

2007 Issue 2



THE AMERICAN COCKER SPANIEL CLUB OF CANADA

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A Message From the Editor

Summer is upon us and the show circuit is in full swing. Good luck to everyone. In this issue of the newsletter you will find two interesting articles to read, submitted by Daria Edgecomb, with the permission of Jerold Bell to print them in the newsletter, minutes of the meetings of the last two meetings of the ACSCC, The Top Cocker Stats will be available on the website updated on a regular basis rather than actually putting them into the newsletter. If you find any errors or omissions, please contact me at cwagner@golden.net so I can submit the corrections. Deadlines for the next three issues are: August 30 and November 30, February 29.

Christine Wagner

REMINDER FOR ALL MEMBERS

When renewing your membership for 2007, please complete and include a Membership/Renewal Application when sending in your payment made out to the American Cocker Spaniel Club of Canada. This helps me to keep up-to-date records on all the members. Thanks in advance.

**Olive Simmons, Secretary/Membership
14 Caldwell St.
St Thomas, ON N5R 5J2**

New Member Applicants

The following have applied for membership in the American Cocker Spaniel Club of Canada. If you have any objections please for in writing to Olive Simmons, Secretary within 30 days of publication of this Newsletter.

JANET C. ROSS-KERR, 9123-118TH STREET, EDMONTON, AB T6G 1T6
ERDNA PUMMELL, 11868-221ST STREET, MAPLE RIDGE, BC V2X 5S6

If your personal information has changed, please fill out the attached and mail it to Olive Simmons,

Change of Personal Information

Name:		
Address:		
City:	Province:	Postal Code:
Phone Number:	Email Address:	

**AMERICAN COCKER SPANIEL CLUB OF CANADA
MINUTES OF MEETING HELD MAY 20TH, 2007**

A meeting of the American Cocker Spaniel Club of Canada was held in conjunction with the K-W Kennel Club shows in Kitchener, Ontario. The meeting was called to order at 12:55 pm by Lorraine (Lorri) Smith.

Present: Chris Wagner, Lorri Smith, Pat Wick, Kelly Gerritsen, Jean Ashley and Olive Simmons.

The minutes of the previous meeting were read. Pat Wick motioned to accept the minutes as read, seconded by Kelly Gerritsen. Motion carried.

The following new members were voted in: Christine Tippett, Syvlie D'Amboise, Cathy Richardson and Angela Luke.

Treasurer's Report: Bank balance as of April 30/07 \$5,131.57

A cheque in the amount of \$300.00 is to be sent to the ACSCC-Alberta Chapter as they are holding the ACSCC National Specialty for 2007.

Website: There was a discussion regarding this and Kelly Gerritsen motioned that we try to get a club member to manage the site. Seconded by Jean Ashley. Motioned carried.

Motion to adjourn by Chris Wagner, seconded by Jean Ashley. Motion carried.

**Olive Simmons
Secretary**

**AMERICAN COCKER SPANIEL CLUB OF CANADA
MINUTES OF MEETING HELD JUNE 2ND, 2007**

A meeting of the American Cocker Spaniel Club of Canada was held in conjunction with the NACA shows in Edmonton, Alberta. The meeting was called to order at 9:30 pm by Lorraine (Lorri) Smith, President.

Present: Kelly Ladouceur, Monique Malcolm, Jackie Forchuck, Marnie Wood, Donna Kjorsvik, Lorri Smith, Candice Sherbo, Carmen Lorenz and Olive Simmons.

There was no Treasurer's Report.

Daria Edgecombe (Nfld.) had emailed about having the "Blue Book" sent to new judges. This is a publication done by the American Spaniel Club. After a discussion regarding this, it was motioned by Candice Sherbo that we write our own "Book" for the new judges here in Canada. Seconded by Monique Malcalm. Motion Carried.

Website: There was a discussion of the website. Kelly Gerritsen had contacted Jackie Forchuck previously about taking care of our website in the future. Jackie has kindly volunteered to be our webmaster.

The members present were informed that the ACSCC - Ontario Chapter will host the ACSCC National Specialty for 2008. It is planned that an ACSCC Obedience Specialty and possibly Rally-O will be included at that time.

Other ACSCC Chapters will be contacted to see if they would like to host the National Specialty for 2009.

Members were reminded that our next Election Meeting will be in January 2008. Members will be sent information and nomination forms at the appropriate time this fall.

Motioned to adjourn at 9:55 pm by Donna Kjorsvik, seconded by Kelly Ladouceur.

**Olive Simmons,
Secretary**

BRAGS

On May 12th, attended another CARO trial at the 'Canine Sports Complex' in Buffalo, where Abbey earned her 3rd leg for her Rally Novice title, and also completed a 4th leg (1st place) as a bonus! Very well done considering she had never attended a rally obedience class, only learning from what I had gained through a previous class a year before. Then on June 2nd in nearby Fenwick, Ont., as another confirmation to Abbey's status as a SJA Therapy Dog, she very nicely earned her CGN test now making her Lurians Abbey Road RNCL, CGN. Very proud of my young girl (not quite two years of age) and all she has accomplished in such a short time!

Virginia Davies

URGENTLY NEEDED

We want your pictures of your winning dogs from the Trillium Specialties for the website. Please submit your picture along with the placement via email to Christine Wagner at cwagner@golden.net . Remember, the website is only as good as the information we put on it. This is the perfect opportunity to strut your stuff.

Hereditary Hypothyroidism: Understanding the disease process

Jerold S Bell, DVM, Tufts University School of Veterinary Medicine

(This article originally appeared in the Healthy Dog column of the AKC Gazette, August 2001.)

The thyroid gland controls the metabolic rate of the body. When the gland functions insufficiently, a condition known as hypothyroidism occurs. Dogs that are clinically affected may display one or more of the following clinical signs: weakness, lethargy, weight gain to the point of obesity, skin and coat problems, behavioral abnormalities, and infertility. Breed health surveys tell us that hypothyroidism is one

of the most common health concerns expressed by breeders.

Canine hypothyroidism is frequently misunderstood, misdiagnosed, and mistreated. Historically, it has been thought that 50 percent of cases of canine hypothyroidism are caused by autoimmune thyroiditis, and

the rest are caused by idiopathic hypothyroidism. What the experts now understand is that almost all primary hypothyroidism in dogs is caused by thyroiditis (autoimmune destruction of the thyroid gland), and

that this is a genetic disorder. Primary idiopathic hypothyroidism, if it exists at all, is a rare condition. The confusion comes from looking at blood test “snapshots” of hypothyroid dogs, and not understanding the whole “moving picture” of thyroid disease.

Measurable antibodies to the thyroid gland and to thyroid hormones develop in the blood of dogs affected by autoimmune thyroiditis. For months to years, these hormones attack and gradually destroy the normal thyroid gland tissue. It is only after a large portion of the thyroid gland is destroyed that the levels of thyroid hormone in the bloodstream drop. It is at this time that the clinical signs of hypothyroidism mentioned above may appear. Once the thyroid gland is destroyed, the body is no longer stimulated to produce the antithyroid antibodies. The dog is now in end-stage hypothyroidism. Most erroneous diagnoses of primary idiopathic hypothyroidism occur because the blood test is performed at this stage, when the gland is already destroyed and the autoantibodies are gone. Thus, the process that has led to this point is not seen.

In a study by Dr. Raymond Nachreiner and his colleagues Michigan State University, more than 50,000 canine blood samples have been screened for significant levels of autoantibodies to either thyroglobulin (TgAA), thyroid hormone 3 (T3AA), or thyroid hormone 4 (T4AA). Of the blood samples tested, 7.9% tested positive for thyroid autoantibodies. Dogs younger than two years of age tested positive in less than 5

percent of the samples, while the tests were positive between 9 and 11.5 percent of the time for between 2 and 6 years of age. The highest percentage of positive tests occurred at 4 years of age. The peak age for low thyroid hormone levels and no autoantibodies was eight years.

There are metabolic, infectious, endocrinologic, and cancerous illnesses that have no autoimmune components but which can nonetheless cause low thyroid-hormone values. This problem, which occurs less frequently is generally referred to as secondary hypothyroidism. It is not a hereditary thyroid disorder.

There is some controversy as to whether environmental toxins or vaccines cause autoimmune thyroiditis. These act as stresses on a dog’s body and could possibly affect the onset or severity of autoimmune thyroiditis. However, Dr. W. Jean Dodds, founder of the animal blood bank Hemopet, states that only dogs

that have the genetic potential can develop autoimmune thyroiditis. Therefore, any dog that has significant levels of blood-thyroid autoantibodies is considered genetically affected with hypothyroidism, and to carry

a gene (or genes) that causes the disorder. Dr. Dodds also reports that thyroid supplementation may be protective to dogs with thyroid autoantibodies even before their thyroid hormone levels drop.

We now know that measuring autoantibodies is the best available way to diagnose hereditary hypothyroidism. The Orthopedic Foundation for Animals thyroid-registry database states: “As a result of

the variable onset of the presence of autoantibodies, periodic testing will be necessary....Since the majority of affected dogs will have autoantibodies by 4 years of age, annual testing for the first 4 years is recommended. After that, testing every other year should suffice. Any test showing significant levels of thyroid autoantibodies confirms a diagnosis of hereditary hypothyroidism.”

Compounding the problem of just who has hereditary hypothyroidism are the myriad of conditions that can respond to thyroid supplementation. The clinical signs of hypothyroidism can appear in dogs that have conditions which are not related to thyroid problems, and these conditions may respond to thyroid supplementation. Just because a dog has a condition that responds to thyroid supplementation, it should not

be assumed the dog has hypothyroidism. Unless autoantibodies or low thyroid hormone levels are found, most of these dogs are probably thyroid-normal.

Studies on the mode of inheritance of hereditary hypothyroidism/autoimmune thyroiditis in dogs have been

inconclusive to date. What has been established is that some breeds have a much greater likelihood of developing autoimmune thyroiditis than do others, while some breeds have a below-average risk. (see tables). These breeds respectively carry a higher or lower genetic load of hypothyroidism causing genes. As opposed to human autoimmune thyroiditis (Hashimoto's disease), where there is a female to male 10-

to -1 ratio, hypothyroidism affects male and female dogs about equally.

Research into hypothyroidism has concentrated on perfecting the diagnostic tests and, in affected dogs, outlining the progression of the disease. There is no active research at this time into the mode of inheritance of canine hereditary hypothyroidism. It is hoped that with more reliable data on which dogs are

affected (producing autoantibodies at a young age) and the further development of canine genome screening, we can learn how to better control the disease through selective breeding. At this point, the recommendation that can be offered is the standard one for dealing with polygenic hereditary diseases: Breed normal-testing dogs that come from litters which have mostly tested normal.

* * * * *

Breeds with the highest prevalence (>9% affected) of hypothyroidism

(Data from the endocrinology lab at Michigan State University)

English Setter, Dalmatian, Basenji, Rhodesian Ridgeback, Old English Sheepdog, Boxer, Maltese Dog, Chesapeake Bay Retriever, Beagle, Cocker Spaniel, Shetland Sheepdog, Siberian Husky, Border Collie, Husky, Akita, Golden Retriever.

Breeds with the lowest prevalence (<3% affected) of hypothyroidism

(Data from the endocrinology lab at Michigan State University)

Chihuahua, Lhasa Apso, Pomeranian, Miniature Pinscher, Cairn Terrier, Basset Hound, Schnauzer, Yorkshire Terrier, Boston Terrier, Norwegian Elkhound, Greyhound, Portuguese Water Dog, Newfoundland, Bichon Frise, Welsh Corgi, Miniature Schnauzer, Cavalier King Charles Spaniel, Flat Coated Retriever.

Breeding Strategies for the Management of Genetic Disorders - Jerold S Bell, DVM Tufts University School of Veterinary Medicine, N. Grafton, MA

With each new generation of dogs, breeders ask, “How can I continue my line and improve it?” Aside from selecting for conformation, behavior and ability, breeders must consider how they are going to reduce the incidence of whichever genetic disorders are present in their breed. There are no answers that will fit every situation. There are, however, guidelines you can follow to preserve breeding lines and genetic diversity while reducing the risk of producing dogs that carry defective genes, or are affected with genetic defects.

Autosomal Recessive Disorders

In the case of a simple autosomal recessive disorder (in other words, a disorder caused by a single, recessive gene that is not sex-linked) for which a test for carriers is available, the recommendation is to test your breeding-quality stock, and breed carriers to normal-testing dogs. The aim is to replace the carrier breeding-animal with a normal-testing offspring that equals or exceeds it in quality. You don’t want to diminish breed diversity by eliminating quality dogs from the gene pool because they are carriers. As each breeder tests and replaces carrier dogs with normal-testing dogs, the problem for the breed as a whole diminishes. (See “The Effects of Genetic Testing: Constructive or Destructive?” in the “Healthy Dog” section of the June 2001 *AKC Gazette*.)

For some disorders there are tests known as linkage-based carrier tests, which can generate a small percentage of false positive and negative results. When using these tests to make breeding decisions, it’s advisable to first determine whether the results correlate with the test results and the known genotypes of relatives.

When dealing with a simple autosomal recessive disorder for which no carrier test exists, breeders must assess whether each individual dog in their breeding program is at high risk of being a carrier. This requires knowledge of the carrier or affected status of close relatives in the pedigree. An open health registry that is supported by the parent club makes it easier for breeders to objectively assess these matters. By determining the average carrier-risk for the breeding population, breeders can select matings that have a projected risk which is lower than the breed average.

If breeding a dog that is at high risk of being a carrier, the best advice is to breed to a dog that has a low risk. This will significantly diminish the likelihood that affected dogs will be produced, and can reduce by up to half the risk that there will be carriers among the offspring. Using relative-risk assessment as a tool, breeders should replace higher-risk breeding dogs with lower-risk offspring that are equal to or better than their parents in quality. Relative-risk assessment allows for the continuation of lines that might otherwise be abandoned due to high carrier risk.

Breeding a dog only once and replacing it with an offspring allows breeders to improve their chances of moving away from defective genes and also limits the dissemination of defective genes. When dealing with disorders for which carriers cannot be identified, the number of offspring placed in breeding homes should be kept to a minimum.

Autosomal Dominant Disorders

Autosomal dominant genetic disorders are usually easy to manage. Each affected dog has at least one affected parent, but it can be expected that half of the offspring of an affected dog will be free of the defective gene. With disorders that cause death or discomfort, the recommendation is to not breed affected dogs. To produce the next generation of a line, a normal full sibling of an affected dog can be used, or the parent that is normal can be used. A problem with some autosomal dominant disorders is incomplete penetrance. In other words, some dogs with the defective gene may not show the disorder. Roughly half their offspring, however, may be affected. If a genetic test is available, this is not a problem. Otherwise,

relative-risk assessment can identify which dogs are at risk of carrying incompletely penetrant dominant genes.

Sex-Linked Disorders

For sex-linked (also known as x-linked) recessive defective genes for which carrier tests exist, breeders should follow the same “breed and replace” recommendations as are outlined above in the discussion of autosomal recessive disorders. If there is no test, the defective gene can be traced through the pedigree. If a male is affected, he would have received the defective gene from his carrier mother. All of his daughters will be carriers, but none of his sons. By using relative-risk assessment to breed him to a female that is at low risk of being a carrier, you can prevent affected offspring, and select a quality son for replacement.

There are rare instances in which a female is affected with a sex-linked disorder. In such cases, she would have received the defective gene from both parents; specifically, an affected father and a mother who is either a carrier or is affected herself. If an affected female is bred, all the sons will be affected, and all the daughters would be carriers, so affected females clearly should not be bred. A normal male that is a littermate to an affected female, however, would be able to carry on the line without propagating the defective gene.

Sex-linked dominant disorders are managed the same way as autosomal dominant disorders are. The difference is that affected males will *always* produce all affected daughters.

Polygenic disorders

Polygenic disorders are those caused by more than one pair of genes. Most polygenic disorders have no tests for carriers, but they do have phenotypic tests that can identify affected dogs. (For a detailed discussion of polygenic disease management, see “Choosing Wisely” in the August 2000 *AKC Gazette*.)

With polygenic disorders, a number of genes must combine to cross a threshold and produce an affected dog. These are known as *liability genes*. In identifying a dog’s liability for carrying defective genes for a polygenic disorder, the breadth of the pedigree (that is, consideration of all siblings of individuals in the pedigree) is more important than the depth of the pedigree (consideration only of parent-offspring relationships.) A clinically normal dog from a litter that had one or no individuals affected with hip dysplasia (which is a polygenic disorder) is expected to carry a lower amount of liability genes than a dog with a greater number of affected littermates. This is why it is important to screen both pet and breeding dogs from your litters for polygenic disorders. Information on the siblings of the parents of potential breeding dogs provides additional data on which to base your breeding decisions.

Genetic disorders without a known mode of inheritance should be managed in the same way as polygenic disorders. If there are multiple generations of normalcy in the breadth of the pedigree, then you can have some confidence that there is less risk that liability genes are being carried. If a dog is diagnosed with a genetic disorder, it can be replaced with a normal sibling or parent and bred to a mate whose risk of having liability genes is low. Replace the high-risk parent with a lower-risk offspring that equals or exceeds it in other aspects, and repeat the process.

Genetic tests are extremely useful tools to help manage genetic disorders. Even when there is no test, or a known mode of inheritance, much can still be done to reduce the incidence of affected and carrier animals. The use of these guidelines can assist breeders in making objective breeding decisions for genetic-disease management, while continuing their breeding lines.

(This article was presented at the AKC Canine Health Foundation 2001 National Parent Club Health Conference, and appears in the “Healthy Dog” section of the November 2001 *AKC Gazette*.)

E-mailed permission from Dr. Bell to re-print articles. Thanks Christine!
Daria Edgecombe
Sunnycapp Reg'd.

--- Jerold Bell <jerold.bell@tufts.edu> wrote:

> Date: Mon, 12 Feb 2007 07:49:41 -0500
> From: Jerold Bell <jerold.bell@tufts.edu>
> To: Daria Edgecombe <sunnycapp2000@yahoo.ca> Subject:> Dear Ms.
> Edgecombe:
>
> You have my permission to reprint the articles in your newsletter.
> They must be reprinted unedited, and in their entirety. You may
> reformat them to your needs. Thank you for your interest in the
> articles.
>
> Sincerely,
>
> Jerold S Bell, DVM
> Clinical Associate Professor of Genetics Department of Clinical
> Sciences Tufts Cummings School of Veterinary Medicine 860-749-8348,
> fax 860-749-4760
>
>
> Quoting Daria Edgecombe <sunnycapp2000@yahoo.ca>:
>
>> > On another note...the American Cocker Spaniel
Club
> of
> > Canada has a quarterly newsletter - these articles would be so
> > beneficial for our membership. Would
> you
> > possibly give us permission to run both articles
> in an
> > upcoming issue?
> > Daria Edgecombe
> > Sunnycapp Reg'd.
> >
>
>
>