



"FITZ"

THE KEEP HEIR TO PEMBERLY




DNA Test Report

Test Date: June 2nd, 2018

embk.me/fitz12

BREED MIX

 **Pomeranian: 100.0%**

GENETIC STATS

Wolfiness: 0.6 % **LOW**

Predicted adult weight: **9 lbs**

Genetic age: **24 human years**

TEST DETAILS

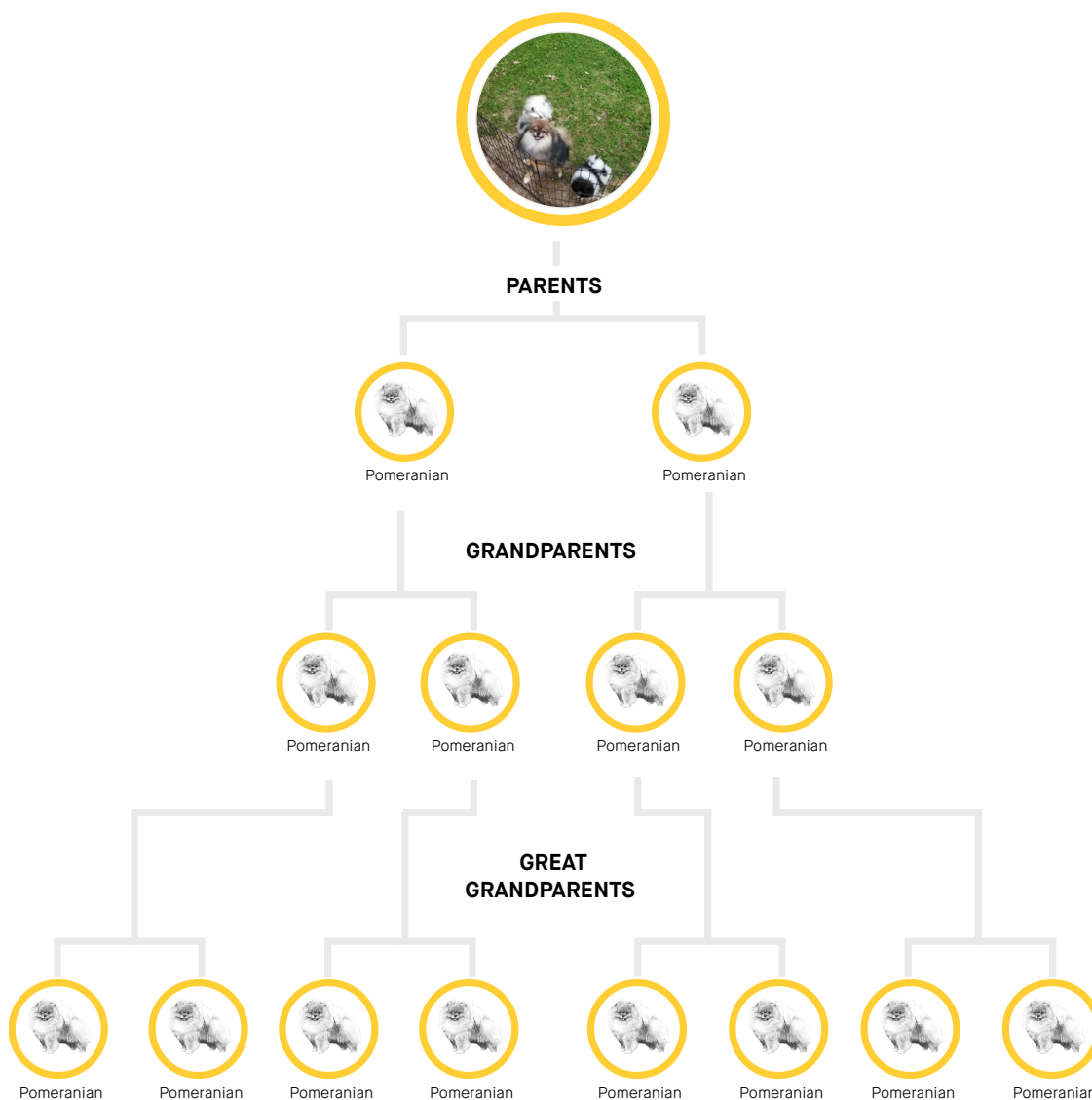
Kit number: EM-3673810

Swab number: 31001801022904



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FAMILY TREE



Our algorithms predict this is the most likely family tree to explain Fitz'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



"FITZ" THE KEEP HEIR TO PEMBERLY

MATERNAL LINE



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

HAPLOGROUP: A1d

This female lineage can be traced back about 15,000 years to some of the original Central Asian wolves that were domesticated into modern dogs. The early females that represent this lineage were likely taken into Eurasia, where they spread rapidly. As a result, many modern breed and village dogs from the Americas, Africa, through Asia and down into Oceania belong to this group! This widespread lineage is not limited to a select few breeds, but the majority of Rottweilers, Afghan Hounds and Wirehaired Pointing Griffons belong to it. It is also the most common female lineage among Papillons, Samoyeds and Jack Russell Terriers. Considering its occurrence in breeds as diverse as Afghan Hounds and Samoyeds, some of this is likely ancient variation. But because of its presence in many modern European breeds, much of its diversity likely can be attributed to much more recent breeding.

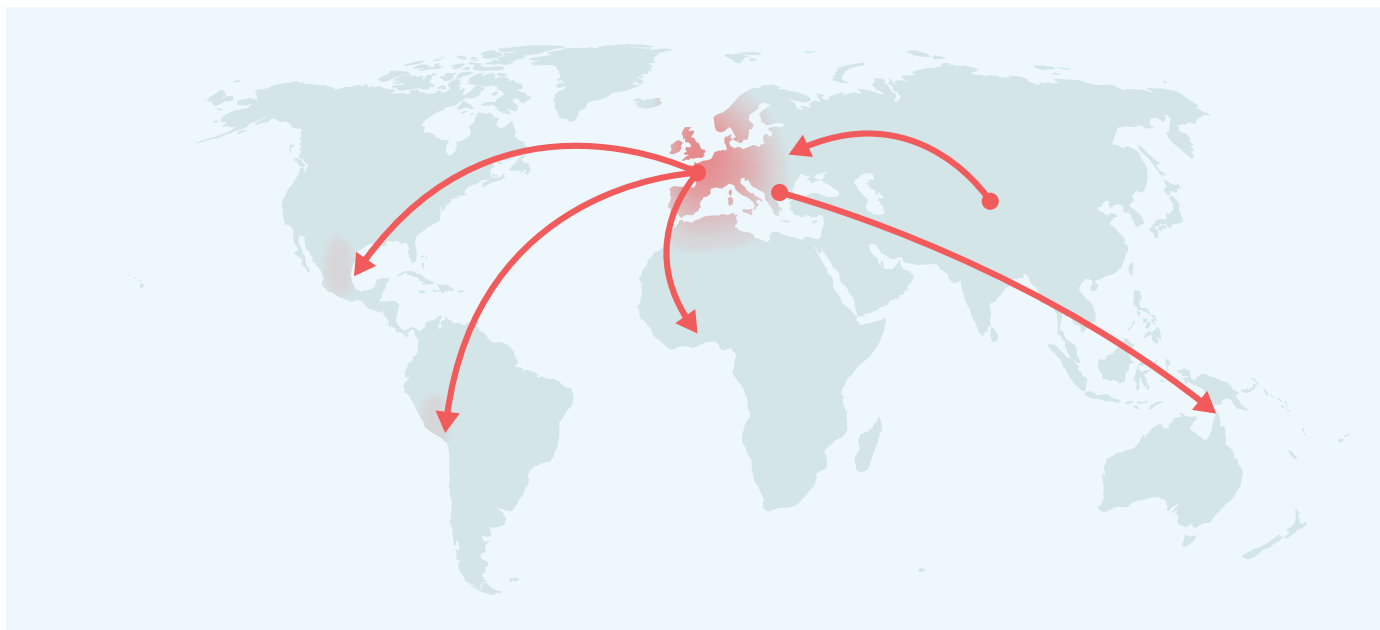
HAPLOTYPE: A247

Part of the large A1d haplogroup, this common haplotype occurs in village dogs all over the world. Among the 32 breeds we have sampled it in, the most common occurrences include Boxers, Labrador Retrievers, and Papillons.



"FITZ" THE KEEP HEIR TO PEMBERLY

PATERNAL LINE



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

HAPLOGROUP: A1a

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

HAPLOTYPE: H1a.1

Part of the large A1a haplogroup, this common haplotype occurs in village dogs all over the world (outside of Asia), with many occurring in Central and South America. We have found this haplotype frequently in Bernese Mountain Dogs, Australian Shepherds, and Boston 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^wa^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

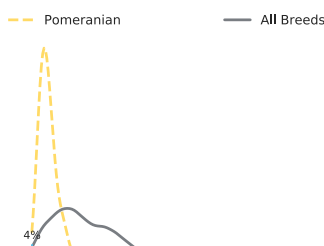
Altitude Adaptation (EPAS1)	GG
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Body Size

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

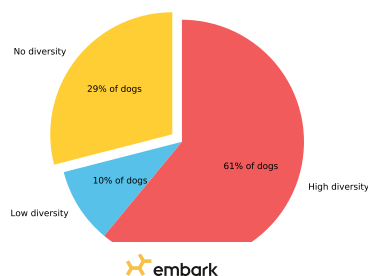
Genetic Diversity

Inbreeding Coefficient **4%**



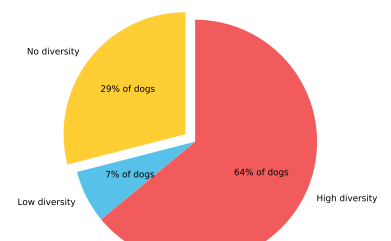
MHC Class II - DLA DRB1

No Diversity



MHC Class II - DLA DQA1 and DQB1

No 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 Heir to Pemberly has one copy of a mutation associated with reduced ALT activity as measured on veterinary blood chemistry panels. Please inform your veterinarian that The Keep Heir to Pemberly has this genotype, as ALT is often used as an indicator of liver health and The Keep Heir to Pemberly is likely to have a lower than average resting ALT activity. As such, an increase in The Keep Heir to Pemberly'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! Fitz did not test positive for any of the genetic diseases that Embark screens for.

0
AT RISK

1
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: **Progressive Retinal Atrophy - prcd Progressive rod-cone degeneration (PRCD Exon 1)**



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PROGRESSIVE RETINAL ATROPHY - PRCD

Progressive rod-cone degeneration (PRCD Exon 1)

Carrier



PRCD Exon 1

GENE NAME

GG

CLEAR

GA

CARRIER

AA

AT RISK

The Keep Heir to Pemberly is a carrier for a mutated allele at PRCD. As a carrier, he or she is unlikely to show any signs of disease. If you choose to breed The Keep Heir to Pemberly, we recommend genotyping any potential mates, and avoiding any matches with other carriers as this could produce puppies at risk for developing PRA.

DESCRIPTION

This retinal disease causes progressive, non-painful vision loss. The retina contains the cells, photoreceptors, that collect information about light: that is, they are the very beginning of how we see. There are two types of photoreceptors: rods, which gather information about light intensity and are the major contributors to night vision, and cones, which distinguish color and are the major contributors to day vision. In nearly all forms of PRA, the rod cells are affected first, leading to night blindness. They are followed by the cone cells, leading to day blindness. The mechanisms by which the photoreceptors degenerate vary depending on the specific mutation that causes PRA. However, the readout is the same: the dog experiences a slow loss of vision, often leading to complete blindness. PRA is a subtle disease: most owners do not even know that their dog has gone blind--you may notice that your dog is reluctant to go down the stairs, or bumping into door frames or corners, or taking a very long time to fetch a ball or toy. A peek at your dog's eyes in bright light may also reveal a sluggish pupillary constriction, because the retina is no longer telling your pupils that it is letting in too much light; however, definitive diagnosis of PRA requires a trip to the vet. Because of the slow progression of PRA, most dogs adapt very well to their condition. Over time, affected dogs can develop cataracts, thought to be due to buildup of reactive oxygen species and other toxic metabolites released from the degenerating retinal cells. This can lead to other complications and requires close monitoring in consultation with your vet.

A late-onset form of PRA resulting from a mutation in the PRCD gene has been observed in many breeds and is inherited in an autosomal recessive manner.

More information

To learn more about this condition, you can visit <http://www.peteducation.com/article.cfm?c=2+2092&aid=343> (<http://www.peteducation.com/article.cfm?c=2+2092&aid=343>).

CITATIONS



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OTHER CONDITIONS

Good news! Fitz tested clear for 3 genetic conditions that are common in his breed.

- Progressive Retinal Atrophy - rcd3
Rod-cone dysplasia, rcd3 (PDE6A)
- Hereditary Vitamin D-Resistant Rickets
(VDR)
- Degenerative Myelopathy
(SOD1A)



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FULL TEST PANEL

Fitz 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 Fitz 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 (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 cmr1 (BEST1 Exon 2) (Chromosome 18)
- 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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CLEAR CONDITIONS

- 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)