

The Gather

Winter/Spring 2005

May 2005

President's Message

A new year is upon us, or passing us by, that has started off well for the club. I would like to begin this message by thanking all the members who attended the annual meeting at Marie Callendar's in San Ramon. The meeting was well attended and it was nice to meet those of you I had not met before. This year we added a raffle to the annual meeting, which I felt, was well received. Thank you Maryln and Sandy for donating the wonderful gift baskets. They were wonderful baskets and a nice addition to the annual meeting.

For those that were not able to attend the February herding aptitude test at Debbie Pollard's in Dixon, we are considering hosting another. The February test was a success with several breeds being represented. It was a great day, although a little cold. Debbie was wonderful with each of the dogs and we received a lot of positive feed back from those in attendance. This was a wonderful event to show that the club is continuing to host events that emphasize the breed's natural herding ability as stated in our Constitution. Thank you Jeanne for all your hard work in putting on this event.

As many of you may know, a DNA test is now available for CEA (Collie Eye Anomaly). This is a great tool that border collie owners and breeders can

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Club News

Membership Meeting

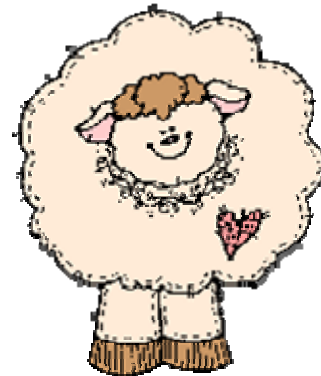
The next membership meeting will be held July 16 in W. Sacramento, CA in conjunction with BCCNC's Membership Fun Day.

20/20 Optigen Clinic

BCCNC, along with the Northern California Herding Group Association, will be hosting a 20/20 clinic Sunday, May 22 at the Coyote Hills Kennel Club show in Vallejo, CA. Flyer enclosed for additional information. All Participants must signup in advance. For additional information, contact Jeanne Hagen-Santiago (borderbrats@jps.net).

2005 A/OA Match

BCCNC will be holding an A-OA match and sweepstakes August 6, 2005 at the Richmond Dog Fanciers show in Dixon, CA. **Volunteers are needed for this event!**



Member Brags

Jamie Jackson's Domnik (Ch. Bruxly Midnight Adventure FdCh) earned his HXAd title at the Coso Dog Fanciers herding trial in Inyokern, CA January 17, 2005. This was Domnik's first herding trial and was handled by Debbie Pollard. He also gained two legs toward his HXAs title.

Jamie Jackson's Eoin (Ch. Shannara Voyage O'The Jerle finished his Novice Standard (NA) Agility title at the Mt. Diablo Dog Training Club's trial March 6.

Jayne Martinez's Panda gained his Novice Versatility Title in NADAC on New Year's Eve!!! Panda also gained his novice standard and novice jumper titles at the AKC TRACS trial at Rancho Murieta in December. On Thanksgiving weekend at WAG he also got his Level 3 Standard title in CPE and is now in Level 4 in everything except colors and wildcard.

Ch. Cymric Lochinbrae Christmas and Ch. Bayshore Shows Off at Shoreland are the proud parents of two girls and a boy. Carol Braden, Gary Guckes, and Pam & Richard Hartman are the proud Grandparents.

Patty Dudte's Neo (Hitop's The One) earned 2 legs in Novice Jumpers (1 first, 1 second) and 1 leg (first place) at the AKC trials in Portland in January.

Carol Braden and Gary Guckes' Taylor (Kensington's Made For Ewe) finished her championship at the Sacramento Kennel Club in April. Taylor was exclusively handled by Jamie Jackson.

Ch. Trumagik Tartan Ozzie, CD, OAJ, OA, CGC finished his Open Agility Jumpers at the Mt. Diablo DTC trail on March 6th. He ran the 39-second course in 24 seconds good for second place! Jan Teichman and AnneMarie Silverton own "Robbie".

Susan and Tim Joder's Hitop Sky's the Limit picked up a three point major at Sun Maid KC on Saturday March 12, 2005 under Judge Lesley E. Hiltz. We would also like to congratulate Maryln Fiscus on Handy finishing her Championship at Hanford.

Ch. Caliente's High and Handy finished her Championship at the Kings Kennel Club show. YEH!! WAY TO GO MARYLN AND HANDY. This is Maryln's first owned Champion and she has bred two finished Champions, Ch. Caliente's Kodak Moment and Ch Caliente's Casual Attire.

Ch Caliente's Kodak Moment, CDX, AKA "Kodi" just finished her CDX at Santa Clara DTC. We took a 2nd place, score 195 out of 48 dogs entered and if her handler (me) had not made a handler error by jamming the halt causing a no sit she would of had a 198. Darn those handlers.

Jack and Barbara Sciaroni's Kelsey received 2Q #21 and 17 points at the Mt.Diablo DTC agility trial on March 5. That is one more 2Q than needed for Kelsey's MACH. Still need those last 118 points.

Celestria Border Collies and Tonalee Border Collies would like to announce the arrival of their litter from the late CH Clan-Abby X-mas Cracker and CH Tonalee's Hiland Hope, FDCH (Laura).Laura delivered two girls and a boy (15 oz. each!) via a C-section on March 11. Mama and babies are all doing fine, although Laura is raring to get back to her agility and herding :-). Jeanne Hagen-Santiago and Tonya Stanley are the proud grandparents.

Membership Update

If you have not renewed your BCCNC membership, this will be your final newsletter.

Download your application form

If you need a renewal form please contact Barbara Sciaroni. Membership applications can be downloaded from the BCCNC website (www.bccnc.org).

2005 Member Statistics

<i>Renewals</i>	38
<i>New Members</i>	3
Total	41

2005 Officers and Directors

President – Jamie L. Jackson
Jamie@shannarabc.com

Vice President – Jeanne Hagen-Santiago
borderbrats@jps.net

Secretary – Barbara Sciaroni
busybc@comcast.net

Treasurer – Jack Sciaroni
agilbcs@comcast.net

Directors

Sandy Bohr
skiptobc@caltel.com

Maryln Fiscus
maryln@mindspring.com

Linda Hurlbut
Ljh777@softcom.net

CALENDAR OF EVENTS

May	22	Optigen 20/20 Clinic Vallejo, CA (Flyer enclosed)
July	16	Membership Meeting & Fun Day W. Sacramento, CA (Flyer enclosed)
August	6	Match (Following BIS) (Border Collies Only)
	27	Membership Meeting & Potluck Dinner Maryln Fiscus' "Club house" Angeles Camp
October	29	Membership Meeting

Thank You

I would like to thank the members who volunteered at the Scottish Games and brought their dogs - Marlyn Fiscus, Sandy Bohr, Linda Hurlbut, Katie Rodgers, Jayme Martinez, Kathleen Abbott. Also thanks to non-members -Kathleen Abbott's husband, Dan Santiago and Brian Santiago, and to previous BCCNC members Carla Popeney and Corie Meyers.

Jeanne Hagen-Santiago

Volunteers are needed for the following events:

***20/20 Clinic
Fun Day
August Match***

Please contact:

***Jeanne Hagen-Santiago (borderbrats@jps.net) or
Jamie Jackson (Jamie@shannarabc.com)
if you can help.***

use to their advantage. BCCNC, along with the Northern California Herding Group Association will be hosting an Optigen DNA 20/20 Clinic Sunday, May 22, 2005, in conjunction with the Coyote Hills Kennel Club show. This clinic will be open to all herding breeds that Optigen offers a DNA test for. This is not strictly a CEA 20/20 clinic. For more information on Optigen please visit their website at www.optigen.com. More information regarding the CEA DNA test is also available on their site. Jeanne is working diligently on making this clinic a success. A flyer containing more details about this event is enclosed in this issue. I am sure we could use more volunteers to help with this club event.

Once again, we will be hosting a fun day for all current and prospective members. Jeanne Hagen-Santiago has graciously volunteered her home for this event that will be held July 16. A flyer is included in this issue with all the details. I hope to see many of our members at this function.

The club is currently preparing to host an A/OA match following Best In Show at the Richmond Dog Fanciers Kennel Club show Saturday, August 6, 2005. More information will be available on this event as time draws nearer. However, we are currently seeking volunteers to help prepare for this event. Jeanne Hagen-Santiago will be serving as Match Chair and Jamie Jackson will be Match Secretary. Our other volunteers thus far are Katie Rogers, Barbara Sciaroni, Jack Sciaroni, Jayme Martinez, Linda Hurlbut, Sandy Bohr, and Darlene Atkinson.

If you, or anyone you know, would be interested in volunteering to help with this event, please contact Jamie or Jeanne.

An Events committee is currently in place working on ideas for a moneymaker event. If you would like to help with this task, please contact Margery Cavins at dharmadog@pacbell.net. A googles discussion group has been setup for members of the event committee to discuss all options. I would like to thank Margery for chairing this committee and keeping everyone focused on the task at hand, and thank you to all the other club members assisting Margery.

Know Your Breed

The Problem With Recessives: Genetics and Breeding

Written by and published with permission from Lisa Pruka

Most people with a basic understanding of genetics understand the principles of dominant and recessive as far as genes and their expressed traits. However I'd like to begin this discussion of recessives and their impact with a brief definition: a recessive gene is one in which the trait is only expressed when paired with another recessive. An easy example of this is the color gene: black is dominant and red (or chocolate) is recessive. When a pair of genes consists of a black (B) gene and a red (b) gene, the black is expressed because it masks the red recessive color. On the other hand when there are two red genes (bb) red is expressed because there is no dominant (B) gene to mask the recessive trait. I promise that this discussion will not get any more complicated than the last couple of sentences but this basic principle is vital to the rest of this article.

My focus is not going to be color, most people are completely comfortable with dominant and recessive genes where color is concerned. My focus will rather be genetic diseases. There are several genetic disease that are recessive in nature: Collie Eye Anomaly (CEA), Progressive Retinal Atrophy (PRA), congenital deafness and even Ceroid Lipofuscinosis (CL). In most cases these diseases are simple recessives which means that, just like the color example illustrated above, two copies of the gene are necessary for the trait to be expressed. The entire genetic sequences of these diseases has not been mapped at this point but we as guardians of the Border Collie breed need to be aware of the recessive and cognizant of what we do know about how it works.

Now for a couple of general examples.

Example 1: Let's say that a dog, I'll call him Joe, is bred to 9 different bitches and never produces a puppy with a given genetic disease. However when he is bred to bitch number 10, there is an affected puppy. Joe can now be considered a carrier of the recessive gene that causes the particular disease. When Joe was bred the first 9 times, those bitches did not possess the recessive gene, bitch number 10 however was also a carrier. As a result, the recessive gene was allowed the opportunity to be expressed.

Example 2: Let's say that a bitch is bred to 3 different dogs and none of the puppies have a given genetic disease. However, one of those puppies, I'll call her Meg, is subsequently bred and produces a puppy with the disease. Since we know that both parents have to be at least carriers of the recessive gene we can now say with some certainty that Meg, as well as the sire of her puppies, is a carrier of the recessive gene. However, we don't know if she got the recessive gene from her sire or dam.

Example 3: For this example I'll consider two dogs, Ben and Jill. Both of them have been bred before to other dogs and all of the puppies have been clear of any genetic disease. However when Ben and Jill are bred together, there are puppies affected with the genetic disease in the litter. In this example both Ben and Jill are carriers of the recessive gene, but each time they were bred before it was to a dog that was not a carrier of the recessive gene.

These three examples illustrate a couple of things about recessive genes. First, at the present time, a "random" occurrence of an affected puppy is the only way to find out if a dog or bitch carries a particular gene for a genetic disease. In this way our color example is very different because color is easy to see and often research into the appearance of a dog's ancestors will reveal whether or not that dog carries a particular recessive (i.e if a black dog has a red parent, it will carry the red gene recessively because there is no other color gene that it could have gotten from its red parent). However, the examples also illustrate the problem with recessives. In the first example not only is Joe a carrier of the "bad" gene, but ALL of his puppies – from all ten litters – are potential carriers of that same "bad" gene. In the second example, not only are the other puppies in Meg's litter potential carriers, but so are all the puppies produced by both of her parents with other mates. The third example is much like the second in that puppies from BOTH parents' matings with other sires/dams should be considered potential carriers as well. This is the case whether there is one affected puppy in a litter of ten or three in a litter of five.

This problem is one reason that recessive genes can become so predominant within a breed. Unless dogs are tested to find the genetic problem many of these diseases can lie undetected even in an affected dog. CEA and unilateral deafness are perfect examples because the affected dog often learns to adapt. Since carriers of the recessive gene do not show any symptoms of the trait that the gene affects it is even harder to identify those dogs. Some estimates indicate that a very high percentage of Collies (Rough & Smooth) are at the very least carriers of the gene that produces CEA.

Now let's pick a random genetic disease to give some substance to our examples. Since I've already introduced CEA in part of the discussion, I'll stick with CEA. CEA is actually close to having a test for finding the genetic marker for the recessive gene (more on that later). CEA is a genetic disease in which the retina, or back of the eye, does not form correctly and there are "blank spots" where the light does not focus. It is not a progressive disease and it is generally believed that there is little or no loss of sight in most affected dogs as they have adjusted to seeing with the blank spots since their eyes opened at a very early age. CEA is

also an easy disease to find at a very early age: the CERF exam when done before the age of about 8 weeks will find a puppy affected with CEA.

The problem with the recessive nature of the gene that causes CEA, again using my examples from above, is that there are an overwhelming number of potentially affected puppies that are only identified when a problem is found. Joe from example #1 has sired dogs in 10 litters; if an average litter is 6 puppies that means that there are potentially 60 puppies that could carry the recessive gene for CEA. If Joe had never been bred to bitch #10, the fact that he was a carrier would never have been discovered. It is also important to make note of dogs that have either produced the problem themselves or been the sire or dam of dogs that have produced the problem. In example #2 above, Meg's sire and dam should be noted as potential carriers of the recessive gene that causes the problem.

CEA has been discussed previously in the pages of *Borderlines* when Robyn Powley wrote about her affected litter in 1999. She wrote about example number 3 from above: a dog and bitch that had been bred previously to others were bred together and had affected puppies. As a result all pedigrees containing these two dogs were marked with "CEA carrier" identifying them as carriers of the disease. It is important to note that these dogs were not affected with CEA, they were simply carriers of the recessive gene that produce it. In the *Border Recorder*, Karen Hall wrote an article about CEA and identified an additional two dogs who were bred together and also produced an affected puppy. Anecdotally I know of at least three other litters where CEA has been produced. In each case, the breeder was surprised to discover a problem when the puppies went in for an eye exam. All of these cases illustrate the importance of doing the CERF exam on puppies before the age of 8 weeks.

So what is to be done with dogs that are either positively identified as carriers or are suspected or potential carriers of a recessive gene that causes a genetic disease? A lot of the answer depends on the particular genetic disease since some are fatal. First and foremost these dogs need to be clearly identified as carriers on pedigrees. However, many carriers may have other desirable qualities so it is important to keep those particular carriers in the breeding program. Owners of these dogs need to be sure not to breed them to other carriers. In this way, the presentation of the disease is prevented but the other desirable traits are passed on.

This process has been used in the case of CL. There are dogs with a history of CL in their pedigree that have exhibited other very desirable qualities. These dogs are still within breeding programs but their owners are very careful not to breed them to another dog with a history of CL in the pedigree. This method isn't always effective at keeping the disease at bay since it is the nature of recessives to show up unexpectedly. As such there are still a few random cases of

CL that occur but I think the practice has had a great effect in reducing the overall number of cases.

There is good news on the horizon as far as the recessive that causes CEA. They have developed a genetic test to find the recessive gene in carrier animals. The test hasn't been released yet, my understanding is that there is concern over how the test will be used. The developers, while recognizing the problem with recessives, also understand that simply removing ALL carriers from the breeding population may cause irreparable harm since those dogs may still have other desirable traits that are important to preserve.

An example of the negative effect caused by simply removing all carriers animals from the population happened about 20 years ago with Basenjis. In seeking to eliminate a kidney disease, pyruvate kinase hemolytic anemia (HA), they removed all affected animals as well as ALL carrier animals. As a result, they virtually eliminated HA but they increased the incidence of both Progressive Retinal Atrophy (PRA) and a kidney disorder called Fanconi's Disease. The theory is that when they eliminated the healthy carriers of the HA gene, they somehow eliminated the freedom from genes for PRA and Fanconi. (1998 C A Sharp, ASCA website)

Collie breeders in the US have been working to eliminate CEA, which is almost endemic within the breed, by breeding dogs with clear CERFs to mildly affected dogs. In this way all of the puppies will be carriers of the gene but only some will be affected. If by chance none of the dogs are affected, they will know that the dog with a clear CERF is in fact not a carrier. The incidence of CEA among Rough and Smooth Collies is being reduced in this way and at the same time, they are preserving all of the positive traits of their dogs.

Breeding for one trait is often discussed within BC circles. Whether that trait is color, conformation, height, or the presence of a recessive gene, other traits are affected in unknown ways. Maintaining breed quality and integrity is not always an easy straightforward task.

In conclusion, I'd like to restate several points: we must understand the nature of recessives, be ready to clearly identify those dogs identified as carriers or potential carriers of a recessive gene, use great care when breeding these animals and work to keep the great diversity of the wonderful breed we all love while making them healthier

