



PAWSITIVE I.D.™

INTERPRETIVE GUIDE

Thank you for your purchase of the Pawsitive ID™ genotyping product. This is your interpretive guide to understanding the DNA genetic results listed on the certificate. Pawsitive ID™ gives you genetic information about your pet. This information consists of DNA Fingerprinting for identification and parentage verification purposes, disease screening and color trait testing. Pawsitive ID™ uses the patent pending VeriSNP™ Universal Genetic Evaluation Process to determine all of its genetic markers. The following is your guide to understanding these results:

DNA FINGERPRINTING/PARENTAGE VERIFICATION

The information in this category is your pet's unique DNA fingerprint. Use this information to positively identify your pet. The information presented is a string of letters (A,C,G,T) that represents components of genes at a certain chromosome location. This is a unique string of information that is almost impossible to duplicate in another animal. If you are a breeder and need to verify parentage of puppies/kittens you have for sale, you can use this information to verify parentage if you have previously obtained a DNA fingerprint of the mother and father.

Pawsitive ID™ will keep this DNA fingerprint information in a secure database and will make it available to owners who have their pets in the system if the pet ever becomes lost or legal ownership is ever in question.

Q. What does the A,C,G,T stand for?

A. The A,C,G,T are nucleotides that are detected by the SNP test and are critical components for the designated gene at the particular chromosome location

Q. What does the CF (for dogs) or FC (for cats) and the number next to it mean?

A. CF (for dogs) is Latin for *Cannis Familiaris* and FC (for cats) is Latin for *Felineus Cat*. The number is the Chromosome location number.

DISEASE SCREENING

Pawsitive ID™ can screen for the following inherent, genetic diseases. Although these may be listed as breed specific, it is possible that these diseases can be detected in other breeds.

TEST #01 MYOTONIA CONGENITA

Myotonia Congenita is a disease that is inherited as an autosomal recessive mutation in the canine chloride channel skeletal muscle gene. The gene codes for specific proteins in muscles. When a mutation occurs in the gene, the nature of the amino acid building blocks of specific proteins is modified. Thus, these changes affect the ability of muscles to quickly relax after a voluntary contraction. The delay in skeletal muscle relaxation is not accompanied by cramping or pain to the animal. Affected dogs often have a rigid gate, probably due to excessive growth of the muscles. However, after exercise, the gate improves. The animals may also have an abnormal bark, and superfluous salivation with difficulty in swallowing. There is no known cure for the disorder.

Clinical Signs:

- Muscle stiffness that partially resolves with exercise.
- Increase in muscle stiffness in response to any rapid change in posture.
- Abnormal upper respiratory sounds especially when beginning to move.
- Regurgitation that may occur when eating.
- Protrusion of the tongue from the mouth at rest.
- Compression of the tongue resulting in dimpling that persists for up to 30 seconds.
- Diffuse skeletal muscle hypertrophy.
- Shortening of the lower jaw (mandible).
- Electromyographic (EMG) evidence of myotonic discharges of approximately 40 seconds duration.
- Breeds most often seen with this ailment are the Miniature Schnauzer.

Reference: Rhodes, T. H., Vite, C. H., Giger, U. et al. 1999. A missing mutation in canine C1C-1 causes recessive Myotonia Congenita in the dog, FEBS Lett., Vol. 456, pp. 54-58.

TEST #02 PROGRESSIVE RETINAL ATROPHY

Progressive retinal atrophy, or PRA as it is frequently termed, is a hereditary form of macular degeneration that results in progressive and irremediable loss of visual. In its simplest form, PRA is a disease of the retina. This tissue, located inside the back of the eye, contains specialized cells called photoreceptors that absorb the light focused on them by the eye's lens, and converts that light, through a series of chemical reactions into electrical nerve signals. The nerve signals from the retina are passed by the optic nerve to the brain where they are perceived as vision. The retinal photoreceptors are specialized into rods, for vision in dim light (night vision), and cones for vision in bright light (day and color vision). PRA usually affects the rods initially, and then the cones in later stages of the disease. In human families, the diseases equivalent to PRA (in dogs) are termed retinitis pigmentosa.

The disease is inherited in an autosomal recessive mode.

Different types of PRA are found in different breeds of dogs.

TEST 2-A: The SNP in the gene detected in this test is generally for the Cardigan Welsh Corgi.

TEST 2-B: The SNP in the gene detected in this test is generally for the English Mastiff.

TEST 2-C: The SNP in the gene detected in this test is generally for the Irish Setter.

NOTE: Although the PRA tests offered by Pawsitive I.D. detect defective genes in the above referenced breeds (Test A, B & C), other breeds as noted below can also be screened as well.

Many breeds are affected by one or sometimes more than one form of PRA.

Generalized PRA - early onset: Cardigan Welsh Corgi, Collie (rod-cone Dysplasia type II), Cairn Terrier, Gordon Setter, Great Dane, Irish Setter (rod-cone Dysplasia type I), Miniature Schnauzer (photoreceptor Dysplasia), Norwegian Elkhound (rod Dysplasia, also early retinal degeneration), Tibetan Terrier (progressive rod degeneration causing night blindness only)

Alaskan Malamute - progressive cone degeneration causing hemeralopia (day blindness) - this condition occurs rarely.

Generalized PRA (progressive rod-cone degeneration) - later onset (usually older than 1 year): Akita, Australian Cattle Dog, Australian Shepherd, American and English Cocker Spaniel, Basenji, Beagle, Belgian Sheepdog, Briard, Brittany Spaniel, Chesapeake Bay Retriever, Collie (rough and smooth), Dachshund, English Springer Spaniel, German Shepherd, German Short-Haired Pointer, Golden Retriever, Greyhound (without typical initial night blindness), Irish Setter, Labrador Retriever, Mastiff, Nova Scotia Duck Tolling Retriever, Old English Sheepdog, Papillon, Pekingese, Poodle (miniature and toy), Portuguese Water Dog, Rottweiler, Samoyed, Shetland Sheepdog, Shih Tzu, Siberian Husky, Tibetan Spaniel, Tibetan Terrier, Welsh Springer Spaniel, Yorkshire Terrier.

Central PRA - retinal pigment epithelial dystrophy (RPED): This disease occurs mostly in dogs in the United Kingdom, of the following breeds: Border Collie, Cardigan Welsh Corgi, English Cocker Spaniel, English Springer Spaniel, Golden Retriever, Labrador Retriever, Rough and Smooth Collie, Shetland Sheepdog.

Retinal degeneration in the Borzoi: Unlike other forms of PRA, the eyes are affected asymmetrically and the retinal lesions appear inflammatory. Males are affected more often than females. Ultimately, the ophthalmoscopic lesions are similar to those of PRA.

Reference: Suber ML, Pittler SJ, Qin N, Wright GC, Holcombe V, Lee RH, Craft CM, Lolley N, Baehr W, Hurwitz RL. 1993. Irish setter dogs affected with rod/cone Dysplasia contain a nonsense mutation in the rod cGMP phosphodiesterase β -subunit gene. PNAS USA 90:3968-3972.

Ray K, Baldwin VJ, Acland GM, Blanton SH, Aguirre GD. 1994. Cosegregation of codon 807 mutation of the canine rod cGMP phosphodiesterase β gene and rcd1. Inv Ophthalmol Vis Sci 35(13):4291-4299.

Petersen-Jones S.M., Entz, D.D. and Sargan, D. R. 1999. cGMP Phosphodiesterase- Mutation Causes Progressive Retinal Atrophy in the Cardigan Welsh Corgi Dog. Investigative Ophthalmology and Visual Science. 40:1637-1644.

Kijas, J.W., Miller, B.J., Pearce-Kelling, S.E., Aguirre, G.D., and Acland, G.M. 2003. Canine Models of Ocular Disease: Outcross Breedings Define a Dominant Disorder Present in the English Mastiff and Bull Mastiff Dog Breeds. J. Heredity 94: 27-30.

TEST #03 CONGENITAL HYPOTHYROIDISM WITH GOITER

Hypothyroidism refers to any state in which thyroid hormone production is below normal. Thyroid hormones have an effect on virtually every organ system in the body. There are many disorders that result in hypothyroidism. The disease is

transmitted as an autosomal recessive gene. The genetic or congenital hypothyroidism may exist in a carrier state in as many as 30% of normal Toy Fox Terriers. About 1 in 4 members of a litter are affected if both parents are carriers.

Symptoms of the disease usually are manifest as lethargy, mental dullness, inadequate wound healing, inferior skin and hair coat, including hair loss, increased appetite leading to obesity, cold intolerance (pups try to find warm places), or altered pigmentation. A lump (goiter) in the neck increases with age, even with treatment. The normal process of bone lengthening is delayed and is seen in the spine, legs and muzzle. Usually, affected animals die by the age of 3 or 4 weeks.

Treatment in standard cases of hypothyroidism consists by supplementing the daily diet with a thyroid hormone called L-thyroxine. However, treatment for congenital hypothyroidism in Toy Fox Terriers is usually not pursued because it is not worthwhile.

Congenital hypothyroidism has also been reported in several breeds, including Scottish Deerhounds, Bullmastiffs, Boxers, German Shepherds, and Giant Schnauzers. It has also been seen in crossbreeds.

Reference: Dixon, R. 2001. Recent developments in the diagnosis of canine hypothyroidism In Practice 23: 328-335.

TEST #04 CANINE LEUKOCYTE ADHESION DEFICIENCY

Canine Leukocyte Adhesion Deficiency (CLAD) is rare but fatal disease inherited as an autosomal recessive trait. CLAD is an abnormality of the immune system where the white blood cells are unable to fight infection. Animals that are affected display early and serious infections and usually die early in life. This disease was first identified in 1975 in Irish Setters. Several animals displaying various forms of recurrent infectious and immunological complications were found to have an aberrant expression of the CD18 molecule.

The identification of a single nucleotide polymorphism (SNP) gene mutation responsible for the disease in Irish Setters has been determined by mutational analysis of CD18 in Irish Setter CLAD pedigrees. This single missing mutation showed complete association with CLAD in Irish Setters. This mutation is thought to be responsible for incomplete disulphide bonding within the β -integrin protein, causing defects in its function and hence impaired immune function.

Reference: Jobling AI, Ryan J, Augusteyn RC. 2003. The frequency of the canine leukocyte adhesion deficiency (CLAD) allele within the Irish Setter population of Australia. Aust Vet J. 81(12):763-5.

TEST #05 NEURONAL CEROID LIPOFUSCINOSIS

Neuronal Ceroid Lipofuscinosis NCL, (also known as Batten Disease) is the general name for a family of at least eight genetically separate neurodegenerative disorders that result from excessive accumulation of fats and proteins known as lipopigments in the body's tissues. The lipopigments build up in cells of the brain and the eye as well as in skin, muscle, and many other tissues and assume a greenish-yellow color when viewed under ultraviolet light. The end result is a progressive neurological disease including blindness. At the present, there is no known cure. This disease manifests itself as a progressive dementia that resembles Alzheimer's symptoms. For example, some animals exhibit aggressive behavior, confusion, and problems adapting to environmental changes.

This disease is generally inherited as an autosomal recessive gene.

TEST 5-A: SNP test CLN 2 is primarily found in Dachshunds.

TEST 5-B: SNP test CLN 8 is primarily found in English Setters. In setters, motor irregularities are observed as the disease progresses. Additionally, hyperactivity followed by violent behavior occurs when the animal is 15 to 24 months old. Most likely, death occurs by the time the dog is around three years old.

TEST 5-C: SNP test CLN 5 is primarily found in Border Collies. In Collies, motor irregularities are observed as the disease progresses. Additionally, hyperactivity followed by violent behavior occurs when the animal is 15 to 24 months old.

Reference: Drögemüller, C., Wöhlke, A., and Distl, O. 2005. Characterization of Candidate Genes for Neuronal Ceroid Lipofuscinosis in Dog. Journal of Heredity 96(7):735-738.

TEST #06 CYSTINURIA

Cystinuria is a disorder characterized by stones in the kidney, ureter, and bladder. It is caused by excessive excretion of certain amino acids (protein building blocks – specifically cystine, lysine, arginine and ornithine) due to a genetic abnormality that affects the renal tubular transport of these compounds.

The filtering action of the kidney fails to remove the excessive amino acids and they tend to form crystals, or stones, in the urine. These stones can block the urethra, especially in males, and impede the urinary stream. It generally takes a few years for the symptoms to appear.

The disease is inherited as an autosomal recessive gene.

The SNP test is primarily for the Newfoundland dog breed.

Reference: Henthorn PS, Liu J, Gidalevich T, Fang J, Casal ML, Patterson DF, Giger U. 2000. Canine Cystinuria: polymorphism in the canine SLC3A1 gene and identification of a nonsense mutation in cystinuric Newfoundland dogs. *Hum Genet* 107: 295-303.

TEST #07 NARCOLEPSY

Narcolepsy is a chronic, neurological sleep disorder characterized by uncontrollable sleep attacks that result in excessive sleepiness. These sleep attacks usually occur multiple times a day even when an animal gets adequate sleep. Other symptoms include sleep paralysis, cataplexy, and hypnologic hallucinations.

The disease gene is inherited as an autosomal recessive mode.

The SNP test for this disease is primarily found in the Dachshund breed although it may target the same disease in Doberman Pinschers, Poodles and Labrador Retrievers

Reference: Marcel Hungs, Jun Fan, Ling Lin, Xiaoyan Lin, Richard A. Maki and Emmanuel Mignot. 2001. Hypocretin (Orexin) Genes of Narcoleptic Canines: Identification and Functional Analysis of Mutations in the Hypocretin (Orexin) Genes of Narcoleptic Canines. *Genome Res.* 11: 531-539.

TEST #08 MUSCULAR DYSTROPHY

Canine Muscular dystrophy is a broad term that describes an inherited disorder of the muscles. The most common form of MD is called Duchenne muscular dystrophy (DMD). MD causes the muscles in the body to become very weak with degeneration and sporadic contractures. The muscles ultimately break down and are replaced with fatty deposits. When a mutation occurs in the dystrophin gene, the one that codes for a muscle cell membrane protein called dystrophin, the disruption causes a break in the mechanical links that work together to stabilize the muscle. This gene is located on the X chromosome.

Clinical signs first appear at 6-9 weeks of age in the form of weakness and atrophy of the muscles. However, an early diagnostic can be administered as early as 2 days of age. A blood sample is taken and serum creatine kinase (CK) levels are checked for strikingly elevated values. Likewise, a Single Nucleotide Polymorphism (SNP) test can be used to identify dogs that carry the mutant X chromosome.

This disease is inherited in an X-linked mode and is primarily found in the Golden Retriever.

Reference: Brumitt, J. W., Essman, S. C., Kornegay, J. N., Graham, J. P., Weber, W. J. & Berry, C. R. 2006. Radiographic Features of Golden Retriever Muscular Dystrophy. *Veterinary Radiology & Ultrasound* 47 (6), 574-580.

Kornegay, J.N., Tuler S.M., Miller D.M. et al. 1998. Muscular dystrophy in a litter of Golden retriever dogs. *Muscle and Nerve*, New York, v.11, n.10, p.1056-1064.

Nguyen, F., ChereL, Y., Guigand, L., Goubault-Leroux, I., Wyers, M. 2002. Muscle lesions associated with dystrophin deficiency in neonatal golden retriever puppies, *Journal of Comparative Pathology* 126:100-108.

Valentine, B.A., Cooper, B.J., Cummings, J.F., Delahunta, A. 1990. Canine X-Linked Muscular Dystrophy - Morphologic Lesions, *Journal of the Neurological Sciences* 97:1-23.

TEST #09 GLOBOID CELL LEUCODYSTROPHY

This disease is often called Krabbe disease (also known as globoid cell leukodystrophy or galactosylceramide lipidosis or GCL) and is a rare, often fatal degenerative disorder that affects the brain and nervous system. As part of a group of disorders known as leukodystrophies, Krabbe disease results from the imperfect growth and development of myelin. GCL disease is caused by mutations in the GALC gene, which causes a deficiency of a critical enzyme. The official name of the GALC gene is "galactosylceramidase but is often called -galactosidase. The buildup of undigested fats affects the β galactocerebroside growth of the nerve's protective myelin sheath (the covering that insulates many nerves) and causes severe degeneration of mental and motor skills.

The disease is manifest as general muscle weakness, awkward gait, lack of coordination, deficiency of hindquarter control and tail tremors.

The disease is most often found in West Highland White and Cairn Terriers.

This condition is inherited as an autosomal recessive pattern.

Reference: Cozzi F, Vite CH, Wenger DA, Victoria T, Haskins ME. 1998. MRI and electrophysiological abnormalities in a case of canine globoid cell leukodystrophy. *J Small Animal Practice* 39: 401-405.

TEST #10 VON WILLEBRAND DISEASE

Von Willebrand Disease (vWD) is a common genetic bleeding disorder that can occur in dogs. In fact, it is not a single disease, but a family of related diseases of variable severity. All the different types in humans and in dogs are caused by a problem with the Von Willebrand Factor (vWF). This is a protein in blood which is necessary for proper blood coagulation, or clotting. When there is not enough of the protein in the blood, bleeding can be uncontrolled and sometimes life threatening. Symptoms can include undue bleeding of the umbilical cord at birth, extended bleeding at the time of tail docking, blood in the urine, or swelling in various body parts. Not all animals show clinical symptoms

The disease is inherited in an autosomal recessive mode.

Different types of vWD are found in different breeds of dogs.

TEST 10-A: The SNP gene detected in this test is generally for the German Shorthaired Pointer breed.

TEST 10-B: The SNP gene detected in this test is generally for the Dutch Kooiker breed and is of the Type III von Willebrand Disease.

Reference: van Dongen AM, van Leeuwen M, Slappendel RJ. 2001. Canine von Willebrand's disease type 2 in German wirehair pointers in the Netherlands. *Vet Rec.* 148(3):80-2.

Slappendel RJ, Beijer EG, van Leeuwen M. 1998. Type III von Willebrand's disease in Dutch kooiker dogs. *Vet Q.* 20(3):93-7.

Venta PJ, Li J, Yuzbasiyan-Gurkan V, Brewer GJ, Schall WD. 2000. Mutation causing von Willebrand's disease in Scottish Terriers. *J Vet Intern Med.* 14(1):10-9.

TEST #11 GM1 GANGLIOSIDOSIS

GM1 Gangliosidosis is a genetic lipid storage disorder that is similar in certain respects to Hurler syndrome and Tay-Sachs disease in humans. GM1 Gangliosidosis is one of the classic lipid storage diseases. It affects both the brain and the viscera (the internal organs) of the dog. GM1 Gangliosidosis causes skeletal deformities and imparts severe effects on the brain and internal organs. Death usually occurs by the age of 2. The gene responsible for it maps to chromosome 23 in the dog. There is no treatment for the disease. It is also known as familial neurovisceral lipidosis or Landing disease in the human.

Ganglioside storage diseases are defects of lysosomal hydrolase enzymes that result in accumulation of gangliosides (glycosphingolipids that are major constituents of plasma membranes in a variety of cells, especially neurons) and glycolipid substrates of these hydrolases within lysosomes of most neurons throughout the nervous system, including brain, spinal cord, and autonomic ganglia. The disease has been described in a variety of species, including cats, dogs, cattle, sheep, mice, and humans. In dogs, the accumulation of ganglioside in the brain is due to deficiency of acid beta-galactosidase. In most of the gangliosidosis, total ganglioside content of the brain is high in clinically affected animals. Asialo (sialic acid free) derivatives of the gangliosides also accumulate in the brain and liver. High levels of other neutral glycosphingolipids may also be found. In some instances, different substrates are stored in neural and visceral tissues, probably reflecting the heterocatalytic activity of the deficient enzyme. In affected Portuguese Water dogs, clinical signs of GM1 Gangliosidosis are typically observed at around 4 to 5 months of age. In most cases, the disease is clinically manifested as a neurodegenerative disorder. Common signs of disease include vision problems, lethargy, difficulty walking, and death in a period of approximately 8 months to 1 year of age.

The disease is inherited in an autosomal recessive mode.

TEST 11-A: The SNP gene detected in this test is generally for the Portuguese Water Dog.

TEST 11-B: The SNP gene detected in this test is generally for the Shiba Inu breed.

Reference: Wang, Z. H.; Zeng, B.; Shibuya, H.; Johnson, G. S.; Alroy, J.; Pastores, G. M.; Raghavan, S.; Kolodny, E. H. 2000. Isolation and characterization of the normal canine beta-galactosidase gene and its mutation in a dog model of GM1-gangliosidosis. *J. Inherit. Metab. Dis.* 23: 593-606, 2000.

Yamato O, Ochiai K, Masuoka Y, Hayashida E, Tajima M, Omae S, Iijima M, Umemura T, Maede Y. 2000. GM1 gangliosidosis in shiba dogs. *Vet Rec.* 146(17):493-6.

TEST #12 MUCOPOLYSACCHARIDOSIS TYPE VII [Dogs]

MPS VII is one of the least common forms of the mucopolysaccharidoses. The disorder is caused by deficiency of the enzyme beta-glucuronidase (GUSB). In its rarest form MPS VII causes puppies to be born with hydrops fetalis, in which extreme amounts of fluid are retained in the body. Neurological symptoms may include mild to moderate mental retardation, communicating hydrocephalus, nerve entrapment, corneal clouding, and some loss of peripheral and night vision. Other

symptoms include short stature, some skeletal irregularities, joint stiffness and restricted movement, and umbilical and/or inguinal hernias.

The disease is inherited as an autosomal recessive lysosomal storage disorder.

The SNP test for this disease is directed to the gene that codes for the enzyme b-glucuronidase. Dog breeds most affected by this disorder are the German Shepard as well as many types of Mixed Breed dogs. Many cat breeds affected by Type VI & VI MILD.

Reference: Ponder, K. P., Melniczek, J. R., Xu, L. et al. 2002. Therapeutic neonatal hepatic gene therapy in mucopolysaccharidosis VII dogs. Proc. Natl. Acad. Sci. USA, Vol. 99:13102–13107.

Ray J, Scarpino V, Laing C, Haskins ME. 1999. Biochemical basis of the beta-glucuronidase gene defect causing canine mucopolysaccharidosis VII. J Hered. 90(1):119-23.

TEST #13 HEMOPHILIA B

Hemophilia B is a hereditary blood coagulation disorder. It is caused by a deficiency of a blood plasma protein called factor IX that affects the clotting property of blood. Dogs that have this disorder bleed very easily, as after customary vaccinations. They often have bloody diarrhea and/or lameness.

The disease is inherited as X-linked, meaning that the male dog (carries a Y and an X chromosome) is either clear or affected – he cannot be a carrier. However, the female dog carries two X chromosomes and can be homozygous for the disease on both X chromosomes and thus, affected; or she can carry the disease on one X chromosome and show no clinical signs; or can be clear of the disease and have no point mutations that code for the disease on the X chromosomes.

The disorder does not appear to be breed specific although it is frequently found in the Labrador Retriever, Lhasa Apso, and various terriers.

Reference: Chao, H.J., Walsh, C.E. 1999. Endogenous canine FIX antigen exists in Chapel Hill strain hemophilia B canine. Thrombosis & Haemostasis 82:1378.

Gu, W.K., Brooks, M., Catalfamo, J., Ray, J., Ray, K. 1999. Two distinct mutations cause severe hemophilia B in two unrelated canine pedigrees, Thrombosis & Haemostasis 82:1270-1275.

TEST #14 PHOSPHOFRUCTOKINASE DEFICIENCY

The enzyme PFK is important in energy metabolism in red blood cells and in skeletal muscle during intense exercise. Canine Phosphofructokinase Deficiency is an inherited disorder that causes premature breakdown (hemolysis) of red blood cells, and a reduced tolerance for exercise. Affected dogs have chronic mild anemia with intermittent bouts of acute hemolysis, often associated with intense exercise, overheating or prolonged barking. Affected dogs have a persistent mild anemia (low level of red blood cells) for which they are generally able to compensate. Intermittently, they will have acute episodes of red blood cell breakdown when they become lethargic and weak. This is usually associated with intense exercise or excessive barking or panting. Their mucous membranes (e.g. gums) are pale or jaundiced and they usually run a high fever. You may notice the urine is brown due to the excretion of blood breakdown products. At these times, your dog will require veterinary attention.

Commonly affected breeds are Cocker Spaniel, English Springer Spaniel.

TEST #15 SEVERE COMBINED IMMUNODEFICIENCY DISEASE

Severe combined immunodeficiency, or SCID, is a genetic disorder in which both B cells and T cells of the adaptive immune system are crippled, due to a defect in one of several possible genes. SCID is a severe form of heritable immunodeficiency. It is also known as the "bubble boy" disease because its victims are extremely vulnerable to infectious diseases and must be kept isolated from others.

The disease is transmitted in an X-linked recessive pattern. Thus, only males are affected and show symptoms of the disease. As in other X-linked genetic diseases, females may be carriers of the trait. Therefore, one-half of her male puppies will have the disease gene. After weaning, and thus being deprived of the mother's immunoprotective milk, pups become afflicted with physical problems such as respiratory infections, ear and skin infections and diarrhea. None of these conditions responds well to antibiotic therapy. There is no known cure for this disease.

TEST 15-A: The SNP test for this gene is primarily found in the Cardigan Welsh Terrier.

TEST 15-B: The SNP test for this gene is primarily found in the Basset Hound.

TEST 15-C: The SNP test for this gene is primarily found in the Jack Russell Terrier.

Reference: Felsburg, P.J. 1992. Primary immunodeficiencies. In J.D. Bonagura and R.W. Kirk (eds) Kirk's Current Veterinary Therapy XI Small Animal Practice p. 448-453. W.B. Saunders Co., Toronto.

Henthorn, P. S., Somberg, R.L. Fimiani, V.M., Puck, J.M., Patterson, D.F. gamma gene micro deletion demonstrates that canine Felsburg, P.J. 1994. IL-2R X-linked severe combined immunodeficiency is a homologue of the human disease. *Genomics* 23(1), 69–74.

Bell, T.G., Butler K.L, Sill, H.B., Stickle, J. E., Ramos-Vara, J.A., Dark, M. J. 2002. Autosomal recessive severe combined immunodeficiency of Jack Russell Terriers. *J Vet Diagn Invest* 14:194–204

TEST #16 THROMBASTHENIC THROMBOPATHIA

Thrombopathia means a disorder of small blood cells called platelets or thrombocytes. Platelets play an important role at several stages of the body's response to any injury that causes bleeding. One function of platelets is to aggregate or "clump" at the site of blood vessel injury to form an initial plug. Platelets also facilitate blood clotting, in conjunction with the clotting factors, and release substances active in inflammation and tissue repair. Affected animals have lengthened times of bleeding after injury or blood drawing. The animals often develop deep bruises at the site of an injury.

The SNP test for this disorder detects changes in the gene that codes for a glycoprotein that is part of the platelet. There is no explicit procedure to treat this disorder. However, plasma from normal donor dogs can help acute bleeding. Also, if the animal is anemic, a transfusion of whole blood may be given.

The disease is most often seen in Otterhounds, although it has been detected in the Great Pyrenees breed.

This disease is inherited in an autosomal recessive mode.

Reference: Boudreaux MK and Catalfamo JL. 2001. Molecular and genetic basis for thrombasthenic thrombopathia in Otterhounds. *Am J Vet Res* 62(11):1797-1804.

TEST #17 CONE DEGENERATION

Cone Degeneration (CD) disease causes day blindness due to degeneration of the retinal "cones" – cone-shaped cells in the retina that respond primarily to bright daylight. CD can be diagnosed in the early weeks of the German Shorthaired Pointer's life. Between 8 and 12 weeks of age, when retinal development is normally completed in dogs, signs of vision problems are noticeable. The pups become day-blind and are photophobic – meaning that exposure to bright light is irritating or even painful. The pup will shun brightly-lit areas. Vision in dim light remains normal, in contrast to PRA affected dogs which is the more common type of retinal disease. The retina of the affected dog initially appears normal when examined by an ophthalmologist and initially the ERG (electroretinogram) recording is normal. However, the ERG response from the degenerating cones declines with age and is non-recordable in the mature CD-affected dog. There is no treatment or cure available for either canine or human disease.

The disease is autosomal recessively inherited

The dog most affected by CNGB is the German Shorthaired Pointer.

Reference: Aguirre G.D., Rubin L.F. 1977. Postnatal development of the retina in Alaskan malamute dogs with inherited cone degeneration. *Proc Am Coll Vet Ophthalmol* 8:51.

Akhmedov, N.B., Piriev, N.I., Ray, K., Acland, G.M., Aguirre, G.D. and Farber, D.B. 1997. Structure and analysis of the transducin beta3-subunit gene, a candidate for inherited cone degeneration (cd) in the dog. *Gene*, 194, 47–56.

Akhmedov, N.B., Piriev, N.I. 1998. Pearce-Kelling, S., Acland, G.M., Aguirre, G.D. and Farber, D.B. Canine cone transducin-gamma gene and cone degeneration in the cd dog. *Invest. Ophthalmol. Vis. Sci.*, 39, 1775–1781.

Sidjanin DJ, Lowe JK, McElwee JL, Milne BS, Phippen TM, Sargan DR, Aguirre GD, Acland GM, Ostrander EA. 2002. Canine CNGB3 mutations establish cone degeneration as orthologous to the human achromatopsia locus ACHM3. *Hum Mol Genet* 11(16):1823-1833.

TEST #18 RETINAL DYSTROPHY

Mutations of genes encoding various retina-specific proteins are known to cause a wide spectrum of inherited retinal dystrophies in different species. In the canine, several types of genetic retinal dystrophies have been described affecting primarily the photoreceptors and/or the retinal pigment epithelium. Retinal tissue, located inside the back of the eye, contains specialized cells called photoreceptors that absorb the light focused on them by the eye's lens, and converts that light, through a series of chemical reactions into electrical nerve signals. The nerve signals from the retina are passed by the optic nerve to the brain where they are perceived as vision. The retinal photoreceptors are specialized into rods, for vision in dim light (night vision), and cones for vision in bright light (day and color vision). With Retinal Dystrophy the abnormal development of the retina usually affects the rods initially, and then cones in later stages of the disease. This disease is very closely related to various Progressive Retinal Atrophy disorders.

The disease has been observed in the Briard breed, Cavalier King Charles Spaniels, Irish Setters, Yorkshire Terriers, Doberman Pinschers, Golden Retrievers and in the American Cocker Spaniel.

While there is no known cure for the disease, studies with laboratory animals such as mice suggest that there may be beneficial results if the animal is kept in a reduced light situation and that detection of the gene defect be accomplished as early as possible.

This disease is inherited in an autosomal recessive mode.

Reference: Narfström K, Wrigstad A, Ekesten B, Nilsson SEG. 1994. Hereditary retinal dystrophy in the Briard dog: Clinical and hereditary characteristics. *Prog Vet Comp Ophthalmol* 4: 85-92.

Veske A, Narfström K, Finckh U, Sargan DR, Nilsson SE, Gal A. 1997. Isolation of canine retinal arrestin cDNA and exclusion of three candidate genes for Swedish Briard retinal dystrophy. *Curr Eye Res.* 16(3):270-4.

TEST #19 PROGRESSIVE ROD-CONE DEGENERATION (PRCD)

The genetic disorder, prcd-PRA, causes cells in the retina at the back of the eye to degenerate and die, even though the cells seem to develop normally early in life. The “rod” cells operate in low light levels and are the first to lose normal function. Night blindness results. Then the “cone” cells gradually lose their normal function in full light situations. Most affected dogs will eventually be blind. Typically, the clinical disease is recognized first in early adolescence or early adulthood. Since age at onset of disease varies among breeds, you should read specific information for your dog. Diagnosis of retinal disease can be difficult. Conditions that seem to be prcd-PRA might instead be another disease and might not be inherited.

Prdc-PRA is inherited as a recessive trait. This means a disease gene must be inherited from each parent in order to cause disease in an offspring. Parents were either “carrier” or affected. A carrier has one disease gene and one normal gene, and is termed “heterozygous” for the disease. A normal dog has no disease gene and is termed negative – both copies of the gene are the same. And a dog with two disease genes is termed positive – both copies of the gene are abnormal.

Unfortunately, at this time there is no treatment or cure for PRA. If your dog is affected, you may find it helpful to read about other owners’ experiences living with blind dogs. (suggested links: www.eyevet.org and www.blinddogs.com).

Commonly affected breeds are the American Cocker Spaniel, American Eskimo Dog, Australian Cattle Dog, Australian Shepherd, Australian Shepherd, Miniature, Australian Stumpy Tail Cattle Dog, Chesapeake Bay Retriever, Chinese Crested, Cockapoo, Dwarf Poodle, English Cocker Spaniel, Entlebucher Mountain Dog, Finnish Lapphund, Golden Retriever, Golden Doodle, Karelian Bear Dog, Kuvasz, Labradoodle, Australian Labradoodle, Labrador Retriever, Laponian Herder, Miniature & Toy Poodle, Norwegian Elkhound, Nova Scotia Duck Tolling Retriever, Portuguese Water Dog, Spanish Water Dog, Swedish Lapphund, Yorkshire Terrier.

TEST #20 NEONATAL ENCEPHALOPATHY (NEWS)

Neonatal encephalopathy with seizures (NEWS) is a previously undescribed autosomal recessive disease of standard poodle puppies. Affected puppies are small and weak at birth. Many die in their first week of life. Those surviving past 1 week develop ataxia, a whole-body tremor, and, by 4 to 6 weeks of age, severe generalized clonic-tonic seizures. None have survived to 7 weeks of age. Cerebella from affected puppies were reduced in size and often contained dysplastic foci consisting of clusters of intermixed granule and Purkinje neurons.

TEST #21 GLYCOGEN STORAGE DISEASE ia

Of the 3 glycogen storage diseases reported in dogs, types I and III directly affect the liver. In general, glycogen storage diseases are caused by a deficiency of certain enzymes and result in failure of glycogen to be released from the cell. Therefore, glycogen accumulates within the liver and other organs and is unavailable for conversion to glucose. Type ia glycogen storage disease results from a deficiency in glucose-6-phosphatase and has been reported in toy-breed dogs. Type III is caused by a deficiency in amylo-1,6-glucosidase and has been reported in German Shepherds. Clinical signs for both include hepatomegaly, retarded growth, and weakness and depression due to hypoglycemia. Enzyme analysis of fresh frozen samples of liver, muscle, or skin is needed for diagnosis. Treatment is symptomatic and includes frequent small meals of high-carbohydrate food. Prognosis is poor, and most dogs succumb to these diseases at a young age.

Commonly affected breeds: Maltese, Beagle, German Shepherd and Lapland Dogs.

TEST #22 L-2-HYDROXYGLUTARIC ACIDURIA

l-2-Hydroxyglutaric Aciduria (l-2-HGA) is a neurometabolic disorder that produces a variety of clinical neurological deficits, including psychomotor retardation, seizures and ataxia. The biochemical hallmark of l-2-HGA is the accumulation of l-2-hydroxyglutaric acid (l-2-HG) in cerebrospinal fluid, plasma and urine. Mutations within the gene L2HGDH (Entrez Gene ID 79944) on chromosome 14q22 encoding L-2-hydroxyglutaric acid dehydrogenase have recently been shown to cause l-2-

HGA in humans. Using a candidate gene approach in an out bred pet dog population segregating 1-2-HGA; the causal molecular defect was identified in the canine homologue of L2HGDH and characterized. DNA sequencing and pedigree analysis indicate a common founder effect in the canine model. The canine model shares many of the clinical and MRI features of the disease in humans and represents a valuable resource as a spontaneous model of 1-2-HGA.

Commonly affected breeds are Staffordshire Bull Terriers.

TEST #23 MDR1 MULTI DRUG RESISTANCE 1

Some breeds of dogs are more sensitive to certain drugs compared to other breeds. For example, Collies, Australian Shepherds and other breeds are often more sensitive to the antiparasitic drug, Ivermectin. It is well known that Collies and related breeds can have adverse reactions to drugs such as Ivermectin, Loperamide (Imodium®), and others. It was previously unknown why some individual dogs were sensitive and others were not. The problem is due to a mutation in the multi-drug resistance gene (MDR1). This gene encodes a protein, P-glycoprotein, which is responsible for pumping many drugs and other toxins out of the brain. Dogs with the mutant gene can not pump some drugs out of the brain as a normal dog would, which may result in abnormal neurological signs. The result may be an illness requiring an extended hospital stay--or even death. Approximately 3 of every 4 Collies in the United States have the mutant MDR1 gene. The frequency is about the same in France and Australia, so it is likely that most Collies worldwide have the mutation. The only way to know if an individual dog has the mutant MDR1 gene is to have the dog tested. As more dogs are tested, more breeds will probably be added to the list of affected breeds.

Breeds found to be effected as of late 2007 include Collies, Shetland Sheepdogs (Shelties). Australian Shepherds, Old English Sheepdogs, German Shepherds, Long-haired Whippets, Silken Windhounds, and a variety of mixed breed dogs.

TEST #24 NEURONAL CEROID-LIPOFUSCINOSES

The Neuronal Ceroid-Lipofuscinoses (NCLs) are a class of inherited neurological disorders that have been diagnosed in dogs, humans, cats, sheep, goats, cynomolgus monkeys, cattle, horses, and lovebirds. Among dogs, NCL has been reported in many breeds, including English Setters, Tibetan Terriers, American Bulldogs, Dachshunds, Polish Lowland Sheepdogs, Border Collies, Dalmatians, Miniature Schnauzers, Australian Shepherds, Australian Cattle Dogs, Golden Retrievers, and other breeds. NCL is almost always inherited as an autosomal recessive trait.

TEST #25 PYRUVATE DEHYDROGENASE PHOSPHATE DEFICIENCY- (PDH)

Complex (PDHc) deficiency is a clinically heterogeneous mitochondrial disorder with phenotypes ranging from fatal infantile lactic acidosis in newborns to chronic neurological dysfunction. Milder forms may present with intermittent ataxia. The role of PDHc is to catalyze the oxidative decarboxylation of pyruvate to acetyl-coenzyme A (acetyl-CoA). PDHc is a multi-enzyme complex comprised of three catalytic domains: pyruvate decarboxylase (E1), an 2_2 tetramer; dihydrolipoamide acetyltransferase (E2), and dihydrolipoamide dehydrogenase (E3). The complex plays a pivotal role in metabolism, limiting the rate of oxidative glucose consumption, and is highly regulated to respond to all metabolic requirements. Several cofactors as well as reversible phosphorylation are required for this regulated mechanism (1-3).

Commonly affected breeds include: Clumber and Sussex Spaniels.

TEST #26 PYRUVATE KINASE DEFICIENCY

Canine Pyruvate Kinase Deficiency, also known as Erythrocyte Pyruvate Kinase Deficiency, is a syndrome of hemolytic anemia. Pyruvate Kinase (PK) is a regulatory enzyme found within red blood cells. Red blood cells (erythrocytes) that are deficient in the PK enzyme rupture prematurely causing hemolytic (red cell rupture) anemia. Indications of anemia include: very pale mucous membranes (gums, eye lids, etc.), increased heart rate and pounding pulses, generalized weakness and intolerance to exercise or activity. The liver and spleen of anemic dogs may be enlarged. In dogs over one year of age, the density of the bones appears radio-graphically, increased. If the dog has a relatively inactive life, it may not exhibit any obvious indications of a problem. Most often this condition is detected, through clinical indications, in dogs between the ages of four months and one year, although this condition can remain undetected until later in life. Most sufferers of Pyruvate Kinase Deficiency die between the ages of one to four years, due to progressive anemia or liver failure.

Commonly affected breeds are Basenji, West Highland White Terrier.

TEST #27 CANINE MULTIFOCAL RETINOPATHY

Canine Multi-focal Retinopathy (CMR) is a recessively inherited eye disease. The condition observed in an ophthalmologist's exam includes numerous distinct (i.e. multi-focal), roughly circular patches of elevated retina with accumulation of material that produces gray-tan-pink colored lesions. These lesions, looking somewhat like blisters, vary in location and size, although typically they are present in both eyes of the affected dog. The disease generally develops in young dogs before 4 months and might progress slowly, might appear to heal, or might even appear and then go away again. Some lesions disappear with no

remaining sign, while some lesions leave a wrinkled area – a fold. Some leave the lasting lesion of a blister formation. Most dogs exhibit no noticeable problem with vision despite their abnormal appearing retinas. And in almost all cases, CMR does not progress significantly over time.

Commonly affected breeds are Great Pyrenees (GP), Coton de Tulear (CdT), English.

TEST #28 CANINE RETINOPATHY

This test is similar to #27, Canine Multi-focal Retinopathy but is specific to Mastiff dogs

Commonly affected breeds are Mastiff (EM), Bullmastiff (BM)

TEST #30 AUTOSOMAL RECESSIVE HEREDITARY NEPHROPATHY

The genetic disease, hereditary nephropathy; is an inherited glomerular disease in the domestic dog that is similar to Alport syndrome of the human. Both diseases are caused by mutations in the type IV collagens genes, and the disease has nearly identical pathology and clinical presentations in the dog and human. By studying this disease in the dog, our laboratory hopes to increase understanding of the disease so that information that can be applied to both the human and the dog. Reported here is 1) the development of a genomic based test to determine genotypes of mixed breed dogs in a colony presenting with X-linked hereditary nephropathy, 2) the determination of patterns of X-chromosome inactivation in normal dogs and dogs that are carriers of X-linked hereditary nephropathy, 3) the design of a synthetic COL4A5 cDNA to be used for gene therapy treatment of dogs with X-linked hereditary nephropathy, 4) the investigation of type IV collagen gene expression changes in normal dogs and those affected with X-linked and autosomal recessive hereditary nephropathy, and 5) the discovery of the mutation causative for autosomal recessive hereditary nephropathy in the English Cocker Spaniel. Utilization of the colony of dogs affected with X-linked hereditary nephropathy (for which the causative mutation was previously identified) allowed for comparisons of type IV collagen gene expression to English Cocker Spaniels with autosomal recessive hereditary nephropathy. These data were critical to identification of the gene harboring the causative mutation for autosomal recessive hereditary nephropathy. Sequencing was performed to identify the mutation. With the ability to test for carriers of this disease, it is our hope that breeders will use it to maintain the desired traits in the ECS while simultaneously eliminating the production of affected offspring.

Commonly affected breeds are: American Cocker Spaniel, English Cocker Spaniel

Reference: Bell, Rebecca Jane, 1981-Genetics of x-linked and autosomal recessive hereditary nephropathy in the domestic dog, Published by Texas A&M University.

TEST #31 TYPE I GLANZMANN'S THROMBASTHENIA

Glanzmann's Thrombasthenia (GT) is an autosomal recessive bleeding disorder caused by qualitative or quantitative deficiencies of the platelet membrane glycoprotein aIIb β 3. This is the first report of a molecular genetic basis for type I GT in dogs. As previously reported, a thrombasthenic Great Pyrenees dog (dog No. 1) experienced uncontrolled epistaxis despite results of coagulation screening tests, platelet quantitation, and von Willebrand factor quantitation that were within reference ranges. Platelet aggregation was minimal in response to agonists. Flow cytometry, autoradiography, and immunoblot experiments demonstrated either marked reduction or absence of glycoproteins aIIb and β 3. In this study, we report the presence of a 14-base insertion in exon 13 and defective splicing of intron 13 in the aIIb gene of two thrombasthenic dogs (Nos. 1 and 8). The insertion disrupted the fourth aIIb calcium-binding domain, caused a shift in the reading frame and resulted in a premature termination codon. Possible consequences of this mutation include decreased aIIb mRNA stability and production of truncated aIIb protein that lacks the transmembrane and cytoplasmic domains and a large portion of the extra-cellular domain. We identified the dam, sire, and three littermates of dog No. 8 as carriers of the aIIb mutation. Canine aIIb and β 3 genes share significant homology with the genes in human beings, making canine GT an excellent translational model for human GT. A defined molecular basis for canine GT will enhance ongoing gene therapy research and increase the understanding of structure–function relationships of this integrin.

Commonly affected breeds are Great Pyrenees.

CATS

TEST #1 POLYCYSTIC KIDNEY DISEASES

Polycystic disease is a disease that shows up later in life (late onset) with enlarged kidneys and kidney dysfunction occurring between three and 10 years of age (on average at seven years of age). The condition is inherited and cysts are present from birth, but are smaller in younger animals. Cyst size can vary from less than 1 mm to greater than 1 cm in size, with older animals having larger and more numerous cysts. Problems occur when these cysts start to grow and progressively enlarge the kidney, reducing the kidney's ability to function properly. The ultimate end is kidney failure. Some of the clinical signs are depression, lack of or reduced appetite, excessive thirst, excessive urination and weight loss. There is a marked difference in when and how quickly individual cats succumb, with the possibility of this developing late enough in life that the cat can die of other causes before kidney failure. However, kidney failure is certain when the cysts can grow and cause problems. Rarely, cysts are also seen in other organs such as the liver and uterus.

This disease is most commonly seen in **Persians** and **Exotic Shorthairs**.

TEST #2 MUCOPOLYSACCHARIDOSES TYPE VI & VI Mild

MPS VII is one of the least common forms of the mucopolysaccharidoses. The disorder is caused by deficiency of the enzyme beta-glucuronidase (GUSB). In its rarest form MPS VII causes kittens to be born with hydrops fetalis, in which extreme amounts of fluid are retained in the body. Neurological symptoms may include mild to moderate mental retardation, communicating hydrocephalus, nerve entrapment, corneal clouding, and some loss of peripheral and night vision. Other symptoms include short stature, some skeletal irregularities, joint stiffness and restricted movement, and umbilical and/or inguinal hernias.

The disease is inherited as an autosomal recessive lysosomal storage disorder.

The SNP test for this disease is directed to the gene that codes for the enzyme b-glucuronidase. Many cat breeds affected by Type VI & VI MILD.

Reference: Ponder, K. P., Melniczek, J. R., Xu, L. et al. 2002. Therapeutic neonatal hepatic gene therapy in mucopolysaccharidosis VII dogs. Proc. Natl. Acad. Sci. USA, Vol. 99:13102–13107.

Ray J, Scarpino V, Laing C, Haskins ME. 1999. Biochemical basis of the beta-glucuronidase gene defect causing canine mucopolysaccharidosis VII. J Hered. 90(1):119-23.



Visit www.ingen.bs for a full description of each of these diseases and check for newly added screenings.

Your Pawsitive ID™ Genotyping certificate will show if your pet test either positive, otherwise known as “Affected” (Homozygous), Negative or is a Carrier (Heterozygous). Most of the time, pets test negative for these diseases. Obviously this result is a relief. Its also valuable information to know that your pet *DOES NOT* have one of these inherent, genetic diseases, particularly if you are a breeder.

Unfortunately there are cases where pets test positive (Homozygous). Your certificate will alert you to this. Please see you vet as soon as possible if this is the case. Your veterinarian can determine the extent of the problem and prescribe medication or therapy. This will keep you pet as comfortable as possible as long as possible.

In some cases test results indicate that your pet is a carrier (Heterozygous) of a certain disease. Your certificate will alert you to this condition as well. Being a carrier does not mean your pet has the disease but could pass these genes on to offspring. Depending on the disease and the breed, you should consider carefully if you should breed this animal. Please see you vet for additional treatment options and consultation.

PHYSICAL ATTRIBUTES

Pawsitive ID™ can screen for the following physical traits:

YELLOW

MELANISTIC MASK

AGOUTI (A82S)

AGOUTI (R96C)

DILUTE COAT COLOR

3 BROWNS

LONG HAIR

FEMALE/MALE MARKERS

This information is typically more important to breeders where it can help determine coat color probability in future litters between two candidate animals. A very informative website which explains more about Physical Attributes and how the test results provided by Pawsitive ID™ could affect future characteristics of offspring from the tested animal is available at:

www.homepage.usask.ca/~schmutz/dogcolors.html

UNDERSTANDING THE RESULTS

The results listed on your Certificate of Genotyping for Physical Attributes are explained below:

Agouti The alternation of light and dark bands of color in the fur of various animals, producing a grizzled appearance.

Agouti Tests (AY) **Ay** is a fawn or sable (with ranges from yellow to red with darker tips or the reddish hair of sable with intermixed black hairs).

+/+ = ay/ay or homozygous for agouti ay

+/- = ay/- or heterozygous carrier of ay

-/- = -/- or negative for ay

Agouti Tests (a) **a** is the recessive black and is black in color.

+/+ = a/a or homozygous for agouti a

+/- = a/- or heterozygous for agouti a

-/- = -/- or negative for **a**

Melanistic Mask Certain breeds of dogs that have a tan, yellow, fawn, or other pale coat color over most of their body may also have a black, brown or grey mask over their muzzle. This black muzzle can sometimes extend up over their ears. Breeds that have such a black mask include the Akita, Bullmastiff, Boxer, German Shepherd, Great Dane, Greyhound, Keeshound, Leonberger, Mastiff, Pekinese, Pug, Rhodesian Ridgeback, Sloughi, Tibetan Spaniel, and Whippet.

+/+ = Em/Em or homozygous for Melanistic mask

+/- = Em/- or heterozygous for Melanistic mask

-/- = -/- or negative for Melanistic mask

Em is positive for Melanistic mask

Dilute Coat Color **A coat color that appears lighter in intensity or paler than normal**

+/+ = D/D or homozygous for dilution

+/- = D/d or heterozygous for dilution

-/- = d/d or negative for dilution

D is positive for dilution. This dilution does not express in all breeds.

There are other dilutions that are not yet mapped.

These color tests are not tested in all breeds at this time and are therefore not verified to be informative in all breeds.

Visit www.ingen.bs for a Glossary of common genetic terms



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