LIGHTING THE WAY

BEACON for Health

The Bearded Collie Foundation for Health

The Official Newsletter of the Bearded Collie Foundation for Health

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In This Issue

President's Reflections
Elsa Sell

Welcome to another issue of Lighting The Way, which passes its sixth birthday with this printing. Newsletter recipients are diverse in interests and background, but we hope there is always something for everyone. Authors devote their time to developing a topic. The editor spends lots of time arranging the articles, sending the draft to BeaCon's board of directors for proofing, managing the mailing list, and then setting up the printing and mailing.

BeaCon is pleased to announce that Cindy Alspaugh and Jana Jezkova have joined our board of directors. They were on board in time for the September "meeting", which is held electronically. Their resumes will be on the web site soon.

Understanding article content avoids incorrect assumptions. Here's an example of what happens when you don't read/get the written word. During the summer we did a small survey for breeders to gather their ideas about the significant decline in number of Beardie puppies recorded with AKC. Note: this means decline in number of puppies, **not** decline in number of reg-

survey was in the spring newsletter. The article was also linked in the introductory paragraph to the survey, just in case someone hasn't seen the newsletter article. Yet, one respondent stated "Perhaps the decrease is due to the increase in pet owners not competing in performance events and not seeing the need to register their beardie." This person and perhaps others missed reading a sentence in the article, which clearly stated "These new data are from litter registration forms that breeders submit. "

We'll have more on the state of both the number of registered Beardies and the number of puppies registered via litter registrations for 07 in the spring.

If you have comments on articles in the newsletter, suggestions for topics, or an article of your own, just contact myself or the editor, Gordon Fitzgerald.

Jana Jezkova won the sponsor contest by encouraging the largest number of persons to enter their Beardie in the open health registry. She won a Mike Sibley print. Perhaps we will have another such contest in the future.

I've included a few techie tidbits for those who want to improve their on-line safety or browser skills.

Finally, BeaCon's web site will be refurbished during the fall and when testing is completed, this will be announced on the respective Beardie lists.
Let us look forward to fall with its cooler and kinder weather.

Diagnosis: Kidney Disease
Or is it?
Jo Tucker

The difference between the clinical signs and blood analysis of kidney disease (causing renal failure) and Addison's disease is very subtle, that is unless you are aware of what you are looking for, and then the possibility of primary Addison's disease can become so obvious it stares you in the face!

Vets could be forgiven for not recognising the vague, non-specific clinical signs of Addison's disease if the consequences of a misdiagnosis were not so dire. It will most certainly result in the premature and unnecessary death of your dog.

In just three months, two Addisonian cases I have been involved in were originally misdiagnosed as definite cases of kidney disease. The dogs were put on a K/D diet (a special veterinary diet for dogs with kidney disease) and no improvement was seen. Both dogs were deteriorating, and when Addison's disease was mentioned to the vets they dismissed it. After the owners were made aware of the clinical signs and typical blood results of dogs with Addison's disease, it seemed very possible to them that their vets had misdiagnosed their dogs' condition. The information gave them enough courage to challenge their vets' diagnosis and in both cases this resulted in a confirmed diagnosis of Addison's disease, which undoubtedly saved the dogs' lives.

These two cases were almost identical, so how many other dogs are being misdiagnosed and dying needlessly? Many years ago when I had to take one of my dogs to the Royal Veterinary College, the vet looking after him told me that in the previous three days, four dogs had been admitted to the hospital with Addison's disease (three of them were Beardies). He said if veterinary surgeons only looked at their text books it would not be necessary for these dogs to be referred to the vet college! No one is saying that it is an easy diagnosis to make, as Addison's shares so many similarities with kidney disease (and other diseases), but there are tell-tale signs that not only should the vet be aware of but also the owners, and if these signs are apparent it should be enough reason to suspect Addison's disease.

Although Addison's disease has been diagnosed in a dog as old as 14, it is more likely for an aged dog to have degenerative kidney disease than Addison's disease. This article is more relevant to dogs of young to middle age, as this is when Addison's disease is more likely to occur, especially in a breed known to be genetically predisposed to this disease.

Much has been written about Addison's disease in the past. This article is intended to give a brief outline of the disease, concentrate mainly on the common factors, and more importantly the subtle differences between kidney and Addison's disease, and how the tell-tale signs can be identified from the blood results. You don't have to be a vet or scientifically minded, just look at the overall picture and be brave enough to challenge your vet's diagnosis and ask for an ACTH stim test, if for no other reason than to rule it out and give you peace of mind.

Primary Addison's disease (hypoadrenocorticism) is the result of an autoimmune destruction of the adrenal glands. The adrenal glands produce several hormones; the most important of these are aldosterone (a mineralocorticoid) and cortisol (a glucocorticoid). Insufficient production of aldosterone alters the electrolyte balance in the body, in particular the sodium and potassium levels, while too little cortisol makes the animal more vulnerable to stress whether physical, physiological or mental. Addison's disease can produce clinical signs such as:

Lethargy, depression, nervousness, weight loss, anorexia (no appetite), vomiting, weakness (particularly of the back legs), shaking or muscle tremors, limping, diarrhoea (with or without traces of blood), abdominal pain, dehydration, excessive thirst and urination, weak pulse, slow heart rate and abnormal heart rhythm, anaemia (pale gums) and collapse. One important difference between acute/chronic renal failure and Addison's disease is that, the heart rate in renal failure is more likely to be fast (tachycardia) and in Addison's disease it is more likely to be slow (bradycardia). Signs of the disease are most apparent when the dog is stressed.

Addison's disease is progressive and it can take many months before clinical signs are seen. Sadly, in some cases - if subtle changes are missed - the first sign can be death. Clinical signs are often vague to start with and can seem insignificant, but this is a common observation of the disease. Outward signs wax and wane and respond to fluid therapy or can worsen with stress. As the autoimmune destruction continues the adrenal glands will reduce in size until they are unable to produce enough adrenal hormones to sustain the dog. At this point the clinical signs, and abnormalities in the blood, are increasing and the dog is at risk of collapse, and subsequent death. It then becomes a serious veterinary emergency, and it is very desirable to achieve a diagnosis before this occurs. Unfortunately, it can become a fight against time to get the vet to even consider that the dog may have Addison's disease. A common remark is 'Oh, it won't be Addison's, we never see it'. ***Never see it, or never diagnose it?***

If your dog is visiting the vet regularly because of continuing symptoms, ask for a Complete Blood Count (CBC) and a full biochemistry blood test to be

done, and don't forget to ask for a printout of the results for your own records. The printout will show you a list of different chemical values, some of which are mentioned below. The most important ones to look at in a case of Addison's disease are the sodium and potassium levels. However, not all cases of primary Addison's disease will have all the clinical signs and blood abnormalities, and this has to be borne in mind. Also, there is a less common condition called atypical Addison's disease, or perhaps more accurately, glucocorticoid hypoadrenocorticism (GHA), in which the dog is deficient in cortisol only. At one time it was thought that this condition always progressed into true Addison's disease, mineralocorticoid and glucocorticoid hypoadrenocorticism (HGHA), but recent research has shown that this is not the case in the majority of cases of GHA. The researchers also found that in their referral hospital there was about 1 case of GHA for every 3 cases of HGHA. [3] Cortisol enables the body to cope with stress, and clinical signs relating to a deficiency may include: depression, nervousness, anorexia, weak pulse and collapse, particularly in stressful situations. The adrenal mineralocorticoid hormone regulates the conservation of sodium and excretion of potassium from the body.

HGHA cases will have **high potassium** and **low sodium** values, with a **ratio of less than 27**. Prior to diagnosis, Addisonian dogs often show a ratio of less than 23. However, the ratio alone is only suggestive, and not diagnostic, of Addison's disease. Individual electrolyte concentrations are also of relevance. As the disease progresses, the ratio will drop even further and the dog may collapse and become critically ill. [1]

Another possible difference between kidney disease and Addison's may be seen in the white blood cells (eg., neutrophils, eosinophils, lymphocytes). When a dog is poorly, he becomes stressed and this is reflected in the white cells. The neutrophil numbers would be expected to be high/normal or increased, and the eosinophils and lymphocytes would be decreased or low/normal numbers. This is called a 'stress leucogram' and is seen in both chronic and acute renal failure, but not in Addison's disease. A dog with Addison's disease may show a much lower white cell reading than would be expected in such a poorly dog. In fact there may even be reverse of what would normally be expected, eg., low/normal values of neutrophils and a higher value of lymphocytes and eosinophils (called a 'reverse stress leucogram'). [1] [2]

Routine Laboratory Abnormalities - Haematology & Biochemistry

INCREASED:
High Potassium (K)
High Creatinine,
High Urea, (BUN – blood urea nitrogen; or SUN - serum urea nitrogen)
High Urea/creatinine ratio (Azotaemia)
Increased Eosinophils
Increased Lymphocytes
High Bilirubin - in some patients
High Calcium (mild to moderate) – in some patients.
ALT- ALP - AST (Mild to moderate increase of liver enzymes) – in some patients

DECREASED:
Low Sodium (Na)
Low Sodium/potassium ratio (K:Na ratio - less than 27) Addisonian dogs often have a ratio of <23.
Low Chloride (80% of Addisonian dogs will have low chloride values)
Low Glucose – in some patients
Low Albumin (moderate to severe) – in some patients

Total white blood cell count (WBC) – in some patients
Red blood cell count (RBC or HCT)

The terms used above might sound a bit too scientific, but all of these values are clearly written on the laboratory report with the reference range, so it is not difficult to see if something is high or low and you may be able to build up a picture that may lead to a possible diagnosis. One point to remember, as Addison's is a progressive disease, remarkable values may not be present in the earlier stages. If the problems are ongoing regularly, recheck the subsequent blood results for any of the above abnormalities.

Points to consider:
Is your dog young / middle aged? Over a period of time, has your dog experienced several of the symptoms listed above, and has he/she responded well to fluid therapy?

Has your young to middle aged dog been diagnosed with kidney disease? Is he/she improving on the treatment/ diet provided by your vet? If the answer is no, then consider Addison's disease.

Do you have a dog whose breed is known to be genetically predisposed to Addison's disease?

Does your dog have relatives who have been diagnosed with Addison's disease or kidney failure at a young age, or other autoimmune disease? Speak to your dog's breeder; he/she may be able to give you valuable information.

Have a Full Serum Biochemistry panel and a Complete Blood Count test done and ask your vet for a copy of the results for your own records. Study the results yourself and check for the abnormalities listed above. If symptoms persist, have a further blood test done to see if there are any changes, but don't leave it too long in between (a week or less) as deterioration seems to quicken in the last stages. Keep each laboratory report for comparison. Blood testing is never a waste of money (in the long term it can save you money), and it provides a 'bench mark' on which to base further tests).

What to do if your dog continues to deteriorate and you suspect that he/she may have Addison's disease.

If your dog is extremely poorly, eg. very lethargic, call your vet straight away, regardless of the time, or if it is a weekend or bank holiday. Treat this as an extreme emergency.

Tell your vet of your concerns and suspicions. Relay clinical signs and draw attention to any significant blood results. It is surprising how many vets miss the significant sodium/potassium clue.

If the breed is genetically predisposed to Addison's, stress this to your vet. The vet should respond by asking you to take your dog to the surgery immediately. If your vet shares your suspicions he may carry out an ACTH stimulation blood test. (Unfortunately this is not possible over a weekend). If your vet does not respond to your concerns then start making telephone calls until you find one who does. Don't worry about offending your existing vet; your dog may be in extreme and immediate danger.

If your dog is too poorly for an ACTH stim test and has typical electrolyte imbalance usually seen in primary Addison's disease, then your vet will need to give **life saving treatment**, eg., saline (sodium) intravenous fluids to rehydrate, and bring down the dangerously high potassium levels and raise the sodium levels in the blood, and an intravenous injection of dexamethasone

to enable your dog to cope with the stress. [2]

NOTE: Dexamethasone is a glucocorticoid but unlike prednisolone it does not interfere with the ACTH stim test.

Giving intravenous fluids and dexamethasone will hopefully support your dog until the result of the ACTH stim test is known. Do not leave your dog without supportive treatment at this critical time. It wouldn't be the first time that a dog has died waiting for the results of the ACTH stim test to come through.

If the result is positive, your dog will be prescribed a primarily mineralocorticoid hormone called Florine-f. The initial dose is usually on the low side, and is gradually increased until the sodium and potassium levels are within normal range and clinical signs have resolved. The treatment must be given every day for life (usually the dose is split and given twice a day). For the first few weeks or so your dog will also be on prednisolone (a glucocorticoid). Once stabilised, there is usually enough glucocorticoid in Florine-f to control everyday stress levels, so the prednisolone can be withdrawn. At times of added stress, however, (caused by fireworks or even going to the groomers etc.,) the owner must give supplemental prednisolone to enable the dog to cope with the trauma. These are replacement doses and are vital to an Addisonian dog's survival and should not be confused with higher treatment doses of steroids used to treat inflammatory conditions. The body is designed to produce these hormones on a supply and demand basis. An Addisonian dog no longer has the ability to do this and they rely on the owner to anticipate stressful situations and administer the appropriate medication. It is not a difficult regime to follow once the dog is stabilised and you are familiar with the condition.

"We cannot change yesterday. We can only make the most of today, and look with hope toward tomorrow." Author Unknown

Initially, blood tests to check electrolytes should be done every 5-7 days and as clinical signs improve and blood results return to within normal limits the tests can become less frequent. Electrolyte blood tests should still be carried out every 6 months when the dog is stable.

In the United States, dogs with Addison's disease are more often treated with intramuscular or subcutaneous injections of desoxycorticosterone pivalate (DOCP – trade name Percorten-V.) Dogs seem to do better with these injections than with Florine-f. The dosage must be balanced by effect, both in the amount of the drug given and the interval between injections. This can range from every 20 to 35 days, but usually is every 26-28th day. Dogs usually do well on far less than the manufacturer's recommended dose by weight, so again initial treatment starts with a low dose, which can be increased as needed. Initially monitoring is also quite intense, but levels off once the appropriate dose has been established. After the initial injection sodium and potassium levels will be evaluated at 12 and 25 days to determine the size and timing of the next injection. DOCP has purely mineralocorticoid activity, and so it is necessary to give a small dose of prednisolone – usually 2.5 to 5.0 mg every 24 or 48 hours.

Many dogs with Addison's disease also are hypothyroid, and treating their thyroid problem tends to make treating the Addison's easier. Usually the dog will require less Florine-f or DOCP, and will handle stress better too.

Dogs with atypical Addison's disease do not have a deficiency of mineralocorticoid and so do not need to be treated with DOCP or Florine-f. They are successfully maintained on low doses of prednisolone every day or every other day. However, they should continue to be monitored to make sure that their sodium and potassium levels remain within the normal range.

The resolution of both clinical signs and blood results must be taken into consideration when evaluating the dog's progress and should not be used in isolation. Remember in Addison's disease especially, clinical signs usually reflect blood results and vice versa, but one may lag behind the other. If your dog is receiving an appropriate dose of Florine-f (this can vary from dog to dog) and his/her clinical signs or blood results do not stabilise after a few months, a full blood test to check kidney/liver/thyroid function etc., may be useful, or ask your vet for a referral to an endocrinologist, just in case there is an unidentified, underlying problem.

If the ACTH stim test does not confirm Addison's disease, it may have to be repeated at a later date if clinical signs and blood abnormalities persist. The result of the ACTH stim test reflects the 'current' adrenal function status and may not show a 'flat line response'- which is diagnostic of Addison's disease - until much later in the disease process.
The good news is that the prognosis for Addison's disease is usually excellent. Dogs can go on to live a happy, normal life on daily medication for many years, living well into old age, 15 or 16 years. It is certainly a much better prognosis than kidney disease.

There is plenty of general and practical information on Addison's disease on the internet, however, if anyone would like further, specific, information please email :

jo@cimda.fsnet.co.uk

Reliable information can be obtained by visiting BeaCon's website on:

www.beaconforheath.org

References:
1. BSAVA Manual of Canine & Feline Clinical Pathology
2. BSAVA Manual of Small Animal Endocrinology
3. Thompson AL, Scott-Montcrieff JC, Comparison of classic hypoadrenocorticism with glucocorticoid-deficient hypoadrenocorticism in dogs: 46 cases (1985-2005) j Am Vet Med Assoc. 2007; 230:1190-1194
Additional information obtained from Linda Aronson DVM

Stillbirths and Canine Herpes
Elsa Sell

Charlotte Lanning wrote to me as the newsletter was being prepared. She very recently had an unfortunate puppy loss experience that is worth sharing, in light of the recent breeder survey on declining puppy in litter registration numbers. If you're not acquainted with that information please see:
http://www.beaconforhealth.org/Newsletter%20Spring%202007%20E-mail1.pdf

In Charlotte's words: I remembered this survey as I just had my first experience with canine herpes in a pregnant bitch. She carried 10 puppies, six were stillborn, two died the first day and the other two are doing great so far; they are now 11 days old. Necropsy showed canine herpes. All I noticed was that all our dogs were sneezing for about a week when she was 4-5 weeks pregnant. As I read about it, it seems to be more of a consideration in Europe where many breeders vaccinate for it. I was just thinking that maybe that can

be the cause of many breeding problems. It seems breeders in Europe who start to vaccinate say they have fewer misses, larger litters etc.

Charlotte also gave the following details: There is absolutely nothing I could have done about it. I hadn't even been showing or taken my dogs anywhere before they got this virus. I have absolutely no idea where they picked it up. When the first dog started sneezing (10 yr old neutered male) he sneezed so hard that he hit his head on the floor. I thought he probably had something stuck in his nose so took him to the vet. Days later more dogs started sneezing and pretty soon they all did it. It was over in a few days and we had no other signs of a virus, they ate and were happy.

About the puppies, one of them was born about 2 inches long, completely wrapped in a greyish, small placenta. I didn't even realize at first that it was a puppy. Two were almost full size (they were all small though, the biggest ones - which are the ones that made it - were 7 and 8 oz, most of them were about 5 ounces) but not completely developed, they had some of their organs on the outside, something I have heard of but never seen before. One of them had his eyes slightly open. The other three stillborns looked normal. But those three looked like they could have been alive before birth, they were pink, full grown etc. I had a vague feeling that something may not be right because the bitch expanded very quickly between the 6th and 7th week but then she didn't get that much bigger.

It was pretty obvious that one of the remaining puppies would not make it. He died the first day. The second puppy that died was one I thought would survive, he was small but did OK. He then suddenly didn't want to nurse and died very quickly. From what I understand, the two remaining puppies should be OK now. They are 11 days old and doing great, very strong and chubby. The vet who did the necropsy said that the bitch (and I'm assuming my other dogs who had the virus) will now be immune, but I have also read that immunity doesn't last for life so I'm not sure which is correct. I guess there is a vaccine in Europe that has been used for years.

Addendum (Elsa): I did a google search on Eurican®Herpes 205 (the vaccine) on the Merial UK and USA sites to find nothing. We'll have a followup article about canine herpes in the spring newsletter. If anyone has a stillborn or a fading puppy death, please save the fetus (cold in plastic bag) and request a necropsy to rule out herpes infection, if you can manage financially to do so. Also, please examine the pup for physical abnormalities and record those for each puppy. Then contact me with your information.

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Techie Tidbits
Elsa Sell

Browsers.

Have you wondered why some web sites look odd, the navigation system buttons/bars are not well spaced or sit right on top of each other, the color seems strange, or the print is too small?

There can be several reasons. One is the design process itself and that is beyond control of the viewer. Let it be said that good design should take into consideration many different factors including accessibility and possible browsers that will be used to view the site.

Browsers (e.g., Internet Explorer (IE), Firefox, Opera, Netscape, Safari, or Internet Explorer for Macintosh) "look" at web sites in their own unique way. Not only does each browser "see" through different eyes, but also each browser has different versions such as IE 5, 6, or 7, and the different versions also "see" through different eyes.
You have some control then with respect to what you yourself can see with your browser. Optimal viewing comes almost always in the most recent version of the browser. If you don't know how to find the most recent version of your browser, try opening your browser, look for the help button in the top line, then look for "About … browser". Once you know what version you have, then google your browser, then search for the most recent version. Then download and install the newest version. These are free and every new computer comes with at least one browser already installed. From my several weeks' experience in working on BeaCon's web site revision, I see much greater stability and true representation of the web site in Firefox than in IE, even version 7.

If the print display is too small, enlarge the font size by going to "view" on the top tool bar of the browser and change to a larger size. Be forewarned, that unless the web site designer has accounted for visual accessibility (meaning you the user are going to ask for a larger font size to enhance readability), the text may run off the right side and you will have to scroll to see – each and every sentence.

When I was upgrading to IE7 recently, there was an option to improve readability. I clicked yes, and so far I have not had to enlarge print size of any web site.

You may be reluctant to upgrade your browser. Be brave. This is the technology age and keeping up with advances can make your internet experience more enjoyable.

Password Protection.

Did you know that information security experts recommend changing passwords every 30-90 days? Did you know that it is important to choose passwords that are hard to crack? Don't use proper names or any word that appears in an English or foreign-language dictionary. Even spelling words backward is a bad idea. To keep hackers at bay, select a combination of at least 8 alphabetic and numeric characters to include both uppercase and lowercase letters.

Suggestions include coming up with a phrase they can easily remember, like a movie title or riddle and creating a password with the first letter in each word. Other options include using a password managing software which stores login data in your hard drive and then retrieves it automatically each time you access an online account. Another option would be buying an encryption software that allows users to encode archived information, such as a word file with passwords. You might use two kinds of passwords—easier ones for sites that don't require much security such as online newspapers, and more complex ones for online banking and other security-sensitive transactions. Paola Singer, WSJ, 7/11/07, D1

A google search turned up several free on-line password generation programs:
http://www.thebitmill.com/tools/password.html
&
http://www.freerandompasswordgenerator.com

Test My Beardie for Addisons — Again???
Some FAQs
Lynne Corn

Many people are astonished to be

asked to update their Beardies for the Addisons Disease research project. Here's a rundown on what's going on.

What's involved in a test for Addisons?

There are very good tests right now to tell whether a dog actually has Addisons Disease (AD). The main one is called the ACTH stimulation test. It involves taking a blood sample, which is sent to one of several testing facilities around the country. This test is considered very reliable.

What about the test my club offered a few months ago?

That was not a test for AD. There is no test right now that will predict whether a dog will eventually get AD. What your dog did was provide a sample of cells from his cheeks. These samples were sent to a research lab so that the DNA of your dog could be studied. A crucial part of the test was that you provided at least a three generation pedigree. By the way, if you have a rescue dog and don't know its pedigree, or for any other reason can't provide a pedigree, there really isn't much point in having this test done, at least not at this time.

What happens to this sample?

The idea is to get a huge number of DNA samples, study their DNA, and compare the pedigrees and DNA of dogs with and without the disease. Then, with enough data, it may be possible to see if any particular DNA segment seems to be responsible for AD. And, knowing the segment involved, to see what DNA pattern results in dogs with a high probability of getting the disease.

Has this study produced any results so far?

Yes! There is already enough information to know some things. First, it is clear that the responsible gene is not on the sex chromosome, so either sex is can develop AD. Second, it appears that although multiple genes may be involved, there is one major gene that controls the disease and it is a recessive gene. That means that of the two copies of the gene (one from each parent), both have to lead to AD. (The dogs with two identical copies — in this case bad — are called homozygotes.) If only one copy is defective, the other, healthy one seems to be adequate to keep the dog healthy. A dog with one healthy and one defective gene is called a "carrier", and you can't tell by looking at the carrier that it is any different from any other healthy dog. AND there is another complicating factor. For some reason that no one understands right now, some small fraction of Beardies with two bad copies just never get the disease. And some who do get it, have forms that are not as severe as others. (This makes life interesting for the scientists!) If you want to learn more technical details, go to the BEACON website.

If this research pans out, what would be the practical effect for my breeding program?

It would be up to the breeder. Many breeders might want to have every dog tested in order to avoid breeding from a dog that was likely to develop the disease later, and some might decide not to breed even from carriers. Or, if they do chose to breed from a carrier, they might want to know the mate's status (normal, carrier, affected). Some breeders might test all the puppies in a litter and require that the puppy purchaser spay or neuter any carrier dog. One thing that probably *won't* happen, at least not very soon, is that all breeders decide to avoid breeding even from carriers. If that were to happen, the fairly widespread distribution of this gene in our breed (remember those carriers?) would mean that the remaining breeding population would be limited, and we Beardie lovers might miss out on some really nice genes for herding or temperament or structure or whatever if we limited breeding programs to non-carriers. In any case, I would probably ask the breeder if both parents had been checked as to their genetic status. I don't like unnecessary surprises when I buy a puppy. Besides, I would be more likely to trust a breeder who had done such testing.

My Beardie already donated a cheek sample for Addisons research. Why should I do it again?

You *don't* need to send another DNA sample – all you need to do is update your information about your dog's health status. It goes back to the fact that AD doesn't usually show up until the dog is 2-7 years old. Suppose that in 2005 your club got samples from 100 dogs. Right then, you could divide those dogs into two categories – the ones that had AD and the ones that didn't. Suppose there were 2 dogs with AD. Note that if there were a bunch of young dogs donating, odds are that they wouldn't have any symptoms yet. Move ahead to 2007. Now those dogs can be divided into THREE categories: (A) the 2 that were already diagnosed in 2005; (B) a few more (x dogs) that developed the disease since then; and (C) the majority, which are still healthy (98-x dogs). Suppose you don't send in the information about how your dog's health current status? Whether your dog has AD or not, the researchers won't know whether your dog is in group B or C. If they get reports about 40 of the dogs, and 3 more have AD now, then there are still 55 dogs out there and the researchers have no clue how they are. It will be assumed that they are normal. If they have developed Addison's and you don't report it, the various analyses will be interpreted incorrectly! And in genetic research, the ones without AD are just as interesting as the ones that are.

Are some Beardies of more interest than others?

Yes. If your Beardie has an affected close relative (parent, littermate, full-sib, half-sib, grandparent, offspring, etc.) there is a chance that your Beardie will share some crucial DNA with that relative that could be a major clue in understanding the disease. So please be sure to contribute a sample from that dog, and to keep information on your dog updated in the data base. Similarly, if you have an affected Beardie, do your best to persuade owners of related dogs to have theirs sampled and reported as well. Besides, since many Beardies benefit from early diagnosis of AD, you'll be doing those owners a favor, since they can be on the lookout, and get their dogs tested promptly if any symptoms appear.

So what am I updating?

You're just reporting whether your Beardie has developed AD since the cheek swab or previous update.

Update yearly.

Link to the update page: http://cgap.ucdavis.edu/healthupdateform.htm

Lynne Corn (and Cap, Ch. Desertstorm Made in America, HSAs, JHD)
Falls Church, VA

We are not passive spectators, but active contestants in the drama of our existence. We need to take responsibility for the kind of life we create for ourselves.
Nathaniel Branden, Ph.D.

"The greatest discovery of any generation is that a human being can alter his life by altering his attitude." William James

UPDATES
Elsa Sell

There are two sets of information on your Beardie that may need to be updated. People are understandably confused and think one update does it all. Not quite and here's why.

The Addison's Research Project (see more in Lynne Corn's article). Here you are updating your Beardie's health so the researchers will know that he/she still is healthy or has a new health problem. This is absolutely critical for the various analyses done in the lab. If you don't know when you last updated, go to the link below and fill in the information. Submit the form and the researchers will connect the information with your dog's file. The data are maintained confidentially by the research lab.

http://cgap.ucdavis.edu/
healthupdateform.htm

You do not need a username or password to obtain the form or to submit information.

BeaCon's Open Health Registry (OHR).

The registry is completely separate from the Addison's research project. Information in the OHR can be updated either by filling in a hard copy form –

http://www.beaconforhealth.org/
forms.htm
or going on-line to:

http://www.beaconforhealth.org/sqlweb

You will need your username and password to log in. If you do not have that information send me an email.

Reminders are sent by email (or letter for those lacking an email contact) every fall to owners of dogs who were living at the time of data entry or the last update. PLEASE respond by updating. DO THE UPDATE even if there are no changes in your or your Beardie(s) information. That simple step makes the spring yearly report far more accurate and your help is very much appreciated.

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HEARTWORM PRIMER
Cindy Mendonca

Every year we take our Beardies to the vet to be tested for heartworm. Hopefully, the vet tells us that the test is negative, sells us a preventative, and says to use it until after the first hard freeze. How many of us really know what heartworm is and how the preventatives work? What happens if we just skip giving the pills? Do we know why we have to give it even after the mosquitoes have all died off?

Heartworms have a five stage life cycle. I'll start with Larval Stage 1 (L1), although it's a bit of the chicken and egg. You see, you can't have L1 microfilaria in the blood unless you have adult heartworms (L5) infecting the heart and producing offspring. Unlike most parasites that produce eggs, heartworms bear live young. The L1 microfilaria have to be removed from the dog in a blood meal by the mosquito where they undergo two moults (Larval stages 2 and 3). They are only infective in the L3 stage and must be reintroduced into a dog in another blood meal. The L3 stage then migrates through the dog's system while changing to the L4 stage, ending up in the heart where it undergoes L5 or adulthood. It takes about 5-8 months for them to develop into full adulthood, that is, to mate and begin producing more L1 microfilaria.
The microfilaria produced by the adult heartworms in the body can *not* develop to adulthood inside the dog's body without ever having been in the secondary host (the mosquito). If the adult heartworms are not killed, they are still doing damage in the heart. The L1 microfilaria do nothing to *your* dog. But if they aren't destroyed, your dog becomes a "carrier" of heartworm when a mosquito decides to have a quick meal on it and starts the cycle all over again.

If your dog is infected by all male or all female heartworms, there won't be any microfilaria produced, but your dog will still have adult worms damaging its heart. While the Membrane Test, Knott's Test, and microscopic review of a hematocrit or whole blood MAY show evidence of microfilaria, it is hard to pick out the presence of heartworm. The tests involve drops of blood and microfilaria may not be in that portion tested. That's why when you have your animal checked for any type of parasite, the term isn't "negative" for whatever, but rather it is "not seen at this time." The most accurate test at 98% is the antigen test. It does not test specifically for microfilaria. It tests for a uterine protein from the female heartworm and can not detect male heartworms. Please note that all of these tests mean that, if positive, your dog ALREADY has adult heartworms.

It's possible to kill the infective microfilaria before they become adults through the use of heartworm preventatives and the specifics of each preventative should be discussed with your vet. To give you a headstart on the four main products, the following information is provided.

Diethylcarbamazine (Filaribits, Caricide, Nemacide) is an effective preventative that targets the L3 stage. L3 "lives" for three days before moulting into L4. These products, if available, are given daily. Adverse reactions are very rare with DEC unless the animal has an existing case of heartworm. It is very important to have the animal tested prior to beginning this drug. Filaribits are no longer available, but the basic chemical is still available in some markets.

Milbemycin oxime (Interceptor) and Ivermectin (Heartgard) are the other two main preventatives and they are designed to kill the L4 stage. They prevent the microfilaria from surviving to the adult stage which again is the stage that damages and eventually kills the dog through congestive heart failure if not treated. The L4 stage has a life span of *approximately* 45 days before it moults into L5. Why do we give the pill every 30 days then? It is not uncommon for people to forget and it's better to have some overlap time than to miss a cycle. Because there is also a slight risk that a dog with a high number of circulating microfilaria may exhibit a mild shock-like syndrome with either milbemycin or ivermectin, it's important to test before starting the monthly preventatives as well.

Revolution contains selamectin, but the principle to prevention is the same as milbemycin oxime and ivermectin.

If your dog is diagnosed with heartworm, the vet will decide what treatment to give by assigning it to a class. These are:
Class 1: Mild, asymptomatic.

Class 2: Moderate, anemic, heart enlargement (determined by x-rays), cough, intolerant to exercise.

Class 3: Severe, Right side of the heart is in failure, constant fatigue and cough
Class 4: Extremely severe, surgery must be done to remove the worms from the heart rather than attempting any type of chemical treatment.

There is currently one approved and proven treatment to kill adult heartworms other than surgery to remove

them. This is melarsomine (Immiticide). It is injected into the dog's back, two injections 24 hours apart, on either side of the spine in the case of Classes 1 and 2. With Class 3, one dose is injected and then a month later the two doses are given 24 hours apart. These injections are given between the 3rd and 5th vertebrae of the epaxial lumbar group *deep* into the muscle. Up to 30% of dogs will experience several days or more of pain. Less than 1% will have abcesses at the injection site or anaphylactic shock leading to death. In Class 3 cases where the dogs are hospitalized for treatment because of possible side effects, up to 10-20% may die. Death can occur with Class 1 and 2 if the dead worm breaks into a clot and goes into the lungs. In some cases, the vet may choose to forego treatment as possibly too hard on your dog, but elect to treat symptoms and use a strong dose of microfilaricide to keep it from becoming a "carrier." After that, the vet will likely prescribe a monthly to keep the microfilaria in check, but remember, it does nothing to the adult heartworms still present.

Preventatives kill what's already in the blood. That is, they are "retroactive," not proactive. Many people have the mistaken idea that heartworm kills the infective agent in the next 30 days (or 24 hours in the case of dailies) after giving the pill, so they stop giving the pill as soon as it gets cold -- instead of giving the last one *after* the freeze as directed by their vet. After all, it saves them money for next heartworm season if they already have a dose or two. BUT if there are L4s in the blood since the last dose, the heartworm then has the entire winter to grow before the next antigen test.
Treatment is very expensive and can run over $1000 depending on where you live. The first treatment may not always kill 100% of the adult worms either and a second and even a third treatment may have to be done.

Heartworm is definitely a case where "prevention is far better than the cure."

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BeaCon/BCCA Breeder Survey

Unfortunately, due to a glitch in the program, and despite extensive testing, several of the categories of answers were misrecorded so that we could not use them in this survey. The following is based on the usable results and the comments of the participants.

There were 76 respondents, not everyone responded to every question. The majority of responders were from the US - 44, but the UK - 14 and Canada – 9 were well represented and there was one response each from Australia and The Netherlands. Some were relatively new to the breeding arena while others were long time breeders. Time in the breed did not necessarily correspond to number of litters bred. Number or litters ranged from one failed breeding to 100, and number of years as breeders ranged from 0 to 34.

Of those responding, 50 of 76 (78%) reported no decline in fertility, while 17 of 76 (22%) did feel fertility had declined in their dogs. Interestingly, even those who didn't feel fertility was declining offered several explanations that were similar to those who did see a decline. Among those reasons cited for a decline in fertility common suggestions were:

1. The reliance on artificial insemination – this was seen to result in impaired sperm quality from fresh chilling or freezing, as well as failure to pick up nuances in a bitch's cycle, such as split or silent heats, which a skilled stud dog could remedy.

2. Reduced genetic variability in the breed – close in-breeding reducing the gene pool and creating breed bottlenecks. It was observed that sticking too closely to any particular line could be problematic, not just the popular show sires. Dropping dogs for perceived health problems could also reduce the gene pool – throwing out the baby with the bath water.

3. Dog factors: Poor sperm morphology – a high incidence of corkscrew tails was noted within the breed. Reduced libido in the dog.
4. Bitch factors: Delaying breeding until bitches have completed their show careers and so are past the optimal age. Unrecognized morphological problems – strictures in the vaginal tract. Increased resorption. An increase in primary inertia and bitches requiring C-sections.

5. Environmental factors, diet, climatic change.
Few comments were made regarding the number of stillbirths. Poor treatment by emergency veterinarians was cited as a reason for delayed C-section and puppy loss. This seems to be a theme I hear a lot. Most ERs have little to no experience with emergency reproductive issues, and tend to keep giving oxytocin injections rather than taking the dog straight to surgery when a C-section is indicated. Do check out the local facilities before you need them. Talk to your veterinarian, and make sure your back up is skilled and experienced in emergency reproductive issues. Several participants commented on smaller litter size, although some felt that this had been a problem a few years ago, but now the numbers had increased again.

While some people felt that there was a constant ebb and flow in the number of people looking for puppies, most seemed to feel that with two working parents and a busy life-style, smaller dogs that required less care than the hairy Bearded Collie were more appealing. More sedentary owners were also cited. Several felt that the efforts to combat a surge in popularity following the release of the Shaggy Dog movie had stressed the drawbacks of the breed, and made many people reject it. The comments that the Bearded Collie requires an experienced dog owner, and is not a breed for everyone that preface its appearance in the group ring at the Westminister KC show might have a similar effect.

Breeders too seem to be aging out and fewer new breeders seem to be coming into the breed, at least according to the survey. Lack of a clear, long term breeding goal and loss of the large kennel were also cited. Many people commented that perceived or actual health issues, and a small breeding population that had to be removed from the breeding pool due to age or health reasons terminated or at least slowed their breeding programs. Local competition by larger kennels was also cited as a reason to stop breeding.

Screening and finding the right owners for their puppies discourages several of our breeders. Several people report having puppies long beyond the traditional 8-12 weeks, not from choice but because there aren't the right homes available for them.

One person felt it is not politically correct to breed dogs when there are so many dogs in shelters – or at least in the public perception. Some cited people losing interest in dog shows due to politics – perceived or actual - or the increased expense. Others felt many people preferred competition in conformation and performance events as well as the social aspects of dog sports, but didn't want to deal with the risks and expense of breeding.

Some of the breeders were concerned about the drop in registration numbers, and feared that the breed numbers would fall below a critical minimum, and that there could be too small a gene pool to be sustainable. Others felt somewhat less pressure as the working Bearded Collies provide a back up gene pool. Others felt diminished breeding was a good thing, that demand should exceed supply so that only the most suitable owners got Beardies. No-one addressed how we might maximize genetic diversity while facing the apparent reality that there is less demand for the breed beyond avoiding the popular sire effect and all rushing to breed to the latest "it" dog.

Our study found that 44 out of 72 (61.1%) of our breeders encourage their puppy buyers to register with AKC and 28 (38.89%) do not. Many felt for the typical pet owner who had no interest in conformation or performance there were no perceived benefits. Many breeders do get close to 100% of their puppies registered, some by sending the entries in themselves, whereas a couple reported 0-5% registration. As with most things, falling registration figures are not unique to our breed. Each year AKC registrations fall 5 to 7% across all breeds.

This survey has produced no rock solid conclusions. We do hope though that it will set more Beardie breeders thinking, and open dialog within the breed so that we can all work together for its future well-being.

Linda Aronson, DVM, Elsa Sell and Eileen Beachell

"What you leave behind is not what is engraved in stone monuments, but what is woven into the lives of others .

Pericles

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Elsa Sell -
beaconbb@bellsouth.net
Chris Walkowicz-
walkoway.dogbooks@mchsi.com
Cindy Alspaugh
stonebaybeardies@yahoo.com
CJ del Valle -
mhari@mhari.cnc.net
Linda Aronson -
dvm@petshrink.com
Karen Drummond -
beardiemom@worldnet.att.net
Judy Howard -
beardiebunch@cabarrus.com
Jo Tucker -
jo@cimda.fsnet.co.uk
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ram5@po.cwru.edu
Jana Jezkova
glenalbyn@gmail.com

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TA DA!!!

Winner of the 1000th Beardie in BeaCon's OHR was :

Jennifer Sander's boy Phinn. Jennifer selected the prize of personalized USPS Stamps. Her photo was Phinn at White Sands National Monument in Colorado. His beautiful photo can be found in the contests link on BeaCon's web site.

E-Mail Contest

Don't forget, if you elect to receive your copy of the BeaCon Newsletter by e-mail, you will be entered into the drawing for some great Beardie "stuff". Just contact the editor at;
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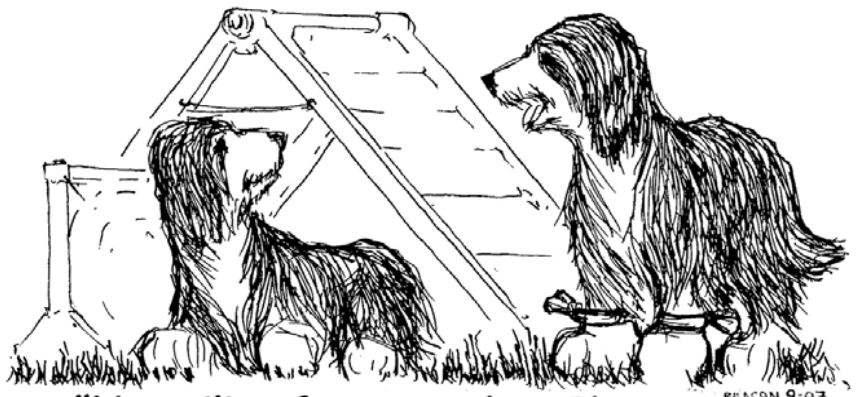

"No agility for me today. I've got athlete's paw."

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