CANINE HEREDITARY AUTOIMMUNE HYPOTHYROID DISEASE
Implications for Your Dogs' Health and Breeding Programs

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Article was reviewed for accuracy by Dr. Steven Friedenberg, DVM, PhD, DACVECC, Assistant Professor who is currently researching Addison's Disease and Autoimmune Disease at the University of Minnesota College of Veterinary Medicine.

SIGNIFICANCE

Hypothyroidism, an abnormal reduction in thyroid hormones, is the most common endocrine disease in dogs {1,24,28,45}, currently approaching 30% incidence in some breeds. All organ systems are affected by a deficiency of thyroid hormones due to slowing of metabolism. Hypothyroidism tends to emerge at the age of 4 to 10 years, is more common in larger breeds, and less common in miniature and toy breeds.

The focus of this article is autoimmune thyroiditis (AITH), which causes about 80% [28] to 95% [8,29,33,43] of hypothyroidism via progressive destruction of the thyroid gland. AITH is proven hereditary in certain breeds of dogs (familial) [6,7,8,28]. Hopefully, this article will encourage breeders and those doing performance competition to screen for hereditary hypothyroid disease, thereby promoting optimal health and reducing the incidence of the disease via selective breeding.

The overall incidence of hypothyroidism in middle-aged to older dogs is cited as 0.2-0.8% [4]. Most sources that mention incidence in dogs are limited primarily to hypothyroidism, of which we know at least 80-95% of those dogs are afflicted with AITH. Dr. Raymond Nachreiner at the endocrinology lab at Michigan State University cited actual cases of AITH: out of 50,000 canine blood samples, results indicated 9-11.5% of dogs ages 2-6 years tested positive for thyroid autoantibodies that indicate autoimmune thyroiditis [6]. According to Dr. Jean Dodds, canine thyroid disorder "has reached epidemic proportions". [42]

Determining the incidence of **AITH** in most breeds is challenging, often based on inadequate numbers and owner-reported data, often failing to differentiate the incidence of hypothyroidism versus AITH, thus preventing accurate conclusions. The data posted on the Orthopedic Foundation for Animals (OFA) website contains inadequate numbers of samples in most breeds, plus some owners chose to withhold abnormal results from the OFA.

OFA data from 1997 to 2020 https://www.ofa.org/diseases/breed-statistics#detail indicates the highest incidence of dogs afflicted with **AITH** is the English Setter at 25%, equivocal 16% (results are questionable, need to be retested in 3-6 months), and 0.3% idiopathic (unknown cause) hypothyroidism [5] out of a sample size of 1,625 dogs. OFA lists incidence of Bichons Frises affected at 0% and equivocal 2.7% out of a sample size of 110 dogs tested [5]. The Bichon Frise 2005 health survey indicated a **hypothyroid** incidence of 0.2% (2 cases) with a sample size of

1000 dogs, as well as high incidences of several health issues that could be the result of undiagnosed hypothyroidism. The incidence of **hypothyroid** disease from data collected on 1267 Bichons via internet reports from 2002 to 2007 was 1%.

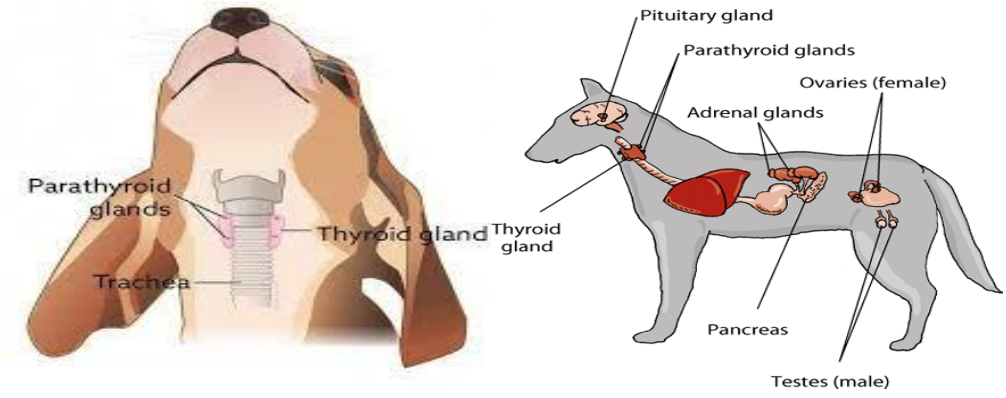
PERCEPTIONS ABOUT HYPOTHYROID DISEASE THAT ARE FALSE!

- It's a simple and rare disease in dogs that is not life threatening.
- I will know if my dog has this disease.
- It's easy to diagnose with just one blood test.
- I don't need to screen for this prior to breeding.
- It will not deter my dog from competing in conformation or performance activities.
- It will not prevent my dog from breeding successfully or having puppies.

In my opinion, screening for thyroid disease prior to breeding is as important as obtaining an ophthalmologist exam to rule out Bichon hereditary juvenile cataracts. "There is an indirect and strict association between thyroid dysfunction and reproductivity." {41} I believe that as a breeder you need to know if your male has abundant/active sperm and the stamina to successfully breed a bitch, and that your bitch is fertile and has the stamina to endure pregnancy, whelping, nursing, and raising a litter. AITH can affect fertility in dogs and bitches, viability of sperm, pregnancy & whelping, sizes of litters and puppies, survival of puppies, behavior, mental abilities, exercise tolerance, muscle strength, and the ability to compete in performance competitions**.** If your dog is afflicted with this genetic disease, do you want to pass it on to the offspring?

DESCRIPTION

This review of anatomy and physiology will help you understand autoimmune thyroiditis (AITH). The thyroid gland surrounds the trachea in the neck. It synthesizes and secretes hormones that regulate the metabolism via stimulating the utilization of oxygen by the tissues, which helps turn food into fuel. The thyroid secretes thyroxine (T4) and triiodothyronine (T3), active hormones that are sent to and exist in every cell of body. By regulating metabolism, the thyroid gland affects all organs of the body. T3 and T4 are synthesized from a protein in the thyroid gland called thyroglobulin (Tg). When T3 and T4 levels are low, the thyroid stimulating hormone (TSH, aka thyrotropin, from the pituitary gland) level increases, indicating that the endocrine system is working harder to produce sufficient T3 and T4 to meet the metabolic demands of the body.



https://rawfoodforpets.com/the-endocrine-system/ https://vcahospitals.com/know-your-pet/parathyroid-tumors

Some definitions to augment your understanding:
Hypothyroid = thyroid gland fails to produce sufficient amounts of thyroid hormones
Thyroiditis = inflammation of the thyroid gland that leads to damage
Lymphocytes = small white blood cells that are part of the immune system
B cells = one type of lymphocyte that produces antibodies to combat invading bacteria & viruses
T cells = another type of lymphocyte that combats invading toxins and foreign substances
Antigen = a toxin, foreign substance, or microorganism that elicits an immune response
Microorganism = microscopic bacteria, viruses, fungi (molds, yeast, mushrooms)
Antibodies = specialized proteins that inactivate invading foreign substances and microorganisms
Autoantibodies = aberrant antibodies generated by the body that attack itself
Thyroglobulin = a thyroid protein that synthesizes T4 and T3
Synonyms: autoimmune thyroiditis (AITH) = autoimmune hypothyroidism = lymphocytic thyroiditis = similar to Hashimoto's thyroiditis in humans

The brain is involved in regulating thyroid hormones as well as the thyroid gland. The hypothalamus (located in the forebrain) releases thyrotropin-releasing hormone (TRH) that directs the pituitary gland (located in the base of the brain) to release the thyroid stimulating hormone (TSH). The TSH instructs the thyroid gland on how much T3 and T4 to produce.

The two major components of the normal immune system that reside in the blood (lymphocytes and antibodies) are normally activated when antigens (foreign cells) enter the body such as allergens, cancer cells, bacteria, viruses, toxins, fungi, and parasites. A healthy immune system combats the foreign cells while it recognizes and differentiates the body's normal cells.

To help define the autoimmune response, Dr. Steven Friedenberg, Assistant Professor Department of Veterinary Clinical Sciences, College of Veterinary Medicine, University of Minnesota, states "In autoimmune disease, the body aberrantly recognizes its own cells, tissues, or organs as abnormal, and mounts an immune response against itself." Depending on the dog's defective genes, particular organs or glands are the target of the attack of autoantibodies in contrast to the entire body. The outcome is chronic inflammatory disease with damage of the target organ or gland. The presence of autoantibodies that attack specific organs, tissues, glands, hormones, and cells can sometimes be detected via blood tests [20,22].

The hallmark of the autoimmune process with thyroiditis is that damaged immune cells (lymphocytes) invade the thyroid gland, destroying cells, tissue, and blood vessels in the gland. Initial symptoms are mild and can mimic symptoms caused by other diseases. Early screening of thyroid hormone levels for this disease is of paramount importance for diagnosing it in the early stages, prior to onset of symptoms, to prevent permanent thyroid damage. Autoimmune thyroiditis is unlikely to be life threatening if identified early.

Potential autoimmune diseases are too numerous to list in this article, but some of the more common conditions seen in dogs in addition to thyroiditis include encephalitis, dry eye, lupus, hemolytic anemia, pemphigus (a skin disorder), rheumatoid arthritis, Addison's disease, type 1 diabetes, vasculitis, and inflammatory bowel disease. Consider that dogs with autoimmune thyroiditis may have coexisting autoimmune conditions.

CAUSES

The major cause of **primary** hypothyroidism is direct destruction of the thyroid gland via the autoimmune process. In rare cases, hypothyroidism is **secondary** to other causes that damage or suppress the thyroid gland [29,33], requiring resolution of the underlying cause:

1. Dysfunction of the hypothalamus or pituitary gland in the brain due to disease or trauma.
2. Damage to the gland due to infections, drugs, toxins, excessive dietary iodine, cancer.
3. Other endocrine diseases can impair thyroid function, at least temporarily until stabilized.
4. Recent studies suggest that gonadectomy (spay/neuter) increases the risk of autoimmune diseases, including thyroiditis [12,13,14].
5. There are rare cases of isolated congenital hypothyroidism that are inherited, appear near birth and may cause dwarfism and mental dullness.

The causes of autoimmune disease are complex, likely a combination of genetics, environmental factors that can influence the development and progression of the disease, and overstimulation or imbalance of the immune system. Typically, a trigger (inducer) occurs that turns on the autoimmune process in a dog with a specific genetic mutation that allows only the thyroid gland to be damaged. Per Dr. Ray Nachreiner, DVNM, PhD, Michigan State University, "Even though a dog may have the genes for thyroiditis, without the inducer the genes may remain dormant." [11} Simply put, dog owners have the power to influence how genes are expressed by modifying lifestyle choices for our dogs!

The major factors for development of autoimmunity in humans, which are not yet proven in dogs:

1. Intrinsic factors: polygenic inheritance, age, sex, poor nutrition, stress, and major viral or bacterial infections
2. Extrinsic factors: some cardiac or psychiatric drugs, some antibiotics or antiviral agents, chemotherapy [34], steroids [32], and potentially toxic additives found in vaccine diluent (metals, mercury, carcinogenic agents) [31]
3. Environmental factors: pesticides, toxic chemicals [36,37] that can cause endocrine disruption, and excessive pollution [32])
4. Overstimulation of the immune system by excessive vaccinations [30]
5. Interruption of the immune system's ability to regulate itself via imbalances in intestinal flora or leaky gut syndrome that can cause persistent inflammation in the body

GENETICS

Autoimmune thyroiditis is proven familial and inherited [6,7,8,11], which raises significant genetic implications for breeding dogs. The mode of inheritance has not been identified but is suspected to be polygenic, meaning **more than one** defective gene is contributed by both sire and dam. Some studies have generated suspicion of an autosomal recessive mode of inheritance, meaning **one** defective gene comes from sire and one from dam [6,11,25], but this is unlikely. Further research into the DNA marker and mode of inheritance is crucial to find a gene-based test. Hopefully, the Bichon Frise Club of America (BFCA) will consider donations to studies in search of a gene-based test to identify affected and carrier dogs, allowing for more selective breeding that can reduce the incidence of AITH.

Recent research has focused on determining the most diagnostic blood tests and describing the progression of the disease. What is known is that hypothyroidism affects male and female dogs equally in some studies and females more often in other studies. Of note is that if a pregnant bitch's level of circulating thyroid autoantibodies is at peak, during advanced stages of AITH prior to destruction of the thyroid gland, the autoantibodies may be passed to puppies via the placenta and colostrum [1], first milk produced by the mammary glands that is rich in antibodies.

POTENTIAL CLINICAL SIGNS AND SYMPTOMS

If left untreated autoimmune thyroiditis destroys the thyroid gland, which can progress to the point of heart failure, kidney failure, and/or coma, all of which can lead to death. For example, myxedema can develop in late stages of undiagnosed autoimmune thyroiditis which is evidenced by swelling of the brain, legs, face, skin, and underlying tissues (causing a waxy appearance to skin). Myxedema coma eventually develops that is a life-threatening condition.

Clinical signs are primarily the result of decreased levels of T3 and T4 due to reduced synthesis and secretion from the thyroid gland, causing reduced metabolism. The most common symptom of all the autoimmune diseases is fatigue, which may reflect in your dogs as changes in energy, appetite, and/or behavior.

The onset of AITH symptoms is variable, tending to manifest at 3 to 5 years of age [5,11]. Early subtle symptoms become more apparent when the gland is about 70-75% destroyed [29,38]. Symptoms can involve all organs of the body demonstrated by a wide ride range of clinical manifestations. What can be confusing to health care providers is that many symptoms are subtle and may mimic symptoms caused by other organic or autoimmune diseases. Once treatment is initiated, some symptoms may quickly resolve if the dog is in the early stages of the disease.

Working issues

- exercise intolerance, fatigue
- less interest in working and competing
- cold intolerance
- aching/stiffness in muscles and joints

Skin issues

- scratching, chewing feet and skin, hot spots, skin infections such as pyoderma, hair loss or thinning, dry coat/skin, dull coat, excessive shedding, seasonal allergies, frequent ear infections, thickening of skin, seborrhea (greasy, flaky skin), hyperpigmentation

Behavioral changes

- sudden personality changes at onset of puberty, incessant whining, nervousness, increased reactivity, moodiness, phobias, fear of strangers, hyperventilating, undue sweating, disorientation, failure to be attentive, aberrant aggression

Eye issues

- fat deposits in corneas (corneal dystrophy), keratoconjunctivitis sicca (dry eye) due to inadequate tear production

Neurological symptoms are evident in 7.5% of dogs with hypothyroidism [4]

- changes in mentation (dullness or dementia)
- Seizures / epilepsy

Strokes / coma
generalized weakness of skeletal muscles
polyneuropathy (muscle weakness or paralysis of one or more limbs)
myasthenia gravis (MG) = weakness of muscles under voluntary control
facial nerve paralysis in up to 70% of dogs with hypothyroidism [4]
vestibular disease (head tilt, difficulty walking, circling, involuntary movement of eyes)
dysphagia (difficulty swallowing)
laryngeal paralysis (noisy breathing, coughing after exertion, change in bark)
megaesophagus (loss of tone in esophagus causing dilatation, regurgitation of food)
myxedema in severe cases can cause coma and death

Cardiovascular changes

elevated cholesterol (adequate thyroid hormone levels keep lipids under control)
atherosclerosis
anemia (low production of erythropoietin in kidneys that produces red blood cells)
bradycardia (slow heart rate)
elevated blood pressure, retention of fluid (edema)
heart can be weakened to the point of heart failure causing death in severe cases

Gastrointestinal issues

Reduced appetite
weight gain
vomiting/diarrhea, possible inflammatory bowel disease symptoms
constipation
liver or pancreas disease

Kidney damage that may lead to renal failure

Reproductive

Dogs: poor semen quality, reduction in sperm count, infertility
testicular atrophy (shriveled)
reduction of libido (sex drive)
erectile dysfunction
delayed ejaculation
low testosterone hormone level
asymptomatic prostate inflammation

Bitches: irregular or lack of heat cycles that decreases fertility
small litter sizes
lower birth weights
higher mortality rates just prior to or shortly after birth
more stillborn puppies
duration of uterine contractions longer and weaker
higher incidence of uterine inertia (contractions stop part way through whelping)
higher incidence of dystocia (inability to complete vaginal delivery without help)
issues with carrying pregnancy to term (abortion, fetal resorption)
reduction of libido (sex drive)
gynecomastia (enlarged breasts)

DIAGNOSIS AND THYROID FUNCTION BLOOD TESTS

Autoimmune thyroiditis is difficult to diagnose due to a wide range of clinical signs that can mimic those caused by other diseases, while interpretation of thyroid function blood tests can be precarious. Clinical signs may be absent in early stages of AITH when blood tests may be abnormal, reinforcing the need for early thyroid function testing. Confirmation of AITH requires a high index of suspicion, accurate testing of thyroid hormone levels, and presence of thyroid autoantibodies in the blood (TgAA) -- proteins that attack one's own thyroid gland. However, sometimes the test results can be discordant with the clinical signs, making interpretation complicated.

Interpretation of thyroid function test results is further complicated by these caveats:

1. Autoimmune diseases such as Addison's disease can cause depressed thyroid function in early stages of treatment until Addison's disease is stabilized (2).
2. False positive TgAA (autoantibody) can result if your bitch is in heat or your dog had a vaccination within 30-45 days [1].
3. Some drugs can affect T4 levels.
4. Breed-specific reference ranges for T4 and FT4D are being researched.

Available thyroid function tests for a complete baseline and definitive diagnosis:

total T3 and total T4 (TT3 and TT4) = a portion may be attached to proteins in blood
free T3 and free T4 (FT3 and FT4) = floating in blood, not attached to proteins in blood
free T4 by dialysis method (FT4D) = most accurate test for levels of free T4
TSH (thyroid stimulating hormone) = regulates production of T3 and T4
TgAA = thyroglobulin (synthesizes T3 and T4) autoantibody
T3AA and T4AA (AA = autoantibody) = subsets of TgAA

Two forms of T4 exist in the body: (1) Total T4 -- the quantity attached to protein in the blood plus the quantity floating in the blood and (2) Free T4 -- the quantity floating in the blood that is utilized by tissues. All T4 in the blood is produced by the thyroid gland, but more than 90% is bound to proteins in the blood that prevent T4 from passing into tissues that need it [40]. When free T4 can reach the body tissues and organs it can exert its effects. The most reliable test for free T4 is accomplished via the direct **equilibrium dialysis** (ED) method where the blood is dialyzed (purified) against a buffer for 16-18 hours. ED then separates the free T4 molecules from the protein bound T4 molecules. This test is abbreviated as FT4D. T3 develops when T4 is converted to T3 by an enzyme. This clarifies why T4 changes develop prior to T3 changes, making T4 more important to measure [40].

Screening is crucial for obtaining the clinical diagnosis. Some veterinarians only include in their wellness panels a test for T4, not a free T4 which is more diagnostic, and rarely a TSH level. Abnormal TSH and T4 tests that indicate **late** thyroiditis stages will raise suspicion of the disease, encouraging further testing, but alone are not diagnostic of autoimmune thyroiditis. Many factors and drugs (phenobarbital for seizures, rimadyl for pain, and steroids) not related to thyroid can cause artificial reductions in T4, so additional thyroid tests are needed to confirm the diagnosis.

Research has proven that the presence of **thyroid autoantibodies** (T3AA, T4AA, TgAA), **markers** for autoimmune damage to the thyroid gland, **confirms the diagnosis** [6]. According to OFA "the

majority of affected dogs will have autoantibodies by 4 years of age", explaining their rationale of recommending yearly testing until 4 years of age [5]. OFA also states that thyroid autoantibodies "usually occur prior to occurrence of clinical signs". Low levels of T3 and T4 along with the presence of autoantibodies help confirm the diagnosis.
Of the 3 types of autoantibodies, TgAA is the most diagnostic. T3AA and T4AA are subsets of TgAA and will interfere with the measurement of both T3 and T4, but not FT4D. Most dogs with thyroiditis have TgAA present despite an 8% chance of false negatives [38]. Thus, the rationale for testing T3AA and T4AA along with TgAA to help clinch the diagnosis. The Orthopedic Foundation for Animals (OFA) offers autoimmune thyroiditis certification beyond the age of 1 year. They recommend retesting at ages 2, 3, 4, 6, and 8 years. **Consider that most affected dogs develop thyroid autoantibodies (TgAA) by the age of 3-4 years.** Tests included in the OFA certification panel that provide a complete thyroid screen are FT4D, TT4, TT3, TSH, TgAA, T4AA, and T3AA. Obviously, these 7 thyroid function tests required for OFA certification will accurately diagnose or rule out autoimmune thyroiditis.

Four stages of autoimmune thyroiditis are based on the degree of damage to the thyroid gland, with corresponding changes in thyroid panel blood tests. Focal (early) stage indicates the damage is limited to a small area and is evidenced by only one test, a positive TgAA. The diffuse stage means the damage is spread out [11] causing abnormalities in TgAA, TSH and FT4D. This chart exemplifies why testing only T4, even FT4D, is inadequate to diagnose thyroiditis since FT4D is normal until the late diffuse stage. **Testing TgAA will pick up thyroiditis in the earlier stages, often preceding symptoms.**

Thyroiditis	***TGAA***	***TSH***	***FT4D***
None	Neg	Norm	Norm
Focal	Pos	Norm	Norm
Multifocal	Pos	High	Norm
Diffuse	Pos	High	Low
End stage	Neg	High	Low

Chart courtesy of Ray Nachreiner DVM, Tufts University, from 2009 presentation [11]

Definitive diagnosis can be established by the presence of symptoms combined with abnormal results of thyroid function tests, or just the presence of thyroid autoantibodies TgAA. If you decide against OFA thyroid certification, advocate for your dog by encouraging your veterinarian to complete at least **the three most diagnostic tests prior to breeding -- TgAA, TSH, and FT4D**.

TREATMENT

Per Dr. Jerold Bell "Canine hypothyroidism is frequently misunderstood, misdiagnosed, and mistreated." [6] Once the diagnosis is confirmed, canine AITH is a permanent state of health that

requires lifelong treatment. No cure exists unless AITH is in very early stages and/or the causes of secondary hypothyroidism can be reversed. The goals of treatment are to reverse symptoms and to restore the thyroid hormones (primarily TSH and T4) to normal levels with synthetic thyroxine (T4). The standard replacement therapy drug is Levothyroxine, the FDA-approved drug for dogs and humans. Treatment is simple via a daily pill if AITH is discovered early, prior to damage to other organs, but frequent thyroid function tests are required to assure the medication is properly dosed. Supplementation causes the thyroid gland to stop making natural hormones leading to atrophy and destruction, so never give the hormone without an accurate diagnosis.

Recommendations for optimal absorption of Levothyroxine:

1. Administer the medication at the same time each day. Half-life is about 16 hours.
2. Optimally give it on an empty stomach, such as 30-60 minutes prior to a morning meal, or at bedtime which is often a few hours after a meal.
3. If not able to give it on an empty stomach, give it the same time each day to keep the T4 and TSH levels steady. Food in the stomach can alter the absorption of Thyroxine [42].
4. To ensure absorption, do not give it with other medications or foods containing soy or calcium.

About 6 weeks after thyroid supplementation is initiated, a repeat serum free T4 and TSH measurement is required to assess the effectiveness of the hormone doses. If doses are adjusted another measurement is needed to assure free T4 and TSH are in the reference ranges. Once the optimal dose is established, measurement of free T4 and TSH yearly or every 6 months will determine if the drug remains therapeutic.

THYROID HEALTH: BREEDER STRATEGIES

Goal: Directing our objectives toward selective breeding to reduce disease incidence

The mode of inheritance for autoimmune thyroiditis is likely polygenic, with the possibility of autosomal recessive. Breeding advice for dogs affected with **any type of autoimmune disease** is to avoid breeding the affected dog due to the risk for genetic susceptibility to the disease. If more than 2 puppies are produced in a litter that later develop autoimmune disease, do not repeat that breeding. Also, you may want to avoid breeding two dogs who have close relatives with autoimmune disease.

More specifically regarding strategies to minimize the risk of producing dogs with **autoimmune thyroiditis**, Dr. Jerold Bell recommends the standard goal for dealing with polygenic diseases such as hip dysplasia. He states, "Breed normal-testing dogs that come from litters which have mostly tested normal." Per Dr. Nachreiner of Michigan State University "I would recommend testing all breeding stock to reduce the amount of thyroid disease." [11]

If autoimmune thyroiditis proves to be autosomal recessive as a pattern of inheritance, which is highly unlikely, the strategy is similar – avoid breeding affected dogs or dogs that are identified as being carriers to other dogs that are carriers, which will decrease the load of carriers in the bloodline. Either strategy is a challenge for breeds with scant testing for the disease [6,11]. The more dogs tested in a pedigree can enhance the breeder's ability to identify carriers (11).

Dr. George Padgett DVM, expert on canine genetic diseases, recommends specific breeding strategies for dogs with autoimmune disorders that are recessive or polygenic when an effective DNA test has not been established. In either case, both parents contribute defective genes to offspring. Dr. Padgett recommends developing a risk assessment by objectively analyzing your pedigrees, gathering data on dogs in your pedigrees, and sharing that data with owners of potential breeding partners to avoid breeding carriers to carriers [25].

Until we have a gene-based test, defective genes can eventually be eliminated from the breed via:

1. thyroid screening of all breeding dogs, even prior to manifestation of symptoms.
2. attempting to find carriers in pedigrees.
3. selecting those dogs with lower genetic disease susceptibility.
4. avoiding mating of dogs that are affected or carriers.

SUMMARY

Ethical breeders place emphasis on screening for hereditary diseases to promote a long healthy life for their dogs, in addition to assuring the dogs are healthy enough to work, compete, and withstand breeding and pregnancy. Autoimmune thyroiditis (AITH) is proven genetic and familial, causing almost all cases of canine hypothyroidism that can manifest in significant breeding and health issues. Analyzing pedigrees allows breeders to pinpoint carriers, knowing the mode of inheritance is likely polygenic.

Affected dogs can accurately be detected via the blood tests TgAA, TSH, and FT4D. A positive TgAA (thyroid autoantibodies) is the most diagnostic test for AITH, the earliest change in thyroid blood panels, prior to symptoms. The variable age of onset of AITH symptoms and presence of autoantibodies in the blood is at 2 to 5 years. The cost of OFA thyroid certification in Minnesota is approximately $200 that includes veterinary costs of the blood draw (venipuncture), shipment of the blood to Michigan (MSU), evaluation of sample, and posting the results of a thyroid panel of seven on the OFA website. Costs may be less if blood is sent to a local lab, or Hemopet https://hemopet.org, or testing only a thyroid panel of three (TgAA, TSH, FT4D). List of approved OFA thyroid labs: https://www.ofa.org/diseases/other-diseases/hypothyroidism/thyroid-labs

Early screening of thyroid hormone levels and TgAA is of paramount importance for diagnosing AITH in the early stages before other organs are damaged, prior to onset of their breeding or performance competition debuts, to prevent permanent thyroid damage, and to reduce the incidence of AITH in the breed. Autoimmune thyroiditis is unlikely to be life threatening if identified early, easily diagnosed with pertinent blood tests, and uncomplicated to treat with daily thyroid supplement and intermittent blood tests to assure adequate T4 levels.

Vickie Halstead RN *has been actively involved in breeding and showing Bichons Frises since 1990 and Portuguese Water Dogs since 2017, producing 36 litters and >50 AKC champions, one of which achieved Best in Show at the 2005 Bichon national specialty. She has been a member of the Bichon Frise Club of America (BFCA) since 1997, a member of BFCA's Health Committee since 2001 & chairwoman 2003-2010, plus was a member of the Board of Directors of BFCA 2005-2007. She published articles in the Bichon Frise Reporter, dog magazines, newspapers, and on*

www.bichonhealth.org. She is a member of the Portuguese Water Dog Club of America (PWDCA) and its health committee since 2018. Vickie has been practicing as a Registered Nurse since 1973, retired in 2017 after previous experience in ICU (adult & pediatric), flight nursing (helicopters & fixed-wing aircraft), ER for 25 years, legal nurse consultantant, speaker for nursing classes/seminars, and lastly as a Coumadin (blood thinner) Nurse at a clinic. Currently she operates a healing service primarily for dogs, but also humans, that offers energy healing (reiki, healing touch) and aromatherapy https://www.victoireshealingandbreeding.com.

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