WESTIE FOUNDATION OF AMERICA, INC
Leading the Way to Westie Health

Westie WELLNESS

Fall / Winter 2016

Questions? Comments? Suggestions?
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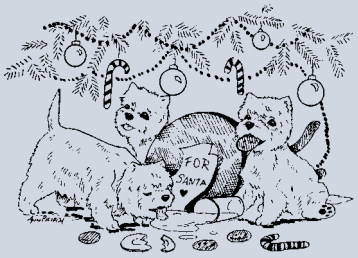

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PRESIDENT'S MESSAGE

Bebe Pinter

Westie owners are amazing! They exchange with each other the many stories about their Westies and chuckle as everyone agrees what a wonderful breed this is. Without our common love of Westies, many of us would never have met.

Such is the case with the Westie Foundation of America's board of directors which is a grouping of people who love the Westie breed and are working together to improve the health and well-being of all Westies.

On October 5, 2016, the board met for its annual board of directors meeting. Directors celebrated the dedication of Kenneth Fodill and Ann Marie Hollowathy as they will retire to the Advisory Council effective January 1, 2017. We are delighted that both of them will continue to work as members of WFA committees. Additionally, Renee Glover, MD and Randy Cantrell were elected as new members to the Advisory Council. Renee possesses medical knowledge especially in the area of dermatology and Randy brings strong executive coaching and technical skills to support our board and mission.

This Fall/Winter issue of *Westie Wellness* focuses on several areas. Tops in her field of dermatology, Valerie Fadok, DVM, PhD, Diplomate, ACVD wrote an article especially for you—"*What role does food allergy play in canine Atopic Dermatitis*?" She speaks to when we should consider food allergy, diagnosis, myths, and treatment.

Please read Robert McCaskill's update on the WFA's E-book project "*A History of the Westie E-Book*". Dr. McCaskill expressed the WFA's gratitude when he said, "We are indeed grateful to Virginia-Maryland Regional Veterinary College and the University of Georgia College of Veterinary Medicine for assisting the WFA in our efforts to provide us with a unique source of health information for the West Highland White Terrier."

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(President's Message continued from page 1)

"Endocrine System Addison's Disease (Adrenal Gland Insufficiency" a WFA Westie Health E-books article that has been updated on the disease. We hope you find it informative. Discussion topics include Anatomy and Physiology of the Adrenal Glands, How does this disease develop?, Which Clinical signs occur in dogs with Addison's disease?, How is Addison's disease diagnosed?, and Current Research About Addison's Disease.

Also of great interest is an article on a possible inheritable condition affecting eyes—"*Persistent Pupillary Membranes (PPM)"* courtesy of Dog Health by LowchensAustralia.com.

To keep abreast of how the WFA is leading the way in novel approaches to disease that affect both our Westies and humans, read Vice President Communications Teresa Barnes' article "*WFA to Convene Pivotal Workshop in Pulmonary Fibrosis and Cancer*". The WFA is honored to continue our work to drive research efforts in diseases that profoundly affect our beloved Westies.

Finally, discover "Why Do Dogs Eat Poop?" Staff writers for the American Veterinary Medical Association explain facts, why, and how to stop this habit.

Don't forget that if you wish to submit a Westie Remembrance or Tribute for that special Westie who has crossed to The Rainbow Bridge, please visit our website at http://www.westiefoundation.org/remembrances.html for the form. The form provides text and photo requirements as well as payment information. Allison Platt, WFA Webmaster, can assist you. Remembrances and Tributes remain on the WFA website indefinitely.

Thank you for your continued involvement and support of the WFA but most of all, your love of Westies!

Bebe Pinter

The Westie Foundation of America, Inc is a nonprofit corporation, recognized by the IRS as a 501 (C) (3) organization. The mission of the Foundation is to advance and support medical research to benefit the health and quality of life of West Highland White Terriers: and to further develop and communicate information regarding the health, care, breeding and quality of life of Westies to Westie owners, Westie breeders and veterinarians.

WFA
WESTIE FOUNDATION OF AMERICA, INC
Leading the Way to Westie Health

Request for Samples

RESEARCH PROJECT	SAMPLES NEEDED	CONTACT INFORMATION
Genetic marker for **Atopic Dermatitis**	Saliva swabs or blood samples from dogs with skin disease or from normal dogs 5 years of age or older from family lines free of allergies	Kim Williams North Carolina State University 919-513-7235 kdwilli4@ncsu.edu
Genetic susceptibility of **Transitional Cell Carcinoma (TCC) (Bladder Cancer)**	Blood samples from dogs diagnosed with TCC and dogs over the age of nine who have no known cancers	Gretchen Carpintero Ostrander Lab National Human Genome Research Institute 301-451-9390 Dog_genome@mail.nih.gov
Genetic marker for **Addison's Disease**	DNA from cheek cells and/or blood from affected dogs and unaffected dogs over the age of 7	Dr. A.M. Oberbauer UC Veterinary School (Davis) 530-752-4997 http://cgap.ucdavis.edu/
Clinical Features and Genetic Basis of **Idiopatic Pulmonary Fibrosis (IPF)**	Blood samples from dogs diagnosed with PF and healthy dogs over age 8 without lung disease	Drs. Ned Patterson and Peter Bitterman Katie Minor (contact) University of Minnesota 612-624-5322 minork@umn.edu
Idiopatic Pulmonary Fibrosis (IPF)	Cheek and/or blood samples from dogs diagnosed with pulmonary fibrosis	Dr. Victor J. Thannickal University of Alabama Sample collection coordinated by Dr. Pamela Whiting, DVM pgwhitingdvm@aol.com 707-529-9222 (cell/text) 707-837-8101 (clinic)
Dry Eye Syndrome (keratoconjunctivitis sicca)	Dogs diagnosed with dry eye and dogs over 7 years old with no ocular abnormalities *participants must be available for appointments at UC Davis Veterinary Center (CA)	Dr. Sara Thomasy UC Veterinary School (Davis) 530-752-1770 smthomasy@ucdavis.edu

For more information about any of the above projects visit
www.westiefoundation.org

On The Healthfront

By Kay McGuire, DVM, MS

One of the Westie Foundation's (WFA) goals for the upcoming years is the development of a tissue and DNA repository. The WFA is contributing $5000 towards a meeting to convene many of the top geneticists at Texas A&M University in late March, 2017. This working meeting is to establish ultimate guidelines for procuring and successfully storing tissue and DNA samples. We anticipate that these guidelines will insure that all samples stored for future use will be safe and useful.

The Canine Health Foundation 2016 Grant Cycle included our support on Atopy, lymphoma, and inflammatory bowel disease. With Atopic Dermatitis being the number one Westie health issue, money is spent not only looking as to how to modify allergies at the cellular level, but also on further treatment options.

There is a new study at the University of Alabama with Dr. Victor Thannickle comparing results of the amino acid thyrosine uptake in humans having Pulmonary Fibrosis (PF) with the rate in Westies also having the same disease.

The West Highland Club of America again hosted and subsidized the cost of a Westie eye clinic in conjunction with our National Sweepstakes competition at Kimberton, October 5, 2017. Cataracts and dry eye, KCS, continue to be the most common eye defects.

Our biggest news is the revamping of the existing 'E-BOOK' chapters for our website. The University of Georgia contracted with the WFA to update the chapters on the conditions that most commonly affect our Westies. We are currently launching this new information.

What Role Does Food Allergy Play In Canine Atopic Dermatitis?

By Valerie A. Fadok, DVM, PhD, Diplomate, ACVD

Traditionally we have thought of food allergy in dogs as an entity separate from canine atopic dermatitis. We have learned, though, that pure food allergy, where all the signs are controlled by diet alone, is uncommon, probably occurring in less than 5% of allergic dogs. More commonly, we find food allergies as part of atopic dermatitis. It has been estimated that anywhere from 5-20% of atopic dogs could have some food triggers, but we honestly don't know the exact percentages.

Atopic dermatitis in dogs is very similar to that in people. This

knowledge can be helpful to us because we can apply some of what is known about the disease in people to the disease in dogs, with the caveat that we verify some of the key points. Atopic dermatitis is an inherited predisposition to develop hypersensitivity reactions to environmental triggers. These include pollens, molds, dusts, danders, and mites. A subset of children with severe atopic dermatitis is known to have concurrent food hypersensitivities. Likewise, a subset of atopic dogs will also have food hypersensitivities. Sorting out these possibilities takes a thorough investigation and patience.

Atopic dermatitis is characterized by an altered immune system as well as skin barrier defects. Given the location of the lesions on dogs, we have come to believe that dogs absorb allergens through their skin because of the defective barrier. This hypothesis fits environmental allergens, but it is harder to understand how food allergy contributes to skin disease. Research in human infants though suggests that in addition to introduction through the gut, food allergens can actually penetrate the skin!

When should we consider food allergy in our itchy allergic dogs? Dogs with food allergies tend to have a nonseasonal itch and inflammation. If the skin disease is seasonal, then food allergy is highly unlikely. Other clues to suggest a food allergy are gastrointestinal signs, which may be mild or intermittent. We can see intermittent vomiting, slightly loose stools, more than 4 bowel movements a day, excessive flatulence, or burping with food allergic dogs.

Diagnosis of food allergic requires an elimination diet. We do not have a diagnostic test for food allergy. Serum testing and skin testing are not accurate because they test only for the allergic antibody IgE; there are other mechanisms possible for food allergy that would not be detected by the serum allergy tests. In order to pick the appropriate diet for a dog, we need to know what he or she has been eating prior for meals as well as for treats. The ideal diet would consist of a novel protein and a novel carbohydrate source. Either a home cooked diet or a veterinary prescription diet is required, because the over-the-counter diets are not prepared to the needed level of purity. Many over-the-counter diets now contain the novel proteins we have used in our veterinary prescription diets, including rabbit and kangaroo. For that reason, many veterinarians recommend the use of prescription hydrolyzed diets including Hill's ZD® (hydrolyzed chicken), Royal Canin's Ultamino® (hydrolyzed poultry byproduct (chicken feathers), and Purina's HA® (hydrolyzed soy). We often recommend Royal Canin's vegetarian diet as well, since most food allergic dogs are reactive to the animal proteins. During the hydrolysis process, the protein is chopped up into very small pieces, which are less likely to be recognized by the immune system. Any of the prescription diets are complete and balanced so they could be continued indefinitely, but it is not required as we can find out what foods trigger reactions and then simply avoid them. Many dogs can

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(Canine Atopic Dermatitis continued from page 4)

go back to an over-the-counter diet once we learn what triggers their reaction. The alternative to a veterinary prescription diet is a home-cooked diet. These diets should be formulated by a veterinary nutritionist, based on the diet history.

We now have some good evidence in the veterinary literature that over 96% of dogs with food allergy can be diagnosed with an 8 week diet trial. This is great information because some veterinary dermatologists have suggested that 12, 16, or 20 week diets were required. The longer we have to feed a restricted diet, the harder it is! But most pet owners can feed a restricted diet for 8 weeks. When the prescription diet is fed, dogs should eat nothing else during the 8 weeks they are on the diet. Treats, table scraps, rawhides, and even some flavored medications need to be stopped during this period. If in 8 weeks there is no improvement, then food allergy is highly unlikely and we move on to managing environmental allergies. If there is improvement, then we can do diet challenges to determine what foods cause an outbreak and therefore what to avoid in future. Many dogs can then go back to an over-the-counter diet that does not contain the offending ingredients.

The first challenge is to mix a small amount of the original diet to the prescription diet (usually ¼ old diet to ¾ new diet) and feed that over a week. If there is no increase in itch, then food allergy is ruled out. But if there is an increase in itch, then single food challenges are done until we can develop a list of foods to avoid in future. Usually we do this by adding 1-2 tbsp. of the food to the prescription diet for one week. The single foods that we use are chicken, beef, lamb, milk products, soy, wheat, corn. Any treats desired can be used as challenges, too, as long as they are given one at a time over a period of a week. As an example, a dog might start to itch any time within a few hrs. to a few days after eating chicken, if they are allergic to it. If chicken is the only food that induces itch, then that dog could do very well on a single ingredient OTC diet containing lamb or beef, but no chicken or chicken byproducts. These restrictions apply to treats as well.

There are some myths associated with food allergy in pets that we need to dispel. First, most dogs having an allergy to a food have been eating that diet for some time. A change in diet is not what precipitates a food allergy. Second, most dogs are not allergic to grains. The three most common food allergens reported in dogs are chicken, beef, and milk products. Feeding a grain-free diet rarely rules out food allergy in dogs. While a grain allergy is possible, it is not common. Third, simply changing brands of dog foods also is rarely effective, because many canine diets contain common ingredients. Even the label can be misleading. Some diets called lamb and rice actually contain chicken or fish or beef. It is the small print in the list of ingredients we need to check when we are looking at diets for dogs. Fourth, when the elimination diet is fed, it is very important to avoid treats as well. The elimination diet does not negate or overcome the effects of treats. Dogs on diet trials may be allowed to have some treats, including bananas or apples, but nothing should be fed without consulting with your veterinarian. Again, this is only for 8 weeks, until it can be determined whether the diet trial reduces itch. Once the diet trial is over then treats can be reintroduced again. Fifth, there is no naturally hypoallergenic protein for dogs. A diet is called hypoallergenic only because it hasn't been fed to the individual dog before. Fish or venison or rabbit or kangaroo can cause allergies in predisposed pets if these ingredients are part of their regular diet.

To Summarize, What Can We Say About Food Allergy In Dogs?

1. It rarely occurs by itself; it is more often associated with atopic dermatitis. Both the food and the environmental allergens need to be managed for best control of the disease.
2. Food allergy is associated with nonseasonal itch and inflammation. The value of eliminating food triggers is that you may be able to take a dog that is itchy year round and reduce that itch to only a few months out of the year. Less medication will be needed in the long run.
3. The only proven way to diagnose food allergy is with a diet trial and challenges.
4. The most common food allergens are animal proteins, not grains.
5. The best treatment for food allergy is avoidance of the offending foods.

The History of the Westie E-book

By Robert McCaskill, DVM, MPH, DACVPM

Past president of the Westie Foundation of America, Inc. (WFA) Wayne Kompare originated the concept of the Westie E-book. At one of the annual board meetings of the WFA back in the early 2000's, Wayne tasked the Health Committee to develop a health book that would discuss the common health issues of the West Highland White Terrier. His idea was that the book would be in an electronic form. This would allow Westie owners and veterinarians to quickly obtain this information. The Health Committee embraced this concept and began to consider options.

Daphne Gentry, while on a cruise one summer, met a veterinarian who was on the faculty of the Virginia-Maryland Regional College of Veterinary Medicine (VMR). We contacted him, and he connected us with the dean of the research office at the school. A grant proposal was discussed to have VMR develop an electronic health book that would consist of chapters discussing the most common diseases found in the West Highland White Terrier.

John Robinson, DVM, PhD, the Director of the Center of Comparative Center for Oncology at VMR, took on the grant. He collaborated with Elizabeth McStay, BSVetMed, MS, a clinical resident, to oversee the project. Each summer for the next three years, a team of rising third-year veterinary students who needed a work study project were assigned clinical areas to research. Dr. McStay edited their work with Dr. Robinson providing the final review and edits. At the end of three years, the WFA was presented a CD with the work of their grant. It was an immediate success!

We heard many accolades from Westie owners who found the information extremely helpful. We also heard from numerous veterinarians who also found the information beneficial in their practices.

By 2015, some of the information in our E-book was becoming outdated. We again approached the Virginia-Maryland College of Veterinary Medicine for an update. We learned that Dr. Robinson had transferred to another college in the Virginia Tech University and was not available to assist us. We then reached out the University of Georgia, College of Veterinary Medicine. They agreed to a WFA grant to review our E-book and to include

two additional chapters on Cushing's Disease and Cancer. The University of Georgia used the same process as VMR, hiring third-year veterinary students in a summer work study program to complete assigned chapters. The college also used two medical illustrators which added a degree of professionalism to this work. Review and editing of the completed work was done by Drs. Jim Moore and Robert Scott.

We are indeed grateful to Virginia-Maryland Regional Veterinary College and the University of Georgia College of Veterinary Medicine for assisting the WFA in our efforts to provide us with a unique source of health information for the West Highland White Terrier.

The current topics of our Health E-books include: Addison's Disease, Aggression, Cushing's Disease, Craniomandibular Osteopathy (CMO), Atopic Dermatitis, Idiopathic Pulmonary Fibrosis (IPF), Legg-Calve Perthes Disease, Patella Luxation, White Dog Shaker Disease, Diabetes Mellitus, Inflammatory Bowel Disease, Copper Toxicosis, Juvenile Cataracts, Keratoconjunctivitis Sicca, and Cancer such as Transitional Cell Carcinoma and Lymphosarcoma.

Endocrine System
Addison's Disease (Adrenal Gland Insufficiency)

Westie Heath E-Book

Addison's disease, also known as adrenal gland insufficiency or hypoadrenocorticism, is an uncommon condition in which the patient's adrenal glands no longer supply the body with two classes of hormones, called glucocorticoids and mineralocorticoids. These hormones help regulate cellular metabolism and electrolyte balance in the body. According to the most recent edition of the Merck Veterinary Manual (Merck, 2015), this disease is characterized by gastroenteritis (vomiting and diarrhea), loss of body condition, lethargy and weakness, and inability to respond to stress. Although this condition has been recognized in dogs for more than 60 years, it remains difficult to diagnose, primarily because the animal's symptoms mimic those associated with several other diseases. However, when the disease is identified, treatment is very effective, allowing affected dogs to lead normal healthy lives.

Anatomy and Physiology of the Adrenal Glands

The adrenal glands, which exist as a pair, are complex, multifunctional organs. The adrenal glands are located on top of the kidneys ('ad renal' – near the kidney). The outer layer of the gland (the cortex) produces three types of hormones: glucocorticoids, mineralocorticoids and small amounts of sex hormones.

Common Clinical Findings

- Lethargic and Listless
- Vomiting or Regurgitation
- Weight Loss or Weakness
- Abnormal Cortisol Response to ACTH Stimulaton

In healthy animals, production of glucocorticoids is regulated by signals received from the brain. The hypothalamus is the region in the brain that produces a hormone called corticotrophin-releasing hormone (CRH), which stimulates another part of the brain, the pituitary gland, to release a hormone called adrenocorticotrophic hormone (ACTH). ACTH is released into the bloodstream and travels to the adrenal glands where it causes them to release glucocorticoids in the form of cortisol. When there is a healthy amount of cortisol circulating in the blood, this is sensed by the hypothalamus, which then reduces its production of CRH, and this causes the pituitary gland to stop releasing ACTH. The end result is a reduction in the production of cortisol by the adrenal glands. Because the healthy level of cortisol in the blood is exerting a negative influence on the production of CRH and ACTH by the brain, this is known as negative feedback. When the concentration of cortisol in the blood decreases, the hypothalamus and pituitary gland respond by releasing more CRH and ACTH, respectively, which stimulates the adrenal glands to produce more cortisol until circulating concentrations are restored.

Unlike the glucocorticoids, production of the mineralocorticoids is regulated by a system that starts with special cells in the kidneys, called the juxtaglomerular cells. These cells, which are located near the functional unit of the kidney called the glomerulus, sense the concentration of sodium in the blood, which is very important in the regulation of blood pressure. When the sodium concentration in the blood is low, the juxtaglomerular cells produce a chemical called renin, an enzyme that converts a substance in the blood called angiotensinogen to angiotensin I. Angiotensin I is then converted by another enzyme, which is located primarily in the blood vessels in the lungs, to angiotensin II. Angiotensin II has two effects: 1) stimulating the adrenal glands to produce aldosterone, the main mineralocorticoid, and 2) constricting small blood vessels to increase blood pressure. Aldosterone then causes the kidneys to absorb additional sodium and water from the fluid that it has filtered, which helps return blood sodium concentrations towards normal and increase blood pressure. At the same time, aldosterone causes the kidney to excrete potassium into the urine, which helps balance the electrolytes in the body.

How does this disease develop?

Addison's disease is characterized by the lack of production of glucocorticoids and mineralocorticoids. The disease can occur either as a result of an abnormality in the brain that then fails to stimulate the adrenal glands to perform their functions or in the adrenal glands themselves. When the problem originates in the brain, there is insufficient production of either CRH by the hypothalamus or ACTH by the pituitary gland. Lacking sufficient production of CRH or ACTH, the adrenal glands fail to function normally, production of cortisol and aldosterone is reduced, and the glands shrink in size (atrophy). This form of Addison's disease occurs infrequently.

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(Addison's Disease continued from page 7)

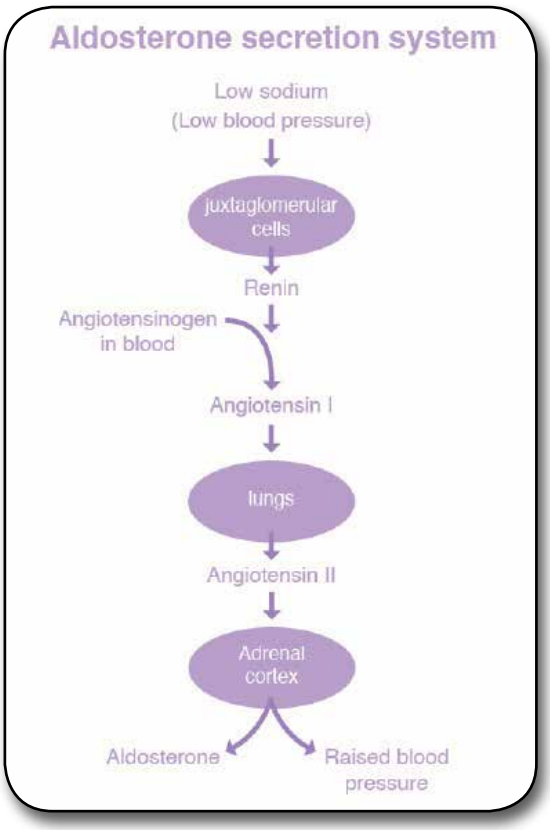

Figure 1 - A graphic representation of the production of aldosterone by the adrenal cortex. In response to low blood sodium concentration, the juxtaglomerular cells in the kidney release renin, which converts angiotensinogen to angiotensin I, which then is converted to angiotensin II. This latter compound stimulates the adrenal glands to secrete aldosterone, which returns blood sodium concentration to normal and increases blood pressure.

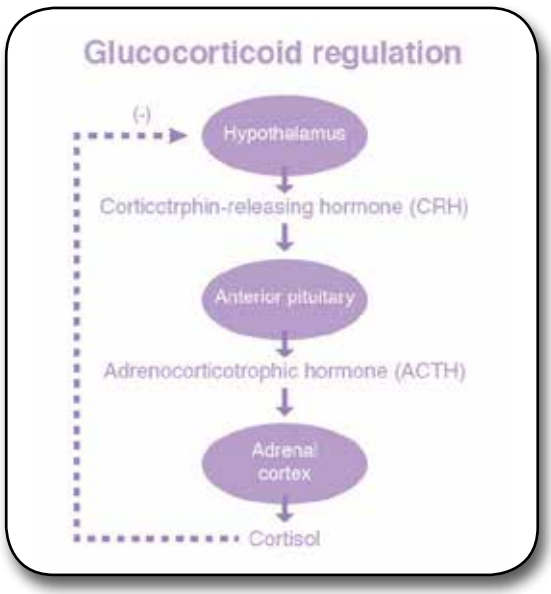

Figure 2 - A graphic representation of the negative feedback regulation of cortisol production by the adrenal cortex.

Most cases of Addison's disease occur because the adrenal glands have been damaged and are no longer able to make cortisol and aldosterone, even when stimulated by ACTH and angiotensin II, respectively. In rare instances, special chronic inflammatory diseases (i.e., granulomatous diseases), hemorrhagic infarctions (blood clots forming and lodging in the adrenals and other tissues), cancer of the adrenals, and trauma can induce enough damage to the adrenal glands to cause Addison's disease. In the majority of cases of Addison's disease in dogs and people, an autoimmune process is responsible for destroying the adrenal glands. That means that the patient's own antibodies have destroyed the cells in the adrenal glands, much like other antibodies destroy foreign invaders like bacteria or viruses. The underlying processes that stimulate this autoimmune attack on the adrenals are not known, but are the subject of active research. For some reason, females are twice as likely to develop Addison's disease as males.

Which clinical signs occur in dogs with Addison's disease?

Clinical signs of Addison's disease often are vague and nonspecific, with many affected dogs being lethargic, listless, anorexic, and reluctant to exercise or even do normal activities. Very often, these signs appear to wax and wane, making it even more difficult for owners to decide when to seek veterinary care. More than half of affected dogs have episodes of vomiting or regurgitation of food, weakness, and weight loss. Diarrhea occurs in approximately one-third of dogs with the disease. The severity of the clinical signs may progress rapidly in some dogs and very slowly in others. Acute exacerbation of the condition may occur when the dog's lifestyle is changed, for instance this may occur when the dog is moved, boarded or is examined by a veterinarian.

Although dogs with Addison's disease may vary in age, the typical dog is 4-5 years old and female. These characteristics should not be surprising as many immune-mediated diseases occur more commonly in females than males.

All clinical signs of Addison's disease are due to the deficiencies of glucocorticoids (cortisol) and mineralocorticoids (aldosterone). For example, cortisol deficiency affects the body's metabolism, which results in a loss of appetite, vomiting, abdominal pain, weight loss and lethargy. Because aldosterone is critical for balancing electrolytes (reabsorbing sodium and excreting potassium) and maintaining blood pressure, a deficiency in aldosterone reduces serum sodium concentration, and lowers blood pressure as the result of reduced circulating blood volume. Dogs with low blood sodium concentration may lose weight, feel weak, have small hearts and produce dilute looking urine even though they may be dehydrated. High blood potassium concentrations can cause life-threatening problems with heart rhythm (called 'arrhythmias'). In fact, some affected dogs may develop such high blood concentrations of potassium that severe alterations occur in heart function and blood pressure, resulting in the development of shock. This clinical scenario is often referred to as an "Addisonian crisis".

Unfortunately, Westies appear to be at a high risk for developing Addison's disease, as are Great Danes, Poodles, Portuguese Water Dogs, Soft-coated Wheaten Terriers, Nova Scotia Duck Tolling Retrievers and others. The results of recent studies suggest that there is a genetic predisposition for the disease in some breeds.

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(Addison's Disease continued from page 8)

How is Addison's disease diagnosed?

Due to the wide variety of clinical signs that can occur and the fact that many of these are nonspecific (i.e., can occur in dogs with other diseases), Addison's disease is difficult to diagnose. As a result, many more dogs are suspected of having Addison's disease than end up being diagnosed with the condition. In one report, 15% of dogs tested for Addison's disease ended up having it (Lennon et al, 2007).

A reliable screening test for Addison's disease involves the measurement of cortisol in the blood. Most dogs with the disease have low resting levels of cortisol, whereas dogs with a high resting cortisol level are extremely unlikely to have the disease. When a low resting cortisol concentration is measured, the follow-up approach is to determine whether or not the adrenal glands will respond when stimulated. This is achieved by administering ACTH and measuring changes in cortisol concentration an hour later. If the dog's adrenal glands are normal, they should respond to the ACTH by increasing their production of cortisol. As a result, the blood concentration of cortisol will be significantly increased when measured an hour later. In contrast, the adrenal glands of a dog with Addison's disease will not respond to the ACTH, and the blood cortisol value measured after ACTH administration will be unchanged. It is important to know that any corticosteroids being given as a treatment as a result of the animal's clinical signs will interfere with this diagnostic approach. Consequently, it is important for these treatments to be stopped at least 24 hours before an ACTH stimulation test is performed.

While it is common to measure cortisol concentrations before and after an ACTH stimulation test, much less is known about circulating concentrations of aldosterone in dogs with Addison's disease. In a recent study, however, aldosterone concentrations were measured in healthy dogs, in dogs with clinical signs similar to those associated with Addison's disease, and in dogs with the disease. Concentrations of aldosterone were significantly lower in dogs with confirmed Addison's disease when compared with dogs in the other two groups. Furthermore, aldosterone concentrations were not increased after administration of ACTH in the dogs with Addison's disease. These findings confirm that damage to the adrenal cortex affects production of both glucocorticoids and mineralocorticoids similarly.

The ACTH stimulation test does not distinguish between hypoadrenocorticism due to abnormalities of the adrenals and the pituitary gland. In order to make this distinction, blood concentrations of ACTH must be measured. When the abnormality primarily affects the adrenal glands, ACTH concentrations will be high as the lack of cortisol production will not provide the normal negative feedback effect on the pituitary gland. As a result, it will continue to produce ACTH.

In contrast, if the abnormality primarily affects the pituitary gland, blood concentrations of ACTH will be low, due to the fact that it is not being produced by the pituitary gland. Dogs with the pituitary gland abnormalities may eventually respond to enough ACTH given by the veterinarian, whereas those with abnormal adrenal glands will not (i.e., their adrenal glands will continue to fail to produce cortisol).

In addition to the aforementioned blood tests, veterinarians may also use radiography (x-rays), ultrasonography, and electrocardiography (ECG; measurements of the heart's electrical output) to help make a definitive diagnosis of Addison's disease. Radiographic findings detected in many dogs with Addison's disease include reduced size of the heart, liver or specific blood vessels in the lung or abdomen. Ultrasound findings in affected dogs often include adrenal glands that appear smaller than normal, although this is not a consistent finding. The most commonly identified ECG abnormalities include those associated with excessively high blood concentrations of potassium.

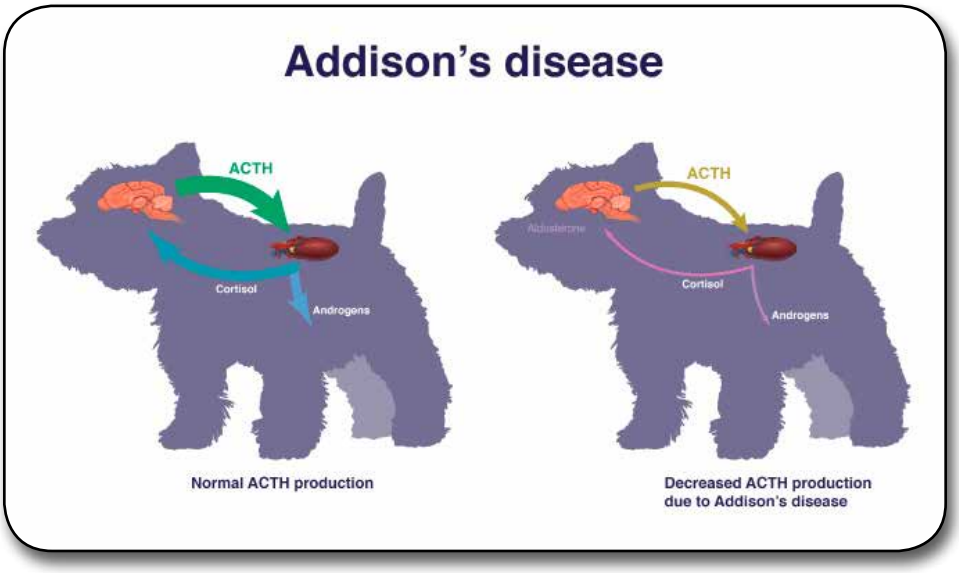

Figure 3 - This illustration depicts the normal interaction between the pituitary and adrenal glands. This results in stimulation of the adrenal glands by ACTH and production of cortisol and androgens, and the normal negative feedback effect of blood cortisol levels on the pituitary gland. In contrast, decreased production of ACTH in a dog with Addison's disease results in reduced synthesis of cortisol and androgens.

(Continued on page 10)

(Addison's Disease continued from page 9)

Treatment

The key to treating dogs with Addison's disease is to address immediate life-threatening aspects of the disease first and then to consider what needs to be done long-term. Clearly, dogs with poorly functioning adrenal glands will need to be treated for the rest of their lives; owners should be made aware of this immediately and be willing to accept the responsibilities associated with the need for life-long therapy. Fortunately, the prognosis for a healthy, happy life is extremely good.

For dogs in a hypoadrenocortical crisis, the veterinarian's initial focus is to restore blood volume with IV fluids, correct electrolyte abnormalities by slowly, but consistently increasing the sodium concentration in the blood with sodium-containing fluids IV, restoring blood glucose and glucocorticoid levels to normal. The dog's responses to these initial treatments are monitored closely to ensure that tissue perfusion and blood pressure increase appropriately. Fluid therapy also is important to rehydrate the animal, reestablish normal kidney function and correct all serious electrolyte imbalances (e.g., reduce high blood potassium concentrations) that could adversely affect metabolism and heart function. Blood glucose concentrations are restored by administering IV fluids containing dextrose and closely monitoring changes in blood glucose levels.

Finally, a fast acting glucocorticoid is given to replace the glucocorticoids not being produced by the animal's adrenal glands. Typically this is done with an injectable glucocorticoid, such as dexamethasone, until the dog has recovered sufficiently to be treated with oral glucocorticoids. During the acute crisis, treatment with a mineralocorticoid is not critical, and many veterinarians prefer to incorporate this as part of the long-term care plan.

Current Research About Addison's Disease

Because there is a relatively high incidence of Addison's disease in Westies, a genetic basis for the disease is strongly suspected. Consequently, there is a great deal of interest in determining whether or not this is true, and, if so, which genes might be associated with development of the disease. There also is convincing evidence that the disease may have an autoimmune component to its development. In this section, we summarize two recent studies about the genetic basis for the disease and one about the autoimmune nature of the condition.

Boag AM, Catchpole B. A review of the genetics of hypoadrenocorticism. Top Companion Anim Med. 2014 Dec;29(4):96-101.

There is good evidence that hypoadrenocorticism in people has an autoimmune component to its pathogenesis, as several immune response genes have been implicated in increasing the susceptibility of humans to development of Addison's disease. There also is good evidence that a similar situation exists with regard to this disease in dogs. For example, specific breeds of dogs are over-represented in epidemiologic studies of the disease, and some recent molecular genetic studies have determined that some of the same genes and cellular signaling pathways that are involved in Addison's disease in people are associated with increased susceptibility of dogs to the disease. Examples of these include genes associated with immune responses, such as the dog leukocyte antigen and cytotoxic T-lymphocyte–associated protein 4 (CTLA4) genes. The authors of this review paper suggested that this increased understanding of the molecular mechanisms involved in the progression of Addison's disease may make it possible to establish genetic tests to identify dogs at risk of developing the disease and for the development of new treatments.

Short AD, Catchpole B, Boag AM, Kennedy LJ, Massey J, Rothwell S, Henthorn PS, Littman MP, Husebye E, Ollier B. Putative candidate genes for canine hypoadrenocorticism (Addison's disease) in multiple dog breeds. Vet Rec. 2014 Nov 1;175(17):430.

In this study, the authors performed candidate gene analyses for canine hypoadrenocorticism in several breeds in the UK: bearded collie, border collie, German shepherd, standard poodle, Jack Russell terrier, West Highland White terrier and Soft-coated Wheaten terrier. They identified that some putative genetic loci for disease susceptibility form part of the T-cell receptor pathway, supporting the involvement of an autoimmune response. However, other genes that were identified are not involved in these responses, providing additional basis for the heterogeneity and complexity of the condition.

The authors cautioned that the animals involved were part of a laboratory-based collaboration, and thus may not be representative of all dogs in the UK. They also noted that a relatively small number of dogs were available for each breed, which meant that they may not have been able to detect small or moderate effects. Furthermore, the most aggressive forms of hypoadrenocorticism may be missing due to euthanasia or death before an accurate diagnosis was made. While the authors concluded that there is clinical heterogeneity between breeds, it is likely that the cause of hypoadrenocorticism within dogs of one breed is the same.

Boag AM, Christie MR, McLaughlin KA, Syme HM, Graham P, Catchpole B. Autoantibodies against cytochrome P450 side-chain cleavage

(Continued on page 11)

(Addison's Disease continued from page 10)

enzyme in dogs affected with hypoadrenocorticism (Addison's Disease). PLoS One 2015; 10(11): e0143458

There is ample evidence that hypoadrenocorticism in dogs occurs as a result of immune-mediated destruction of portions of the adrenal glands and leads to deficiencies in glucocorticoid and mineralocorticoid production. In people with Addison's disease, circulating autoantibodies directed against some of the enzymes responsible for the synthesis of adrenal gland hormones have been identified. This study was performed to determine whether or not similar autoantibodies against enzymes of the corticosteroid synthesis pathway are present in dogs with hypoadrenocorticism, and whether a relationship exists between autoantibody status and clinical features of the disease. The results of this study indicated that autoantibodies directed against a key enzyme in this pathway exist in a proportion of dogs affected with hypoadrenocorticism, are more prevalent in affected female dogs, and appear to be related to breed and DLA-type. Further work is required to determine whether the presence of these autoantibodies is associated with reproductive dysfunction in affected female dogs and whether measurement of these autoantibodies is of use as part of the diagnostic approach for canine hypoadrenocorticism.

Acknowledgements

Mr. Matthew Crotts, a medical illustrator in Educational Resources in the College of Veterinary Medicine at the University of Georgia, created the illustration used in this chapter.

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Short AD, Boag A, Catchpole B, Kennedy LJ, Massey J, Rothwell S, Husebye E, Ollier B. A candidate gene analysis of canine hypoadrenocorticism in 3 dog breeds. J Hered. 2013 Nov-Dec;104(6):807-20.

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Persistent Pupillary Membranes (Ppm)

Courtesy of Dog Health by LowchensAustralia.com

What Are Persistent Pupillary Membranes (Ppm)?

Persistent pupillary membranes are strands of tissue in the eye. They are remnants of blood vessels which supplied nutrients to the developing lens of the eye before birth. Normally these strands are gone by 4 or 5 weeks of age.

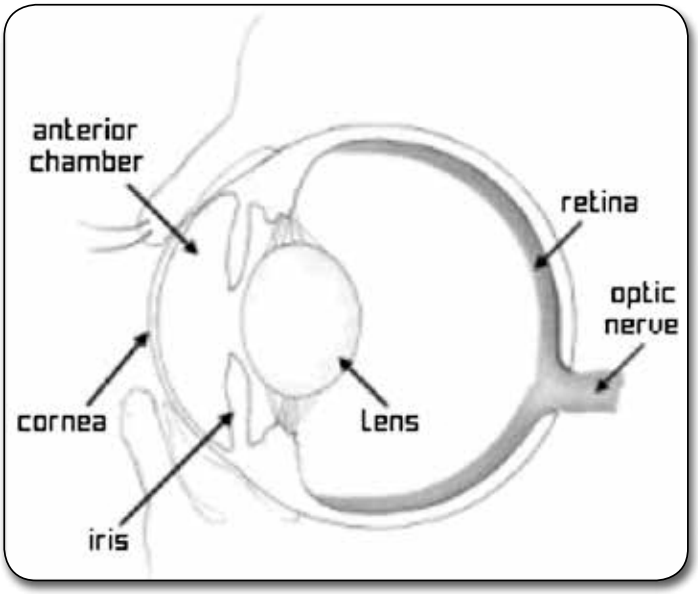
Depending upon the location and extent of these strands, they may interfere with vision. They may bridge from iris to iris across the pupil, iris to cornea (may cause corneal opacities), or iris to lens (may cause cataracts), or they may form sheets of tissue in the anterior chamber of the eye. In many dogs these tissue remnants cause no problems.

How Are Persistent Pupillary Membranes Inherited?

Inheritance is not defined.

What Breeds Are Affected By Persistent Pupillary Membranes?

PPM are known or strongly suspected to be inherited in the Basenji, Pembroke and Cardigan Welsh Corgi, Mastiff, and Chow Chow. This problem is particularly significant in the Basenji where the strands often bridge to the cornea, causing opacities which may impair sight. In the basenji the condition has been seen with optic nerve coloboma - a cavity in the optic nerve which, if large, causes blindness.



PPM are also seen in many other breeds, including the Akita, Alaskan Malamute, American and English cocker spaniel, Australian Shepherd, Basset Griffin Vendeen (petite), Beagle, Bearded Collie, Belgian Sheepdog, Belgian Tervuren, Bichon Frise, Bouviers des Flandres, Chesapeake Bay Retriever, Collie (rough and smooth), Doberman Pinscher, English Springer Spaniel, Golden Retriever, Gordon Setter, Havenese, Irish Setter, Labrador Retriever, Lakeland Terrier, Lowchen, Miniature Bull Terrier, Norwegian Elkhound, Nova Scotia Duck Trolling Retriever, Old English Sheepdog, Papillon, Poodle (all sizes), Portuguese Water Dog, Samoyed, Scottish Terrier, Shetland Sheepdog, Soft-Coated Wheaten Terrier, Tibetan Terrier, Welsh Springer Spaniel, West Highland White Terrier, and Yorkshire Terrier.

For many breeds and many disorders, the studies to determine the mode of inheritance or the frequency in the breed have not been carried out, or are inconclusive. We have listed breeds for which there is a consensus among those investigating in this field and among veterinary practitioners, that the condition is significant in this breed.

What Do Persistent Pupillary Membranes Mean To Your Dog & You?

Generally persistent pupillary membranes cause no problems. However if attached to the cornea or lens, the strands can cause opacities which may interfere with vision. The cataracts that can occur with PPM usually don't worsen.

How Are Persistent Pupillary Membranes Diagnosed?

PPM are seen in young dogs. You or your veterinarian may notice small white spots in your dog's eyes, or you may suspect that your dog's vision is impaired if the condition is severe. With an ophthalmoscope, your veterinarian will be able to see the membranous strands, and whether they adhere to the lens or cornea.

(Continued on page 13)

(Persistent Pupillary Membranes (Ppm) continued from page 12)

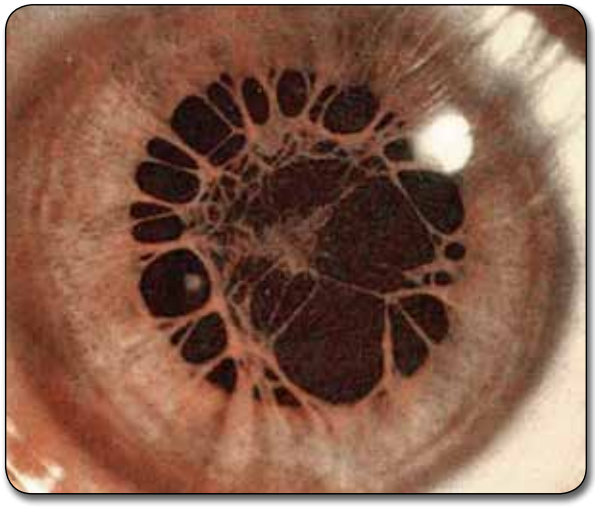
How Are Persistent Pupillary Membranes Treated?

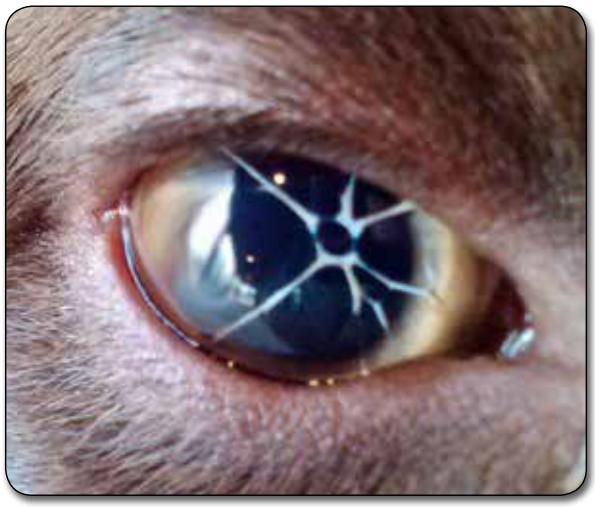
There is no treatment for the membranes themselves and in most cases there are no associated problems. If there is significant edema or "bluing" of the cornea due to adhesions, hyperosmotic eyedrops may help. Surgery may be required if there are extensive cataracts.

Breeding Advice

PPM's may or may not be a problem in a breed and/or individual dogs. PPMs are remnants of a fetal structure called the pupillary membrane. This membrane covers the pupil before an animal is born. It is part of the blood supply to the developing lens (the structure in the eye that focuses light on the retina). Normally the pupillary membrane completely absorbs before birth in foals and calves but is partially present and continues to disappear in neonatal dogs. Absorption may not be complete in puppies when the eyes first open and small strands or a web-like structure may be seen across the pupil. These strands normally disappear by four to five weeks of age. In some dogs these strands do not disappear and become PPMs.

PPMs may be found in several configurations in the anterior chamber. They may span across the pupil (iris to iris), from the iris to the lens, from the iris to the cornea, or they may float free on one end, only connected to the iris. In general, iris to iris PPMs cause no problems. They may be single strands or a forked structure. These PPMs may break and become less prominent as the puppy gets older, but they usually do not disappear completely. Iris to lens PPMs are more problematical. These PPMs cause opacities (cataracts) at the point where they are attached to the lens capsule. The cataracts do not usually progress and cause only minor visual deficits. Iris to cornea PPMs cause opacities on the cornea due to their ability to damage the corneal endothelium (the inner lining of the cornea). These opacities may be small or may be severe due to the development of corneal edema (fluid in the cornea). Severely affected puppies (with numerous strands) may be blind (they may improve as they get older). The strands may regress but do not disappear.





PPMs are found in many breeds of dog. In some breeds, PPMs are known to be hereditary. The Basenji is the most well-known but it is also found frequently in Chow Chows, Mastiffs, Pembroke Welsh Corgis, or Yorkshire Terriers. Members of these breeds have been shown to produce offspring with blindness directly associated with their PPMs. In these breeds, the mechanism of inheritance is not known but breeding any of these dogs with PPMs is highly discouraged.

WFA to Convene Pivotal Workshop in Pulmonary Fibrosis and Cancer

By Teresa Barnes

Leading the way in novel approaches to diseases that affect both West Highland Terriers and humans, the WFA is working with top researchers to better understand two independently life-threatening diseases that together pose an even greater threat. Scientists believe that by better understanding the relationship between Pulmonary Fibrosis (PF) and cancer, lung cancer in particular, new therapies can be found faster to save the lives of canines and humans.

Pulmonary Fibrosis is increasingly common in humans and domestic animals with more than 200,000 known cases in humans. Lung cancer, the deadliest form of cancer, affects approximately the same number of people in the U.S. with more than a million and a half deaths around the globe attributed to the disease, according to the World Health Organization (WHO).

In late February, the WFA along with National Jewish Health and the University of Louisville, will convene the Pulmonary Fibrosis & Lung Cancer Workshop in Louisville, Kentucky to move efforts forward towards targeting new therapies that save lives.

"Researchers from pulmonary and oncologic medicine will come seeking to advance collaboration between researchers to facilitate the development of a cohesive plan for novel comparative research efforts in the combined space," said Jesse Roman, M.D., Chair of Medicine at the University of Louisville in Louisville, Kentucky.

Roman, a top pulmonary physician and researcher, will lead the one-and-a-half-day workshop in Louisville. "Interestingly, the ongoing research in pulmonary fibrosis and lung cancer is starting to converge and has led to the understanding that these conditions share similar underlying mechanisms of susceptibility, development, and progression," he said. This further strengthens the noted epidemiological association between these two conditions."

"The WFA is honored to continue our work to drive research efforts in diseases that profoundly affect our beloved Westies," said Bebe Pinter, President of the WFA. "We, in partnership with our members, are making an impact on the scientific direction of research efforts."

Background

Great advancements occurring over the past two decades in the area of pulmonary fibrosis led to a better understanding of this disorder and culminated in the development of not one, but two anti-fibrotic drugs approved in 2014 for clinical use by the U.S. Food and Drug Administration. Intense work in this area continues and it is anticipated that further significant insights into pulmonary fibrosis will soon emerge. While this work is ongoing, much research is taking place in the area of cancer, especially lung cancer, where new biological pathways have been unveiled and effective drugs improving survival have been developed.

Despite much data generated linking pulmonary fibrosis to lung cancer, whether pulmonary fibrosis directly enhances susceptibility to lung cancer development is unclear, and the mechanisms by which pulmonary fibrosis may promote cancer progression remain uncertain.

(Continued on page 15)

(WFA ti Cinvene Pivotal Worshop continued from page 14)

Sponsorship

If you or your organization would like to become sponsors of the Pulmonary Fibrosis & Lung Cancer Workshop, see sponsorship levels below and contact WFA board member Teresa Barnes at 303-521-4080 or TeresaRBarnes@hotmail.com

$25,000

- Prominent placement of company logo on workshop signage by sponsorship level
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- Attendance to workshop for three (3) representatives

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- Prominent placement of company logo on workshop signage by sponsorship level
- Acknowledgement by sponsorship level on printed workshop materials
- Acknowledgement in pre and post workshop national press releases
- Verbal recognition by sponsorship level at meeting introduction and dinner functions
- Inclusion of one (1) company brochure in Workshop tote bags
- Attendance to workshop for two (2) representatives

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- Attendance to workshop for one (1) representative

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Why Dogs Eat Poop and How To Stop It

By Staff Writers | July 01, 2015

With permission of the American Veterinary Medical Association

Of all the repulsive habits our canine companions have—drinking from the toilet, rolling in swamp muck, licking their butts—nothing tops the disgusting practice of eating poop. Their motivation may not be to gross us humans out, but it certainly does. So much so, in fact, that poop eating is often a reason people try to rehome a dog or even opt for euthanasia.

There's a scientific name for this habit—coprophagia (kop-ruh-fey-jee-uh)—and also both behavioral and physiologic reasons why some dogs view dung as a delicacy.

If you have a poop eater, don't despair. There are ways to discourage the habit.

Although not deeply probed by science—there are few studies on it—poop eating is a relatively common phenomenon. In a 2012 study presented at the American Veterinary Society of Animal Behavior annual conference, researchers led by Dr. Benjamin Hart, from the University of California, Davis, found that:

- 16 percent (one in six) of dogs are classified as "serious" stool eaters, which means that they were caught in the act five times.
- 24 percent of the dogs in the study (one in four) were observed eating feces at least once.

Hart wrote, "Our conclusion is that eating of fresh stools is a reflection of an innate predisposition of ancestral canids living in nature that protects pack members from intestinal parasites present in feces that could occasionally be dropped in the den/rest area." His study consisted of two separate surveys sent to about 3,000 dog owners.

While it is repulsive to human sensibilities, it's not really all that bad from a canine point of view. Dogs evolved as scavengers, eating whatever they found on the ground or in the trash heap, so their ideas of haute cuisine is somewhat different from ours. In his Handbook of Applied Dog Behavior and Training, animal behaviorist Steven R. Lindsay says, that coprophagia "may be one of several appetitive survival behaviors that have evolved to cope with the periodic adversity of starvation." In other words, when food is scarce, you can't be picky.

Poop Eating is Normal For Mothers and Pups

For some species, such as rabbits, eating fecal droppings is a totally normal way of obtaining key nutrients. In fact, if you prevent rabbits from doing this, they will develop health problems, and young ones will fail to thrive.

Fortunately, dogs do not need to get nutrients in this manner.

It is, however, a normal, natural behavior at some canine life stages. Mother dogs will lick their puppies to urge them to eliminate, and clean their feces, for about the first three weeks. Puppies will also naturally engage in this behavior, eating both their own fecal droppings (known as autocoprophagia), and those of other dogs (allocoprophagia), as well as cats and other animals. Some dogs find horse manure and goose droppings particularly appealing.

Eating their own poop is harmless, but consuming that of other animals may cause health problems if the stool is contaminated with parasites, viruses, or toxins. In most cases, this behavior will fade before the puppy is about nine months old.

Some Facts About Dogs Who Eat Poop

When it occurs in puppies, coprophagia is generally considered part of the process of exploring the world around them. Most will be satisfied with a sniff, but a few will want, like human children, to put everything in their mouths. One bizarre fact: Dogs will rarely eat soft, poorly formed stools or diarrhea. They appear to be attracted most to hard stools. Frozen ones, in

(Continued on page 17)

(Why Dogs Eat Poop continued from page 16)

particular, are gulped down with relish. There is a reason why dog owners have coined the term, "poopsicle."

In his study, Hart made some other observations about why dogs eat poop:

- Coprophagia was more common in multi-dog households. In single-dog homes, only 20 percent of dogs had the habit, while in homes with three dogs, that rose to 33 percent.
- Poop eaters are no harder to house train than any other dogs.
- Females are more likely to eat poop, and intact males were least likely.
- 92 percent of poop eaters want fresh stuff, only one to two days old.
- 85 percent of poop eaters will not eat their own feces, only that of other dogs.
- Greedy eaters—dogs who steal food off tables—tend to also be poop eaters.

Why Do Dogs Eat Poop?

If your adult dog starts to dine on dung, you should consult with your vet to rule out such health problems as:

- **parasites**
- diets deficient in nutrients and calories
- malabsorption syndromes
- diabetes, Cushing's, thyroid disease, and other conditions that might cause an increase in appetite
- drugs, such as steroids

In many cases, dogs start to eat their own poop because of some kind of environmental stress or behavioral triggers, including:

- Isolation: Studies have shown that dogs who are kept alone in kennels or basements are more likely to eat poop than those dogs who live close to their people.
- Restrictive confinement: Spending too much time confined in a small spaces can cause the problem. It's not unusual to see coprophagia in dogs rescued from crowded shelters.
- **Anxiety:** often a result of a person using punishment or harsh methods during **housetraining**. According to this theory, dogs may eliminate and then eat their own poop to get rid of the evidence, but then they are punished more. It becomes a vicious cycle.
- **Attention-seeking:** Dogs eat their own poop to get a reaction from their humans, which they inevitably will. So if you see your dog doing this, don't overreact.
- **Inappropriate association with real food:** Dogs who are fed in close proximity to their feces may make a connection between the odors of food and those of poop and will be unable to tell the difference.
- **Scenting it on their mothers:** Lindsay writes that in some cases, puppies will get confused by sniffing fecal odors on their mother's breath after she has cleaned them. Also, sometimes mothers may regurgitate food that is mixed with puppy fecal matter. He calls this an "appetitive inoculation," which may set a puppy up to develop this bad habit.
- **Living with a sick or elderly dog:** Sometimes a healthy dog will consume stools from a weaker canine member of the household, especially in cases of fecal incontinence. Scientists hypothesize that this may be related to the instinct to protect the pack from predators.

How to Stop Your Dog From Eating Poop

Veterinarians and dog owners have seen improvements with a handful of strategies, including:

- **Vitamin supplementation:** There's been a long-standing theory that dogs eat feces because they are missing something in their diets. Vitamin-B deficiency, in particular, has been a prime suspect, and studies have backed this up. In 1981, scientists showed fecal microbial activity synthesized thiamine, a B-vitamin. Other research found other missing nutrients.
- **Enzyme supplementation:** The modern canine diet is higher in carbohydrates and lower in meat-based proteins and fats than the canine ancestral diet. Some people have had success with a meat tenderizer that contains papain, an enzyme.
- **Taste-aversion products:** The theory is that certain tastes and smells are as disgusting to dogs as the idea of stool eating is to us and that spraying certain substances on poop will make it less appealing. Many of these products contain monosodium glutamate, chamomile, pepper-plant derivatives, yucca, garlic, and parsley.

Perhaps the best way to stop the problem is through training and environmental management methods, including:

- **Keep the dog's living area clean**, including the yard, so there will be no poops for him to pick up.
- Cat owners **should keep that litter box clean or out of the dog's reach.**
- **Supervise your dog on walk**s, and pick up after him immediately.
- Training. **Work hard on the commands** "leave it" and "come." One simple exercise, suggested by *Debra Horwitz, DVM, Diplomate ACVB* and Gary Landsberg, DVM, Diplomate ACVB, is to teach your dog to come to you for a food treat as soon as he has eliminated. That way, the dog will develop a habit to run to you for a tasty tidbit, instead of reaching for the revolting recyclable on the the ground.

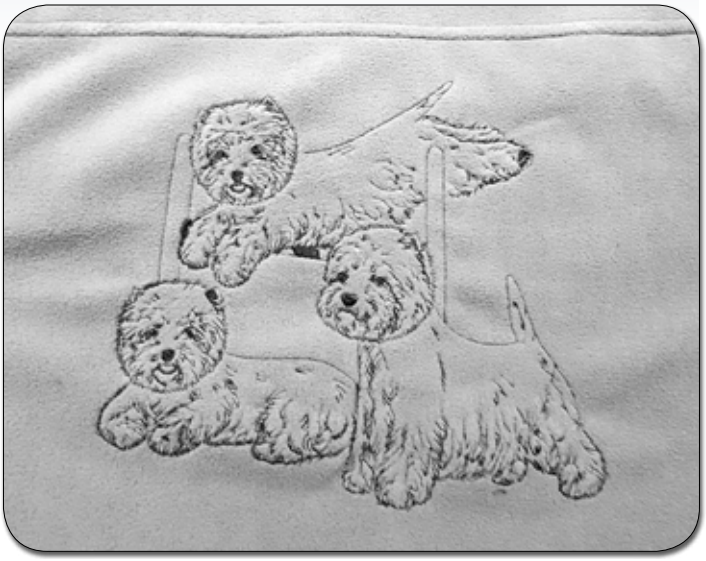
Sources: *Applied Dog Behavior and Training*, by Steven R. Lindsay; "Coprophagia in Dogs—Behavior," VCA Animal Hospitals fact sheet; "Coprophagia: The Scoop on Poop Eating in Dogs," Dr. Sophia Yin fact sheet

Fundraising Report

By Marianne Jacobs

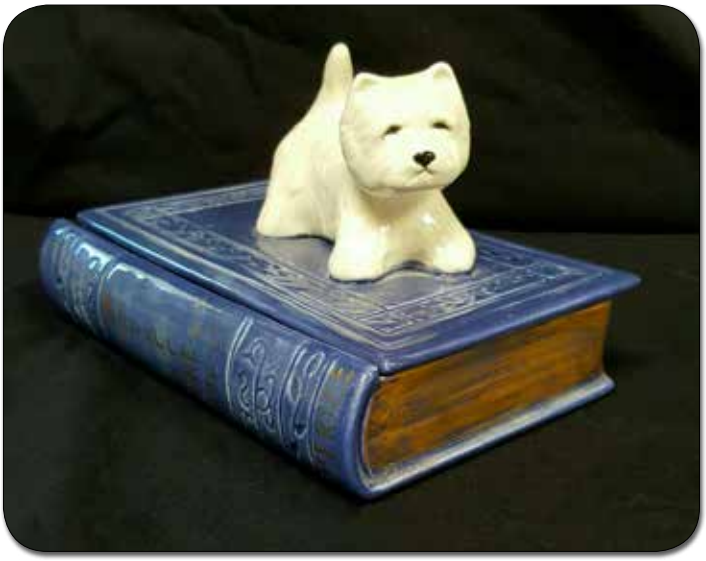
Montgomery Week Fundraising

The proceeds from the auction at the dinner and the Kimberton sales table were approximately $2,400. We send a big thank you to everyone who participated and donated to the Westie Foundation of America, Inc. (WFA).



Kirsten Fox Fleece Jackets

We have a limited number of jackets from the Kimberton sales table left. They are medium weight, no pill fleece. We have some L & XL left in raspberry and M, L & XL in the Medium Blue. They have the Kirsten Fox design embroidered on the back. Price is $59.00 + shipping. If you are interested in purchasing a jacket, please e-mail us at info@westiefoundation.org. We recommend ordering a size up.



Fundraising Auction

The WFA 2017 Facebook auction is scheduled for March 10-12. With your help, we will have tons of unique Westie items and some nice buy it now items. Last year we auctioned more than 400 items, including the buy it now item. We hope to surpass this number in 2017. What a great, fun way to support the WFA cause!

If you would like to participate in Facebook auction or donate to the auction, please join our Facebook group "Westie Foundation of America Fundraising Auction" and you will receive all the posts and updates on the auction.

For auction donations, you can also contact us by e-mail at info@westiefoundation.org

Or you can always make a donation by visiting our website www.westiefoundation.org

And, don't forget to shop on Amazon, via the www.westiefoundation.org website where the WFA gets 4 – 15% of each purchase made on Amazon. Please go to www.westiefoundation.org and click on the Amazon button on the bottom left of the home page.

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WFA's Wills, Gifts and Bequests package can help you make arrangements to ensure our Westie breed's health will be cared for into perpetuity.

www.westiefoundation.org/legacy-alliance.html

Westie Cartoon Caption Contest

Create the winning caption for this Westie cartoon. The winner will receive a WFA magnet. Please send your caption to bjpinter@msn.com before February 1, 2017. The winner will be announced in the next newsletter with his/her caption.

Create a Caption for this Cartoon

Copy of original watercolour by Ruth Sutcliffe, England

Winning Caption of Last Cartoon!

Mary Lowden

"Look--it's a rat in turtle clothing--let's get it!"

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