

Swedish Vallhund - Memento notes from the World Congress 2011 June 3-4.

These notes are a brief summary of the different lectures held during the congress and it may sometimes be difficult to find the right connection.

Contact us in the Breed Committee if you have any questions. We are happy to explain if you find something unclear!

Engelsk version/October 2011/Revised part of HD by Math's Lindberg/Notes from 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 of working together internationally and hoped this conference from now on will be hosted in different countries, and suggested Finland would take over as host the next time. The chairman of the Community Council of Vårgårda, Mr. Bengt Hilmersson, wishes everyone welcome to the community where this breed once started. The moderator, Mr. Jahn Stääv, greeted the club's overseas guests especially welcome and suggested that they should introduce themselves and the breed's status in their country.

Presentations

Australia

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

Finland

Eight guests including the chairman, secretary and editor of the breed club and magazine. Finland has approx. 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 in total in the country.

France

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

USA

The American guest looks forward to bringing a lot of information back home to the people who are involved in the breed. In the USA they have around 1000 dogs in the breed.

Switzerland

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

England

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

Ms. 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 changed name to *Specialklubben för Västgötaspets* (The Breed Club of the Swedish Vallhund). The club currently has nearly 800 members and registers about 200 dogs a year. The breed is in numbers 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 the clubs members lives in the mid-region, this is where most of the gatherings are organized. The Club is working 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 are some difficulties to get hold of cows for them to train on. Some owners are active with agility, obedience and scenting.

STANDARDS - THE STRUCTURE of FCI and, What's on in the DOG SHOWRING

(*FCI - Federation Cynologique Internationale/The International Kennel Club Organization in Europe*)

Dr GÖRAN BODEGÅRD

Dr Göran Bodegård is a member of board at The Swedish Kennel Club SKKs. He is giving a lecture on the structure of the FCI's organization in which the Swedish Kennel Club is one of 85 member countries. FCI's members have 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 FCI's member countries.

Dog show judges have theoretically a greater responsibility today than they used to have. FCI sets the rules and recommendations which FCI-judges have to follow. FCI's work is in many ways hard work. The judge's education differs between member-countries, quite a lot for some. In some countries an all-round judge is only allowed to judge a small number of all the FCI's registered breeds for a period of 10 years before he or she is accepted to judge a few more.

GB poses a few questions to consider – What about the difference in competence between the 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 Club's educational system with producing compendium of a breed standard and arranging breed conferences where discussions about standards are held.

Breed Specific Instructions - BSI

Breed experts is in the breed club. The breed club is where the breed experts are found. 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.*) Mr. Bodegård gives an example; “If SKK should say ‘a long neck is unsound’... and with this statement they change the standard and a giraffe with shorter neck is bred for... Question will be – is it a giraffe?”

Could this give a result of seeking 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 Committee) contacts 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 like some 60 years ago? How big an influence will it have on the exterior if the breeding turns toward different ideals? For example: Is a longer legged dog sounder than a short legged? (*...short-legged and low legged are a distinctly different. Mr. Bodegård continues: "short legged is not something to look for. But, are the longer legged dogs sounder? I don't know, but usually the short legged dogs are higher in rear and most of the times they also have curved front legs."*)

How about color markings? And the description of mask in head? Will, what is called “faults”, follow with a different way of breeding?

It is important to communicate within and outside the breed club? Also, if the language barrier makes wrongly interpreted words in a breed standard – what will then be the clear consequence of this in other countries?

Dr Göran Bodegård finishes his lecture with: It is important to communicate in between each other and to have these kinds of meetings.

HD within VÄSTGÖTASPETS – by MATHS LINDBERG

Maths Lindberg, is an X-ray technologist and radiology specialist. He is a teacher at The Swedish Board of Agriculture (for District Veterinarians). ML has for many years followed the Swedish Vallhund breed with great interest. His lecture here was held in dialogue with the audience.

Note: It was suggested at the meeting that the club should send a proposal to the Swedish Kennel Club for forwarding to the Nordic Kennel Union, and their panel, which is investigating HD within FCI. If there is a problem in some breeds and specific instruction for a single breed are in hand, there might be a chance of a rule being changed for a certain breed. All condrostryfokial breeds should be treated the same – if given wrong HD-results. If more breed clubs would send in an application with an explanation of the problem, this could 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 text regarding HD in the breed Västgötaspets, given by Mr. Maths Lindberg, made a note about the significant parts of the text. The Swedish Vallhund Club has therefore decided to take this part out from the Notes. 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 about hip dysplasia described at the lecture are unfortunately faulty, incomplete or unclear. To change them and to give their full completion and correction in many of the things said in the text, requires a lot of time. Since several of the things written already are published, either at the Swedish Kennel Club's website or in different articles in the Swedish magazine 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 situated at her and her husband's farm. She is breeding Dachshund 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 on questions 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 get 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 matings 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, the figure may be 75-80 % of our bitches will get pregnant after mating. That is my own guess.

When should the bitch be mated?

During the first days of the bitch's season the bitch won't show any interest in being mated. After 5-6 days there are bitches that show signs that they want to mate even if it is far from ovulation time.

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 with 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 matings with one day's rest results in a higher percentage of bitches getting pregnant and larger litters. The best time to mate is when progesterone level is between 30 and 75.

A vaginal smear can also provide some assistance to see if the bitch is in high season (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 high season?

Day 1 in high season is when the bitch allows mating. High season can vary from 4 to 20 days.

Many people consider that bitches should be mated between days 10 and 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 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 70 000 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 male, 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-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 this is 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 fetuses 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 kennels that used to have problems after the Axilur-program had 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. They all provide the trace elements and vitamins needed. An excess of calcium 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.

Caesarean 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 categorically. There are so many examples of bitches that after a caesarean, 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). Prostaglandin 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 carrying only 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 decreases/stops.

5. More than 6 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 to help the bitch?

Go for a run with the bitch (2-3 minutes).

Stimulate the push-reflex with one or two well-washed fingers in the roof of vulva.
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).
You can give the bitch a "Power-kick" (honey and egg yolk).

After the lecture some answers were given:

It is recommended not to use males before one 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 a "dry season" could be that they at their first season are showing signs of being in season, but are not, and after a short while they will have a correct season. Others can early in the season allow mating, even if they don't have an ovulation.

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

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

Cryptorchism 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 a Swedish Vallhund male need not be used that much.

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

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

Her first dog was a Sky Terrier (in 1983). Through two very special friends she has followed this breed very closely in Finland. In 1993 she was authorized as an International FCI Dog Show Judge.

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

Short tail phenotype has probably existed 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 homolog 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 this mutation early on and this made scientists investigate if there could be the same mutation in dogs. All mammals have similar spinal structure, a giraffe also has the same number of vertebrae as we have: 7 cervical, 13 thoracic, 7 lumbar and 3 sacral. The tail has a variation of vertebrae.

The shape of the vertebrae is a structure called mesoderm. One part of it forms a paraxial mesoderm. Paraxial mesoderm gives rise to vertebrae and bone structures of the trunk.

Vertebrae are formed from structures called somite. Every somite follows exactly the same shape and form and stays side by side exactly the same on both sides of the spine. It is one of the most studied structures.

What is a T-box? A T-box is part of a DNA structure (all this is showed in Power Point pictures).

A transcription factor consists of three factors: Eomes, T and Tbx6. T-box is the factor that causes the short-tail. 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 are 5.5 billion base-pairs in dogs. A dog has in its total genome approx. 2.5 billion base-pairs and 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 Sainio says: No one has yet found all the keys to how it really works in this structure.

For somite formation and for formation tail structure, transcription factors contains regulatory elements and coding regions, in under are Enhancer and Promoter together with Intron and Exon.

Mendelian are by heredity out of the short-tail Phenotype.

T-dominant and T-recessive

TT	Tt	tt
25%	50%	25%

TT = without Tt = short-tail tt = lethal mutation of short-tail

Newborn with tt will never survive, they will die before they are born or soon thereafter. The homozygote mutation seems OK, but when looked at closely, the puppy's vertebrae looks developed, but its hind legs does not look well and the puppy 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 phenotype but are not breeds with short-tail, not the same mutation. In breeds like Boston terrier, English bulldog, French bulldog, King charles spaniel, Miniature schnauzer, Parson russell terrier and Rottweiler it looks recessive.

What does a breeder need to know about the T-box?

It affects the development of vertebrae and tail. In short-tail phenotype dogs 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, and 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 mate in Finland. The same goes with two merles and two harlequins. Any breeding between two mutations is forbidden – despite the length of the tail as it is a mutation. The only way to see if it is a mutation is to have a genetic-test done. In countries where it is allowed to breed two mutations, breeding short-tail with short-tail to have short-tail puppies is not recommended. It will give smaller litters and it will lose the homozygote line that long-tails have.

From the beginning all mammals developed a tail. Since we don't really know what causes 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 that there is always Modifiers that promote for or against effects or defects.

Years ago when a puppy died it was called a "fading puppy-syndrome". But, it was not always due to this. Puppies described here, look different all over and also they do not have a developed opening of anus.

COLOURS

Geneticist Clarence Little found out the color genes in 1957. Proteins are coded by the color genes. It contains two pigments – 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 gene-combinations and gives a description that I was not able to write down.)

Dr Mendelians, Inheritance of Colors, is only seen when you have enough puppies in a litter (i.e. hundreds). Breeders of Labrador retriever say they do not have these colours. KS say they do. A brown Labrador retriever 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) grey.

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 have nothing to do with specific genes. White patches are lack of color and will therefore show white hairs.

When can it cause problems?

In the development of the inner ear, there is a region named Stellata Vascularis, which produces liquid for the inner ear and makes the inner ears cells move and hearing will develop. If Melanocytes are missing it will not develop the Stellata Vasculatis and the dog will not hear, i.e. deaf. If Melanocytes are missing this will also affect other areas in the dog. Pie-baldism. In dogs the white color, or lack of pigmentation, is recessive. White spotting alleles sp and sw. Pie-baldism comes from two words: "magpie" and "bald eagle".

S-genes (white spotting)

S

si = Irish spotting in 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.

The 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 situated in County Västmanland in mid-Sweden. The Animal Hospital Strömsholm has 140 employees, including 40 veterinarians. They have 26.000 cases a year. Strömsholm has a section for Ophthalmology with two specialists who were educated in the United States. Berit WH is to retire from Strömsholm, but will be continuing her work as a consultant at the Swedish Kennel Club and within The Board of the Swedish Eye Panel.

The following results are from the Swedish Kennel Club Eye data from registrations in between 1991-2010. 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 consideration that these numbers are based on year of birth.

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

Explanation to PowerPoint(quick description of the eye.): The Retina – grey arrows. Under retina is the Choroid. Eye-color comes from the Choroid. The Retina itself looks like "wet toilet paper". Behind the Retina is Tapetum (colored), non-Tapetum and Tapetal area.

Following are all eye-forms with any comment on:

- Bullous Gray Hyper-hypo reflective – 14 cases.
- Changes in the Lateral Tapetum (roundish changes in a cluster in the lateral 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 are 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 signs = 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 eyes.

Future?

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

ANDRAS M KOMAROMY Assisting Professor in Ophthalmology - 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 in 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), (Retinopathy-research)

Retinopathy 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, ophthalmoscopy 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 SV-population in the United States, 64 percent 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 Retinopathy. Out of the 1000 dogs they have selected 414 blood tests. In this pedigree there are several affected dogs, with non-affected parents (but probably carriers). This sustains this disease is autosomal recessive. There are not more affected males than females, therefore Retinopathy 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 dog is affected late, it will be a problem for the breeder. In Sweden this breed does not require eye-examination, but some owners are having their dogs checked anyway.

A genome includes 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 by 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).

Retinopathy

Stage 1.

Shows no sign of Retinal degeneration. Could probably be used for breeding, but only with healthy individuals. 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 not be used for breeding. What was seen 4-5 years ago is still true. To begin with, red-brown dots are seen and grey "mottling" – multifocal 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. Symptoms can stop at stage 1 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 degeneration. 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 also differ from earlier experiences. The process has now gone from progressive Focal Retinal Degeneration to General Retinal Degeneration. Dog's 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. What will also help science is to receive eyes from affected dogs after they have died.

Examinations with electro-retinography 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 is put in over the eye. Dr Komaromy has made a portable kit for him to easily do this at any vet clinic when required.

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

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

No signs of possible vitamin E-relation have 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 Retinopathy.

They do not yet know how big a percentage that has developed from stage 1 to stage 2 and 3. In the USA AKC has only seen one dog with a developed stage 3. Most dogs stops at stage 1.

It is important for this breed's gene-pool to continue to breed your dogs until confirmation of the disease and not to panic.

A proposal was put forward that clubs all over the world should start their own help programs with matching funds to support this project. Economically the figures come close to 100.000 US-dollar a year to support this 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 cooperate and to share information with each other. What we know today is thanks to all help from Finland and the US.

Three important documents that have to be included at the DNA-test is a blood sample, the pedigree of the dog and a copy of his/her 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 the 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-sample.
2. If the dog has been eye-examined send also a copy of this with the sample.

3. Ask your vet to get you 3-5 ml of blood in an EDTA-tube.
4. Write the name of the dog and his/her registration number on the tube. Check that all information is the same in all papers you are sending.
5. Mix the tubes 8-10 hours so that the EDTA is mixed in the blood.
6. Send the blood sample, blood-test form and the eye-examination to the address that is on the blood-test form. Keep the blood-test in the refrigerator just until you go to the post-office.

If the blood sample was taken on a Friday, keep it in the refrigerator over the weekend and send it by mail on the following Monday.

All given information will be strictly confidential. The dog's and owner's data are archived safely in a database for research purposes only.

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 will know how continue. Both eyes are often affected similarly. 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 fool his owner.*

I have seen dogs which have barely any eyesight (which I can 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 Program Profile

IB AHLEN.

Product and business analyst, all stock, at Agria Insurances, 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 been produced to meet the information of 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 16 diagnoses. The database is primarily for insurance policies in various fields. A researcher must evaluate to see to that 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 our breed in Agria statistics is the number of dogs hit by cars.

Conclusion for the Swedish Vallhund is – it is a healthy breed!

Explanations

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

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