NEWSLETTER

Issue 2

August 2007

Hon Secretary:
Keith Gullis
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24c Lowden Avenue
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Special points of interest:

- Update on research into SA-auto immune disease
- Judges Assessment day October 13th 2007
- Next breed classes are scheduled at Darlington Championship Show 14th September 2007
- Latest imports to the UK

Judges Assessment Day

Saturday

13th October

2007

contact

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Er!
What time is Dinner..?

What's in a name….?

In the words of Shakespeare we ask…. Which of you can think of a title for the club newsletter ? See how creative you can be and submit your ideas for a catchy title… We will publish ideas and ask members to vote and choose the best title.

Can you be creative…?

If so, we would love to hear from you! If you have a story to share about your dogs, a funny picture or any information that other members may benefit from please send it to us and we will include it in a future edition of the club newsletter.
Welcome to issue two for this year but ideally, we would like to publish in the seasonal branding of Spring, Summer, Autumn and Winter each year.
Material that would make interesting and informative reading will be most welcome.
Maureen Thompson 0115 9637962

**** NEW IMPORTS ****

Birin Go Musashi Aiwa
A red male from Japan.
Imported by Keith Gullis + Erika Mason.

Unsyuhime Go Shun'You Kensha
A red brindle female from Spain. Imported by Maureen Thompson, Peter Chapman, Cherry + Alan Wilkinson.

Dei Laghi Tailly Zanghez No Hiasi Anshee
A red female from Italy. Imported by Andy + Sheena Clarke.

JAIC Banner….

Materials and labour have been generously donated to produce a banner in a rich velvet fabric that displays an embossed club logo for use at club events. For a **one off fee of £15** you can have your kennel name embroidered onto this banner and make use of the advertising opportunity at all future club events.

Contact Cherry Wilkinson **01205 870414**
cherrylynn@hotmail.co.uk

Discover Dogs… volunteers wanted!!

Can you spare some time to help?

As the recognised club we have already been approached by the Kennel Club to provide a number of dogs to represent the breed at their prestigious 'discover dogs' events in the near future.

The annual event at Earls Court, London will be held on 10/11th November 2007. It is well attended by members of the public and as such is an ideal opportunity to meet and inform members of the public about our breed and it's requirements.

Many of you will already know that a similar event is also staged at Crufts each year which runs for four days during March 2008 and can be very demanding on numerically small breeds.

If you are willing to help by bringing along your dogs or making yourself available to be on the stand at any of these events for one or more days we would love to have your help. Please contact the club secretary to express interest and obtain more information.
Keith Gullis

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“Sebaceous Adenitis” – Realities in Akita dogs

Overview

In 1985 the first report about Sebaceous Adenitis (SA) was published.
SA is an uncommon inflammatory disease centred on the destruction of the sebaceous glands. The disease has been reported in many different dog breeds and also in mixed breeds.
Nevertheless, a predisposition of Akita, Poodles, Hovawart, Vizsla and Samoyed is known. In the United States SA affected dogs were collected in a database administrated by the Orthopedic Foundation of America.
Today only one skin disease, coupled with attacked glands was described in veterinary medicine literature: "Sebaceous Adenitis". The following part explains the actual situation of the scientific investigations / knowledge.

Sebaceous Adenitis

In general, typical outer signs of Sebaceous Adenitis are, the coat is dull, dry and fragile like moth-eaten and the odor of the dog changes: They smell like “three days worn socks ".
The first signs of the disease are subtle and appear at the head, the ears and in the trunk. The hair seems thinner, almost "similarly to moth damage". In some cases the coat-colour changes.
SA is normally non-pruritic unless there is a secondary staphyloccal skin infection and even furunculosis may develop.
As already mentioned, different inflammation degrees of the skin can be ascertained by microscopic investigations. At the beginning of the disease, histopathologic findings demonstrate different cell types of the immune system in the sebaceous glands. The cells were described as perifollicular granulomes (aggregations of many cells). The aggregations consist of defensive cells, macrophages, lymphocytes, plasma cells and neutrophile granulocytes.
As microscopic investigations (histological examinations) of skin biopsies pointed out, the inflammation character of SA goes through several different stages. Only after complete loss of the sebaceous glands the inflammation intensity decreases.
The etiology of microscopic investigations showed cells or other components of the immune system participating massively in the destruction process. However, no bacteria, viruses or other pathogens are proven to be the cause of the disease, but all autoimmune reactions are focused against self substances/cells.
As demonstrated by several statistical calculations (Segregation Analysis) our new investigations have shown that **SA in Akitas follows an autosomal recessive inheritance *1**, a so called genodermatose.
In this context we check lots of pedigrees available from affected and non affected dogs and their relatives. Nevertheless we can exclude environmental factors as the main cause of the disease.
Beside this SA can be triggered by stress: eg. pregnancy, changes in the ownership. Additionally no significant relations can be derived between the duration of the disease and the degree of SA. It has turned out, that it is very difficult to find out in which stage of the disease a SA-dog actually is. There are dogs with a nearly complete destruction of all glands after a timespan of 2 months. On contrary, individuals were monitored with a destruction of the glands for many years eg. 8-9 years, similar to a slowly creeping process. Typical for the disease is a very “individual character” in each case.
The reason for the destruction of the sebaceous glands, is actually unknown. **In any case, no viruses, bacteria or other pathogens are observed as a primary cause.**
From this point of view the scope of SA-research is actually focused on mechanisms triggering the destruction of the cells by the immune system and what kind of genetic reason(s) plays a key role.

Research

Due to the etiology of the disease microscopic investigations showed cells or other components of the immune system participating massively in the destruction process. However, no bacteria, viruses or other pathogens are cause of the disease, but the autoimmune reactions run against substances/cells of the showed cells or other components of the immune system participating massively in the destruction process. However, no bacteria, viruses or other pathogens are proven as a cause of the disease, but the autoimmune reactions run against substances/cells of the dog.
What causes exactly the “false control” of the immune system?
Until today, the research has shown that **SA positive dogs have “changes” in the blood,** which we cannot assign. In the blood many components of the immune system are located. Finally these components can be found later in the sebaceous glands,- so the question is:
1. What kind of connecting links/cross-reactions exists between the immune system / blood and the sebaceous Glands?
2. What kind of genetic background is responsible for this process?

At the moment both questions were investigated by different methodical attempts.
The organisation of immune system is very complicated and a huge number of interactions run off, so our search is multi-layered. But to observe the key and the connections to the triggered reasons, lots of investigations are necessary.
Actually we investigate a so called “Hot Spot” on genetic level, because this mutation seems to be very prominent in affected dogs. **But we need more EDTA-blood samples from affected dogs to undertake a “risk calculation”.**
Furthermore we **need pedigrees from affected dogs with their relatives** to investigate the mendelian inheritance of the disease. This pedigree information we will incorporate into our segregation matrices (statistical calculations) to generate powerful results.
But without the support of engaged breeders and Akita-friends the progress of the SA-research would not be conceivable and we thank all who help us.

PD Dr. Ina Pfeiffer
Dipl Biol. Tina Roth
Dipl. Mathem. Robin Wellmann

University of Kassel

Club Web Site

www.japaneseakita-inu.co.uk
Is the official club web address
Why not take a look!!

Come on slow coach…?