

# Certificate of Breed

OWNER'S NAME: Nick Staab DOG'S NAME: "Duke" TEST DATE: April 12th, 2019

Hickory's Point of Pause

This certifies the authenticity of **Duke**'s canine genetic background as determined following careful analysis of more than 200,000 genetic markers.

WOLFINESS 1.5% HIGH

MATERNAL **A414/643** 

**HAPLOTYPE** 

PATERNAL H1a.8/32/43/44

HAPLOTYPE

Good news! Duke did not test positive for any of the genetic diseases that Embark screens for, however Duke is a carrier for 1 of the genetic diseases that Embark tests for. We highly recommend genotyping any potential mates to ensure they are not also carriers as any mating to another carrier of the disease could produce affected puppies.

Welcome to the **Embark** family!

LLEWELLIN SETTER

Adam Boyko, Ph.D.

Ryan Boyko
CHIEF EXECUTIVE OFFICER

O AT RISK

CARRIER





# **GENETIC STATS**

Wolfiness: 1.5 % **HIGH**Predicted adult weight: **64 lbs**Genetic age: **34 human years** 

# **TEST DETAILS**

Kit number: EM-5636849 Swab number: 31001809345972

Registration: FDSB 1664012 ★ embark Microchip: 985 112 005 410 7 74





# **LLEWELLIN SETTER**



The Llewellin Setter is widely cherished as one of the best upland bird hunting dogs. A strain of the English Setter, the Llewellin has a great many devotees and is recognized as its own breed by The American Field Dog Stud Book, the oldest dog registry in the USA. These dogs are well-loved for their hunting prowess in intelligence, stamina, nose, biddability, in addition to its gentle nature. While the history of the English Setter goes back to the 1500s, the Llewellin Setter was developed in a breeding program by R. L. Purcell Llewellin in the 1860s, using dogs from Laverack. These dogs were intended to excel in field work, and Llewellin succeeded in that task. Today, a great number of field-type English Setters have Llewellin blood from the historical crossing of the lines.

Fun Fact When a Llewellin Setter smells a bird nearby, rather than barking, they'll start wagging their tail to alert their owner.

## **RELATED BREEDS**



Gordon Setter
Cousin breed



**Irish Setter**Cousin breed







# **MATERNAL LINE**



Through Duke'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: A1b

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

#### **HAPLOTYPE: A414/643**

Part of the A1b haplogroup, this haplotype occurs most frequently in the English Springer Spaniels.

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# **PATERNAL LINE**



Through Duke'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.8/32/43/44

Part of the A1a haplogroup, the H1a.8/32/43/44 haplotype occurs most commonly in Llewellin Setters, Gordon Setters and German Wirehaired Pointers. We've also spotted it in Southeast Asian Village Dogs, European Village Dogs and East Asian Village Dogs.





# **TRAITS**

# **Coat Color**

E Locus (Mask, Grizzle, Recessive Red) ΕE k<sup>y</sup>k<sup>y</sup> K Locus (Dominant Black) a<sup>t</sup>a<sup>t</sup> A Locus (Agouti, Sable) No Call D Locus (Dilute, Blue, Fawn) B Locus (Brown, Chocolate, Liver, Red, Dudley) bb Saddle Tan П

#### **Other Coat Traits**

Furnishings / Improper Coat (RSPO2)	II
Long Haircoat (FGF5)	TT
Shedding (MC5R)	TT
Curly Coat (KRT71)	CC
Hairlessness (FOXI3)	N/N
Hairlessness (SGK3)	NN
Oculocutaneous Albinism Type 2 - OCA2, Doberman Z Factor Albinism	N/N

(SLC45A2)

# **Other Body Features**

Brachycephaly (BMP3)	CC
Natural Bobtail (T)	CC
Hind Dewclaws (LMBR1)	CC
Blue Eye Color	N/N
Back Muscling & Bulk, Large Breeds	CC
Only	

# Performance

GG Altitude Adaptation (EPAS1)

# **Body Size**

Body Size - IGF1	NN
Body Size - IGF1R	GG
Body Size - STC2	TT
Body Size - GHR (E195K)	GG
Body Size - GHR (P177L)	СС

#### **Genetic Diversity**

Inbreeding Coefficient 20%

**High Diversity** All Breeds

**H**embark

MHC Class II - DLA DRB1

MHC Class II - DLA DQA1 and DQB1

**High Diversity** 



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-- Llewellin Setter





# **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: Normal

Hickory's Point of Pause has two normal alleles at ALT.

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! Duke did not test positive for any of the genetic diseases that Embark screens for.

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ISK 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: Urinary

Condition: Hyperuricosuria and Hyperuricemia or Urolithiasis (SLC2A9)

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### HYPERURICOSURIA AND HYPERURICEMIA OR UROLITHIASIS

(SLC2A9)

Carrier



SLC2A9 (Exon 5) CC CT TT

GENE NAME CLEAR CARRIER AT RISK

Hickory's Point of Pause is a carrier for a mutation in the SLC2A9 gene. As a carrier, he or she is unlikely to develop HUU. If you decide to breed Hickory's Point of Pause, we highly recommend genotyping any potential mates as breeding to another carrier could produce affected puppies.

# **DESCRIPTION**

This condition causes kidney and bladder stones composed of urate; if caught early, it is responsive to dietary management. Uric acid is an intermediate of purine metabolism. In most dogs, uric acid is converted to allantoin, an inert substance that is then excreted in the urine. Dogs with HUU have defects in the pathway that converts uric acid to allantoin. As such, uric acid builds up, crystallizes and forms urate stones in the kidney and bladder. While hyperuricemia in other species (including humans) can lead to painful conditions such as gout, dogs do not develop systemic signs of hyperuricemia. Urate stones are invisible on X-rays and must be diagnosed by a veterinarian via ultrasound or urine sediment analysis. If left undiagnosed, bladder stones can lead to urinary obstruction, which can be life-threatening.

# More information

To learn more about this condition, you can visit http://www.vetbook.org/wiki/dog/index.php?title=Hyperuricosuria (http://www.vetbook.org/wiki/dog/index.php?title=Hyperuricosuria).

# **CITATIONS**

Bannasch et al 2008 (http://www.ncbi.nlm.nih.gov/pubmed/18989453), Karmi et al 2010 (http://www.ncbi.nlm.nih.gov/pubmed/21054540), Donner et al 2016 (http://journals.plos.org/plosone/article? id=10.1371/journal.pone.0161005#pone-0161005-t001)







# **OTHER CONDITIONS**

Good news! Duke tested clear for 6 other common genetic diseases that Embark tests for.

- MDR1 Drug Sensitivity (MDR1)
- Primary Lens Luxation (ADAMTS17)
- Dilated Cardiomyopathy (PDK4)

- Progressive Retinal Atrophy prcd
   Progressive rod-cone degeneration (PRCD Exon 1)
- Degenerative Myelopathy (SOD1A)
- Exercise-Induced Collapse (DNM1)

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# **FULL TEST PANEL**

Duke is also clear of 165 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 Duke tested clear for.

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# CLEAR CONDITIONS

- 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 III (VWF Exon 4) (Chromosome 27)
- Von Willebrand Disease Type I (VWF) (Chromosome 27)
- Von Willebrand Disease Type II (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 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 rcd3 Rod-cone dysplasia, rcd3 (PDE6A) (Chromosome 4)
- Progressive Retinal Atrophy CNGA (CNGA1 Exon 9) (Chromosome 13)
- · Progressive Retinal Atrophy (CNGB1) (Chromosome 2)





# **CLEAR CONDITIONS**

- 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)
- 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)
- Glaucoma Primary Open Angle Glaucoma (ADAMTS17 Exon 2) (Chromosome 3)
- Hereditary Cataracts, Early-Onset Cataracts, Juvenile Cataracts (HSF4 Exon 9 Shepherd Variant) (Chromosome 5)
- 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)
- 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 Dermatosis (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)
- 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)

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# **CLEAR CONDITIONS**

- Mucopolysaccharidosis Type I (IDUA) (Chromosome 3)
- 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)
- Deafness and Vestibular Syndrome of Dobermans (DVDob, DINGS) (Chromosome 21)
- Shar-Pei Autoinflammatory Disease (SPAID, Shar-Pei Fever) (MTBP) (Chromosome 13)
- 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)
- Fetal-Onset Neonatal Neuroaxonal Dystrophy (MFN2) (Chromosome 2)

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# CLEAR CONDITIONS

- 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)
- Spongy Degeneration with Cerebellar Ataxia 1 (SDCA1), SeSAME/EAST (KCNJ10) (Chromosome 38)
- Spongy Degeneration with Cerebellar Ataxia 2 (SDCA2) (ATP1B2) (Chromosome 5)
- · 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)
- Inherited Myopathy of Great Danes (BIN1) (Chromosome 19)
- Myostatin Deficiency, Bully Whippet Syndrome (MSTN) (Chromosome 37)
- 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, Acatalasemia (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)
- Ichthyosis (NIPAL4) (Chromosome 4)

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# **CLEAR CONDITIONS**

- Focal Non-Epidermolytic Palmoplantar Keratoderma, Pachyonychia Congenita (KRT16) (Chromosome 9)
- 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)
- Hereditary Vitamin D-Resistant Rickets (VDR) (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)