



"BO"

THE KEEP HELL'S BELLE BOADICEA



DNA Test Report

Test Date: July 13th, 2018

embk.me/bo54

BREED MIX

 **Pomeranian: 100.0%**

GENETIC STATS

Wolfiness: 1.7 % **HIGH**

Predicted adult weight: **12 lbs**

Genetic age: **22 human years**

TEST DETAILS

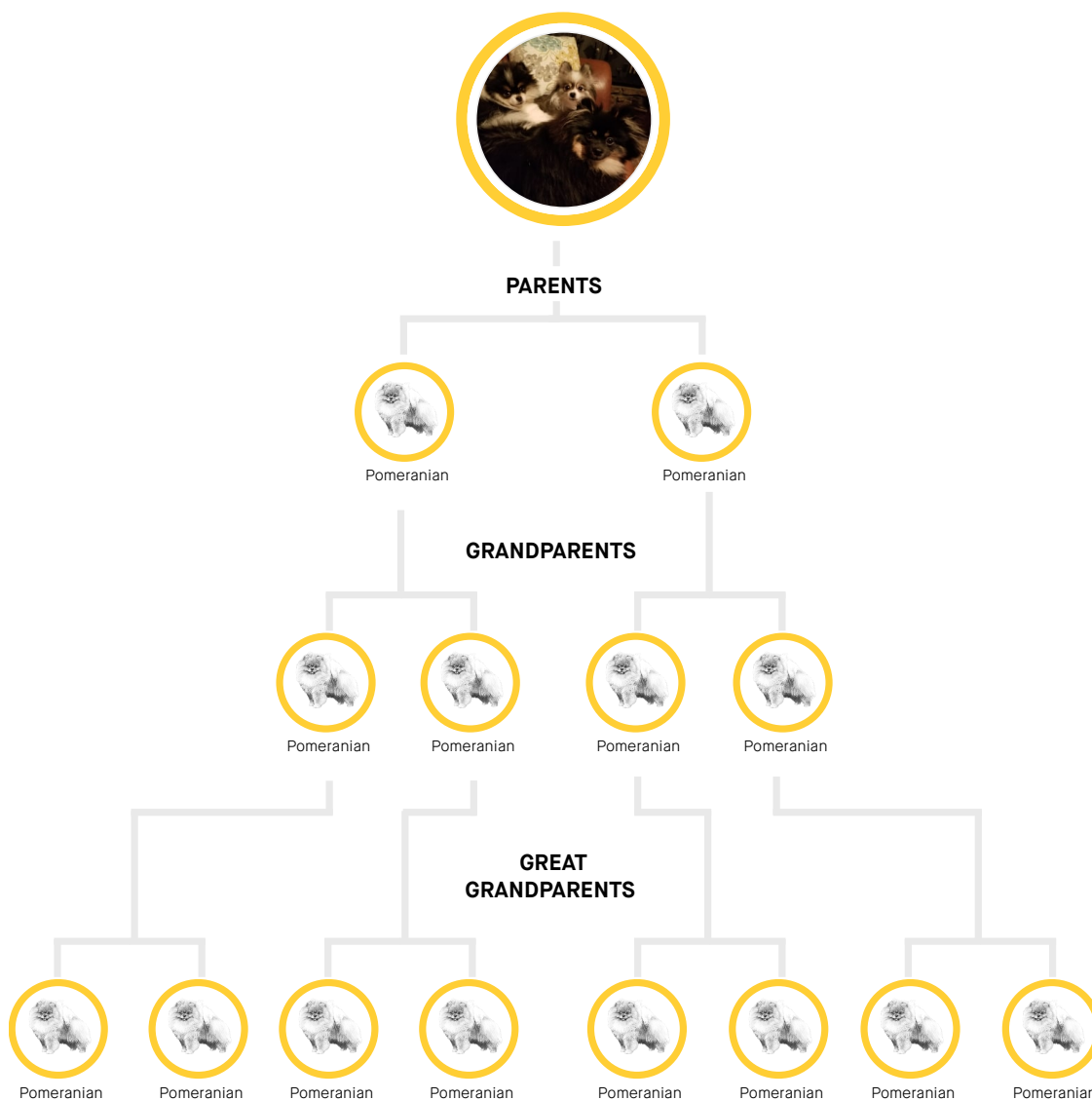
Kit number: EM-4297582

Swab number: 31001802022494



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FAMILY TREE



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



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POMERANIAN



Fun Fact Pomeranians boast one of the widest variety of color options in one breed. The American Kennel Club lists 23 accepted colors

Cute, feisty and furry, Poms are intelligent and loyal to their families. Don't let their cuteness fool you, however. These independent, bold dogs have minds of their own. They are alert and curious about the world around them. Unfortunately, in their minds, they are much larger than they really are, which can sometimes lead them to harass and even attack much larger dogs. Luckily, if they are properly socialized with other dogs and animals, they generally get along quite well with them. Poms take their name from the province of Pomerania, in Germany. They became especially popular when Queen Victoria allowed some of her Pomeranians to be shown in a conformation show, the first Pomeranians ever to be shown. Pomeranians make excellent pets for older people and those who are busy, because they aren't an overly dependent breed. They are also good for apartment dwellers or homes that don't have a backyard. Because of their small size, they aren't recommended for families with small children who might injure them accidentally. While Poms are good with children, they are not a good choice for very young or highly active children because of their small size. Never let your small children and your Pom play without supervision. Because they are so small, Poms can be perceived as prey by owls, eagles, hawks, coyotes, and other wild animals. Never leave them outside unattended, and be watchful if there are predatory birds in your location. If this is the case, stay close to your Pom to discourage birds from trying to carry them off!

RELATED BREEDS



**American Eskimo
Dog**
Cousin breed



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MATERNAL LINE



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

HAPLOGROUP: 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: B1c

Part of the large B1 haplogroup, we have detected this haplotype in Mexico and Lebanon village dogs. Among the 12 breeds that we have spotted this haplotype in, it occurs most frequently in Border Collies, Australian Shepherd Dogs, and West Highland white Terriers.



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TRAITS

Coat Color

E Locus (Mask, Grizzle, Recessive Red)	EE
K Locus (Dominant Black)	k^Yk^Y
A Locus (Agouti, Sable)	a^ta^t
D Locus (Dilute, Blue, Fawn)	DD
B Locus (Brown, Chocolate, Liver, Red, Dudley)	Bb

Other Coat Traits

Furnishings / Improper Coat (RSPO2)	II
Long Haircoat (FGF5)	TT
Shedding (MC5R)	CC
Curly Coat (KRT71)	CC
Hairlessness (FOXI3)	N/N
Oculocutaneous Albinism Type 2 - OCA2, Doberman Z Factor Albinism (SLC45A2)	N/N

Other Body Features

Brachycephaly (BMP3)	AC
Natural Bobtail (T)	CC
Hind Dewclaws (LMBR1)	CC
Blue Eye Color	N/N

Performance

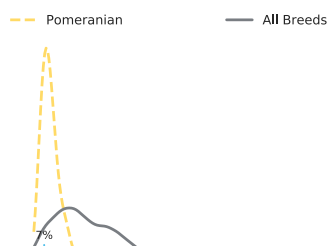
Altitude Adaptation (EPAS1)	GG
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Body Size

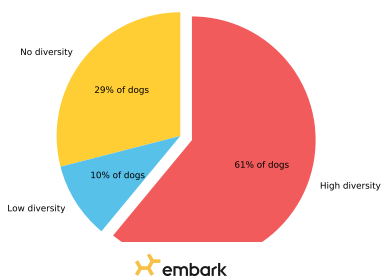
Body Size - IGF1	II
Body Size - IGF1R	GA
Body Size - STC2	TA
Body Size - GHR (E195K)	AA
Body Size - GHR (P177L)	CT

Genetic Diversity

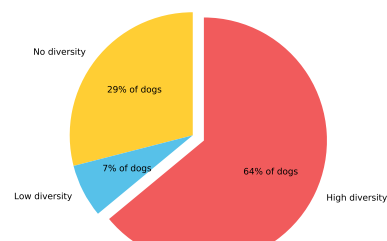
Inbreeding Coefficient **7%**



MHC Class II - DLA DRB1
High Diversity



MHC Class II - DLA DQA1 and DQB1
High Diversity





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CLINICAL TRAITS

These clinical genetic traits can inform clinical decisions and diagnoses. These traits do not predict a disease state or increased risk for disease. We currently assess one clinical trait: Alanine Aminotransferase Activity.

Alanine Aminotransferase Activity result: Low Normal

The Keep Hell's Belle Boadicea has two copies of a mutation associated with reduced ALT activity. Please inform your veterinarian that The Keep Hell's Belle Boadicea has this genotype, as ALT is often used as an indicator of liver health and The Keep Hell's Belle Boadicea is likely to have a lower than average resting ALT activity. As such, an increase in The Keep Hell's Belle Boadicea's ALT activity could be evidence of liver damage, even if it is within normal limits by standard ALT reference ranges.

More information on Alanine Aminotransferase Activity:

The liver enzyme alanine aminotransferase, or ALT, is one of several values your veterinarian measures on routine blood work to gauge liver health. Dogs with one or more copies of the “A” allele are likely to have a lower baseline ALT activity (“low normal”) than dogs with zero copies of the “A” allele (“normal”). This means that your veterinarian may recommend blood work to establish an individualized baseline ALT value during an annual wellness exam or before starting certain medications. You and your veterinarian would then be able to monitor your dog for any deviation from this established baseline. Please note that this mutation should never cause an increase in your dog's ALT activity and does not cause liver disease. If your dog has high ALT activity, please consult your veterinarian.



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HEALTH

Good news! Bo did not test positive for any of the genetic diseases that Embark screens for.

0

AT RISK

2

CARRIER

CARRIER CONDITIONS

CARRIER status: This indicates the dog has inherited a recessive allele for a genetic trait or mutation. This is not enough to cause symptoms of the disease, but is important to bear in mind if the dog ever has offspring.

Carrier

System: **Ophthalmologic**

Condition: **Canine Multifocal Retinopathy cmr1 (BEST1 Exon 2)**

Carrier

System: **Neurologic**

Condition: **Degenerative Myelopathy (SOD1A)**



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CANINE MULTIFOCAL RETINOPATHY

cmr1 (BEST1 Exon 2)

Carrier



BEST1/VMD2 (Exon 2)

GENE NAME

CC

CLEAR

CT

CARRIER

TT

AT RISK

The Keep Hell's Belle Boadicea is a carrier for a mutation in the BEST1 gene. As a carrier he or she is unlikely to show signs of the disease. If you choose to breed The Keep Hell's Belle Boadicea, we highly recommend genotyping any potential mates as breeding to another carrier or an affected dog could produce affected puppies.

DESCRIPTION

This is a non-progressive retinal disease that, in rare cases, can lead to vision loss. CMR is typically only identified when a vet examines the eye which, in dogs with CMR, reveals multiple retinal abnormalities that range from small, flat folds (called "retinal folds") to larger, irregularly edged raised lesions (called "geographic lesions"). Dogs with larger lesions can suffer from vision loss. CMR is fairly non-progressive; new lesions will typically stop forming by the time a dog is an adult, and some lesions will even regress with time.

CITATIONS

Guziewicz et al 2007 (<http://www.ncbi.nlm.nih.gov/pubmed/17460247>), Hoffman et al 2012 (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3787078/>), <http://omia.org/OMIA001554/9615/> ()



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DEGENERATIVE MYELOPATHY

(SOD1A)

Carrier



SOD1

GENE NAME

GG

CLEAR

GA

CARRIER

AA

AT RISK

The Keep Hell's Belle Boadicea is a carrier for a mutation in the SOD1 gene. While he or she is unlikely to exhibit signs of disease, as a carrier, he or she will pass the mutation on to the next generation. If you choose to breed The Keep Hell's Belle Boadicea, we highly recommend testing any potential mates for this mutation. Breeding to another carrier is not recommended as this will produce a number of affected puppies.

DESCRIPTION

A disease of mature dogs, this is a progressive degenerative disorder of the spinal cord that can cause muscle wasting and gait abnormalities. Affected dogs do not usually show signs until they are at least 8 years old, where the first signs of neural degeneration appear in the nerves that innervate the hind limbs. You may notice your dog scuffing the tops of his or her hind paws, or walking with a hesitant, exaggerated gait. In advanced cases, lower motor neurons are also affected leading to weakness or near-paralysis of all four legs and widespread muscle wasting. Given the advanced age at the time of onset, the treatment for DM is aimed towards making your dog comfortable in his or her old age and includes lifestyle changes and physical therapy. SOD1 codes superoxide dismutase, an enzyme important in neutralizing free radicals and reactive oxygen species, both of which are produced as a byproduct of cell metabolism. If not neutralized, these are injurious to the cell and will cause premature cell death. The first system to show effects of this is the nervous system given the highly specialized and delicate nature of these cells. Please note that these mutations are reported to have incomplete penetrance: that is, while a dog with two copies of this mutation has a much greater chance of developing DM than a dog with one copy of the mutation, or none at all, other genetic and environmental factors will also contribute to whether your dog develops DM.

More information

To learn more about this condition, you can visit <http://www.caninegeneticdiseases.net/DM/basicDM.htm> (<http://www.caninegeneticdiseases.net/DM/basicDM.htm>).

CITATIONS

Awano et al 2009 (<http://www.ncbi.nlm.nih.gov/pubmed/19188595>), Shelton et al 2012 (<http://www.ncbi.nlm.nih.gov/pubmed/22542607>), Capuccio et al 2014 (<http://www.ncbi.nlm.nih.gov/pubmed/24390315>)





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OTHER CONDITIONS

Good news! Bo tested clear for 2 genetic conditions that are common in her breed.

- Progressive Retinal Atrophy - rcd3
Rod-cone dysplasia, rcd3 (PDE6A)
- Hereditary Vitamin D-Resistant Rickets
(VDR)



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FULL TEST PANEL

Bo is also clear of 163 other genetic health conditions that Embark tests for.

To help ensure healthy breeds, every test includes analysis of our full panel of over 160 genetic health conditions.

The following pages list out all the other genetic health conditions that Bo tested clear for.



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CLEAR CONDITIONS

- MDR1 Drug Sensitivity (MDR1) (Chromosome 14)
- P2Y12 Receptor Platelet Disorder (P2RY12) (Chromosome 23)
- Factor IX Deficiency, Hemophilia B (F9 Exon 7, Terrier Variant) (Chromosome X)
- Factor IX Deficiency, Hemophilia B (F9 Exon 7, Rhodesian Ridgeback Variant) (Chromosome X)
- Factor VII Deficiency (F7 Exon 5) (Chromosome 22)
- Factor VIII Deficiency, Hemophilia A (F8 Exon 10, Boxer Variant) (Chromosome X)
- Factor VIII Deficiency, Hemophilia A (F8 Exon 11, Shepherd Variant 1) (Chromosome X)
- Factor VIII Deficiency, Hemophilia A (F8 Exon 1, Shepherd Variant 2) (Chromosome X)
- Thrombopathia (RASGRP2 Exon 5, Basset Hound Variant) (Chromosome 18)
- Thrombopathia (RASGRP2 Exon 8) (Chromosome 18)
- Thrombopathia (RASGRP2 Exon 5, American Eskimo Dog Variant) (Chromosome 18)
- Von Willebrand Disease Type II (VWF Exon 28) (Chromosome 27)
- Von Willebrand Disease Type III (VWF Exon 4) (Chromosome 27)
- Von Willebrand Disease Type I (VWF) (Chromosome 27)
- Canine Leukocyte Adhesion Deficiency Type III (LAD3) (FERMT3) (Chromosome 18)
- Congenital Macrothrombocytopenia (TUBB1 Exon 1, Cavalier King Charles Spaniel Variant) (Chromosome 24)
- Canine Elliptocytosis (SPTB Exon 30) (Chromosome 8)
- Cyclic Neutropenia, Gray Collie Syndrome (AP3B1 Exon 20) (Chromosome 31)
- Glanzmann's Thrombasthenia Type I (ITGA2B Exon 13) (Chromosome 9)
- Glanzmann's Thrombasthenia Type I (ITGA2B Exon 12) (Chromosome 9)
- May-Hegglin Anomaly (MYH9) (Chromosome 10)
- Prekallikrein Deficiency (KLKB1 Exon 8) (Chromosome 16)
- Pyruvate Kinase Deficiency (PKLR Exon 5) (Chromosome 7)
- Pyruvate Kinase Deficiency (PKLR Exon 7 Labrador Variant) (Chromosome 7)
- Pyruvate Kinase Deficiency (PKLR Exon 7 Pug Variant) (Chromosome 7)
- Pyruvate Kinase Deficiency (PKLR Exon 7 Beagle Variant) (Chromosome 7)
- Pyruvate Kinase Deficiency (PKLR Exon 10) (Chromosome 7)
- Trapped Neutrophil Syndrome (VPS13B) (Chromosome 13)
- Ligneous Membranitis (PLG) (Chromosome 1)
- Congenital Hypothyroidism (TPO, Tenterfield Terrier Variant) (Chromosome 17)
- Complement 3 (C3) deficiency (C3) (Chromosome 20)
- Severe Combined Immunodeficiency (PRKDC) (Chromosome 29)
- Severe Combined Immunodeficiency (RAG1) (Chromosome 18)
- X-linked Severe Combined Immunodeficiency (IL2RG Variant 1) (Chromosome X)
- X-linked Severe Combined Immunodeficiency (IL2RG Variant 2) (Chromosome X)
- Progressive Retinal Atrophy - rcd1 Rod-cone dysplasia, rcd1 (PDE6B Exon 21 Irish Setter Variant) (Chromosome 3)
- Progressive Retinal Atrophy Rod-cone dysplasia, rcd1a (PDE6B Exon 21 Sloughi Variant) (Chromosome 3)
- Progressive Retinal Atrophy - CNGA (CNGA1 Exon 9) (Chromosome 13)



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CLEAR CONDITIONS

- Progressive Retinal Atrophy - prcd Progressive rod-cone degeneration (PRCD Exon 1) (Chromosome 9)
- Progressive Retinal Atrophy (CNGB1) (Chromosome 2)
- Progressive Retinal Atrophy (SAG) (Chromosome 25)
- Golden Retriever Progressive Retinal Atrophy 1 (SLC4A3) (Chromosome 37)
- Golden Retriever Progressive Retinal Atrophy 2 (TTC8) (Chromosome 8)
- Progressive Retinal Atrophy - crd1 (PDE6B) (Chromosome 3)
- Progressive Retinal Atrophy - crd2 (IQCB1) (Chromosome 33)
- Progressive Retinal Atrophy - crd4/cord1 (RPGRIP1) (Chromosome 15)
- Collie Eye Anomaly, Choroidal Hypoplasia (NHEJ1) (Chromosome 37)
- Day blindness, Achromatopsia, Cone Degeneration (CNGB3 Exon 6) (Chromosome 29)
- Achromatopsia (CNGA3 Exon 7 German Shepherd Variant) (Chromosome 10)
- Achromatopsia (CNGA3 Exon 7 Labrador Retriever Variant) (Chromosome 10)
- Autosomal Dominant Progressive Retinal Atrophy (RHO) (Chromosome 20)
- Canine Multifocal Retinopathy cmr2 (BEST1 Exon 5) (Chromosome 18)
- Canine Multifocal Retinopathy cmr3 (BEST1 Exon 10 Deletion) (Chromosome 18)
- Canine Multifocal Retinopathy cmr3 (BEST1 Exon 10 SNP) (Chromosome 18)
- Glaucoma Primary Open Angle Glaucoma (ADAMTS10 Exon 9) (Chromosome 20)
- Glaucoma Primary Open Angle Glaucoma (ADAMTS10 Exon 17) (Chromosome 20)
- Glaucoma Primary Open Angle Glaucoma (ADAMTS17 Exon 11) (Chromosome 3)
- Hereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts (HSF4 Exon 9 Boston Terrier Variant) (Chromosome 5)
- Hereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts (HSF4 Exon 9 Shepherd Variant) (Chromosome 5)
- Primary Lens Luxation (ADAMTS17) (Chromosome 3)
- Congenital stationary night blindness (RPE65) (Chromosome 6)
- Macular Corneal Dystrophy (MCD) (CHST6) (Chromosome 5)
- 2,8-Dihydroxyadenine (2,8-DHA) Urolithiasis (APRT) (Chromosome 5)
- Cystinuria Type I-A (SLC3A1) (Chromosome 10)
- Cystinuria Type II-A (SLC3A1) (Chromosome 10)
- Cystinuria Type I-A (SLC7A9) (Chromosome 1)
- Hyperuricosuria and Hyperuricemia or Urolithiasis (SLC2A9) (Chromosome 3)
- Polycystic Kidney Disease (PKD1) (Chromosome 6)
- Primary Hyperoxaluria (AGXT) (Chromosome 25)
- Protein Losing Nephropathy (NPHS1) (Chromosome 1)
- X-Linked Hereditary Nephropathy (Samoyed Variant 2) (COL4A5 Exon 35) (Chromosome X)
- Autosomal Recessive Hereditary Nephropathy, Familial Nephropathy (COL4A4 Exon 3) (Chromosome 25)
- Primary Ciliary Dyskinesia (CCDC39 Exon 3) (Chromosome 34)
- Congenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatitis (CKCSID), Dry Eye Curly Coat Syndrome (FAM83H Exon 5) (Chromosome 13)
- X-linked Ectodermal Dysplasia, Anhidrotic Ectodermal Dysplasia (EDA Intron 8) (Chromosome X)
- Renal Cystadenocarcinoma and Nodular Dermatofibrosis (RCND) (FLCN Exon 7) (Chromosome 5)





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- Canine Fucosidosis (FUCA1) (Chromosome 2)
- Glycogen Storage Disease Type II, Pompe's Disease (GAA) (Chromosome 9)
- Glycogen Storage Disease Type Ia, Von Gierke Disease (G6PC) (Chromosome 9)
- Glycogen Storage Disease Type IIIa (GSD IIIa) (AGL) (Chromosome 6)
- Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A (SGSH Exon 6 Variant 1) (Chromosome 9)
- Mucopolysaccharidosis Type IIIA, Sanfilippo Syndrome Type A (SGSH Exon 6 Variant 2) (Chromosome 9)
- Mucopolysaccharidosis Type VII, Sly Syndrome (GUSB Exon 5) (Chromosome 6)
- Mucopolysaccharidosis Type VII, Sly Syndrome (GUSB Exon 3) (Chromosome 6)
- Glycogen storage disease Type VII, Phosphofructokinase deficiency (PFKM Whippet and English Springer Spaniel Variant) (Chromosome 27)
- Glycogen storage disease Type VII, Phosphofructokinase deficiency (PFKM Wachtelhund Variant) (Chromosome 27)
- Lagotto Storage Disease (ATG4D) (Chromosome 20)
- Neuronal Ceroid Lipofuscinosis 1 (PPT1 Exon 8) (Chromosome 15)
- Neuronal Ceroid Lipofuscinosis 2 (TPP1 Exon 4) (Chromosome 21)
- Neuronal Ceroid Lipofuscinosis 1, Cerebellar Ataxia - NCL-A (ARSG Exon 2) (Chromosome 9)
- Neuronal Ceroid Lipofuscinosis 1 (CLN5 Border Collie Variant) (Chromosome 22)
- Neuronal Ceroid Lipofuscinosis 6 (CLN6 Exon 7) (Chromosome 30)
- Neuronal Ceroid Lipofuscinosis 8 (CLN8 English Setter Variant) (Chromosome 37)
- Neuronal Ceroid Lipofuscinosis (MFSD8) (Chromosome 19)
- Neuronal Ceroid Lipofuscinosis (CLN8 Australian Shepherd Variant) (Chromosome 37)
- Neuronal Ceroid Lipofuscinosis 10 (CTSD Exon 5) (Chromosome 18)
- Neuronal Ceroid Lipofuscinosis (CLN5 Golden Retriever Variant) (Chromosome 22)
- Adult-Onset Neuronal Ceroid Lipofuscinosis (ATP13A2) (Chromosome 2)
- GM1 Gangliosidosis (GLB1 Exon 15 Shiba Inu Variant) (Chromosome 23)
- GM1 Gangliosidosis (GLB1 Exon 15 Alaskan Husky Variant) (Chromosome 23)
- GM1 Gangliosidosis (GLB1 Exon 2) (Chromosome 23)
- GM2 Gangliosidosis (HEXB, Poodle Variant) (Chromosome 2)
- GM2 Gangliosidosis (HEXA) (Chromosome 30)
- Globoid Cell Leukodystrophy, Krabbe disease (GALC Exon 5) (Chromosome 8)
- Autosomal Recessive Amelogenesis Imperfecta (Italian Greyhound Variant) (Chromosome 13)
- Persistent Mullerian Duct Syndrome (AMHR2) (Chromosome 27)
- Alaskan Husky Encephalopathy, Subacute Necrotizing Encephalomyelopathy (SLC19A3) (Chromosome 25)
- Alexander Disease (GFAP) (Chromosome 9)
- Cerebellar Abiotrophy, Neonatal Cerebellar Cortical Degeneration (SPTBN2) (Chromosome 18)
- Cerebellar Ataxia, Progressive Early-Onset Cerebellar Ataxia (SEL1L) (Chromosome 8)
- Cerebellar Hypoplasia (VLDLR) (Chromosome 1)
- Spinocerebellar Ataxia, Late-Onset Ataxia (CAPN1) (Chromosome 18)
- Spinocerebellar Ataxia with Myokymia and/or Seizures (KCNJ10) (Chromosome 38)
- Benign Familial Juvenile Epilepsy, Remitting Focal Epilepsy (LGI2) (Chromosome 3)





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- Fetal-Onset Neonatal Neuroaxonal Dystrophy (MFN2) (Chromosome 2)
- Hypomyelination and Tremors (FNIP2) (Chromosome 15)
- Shaking Puppy Syndrome, X-linked Generalized Tremor Syndrome (PLP) (Chromosome X)
- L-2-Hydroxyglutaricaciduria (L2HGDH) (Chromosome 0)
- Neonatal Encephalopathy with Seizures (NEWS) (ATF2) (Chromosome 36)
- Polyneuropathy, NDRG1 Greyhound Variant (NDRG1 Exon 15) (Chromosome 13)
- Polyneuropathy, NDRG1 Malamute Variant (NDRG1 Exon 4) (Chromosome 13)
- Narcolepsy (HCRTR2 Intron 6) (Chromosome 12)
- Progressive Neuronal Abiotrophy (Canine Multiple System Degeneration) (SERAC1 Exon 15) (Chromosome 1)
- Progressive Neuronal Abiotrophy (Canine Multiple System Degeneration) (SERAC1 Exon 4) (Chromosome 1)
- Juvenile Laryngeal Paralysis and Polyneuropathy, Polyneuropathy with Ocular Abnormalities and Neuronal Vacuolation (POANV) (RAB3GAP1, Rottweiler Variant) (Chromosome 19)
- Hereditary Sensory Autonomic Neuropathy (HSAN), Acral Mutilation Syndrome (GDNF-AS) (Chromosome 4)
- Juvenile-Onset Polyneuropathy, Leonberger Polyneuropathy 1 (LPN1, ARHGEF10) (Chromosome 16)
- Dilated Cardiomyopathy (PDK4) (Chromosome 14)
- Long QT Syndrome (KCNQ1) (Chromosome 18)
- Muscular Dystrophy Cavalier King Charles Spaniel Variant 1 (Chromosome X)
- Muscular Dystrophy Muscular Dystrophy (DMD Pembroke Welsh Corgi Variant) (Chromosome X)
- Muscular Dystrophy Muscular Dystrophy (DMD Golden Retriever Variant) (Chromosome X)
- Centronuclear Myopathy (PTPLA) (Chromosome 2)
- Exercise-Induced Collapse (DNM1) (Chromosome 9)
- Inherited Myopathy of Great Danes (BIN1) (Chromosome 19)
- Myotonia Congenita (CLCN1 Exon 7) (Chromosome 16)
- Myotonia Congenita (CLCN1 Exon 23) (Chromosome 16)
- Myotubular Myopathy 1, X-linked Myotubular Myopathy (MTM1) (Chromosome X)
- Hypocatalasia, Acatlasemia (CAT) (Chromosome 18)
- Pyruvate Dehydrogenase Deficiency (PDP1) (Chromosome 29)
- Malignant Hyperthermia (RYR1) (Chromosome 1)
- Imerslund-Grasbeck Syndrome, Selective Cobalamin Malabsorption (CUBN Exon 53) (Chromosome 2)
- Imerslund-Grasbeck Syndrome, Selective Cobalamin Malabsorption (CUBN Exon 8) (Chromosome 2)
- Congenital Myasthenic Syndrome (CHAT) (Chromosome 28)
- Congenital Myasthenic Syndrome (COLQ) (Chromosome 23)
- Episodic Falling Syndrome (BCAN) (Chromosome 7)
- Dystrophic Epidermolysis Bullosa (COL7A1) (Chromosome 20)
- Ectodermal Dysplasia, Skin Fragility Syndrome (PKP1) (Chromosome 7)
- Ichthyosis, Epidermolytic Hyperkeratosis (KRT10) (Chromosome 9)
- Ichthyosis (PNPLA1) (Chromosome 12)
- Ichthyosis (SLC27A4) (Chromosome 9)
- Focal Non-Epidermolytic Palmoplantar Keratoderma, Pachyonychia Congenita (KRT16) (Chromosome 9)





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- Hereditary Footpad Hyperkeratosis (FAM83G) (Chromosome 5)
- Hereditary Nasal Parakeratosis (SUV39H2) (Chromosome 2)
- Musladin-Lueke Syndrome (ADAMTSL2) (Chromosome 9)
- Cleft Lip and/or Cleft Palate (ADAMTS20) (Chromosome 27)
- Oculoskeletal Dysplasia 1, Dwarfism-Retinal Dysplasia (COL9A3, Labrador Retriever) (Chromosome 24)
- Osteogenesis Imperfecta, Brittle Bone Disease (COL1A2) (Chromosome 14)
- Osteogenesis Imperfecta, Brittle Bone Disease (SERPINH1) (Chromosome 21)
- Osteogenesis Imperfecta, Brittle Bone Disease (COL1A1) (Chromosome 9)
- Osteochondrodysplasia, Skeletal Dwarfism (SLC13A1) (Chromosome 14)
- Skeletal Dysplasia 2 (COL11A2) (Chromosome 12)
- Craniomandibular Osteopathy (CMO) (SLC37A2) (Chromosome 5)