hemophilia B (Discovered in the Lhasa Apso)** | FIX | Deletion | 0 | XR | Clear |
| **Hereditary Ataxia (Discovered in the Belgian Malinois)** | SLC12A6 | Insertion | 0 | AR | Clear |
| **Hereditary Ataxia (Discovered in the Norwegian Buhund)** | KCNIP4 | T>C | 0 | AR | Clear |
| **Hereditary Elliptocytosis** | SPTB | C>T | 0 | AD | Clear |
| **Hereditary Footpad Hyperkeratosis** | FAM83G | G>C | 0 | AR | Clear |

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| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Hereditary Nasal Parakeratosis (Discovered in the Greyhound)** | SUV39H2 | Deletion | 0 | AR | Clear |
| **Hereditary Nasal Parakeratosis (Discovered in the Labrador Retriever)** | SUV39H2 | A>C | 0 | AR | Clear |
| **Hereditary Vitamin D-Resistant Rickets Type II** | VDR | Deletion | 0 | AR | Clear |
| **Hypocatalasia** | CAT | G>A | 0 | AR | Clear |
| **Hypomyelination** | FNIP2 | Deletion | 0 | AR | Clear |
| **Hypophosphatasia** | Confidential | - | 0 | AR | Clear |
| **Ichthyosis (Discovered in the American Bulldog)** | NIPAL4 | Deletion | 0 | AR | Clear |
| **Ichthyosis (Discovered in the Great Dane)** | SLC27A4 | G>A | 0 | AR | Clear |
| **Ichthyosis Type 2 (Discovered in the Golden Retriever)** | ABHD5 | Deletion | 0 | AR | Clear |
| **Inflammatory Myopathy (Discovered in the Dutch Shepherd Dog)** | SLC25A12 | A>G | 0 | AR | Clear |
| **Inflammatory Pulmonary Disease (Discovered in the Rough Collie)** | AKNA | Deletion | 0 | AR | Clear |
| **Intestinal Cobalamin Malabsorption (Discovered in the Beagle)** | CUBN | Deletion | 0 | AR | Clear |
| **Intestinal Cobalamin Malabsorption (Discovered in the Komondor)** | CUBN | G>A | 0 | AR | Clear |
| **Intestinal Lipid Malabsorption (Discovered in the Australian Kelpie)** | ACSL5 | Deletion | 0 | AR | Clear |
| **Junctional Epidermolysis Bullosa (Discovered in the Australian Cattle Dog Mix)** | LAMA3 | T>A | 0 | AR | Clear |
| **Junctional Epidermolysis Bullosa (Discovered in the Australian Shepherd)** | LAMB3 | A>G | 0 | AR | Clear |
| **Juvenile Cataract (Discovered in the Wirehaired Pointing Griffon)** | FYCO1 | Deletion | 0 | AR | Clear |
| **Juvenile Dilated Cardiomyopathy (Discovered in the Toy Manchester Terrier)** | ABCC9 | G>A | 0 | AR | Clear |

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| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Juvenile Encephalopathy (Discovered in the Parson Russell Terrier)** | Confidential | - | 0 | AR | Clear |
| **Juvenile Laryngeal Paralysis and Polyneuropathy** | RAB3GAP1 | Deletion | 0 | AR | Clear |
| **Juvenile Myoclonic Epilepsy** | DIRAS1 | Deletion | 0 | AR | Clear |
| **L-2-Hydroxyglutaric aciduria (Discovered in the Staffordshire Bull Terrier)** | L2HGDH | T>C | 0 | AR | Clear |
| **L-2-Hydroxyglutaric Aciduria (Discovered in the West Highland White Terrier)** | Confidential | - | 0 | AR | Clear |
| **Lafora Disease (Linkage test)** | NHLRC1 | Insertion | 0 | AR | Clear |
| **Lagotto Storage Disease** | ATG4D | G>A | 0 | AR | Clear |
| **Lamellar Ichthyosis** | TGM1 | Insertion | 0 | AR | Clear |
| **Laryngeal Paralysis (Discovered in the Bull Terrier and Miniature Bull Terrier)** | RAPGEF6 | Insertion | 0 | AR | Clear |
| **Leigh-like Subacute Necrotizing Encephalopathy (Discovered in the Yorkshire Terrier)** | SLC19A3 | Insertion | 0 | AR | Clear |
| **Lethal Acrodermatitis (Discovered in the Bull Terrier)** | MKLN1 | A>C | 0 | AR | Clear |
| **Leukodystrophy (Discovered in the Standard Schnauzer)** | TSEN54 | C>T | 0 | AR | Clear |
| **Ligneous Membranitis** | PLG | T>A | 0 | AR | Clear |
| **Limb-girdle Muscular Dystrophy (Discovered in the Boston Terrier) Variant 1** | SGCD | Deletion | 0 | AR | Clear |
| **Limb-girdle Muscular Dystrophy, Type L3 (Discovered in the Miniature Dachshund)** | SGCA | G>A | 0 | AR | Clear |
| **Lung Developmental Disease (Discovered in the Airedale Terrier)** | LAMP3 | C>T | 0 | AR | Clear |
| **Macrothrombocytopenia (Discovered in Norfolk and Cairn Terrier)** | TUBB1 | G>A | 0 | AR | Clear |
| **May-Hegglin Anomaly** | MYH9 | G>A | 0 | AD | Clear |
| **Microphthalmia (Discovered in the Soft-Coated Wheaten Terrier)** | RBP4 | Deletion | 0 | AR | Clear |

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|---|---|---|---|---|---|
| **Mucopolysaccharidosis Type IIIA (Discovered in the Dachshund)** | SGSH | C>A | 0 | AR | Clear |
| **Mucopolysaccharidosis Type IIIA (Discovered in the New Zealand Huntaway)** | SGSH | Insertion | 0 | AR | Clear |
| **Mucopolysaccharidosis Type VII (Discovered in the Brazilian Terrier)** | GUSB | C>T | 0 | AR | Clear |
| **Mucopolysaccharidosis Type VII (Discovered in the German Shepherd Dog)** | GUSB | G>A | 0 | AR | Clear |
| **Mucopolysaccharidosis VI (Discovered in the Miniature Pinscher)** | ARSB | G>A | 0 | AR | Clear |
| **Muscular Dystrophy (Discovered in the Cavalier King Charles Spaniel)** | Dystrophin | G>T | 0 | XR | Clear |
| **Muscular Dystrophy (Discovered in the Golden Retriever)** | Dystrophin | A>G | 0 | XR | Clear |
| **Muscular Dystrophy (Discovered in the Landseer)** | COL6A1 | G>T | 0 | AR | Clear |
| **Muscular Dystrophy (Discovered in the Norfolk Terrier)** | Dystrophin | Deletion | 0 | XR | Clear |
| **Muscular Dystrophy-Dystroglycanopathy (Discovered in the Labrador Retriever)** | LARGE | C>T | 0 | AR | Clear |
| **Muscular Hypertrophy (Double Muscling)** | MSTN | T>A | 0 | AR | Clear |
| **Musladin-Lueke Syndrome** | ADAMTSL2 | C>T | 0 | AR | Clear |
| **Myeloperoxidase Deficiency** | MOP | C>T | 0 | AR | Clear |
| **Myotonia Congenita (Discovered in Australian Cattle Dog)** | CLCN1 | Insertion | 0 | AR | Clear |
| **Myotonia Congenita (Discovered in the Labrador Retriever)** | CLCN1 | T>A | 0 | AR | Clear |
| **Myotonia Congenita (Discovered in the Miniature Schnauzer)** | CLCN1 | C>T | 0 | AR | Clear |
| **Myotubular Myopathy** | MTM1 | A>C | 0 | XR | Clear |
| **Narcolepsy (Discovered in the Dachshund)** | HCRTR2 | G>A | 0 | AR | Clear |
| **Narcolepsy (Discovered in the Labrador Retriever)** | HCRTR2 | G>A | 0 | AR | Clear |

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## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Nemaline Myopathy** | NEB | C>A | 0 | AR | Clear |
| **Neonatal Cerebellar Cortical Degeneration** | SPTBN2 | Deletion | 0 | AR | Clear |
| **Neonatal Encephalopathy with Seizures** | ATF2 | T>G | 0 | AR | Clear |
| **Neuroaxonal Dystrophy (Discovered in Spanish Water Dog)** | TECPR2 | C>T | 0 | AR | Clear |
| **Neuroaxonal Dystrophy (Discovered in the Papillon)** | PLA2G6 | G>A | 0 | AR | Clear |
| **Neuroaxonal Dystrophy (Discovered in the Rottweiler)** | VPS11 | A>G | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 1** | PPT1 | Insertion | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 12 (Discovered in the Australian Cattle Dog)** | ATP13A2 | C>T | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Golden Retriever)** | CLN5 | Deletion | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 7** | MFSD8 | Deletion | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Alpine Dachsbracke)** | CLN8 | Deletion | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Australian Shepherd)** | CLN8 | G>A | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 8 (Discovered in the English Setter)** | CLN8 | T>C | 0 | AR | Clear |
| **Neuronal Ceroid Lipofuscinosis 8 (Discovered in the Saluki)** | CLN8 | Insertion | 0 | AR | Clear |
| **Obesity risk (POMC)** | POMC | Deletion | 0 | AD | Clear |
| **Osteochondrodysplasia** | SLC13A1 | Deletion | 0 | AR | Clear |
| **Osteochondromatosis (Discovered in the American Staffordshire Terrier)** | EXT2 | C>A | 0 | AR | Clear |
| **Osteogenesis Imperfecta (Discovered in the Beagle)** | COL1A2 | C>T | 0 | AD | Clear |
| **Osteogenesis Imperfecta (Discovered in the Dachshund)** | SERPINH1 | T>C | 0 | AR | Clear |

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## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **P2RY12-associated Bleeding Disorder** | P2RY12 | Deletion | 0 | AR | Clear |
| **Palmoplantar Hyperkeratosis (Discovered in the Rottweiler)** | DSG1 | Deletion | 0 | AR | Clear |
| **Paroxysmal Dyskinesia** | PIGN | C>T | 0 | AR | Clear |
| **Persistent Müllerian Duct Syndrome** | AMHR2 | C>T | 0 | AR | Clear |
| **Phosphofructokinase Deficiency** | PFKM | G>A | 0 | AR | Clear |
| **Pituitary Dwarfism (Discovered in the Karelian Bear Dog)** | POU1F1 | C>A | 0 | AR | Clear |
| **Polycystic Kidney Disease** | PKD1 | G>A | 0 | AD | Clear |
| **Prekallikrein Deficiency** | KLKB1 | T>A | 0 | AR | Clear |
| **Primary Ciliary Dyskinesia** | CCDC39 | C>T | 0 | AR | Clear |
| **Primary Ciliary Dyskinesia (Discovered in the Alaskan Malamute)** | NME5 | Deletion | 0 | AR | Clear |
| **Primary Lens Luxation** | ADAMTS17 | G>A | 0 | AR | Clear |
| **Primary Open Angle Glaucoma (Discovered in Basset Fauve de Bretagne)** | ADAMTS17 | G>A | 0 | AR | Clear |
| **Primary Open Angle Glaucoma (Discovered in Petit Basset Griffon Vendeen)** | ADAMTS17 | Insertion | 0 | AR | Clear |
| **Primary Open Angle Glaucoma and Lens Luxation (Discovered in Chinese Shar-Pei)** | ADAMTS17 | Deletion | 0 | AR | Clear |
| **Progressive Early-Onset Cerebellar Ataxia** | SEL1L | T>C | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Basenji)** | SAG | T>C | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA 2 variant)** | TTC8 | Deletion | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Golden Retriever - GR-PRA1 variant)** | SLC4A3 | Insertion | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Lapponian Herder)** | IFT122 | C>T | 0 | AR | Clear |

# Paris

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Border Collie
**Birth date:** 2025-08-05
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Progressive Retinal Atrophy (Discovered in the Lhasa Apso)** | IMPG2 | Insertion | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Miniature Long Haired Dachshund)** | RPGRIP1 | Insertion | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Papillon and Phalène)** | CNGB1 | Deletion | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - BBS2 variant)** | Confidential | - | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Shetland Sheepdog - CNGA1 variant)** | CNGA1 | Deletion | 0 | AR | Clear |
| **Progressive Retinal Atrophy (Discovered in the Swedish Vallhund)** | MERTK | Insertion | 0 | AR | Clear |
| **Progressive Retinal Atrophy 1 (Discovered in the Italian Greyhound)** | Confidential | - | 0 | AR | Clear |
| **Progressive Retinal Atrophy Type III** | FAM161A | Insertion | 0 | AR | Clear |
| **Progressive Rod Cone Degeneration (prcd-PRA)** | PRCD | G>A | 0 | AR | Clear |
| **Protein Losing Nephropathy** | NPHS1 | G>A | 0 | AR | Clear |
| **Pyruvate Dehydrogenase Phosphatase 1 Deficiency** | PDP1 | C>T | 0 | AR | Clear |
| **Pyruvate Kinase Deficiency (Discovered in the Basenji)** | PKLR | Deletion | 0 | AR | Clear |
| **Pyruvate Kinase Deficiency (Discovered in the Beagle)** | PKLR | G>A | 0 | AR | Clear |
| **Pyruvate Kinase Deficiency (Discovered in the Pug)** | PKLR | T>C | 0 | AR | Clear |
| **Pyruvate Kinase Deficiency (Discovered in the West Highland White Terrier)** | PKLR | Insertion | 0 | AR | Clear |
| **QT Syndrome** | KCNQ1 | C>A | 0 | AD | Clear |
| **Renal Cystadenocarcinoma and Nodular Dermatofibrosis** | FLCN | A>G | 0 | AD | Clear |
| **Rod-Cone Dysplasia 1** | PDE6B | G>A | 0 | AR | Clear |
| **Rod-Cone Dysplasia 1a** | PDE6B | Insertion | 0 | AR | Clear |

# Paris

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Border Collie
**Birth date:** 2025-08-05
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Rod-Cone Dysplasia 3** | PDE6A | Deletion | 0 | AR | Clear |
| **Sensorineural Deafness (Discovered in the Rottweiler)** | LOXHD1 | G>C | 0 | AR | Clear |
| **Sensory Ataxic Neuropathy** | tRNATyr | Deletion | 0 | MT | Clear |
| **Severe Combined Immunodeficiency (Discovered in Frisian Water Dogs)** | RAG1 | G>T | 0 | AR | Clear |
| **Severe Combined Immunodeficiency (Discovered in Russell Terriers)** | PRKDC | G>T | 0 | AR | Clear |
| **Shaking Puppy Syndrome (Discovered in the Border Terrier)** | Confidential | - | 0 | AR | Clear |
| **Skeletal Dysplasia 2** | COL11A2 | G>C | 0 | AR | Clear |
| **Spinocerebellar Ataxia (Late-Onset Ataxia)** | CAPN1 | G>A | 0 | AR | Clear |
| **Spinocerebellar Ataxia with Myokymia and/or Seizures** | KCNJ10 | C>G | 0 | AR | Clear |
| **Spondylocostal Dysostosis** | HES7 | Deletion | 0 | AR | Clear |
| **Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA1)** | KCNJ10 | T>C | 0 | AR | Clear |
| **Spongy Degeneration with Cerebellar Ataxia (Discovered in Belgian Malinois - SDCA2)** | ATP1B2 | Insertion | 0 | AR | Clear |
| **Stargardt Disease (Discovered in the Labrador Retriever)** | ABCA4 | Insertion | 0 | AR | Clear |
| **Startle Disease (Discovered in Irish Wolfhounds)** | SLC6A5 | G>T | 0 | AR | Clear |
| **Startle Disease (Discovered in the Miniature American Shepherd)** | Confidential | - | 0 | AR | Clear |
| **Succinic Semialdehyde Dehydrogenase Deficiency (Discovered in the Saluki)** | ALDH5A1 | G>A | 0 | AR | Clear |
| **Thrombopathia (Discovered in the Basset Hound)** | RASGRP1 | Deletion | 0 | AR | Clear |
| **Thrombopathia (Discovered in the Eskimo Spitz)** | RASGRP1 | Insertion | 0 | AR | Clear |
| **Van den Ende-Gupta Syndrome** | SCARF2 | Deletion | 0 | AR | Clear |
| **von Willebrand's Disease, type 1** | VWF | G>A | 0 | AD | Clear |

# Paris

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Border Collie
**Birth date:** 2025-08-05
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

## Other health conditions tested

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **von Willebrand's Disease, type 2** | VWF | T>G | 0 | AR | Clear |
| **von Willebrand's Disease, type 3 (Discovered in the Kooiker Hound)** | VWF | G>A | 0 | AR | Clear |
| **von Willebrand's Disease, type 3 (Discovered in the Scottish Terrier)** | VWF | Deletion | 0 | AR | Clear |
| **von Willebrand's Disease, type 3 (Discovered in the Shetland Sheepdog)** | VWF | Deletion | 0 | AR | Clear |
| **X-Linked Ectodermal Dysplasia** | EDA | G>A | 0 | XR | Clear |
| **X-Linked Hereditary Nephropathy (Discovered in the Navasota Dog)** | COL4A5 | Deletion | 0 | XR | Clear |
| **X-Linked Hereditary Nephropathy (Discovered in the Samoyed)** | COL4A5 | G>T | 0 | XR | Clear |
| **X-Linked Myotubular Myopathy** | MTM1 | C>A | 0 | XR | Clear |
| **X-Linked Progressive Retinal Atrophy 1** | RPGR | Deletion | 0 | XR | Clear |
| **X-Linked Progressive Retinal Atrophy 2** | RPGR | Deletion | 0 | XR | Clear |
| **X-Linked Severe Combined Immunodeficiency (Discovered in the Basset Hound)** | IL2RG | Deletion | 0 | XR | Clear |
| **X-Linked Severe Combined Immunodeficiency (Discovered in the Cardigan Welsh Corgi)** | IL2RG | Insertion | 0 | XR | Clear |
| **X-Linked Tremors** | PLP1 | A>C | 0 | XR | Clear |
| **Xanthinuria (Discovered in a mixed breed dog)** | Confidential | - | 0 | AR | Clear |
| **Xanthinuria (Discovered in the Cavalier King Charles Spaniel)** | Confidential | - | 0 | AR | Clear |
| **Xanthinuria (Discovered in the Toy Manchester Terrier)** | Confidential | - | 0 | AR | Clear |

**Breed:** Border Collie
**Birth date:** 2025-08-05
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

# Glossary of genetic terms

## Test result definitions

**At Risk:** Based on the disorder's mode of inheritance, the dog inherited a number of genetic variant(s) which increases the dog's risk of being diagnosed with the associated disorder.

**Carrier:** The dog inherited one copy of a genetic variant when two copies are usually necessary to increase the dog's risk of being diagnosed with the associated disorder. While carriers are usually not at risk of clinical expression of the disorder, carriers of some complex variants may be associated with a low risk of developing the disorder.

**Clear:** The dog did not inherit the genetic variant(s) associated with the disorder and will not be at elevated risk of being diagnosed with the disorder due to this genotype. However, similar clinical signs could develop from different genetic or clinical causes.

**Inconclusive:** An inconclusive result indicates a confident call could not be made based on the data for that genetic variant. Health testing is performed in replicates, and on occasion the outcomes do not agree. This may occur due to an unusual sequence of DNA in the region tested, multiple cell genotypes present due to chimerism or acquired mutations, or due to quality of the DNA sample.

## Inheritance mode definitions

**Autosomal Recessive (AR):** For autosomal recessive disorders, dogs with two copies of the genetic variant are at risk of developing the associated disorder. Dogs with one copy of the variant are considered carriers and are usually not at risk of developing the disorder. However, carriers of some complex variants grouped in this category may be associated with a low risk of developing the disorder. Dogs with one or two copies may pass the disorder-associated variant to their puppies if bred.

**Autosomal Dominant (AD):** For autosomal dominant disorders, dogs with one or two copies of the genetic variant are at risk of developing the associated disorder. Inheriting two copies of the variant may increase the risk of development of the disorder or cause the condition to be more severe. These dogs may pass the disorder-associated variant to their puppies if bred.

**X-linked Recessive (XR):** For X-linked recessive disorders, the genetic variant is found on the X chromosome. Female dogs must inherit two copies of the variant to be at risk of developing the condition, whereas male dogs only need one copy to be at risk. Males and females with any copies of the variant may pass the disorder-associated variant to their puppies if bred.

**X-linked Dominant (XD):** For X-linked dominant disorders, the genetic variant is found on the X chromosome. Both male and female dogs with one copy of the variant are at risk of developing the disorder. Females inheriting two copies of the variant may be at higher risk or show a more severe form of the disorder than with one copy. Males and females with any copies of the variant may pass the disorder-associated variant to their puppies if bred.

**Mitochondrial (MT):** Unlike the two copies of genomic DNA held in the nucleus, there are thousands of mitochondria in each cell of the body, and each holds its own mitochondrial DNA (mtDNA). Mitochondria are called the "powerhouses" of the cell. For a dog to be at risk for a mitochondrial disorder, it must inherit a certain ratio of mtDNA with the associated variant compared to normal mtDNA. mtDNA is inherited only from the mother.

# Paris

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Border Collie
**Birth date:** 2025-08-05
**Owner:** Jamie Gardner
**Registry:** American Border Collie Association
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

## Paris's Profile

### Pet information

**Registered name**
Paris

**Sex**
F

### Breed specific genetic health tests

| Genetic Condition | Gene | Risk Variant | Copies | Inheritance | Result |
|---|---|---|---|---|---|
| **Intestinal Cobalamin Malabsorption (Discovered in the Border Collie)** | CUBN | Deletion | 1 | AR | Carrier |
| **Trapped Neutrophil Syndrome** | VPS13B | Deletion | 1 | AR | Carrier |
| **Collie Eye Anomaly (CEA)** | NHEJ1 | Deletion | 0 | AR | Clear |
| **Dental Hypomineralization** | FAM20C | C>T | 0 | AR | Clear |
| **Early Adult Onset Deafness For Border Collies only (Linkage test)** | Intergenic | Insertion | 0 | AR | Clear |
| **Hereditary Calcium Oxalate Urolithiasis, Type 1** | Confidential | - | 0 | AR | Clear |
| **Hyperuricosuria** | SLC2A9 | G>T | 0 | AR | Clear |
| **MDR1 Medication Sensitivity** | MDR1/ABCB1 | Deletion | 0 | AD | Clear |
| **Neuronal Ceroid Lipofuscinosis 5 (Discovered in the Border Collie)** | CLN5 | C>T | 0 | AR | Clear |
| **Sensory Neuropathy** | FAM134B | Insertion | 0 | AR | Clear |

# Paris

Optimal Selection™ powered by WISDOM PANEL™

**Breed:** Border Collie
**Birth date:** 2025-08-05
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**Registry:** American Border Collie Association
**Test date:** 2025-11-11
**ID kit:** DMPJDYH

## Glossary of genetic terms

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# Paris

| Call name | Date of birth | Kennel Club or Registry | Breed |
|---|---|---|---|
| Paris | 2025-08-05 | American Border Collie Association | Border Collie |

Genetic sex
Female

## SNP - ISAG 2020 Panel 1

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 1 | Cfam_1:3962719 | A/G | 39 | Cfam_11:23907101 | C/C | 77 | Cfam_25:2073511 | A/A |
| 2 | Cfam_1:20842130 | G/G | 40 | Cfam_11:65603333 | A/A | 78 | Cfam_25:33986348 | G/G |
| 3 | Cfam_1:70238933 | A/G | 41 | Cfam_12:5579055 | A/A | 79 | Cfam_25:47708600 | A/A |
| 4 | Cfam_1:80971770 | A/A | 42 | Cfam_12:35306641 | A/G | 80 | Cfam_26:20004896 | G/G |
| 5 | Cfam_1:106430955 | A/A | 43 | Cfam_12:55201839 | A/G | 81 | Cfam_26:35071515 | A/A |
| 6 | Cfam_1:119414584 | A/G | 44 | Cfam_12:68125319 | A/G | 82 | Cfam_27:2619058 | A/G |
| 7 | Cfam_2:2610859 | G/G | 45 | Cfam_13:8704192 | A/G | 83 | Cfam_27:22599860 | G/G |
| 8 | Cfam_2:38293797 | G/G | 46 | Cfam_13:59896033 | C/C | 84 | Cfam_27:41049333 | A/C |
| 9 | Cfam_2:77806065 | G/G | 47 | Cfam_14:50063321 | A/G | 85 | Cfam_28:9877730 | A/A |
| 10 | Cfam_3:1252765 | A/C | 48 | Cfam_14:58465266 | A/A | 86 | Cfam_28:18509221 | A/G |
| 11 | Cfam_3:24757939 | A/G | 49 | Cfam_15:19299365 | G/G | 87 | Cfam_28:38885325 | A/G |
| 12 | Cfam_3:73570828 | A/G | 50 | Cfam_15:22834903 | A/A | 88 | Cfam_29:251970 | A/A |
| 13 | Cfam_4:31301072 | G/G | 51 | Cfam_16:29634940 | A/A | 89 | Cfam_29:9625359 | G/G |
| 14 | Cfam_4:64121754 | G/G | 52 | Cfam_16:46884446 | C/C | 90 | Cfam_29:17561258 | A/A |
| 15 | Cfam_4:75910211 | G/G | 53 | Cfam_16:57958947 | G/G | 91 | Cfam_29:36319325 | A/C |
| 16 | Cfam_4:86049027 | A/G | 54 | Cfam_17:10649078 | A/G | 92 | Cfam_30:3896482 | G/G |
| 17 | Cfam_5:5410890 | A/G | 55 | Cfam_17:34462308 | A/G | 93 | Cfam_30:15542105 | A/G |
| 18 | Cfam_5:26320165 | A/A | 56 | Cfam_17:39124697 | A/A | 94 | Cfam_30:32852404 | A/G |
| 19 | Cfam_5:85451804 | G/G | 57 | Cfam_18:6745949 | A/G | 95 | Cfam_31:21068798 | A/A |
| 20 | Cfam_6:11553458 | A/G | 58 | Cfam_18:54361347 | A/A | 96 | Cfam_31:39391935 | A/A |
| 21 | Cfam_6:33976751 | A/G | 59 | Cfam_19:841347 | A/A | 97 | Cfam_32:679380 | A/G |
| 22 | Cfam_6:64006720 | A/G | 60 | Cfam_19:15926130 | A/A | 98 | Cfam_32:17792284 | G/G |
| 23 | Cfam_7:76294 | G/G | 61 | Cfam_19:27288167 | A/C | 99 | Cfam_32:32382778 | A/A |
| 24 | Cfam_7:15011628 | G/G | 62 | Cfam_19:47470564 | A/A | 100 | Cfam_33:15018500 | A/G |
| 25 | Cfam_7:36555518 | G/G | 63 | Cfam_20:13740894 | A/G | 101 | Cfam_33:23742061 | A/G |
| 26 | Cfam_8:5291824 | A/A | 64 | Cfam_20:49900586 | A/A | 102 | Cfam_34:195313 | A/A |
| 27 | Cfam_8:18121580 | A/A | 65 | Cfam_20:57167714 | A/G | 103 | Cfam_34:24396298 | A/G |
| 28 | Cfam_8:45852939 | A/G | 66 | Cfam_21:15558670 | G/G | 104 | Cfam_35:15345329 | C/C |
| 29 | Cfam_8:63196958 | G/G | 67 | Cfam_21:25537675 | G/G | 105 | Cfam_36:3565500 | A/A |
| 30 | Cfam_9:22610227 | A/G | 68 | Cfam_21:35719434 | A/G | 106 | Cfam_36:12714421 | A/G |
| 31 | Cfam_9:40096141 | A/G | 69 | Cfam_22:641125 | G/G | 107 | Cfam_36:23459390 | G/G |
| 32 | Cfam_9:52710991 | A/A | 70 | Cfam_22:26694580 | G/G | 108 | Cfam_37:9398945 | A/G |
| 33 | Cfam_9:60437147 | G/G | 71 | Cfam_22:55308193 | A/C | 109 | Cfam_37:15436615 | G/G |
| 34 | Cfam_10:10652659 | G/G | 72 | Cfam_23:42886681 | A/A | 110 | Cfam_37:27667297 | G/G |
| 35 | Cfam_10:22409408 | A/A | 73 | Cfam_23:50772488 | A/G | 111 | Cfam_38:9224942 | A/A |
| 36 | Cfam_10:30034450 | G/G | 74 | Cfam_24:23393510 | C/C | 112 | Cfam_38:17657161 | G/G |
| 37 | Cfam_10:66922269 | A/G | 75 | Cfam_24:29909901 | A/G | 113 | Cfam_38:20441216 | A/A |
| 38 | Cfam_11:5318488 | A/A | 76 | Cfam_24:47381908 | A/G | | | |

**SNP - ISAG 2020 Panel 2**

| | | |
|---|---|---|
| 1 | Cfam_1:72613047 | G/G |
| 2 | Cfam_1:74450772 | A/G |
| 3 | Cfam_1:119306331 | A/G |
| 4 | Cfam_3:10255068 | A/G |
| 5 | Cfam_3:37849557 | A/A |
| 6 | Cfam_3:43055696 | A/A |
| 7 | Cfam_3:43063677 | A/A |
| 8 | Cfam_3:64084413 | A/G |
| 9 | Cfam_3:90291255 | G/G |
| 10 | Cfam_3:91626907 | A/G |
| 11 | Cfam_4:42104780 | A/G |
| 12 | Cfam_4:67040898 | G/G |
| 13 | Cfam_4:70217695 | A/G |
| 14 | Cfam_5:13080303 | A/A |
| 15 | Cfam_5:36642434 | A/G |
| 16 | Cfam_5:44650576 | G/G |
| 17 | Cfam_5:55349573 | G/G |
| 18 | Cfam_5:64611038 | A/G |
| 19 | Cfam_7:3318809 | G/G |
| 20 | Cfam_7:6423299 | G/G |
| 21 | Cfam_7:15017979 | G/G |
| 22 | Cfam_7:76487265 | G/G |
| 23 | Cfam_8:6188937 | G/G |
| 24 | Cfam_8:19076567 | A/A |
| 25 | Cfam_8:24614720 | A/G |
| 26 | Cfam_8:52381322 | A/A |
| 27 | Cfam_8:67183794 | A/G |
| 28 | Cfam_9:20867959 | G/G |
| 29 | Cfam_9:32506288 | A/G |
| 30 | Cfam_9:50114927 | A/A |
| 31 | Cfam_9:56021221 | A/G |
| 32 | Cfam_10:8085469 | A/A |
| 33 | Cfam_10:14685262 | G/G |
| 34 | Cfam_10:39548483 | A/A |
| 35 | Cfam_10:47923623 | G/G |
| 36 | Cfam_10:57954366 | G/G |
| 37 | Cfam_11:1161870 | A/A |
| 38 | Cfam_11:62157625 | A/G |
| 39 | Cfam_11:70698603 | A/A |
| 40 | Cfam_12:6337286 | A/A |
| 41 | Cfam_12:8532712 | G/G |
| 42 | Cfam_12:23059939 | A/G |
| 43 | Cfam_12:40681020 | A/G |
| 44 | Cfam_12:70657733 | G/G |
| 45 | Cfam_13:40616856 | G/G |
| 46 | Cfam_14:55735620 | G/G |
| 47 | Cfam_16:29675662 | A/C |
| 48 | Cfam_16:58093031 | A/C |
| 49 | Cfam_17:9407683 | G/G |
| 50 | Cfam_17:12787849 | A/C |
| 51 | Cfam_17:57371669 | G/G |
| 52 | Cfam_18:10189759 | A/A |
| 53 | Cfam_18:16385020 | A/A |
| 54 | Cfam_18:16388978 | A/A |
| 55 | Cfam_18:31579269 | A/A |
| 56 | Cfam_18:47325586 | A/A |
| 57 | Cfam_19:30246414 | G/G |
| 58 | Cfam_19:40189405 | C/C |
| 59 | Cfam_19:42756283 | A/A |
| 60 | Cfam_20:6046176 | A/A |
| 61 | Cfam_20:45777531 | A/G |
| 62 | Cfam_20:48602465 | A/G |
| 63 | Cfam_21:22581321 | A/A |
| 64 | Cfam_21:29796784 | A/A |
| 65 | Cfam_21:31751817 | G/G |
| 66 | Cfam_22:20498421 | A/A |
| 67 | Cfam_22:33934047 | A/A |
| 68 | Cfam_22:37522364 | A/G |
| 69 | Cfam_22:39647748 | G/G |
| 70 | Cfam_22:61153661 | G/G |
| 71 | Cfam_23:44497217 | A/A |
| 72 | Cfam_23:48055836 | A/C |
| 73 | Cfam_24:18599997 | A/A |
| 74 | Cfam_24:27925354 | A/A |
| 75 | Cfam_24:30954773 | A/A |
| 76 | Cfam_24:43589304 | G/G |
| 77 | Cfam_24:45191477 | G/G |
| 78 | Cfam_25:4614777 | A/A |
| 79 | Cfam_27:20948372 | A/A |
| 80 | Cfam_27:34444177 | G/G |
| 81 | Cfam_27:42526114 | G/G |
| 82 | Cfam_28:9703418 | A/G |
| 83 | Cfam_28:12804225 | G/G |
| 84 | Cfam_28:34478533 | A/G |
| 85 | Cfam_28:35104850 | G/G |
| 86 | Cfam_29:4020192 | A/G |
| 87 | Cfam_29:4022252 | A/G |
| 88 | Cfam_29:19681270 | A/A |
| 89 | Cfam_29:22992304 | G/G |
| 90 | Cfam_30:10012939 | A/G |
| 91 | Cfam_30:11735245 | A/A |
| 92 | Cfam_30:27619023 | A/A |
| 93 | Cfam_31:20912553 | G/G |
| 94 | Cfam_32:13183511 | G/G |
| 95 | Cfam_33:15233992 | G/G |
| 96 | Cfam_33:22070526 | A/G |
| 97 | Cfam_33:22472901 | A/C |
| 98 | Cfam_33:22648231 | G/G |
| 99 | Cfam_34:24351570 | A/G |
| 100 | Cfam_34:34993916 | A/A |
| 101 | Cfam_34:37323213 | A/A |
| 102 | Cfam_34:41703614 | A/G |
| 103 | Cfam_35:15283717 | A/G |
| 104 | Cfam_36:288045 | G/G |
| 105 | Cfam_36:9241262 | A/G |
| 106 | Cfam_36:10084888 | A/G |
| 107 | Cfam_36:12723744 | A/C |
| 108 | Cfam_36:18627936 | A/A |
| 109 | Cfam_37:18338930 | A/C |
| 110 | Cfam_37:26611359 | A/A |
| 111 | Cfam_37:28611801 | G/G |
| 112 | Cfam_37:30110473 | A/G |
| 113 | Cfam_37:30902202 | A/G |
| 114 | Cfam_38:13098194 | A/C |
| 115 | Cfam_38:15271384 | A/G |
| 116 | Cfam_38:19172567 | A/A |
| 117 | Cfam_38:20930997 | A/A |
| SEX | Cfam_x:7828353 | X/X |