the Poodle club of canada

PCC Fall Newsletter

DEC 2016 SUMMER NEWSLETTER

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Managing Dog Seizures Holistically

by Jodie Gruenstern, DVM, CVA

Seizures are scary, but don't panic. Many of these conditions can be managed successfully with an integrative approach.

What happens during a seizure?
Few things are more distressing than watching your dog have a seizure. The first time you witness it, you may fear he is dying or will need to be euthanized. But it's important not to jump to conclusions. Many seizure disorders can be successfully managed.
During a seizure, dogs do lose control of their jaw muscles and could bite accidentally. They commonly lose colon and bladder control as well. So it is not unusual for them to drool profusely, and urinate or defecate during a seizure episode.

Often, the veterinarian will perform a battery of screening blood tests to rule out the many problems that can cause seizures, but do not produce any definitive answers or specific therapeutic management. The assumption: idiopathic epilepsy, which means the cause is unknown.

Not all seizures are epilepsy

Many people assume that seizures and epilepsy are the same thing, but many seizures are not caused by epilepsy. Unfortunately, **there is no specific test for epilepsy**, so it's what we call a diagnosis of "rule outs". In other words, your veterinarian needs to "rule out" the myriad other causes of seizures before you can assume your dog has epilepsy. The distinction is important because it affects treatment options and prognosis.

Conventional treatment means anticonvulsant drugs

Mainstream veterinarians will usually prescribe anticonvulsant drugs for seizures, but they are not without risks. Phenobarbital is a commonly recommended, addictive narcotic with potential **liver side effects**. Potassium bromide is often piggybacked onto phenobarbital, but **can cause pancreatitis**. Both can be quite **sedating**.
For patients that must remain on a conventional anticonvulsant, an integrative veterinarian can recommend **nutritional support to protect the liver and pancreas.** A popular Chinese herbal is prescribed by Oriental practitioners to assist with liver metabolism. This formula is called Bao Hu Jiang Jun Tang and can help prevent liver failure associated with phenobarbital usage. The formula contains: milk thistle, bupleurum, schisandra, licorice root, salvia root, white peony and skullcap. You might recognize milk thistle as a commonly used liver supportive herbal. Herbal care can be initiated while the dog is still on phenobarbital.

Identify the triggers

When getting to the root of Toby's seizures, the most important additional diagnostic tool we used was journaling. I cannot overemphasize how useful it can be for a diligent dog parent to note every seizure. Record the date, time of day, the current weather, and what the dog has eaten recently, including treats. Also record household activities or environmental exposures that occur just prior to each seizure. Just as there are multiple causes for seizures, there are also multiple triggers for epileptic seizures such as Toby was experiencing. Much as an allergic individual is often allergic to several things, an epileptic individual can be stimulated to seizure for many different reasons. Therefore, one of the main goals of therapy is to identify as many triggers as possible and eliminate them.

1. Food triggers

A weekend of close observation quickly revealed that Toby had a sensitivity to chicken. Eliminating all chicken and replacing it with beef led to a substantial decrease in the frequency of seizure activity! Although Toby had been given many different diets, they all contained chicken and a starch such as corn or rice. Toby was now being fed a balanced all-beef, starch-free raw diet.

2. Environmental triggers

Careful observation and long term journaling also made it clear that lawn treatments and particular household floor cleaners would trigger seizure activity in Toby. Like many epileptics, Toby would also frequently seizure at the time of a full moon. The former triggers could be eliminated or avoided. For those that are unavoidable, like the moon phase, knowing about their involvement allows you to be prepared and predict a seizure. It is also possible to add an herbal or use another modality to try and intervene with the seizure pattern.
For example, if you know a dog has a seizure every three weeks in the evening, an acupuncture treatment could be scheduled for the anticipated day, or an extra dose or additional type of calming herbal might be given. Toby's mom, who is also an acupuncturist, was able to pre-empt a seizure onset with acupressure at GV 26, an acupoint found at the groove between the nostrils. If

Managing Dog Seizures Holistically

necessary, even an extra quantity of anticonvulsant might be given prevemptively.

Western herbs and Eastern medicine

• **Western herbals** used to manage seizure activity have a calming influence. These are helpful when stressful situations trigger the seizures. Most popular in this category would be skull cap, valerian, passion flower and oat straw. They are safe to use with phenobarbital; they may potentiate its effectiveness, but this is generally desirable. It may allow an integrative veterinarian to decrease the dose of phenobarbital.

• **Oriental medicine** should first involve a tongue and pulse diagnosis, and a diagnosis of the dog's constitution. Seizure activity is considered "internal wind" originating from the liver. To understand this theory, think of heat producing fire with a rising wind. A dog with a Fire constitution, red tongue and fast pulse may have internal wind triggered by hot foods such as chicken, lamb or venison. As in Toby's case, a neutral food choice such as beef or bison might be ideal. An herbal blend that includes cooling and "shen"-calming herbals may be beneficial. "Shen" means mind. A shen disturbance may trigger a seizure.

Toby had a red, thin tongue and a rapid pulse. His nose, pads and skin were commonly dry. These are all heat signs. He preferred cool areas like the floor or basement. He had a great appetite. The Oriental diagnosis for him was liver yin deficiency with internal wind, and kidney jing deficiency due to the early onset of his disorder. This diagnosis helped with the selection of a Chinese herbal formulation for Toby.

The initial herbal formula we gave Toby was Tian Ma Gou Teng Yin with added **schisandra for liver protection**. This formula contains 11 herbs. Chinese medical practitioners commonly prescribe it for human patients with internal wind caused by high blood pressure and its associated headaches and dizziness. Di Tan Tang, meanwhile, is commonly called "herbal valium", and Ding Xian Wan is classically used for phlegm conditions.

Toby received a rotation of several different Chinese herbal formulations, and as the frequency and severity of his seizures diminished, he was weaned off the phenobarbital.

Toby's journey is just one example of what can be accomplished with the cooperation of a integrative or holistic veterinarian, and a diligent guardian who is able and willing to identify and manage her dog's seizure triggers. Toby went on to live a full and happy life, and so can many other dogs with similar conditions.

Some other opinions of holistic veterinaries:

Extracted From Shirley's Wellness Cafe

Dee Blanco, D.V.M -

"You take healthy animals and often very quickly after you vaccinate, you can see simple things like itching of the skin or excessive licking of the paws sometimes even with no eruptions. **We see a lot of epilepsy/seizure, often after a rabies vaccination.** Or dogs or cats can become aggressive for several days. Frequently, you'll see urinary tract infections in cats, often within three months after their [annual] vaccination. If you step back, open your mind and heart, you'll start to **see patterns of illness post-vaccination."**

Some dogs are sensitive to chemicals and if you feed and water them from a plastic bowl, they ingest some of the chemicals and it can help induce the seizures. **There has been reports of a reduction of seizures when the immune system was modulated and the inflammation reduced with a natural immune enhancing substance.** Contact Shirley for information: (323) 522-4521 wellnesscafe@twc.com

William Pollak D.V.M. -

"The most common direct cause of seizures seen in clinical practice in our pets is **parasitic infection** combined with **nutritional deficiencies** based on 100% commercial pet food feeding."

"Feeding a natural raw food diet is vital to not only maintaining the health of your pet, but also **keeping ideal immune function alive and well**. Many times after eliminating seizures through improving the diet, seizures return after commercial pet food is re-instituted."

"Today's modern approach to dealing with these problems is the administration of more chemicals, injectable or otherwise and even greater processed "prescription" diets. Seizures are masked by giving chemicals that profoundly dull the CNS, slowing it down and confusing it so as to reduce the likelihood of another seizure. These chemicals oftentimes do not work and further confuse the biological system as already described earlier. The underlying imbalance is not directly addressed. Deranged metabolic disorders due to chemical shortages or imbalances are superficially addressed by further limitations in the diet; i.e.even more severely processed foods."

WHAT CAUSES SEIZURE DISORDERS?

by Amy Snow & Nancy Zidonis

A seizure is a neurological dysfunction or misfiring of the neurons most often in the cerebrum section of the brain. Studies show that the chemical balance of the neurotransmitters is compromised. Some dogs experience mild seizures while others endure lengthy, more severe episodes. Unfortunately, there is another category of more extreme situations when a dog is stricken in quick succession with severe, relentless seizures that often result in death.

The veterinary community is not exactly sure why dogs experience seizures, nor why the number of dogs with seizure disorders is on the rise. There are many possible triggers, including hereditary defects, toxins (household cleaners, tick repellents, fertilizers, foods, etc.), tumors, brain damage, hyper or hypothermia, certain medications, hormonal imbalance, distemper, Lyme disease, kidney disease, liver disease – the list goes on. Many seizure disorders are deemed "idiopathic", meaning "unknown cause", since it is difficult to track the pathology.

ACCUPRESSURE AND ICE CAN WORK TOGETHER TO HELP REDUCE THE SEVERITY AND DURATION OF SEIZURES IN DOGS

Acupressure and seizures

Acupressure has been used for thousands of years to maintain the health of animals and humans. This touch therapy is gentle and non-invasive, yet powerful. Acupressure is based on Traditional Chinese Medicine (TCM) concepts and theories.

According to **Chinese medicine**, most seizure disorders are related to an invasion of Wind that affects the Liver organ system. To the Western mind, associating wind with seizures may seem strange. Take a minute to picture a strong wind blowing the limbs of trees in all directions while other branches and dust are chaotically swirling in the air. Now have that wind blowing inside the dog's body, creating a similar internal chaos and movement. The dog's involuntary movements and loss of consciousness during a seizure are similar to the chaos created by a strong wind.

The Liver organ system is responsible for the smooth and harmonious flow of life-promoting energy called chi (also seen as qi or ki and pronounced "chee"). When the Liver is compromised in any way, by toxins or disease, it becomes vulnerable to an invasion of Wind. In turn, the Wind will disrupt the harmonious flow of chi in the dog's body, leading to a seizure. To prevent and/or minimize seizures, we need to restore the natural balance of Liver chi and calm the nervous system.

A session for seizures

The acupressure chart with this article offers acupoints that can help reduce the frequency and severity of seizures and may even completely resolve the disorder. They can also help reduce the amount of medication a dog may need to receive to control the disorder.

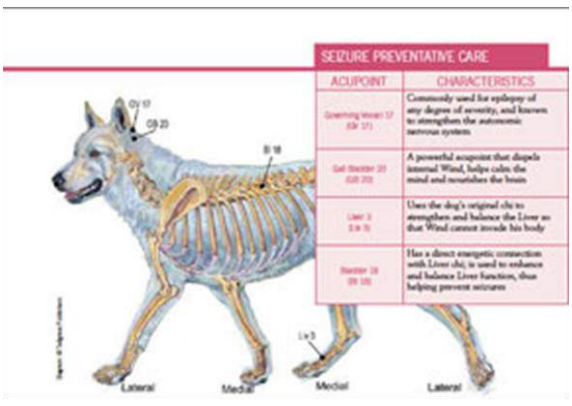
Keep in mind that acupressure is not a substitute for veterinary care. As well, these points are not intended for during a seizure. The intent is for the guardian to consistently offer this acupressure session every five to six days.

This session is designed to specifically dispel internal Wind, strengthen Liver chi, rebalance the autonomic nervous system, and clear the mind of anxiety.

How to do it

The acupoints shown in the chart are to be stimulated in succession (one at a time) on both sides of the dog's body. By stimulating a point, we simply mean applying gentle pressure to the point with the soft, fleshy portion of your thumb at a 45° to 90° angle to the dog's body. You do not have to apply much pressure because this is energetic work, not tissue manipulation.

While holding the acupoint, count to 30 slowly or until the dog moves away or demonstrates some form of release. Energetic releases can include yawning, licking lips, stretching, passing air, demonstrating the need to move, even falling asleep. Remember to repeat this procedure on both sides of the dog since the body is bilateral. If the dog gives any indication of pain, please stop immediately and work the points on his other side. If the dog continues to be uncomfortable, try again at a later date when he's not as sensitive.



Depending on your dog's condition, regular acupressure sessions can go a long way to controlling the disorder. It may not completely eliminate it, but it will make life a lot less stressful for you and your dog.

Other Alternative Treatments for Seizures

Homeopathics are medical formulas based on a theory of like cures like and highly diluted quantities of medicinal substances are given to cure symptoms. acorus, black cohosh, Chinese senega root, ginger, milk thistle bilberry, causticum-injeet (heel) .

Essential Oils:
Every essential oil has a unique and distinctive fragrance which stimulates the immune system, body and emotions in a precise way. The essential oils are diluted with a carrier for delivery because they should not be used in concentrate form. Carriers range from sweet almond oil to watermelon seed oil, each possessing individual healing traits. Essential oils are purposefully matched with individual carries to achieve a specific result. for this disorder:

Source: Shirley's Wellness Cafe

Homeopathic Treatment:

Healing From Within - Drug Treatment and Suppression

Dr. Jeffrey Levy DVM - "The greatest harm of drug treatment is usually not so much the toxicity or side effects as it is the effects of suppression. Allopathic (conventional Western) medical thinking generally seeks immediate gratification: just make the symptom go away. So the patient may be better in the short term, but is usually worse in the longer term. Elimination of the symptom is NOT the same as elimination of the disease. Homeopathy is just the opposite: sometimes the symptoms are worse in the short term (such as with aggravation or the reversal of a previous suppression), but the real benefit is in the longer term. A symptom, say itchy skin, is the body's response to a deeper problem.

Flower Essence: Star of Bethlehem helps ease trauma. Cherry Plum targets loss of control in body functions. Impatiens helps with seizure activity.

When a symptom is suppressed, it is only the outward manifestation of the problem that goes away. Since the deeper problem is still there, the body may, in time, produce the same symptom again. Another possibility is that, as a result of the suppression, the deeper problem progresses to the point that a deeper, more serious symptom is produced. So the itchy skin may go away, but then chronic diarrhea develops. If the diarrhea is then suppressed as well, it may lead to, say, liver disease. But hey, at least the skin is cleared up! I see this pattern, or variations on it, very frequently in reviewing the medical records of new patients. It is the unrecognized, and often high, price that we pay for the quick fix, for immediate gratification, for the shot or pill that seems to make the problem go away."

Dr. Goldstein DVM -
"Holistic pet care essentially revolves around the notion that the best way to cure an animal who is ill is to help the animal cure itself. We are not the true healers of our pets--they are. By treating the root of the problem instead of its symptoms, holistic medicine enables our pets to regain and maintain their own well-being." In his practice, Dr. Goldstein has had extraordinary success treating cancer holistically.

Source: http://www.natural-dog-remedies.com/remedies/seizure-disorder.htm

Description: A dog could experience more than one type of seizure.
For example: **A Petit Mal** seizure resembles day dreaming. Petit Mal seizures are difficult to notice because they happen very fast. They are also referred to as Absence seizures. Partial seizures only effects one area of the body. You will see limbs twitching involuntarily.
The **Grand Mal** is the most noticeable. It involves full blown convulsions and incontinence. Grand Mal seizures are also known as tonic-clonic seizures.

Contributing factors: Neurotransmitters are not working properly. It is still a mystery as to why the neurotransmitters do not work in the manner they are suppose to. Contributing factors to seizures are: stress induced, underlying medical reasons, vaccinations, poisons, brain injuries, some medications, nutritional deficiencies, genetic factors, parasitic infections, low blood sugar, and heavy metal contamination.

Herbal Treatment:

For years dog supplements have included toxic materials to artificially maintain dog fitness as a temporary measure. The time has come to change to a holistic approach toward pet well-being. Native remedies have been shown to achieve more immediate, more complete and longer lasting fitness, vitality, quality and longevity of animal lives. Learn about herbal remedies and other safe alternatives to drugs, chemo, radiation and surgery.
Some herbs that may form part of a formula are: Acorus is a natural antioxidant and **stimulates the immune system** in a unique way. Chinese Senega Root helps to reduce seizures triggered by stress. Ginger helps to protect the body from the side effects of anti-seizure medications.
Get more info on EaseSure - Canine and feline epilepsy natural remedy

Other: Reiki or other types of therapeutic touch is beneficial. It has been our experience dogs respond positively to the healing human touch.

The Importance of a Balanced Immune System

Source: Shirley's Wellness Cafe

The key to self-healing is a strong defense system, which protects dogs and cats from everything from the flu germs to cancer cells. More than their mainstream counterparts, holistic veterinarians believe that a weak immune system plays a key role in causing illness and associated syndromes. Antibiotics fight infection, but they don't affect whatever weakened the immune system in the first place. This is why holistic veterinarians focus less on things that cause diseases and more on those that affect the body's defenses.

Carm's puppy began to experience severe seizures as a result being exposed to organic pyrethrum.

Organic pyrethrum spray paralyzes insects that come into contact with it and is the strongest insecticide allowed under National Organic Standards guidelines.

The veterinarian was helpless. The dog was dying. In her detailed testimonial, Carm explains what she did cure her dog of seizures and how she saved his life.

On Wednesday, May 7, 2014, one of our small moyen standard poodles was exposed to organic pyrethrum and started having severe seizures; flopping down and paddling. We rushed him to the vet and they put him on IV. He was eating a little (on Science Diet from the vet) but still was having seizures and was biting randomly at his bowl, the air, and kept tearing out his IV. He was being sedated to control the seizures and to keep the IV going to keep him hydrated. Basically, he was getting all the prescribed vet care for pesticide poisoning.

Our Veterinarian Was Helpless

We called every morning to see how he was doing while half hoping he was better and half dreading if he'd succumbed to the poison. It seemed he was getting a little better and on Friday the vet wanted to keep him over the weekend and see how he progressed. I still called Saturday morning to check on him again. The vet tech said that they were sorry and they'd done everything they could for him but he was blind, still having seizures, couldn't stand up, couldn't walk, had no control over his back end and was biting everything. They said he wasn't getting any better and we discussed having him put down.

Immunocal to the Rescue

In the meantime, I had spoken with a friend, Cathie Warren of Adonai Red Poodles, which had mentioned Immunocal. She told me she once had 2 puppies that had lost control of their back ends and were paralyzed from a pronounced reaction to vaccinations. She said it took a while of nursing them back to health but gave them Immunocal for about 3 weeks and they recovered and could walk again. While I listened to her story and found it hard to believe, I figured we already had him the hospital four days and owed it to him to try to save this precious puppy's life. After talking to the vet on Friday that Davie wasn't making a lot of progress, we decided to spend quite a bit extra to have Immunocal overnighted to get it on Saturday. We thought that we had nothing to lose to try Immunocal and if he wasn't better we would take him back on Monday to have him put down.

Saturday at noon before the vet's office closed, I went and picked him up. He was just lying in the hospital cage and was very sedated. I thanked the vet for all they had done to care for him but I just didn't want to give up on him yet. My very experienced, kind vet lady said that that's the way to be...to never get up. I scooped up this **blind, crippled** little poodle that reeked of Science Diet, headed to the health food store and prayed that the Immunocal, that had been overnighted, was waiting for me when I got home.

Davie was fighting for his life!

I had brought plastic bags, bottle water, and bentonite clay and made a clay poultice to apply to his skin to help draw out the toxins. I wrapped him in a towel and put him a soft crate in my car's front seat. At the health food store I bought some homeopathic pills like Belladonna 30x for seizures and Thuja 30x to pull out toxins. I gave him the Belladonna and Thuja in the car and found out the Immunocal had arrived. At home after shaving his hair, I got him in a warm Epsom salt bath and applying more clay poultice all over his body, I let him soak for about 45 minutes. While soaking, I gave him some Immunocal by emptying the powder from the capsule on his gums.

After soaking him in the Epson salt bath to help pull out the toxins, I got him wrapped in a towel and held him on a heating pad. His eyes were totally dilated and he was totally blind. He couldn't stand up and had a couple more seizures. However, even in his extremely debilitated shape, he seemed to want to be held, made little moaning sounds and snuggled next to me as best he could. By this time is was about 2 p.m. and Cathie had told me that Immunocal takes 4 to 5 hours to start see a difference. Right on time about 6:30 p.m., Davie seemed more responsive. He ate a little meat but would bite at the bowl because he

couldn't see. He would bite at me if I had to pick him up and move him. He was hurting but also due to his blindness didn't know what was happening to him. He really responded to our sweetly talking to him and cuddling him. Then he'd snuggle, groan and let us know he was hurting but glad to be home. We named Davie in honor of David who fought Goliath since Davie was fighting the big giant death that had its grip on him.

Our poodle gets a natural immune system boost

Round the clock for the next few days, I held him and slept with him, giving him **Immunocal**,

Thuja and **Belladonna homeopathic** pills to help prevent the seizures every few hours. The next day he could stand up a little but then would flop down. Sometimes he'd fall off the couch. When I'd pick him up he'd snap at me. He had no use of his back end at all.

Amazingly though, **everyday he got better**. After 2 or 3 days, he didn't have any more seizures. Within 4 days we could take him outside to go potty but he was still unsteady on his feet. His eyes were dilated but it seemed like maybe he could see shadows but didn't have full eyesight. At this point, that he was alive and was improving was a miracle. We were committed to keeping him for life even if he remained blind and never recovered his sight.

Everyday we gave him Immunocal and everyday he got better and better. He loved to snuggle and was giving us kisses and hugs around our neck. Now we were giving him Immunocal with every meal.

Within one week, he could walk and could see! A few more days later and he was walking fast and then trotting. The next day he was actually running!!

Two weeks after picking him up from the vet when they thought it was time to put him down and now he was playing with other puppies and no one can tell any difference from any other puppies except maybe that he loves to snuggle, give hugs and be picked up even more than the other puppies. It's been 3 weeks now and other than his haircut he looks and acts like any other active, healthy puppy. It's a true miracle and Immunocal saved his life.

News from Optigen

OPTIGEN is REQUESTING RESEARCH SAMPLES

from Miniature or Toy Poodles diagnosed with
Optic Nerve Hypoplasia or Micropapilla or Inherited Cataracts.

The Poodle Club of America Foundation, Inc. is supporting research aimed at identifying the molecular causes of Optic Nerve Hypoplasia & Micropapilla and Inherited Cataracts in Miniature and Toy Poodles. OptiGen in collaboration with the research laboratory of Dr. Gustavo Aguirre at the University of Pennsylvania is collecting samples and clinical data on these diseases with the goal of developing diagnostic DNA tests for the conditions.
Research samples from affected dogs are needed for this study.

Details of these Research Studies

Molecular Genetic Study of Optic Nerve Hypoplasia (ONH) and Micropapilla (Mp) in the Miniature and Toy Poodle

The Poodle Club of America Foundation, Inc. and Gustavo Aguirre, VMD, PhD
University of Pennsylvania and OptiGen, LLC

Background

The Poodle Club of America Foundation, Inc. has funded a three year research study to be carried out at the University of Pennsylvania and OptiGen, LLC to identify the molecular genetic basis of ONH and Mp,
and develop a DNA-based diagnostic test that can be used to identify dogs that are genetically normal or are at risk to transmit the undesirable genetic defect to their progeny. By judiciously using the DNA test information, breeders can minimize the risk of producing affected dogs while maintaining the genetic diversity of the breed.
ONH is a rare genetic defect in which the optic nerve fails to develop normally, leading to blindness; one or both eyes may be affected. Although autosomal recessive inheritance is suspected, there is insufficient genetic information to date to validate the mode of inheritance. It is likely that disease predisposition is controlled by one gene while the additional modifying gene(s) influence the expression
and severity of the disease. Mp **may be** part of the same disease continuum in which dogs with
apparently "hypoplastic" optic nerves on clinical examination retain normal vision and pupillary light
responses (PLR), and optic nerve structure is normal behind the globe. Only by well focused research study can these issues be resolved and DNA based test(s) developed.

Your help is needed

To carry out this study we need the assistance of dog owners/ breeders, as well as board certified veterinary ophthalmologists (ACVO, ECVO) so that samples for the research study can be definitely ascertained. We are very grateful for your interest in participating in the present research study. Please make sure that only one form is used for each study dog.
A research form for submitting samples and information for this study can be found by clicking on ONH/Mp Research Form.
The owner should complete the first section of owner and dog information. The second section of the form requests the examining ophthalmologist to provide brief clinical descriptions and, if possible, fundus photographs. Along with the completed form, we need a copy of the dog's pedigree (5-6 generations), any current/previous eye exam records, and 3-5 ml of whole unclotted blood in EDTA to be sent to OptiGen 767 Warren Rd. Ithaca NY 14850. Please call OptiGen 607-257-0301 or email genetest@optigen.comwith any questions on sample submission.

Molecular Genetic Study of Inherited Cataracts in the Miniature and Toy Poodle

The Poodle Club of America Foundation, Inc. and Gustavo Aguirre, VMD, PhD

Background

The Poodle Club of America Foundation, Inc has funded a three year research study to be carried out at the University of Pennsylvania and OptiGen, LLC to identify the molecular genetic basis of inherited cataracts, and develop a DNA-based diagnostic test that can be used to identify dogs that are genetically normal, carriers or affected. By judiciously using the DNA test information, breeders can minimize the risk of producing affected dogs while maintaining the genetic diversity of the breed.

In the Miniature and Toy Poodle some types of cataracts are an inherited condition which is characterized by loss of transparency in the lens when dogs are young adults/adult. Although some individuals use the term 'Juvenile Cataracts" to imply a genetically inherited defect, the appropriate term for the disease is *Inherited Cataracts*. Dogs with inherited cataracts are born with normal lenses, which then proceeds to degenerate over time, leading to visual impairment and then blindness later in life. The age range generally is variable, and the disease begins sometime between 2-5 years of age, and progresses; the rate of progression and severity of the disease can vary between affected dogs. About 6% of all Poodles are diagnosed with inherited cataracts. The mode of inheritance is not known, although autosomal recessive inheritance is likely; there are no gene-based tests available. We propose to carry out a focused study on inherited cataracts in Miniature and Toy Poodles with the aim of identifying the gene/mutation responsible for the disease.

Your help is needed

To carry out this study we need the assistance of dog owners/ breeders, as well as board certified veterinary ophthalmologists (ACVO, ECVO) so that samples for the research study can be definitively ascertained.

We are very grateful for your interest in participating in the present research study.

Please make sure that only one form is used for each study dog. A research form for submitting samples and information for this study can be found by clicking Inherited Cataracts in Poodles Research Form. The owner should complete the first section of owner and dog information. The second section of the form requests the examining ophthalmologist to provide brief clinical descriptions and, if possible, clinical photographs. Along with the completed form, we need a copy of the dog's pedigree (5-6 generations), any current/previous eye exam records, and 3-5 ml of whole unclotted blood in EDTA to be sent to OptiGen 767 Warren Rd. Ithaca NY 14850.

Please call
OptiGen 607-257-0301 or email genetest@optigen.comwith any questions on sample submission.

NOTE: all the information provided for this study is CONFIDENTIAL and will not be disclosed.

Please note we have focused this study in Miniature and Toy Poodles because it is in these varieties that Optic Nerve Hypoplasia or Micropapilla are more frequently found. However, if there are Standard Poodles diagnosed with Optic Nerve Hypoplasia or Micropapilla, we would be delighted to include samples from them

Newer Genetic Tests available for Poodles

Day Blindness/Retinal Degeneration (DB/RD) Test

***For Breed: Standard Poodle**, Goldendoodle, Labradoodle/Goldendoodle Cross, Labradoodle & Labradoodle, Australian*

The support of the **Poodle Club of America** was critical to a successful collaboration between OptiGen and researchers at the University of Pennsylvania School of Veterinary Medicine that led to the discovery of a mutation causing day blindness and subsequent retinal degeneration in the Standard Poodle. Multiple cases of day blindness in this breed were submitted for investigation to OptiGen's Free DNA testing/ Research program. After OptiGen DNA testing revealed that none of the mutations previously known to cause day blindness in other breeds were responsible for the clinical signs observed in the Standard Poodle cases, the project as turned over to Dr. Karina Guziewicz, Research Assistant Professor of Ophthalmology at the University of Pennsylvania School of Veterinary Medicine.

Dr. Guziewicz is part of the team led by Dr. Gustavo Aguirre and that has included a consortium of researchers who, over the years, have discovered mutations that comprise the majority of OptiGen's

DNA tests for inherited eye diseases in dogs. Dr. Guziewicz carried out genomic analyses on the day blind Standard Poodles that led to discovery of a mutation in one of the genes critical to retinal function.
Further research aimed at developing therapeutic strategies for DB/RD in Standard Poodles is ongoing.

Clinical signs/Disease description:

Day blindness, also known as **achromatopsia**, is characterized by a failure of cone cells in the retina to function properly. Cone cells are responsible for vision in bright light conditions while their retinal counterparts, the rod cells, function in dim light. Similar to signs observed by owners of *Alaskan Malamutes, Australian Shepherds or German Shorthaired Pointers* affected with Cone Degeneration or *Labrador Retrievers* with Achromatopsia and *German Shepherds* caused by other mutations,

Standard Poodle puppies with the DB/RD form of day blindness manifest signs of poor vision in bright light but initially retain normal vision in low light levels. However unlike other forms of Day Blindness observed in other breeds, the DB/RD mutation causes a more complete retinal degeneration in the Standard Poodle and affected dogs eventually lose both cone and rod cell function resulting in vision loss under all lighting conditions.

DB/RD mutation based test:

A DNA-based test has been developed to target the mutation responsible for Day Blindness/Retinal Degeneration (DB/RD) in the **Standard Poodle**. The DB/RD test can be used to determine the genetic status of a dog in respect to the mutation and to make informed decisions about mating options to prevent the occurrence of this undesirable blinding disorder in progeny.
The DNA-based DB/RD test allows genotype determination of a tested dog as being:

NORMAL - the dog has two copies of the normal gene and it is not going to develop Day
Blindness/Retinal Degeneration caused by the DB/RD mutation.
CARRIER - the dog has one copy of the normal gene and one copy of the DB/RD mutation. The dog will
not develop clinical signs of Day Blindness/Retinal Degeneration due to the DB/RD mutation but it will
transmit one copy of the DB/RD mutation to about 50% of its progeny.

AFFECTED - the dog has two copies of the DB/RD mutation and is expected to develop/is already
showing signs of the Day Blindness/Retinal Degeneration caused by the DB/RD mutation.

Testing and Breeding recommendations:

By selecting the mate with the appropriate genotype, it is possible to breed affected or carrier dogs and never produce affected progeny. In this manner the genetic diversity of the breed can be maintained.

Expected results for breeding strategies using the DB/RD test

Parent 1 Genotype	Parent 2 Genotype		
	Normal/ Clear	Carrier	Affected
Normal/Clear	All = Normal/ Clear	1/2 = Normal/ Clear 1/2 = Carrier	All = Carrier
Carrier	1/2 = Normal 1/2 = Carrier	1/4 = Normal/ Clear 1/2 = Carrier 1/4 = Affected	1/2 = Carrier 1/2 = Affected
Affected	All = Carrier	1/2 = Carrier 1/2 = Affected	All = Affected

Canine Degenerative Myelopathy (DM) Test

For Breeds: *American Eskimo Dog, Bernese Mountain Dog**, Borzoi, Boxer, Cardigan Welsh Corgi, Chesapeake Bay Retriever, German Shepherd dog, Golden Retriever, Great Pyrenees, Kerry Blue Terrier, Pembroke Welsh Corgi, Poodle (all varieties), Pug, Rhodesian Ridgeback,*

*These are breeds for which there is evidence of a strong correlation between the SOD1 mutation and clinical symptoms of Degenerative Myelopathy (DM). Upon request from the owner, OptiGen will perform the DM test for any breed NOT listed here.
**Two different mutations in the SOD1 gene can cause Degenerative Myelopathy in *Bernese Mountain Dogs (BMD)*. A test for the second mutation, called DM-BMD, is expected to be available at OptiGen for BMDs in the near future.

Symptoms/Disease Description:

Canine Degenerative Myelopathy (DM) has been recognized for more than 35 years as a spontaneously occurring **spinal cord disorder in older dogs,** with age of onset ranging between 8 and 14 years. Initially thought to be specific to *German Shepherds*, DM has been diagnosed in many other breeds, being most prevalent in *Pembroke Welsh Corgis, Boxers, Rhodesian Ridgebacks and Chesapeake Bay Retrievers*.
Degenerative Myelopathy is characterized by a **gradual degen-**

eration of spinal reflexes and muscle weakness. The initial signs of DM typically include loss of coordination (asymmetric ataxia) in the hind limbs. The symptoms worsen with time when the affected dog can no longer support its weight in the hind limbs. The age of onset and the speed of disease progression is variable. Affected small breed dogs often develop DM at an older age and deteriorate more slowly than affected dogs of large breeds. Affected medium to large breed dogs can be difficult to manage and owners often elect euthanasia when their dog can no longer support weight in the hind limbs

For owners and veterinarians it is important to make a distinction between progressive Degenerative Myelopathy and a variety of acquired compressive spinal cord diseases and injuries because the initial symptoms are similar but the outcome and the approach for treatment is very different.

DM Mutation Test:

A genetic test for DM was developed based on a mutation in the SOD1 gene that has been identified as a major genetic factor contributing to the development of DM in *Pembroke Welsh Corgi, Boxer, Rhodesian Ridgeback, German Shepherd, and Chesapeake Bay Retriever* [1]. The mutation is widespread and has been detected in at least 124 dog breeds with the frequency of the mutation ranging from as high as 0.94 in the Wire Fox Terrier to as low as 0.01 in the Rat Terrier [2]. There is strong evidence that homozygosity for the SOD1 mutation is highly associated with the development of DM: out of 115 dogs of various breeds with histologically confirmed DM 105 were homozygotes for the mutation, 8 were heterozygotes and only 2 were free of the mutation.
The DNA-based DM test allows to determine the genotype of a tested dog with respect to the SOD1 mutation as being:

Homozygous Normal - this dog has two copies of the normal gene and is likely to be free of the DM disease.

Carrier - this dog has one copy of the mutation and one copy of the normal gene. The chances that the dog will develop the disease are low.

Homozygous Affected - this dog has two copies of the mutated SOD1 gene and has a high risk of developing the disease during its lifetime.

Testing/Breeding Recommendations:

The identified mutation in the SOD1 gene is a major factor contributing to the development of DM in many breeds. The DM test is effective for estimating relative risk of developing the disease by a given dog but it does not account for unknown genetic factors that influence the trait. There were instances of dogs with two copies of the SOD1 mutation that did not develop the disease, as there were cases when dogs clear of the mutation were diagnosed with DM (2).
The mode of inheritance of the SOD1 mutation is best described as autosomal recessive, meaning that dogs of both sexes have a much higher risk of developing the disease when they receive two copies of the mutated gene, one from each parent.
For detailed recommendations on breeding strategies using results of OptiGen testing, please, refer to the Breeding Strategy Chart below

Expected results for breeding strategies using the OptiGen DM test

Parent 1 Genotype	Parent 2 Genotype		
	Normal/ Clear	Carrier	Affected
Normal/Clear	All = Normal/ Clear	1/2 = Normal/ Clear 1/2 = Carrier	All = Carrier
Carrier	1/2 = Normal 1/2 = Carrier	1/4 = Normal/ Clear 1/2 = Carrier 1/4 = Affected	1/2 = Carrier 1/2 = Affected

This table highlights in yellow the breedings that will NOT produce DM-affected pups due to the presence of the SOD1 mutation, which is the major risk factor associated with the disease. These breedings include at least one parent proven "Normal/ Clear" by the OptiGen DM test. All other combinations are at risk of producing DM-affected pups.

References:

1. *Awano T, Johnson GS, Wade CM, Katz ML, Johnson GC, Taylor JF, Perloski M, Biagi T, Baranowska I, Long S, March PA, Olby NJ, Shelton GD, Khan S, O'Brien DP, Lindblad-Toh K, Coates JR. Genome-wide association analysis reveals a SOD1 mutation in canine degenerative myelopathy that resembles amyotrophic lateral sclerosis. Proc Natl Acad Sci U S A. 2009 Feb 24; 106(8):2794-9.*

2. *Zeng R, Coates JR, Johnson GC, Hansen L, Awano T, Kolicheski A, Ivansson E, Perloski M, Lindblad-Toh K, O'Brien DP, Guo J, Katz ML, Johnson GS. Breed Distribution of SOD1 Alleles Previously Associated with Canine Degenerative Myelopathy. J Vet Intern Med. 2014 Feb 13. doi: 10.1111/jvim.12317.*

Osteochondrodysplasia (OC) Testing - November 11 2015

For: Miniature, Dwarf, Moyen (Klein) and Toy Poodles

A form of severe dwarfism was first described in the Miniature Poodle in Great Britain almost 60 years ago (1). Also known as osteochondrodysplasia (OC), it involves abnormalities in cartilage and bone development.

The initial clinical signs of the disorder appear at 3 weeks of age when affected puppies show stunted growth and abnormal movements. Later, deformity in limbs, jaws and the rib cage become apparent, causing difficulties in breathing and feeding. Affected puppies are typically euthanized. Dogs with less pronounced symptoms of OC can survive into adulthood but the majority have significant physical abnormalities and increased incidence of osteoarthritis.

Combined efforts of the dedicated Miniature Poodle breeders and the research team led by Dr. Mark Neff at the Van Andel Research Institute culminated in the discovery of a partial deletion of the sulfate transporter gene, SLC13A1, as a cause of the osteochondrodysplasia in this breed (2,3). The mutation is inherited as a autosomal recesessive disease. Affected dogs of both sexes develop this form of OC, apparently due to abnormalities in sulfate metabolism, when they receive two copies of the truncated gene, one from each parent.

Prevalence of the SLC13a1 mutation:

The frequency of the SLC13a1 mutation has been estimated in the U.S. subpopulation of the Miniature Poodle, with an average of 9.7 percent of tested dogs being carriers of the mutation (2). Cases of the OC disease have been described in European subpopulation of the breed but the frequency of the mutation has not yet been reported.

DNA test for OC in the Miniature Poodle:

The mutation based test for OC is designed to detect the presence/absence of the SLC13a1 deletion
and to clearly distinguish three groups of dogs corresponding to three genotypes:

AFFECTED homozygotes with two copies of the truncated SLC13a1 gene develop clinical signs of OC and they can only pass the mutated gene on to their offspring.

CARRIERS heterozygotes with one copy of the mutation and one copy of the normal gene do not develop OC but they can transmit the SLC13a1 deletion to progeny.

NORMAL/CLEAR homozygotes with two normal copies of the gene can only transmit the normal gene
to its offspring.

Recommendations for breeding strategies:

Because of the recessive nature of OC inheritance, the mutation can exist in breeding lines without detection for many generations until two carriers of the mutation produce an affected pup. On average, 25 percent of the offspring from a mating between carriers will be affected. Due to random (chance) assortment of gametes and the relatively small litter size, the actual percentage of puppies affected with OC produced in Carrier to Carrier mating often deviates from the expected 25%. It is recommended to test dogs prior to mating to ensure that at least one parent is Normal/Clear of the OC mutation to avoid production of affected progeny.

Expected results for breeding strategies using the mutation test for Osteochondrodysplasia (OC)

Parent 1 Status	Parent 2 Status		
	Normal/ Clear	Carrier	Affected
Normal/Clear	All = Normal/ Clear	1/2 = Normal/Clear 1/2 = Carrier	All = Carrier
Carrier	1/2 = Normal 1/2 = Carrier	1/4 = Normal/Clear 1/2 = Carrier 1/4 = Affected	1/2 = Carrier 1/2 = Affected
Affected	All= Carrier	½ = Carrier ½ = Affected	All = Affected

References:

1. Cotchin E, Dyce K. 1956. A case of epiphyseal dysplasia in a dog. Veterinary Record 68: 427-428.

2. Neff MW, Beck JS, Koeman JM, Boguslawski E, Kefene L, Borgman A, Ruhe AL. Partial deletion of the Sulfate Transporter SLC13a1 is associated with an osteochondrodysplasia in the Miniature Poodle breed. PLoS ONE, V. 7(12), e51917.

3. Collaboration aids discovery of SLC13a1 mutation for dwarfism in Miniatures. Pro Club Update Newsletter, 2013. V.12.

Optimal Wisdom Panel

This is a new test I had never heard of before. Jodie, the lady that bought a show puppy out of Neige and Aiden was told that it is a very important test to do. It is amazing all the information it can provide… It cost $100 and it is done with check swab.

I have ask Dr. Aguirre and Natalie Tessier if it is a reliable test… , their comments are at the end of this article on page 20.

DISORDERDegenerative Myelopathy, (DM)CLEAR
TYPENeurologic Disorders
MODE OF INHERITANCE Autosomal Recessive (Incomplete Penetrance)
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (WITHIN BREED) Not available
PREVALENCE (ALL DOGS)10.26%
DISORDER: Osteochondrodysplasia; mutation originally found in Miniature Poodle CLEAR
TYPE Skeletal Disorders
MODE OF INHERITANCE Autosomal Recessive
GENOTYPET/T
SEVERITY Considerable
PREVALENCE (WITHIN BREED) Not available
PREVALENCE (ALL DOGS)< 1%
141 additional disease mutations found in other breeds were also tested. **No findings for this dog**.

Why are these disorders tested?

Blood Disorders
DISORDERBleeding disorder due to P2RY12 defect CLEAR
MODE OF INHERITANCE Autosomal Recessive
GENOTYPE-/-
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERCanine Cyclic Neutropenia, Cyclic Hematopoiesis, Gray Collie Syndrome, (CN)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERCanine Scott Syndrome, (CSS) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERFactor IX Deficiency or Hemophilia B; mutation Gly379GluCLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERFactor IX Deficiency or Hemophilia B; mutation originally found in Airedale Terrier CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEA/A
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERFactor IX Deficiency or Hemophilia B; mutation originally found in Lhasa ApsoCLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERFactor VII DeficiencyCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)1.18%
DISORDERFactor VIII Deficiency or Hemophilia A; mutation originally found in Boxer CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEC/C
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERFactor VIII Deficiency or Hemophilia A; mutation originally found in German Shepherd DogCLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERFactor VIII Deficiency or Hemophilia A; mutation originally found in Havanese CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERFactor VIII Deficiency or Hemophilia A; p.Cys548Tyr mutation originally found in German Shepherd CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERGlanzmann Thrombasthenia Type I, (GT); mutation originally found in Pyrenean Mountain DogCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERHereditary Elliptocytosis CLEAR
MODE OF INHERITANCE
GENOTYPEC/C
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERHereditary Phosphofructokinase (PFK) Deficiency-CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERMacrothrombocytopenia; disease-linked variant originally found in Norfolk and Cairn Terrier CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERMay-Hegglin Anomaly (MHA)CLEAR
MODE OF INHERITANCEAutosomal Dominant
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%

DISORDERPrekallikrein Deficiency CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERPyruvate Kinase Deficiency; mutation originally found in BeagleCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERPyruvate Kinase Deficiency; mutation originally found in PugCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERPyruvate Kinase Deficiency; mutation originally found in West Highland White TerrierCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERTrapped Neutrophil Syndrome, (TNS)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERVon Willebrand's Disease (vWD) Type II CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEA/A
SEVERITYConsiderable
PREVALENCE (ALL DOGS)1.30%

Dermal Disorders

DISORDEREpidermolysis bullosa, dystrophic; mutation originally found in Central Asian Ovcharka CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDEREpidermolysis bullosa, dystrophic; mutation originally found in Golden RetrieverCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERHereditary Footpad Hyperkeratosis, (HFH) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERIchthyosis; mutation originally found in Great Dane CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERLamellar Ichthyosis, (LI)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERLigneous Membranitis CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERMusladin-Lueke syndrome, (MLS)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERX-Linked Ectodermal Dysplasia, (XHED)CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%

Endocrine Disorders

DISORDERCongenital Hypothyroidism; mutation originally found in Tenterfield TerrierCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERCongenital Hypothyroidism; mutation originally found in Toy Fox- and Rat TerrierCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%

Immunologic Disorders

DISORDERAutosomal Recessive Severe Combined Immunodeficiency, (ARSCID)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERComplement 3 (C3) DeficiencyCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERSevere Combined Immunodeficiency in Frisian Water Dogs, (SCID) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERX-linked Severe Combined Immunodeficiency (XSCID); mutation originally found in Basset HoundCLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERX-linked Severe Combined Immunodeficiency (XSCID); mutation originally found in Cardigan Welsh Corgi-CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%

Metabolic Disorders

DISORDERGlycogen Storage Disease Type II or Pompe's Disease, (GSD II)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G

SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERGlycogen Storage Disease Type IIIa, (GSD IIIa)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERHypocatalasia or AcatalasemiaCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERIntestinal Cobalamin Malabsorption or Imerslund-Gräsbeck Syndrome, (IGS); mutation originally found in Beagle CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERIntestinal Cobalamin Malabsorption or Imerslund-Gräsbeck Syndrome, (IGS); mutation originally found in Border Collie CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERMucopolysaccharidosis Type IIIA, (MPS IIIA); mutation originally found in DachshundCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERMucopolysaccharidosis Type VII, (MPS VII); mutation originally found in Brazilian TerrierCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERPyruvate Dehydrogenase Phosphatase 1 (PDP1) DeficiencyCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%

Muscular Disorders

DISORDERCavalier King Charles Spaniel Muscular Dystrophy, (CKCS-MD)CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERCentronuclear Myopathy, (CNM); mutation originally found in Great Dane CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEA/A
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERCentronuclear Myopathy, (CNM); mutation originally found in Labrador Retriever CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERDuchenne or Dystrophin Muscular Dystrophy, (DMD); mutation originally found in Golden RetrieverCLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEA/A
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERDuchenne or Dystrophin Muscular Dystrophy, (DMD); mutation originally found in Norfolk Terrier CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERMuscular Dystrophy (MDL), Ullrich-type; mutation originally found in Landseer CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERMuscular Hypertrophy (Double Muscling) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERMyotonia Congenita; mutation originally found in Australian Cattle Dog CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERMyotubular Myopathy; mutation originally found in Rottweiler CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEA/A
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%

Neurologic Disorders

DISORDERAlaskan Husky Encephalopathy, (AHE) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERBandera's Neonatal Ataxia, (BNAt)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERBenign Familial Juvenile Epilepsy or Remitting Focal EpilepsyCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEA/A
SEVERITYMild
PREVALENCE (ALL DOGS)1.11%
DISORDERCerebral Dysfunction; mutation originally found in Friesian Stabyhoun CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERDandy-Walker-Like Malformation (DWLM); mutation originally found in Eurasier CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable

PREVALENCE (ALL DOGS)< 1%
DISORDEREarly-Onset Progressive Polyneuropathy; mutation originally found in Alaskan MalamuteCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDEREarly-Onset Progressive Polyneuropathy; mutation originally found in GreyhoundCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERFetal Onset Neuroaxonal Dystrophy, (FNAD)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERHereditary Ataxia or Cerebellar Ataxia; mutation originally found in Old English Sheepdog and Gordon Setter CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEA/A
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERHyperekplexia or Startle DiseaseCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERHypomyelination; mutation originally found in Weimaraner CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERL-2-Hydroxyglutaric aciduria, (L2HGA); mutation originally found in Staffordshire Bull TerrierCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERLagotto Storage Disease, (LSD) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERNeonatal Cerebellar Cortical Degeneration or Cerebellar Abiotrophy, (NCCD)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERNeonatal Encephalopathy with Seizures, (NEWS) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERNeuronal Ceroid Lipofuscinosis 1, (NCL1); mutation originally found in DachshundCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERNeuronal Ceroid Lipofuscinosis 10, (NCL10); mutation originally found in American BulldogCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERNeuronal Ceroid Lipofuscinosis 12, (NCL12)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERNeuronal Ceroid Lipofuscinosis 8, (NCL8); mutation originally found in English Setter CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERNeuronal Ceroid Lipofuscinosis, (NCL7); mutation originally found in Chinese Crested Dog and Chihuahua CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERProgressive Early-Onset Cerebellar Ataxia; mutation originally found in Finnish HoundCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERSpinal DysraphismCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERSpinocerebellar Ataxia with Myokymia and/or Seizures (SCA) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERSpinocerebellar Ataxia/ Late-Onset Ataxia (SCA, LOA) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERX-Linked Tremors; mutation originally found in English Springer Spaniel CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEA/A
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%

Neuromuscular Disorders

DISORDERCongenital Myasthenic Syndrome (CMS); mutation originally found in Labrador Retriever CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERCongenital Myasthenic Syndrome, (CMS); mutation originally found in Old Danish Pointing Dog CLEAR

MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDEREpisodic Falling, (EF)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERExercise-Induced Collapse, (EIC)CLEAR
MODE OF INHERITANCEAutosomal Recessive (Incomplete Penetrance)
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)1.76%
DISORDERGloboid Cell Leukodystrophy or Krabbe's Disease, (GLD); mutation originally found in Irish SetterCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEA/A
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERGloboid Cell Leukodystrophy or Krabbe's Disease, (GLD); mutation originally found in TerriersCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEA/A
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERGM2 Gangliosidosis, mutation originally found in Japanese Chin CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERGM2 Gangliosidosis; mutation originally found in Toy PoodleCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%

Ocular Disorders

DISORDERCanine Multifocal Retinopathy 1, (CMR1); Mastiff-related breeds mutationCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYMild
PREVALENCE (ALL DOGS)1.61%
DISORDERCanine Multifocal Retinopathy 2, (CMR2); mutation originally found in Coton de TulearCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERCanine Multifocal Retinopathy 3, (CMR3); mutation originally found in Lapponian HerderCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERCone Degeneration, (CD) or Achromatopsia CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERCone Degeneration, (CD) or achromatopsia; mutation originally found in German Shepherd CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERCone Degeneration, (CD) or Achromatopsia; mutation originally found in German Shorthaired PointerCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERCone-Rod Dystrophy 1, (crd1); mutation originally found in American Staffordshire Terrier CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERCone-Rod Dystrophy 2, (crd2); mutation originally found in Pit Bull Terrier CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERCone-Rod Dystrophy, (cord1-PRA / crd4)CLEAR
MODE OF INHERITANCEAutosomal Recessive (Incomplete Penetrance)
GENOTYPE-/-
SEVERITYMild
PREVALENCE (ALL DOGS)3.47%
DISORDERCone-Rod Dystrophy, Standard Wirehaired Dachshund, (crd SWD)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERDominant Progressive Retinal Atrophy, (DPRA)CLEAR
MODE OF INHERITANCEAutosomal Dominant
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERGolden Retriever Progressive Retinal Atrophy 2, (GR_PRA 2) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERPrimary Hereditary Cataract (PHC); mutation originally found in Australian ShepherdCLEAR
MODE OF INHERITANCEAutosomal Dominant (Incomplete Penetrance)
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERPrimary Lens Luxation, (PLL)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)1.41%
DISORDERPrimary Open Angle Glaucoma, (POAG); mutation originally found in BeagleCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYConsiderable

PREVALENCE (ALL DOGS)< 1%
DISORDERPrimary Open Angle Glaucoma, (POAG); mutation originally found in Norwegian Elkhound CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERProgressive Retinal Atrophy Type III, (PRA type III); mutation originally found in Tibetan Spaniel and Tibetan Terrier CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERProgressive Retinal Atrophy, (CNGA1-PRA); mutation originally found in Shetland Sheepdog CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERProgressive Retinal Atrophy, (PAP1_PRA); mutation originally found in Papillon and Phalene CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERProgressive Retinal Atrophy, (PRA); mutation originally found in Basenji CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERRod-Cone Dysplasia 1, (rcd1); mutation originally found in Irish SetterCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERRod-Cone Dysplasia 1a, (rdc1a); mutation originally found in SloughiCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERRod-Cone Dysplasia 3, (rcd3)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERX-Linked Progressive Retinal Atrophy 1, (XLPRA1) CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERX-Linked Progressive Retinal Atrophy 2, (XLPRA2) CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%

Other Disorders
DISORDERAmelogenesis Imperfecta, (AI) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERCongenital Keratoconjunctivitis Sicca and Ichthyosiform Dermatosis, (CKCSID)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERDental Hypomineralization; mutation originally found in Border Collie CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERNarcolepsy; mutation originally found in Dachshund CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERNarcolepsy; mutation originally found in Doberman PinscherCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERNarcolepsy; mutation originally found in Labrador Retriever CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERPersistent Müllerian Duct Syndrome, (PMDS); mutation originally found in Miniature SchnauzerCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERPrimary Ciliary Dyskinesia, (PCD)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%

Pharmacogenetics
DISORDERMulti-Drug Resistance 1, (MDR1) or Ivermectin SensitivityCLEAR
MODE OF INHERITANCEAutosomal Dominant
GENOTYPE-/-
SEVERITYModerate
PREVALENCE (ALL DOGS)1.68%

Renal Disorders
DISORDERCystinuria Type I-A; mutation originally found in Newfoundland Dog CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERCystinuria, Type II-A; mutation originally found in Australian Cattle Dog CLEAR
MODE OF INHERITANCEAutosomal Dominant
GENOTYPE-/-

SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERFanconi Syndrome CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERHyperuricosuria, (HUU)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)2.59%
DISORDERPolycystic Kidney Disease in Bull Terriers, (BTPKD) CLEAR
MODE OF INHERITANCEAutosomal Dominant
GENOTYPEG/G
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERPrimary Hyperoxaluria, (PH); mutation originally found in Coton de TulearCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERProtein Losing Nephropathy, (PLN); NPHS1 gene variant CLEAR
MODE OF INHERITANCE
GENOTYPEG/G
SEVERITYModerate
PREVALENCE (ALL DOGS)< 1%
DISORDERRenal Cystadenocarcinoma and Nodular Dermatofibrosis, (RCND) CLEAR
MODE OF INHERITANCEAutosomal Dominant
GENOTYPEA/A
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERX-Linked Hereditary Nephropathy, (XLHN)CLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPEG/G
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERX-Linked Hereditary Nephropathy, (XLHN); mutation originally found in Navasota DogCLEAR
MODE OF INHERITANCEX-linked Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%

Skeletal Disorders

DISORDERChondrodysplasia; mutation originally found in Norwegian Elkhound and Karelian Bear DogCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERCleft Palate; Cleft Lip and Palate with Syndactyly; ADAMTS20 gene mutation originally found in Nova Scotia Duck Tolling Retriever CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERCleft Palate; Cleft Lip and Palate with Syndactyly; DLX6 gene mutation originally found in Nova Scotia Duck Tolling RetrieverCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEC/C
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERCraniomandibular Osteopathy, (CMO)CLEAR
MODE OF INHERITANCEAutosomal Dominant (Incomplete Penetrance)
GENOTYPEC/C
SEVERITYConsiderable
PREVALENCE (ALL DOGS)< 1%
DISORDERHereditary Vitamin D-Resistant Rickets, (HVDRR) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDEROsteogenesis Imperfecta, (OI); mutation originally found in Beagle CLEAR
MODE OF INHERITANCE
GENOTYPEC/C
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDEROsteogenesis Imperfecta, (OI); mutation originally found in DachshundCLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPET/T
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERSkeletal Dysplasia 2, (SD2)CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPEG/G
SEVERITYMild
PREVALENCE (ALL DOGS)< 1%
DISORDERSpondylocostal Dysostosis CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYSevere
PREVALENCE (ALL DOGS)< 1%
DISORDERVan den Ende-Gupta Syndrome, (VDEGS) CLEAR
MODE OF INHERITANCEAutosomal Recessive
GENOTYPE-/-
SEVERITYConsiderable

PREVALENCE (ALL DOGS)< 1%

A majority of the tested genetic disorders follow either a recessive (autosomal or X-linked) or dominant pattern of inheritance. The results are reported as 'Clear', 'Carrier' or 'At risk' for recessively and 'Clear' or 'At risk' for dominantly inherited disorders.
The genotype column shows the actual genotype of your dog at the measured site (locus) of the genome. At each site, your dog carries two alleles (genetic variants), separated by a forward slash: one inherited from its dam, and the other from its sire. E.g. for a dominantly inherited disorder, this column reveals whether an affected dog is heterozygous or homozygous for the disease mutation (i.e., carries one or two copies of it). Without examination of the parents' genomes, it is not possible to tell which one of the alleles is inherited from the dam and which from the sire.
Please read the disease descriptions carefully, should your dog turn out to be a carrier or at risk and share this information also with your veterinarian.

COMMENTS

From Sue Pearce Kelling of Optigen :

Although I think the Wisdom panel approach to testing can offer useful information, it can also be somewhat confusing at best —and unintentionally misleading at worst— for breeders to see test results for various diseases/mutations that are not known to occur in their breed. For example, there are multiple mutations for PRA (including the just recent finding at OptiGen that rcd4 PRA occurs in miniature poodles) but most of these PRA mutations are restricted to only one breed. As with all DNA tests, a strong counseling and educational support is important so that owners can clearly understand what the results mean for their dog and their breed.

In general, I think the Wisdom panel is based on sound science and I appreciate that the company honors any/all intellectual property where it exists on these tests—meaning that they do not sell tests without licensing if there are patents on the tests. I'm not sure but I don't think Wisdom directly funds any research on inherited diseases in dogs—something that OptiGen strives to do and donates significant resources to —including work on the poodle disease projects (e.g. cataracts and Optic Nerve hypoplasia) —the same projects that the poodle health foundation has also supported in Dr. Aguirre's laboratory.
In summary, I'd say the Wisdom panel is among the few "good guys" currently out there in the field of canine DNA testing. There are certainly some examples at the other end of the spectrum. I don't know what level of quality assurance the Wisdom laboratory provides and this is something that is, in my opinion, critical to the value of any test report and is another area of considerable investment by OptiGen. OptiGen achieved the "gold standard" of laboratory certification called ISO17025 in 2014 and has been certified annually by this independent assessment of quality ever since. It is quite a high bar to achieve and few laboratories in the field can claim it.

There are many factors of course that breeders consider when choosing where to test. All of us at OptiGen appreciate the loyalty of the poodle owners who we have served over the years.

From Natalie Tessier:

There are a few tests out now to test for genetic diversity. The Optimal Wisdom panel tests for all kinds of disease genes not even found in Miniature Poodles.
You get color genes, which is good but the diversity information is not super complex or particularly relevant, because they simply count up how many of the markers they test for are homozygous

In particular, note that any condition may exhibit incomplete penetrance on the phenotypic level, and the onset, expression and progressivity of the disease may be influenced by other genetic and environmental factors. For instance, not all dogs with the result 'At risk' will necessarily manifest the condition. Note that the disease severity rating presented here is only suggestive and it should not be used for other purposes. Always consult your veterinarian for the most accurate information on your dog's health status and available treatment options.

RECOMMENDED READING

» Panel Testing of Canine Inherited Disorders Provides Great Opportunities, But Requires a New Level of Information Management» Introduction to panel testing of canine inherited disorders and reporting of the test results
» Genome-wide analysis identifies carriers of a known bleeding disorder in Finnish Hounds and Welsh Springer Spaniels
SEE NEXT: Traits

Better Bred : Using the UC-Davis Canine Diversity Testing

by Joan Harrigan

The Genetic Diversity Test developed by Dr. Niels Pedersen and his team at the University of California-Davis Veterinary School opened a door for breeders. Instead of relying on pedigrees and calculating Coefficients of Inbreeding (COIs), breeders could compare the genes carried by prospective mates. Mating genetically different dogs doesn't eliminate the risk of producing genetic defects in the offspring, but Dr. Pedersen has demonstrated that it can reduce the risk.

Dr. Pedersen's vision was to create the tool; a poodle breeder from upstate New York set out to create a website to facilitate its use. Natalie Green Tessier of Buffalo, N.Y. bought her first Standard Poodle in 1987. "I picked the cute one," she recalls. "And she became my favorite dog ever." Tessier, a journalist, began to read the writings of John B.Armstrong, PhD, a professor of biology and genetics at the University of Ottawa. TheArmstrong family owned poodles, and hislove of the breed and personal experience with the genetic issues they faced inspired his study of the effects of inbreeding. His research expanded to other breeds as well, with the purpose of improving the health and longevity of purebred dogs.

Dr. Armstrong's research was ended by his death more than a decade ago, but Tessier and other breeders with similar interests continued the cause through private email lists and the Poodle Health Registry (www.poodlehealthregistry.org), an independent, international open database for owners to list poodles affected by a variety of health conditions. While helpful, the site depended upon owner registration of affected dogs. And, while a pedigree database facilitated research, it was impossible to know the genetics involved. These breeders recognized the problem—the overuse of popular sires and two significant genetic bottlenecks. The Wycliffe and Mid-Century Bottlenecks occurred in the last century, and today's show pedigrees largely trace back to the five founding Wycliffe poodles and 10 dogs bred between 1948 and 1953 that were behind the influential Wycliffe and Bel Tor show lines.

Tessier and her mentor—Mary Jane Weir, past president of the Poodle Club of Canada, and currently its health officer—joined forces and spent ten years researching pedigrees, studying genetics and health issues, and looking for Standard Poodles with low Wycliffe and MCB factors. In North America, they found a 15% Wycliffe bitch and they imported poodle from Russia and the former Czechoslovakia in hopes of finding genetically diverse dogs. While the Czech imports proved to be heavily Wycliffe-influenced, some of the Russian dogs were found to have uncommon genes. None, however, were totally devoid of Wycliffe ancestry.

"There was no health testing behind the imported dogs," Tessier says. "But, you have to start somewhere." To assess the progress of their breedings, they needed to know the genetics behind their breeding stock. In 2014, Tessier wrote to Dr. Pedersen asking for help. "He told me that if I could get samples from 100 unusual dogs, he could work with us," Tessier recalls. In just a few months, she had genetic material from 150 Standard Poodles – the UCDavis database now contains more than 700 samples. Funding for the testing came from grants from the Poodle Club of America Foundation, as well as assistance from the Poodle Club of Canada, individual breeders, and owners. Thanks to their efforts, Dr. Pedersen could finish his analysis of Standard Poodles, and today, they join Alaskan Klee Kai and Italian Greyhounds as the breeds for which the Canine Genetic Diversity Test is complete.

To read the complete article:
http://www.onlinedigitalpubs.com/publication/?i=356422&m=&id=8717&p=140
124 – November/December, 2016
Short URL: http://caninechronicle.com/?p=116137

Better Bred is an online database using pre-reviewed research based algorithms to analyze the Canine Diversity test data input by owner. Breeders can search the population of dogs on Better bred to find existing genetically diverse mates for their breeding stock.

Annual Full Membership offers access to all articles and user groups. Members can upload their dog's genetic profiles and additional information including health testing and photos. This also includes potential breeding lists and analyses. They can make these dogs public or keep them private. $75 annually.

There are 142 Miniatures in the BetterBred database, 86 of them are public - so you can compare your dogs to others.

https://www.betterbred.com/breeds/breed-comparisons/

The VGL testing is growing quickly, after poodles set a great example. Natalie Tessier will be running a course for breeders in January, This is being run with the full cooperation of the Veterinary Genetics Lab (VGL) at UC Davis. This course will fully prepare you to get a full analysis of your breed. A single person can do it, a small team can do it, or representatives of a breed club can do it.

Lisa Kimberly Glickman is organizing a seminar which should be interesting... Nathalie Tessier will be coming to Montreal !

CARE

CANINE ADVANTAGE RESOURCES & EDUCATION 2017

CONFERENCE INFORMATION
OCTOBER 14 & 15 2017
LAVAL QUEBEC

Some of the biggest talent, movers and shakers in the canine world are coming to Montreal, Quebec, Canada to share their passions and knowledge. Educational Seminars, workshops, hands-on, Demos, Vendors and Educational booths. Judges Education (Masters of Disguise). Sign up for one or two days. There will be time on both days to have sidebars, mini-meetings in dedicated spaces with peers and mentors on topics of special interest. Grassy field to exercise your pet. OFFA Eye Clinic (pending minimum numbers met) & CGN testing. DEMOS: Obedience, Agility, Rally & more.

HEADLINERS, the list is not exhaustive.

1) **Anders Rosell**, Avatar Kennels, Sweden/Spain: will present on Breeding, Showing, and Grooming with a demo and Hands-On workshops.

2) **Christine Scruggs MDV**, Tivin Standard Poodles, Connecticut: will present on canine reproduction issues, genetics, health & structure.

3) **Natalie Green Tessier**, Betterbred.com, Poodles de Grenier, New York: will present on the Dr. Pederson /UC Davis Dog genetics study and how to understand the test results.

4) **Jac Harbour**, Tudorose Standard Poodles, Oregon: will discuss puppy testing and choosing the puppy for the right job, and training techniques.

CARE CONFERENCE DETAILS

You will receive a more precise schedule in which the Seminar(s) in each block will each be identified so that you may choose which seminars you wish to attend. First come, first serve. Everyone will receive the updated information via email on the same date.

Both days: 8.30 a.m. Registration/coffee Start time 9 a.m. SHARP	
SATURDAY 14 OCTOBER 2017	**SUNDAY 15 OCTOBER 2017**
9:00- 11:00 a.m. Block A Seminar #1	**9:00- 11:00 a.m. BLOCK E** Seminar #5
Break	Break
11:15-12:30 BLOCK B Seminar #2 Hands-on	**11:15-12:30 BLOCK F** Seminar #6 Hands-On
Lunch	Lunch
1:30- 3:30 BLOCK C Seminar #3 Group Panel	**1:30- 3:30 BLOCK G** Seminar #7 Group Panel
Break	Break
3:45-5:00 BLOCK D Seminar #4	**3:45-5:00 BLOCK H** Seminar #8

CARE

CANINE ADVANTAGE RESOURCES & EDUCATION 2017

CONFERENCE INFORMATION
OCTOBER 14 & 15 2017
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HEADLINERS BIOS:

Anders Rosell:

A Graphic Designer and Art Director by profession, Anders is a world-renowned top breeder of Standard and Miniature Poodles under the prefix 'Avatar'. Anders has been involved with Poodles since the mid 70' s and bred and owned many Top and BIS winners. As a small scale breeder Avatar has bred champions in 20 different countries all over the world. The Avatar dogs are to be found behind top winners all over the globe. At prestigious shows such as the PCA (Poodle Club of America National Show) his dogs have produced BIS, BIS puppy, BIS Veteran, 3 different Winners Dog, many Award of Merit winners as well as BIS Stud dog and BIS Brood bitch. Anders is the only European Standard Poodle breeder who has bred/owned four generations of American Top Producers. Six different stud dogs bred or owned by Avatar have produced Group winners in North America only. He has also bred and owned World Winners in Europe, Junior World Winners, Veteran World Winners, Dog Of The Year Winner in Australia, as well as Top Poodle Bitch in Canada. Anders is also a licensed grooming judge and has judged at competitions such as Intergroom in America, the Oster Invitational Tournament in Germany, Groomania in Belgium, Artero International Championship in Spain, Scandinavian Master Groom in Sweden. As an educator he has held many grooming seminars and workshops for Poodles, which includes countries such as the U.S, Canada, England, Australia, Russia, Israel, Scandinavia as well as numerous countries all over Europe. He used to be the co-owner and publisher of the famous breed magazines The Scandinavian Poodle Magazine and Scandinavian Sighthounds – The Journal. Anders is currently living and working in Malaga, Spain.

Dr. Christine Scruggs MDV:

Christine started her kennel Tivin in between college and veterinary school, however her relationship with poodles started in childhood as her mother also bred poodles under the prefix Tiva.
Christine breeds for the total dog, as she does compete in performance events as well as conformation. As a veterinarian, she is intimately familiar with the health challenges faced by the breed. One of her areas of expertise is canine reproduction. Christine is also the author of multiple articles published in various journals.

Natalie Green Tessier:

Natalie has had standard poodles since 1987 and bred her first litter in 2006. Learning about the state of the narrow gene pool and health issues in standard poodles inspired her to looks for unusual pedigrees and get involved seriously in an effort to preserve this historic breed. Natalie is one of the founding members of the Standard Poodle Project, and acted as an advisor to Dr Niels Pederson of the UC Davis Veterinary Genetics Lab. More recently, she designed a program and software which analyzes the data of hundreds to thousands of dogs in the database with information gleaned from the aforementioned study to help breeders choose the best mate choice (genetically, for maximum diversity) for a number of breeds facing bottlenecks including Akitas, English Bulldogs, Dobermans & Havanese.

Jac Harbour:

Jac has been breeding Standard poodles conformation, obedience and field titled Standard Poodles as well as family companions, therapy dogs and service dogs under the pre-fix Tudorose since 1972. She has bred some of the most diverse standard poodles in the gene pool today, many service dogs and hunting dogs, and has been a mentor to many. Since 2009 she has been a partner in Trainer's To The Rescue LLC. Jac founded hearing ear dogs & developed special skills dogs both now under the umbrella of the Lions Foundation of Canada. Among numerous awards she has been awarded include Woman of the Year and the Commemorative Medal for the 125th anniversary of the Confederation of Canada in recognition of significant contribution to compatriots, community and to Canada 1991.

On Testing For Every Genetic Mutation Under The Sun...

July 18, 2016 Natalie Green Tessier

Why not offer a huge panel of disease gene identifications?

There are a number of companies who do this, and do a good job of it. This is a wonderful thing for adopted mixed dogs, since there's no way to know what's in them, but our Better-Bred.com service is for breeders.

While we understand the attraction of knowing every possible mutation in a dog, the truth is, our philosophy simply differs, because we aren't assessing pets - we are assessing dogs that will be the parents for the next generation.

Breeders have become accustomed to the great search for The Gene.

We think, once we've admitted there's a problem in a breed, that if we can just find The Gene, then we can breed it out, and save the breed. In some cases this is true and has been an enormous benefit for a breed. Whole lines have been saved, diversity has been salvaged and by breeding out affected and carefully breeding to clear dogs, a recessive mutation can slowly disappear from a population.

How is that not a great thing?

As is often the case for dog breeders, dedicated, enthusiastic, passionate dog breeders, we often think that if some is good, more is better.

We want the best. We want the purest. We want to see in our whelping box the results of all our hard work - perfect puppies.

Playing with genes, which is what breeders do, is an enormous responsibility. That's why we take it so seriously. It's also incredibly complex. Dogs have over 19,000 genes, and over 2.8 billion base pairs in their DNA. (Base pairs are the little rungs on the spiral ladder that makes up the DNA.) One small change in one gene, in a base pair or two, can mean different traits in an animal. The differences can be huge or tiny, evident or not, show up immediately or show up later.

In every animal, there are tiny little blips in the code - a rung or several on the spiral ladder that is a little different from the last generation's spiral ladder. In 2.8 billion of these base pairs, how obvious is it that a tiny fraction of them will reproduce a little wonky when making new sperm and eggs? The miracle is, in fact, that so many reproduce perfectly. It's important to know that flaws happen though, all the time, in all animals, in every generation. These mutations are usually either problematic or harmless, and very occasionally they are a benefit to the animal. The first white snowshoe rabbit was very glad for its new mutation, I'm sure! A similar mutation is not such a happy event in the white tailed deer.

So mutations happen, and only rarely are they of such benefit that they help creatures survive, but we humans do love to spot the pretty mutations and promote them. Think of merle dogs - so pretty, but most likely popular because humans find it pretty, since dogs with two copies of the merle gene do not thrive.

Just as we keep the mutations we like around, there are invisible ones happening as well, and they tend to become common in specific breeds without us knowing. Sometimes this is just because they happened to be in an otherwise fantastic sire that everyone used. Sometimes they are in a popular line. Sometimes we have a breed founded on very few dogs, and those founders had their mutations. This is why we see certain recessive disease mutations in certain breeds - but we only see them once they start meeting mutations of precisely the same type, and this happens most when animals have an ancestor in common.

This is why we breeders are always on the lookout for recessive genes, and researchers are more than happy to look for these recessive mutations. In these breeds, affected dogs ALWAYS have two copies of the recessive mutation, and healthy dogs that produce the disease ALWAYS have a single copy of the mutation. These are what Dr. Niels Pedersen calls the "low hanging fruit" of the genetic diseases. Researchers make the tests and we breeders buy them, for hundreds of dollars each time, for identification of one mutated gene at one locus.

So as genetic research has rapidly improved and become more affordable, a few smart people

We think, once we've admitted there's a problem in a breed, that if we can just find The Gene, then we can breed it out, and save the breed.

affected dogs ALWAYS have two copies of the recessive mutation, and healthy dogs that produce the disease ALWAYS have a single copy of the mutation.

by breeding out affected and carefully breeding to clear dogs, a recessive mutation can slowly disappear from a population.

decided to test for all the known mutations at the same time. This, of course, makes perfect sense from a researcher's standpoint, and in terms of cost effectiveness, why would anyone keep charging breeders for single locus disease tests when you can test for them all in one shot? With a single test a dog can be tested for dozens or hundreds of mutations at a single time. Bonus!! Right? Well…. sort of.

First, as we've already seen, **mutations tend to be breed specific.** This is because genes interact. A deadly gene in one breed may not be so deadly or could even be perfectly harmless in another breed. A mutation may be found in one breed and nonexistent in another. And some markers that are tested for aren't even the cause of the gene at all, they just happen to be found in affected dogs because they are near the actual mutation, so of course in another breed these markers are likely to be meaningless.

So it's important to know a great deal about each of the mutations and each of the markers and their relation to a breeder's breed.

But breeders don't think like scientists - we think like breeders. For a century, our community goal was to produce ever more perfect dogs that were ever more similar to the breed standard. Improvement was the order of the day. Inbreeding became popular because it was the fastest way to improve the next generation - and only later did we realize that it was also **the fastest way to spread an invisible mutation or concentrate the genetic elements for complex diseases.**

Here are some facts we breeders have to learn by heart. Every living creature carries with it mutations. Let's repeat that for emphasis: EVERY LIVING CREATURE CARRIES MUTATIONS.

Breeders, always trying to improve their breed, want to breed out mutations. One mutation is easy to breed out.

How about two? Tricky but doable.

Three? Ugh.

Four? Come on.

Five? "I won't have that dog in my breeding program." Right? And yet your dogs, your beautiful, healthy, robust dogs in fact do have mutations. They are just unseen by you, probably are ones that are exceedingly rare and they will probably never cause a problem.

So what happens when you run a panel and there are several mutations, all from other breed research, in your otherwise healthy dog? The idea of finding unknown mutations seems great - but when a few show up in your dog, what will you do? Many breeders will simply not breed from a dog with several known mutations - even a healthy one. Those mutations may mean nothing about the health of your dog, but can you justify breeding such a dog? How do you explain it to other breeders? You don't. You most likely just eliminate an otherwise fine specimen of the breed, most likely unnecessarily.

Let's imagine another dog - one that passes everything and its large panel test shows no mutations or faults found of any kind. Wow! What a relief. Proudly, you can boast, this dog is perfect. DNA clear of everything. You can breed this handsome boy to anyone and anything and you are only improving the breed by doing so, right? And other breeders may flock to use your boy. Yay!

Right? Well...sort of.

Let's review. EVERY LIVING CREATURE CARRIES MUTATIONS. This one too. You just can't see this dog's mutations. They haven't been found yet, but after he sires hundreds of puppies, eventually you may well find out as generations pass and the lines eventually double up. This will happen more rapidly in breeds with fewer breeding dogs.

So if all dogs CARRY MUTATIONS (and they do), if we don't test for them, how do we know what's there and how do we get rid of them?

You do what nature does - you breed dogs that aren't very similar genetically. And nature doesn't identify the mutations first - it just avoids them being found in both dam and sire.

Most mutations are recessive. They are a one time, random error in the DNA, and only happen in a single sperm or egg. That sperm or egg will meet up with the egg or sperm of another dog that is almost certain not to have the same mutation in the same place. Together, the gene pair - the healthy one from the mate and the new mutation from the first dog will function fine because the healthy gene will do its job. In a recessive disease, you need the new mutation to meet up with one precisely like it to cause the disease. When you breed to very different dogs, you raise the probability that whatever mutations in there are in the two dogs (because ALL DOGS CARRY MUTATIONS) will never, ever meet their match.

Inbreeding as a method has been used a long, long time. When you see a pedigree that has the same dog appear many times, you can bet that there was some culling along the way, because **inbreeding means recessive mutations are more likely to meet**. There isn't an experienced dog breeder who won't tell you that severe inbreeding requires a strong constitution. While it is also true that severe inbreeding can "purify a line" from recessive diseases, there are other biological deficits from repeated generational inbreeding that cause other problems. It's just a trade.

Nature has been at this much longer (about 4 billion years) and the most successful strategies are the ones that improve survivability. All breeding pairs instinctively try to detect genetic differences when looking for a mate. Again, animals can, of course, look just the same and still be very different genetically. (Think of a red fox. Now think of another red fox. See what I mean?)

When you breed for diversity, what you are really doing is breeding so that NO mutations ever meet their precise matches. You can still select for traits you like, but there's no good way to get rid of multiple mutations just because you know they are there. Test all you like - whatever your results are, you already know that your dog has mutations. **Whether you are breeding to get rid of known recessive mutations or trying to avoid doubling up on unknown ones, the most effective way to do that is to breed unrelated animals with similar positive qualities.**

So that's why BetterBred.com won't offer large panel single locus disease gene tests. There are great other companies that will. Instead, we **offer the ability to know whether dogs are genetically unrelated, distantly related, or very related, based on genetics,** and we think that's a better way to breed healthy dogs and preserve gene pools.

81-Year-Old Agility Competitor Defies Stereotypes In The Ring

By: Ranny Green | may. 06, 2016

Maggie makes her way down the A-frame during the 2016 Masters Agility Championship at Westminster. Photo by Steve Surfman, Westminster Kennel Club

Athleticism and age tend to be combative traits and polarized positions for most. But when it comes to 81-year-old Marge Yonda, you can throw the book away.

A mother of five, grandmother of 11 and great-grandmother of one, she has mimicked the Energizer Bunny while segueing from marathon running to American Kennel Club agility competition the past decade.

But it hasn't been easy.

Injuries have served up momentous challenges for her and her 9-year-old Standard Poodle partner, Maggie, but they have only pushed the pause button momentarily before recharging into overdrive and back into the ring.

While in her 50s and 60s, Yonda ran eight marathons – all under 4 hours and with a best time of 3 hours, 45 minutes – and engaged in plenty of bicycle touring worldwide with her husband, Tony, including a 4,650-mile cross-country trip, and worked out regularly on an elliptical trainer.

While the five children were growing up, the family owned five dogs – a Dalmatian, two Brittany Spaniels, Boxer, and an All American dog – but when Tony and Marge became empty nesters they traveled to 53 countries and didn't own a dog for two decades.

"At that time in our lives," Yonda explains, "we spent four months every winter in our RV in Mexico, and were parked next to a couple with a Standard Poodle. We fell in love with the dog -- and the breed – and liked the fact it did not smell or shed. We had no idea how smart the breed is, nor its happy-go-lucky temperament."

After leaving Mexico on one of those trips, they flew to Oregon, where they found their first Standard Poodle puppy. However, she suffered from a stomach ailment and died a short time later from a blood clot following surgery.

They allowed time to heal before searching for another Standard. In the spring of 2007, they purchased Maggie from a Connecticut breeder/veterinarian and were introduced to agility in a puppy obedience training class.

"There I was at age 72 starting with my first agility dog who was 4 months old. She entered her first trial at age 15 months," Yonda recalls. Four years later Yonda began worrying about what would happen if Maggie became ill or injured. She remedied that by getting a second Standard Poodle, Mandy, from the same breeder. She is now 4 and on track for her first MACH.

When a knee replacement forced her to discontinue marathon running, agility proved to be the perfect option for the energetic senior. "It saved me in a sense," says Yonda. "Here I could run short distances in a competitive environment. And it turned out Maggie was great at it, although, at that time, as a handler, I was awful."

There are many sports with partners where verbal communication is commonplace. "Agility gives new meaning to partnership and an unbreakable bond between two species. It is a unique experience, from which I have quickly learned that most mistakes are handler errors," she acknowledges.

Maggie makes her way down the A-frame during the 2016 Masters Agility Championship at Westminster. Photo by Steve Surfman, Westminster Kennel Club

Yonda says the sport offers something for everyone, no matter the age or physical impairment. "I have even seen a handler in a wheelchair," she adds. "However, to do well it is important to be physically fit. I do 40 minutes on the elliptical trainer, 50 push-ups, and 200 crunch sit-ups four times a week. I also play pool volleyball and am enrolled in exercise classes." But she adds fun to the fitness mandate.

"Sometimes that is difficult when one has just made a ridiculous error and the dog does not qualify. The key here is to keep remembering that this is supposed to be enjoyable. The dog does not know it did not qualify, is holding no grudges and continues on as if nothing happened."

Yonda directs Maggie over a hurdle in a recent competition

The Marge and Maggie Show has had it bumps in the road, too. Major ones physically. In addition to her knee replacement, last August Yonda underwent a spinal fusion with bone grafts, requiring the placement of two rods and a dozen screws and bolts. "I never had any pain post-surgery," she adds, "and I was back in the agility ring in 2½ months. I fear falling, however, and the first time back in the ring I went down flat on my back. Horrified, I laid there a short time while the judge held Maggie. I got up and finished the exercise." Ironically, Maggie had a perfect run but time expired because Yonda had been on the ground too long.

Yonda and Maggie were entered in the 2015 Masters Agility Championship at Westminster, but Yonda was hospitalized the week before with a virus and shortly thereafter Maggie fractured her ankle.

> "It took weeks to recover from the disappointment of not being able to compete, since I had been at the press conference days before promoting the event."

Maggie's recovery wasn't so quick. Her fracture was misdiagnosed as arthritis and she underwent rehab for weeks. Eventually, a Cornell University School of Veterinary Medicine practitioner determined that her continued pain was due to a fracture. He cautioned Yonda the dog might never be able to compete in agility again, but after two months of following his recovery protocol she was back in the ring.

The bulk of the team's competition is in the Northeast, although for several years while they wintered in Florida in an RV, they traveled to an agility trial almost every weekend.

Maggie's 2016 Westminster outing didn't go particularly well, either. While she had a perfect run, it was over time, costing her a place in the finals. "What I didn't know at the time," Yonda explains, "is that Maggie was sick. So sick, in fact, that we took her to an emergency veterinarian the next day for severe diarrhea, from which she quickly recovered."

While Yonda's agility ride has been packed with plenty of psychological bumps, she savors the new friendships, physical challenges, and tight partnerships she has cultivated with Maggie and Mandy along the way.

"They have been my soul mates and a grounding force on this journey," she concludes, "which has been both challenging and satisfying."

Header image: Steve Surfman, Westminster Kennel Club

Agility offers a fun activity for you and your dog, no matter your skill level.

Whether your goal is to collect ribbons or just have some fun and meet other dog lovers, agility offers something for everyone.

Learn more about the sport and find events in your area here.

Standard Poodles Over The Centuries

Ciro an

Royal or Standard

France, Germany and Russia have all claimed the Poodle. Poodles were certainly known in Italy in the 16th century and in Holland in the 17th century; they did not arrive to this country until considerable later. On the continent the poodle was used as a water spaniel, hence its name which derives from the German "puddeln" to splash in water.
Recently the larger members of this intelligent family have been less favored than their smaller parvenu brethren but there are signs that their popularity is increasing.
Height 15 inches and over, color Black, white, Brown, Blue or any solid color. Their profuse outstanding coat is of hard texture and is usually clipped in the traditional "Lion" style.

Miniture Poodles Over The Centuries

Miniature

Small Poodles, almost always white or parti-coloured, can be seen in many 17th century paintings and were certainly fashionable pets at that period but it seems likely that this were the results of a cross with a Maltese or other small breed. It was in the early years of the 20th century that the systematic and ultimately successful attempts were made to breed small, typical pure breed Poodles and in 1911 the English Kennel Club acknowledge them as a separate breed. It was not until the end of the Second World War that the bred captured the fancy of dog lovers both here and in the USA_ since then their popularity has been phenomenal.
Height under 15 inches, In all other aspects, the Miniature Poodle is a replica of the Standard poodle.

Toy Poodles Over The Centuries

Toy

The development of the Toy-size Poodle (under ten inches) goes back only 10 years. Before that time they were all white. They Toy is now bred in all the colors of the Miniature and the Standard Poodle whose history goes back more than 2000 years. He is trimmed in the same way and carries itself with the same pride. The dark, almond shape eyes are full of fire and intelligence. The type has changed but little since it was developed from larger ancestors. Cuba was the starting place, Spain next then England. The American kennel Club "approved" them in 1928.

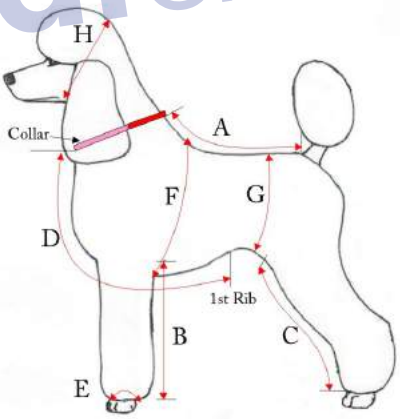
This dogs have always been favorites of the theater, and we hear of troupes of them as early as 1700 in England. They balanced balls, jumped hoops and danced their way into the hearts of the audience. Today more and more of them are joining the fashion parades at Park avenue and Fifth Avenue in New York City.

Custom made Special Items for "YOUR" dog

I started a line of clothing for special KOOL DOGZ.

I mean special because I keep in mind show coats and the special needs of show dogs. Coats are made with legs, waterproof outside and can be lined with satin. They have a coat protector so the zipper will not catch the hair.
Our coats, underwear, collars, leggings and blankets can be made custom to your needs, taste, material and sizes. Reflective material is optional.
Prices vary accordingly.

Dog Measurements



Let's start by taking the measurements of the dog:

A= Length of back
B= Length of Front leg
C= Length of back leg
D= From collar to fist rib
E= Around wider part of the paw
F= Around the widest part of the chest
G= Around waist
H= Around bigger part of the head

Every coat is made to your dog's size.
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U Ch Opus's Glick's Some Like It Hot CGN HIC THD

"We had a nice but very busy weekend in Kingston on November 19 & 20 at The Maple Leaf kennel Club.
In attendance:

U Ch Opus's Glick's Some Like It Hot CGN HIC THD (aka Chilli) who earned her URO1 with three 4th places and decent scores & CA (coursing Aptitude)

U Ch Glick's Opus Get Ready HIC (aka CocoMax - co -owned by OVPC members Gilles Boulais & Michelle Ramsay) who earned his Championship. CKC &

U Ch Glick's Speranza Primavera (aka Enzo) Co-owner handled by Michelle Jessop earned his Championship.

CKC & U Ch Glicks Midsummer Breeze ADC SHDC URO1 HIC HIT (aka Breeze) earned her Championship and her Rally Novice with a HIT and 2 HICs and two 2nd places handled beautifully by her co-owner Carolyn Stevens

All of them had prior wins from different shows but they all finished that weekend (there wee two shows a day and they took turns winning like good sports, except Enzo who deservingly as he showed beautifully won the most times and made it to group every time
On Sunday it was like a mini OVPC meet-up because Rosemary Euringer was there with two of her lovely and happy Claire x Ethan puppies impeccably groomed by Ann Curran along with new OVPC member Daren Auger. And Donna Wilson was there with the illustrious Anderson Cooper who won Bbpiss at the OVPC summer specialty, all grown up and quite the dude.
Unfortunately the venue was absolutely freezing and nobody hung around to socialize, and although I had my camera I was too cold to take many photos.

Chilli earning her new title

CocoMax - co-owned by OVPC members Gilles Boulais & Michelle Ramsay) who earned his Championship. CKC

"U Ch Glick's Speranza Primavera " aka Enzo

Bred by Lisa Kimberly Glickman
Co-owner handled by Michelle Jessop earned his Championship.

********** Rousseau & Remi**********

Glow Rousseau Veritable Lumiere is the son of Cameo Glow Lady in Red and Cameo the Big red Dog

Bred by: Gloria Koolsbergen
Own by : Natalie Tawadawa

Rousseau and Remi are two very talented pups, and long standing fans and supporters of both Ottawa Pet Expo and Ottawa Dog festival.

This clever pair entered our Pet "Expo" sure video contest in 2013 and performed on our top dog contest in 2014. Rousseau was even chosen as a #top model for Ottawa Dog Festival.

BIS BISS BPIS BPISS Am CH Cnd GCH Gardenpath Concours d'Elegante aka "Ella"

Sire: Am. GCH Dacun Kaylens He's A Heartbreaker

Dam: CH Canzone Bella Nina of Gardenpath TD CD RE CGN VCX

ELLA finished her American Championship and is well on her way to her Grand. We are very excited about her future!
Expertly handled by Allison Cowie

Bred, owned & loved by Renee S. Koch

Gardenpath Standard Poodles -

Kitchener, Ontario

Next News Letter

the poodle club of canada

Next News letter planned for March 2017,

Please I need our member's input!!!

Pictures, Interesting Articles, Health issues, Brags,

Stories, Recipes, etc.

Please send input to your editor Gloria Koolsbergen

E-mail Address poodlesglow@live.com

www.poodlesglow.com