

ISAA HEALTH BULLETIN



Sustaining a Healthy Breed

A breed club serves several purposes. We want to share stories, photographs, experiences and to have fun doing so. Your Board supports activities that encourage fun and that will remain a top item on our agenda. Regional events are being planned as this is written and we hope you will attend at least one in the coming year.

We have recently received feedback from folks who do not wish to participate in the "fun stuff" and from those who don't want to participate in "the complex discussions of genetics". I would like to ask that we all consider that the healthiest approach for the club at present is to see these extremes as being on a continuum. It isn't an either/or issue, it's both and everything in-between. It is all important and we invite each of you to participate in whatever way you feel comfortable.

We have recently witnessed four breeders bravely step forward and share health information about their Icelandic Sheepdogs (ISD). From what we have been told, they have received tremendous support. They have set an excellent example of the first step that we must take to control canine genetic disease in our breed. That is, speak openly about health issues as they arise.

Issuing this special health bulletin is a step toward increasing our understanding of canine disease so that we are better prepared to meet that challenge. We must look at the issues logically as we become aware of them. We must also remember that our dogs are not one single trait and that one affected dog in a kennel does not reflect the entire kennel, rather, just one dog in that kennel.

Each dog within a kennel is not necessarily related and we need to be careful to resist the urge to label those who courageously provide us with precious information. The best course of action is to openly discuss dogs with defects or dogs that produce defects so that we can foster a learning environment, not an environment of fear and rumor. We ask you to demonstrate compassionate understanding to these individuals. They are our champions.

We know that all dog breeds, statistically speaking, are likely to be affected by four or five genetic diseases. Therefore, the sooner we discover what those diseases are, the sooner we can work toward finding a workable solution.

Our dogs are not just "hips" or just "eyes" and we cannot begin removing dogs from our precious gene pool without carefully examining what each has to offer. Some of the issues we need to carefully examine are a particular dog's littermates, what those littermates have produced, littermates of parents and what that dog has produced as offspring. We can only do that if we are informed. If you know of genetic defects in your dogs, we invite you to share that information with us.

One person recently asked me if we were experiencing a health crisis in the breed. This very human response to news that is difficult to understand is normal. However, it couldn't be further from the truth. Ours is a healthy breed. Yes, there are cataracts, one identified case of deafness and a handful of other known diseases, but there is not a cause for alarm.

However, there is a need for decisive action. We sit in the position to make decisions now that can affect the future of our beloved breed. We can ignore the issues, stick our heads in the sand and react out of fear or we can methodically study each issue and commit to finding solutions. Your Board has chosen the latter.

As more information is released by breeders and owners, we encourage each of you to carefully consider the issues, demonstrate the compassion that makes you ISD owners and commit, with us, to finding solutions to these very solvable problems.

In reading this bulletin, we have the opportunity to educate ourselves about a number of very topical issues, particularly deafness and eye disorders. Articles explore these issues, one a hereditary issue, one that is not yet determined as hereditary or not and one that is not hereditary. This is one step among many that the Board has taken to proactively inform and educate. We firmly believe that knowledge is power.

Some of the other proactive steps we are taking are:

1. Openly communicating with Iceland/DIF. We have established a direct line of communication. Our Icelandic liaison has done tremendous work gathering data. Maggy Pease, with help from several others, has compiled a comprehensive eye database that will be posted on the Health and Genetics website (www.is-pedigrees.com) site very soon.
2. Continuing the collection of articles and information regarding genetic issues pertinent to our breed and sharing that information.
3. Planning a regional CERF and cataract seminar.
4. Continuing work on the new Health and Genetics Website. Knox Rhine has spent countless hours adding pedigrees so that information is available to everyone. We are very excited about this new commitment to information sharing and openness.
5. Joining CERF as a club so that we will receive a breed eye report each year that will include dogs that are not public. While we will not know who they are, we will know how many affected dogs have been tested in our population.
6. Encouraging breeders to join CHIC with incentives offered for doing so.
7. Attending the Icelandic Sheepdog International Committee gathering in Iceland so that we can discuss these and other issues with representatives of our international ISD community.
8. Tracking cataracts worldwide in an attempt to discover affirmative links so that more informed breeding decisions can be made.
9. Starting a line item dedicated to funding research to find a test that will identify carriers of cataracts.
10. Starting a study group regarding the control of genetic disease. Joining the Purina Pro Club to raise funds for the study of genetic disease.

Most importantly, we call on all ISD owners to take the time and spend the money to CERF test their dogs and puppies and publish the results. To make breeding decisions that will positively affect the gene pool, we need information about the entire gene pool, not just the breeding population, so, we have asked our "pet" ISD owners to assist in our efforts. The best predictors of genetic disease are possible only when we know the genetic makeup of the grandparents and their litters, the parents and their litters, and the siblings their litters.

Our ISD community is international and Iceland sets a good example of testing and publishing results for eyes and hips. A number of other countries follow that practice, some to a greater and some to a lesser degree. We are now one of those setting a positive example by maintaining an open database and pedigree information. However, our information is only as good as what you are willing to release and we thank those who have bravely stepped forward and openly shared information that will only serve to preserve the healthy status of the Icelandic Sheepdog.

Best regards, *Donna McDermott* President, ISAA

Congenital Hereditary Sensorineural Deafness

Jo-Ann Secondino

Recently, we have learned that the Icelandic Sheepdog can be afflicted by congenital hereditary sensorineural deafness; this is most often seen in dogs and cats with white patterning or predominantly white pigmentation. Often referred to as white pattern deafness, this is not so much a genetic disease but a problem with the mechanics of this sensory organ caused by the lack of pigmentation in the skin of the inner ear. To help understand this disorder we need to understand why our dogs have white trim and the importance of melanocytes in sensory development.

During embryonic development melanocytes originate in the same part of the embryo and at the same time as the central nervous systems brain and spinal cord cells. Once the brain and spinal cord cells have formed those that are remaining at the edge of the central nervous system are called Neural Crest cells. These Neural Crest Cells then travel about the body to form the melanocytes of the skin, adrenal glands, dentine of the teeth, some bones of the base of the skull and voice box, the cornea of the eye, sensory cells of the ear and components of the involuntary nervous system in the viscera.

These melanocytes carry granules of pigment throughout the body, where ever one resides will produce a pigmented area. The “S” locus in the Icelandic Sheepdog influences the distribution of these cells resulting the Irish Spotting and Piebald patterns causing “white” patches of skin, the fur present at these locations will be white as well. Geneticists use the following symbols to represent the alleles that reside in this locus **S** = solid, **si** = Irish Spotting, **sp** = piebald patterning

The Irish Spotting “**si**” **pattern** of the **S** locus typically affects the belly, chest, muzzle, collar, blazes/stars, tip of the tail and socks at the feet. When present in a heterozygous pattern **Ssi** (Solid – Irish Spotting) we may see none to only a few of the typical patterns in our dogs. When present in a homozygous pattern **sisi** we will see the pattern interfere with the melanocytes distribution in all these areas. This gene is also governed by plus and minus modifiers which will turn “volume” up or down on the patterns, minus modifiers may result in short socks, skinny blazes for example where plus modifiers can make very wide white collars, very wide blazes which may in an almost predominantly white face.

The piebald pattern interferes with the migration of the melanocytes in a random pattern all over the body. When Heterozygous for **Ssp** (Solid – Piebald) or **sisp** (Irish Spotting- Piebald) there will be no expression of the pied pattern. When heterozygous **spsp** you will see an all over spotting pattern. When combined with minus modifiers the spotting will be minimal, where plus modifiers are present it can result in an extremely pied dog which may be predominantly white.

When the spotting also occurs in the inner ear resulting in unpigmented skin in the inner ear, especially in the cochlea, deafness can result. The absence of the melanocyte cells cause nerve endings to atrophy, the stria vascularis degenerates, this is a vascular bed responsible for the secretion of endocochlear fluid which is essential to the transferring of sound to the sensory hair cells. This degeneration cuts off the blood supply to the sensory hair cells and they will die off in the first few weeks of the puppy's life, resulting in deafness. The deafness can be unilateral or bilateral in an affected dog. Please note that you cannot tell the color of hairs in the inner ear by looking at any visible part of the dog's ears (including the hair around the ear canal). Although some dogs with white hair on their ears might be deaf, deaf dogs can have colored ears as well. When the spotting genes affect the distribution of the melanocytes in the eye, it suppresses the brown in the iris causing it to become blue.

It is often difficult to diagnose a unilaterally or bilaterally deaf puppy at home as response to sound must be done without any other stimuli that can influence their reaction such as visual clues, vibratory clues, touch and air movements. It is even more difficult in cases of unilateral deafness. Behavioral cues may be that a puppy doesn't wake up after weaning for a feeding unless bumped by a littermate or a pup is not deterred by a littermate's yelps when playing aggressively. The only way to be certain is through Baer testing.

Baer testing (Brain Stem Auditory Evoked Response) is an electro-diagnostic test which records the electrical activity in the cochlea and auditory pathways in the brain. Tiny sub dermal electrode needles are inserted subdermally in front of the ear and on top of the head, a ground electrode is placed behind the eyes or on the neck, rarely does a dog exhibit any discomfort during this process and there is no need for anesthesia. Foam earphones are placed into the ear and an air generated stimulus click in most of the frequencies that a dog can hear is computer generated. The test only detects that the dog has or does not have hearing, not which frequencies they are able to hear. Living with a deaf dog may be a challenge but is not impossible. Strides have been made to help people to communicate with deaf canines. They can be trained using sign language, vibrating collars have been developed to get their attention, they can learn to respond to flashing lights, and will key off of other animals in the household, online support groups are also out there. It is thought that deaf dogs make excellent tracking dogs as they have increased olfactory senses and have no auditory distraction. Once you have learned to communicate with these dogs they can live normal lives, compete in obedience and agility, just imagine a dog not distracted by other dogs barking at a performance event!

Tracking Eye Disease in the Icelandic Sheepdog

Jo-Ann Secondino

As our Icelandic Sheepdog community grows, so does the need to increase our efforts to track and record hereditary eye disease in this very special breed. The Icelandic Sheepdog is primarily a healthy one, although there are a few conditions of the eye, some hereditary, others whose origin is yet to be determined, that have the possibility of increasing in frequency as our population grows. Tracking the affected dogs and openly sharing this information can help breeders make informed decisions as to their pairings. Discussing and understanding what these conditions are and how they affect our dogs can help us all grow together from being merely breeders into true stewards of the breed, keeping it healthy for future generations.

Persistent Pupillary Membrane

During the embryological development of the eye, the front or anterior portion of the eye is filled with a solid sheet of mesodermal tissue, called the pupillary membrane. This covers the iris and developing pupil to provide a blood supply to the developing lens connecting with a network of blood vessels known as the Tunica Vasculosa Lentis Anterioris. This tissue in the last 3 weeks of fetal development begins to atrophy and degenerate, as it disappears, the pupil forms. In most puppies this degeneration is complete at six weeks after birth. Occasionally in the Icelandic Sheepdog, the degeneration of the pupillary membrane is incomplete and can manifest itself in several ways:

Fine strands of the mesodermal tissue can remain attached to the collarette of the iris (Center of the iris) crossing the anterior portion of the iris or crossing the pupillary aperture, this will form iris to iris PPMs. This can be a single strand or a cobweb like structure, this form is generally considered benign causing few problems. Although on occasion the tissue may only be attached on one side of the iris, this loose strand of tissue has the possibility of attaching to the cornea, resulting in a lesion on the cornea creating an opacity.

Iris to Cornea PPMs is a bit more problematic. The mesodermal strand attaches to the Corneal Endothelial Surface (inner lining of the cornea) causing opacities. These opacities can remain small and unchanging or mesodermal attachments can cause corneal edema (fluid in the cornea) in severe cases blindness can result. Multiple remnant attachments can result in a large bluish white opacity as the result of extensive corneal endothelial disruption.

Iris to Lens PPMs will cause opacities on the lens capsule; these cataracts generally do not change or become any larger. This form does not generally cause significant loss of sight.

In severe cases an entire sheet of mesodermal tissue may not degenerate obscuring the pupil, causing blindness.

It is unknown at this time if this condition is an inherited one in the Icelandic Sheepdog, continued study is necessary to determine this. In breeds such as the Basenji it is considered heritable. It is recommended that you do not cross a pair affected by PPMs as a precautionary measure. When CERFing young puppies, sometimes minor development delays will result in

portions of the pupillary membrane to remain longer than normal, re-CERFing in 6 months to a year is recommended to insure that the condition has resolved itself.

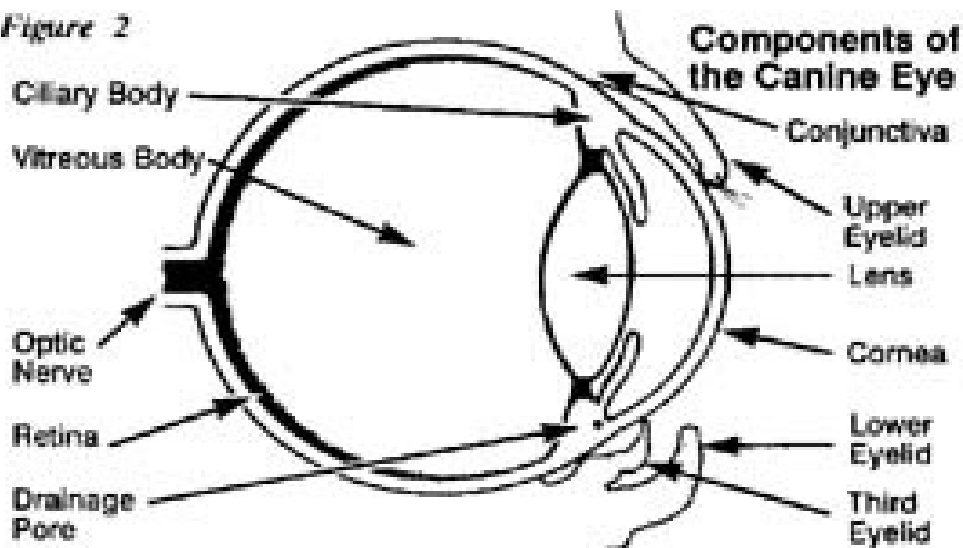


The arrows point to persistent pupillary membranes in this dog. The adjacent white specks are scars on the inside of the cornea due to the membranes touching the cornea.

Persistent Hyperplastic Primary Vitreous

In the posterior of the eye is a jelly like substance known as the vitreous, the vitreous fills the space between the lens and the retina. While in fetal development the vitreous goes through three stages of development, in the primary vitreous phase a large artery forms at the back of the eye growing towards the lens. Known as the Hyaloid artery, this vessel divides and branches out to form a network of blood vessels needed to feed the developing lens, this vascular network is Tunica Vasculosa Lenticis, part of the same network that feeds the Pupillary Membrane. Like the Pupillary Membrane, prior to birth these vessels degenerate and disappear but in Persistent Hyperplastic Primary Vitreous, this system fails to completely degenerate leaving fibrovascular plaque on the posterior lens capsule and a possible posterior cortical cataract. The effect on sight can range from nearly nothing to blindness. In severe cases, the lens is operable but hemorrhaging may result due to the remaining vascular system. While in the Doberman and Staffordshire Terrier it is suspected to possibly be heritable, the origin of the condition in the Icelandic Sheepdog is unknown.

Figure 2



Retinal Dyplasia and Retinal Folds

The retina is a unique structure, during embryonic development the retina is formed from the front portion of the primitive neural tube. The neural tube becomes the central nervous system, brain and spinal cord. The retinal structure is located at the back of the eye, it receives light, converts it into electrical signals, transmits this signal via the optic nerve to the brain; the brain interprets this signal as visual images. Occasionally there are defects or malformations in the forming of the retina; this can be caused by heredity or environmental influences during gestation. Dysplasia is defined as the defective development of an organ or structure; Retinal Dysplasia occurs when the two developmental layers of the retina do not form together properly.

Retinal Folds - folding of 1 or more areas of the inner retinal layer, this folding can be linear, Y shaped or circular and results from the non-attachment of the neural part of the retina, the neurotina, to the underlying retinal pigment epithelium. Complicated folds, where there is a proliferation of photoreceptor and RPE elements are diagnosed as rosettes. Retinal Folds are the mildest form of this condition; they can be accompanied by variable degrees of retinal detachment and degeneration. The effect to the dog's vision is minimal and may represent small blind spots in the eye which the dog does not even notice. This problem can be detected in puppies at 6 to 8 weeks and can at times resolve itself while the dog matures

In **Geographic Retinal Dysplasia** there is a larger area of abnormal retinal development. There will be apparent areas of thinning, folding and disorganization of the retina. This can lead to large fields of visual impairment. **Detached Retinal Dysplasia** is a severe disorganization where the two retinal layers do not come together at all and detachment occurs. Dogs with retinal detachments are completely blind. This eye condition is suspected to be a genetic autosomal recessive in most breeds, although prenatal exposure to the canine herpes virus and parvovirus while the bitch is in whelp has also been attributed to causing Retinal Dysplasia. There is no treatment for Retinal folds or Retinal Dysplasia although it is also not a progressive condition.

Cataracts

A cataract is defined as a partial or complete opacity of the lens and/or its capsule. In cases where cataracts are complete and affect both eyes, blindness results. A cataract can be caused by genetics, age, trauma, ocular inflammation, genetic retinal atrophy, persistent pupillary membranes, persistent hyaloid remnants, specific nutritional deficiencies, congenital abnormalities and by other specific metabolic disease.

The lens is a soft transparent structure located in the eye behind the iris. Like a camera lens, it changes shape to allow the eye to focus on views near and far away when pulled by small muscles within the eyes structure. The lens has no vascular blood supply, so it depends on its surrounding capsule to provide nutrients and to maintain its shape; the capsule also provides an anchoring point for the muscles which manipulate the lens.

Cataracts are the most common intraocular lesion and cause of vision loss in a dog. Their size is highly variable from a very tiny lesion which the dog will barely notice and see around, to one which completely obscures the lens and causes blindness. There are some types of cataracts that will never change in shape or size while others will continue to grow. Other types of cataracts only affect the capsule, which covers the lens, obscuring vision. Genetic cataracts can be diagnosed in dogs between the ages of 2 months to seven years old. From CERF's guideline "breeding is not recommended for any dog demonstrating partial or complete opacity of the lens or its capsule unless the examiner also checks the space for significance of the above punctuate cataract unknown. The prudent approach is to assume cataracts to be hereditary except in unusual cases known to be associated with other causes."

Although many feel it is only important to check breeding dogs for hereditary eye disease, it is also important to receive an early diagnosis of eye disease in our pet population as well. Some types of cataracts cause inflammation to eye, if allowed to go without treatment this can lead to severe dam-

age to the dog's eye, glaucoma or retinal detachment. If not treated promptly, this can not only lead to blindness but also extremely painful conditions for your pet or potential loss of the eye itself. This should be considered as part of your dog's wellness exam periodically throughout the years. The Canine Eye Registration Foundation (CERF), breed and kennel clubs sponsor clinics throughout the US, they are frequently held on weekends and for a reduced rate. For a listing of upcoming CERF clinics or a Veterinary Ophthalmologist near you please go to CERF's website at <http://www.vmdb.org/cerf.html>

There are several types of inherited cataract and they are often breed specific as to their age of onset and severity, some of the cataract diagnoses that other breeds experience are:

Posterior Polar Cataract whose age of onset is 5, impairing vision in the Norfolk terrier

Posterior Cortical Cataract in the Norwegian Elkhound, whose age of onset is 1 to 3 years, mildly affects their vision but severely affecting the Poodles at an age of onset of two months to 3 years

Posterior Subcapsular Cataract in the St. Bernard whose age of onset six months to 8 years while the Norwich Terrier experiences its onset at six months to 2.5 years impairs the vision of both breeds

The Posterior Y-Suture Tip Cataract can often severely affect the West Highland White Terrier, age of onset 4 years.

Posterior Cortical Cataracts can be diagnosed in the Samoyed from 6 months to 3 years and usually impairs their vision while in the Pomeranian age of onset is 4.

A **Posterior Cataract** age of onset is up to the age of 8 years in the West Highland White Terrier. The Samoyed's vision is rarely affected by an **Anterior Subcapsular Cataract** whose age of onset is up to 5 years old; while the Schipperke can be affected up until 8 years old.

The **Anterior Cortical Cataract** age of onset in the Poodle is 2 to 5 years and their vision is only mildly affected, where in the Whippet it is up to 3 years and can impair vision.

Peripheral Cataracts usually appear in a Pointer between the ages of 2 and three impairing their vision.

The Papillon experience inherited **Nuclear Cataracts** between the ages 2 to 8 with a mild impairment of vision. Other breeds may also experience this type of cataract in conjunction with old age.

As our Icelandic Sheepdog population grows it is important to track all canine hereditary disease but of most concern inherited cataracts, as it is the most devastating. The development of a test to determine which of our dogs may carry the hereditary cataract gene could be just over the horizon. This test could help Icelandic Sheepdog breeders make good breeding choices in not pairing two dogs who would both carry the same inherited cataract gene. The ISAA would like to begin down the road to develop that test but we need the Icelandic Sheepdog communities help. If you've received a cataract diagnosis please share it with Jo-Ann Secondino at jasecondino@aol.com. To begin the research for this test we need to know what types of Cataracts our breed is being diagnosed with, what is the age of onset, and if there is a family history. The Icelandic Sheepdog Community is like no other in the dog world, we are a tight cohesive group who work for the betterment of the breed rather than personal gain; together we can make a genetic test for cataracts a real possibility. Please join us in this journey.

The ISAA would like to thank

Dr Dennis Hacker, D.V.M., DIPLOMATE, A.C.V.O. (Veterinary Ophthalmologist) of El Cerrito, Ca. <http://www.animal-eye-specialists.com> 510-559-1755 for allowing us to use his photo of Canine PPM's for this bulletin.

Questions & Answers about Juvenile Cataracts

Juvenile or hereditary cataracts are an opacity or cloudiness of the lens of the eye that appear at a young age, usually as a puppy or young adult. They can be very small and not be noticed by general veterinarians, but still be significant genetically. Other types of cataracts that form in the dog are congenital (present at birth) and senile (common in dogs over 8 years of age due to the aging process). Sometimes a cataract can be caused by environmental factors such as trauma, radiation, toxins, or disease.

Below is a list of commonly asked questions.

Q. I have a brother of a known carrier. Can I breed him safely

A. Yes, as long as he has cleared a CERF exam. As a full-brother (or sister) of a known carrier there is a 50% chance that your dog is a carrier. However you can breed to a "clear" dog with statistics showing that no puppy in the litter will be affected but each puppy will have a 50% chance of being carrier. You have to make selective breeding decisions when choosing a mate for your dog.

Q. Do both parents of a carrier dog have to be carriers as well?

A. No. The carrier gene can be passed from either the sire or dam.

Q. Does age have anything to do with cataracts?

A. Like humans, it sure can. As mentioned above, they can be common in dogs over 8 years of age. If a dog develops a cataract after seven years of age, it may not be hereditary, but this is not set in stone. Age-related cataracts can often be differentiated from hereditary cataracts by ophthalmologists.

Q. My dog has passed her CERF test. Does that mean she doesn't have cataracts?

A. The CERF test results mean that she is not currently "affected" by cataracts but she may still be a "carrier" of the gene and may develop a cataract at a later date.

Q. How will I know if my dog is a "carrier"?

A. By careful evaluation of all close relatives of the dog. Though not foolproof, it is the best way of telling if there is a potential that your dog is a carrier. This is why it is so important that people share information openly. The ISAA is developing public eye records and encourages people to share all results of CERF testing with the club. We also encourage all breeders to get litters CERF tested as this is the best way for us to track the spread of cataracts in our population. Prior to choosing a mate for your dog, a careful examination of these records is imperative.

Q. Isn't there a simple test that can be performed on my dog to check for cataracts?

A. Not yet but hopefully soon. The area of genetic testing in dogs is so active that the prediction is that within a decade that there will be genetic tests for most of the genetic diseases in dogs. The ISAA Health & Genetics Committee is currently trying to set up a study.

Q. This seems so complicated. How can I be expected to learn all of this?

A. A simple chart based on statistical expectations is included in this newsletter. It hones the issues down to the bottom line. If you are a breeder of Icelandic Sheepdogs, we encourage you to take the time necessary to educate yourself so that you can actively participate in the protection of our healthy breed.

To reiterate a very important note to breeders--information must be used carefully, to make proper decisions for breeding in order to maintain genetic diversity in the breed. In the case of recessive disorders, if a carrier is bred to a clear, none of the puppies will be affected; however there is a 50% chance that individuals will be carriers. Likewise, there is a 50% chance that the puppies will be normal. We encourage each breeder and buyer to educate themselves and ask questions. The ISAA Board of Directors is here to help you with this process.

PARENT	Clear Male	Carrier Male	Affected Male
Clear Female	100% Clear	50/50 Carrier/Clear	100% Carrier
Carrier Female	50/50 Carrier/Clear	25/50/25 Clear/Carr/Affctd	50/50 Carrier/Affected
Affected Female	100% Carrier	50/50 Carrier/Affected	100% Affected

Ideal Breeding Pair: Puppies will be **Clear** of the disease gene (neither **Carrier** nor **Affected**).

Breeding is Safe: No **Affected** puppies will be produced. However, some of all puppies will be **Carriers**. Accordingly, it is recommended that **Carrier** dogs which are desirable for breeding be bred with **Clear** dogs in the future, which will produce 50% **Carrier** and 50% **Clear** animals, to further reduce the disease gene frequency. These offspring should be tested for this defective gene, and only the **Clear** animals in this generation should be used.

High Risk Breeding: Some puppies are likely to be **Carriers** and some puppies are likely to be **Affected**. Even though it is possible that there will be some **Clear** puppies when breeding "Carrier to Carrier", in general, neither this type of breeding pair nor "Carrier to Affected" are recommended for breeding.

Breeding Not Recommended: All puppies will be genetically and medically **Affected**.

Clear

This finding indicates that the gene is not present in your dog. Therefore, when used for breeding, a Clear dog will not pass on the disease gene.

Carrier

This finding indicates that one copy of the disease gene is present in your dog, but that it will not exhibit disease symptoms. Carriers will not have medical problems as a result. Dogs with Carrier status can be enjoyed without the fear of developing medical problems but will pass on the disease gene 50% of the time.

Affected

This finding indicates that two copies of the disease gene are present in the dog. Unfortunately, the dog will be medically affected by the disease. Appropriate treatment should be pursued by consulting a veterinarian.

data sourced from www.vetgen.com

What is PennHIP?

PennHIP stands for the University of **Pennsylvania Hip Improvement Program**.

PennHIP is a multifaceted radiographic technology (x-ray) for hip evaluation. The technique assesses the quality of the canine hip and quantitatively measures canine hip joint laxity. The PennHIP method of evaluation is more accurate than the current standard in its ability to predict the onset of osteoarthritis. Osteoarthritis, also known as degenerative joint disease (**DJD**), is the hallmark of canine hip dysplasia (**CHD**). Taken from the PennHIP website. To read more about PennHIP studies related to HD and DJD, go to www.vet.upenn.edu/researchcenters/pennhip.

PennHIP Information as it relates to the Icelandic Sheepdog :

As of February, 2007

Min = .25

Max = .83

Average = .54

.44 = 75th percentile

.47 = 60th percentile

.50 = 50th percentile

.65 = 25th percentile

Total Sample = 96

Special thanks Dr. Tram Le, Burian, WA Animal Hospital

Introducing the ISAA's Breeding Review and Compliance Committee (BRCC)

BRCC Tasks

1. Collect applications from breeders to list litters, both future and current.
2. Collect fee from breeders.
3. Maintain current data on breeding pairs and offspring.
4. Insure that breeders comply with ISAA breeding standards.
5. Other duties as assigned.

BRCC Goals*

1. Generate a list of genetic defects occurring in the Icelandic Sheepdog by surveying members and owners. This list will be made available to the public including the mode of inheritance when known.
2. Form and provide oversight for committees to assess the impact of each trait on the breed.
3. Advocate for the registration of dogs and bitches affected with genetic defects and those known to carry genes.
4. Advocate for the registration of dogs and bitches known to be free of genes for various undesirable traits.
5. Maintain Health and Genetics website to insure that information is available in that open registry.
6. Determine which defects should be attacked on a breed-wide basis.
7. Develop articles for the Health and Genetics website that specifically address diseases that occur in the ISD, clinical signs, methods of diagnosis, age, onset, mode of inheritance, potential treatments and prognosis.
8. Develop a page on the H&G website that discusses the rationale of the various systems that can be used to control disease and how to handle carriers and potential carriers of the various traits.
9. Provide strong support for those breeders and owners with the honesty, courage and foresight to openly register dogs affected with genetic disease, because there is no hope for control without knowledge. Clearly state that the best course is to openly discuss dogs with defects or those that produce defects when selling a show dog, breeding prospect, pet dog or stud service.

As of March 15, 2008, the ISAA is working on goal numbers 1, 2-5, 7 & 9. If you would like to assist the BRCC to achieve these goals, please contact the BRCC Chairperson at eyjahunda@gmail.com.

*Adopted from: George A. Padgett, DVM Control of Canine Genetic Diseases, pgs. 8 & 9 The BRCC was formed by vote to the ISAA's Board of Directors on March 14, 2008.

ICELANDIC SHEEPDOG ASSOCIATION
OF AMERICA

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ISAA Website:
<http://Iceland-Dogs.com>

Opinions expressed herein are those
of individual authors and not
necessarily those of the ISAA.

Want to learn more about these issues? Contact Donna McDermott to join a study group that will use George Padgett's book, Control of Canine Genetic Diseases to study how we can breed better dogs. The group will begin April 15, 2008. Anyone can join, breeder or not, by contacting Donna at eyjahunda@gmail.com. Members will need to purchase the book to participate.

The ISAA applauds our volunteer CHIC (Canine Health Information Center) Breeders. They have gone above and beyond by satisfying each breed specific requirement of CHIC and have agreed to the release of the results into the public domain. Bravo!

ISAA CHIC Breeders as of March 15, 2008

Knox Rhine of Nor'Star Icelandics
Shellie Greyhavens of Havens Icelandics
Jo-Ann Secondino of Fox Meadow Icelandics
Donna McDermott of Eyja Hunda Icelandics
Laurie Ball-Gisch of Lavandel Icelandics
Judi Vittetoe of Vittetoe Icelandics
Margaret Gimblin of Vallhalla Icelandics
If you would like to join the ISAA's CHIC Program, contact Jo-Ann Secondino jasecondino@yahoo.com, our CHIC Program Representative. You can find out more about the CHIC program by visiting www.is-pedigrees.com.

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