English version Nov 2011 by conference secretary Mia Sandgren

A summary of the minutes taken at the The Swedish Vallhund World Congress 3-4 june 2011

65 participants, seven lecturers, Jahn Stääv (moderator), Mia Sandgren (conference Secretary), Gunnel Sandquist and Birgit Svensson (technology) and Anita Whitmarsh (hostess). Overseas guests are from Australia, Denmark, England, Finland, France, Netherlands, Norway, Switzerland and USA.

Chairman of the Svenska Västgötaspetsklubben, Mr Jan Helgesson, wished everyone welcome. He explained the importance in working together internationally and hoped this conference from now on will be hosted in different countries, and suggested Finland would take over the hosting the next time. Chairman of the Community Council of Vårgårda, Mr Bengt Hilmersson, wishes everyone welcome to the community were this breed once started. The moderator, Mr Jahn Stääv, greeted special welcome to the club's overseas guests and suggested that they should introduce themself and the breed's status in their country.

Presentations
Australia has a small number of individuals in the breed. In some of our States people get together every once in a while and have informal meetings with judges.

Finland
Eight guest including the chairman, secretary and editor of the breed club and magazine. Finland has circa 400 members and the registration numbers in the breed are around 100 puppies per year.

Denmark
Denmark has only two breeders of the Swedish Vallhund. They have about 250 dogs totally in the country.

France
France has a small population and few breeders, who in between them register about three litters a year.

USA. Look forward to bring much information back home to the people in the breed. In the USA they have around 1000 dogs in the breed.

Schwitzerland. The club is young, they have four active breeders and there is approx. 120 Swedish Vallhunds in the country.

England has several imports from Sweden. They are not many breeders.

Sweden. Anita Whitmarsh completed the presentations by talking about the breed's status in Sweden. The breed club started 1976. 1986 they received an official status and change name to *Specialklubben för Västgötaspets* (The Specialty Club of the Swedish Vallhund). The club currently has nearly 800 members and registers about 200 dogs a year. The breed is numerically counted as a minor breed. The Club is organizing meetings within regions, but as

Sweden size-wise is a long country and the larger population of its people lives in the mid-region, this is where most of the gatherings are organized. The Club is working closely internationally, and welcomes more clubs and single members to join in.
The Swedish Vallhund Club is arranging two to three shows per year, one of them being the larger one, as this year at Tånga Hed this weekend.
The Swedish club encourages all members to activate their dogs in herding, although there is some difficulties to get them to train on cows. Owners are active with agility, obedience and scenting.

STANDARDS - THE STRUCTURE of FCI and, What's on in the DOG SHOWRING
(FCI - Federation Cynologique International/The International Kennel Club Organisation in Europe)

Dr GÖRAN BODEGÅRD
***Dr Göran Bodegård**, is a member of board at The Swedish Kennel Club SKKs. He is giving a lecture talk on the structure of the FCIs organization to which the Swedish Kennel Club is one of 85 member countries. FCIs members has together 341 official breeds and breed varieties. Each country has its responsibility for its breed's standard as the native country, but as The Governing body, FCI has primary responsibility for any changes to be made in a breed standard within FCIs member countries.*

Dog show judges have theoretically a greater responsibility today than they use to have. FCI sets the rules and recommendations which FCI-judges have to follow. **FCIs** work is in many ways a hard work. The judges-education in between member-countries differs. Quite a lot in some. In some countries an all-round-judge are only allowed to judge a small number of all the FCIs registered breeds for a period of 10-years before he or she are accepted to judge a few more.
GB gives a few questions to consider – What about the differences in competence among judges, and how active/considerate are breed clubs in their choice of a judge to judge the own club shows? He explains that FCI has recently begun to follow the Scandinavian Kennel Clubs educational system with producing compendium of a breed standard, arranging breed conferences where discussions about standards are held.

Special Breed Specific Instructions - BSI

Breed experts is in the breed club. Today, 46 breeds are in a risk zone. The Swedish Vallhund is **not** among them. Extreme typical qualities changes due to soundness. A difficult conflict occurs between soundness and judging of type (*a power-point-picture is shown with a giraffe with a long neck. For instance, if SKK should say "a long neck is unsound"… and they change the standard, and a giraffe with shorter neck is bred for… will be it a giraffe?... That is the question*.)

Could this give a result of seeking of faults instead of looking for an overall type? Today they are training judges in positive thinking. Is the grading of today under FCI rules correct? FCI is the Head Body for any change to be made within FCI breed standards. The country of its origin applies for any changes to be made. *(SKK/SK (Standard Kommitté) is contacting the breed club. The Breed Club should turn to SKK if they want a change to be made.)*

What will happen to a breed when one part decides not to follow the descriptions of details in a breed? Has the Swedish Vallhund changed from what it looked some 60 years ago? How big influence will it have on the exterior if the breeding are turning toward different ideals?

For example; Is a taller legged dog sounder than a short legged? *(…shortleged and lowlegged are a distinct difference. GB –Conclusion; "shortlegged is nothing to look for". But are those more up on leg sounder? I don't know, but the shortlegged are high in rear and most of the times also have curved front legs*).
How about color markings? And the description of masks? Will what is called "faults" follow with a another way of breeding? It is important to communicate within and outside the breed club? If the language barrier gives wrongly interpreted words in a breed standard – what will the clear consequence of this be in other countries?
Dr Göran Bodegård finishes his lecture saying - It is important to communicate and to have these kinds of meetings.

HD within VÄSTGÖTASPETS – by MATHS LINDBERG

Maths Lindberg, X-ray technologist and radiology specialist. CEO in N*ordisk Röntgendiagnostik in Göteborg. He's a teacher at Swedish Board of Agriculture (district vets). ML has followed Swedish Vallhund with a big interest for many years. This is his second lecture for the Club. Lecture is held as a dialogue with the audience.*

Quotation: It was suggested at the meeting that the club should send a proposal to the Swedish Kennel Club forwarding to the Nordic Kennel Union, and their panel, which is investigating in HD within FCI. If this is a problem in some breeds and specific instruction for a single breed are in hand, it might be a chance for a rule to be changed for a certain breed. All condrostryfokial breeds should be treated the same – if given wrong HD-results. If many breed clubs will send an application with an explanation of the problem, this can make a change.

A recommendation has to come from the country of origin of a breed to have a greater impact.

The Swedish Kennel Club has, after they had read the original text regarding HD in the Västgötaspets, given by Mr Maths Lindberg, made a notification in some parts of the text. The Swedish Vallhund Club has therefore decided to exclude this lecture. Mr Lindberg has not responded to the Club.

OBS Following text is from The Swedish Kennel Club----------
The Swedish Kennel Club: Many of the questions described about hip dysplasia are unfortunately either fault, incomplete or unclear. To change them and to give a complete new information and correction in many of the things said in this text requires a lot of time. Since Much information on HD already are published, either on the Swedish Kennel Club web site or in articles of different magazine (i.e. Hundsport Special), we will refer to these sources instead.

Mating and whelping by Vet EVA VON CELSING

Vet Eva von Celsing has her own vet clinic suited at her and her husband farm. She is breeding Dachshound under the prefix Hvarsta.

I would like to thank the organization for inviting me to this congress. My theme is "Mating and whelping". My guess is that most of you have great experience in this field. I have therefore chosen to concentrate my speech with some common issues that we as breeders ask ourselves after a period of breeding.

MATING

Why do bitches not get pregnant?

The two most common reasons that bitches don't get pregnant are
- Bitch mated at the wrong time during the season
- Males have a poor quality of semen.

Both things are easy to control. In the first case with a blood sample and a vaginal smear. In the second case it is easy to take a semen sample.

Many breeders think that the reason may be an infection in the bitch but this is not likely.

How many bitches will be pregnant after mating? We do not know.
422 mating of Guide Dogs (Golden, Labrador retriever and German shepherd) resulted in that 85% had puppies.
A U.S. study of 2507 mating with Beagle in the pharmaceutical industry resulted in 90,2 % having puppies.

Both these institutions breeding programs are very professional. Among us breeders with different skills, maybe 75-80%of our bitches will be pregnant after mating- my own guess.

When will the bitch be mated?

The first days of the bitches season the bitch won't show any interest in being mated. After 5-6 days there are bitches that show signs that they will be mated even if it is far from ovulation. Sperm can survive in the uterus for 4-6 days and still be fertile so some bitches might be pregnant even if they are mated too early.

At the beginning of the heat the progesterone level is about one. In connection to ovulation the level of progesterone rises. When the progesterone is 15 the ovulation will start and 48 hours later the eggs will be able to be fertilized. Two mating with one day's rest gives a higher percentage of bitches in whelp and larger litters. The best time to mate is when progesterone level is between 30 to 75.

A vaginal smear can also provide some assistance to see if the bitch is in estrus but the cell image can look great for many days and is thus more difficult to interpret in order to give a precise answer to the breeder.

When in oestrus?

Day 1 in oestrus is when the bitch allows mating. Oestrus can vary from 4-20 days.

Many people consider that bitches should be mated between days 10-14 in the heat. As a breeder it can help being humble and realize that there are big differences. Some bitches allow mating both when it is too early and some when it is too late. A blood test provides information about ovulation and a vaginal smear provides information when it is too late.

There are certainly preferences for particular partners in the dog world but the most common reason for females to reject males or why males do not want to mate the females is that the bitch is presented to the dog the wrong day/days. Most often they meet too early.

Can a male dog mate daily for a week?

Yes, from the point of view of semen quality, a fully grown male can mate daily for two weeks without any problem. It will not lower the concentration of sperm when they ejaculate.

Where exactly will the fertilization take place?

The newborn female puppy has about 700,000 eggs in the ovaries. Many have degenerated before the bitch is ready to be mated, but it is amazing what an overcapacity nature has enriched mammals with. Ovulation will only take place during the heat.

In the grown up males, it takes 62 days for an immature sperm cell to develop into a mature sperm. An ejaculation should contain at least 200 million sperms.

After mating the sperm reaches the oviduct after 7 hours. Sperm can remain viable in the uterus of the bitch for 4-7 days. Live sperm have been detected as late as 11 days after mating, but whether they are capable of fertilizing, this is not known.

There will be a cleavage of cells in the oviduct. When all fertilized eggs have reached/developed a 32-64-cells morula they will be released to the uterus. Even if the bitch has been mated during several days all the morulas are at the same developed stage.

Is a bleeding 17-18 days after mating a warning sign?

Barely three weeks after mating an implantation will take place in the uterine wall. A fresh bleeding may occur which the observant breeder might notice as a blood discharge. This is normal and will stop after 1-2 days.

Is a vaginal culture of any value?

A bacteriological sample from the vagina may be useful if the bitch has abnormal vaginal discharge - however, almost only seen in connection with uterine infections. Bacteria that causes fetal deaths, which is found in the uterine inflammation, are often also found in the normal vaginal flora. Thus, it is of doubtful value doing routine bacterial culture.

There is no reason to recommend vaginal culture as a routine

Many believe that antibiotics may be wise to order before mating, but this is NOT accurate and can actually cause problems. First, we increase the risk of resistance development that affects both animals and humans. Then we change the individual's normal bacterial flora in the mouth, skin, gastrointestinal tract, uterus etc.

So if the bitch did not have puppies at the last mating, it is probably NOT a bacterial infection causing this to occur. More likely it is due to mating her on the wrong day or poor quality of semen of the dog.

Bacteria causing diseases can be given an opportunity to grow when an incorrect cure of antibiotics has been ordered!

Should the bitch be vaccinated against the herpes virus if I have problems with dead puppies /empty bitches?
There is a vaccine against herpes virus. The bitch will be vaccinated 1 week before mating and 10 days before whelping. The feotuses are not protected against herpes during pregnancy but will be well protected when the puppies have suckled colostrum. Herpes virus thrives in 35 degrees - it is important that newborn puppies are kept warm.

Palpation, ultrasound, X-ray - how can I check if my bitch is pregnant?
With careful palpation you can palpate a pregnant uterus around 21 days. Ultrasound can be done after about three weeks but it may be wise to wait for more than 30 days. The diagnosis becomes safer pregnant or not pregnant. Radiology is not healthy for growing individuals, but if the owner wants to know how many puppies the bitch carries a radiograph can be taken the last week of gestation.

Can I de-worm the pregnant bitch?
Yes, with the Axilur-program the puppies can be born without having any worm infection. Higher conception rates and a larger number of puppies in litters have been seen in kennel with problems after the Axilur-program have been introduced.

From the 40th day of gestation the bitch will be given Axilur daily until the puppies are 14 days of age. The puppies have to be dewormed at 6 and 8 weeks of age.

WHELPING

Can calcium supplements in the food give better contractions and reduce the risk of eclampsia?
Breeders of today almost always offer a quality food in their kennel. There are all trace elements and vitamins needed. An excess of Ca may actually increase the risk of eclampsia instead of preventing it. When the bitch produces milk calcium will be released from the bones into the blood via a hormone (parathyroid hormone). If the bitch at the end of gestation is given an excess of calcium, parathyroid hormone will be inactivated. When calcium is suddenly needed for the increasing milk production there will not be enough parathyroid hormone. Thereby the risk of developing eclampsia will increase.

Cesarean section = no more breeding?
If the bitch needs a caesarean – does it mean that she must be removed from breeding? No,

there is no need to state that categorical. There are so many examples of bitches that after a cesarean, on her next pregnancy, have been able to give birth normally. However, if she needs another caesarian, I don´t think she should be bred from again.

What starts whelping?
To put it simple, you can say that some hormones give signals to start the whelping. Cortisone, which is secreted by both the puppies and their mother's adrenal glands start labor (birth). Postaglandin is released thereafter. When the temperature of the bitch drops, she will be restless, cervix begins to soften and open up. The progesterone from the ovaries decreases drastically one day before whelping.

If the bitch is just carrying one puppy or if all the puppies are dead it might be a reason why a normal delivery would not get started.

Signs of whelping complications?

1. More than 24 hours have passed since the temperature dropped and no puppy has been delivered.

2. Severe, continuous contraction for 30 minutes without any puppy been delivered.

3. Normal contractions for 2 hours without any puppy been delivered.

4. Normal contractions will decrease/stop.

5. More than six hours since the last puppy was delivered and you know that the whelping is not finished.

6. Green discharge before the first puppy has been delivered.

What can I do for helping the bitch?
Go for a run with the bitch (2-3 minutes).
Stimulate the push-reflex with one or two well-washed fingers in vulva roof.
If the bitch has started the whelping and one or more puppies have been delivered the bitch can be given oxytocin spray in the nose (Syntocinon).
Power-kick (honey and egg yolk).

<u>After the lecture some given answers:</u>
It is recommended not to use males before a year old, with reservation of some breeds. In, for example golden retrievers it is OK for breeding at a young age, but in Dachshunds it is recommended an older age.

Females that are going through, what is called, ”dry season”, could be they are at their first season are showing signs of being in season, but are not, and after a short while will have a correct season. Others can early in season allow to be bred to, even if they don’t have an ovulation.

Is there a need to breed a female twice at same season? Yes, we know the result in number of puppies are higher when doing so.

Check the male testicles for any changes. If there is a change of size and consistency could give a result of lower quality of semen or even lack of production.

Cryptorcism is not a problem in this breed.

Generally a male can mate once a day for two weeks without any problem in sperm-quality. But hopefully should a Swedish Vallhound male not be used that much.

BREEDING SHORT-TAIL TO SHORT-TAIL, by KIRSI SAINIO, Finland

Kirsi Saino, Finland, scientist and a teacher in genetics and development as a biologist, also genital defects in newborn in humans (and in dogs).

She got her first dog, a sky terrier, in 1983. In 1993 she was authorized as an international FCI dog show judge. Through two very special friends she has followed this breed very closely in Finland.

"All knowledge the totality of all questions and all answers is contained in the dog" Franz Kafta.

Short tail phenotype has been forever. These breeds are probably not ancestral. This has developed at some time in history and has thereafter spread to different breeds. (*Ref. Magazine Mammalian Genome*) .

Canine omolog of the T-box transcription factor, t-failure of the protein to bind to its DNA target leads to a short-tail phenotype.

Mice showed early this mutation and this made scientists to seek if this gene could be the same in dogs. Mammals has similar spinal structure, a giraffe also have 11 vertebrae, 7 cervical, 13 thoraic, 7 lumbar, 3 sacra and the tail with a variation of vertebrae.

The shape of a the vertebrae, a structure called mesoderm, are in different parts. One part of it forms a paraxial mesoderm. Paraxial M gives rise to vertebrae and bone structures of the truk. Vertebrae form from structures called Somite. Every Somite follows exactly the same form and stays side by side exactly the same on both side of the spine. It is one of the most studied structures.

What is a T-box? A T-box is named out of a DNA structure (all this is showed in power point pictures).

What is the transcription factor? In the T-box family three of the factors are Eomes, T and Tbx6. The T-box is causing the short-tail factor. To understand the transcription factors we have to understand DNA. DNA is short for Deoxyribo Nucleic Acid. It contains paired bases and sugar-phosphate backbone. There is 5.5 billion basepairs in dogs. A dog has in its total genome approx. 2.5 billion base pairs. Also approx. 14-19 thousand genes. DNA has four different bases bound to each other with Hydrogen-bonds - H-bond - and the Sugar phosphate

backbone. The four bases are forming specific pairs, Adenine with Thymidine, Cytosine with Guanine. Kirsi Saino says: No one still not understand the total work in this structure.

For Somite formation and for Formation fo tail structure. Transcription factors contains regulatory elements and Coding regions, inunder are Enhancer and Promoter together with Introni and Eksoni.

Mendelilan are an inheritage of the short-tail Phenotype.

T-dominant and T-recessiv

TT	Tt	tt
25%	50%	25%
TT = without	Tt = short-tail	tt = letal mutation of short-tail

Newborn with tt will never survive, they will die before they are born or soon thereafter.

The Homocygote mutated seems OK, but when looked closely at the puppy the vertebrae will look developed, but its hind legs will not look well and it has no anus and will soon die.

There are 17 breeds with the C189G mutation. As soon as one pair has changed its DNA, it changes its total chains. They are Short-tail Fenotype but are not breeds with short-tail, not the same mutation. In breeds like Boston terrier, English Bull Dog, French Bulldog, King Charles Spaniel, Miniature Schnauzer, Parson Russell Terrier and Rottweiler it looks recessive.

What needs a breeder to know about the T-box? It affects the development of vertebrae and tail. Short-tail phenotype in dogs have this mutation is caused by a single dominant allele. T-box mutation allele causes also Kinky Tails (= kinky tails are a part of the mutation, it cannot be avoided and should be allowed in short-tails). As a Homozygote, T-box mutation is lethal.

Two short-tails are not allowed to breed in Finland. The same goes with two merles and two harlekin. Any breeding between two mutations are not allowed - despite the length of the tail as they from the beginning always is a mutation. The only way to see if it is a mutation is to have a gen-test made. In countries where it is allowed two breed two mutations, breeding short-tail to short-tail to have short-tail should not be made. It will give smaller litters and will lose the homozygote line that long-tails have.

From the beginning all mammals developed a tail. Since we don’t really know what makes a short-tail, we don’t yet know the amount of genes behind the development of the tail. In Genetics the answers are very seldom either black or white. Meaning, the answer will always refer to there is always Modifiers that promote for or against effects or defects.

Years ago when losing a puppy it was called a “fading puppy”-syndrome. It was not always so. These puppies describe here you can see they look different and they do not have a developed opening of anus.

COLOURS

Genetist Clarence Little found out the color genes in 1957. Proteins are coded by the color genes. It contains two pigment – EU-melanin and Fe-melanin.

A (agouti)

B /Black/Brown)

E (extension) =

Recessive allele e = cannot be black or brown.

D (dilution) =

dd= diluted EU-melanin into BLUE (so called blue-dog-gene (not Kerry Blue terrier)

M (merle) = SILV-gene mutation

K (dominant black) - beta-defensin, dominant allele KB give a uniform black, brown or grey color (only production of EU-melanin) recessive alleles k-brindle and k-yellow make it possible for the recessive agouti alleles to be expressed and visible in the phenotype.

(KS show photos of Finnish Lapponian Herder with different genecombinations and gives a description that I was not able to write down.)

Dr Mendelians, Inheritance of Colors, is only seen when you have enough of puppies in a litter (i.e. hundreds). Breeders of Labrador retriever say they do not have theese colours. KS say they do. A brown labrador puppy has the recessive brown allele. One puppy is brown, one puppy is black and the fourth puppy has the double mutation of recessive allele (bbddEE) grå.

Why can the color cause problems?

White is not a color it is lack of pigmentation. Melanocytes... Sternum gets the Melanocytes last, second last is the feet and when there is no more Melanocytes it will show white = i.e. no color. White patches on a dog has nothing to do with specific genes. White patches are lack of color and will therefor show white hairs.

When can it cause problems?

When development of the inner ear, there is a region named Stellata Vascularis, which produces liquid for the inner ear and make the inner ears cells moving and hearing will develop. If Melanocytes are missing it will not develop the Stellata Vasculatis and the dog will have no hearing, i.e. death. If Melanocytes are missing this will affect other areas too in the dog.

Piebaldism. In dogs the white color, or lack of pigmentation is recessive. White spotting-alleles sp and sw. Pibaldism comes from two words: "magpie" and "bald eagle".

S-genes (white spotting)

S

si = irish spotting i Bernese Mountain Dog and Basenji

sp

sw

The amount of white color also depends on other genes, so called modifiers, such as the Merle-gene.

Irish spotting gene was found three years ago (2008), in white bullterriers and boxers. An important developmental gene with a complex regulation implicated in pigmentary and audiotory.

EYES by BERIT WALLIN HÅKANSSON and ANDRAS M KOMAROMY, USA

Berit Wallin Håkansson, European Veterinarian Specialist in Ophthalmology

Working at the Regional Animal Hospital Strömsholm suited in a the small town Strömsholm, in County Västmanland in mid-Sweden. The Animal Hospital Strömsholm has 140 employees, with 40 veterinarians. They have 26.000 cases a year. Strömsholm has a section for Ophtalmology with two specialists educated in the United States. Berit WH is to retire from Strömsholm, but continuing her work as a consult at the Swedish Kennel Club and also within the Board the Swedish Eye Panel.

The following results are from the Swedish Kennel Club Eye data from registrations in between 1991-2010 (year of birth). 128 cases are u.a. (without notification) and 43 cases are diagnosed (more or less important). When pulling out this information you have to take in concideration that these numbers are bases on year of birth.

Retinopathy 2000-2010 (year of birth) 7 cases of cataract are reported.

The Retina. Grey arrows Retina. Under retina is the Choroid. Color comes from the Choroid. The Retina itself looks like "wet toilet paper". Tapetum (colored), non-Tapetum and Tapetal area (quick description of the eye.)

Following are all eye-forms with any comment on. Bullous Gray Hyper-hyporeflective /14 cases.

Changes in the Alteral Tapetum/roundish changes in a cluster in the latter part of the Tapetum/has been seen in field studies mostly in Great Pyrenees Dog/usually mirror images, looks exactly the same on both sides in both eyes.

Yellow, brown, pigmented dark/ 7cases.

PRA-like/3 cases.

Looks exactly like ordinary stage PRA. The differences to be seen are the many arteries. Right eye only shows a smaller amount, quite like a sunset, there is no wild discovery. More on this later (see below).

Watery silk/2 cases. It is made by pressing wet silk by rulers/some parts are dens and dark, others are white and shiny. There is various changes in the grey tone (on the photo).

Retinal fold/2 cases.

Chorio Retinitis/ 4 cases. This is an inflammatory response to the Retina and the Choroid.

Totally 32 cases with commentary at eye examination.

Examined = 170 dogs, 32 with Retinal Designs = 19 percentage, some severe others less significant. 14 percentage show major Retinal signs, i.e. one dog out of seven may have changes in their eye.

Future?

Are these signs serious? Many are followed over time, done, anatomical background, autopsy, breeding restrictions, are dogs related, breeding restrictions. When DNA testing - only for inherited disease – with known inheritance. Test must be relevant and validated. If your dog has changes in eye, please give the dogs eye to science after its dead.

ANDRAS M KOMAROMY *Assisting Professor in Ophtamology - Tenure Track, Med Vet (Veterinary Medicine), University of Zurich, Switzerland, 1993. Dr Med Vet (Veterinary Medicine) University of Zurich, Switzerland, 1996. PhD (Comparative Ophthalmology) University of Florida,2002. AK works at University of Pennsylvania, Philadelphia, komaromy@vet.upenn.edu. A Komaromy studies how various diseases in dogs are related to human diseases. All science in dogs could be fortunate for human with similar diseases. All research is dogs can become an advantage for people with similar diseases. AK works closely with Professor Hannes Lohi and Saija Ahonen at University of Helsinki, Finland, and veterinarian Päivi Vanhapelto from Vetset, Kyrkslätt, Finland. (saija.ahonen@helsinki.fi - www.koirangeenit.fi), (Retinopati-research)*

Retinopati in Swedish Vallhund

This work started 5-6 years ago when he had a phone call from a breeder in USA.

The eye exam could be done in two different ways, ophtalmoskop and slit-lamp.

Every other year the publication “Ocular Disorders” are presented. This publication can be ordered over internet. A number of Swedish Vallhund are among the other breeds. Out of 288 dogs, that is 93 percentage of the whole SW-population in the United States, 64 percentage are without any remarks on their eye exam. Most common eye-defect in this breed is Cataract.

Saija Ahonen, at University of Helsinki, Finland, explains: In Finland we have put together a pedigree with little less than 1000 dogs (Sw.Vallhund). 87 are affected with Retinopati. Out the 1000 dogs they have selected 414 blood tests. In this pedigree it is several affected dogs, but with non-affected parents (but probably carriers). This sustain this disease is autosomal recessive. It is not more affected males than females, therefor Retinopati could be X-chromosomal.

In Finland dogs are having the first eye-exam at two years of age, then every second year up to 6 years of age and then the last time at 8 years of age. If the is affected late, it will be a problem for the breeder. The Sweden this breed do not have requirements in eye-examination, but some owners are having their dog checked.

A genome include all genes. It is like a fingerprint. You have to start with comparing normal print with affected to see the different in what is normal and what is a defect gene.

What causes this disease? Is it an acquired or environmental factor? Inherited or a single gene, complex or multiple genes? Ethological causation is most often environmental related. The overall part of gen-test is too monogenic, but multiple genes are involved and they affect each other (more variable clinical symptoms, more variable age of onset, more variable rate of progression - etiology of disease).

Retinopati

Stage 1.

Shows no sign of Retinal degeneration. Could be probably be uses for breeding, but only with healthy individual. Based on experience, if a dog is diagnosed with Stage 1, it is up to the breeder if he or she will use the dog, but it is recommended to be sure to use a healthy dog. But, if Stage 2 or 3 has occurred, this individual should be used for breeding. What was seen 4-5 years ago are still true. To begin with redbrown dots are seen and grey “mottling” – multifocul discoloration in the tapetal fundus. But no clinical signs of vision loss. That will occur in 2-3 years of age, but the youngest dog was only 7 months old and the oldest 17 years old. Symptom can stop in its first stage without going further to stage 2 or stage 3.

Stage 2. One or two years later larger spots will occur and can then be diagnosed as Retinal. In the beginning this will not cause lack of vision for the dog.

Stage 3.

At this stage the area with dots have increased, they differes also from earlier experiences. The process has now gone from progressive Focal Retina to General Retinal Degeneration. Dogs lack of vision in daylight has now increased in daylight, and no night-vision is left.

Strategy.

Continue with eye-examinations, tissue analysis and blood-tests for DNA-analyses. The search for the gene will continue. Continue to do the eye-exams and to have blood-tests taken. Also what will help science is to receive eyes form affected dogs after they have died.

Examinations with electro-retinografi can be done at laboratory. The dogs will be anaesthetized (so that the dog will stay completely still during the examination) and a special contact-lens are put in over the eye. Dr Komaromy has made a portable kit for him to easily do this at any vet clinic when required.

With the three stages (above) has been possible to discover thanks to donations from owners when their dog has died.

Immunohistochemistry. Technology is so advanced today and with this special technic we can discover so much more in the dog-eye as ever has been seen before and without having to taking the eye out.

No signs of possible vitamin E-related has come up.

Recommendations are to begin testing puppies before they leave their breeder. Continue with eye-examinations up until the dog is 8 years old. If nothing has occurred by this age, it is unlikely it will develop Retinopati.

They do not yet know how big percentage it is that has developed from stage 1 to stage 2 and 3. In the USA AK has only seen one dog with a developed stage 3. Most dogs stops at stage 1. It is important for this breeds gen-pool to continue to breed your dogs before any clearance of this disease, and no panic to occur.

A proposal was put forward that clubs all over the world to start their own help programs with matching funds to support this project. Economically figures come close to 100.000 US-dollar a year to keep this as a science project. A big help would also be to create some guidelines for science/research. It is desirable to have clubs all over the world to cooperate and to send information to each other. What we know today is thanks to all help from Finland and United States.

Three important documents that have to be included with the DNA-test is a blood-sample, the pedigree of the dog and a copy of his/hers eye-examinations. All this should be sent over to Finland to be registered and the result will be forwarded to Andras Komaromy. (See more information below from Saija Ahonen. For mail-contact saija.ahonen@helsinki.fi)

How to participate in the genetic research

(by Saija Ahonen, Finland)
The owner has to fill in a form for the blood test of a dog. You can get this form at website http://www.koirangeenit.fi/osallistuminen/lomakkeet/ or order it from lgl-kyselyt@helsinki.fi

1. Carefully fill in the blood-test-form and send it together with the blood-test.
2. If the dog has been eye-examined send also a copy of this with the test.
3. Ask your vet to get you 3-5 ml of blood in an EDTA-tube.
4. Write your dogs name and registration number on the tube. Check that all information is the same in all the papers you are sending.
5. Mix the tubes 8-10 hours so that the EDTA are mixed in the blood.
6. Send the blood-test, blood-test-form and the eye-examination to the address that stands in the blood-test-form. Keep the blood-test in the refrigerator just until you will go to the post-office. If the blood-test has been taken on a Friday, keep them in the refrigerator over the weekend and send it by mail on the following Monday.
   All given information will be strictly confidential. The dog and owners data are archived in safely in a database only for research purposes.

Vet Berit Håkansson-Wallin concludes: *"The hard part in this is that many do not know about this disease. I believe we should continue to do eye-examinations to be able to see and follow the different stages. As soon as the DNA-gene is found we know how to go further. Both eyes are often likely affected. Andras Komaromy is the predecessor in this research, everyone else is following in the subject. It is important to understand that the pictures you see in stage 2 and stage 3 are serious eye-diseases and that it leads to blindness. It is easy for a dog to foul his owner. I have seen dogs which have barely any eye (which I barely see), but the owner still says that they know that the dog can see. The eye is the third mind in a dog, as long as ear and nose work. One can test them in a totally unfamiliar area, to get an idea of how much vision the dog really has. We can see changes in 14-15 percent of those we examine. I have seen dogs with serious problems that should be examined. But I have also seen several that are not so seriously affected yet. There is no problem for me to inform my colleagues about this problem. Andras Komaromy can inform researchers. But what do you breeders want? "*

AGRIA Breeder Programme Profile

IB AHLEN. *Product and businessanalysts, all stock, at Agria Insurances, in Sweden, Norway and Denmark. His family includes one Labrador retriever and three cats.*

Agria Insurance started with insurances for pet animals in 1924. 1972 cats were included and 1977 parrots were included. Sweden has approx.730 000 dogs, 1.2 cats, 365 000 horses and

400 000 pet birds. Agria Insurances has 50 percent of the market of all insured dogs, 40 percent of all stock in Sweden.

The profile program has produced to meet the information to anyone interested in animals. When SKK started the Breed-project Agria understood the need of information the Swedish breed clubs were calling for. The program shows, in diagrams, the status of all recorded diagnoses. The database is primarily for insurance policies in various fields. A researcher must evaluate to see to the information is helpful before it is published.

The Profile program gives statistics in all breeds in one unit, and on individual breeds in another. It may be a disease problem, or the most common problems in 80 of the most common breeds. It can be a bit harder to get a larger picture of minority breeds who are not seeking compensation.

What are the most common diagnoses and what differences exist between breeds? Highest mortality in breeds in Agria statistics is the number of dogs hit by cars.

Conclusions of the Swedish Vallhund? – It is a healthy breed!

Explanations

Retinopathy *is a vascular change in the retina such as bleeding or edema, which occur in diabetes or hypertension.*

A ***recessive*** *gene is a concept in genetics, which means that for a capacity is that an individual had genes from both parents for the capacity to be proven. The opposite is a* ***dominant*** *gene, which is also shown only inherited from one parent. A recessive gene is hidden by a dominant gene.*

Etiology *is the study of causal connection.*

Histopathology. ***Tissue*** *is a collection of cells in an organism with similar tasks. The study of tissues is called* ***histology*** *or, if there is disease, histopathology.*

Immunohistochemistry (IHC *shortened after English Immunohistochemistry) is that it locates proteins using antibodies that bind to specific antigens.*