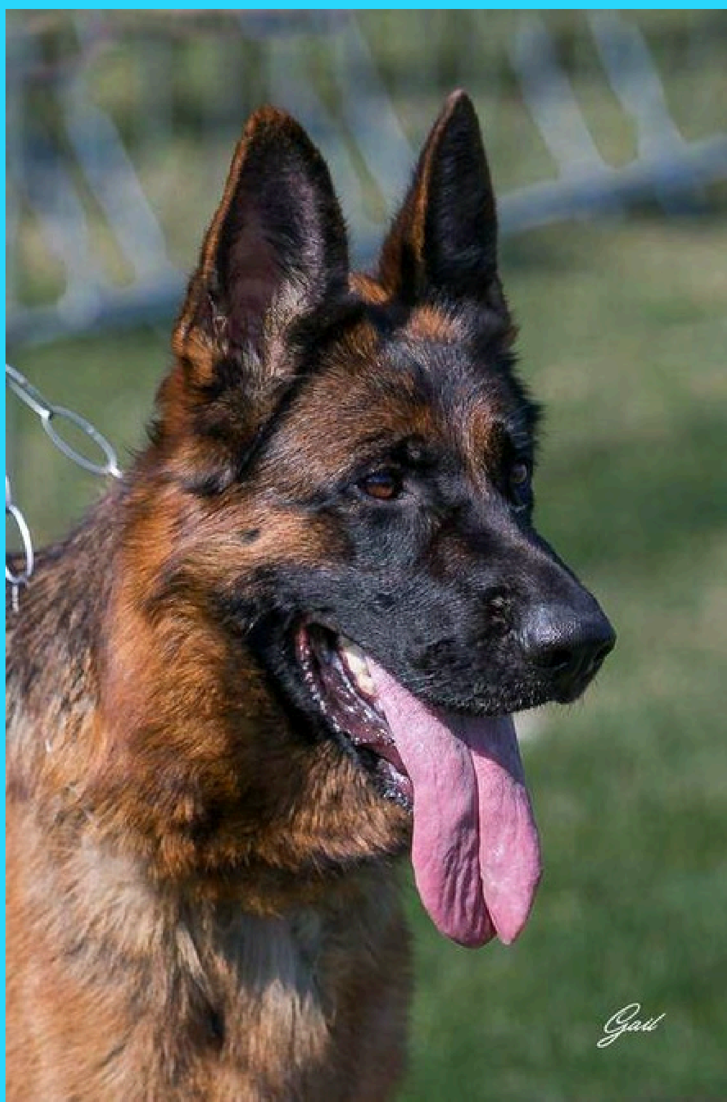
Shepherd News

February 2025

THE GERMAN SHEPHERD DOG CLUB OF S.A. INC.

What's Inside:

Page: 2	President's Report
Page: 3-6	Breed Affairs
Page 7:	65th Member's Competiton Schedule
Page: 8	Breeders Directory & Club Training Times
Pages: 9	Stud Directory
Page: 10	Ground Rules



President's Report
John De Lucia

I would like to welcome back all our members after the Christmas break and also extend a warm welcome to all of our new members.

The club has recently held a working bee, enabling a great deal of tidying up and replacing the gutters on the main verandah. I wish to thank the helpers who turned out to assist.
We also held our very popular annual puppy day with over 65 people in attendance with around 35 puppies. It was a fantastic morning mixing with potential new club members and the club also provided a free BBQ and drinks for all.

As with all things in life the club needs assistance in many areas, especially sponsorship so if you can help out with products or monetary help it would be greatly appreciated with businesses being able to advertise on our website and Facebook page.

I am looking into making an Honor roll board for past executive club committee members to be placed in our club rooms so if we have a sign writer in the club who may be able to assist in this project please reach out to me it would be greatly appreciated.

This is just a general reminder that our Shepherd Shop sells assorted dog-related products and food along with club clothing so check it out as our prices are quite reasonable.
Also, a reminder that all membership payments are now overdue so if this has slipped your mind over the holiday break, please see the Secretary, Stephen as you will not be able to attend training.

Looking forward to seeing you all again.

John J De Lucia
President

ROYAL CANIN

Breed Affairs

Dr. Wolfgang Lauber (Retired SV Judge and Veterinarian)

Thoughts on Degenerative Myelopathy

The Degenerative Myelopathy (DM) is currently a hotly discussed topic of many breeders and unfortunately often ignorance leads to the dissemination of half-truths and to uncertainty in view of handling of the DM. In the US, the situation was similar three years ago, and now we have to work against hysteria in Europe. In dog breeding the deciding factor should not be a fashion trend, but instead it must be well thought out action based on knowledge and science, that are the basis. The DM of dogs is a slowly progressing neurological disorder. There is a destruction of the nerve sheaths in the spinal cord. This initially delays the forwarding of signals, which is completely blocked later on. The animals show a weakness of the hindquarter, which begins with often unnoticed scraping of the claws of the hind legs. The progression of the disease is very slow and can continue for several years.

As with other breeds of dogs, the first symptoms usually occur in the German Shepherd Dog from around the age of eight years. The owners then realize that the dog has difficulty getting up, jumping into the car or the hurdle. Typical here is the asymmetric onset with signs of paralysis in only one hind limb. Unfortunately the diagnosis can only be made on the living dog according to the exclusion principle, i.e one must eliminate other illnesses by means of a thorough examination as well as x-rays and MRI. Diseases such as the Cauda equina syndrome", degeneration of discs in other localization, hip dysplasia, tumors and much more lead to similar clinical symptoms. A definitive diagnosis of DM can only be made by means of a pathological examination of the dead animal. The under the microscope detectable changes in the nerve tissue are the proof. The use of medication can not cause a visible improvement in the signs of paralysis. The disease usually worsens over a period of 1 - 2 years and leads to the complete paralysis of the dog. There is no pain throughout the progression . A mutation in the gene SOD1 could be identified as a risk factor for the disease.

The occurrence of the DM in the German Shepherd was already described in the 1970s. All statements on its frequency or also the frequency of the risk allele should, however, be interpreted with caution because the investigations were based on preselected patient-material. The majority of the dogs were clinically conspicuous, showing neurological disorders that already aroused suspicion of DM. Figures for a total population can therefore not be derived from these studies.

In Germany, there is a gene test to show proof of the mutation associated with the formation of DM on the SOD1 gene. However, the result is no diagnosis of "sick" or "healthy", it only provides information whether the risk- allele exists or not. So it can be estimated how likely it is that the tested dog will contract DM in old age.

The result can be:

Normal (N/N), DM-free
This dog is homozygous, it has two normal copies of the gene. This dog can only pass on the normal variant of the gene to its offspring, and it is very unlikely that this dog will get DM.

DM carrier (A/ N)
This dog is heterozygous A / N, so it has a mutated and a normal copy of the gene. While it is unlikely that such a dog will suffer from DM, he may pass on either the normal or the modified (risky) form of the gene to his offspring.

At-risk, carrier (A/A) d
This dog is homozygous A/A, it has the mutated gene twice and is at risk of developing DM itself in old age. Whether this will indeed be the case, however, can not be predicted. This requires more extensive research in order to clarify which factors are ultimately decisive for whether and when a dog becomes ill with DM. According to current knowledge, the DM is a multi-factorial disease with genetic and non-genetic factors. Dogs with the result A/A always pass on the changed (risky) form of the gene to their offspring. Matings of DM -risk carriers thus always lead to offspring with an increased DM-risk of disease and should be avoided.

The previous research on the DM has been driven substantially by the fact that it bears great resemblance to a disease of humans. Nevertheless, many details are still unknown, and especially the knowledge of what is especially important for the dog owners and breeders is still sketchy: How common is the disease overall ? And how much more often do A/A dogs come down with DM in comparison to dogs with only one or no copy of the mutated SOD1 gene? What does just when a dog shows the first signs of DM, depend on, and how quickly do the symptoms of paralysis increase?

The SV had decided to contribute to the clarification of these and other questions that arise all around the DM, and thus to the objectivity of the discussion. The first step is to collect data that can be expected to deliver meaningful results: Randomly selected dogs whose genetic material (DNA) as well as comprehensive information on how they are kept and their usage, on their development and health, is available, are ideal for this. The SV “study on joints” already provides such a sample of dogs, and the continuation and expansion of data collection on the DM offers itself. All results from the study will be scientifically reviewed and, of course, published only in anonymous form. This ensures that everyone can participate with their GSD without having to fear adverse consequences for themselves or their dog. Instead, they increase their knowledge to which they can contribute and which is especially urgently needed for the DM. Only if there is a solid basis of information can solutions be found in joint undertaking in the association, and measures can be defined that will sustainably serve the health of the breed.

Addition by Federal Breed Warden Lothar Quoll

The Working Group on Health recommended to the VWA in their spring session that the " study dogs" (better known as the "joints- study") be subjected to a gene- test for DM. The VWA unanimously approved the procedure in its April meeting. The costs for the genetic test of the study dogs are borne by the SV. This gives us data representative for the DM in our breed.

Dr. Manz explains:
The DM is not a monogenic genetic defect. Besides the SOD1- Gene, other genetic factors are likely to be involved. (Inheritance with incomplete penetrance). This explains the late occurrence and the observation that 34% of unaffected animals are homozygous for the SOD1 mutation (Awano et al, 2009). In a genetic test, it is essential to properly assess the epidemiological (epidemic-like) situation and to control the application of a gene test.

Findings on the case of the SOD1 mutation and the clinical DM, according to Awano:

- Frequency of the disease: 2.1%

- animals with DM diagnosis and homozygous for the SOD-1 mutation: 96%

- Sound animals and homozygous for the SOD-1 mutation ("at risk"): 34%

From this, the prognosis value of the SOD-1 mutation for the DM disease is calculated to be 5.6%.

That means out of 1000 animals tested homozygous for the SOD1 mutation, only 56 developed a clinical picture.

That means out of 1000 animals tested homozygous for the SOD1 mutation, only 56 developed a clinical picture. The onset of the disease after the reproduction phase and the relative little significance of the SOD-1 mutation explains the high frequency of SOD1 carrier animals.
In ignorance of the epidemiological situation and the significance of the SOD1 mutation, the test is already widely misused as a diagnostic tool: animals with ataxia of the hind limbs and simultaneous homozygous presence of the SOD1 mutation "have DM". Unfortunately, the breeders' ignorance now leads to quite unjustified stigmatization of SOD1 carrier animals.

The "at risk" (carrier) animals too are valuable, because they carry a genetic endowment that compensates for the effect of the SOD1- mutation. The test is therefore not suitable as a diagnostic tool, as has been pointed out frequently.

For the handling of the DM situation, Dr. Mans would suggest the following procedure:
To gain the first insight into the events, one should test a number of breeding animals that are over 10 years old. The studies would show if there are any animals among these, tested homozygous for the SOD1- mutation. These clearly have the genetic makeup that compensates for the effect of the SOD1- mutation, and are therefore very valuable for the breed. To examine the offspring of these animals would then also show whether the effect is passed on.

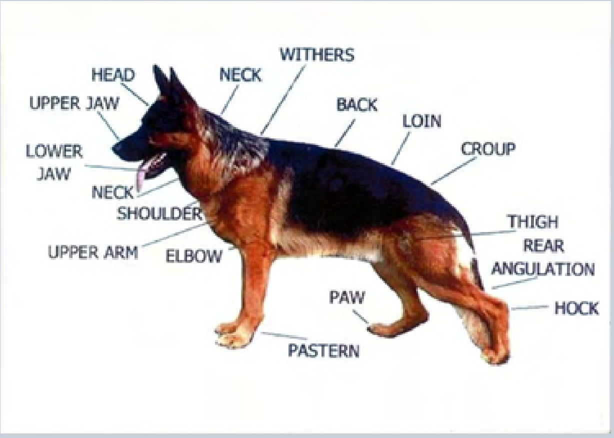
To offer parallel to this, the opportunity on a voluntary basis, for diagnostics when submitting a DNA sample. The "joint study" dogs are included mandatorily. This gives the opportunity to become informed on the disease and the importance of the test for the SOD1 mutation and the SV gains insight on the tested animals. In Combination with 1) we receive further information on the effect of the SOD1 mutation.

Younger animals tested homozygous for the SOD1- Mutation will be accepted for monitoring. I.e. The pet owner should inform the SV when the first symptoms of the DM show themselves. (Volunteers and study dogs)

My thanks go to the working group including Fr.Dr. Stock and Hr.Dr. Tellhelm for their counsel. My special thanks go to Hr.Dr. Lauber for the preparation of the article.

Lothar Quoll
National Breed Warden.

Interpreter's note: Researchers have found that the genetic mutation responsible for degenerative myelopathy (DM) in dogs is the same mutation that causes amyotrophic lateral sclerosis (ALS), the human disease also known as Lou Gehrig's disease. ... Researchers found that dogs with DM also had mutations in their SOD1 gene.
Honey Gross-Richardson



65th Members Speciality Competition

Barratt Reserve, West Beach SA

Sunday 16th March 2025
Commencing At 9-00 am

Judge : Mrs Jean Butterfield (SA)

The club reserves the right to appoint a reserve judge if necessary

Should the Dogs SA heat rule apply, the show will commence at 7-00pm on Saturday 15th March 2025.
There will be no tea break and the Junior Showmanship Competition will be cancelled.

NO CLASS RUN OFFS & NO CHAMPIONSHIP POINTS AWARDED

Classes & Order Of Judging –
Long Stock Coats followed by Stock Coats, Bitches followed by Dogs, in age order alternating in variety. LSC1a, SC1a; LSC2a, SC2a; etc. Followed by Best LSC bitch, Best SC bitch. LSC1, SC1; LSC2, SC2; etc. Followed by Best LSC dog, Best SC dog.

Trophies: 1st place in each class; Best Bitch and Best Dog of each variety.

Sashes: 1st to 4th place in each class where numbers permit.

Gradings: Awarded at the judge's discretion in all classes. Excellent medallions will be awarded only in the Open classes if the animal has been successfully Breed Surveyed and is over 24 months of age. **GRADING CERTIFICATES FOR ALL CLASSES WILL BE AVAILABLE**. **Critiques:** Available for Junior, Intermediate & Open classes only.

Entry Fees: Baby, Minor & Puppy classes - $12-00 per entry
Junior & Intermediate classes - $20-00 per entry
Open classes - $25-00 per entry

Catalogue – $5-00 each. MUST be ordered separately (not included with initial entry).

ALL ENTRIES CLOSE SUNDAY 9/3/25

Entries: Available online at www.showmanager.com.au or by post to *The Secretary, GSDC of SA Inc, PO Box 109, Willunga SA 5172*

Show Manager
Online Entries Available
www.showmanager.com.au

Please ensure all online details are up to date.
Paper entries need a separate entry form for each class. Please ensure that variety is stated on entry form eg SC or LSC and all titles, pedigree names, registration numbers and full details of sire & dam are shown on entry form. Cheques are to be made payable to the GSDC of SA Inc.

Any person double handling at this fixture does so at their own risk. The GSDC of SA accepts no responsibility for any injury that may occur while double handling.

President's Youth Trophy Competition
(Incorporating GSDCA Junior Showmanship Award)
Judge :TBA

Entries taken on the day. To be judged during the lunch break.

Medallions (donated by Alimanda Kennels) will be presented to all participants.

Classes: 7 to under 10 years; 10 to under 13 years; 13 to under 19 years

All Youth Handlers must have control of the animal they are exhibiting. If any handler cannot solely control (hold) the animal they are exhibiting they may be asked to leave the ring.
No adults will be permitted in the ring.

Breeders Directory

ALIMANDA KENNELS
J Butterfield & G Mercer
OLD NOARLUNGA
Mob: 0412 471 802
Email: alimanda@hotmail.com.au

ARKAHLA KENNELS
W & J & A O'Loughlin
PORT NOARLUNGA
Mob: 0412 605 584
Email: arkahla@gmail.com

BABENBERG KENNELS
J & J & I DeLucia GAWLER BELT
Mob: 0408 100 745
Email: jjdelucia@bigpond.com

RHOSYN KENNELS
S & J Collins
WILLUNGA
Mob: 0419 212 749
Email: mail@rhosyngsd.com

SHEPROSE KENNELS
B Owen
TWO WELLS
Mob: 0417 834 941
Email: sheprose01@gmail.com

Training Times

Sunday

9.30 am: Beginners Obedience
Obedience Class 1, 2 & 3
10.00 am: Show Training
10.30 am: Obedience Class 4
Open Obedience & Utility Classes
1**1.00 am**: Class 5

Wednesday

7.30 pm: Beginners Obedience
Obedience Class 1, 2 & 3
Show Training

8.15 pm: Obedience Class 4 & 5
Novice Obedience & Trials Classes

Stud Dog Directory

***CH LENIN VON DER GRAFENBURG IPO2 (Imp Deu) 'a' Normal ED Normal**
Sire: *Pepe v Bordetal IPO2 'a' ED
Dam: *Tatoo v d Grafenberg IPO1 'a' ED
Owner: Mrs C Collins & Mr J De Lucia
WILLUNGA
Phone: 08 8556 2340 **Mob:** 0419 212 749
Email: mail@rhosyngsd.com

***BABENBERG VALENTINO ‘A’ (2-2) ‘Z’ NORMAL (0-0)**
Sire: CH * Lenin vd Grafenburg 'a' ED (Imp Deu)
Dam: *Babenberg Tiffany AZ
Owners: Jenny, John & Isabella DeLucia
Mob: 0408 100 745
Email: jjdelucia@bigpond.com

***YACCO VON EICHENPLATZ 'a' ED Normal**
Sire: *Yoris v Osterberger-Land 'a' ED
Dam: *Milagre v Eichenplatz 'a' ED
Owner: Mr S & Mrs C Collins, WILLUNGA
Phone: 08 8556 2340
Mob: 0419 212 749
Email: mail@rhosyngsd,com

Ground Rules

These rules apply to all handlers and have been formulated for the welfare of you and your dog.

Any Club member, or any person guilty of unseemly conduct or acts harmful to the club, may be asked to leave the Club grounds. They may also be dealt with by the Committee of The GSDC of SA Inc. under clause 6, and relative by-laws of the Clubs Constitution.
Members shall not treat their dog in a manner that may reflect badly on the Club.
The owner and or handler shall be responsible for their dogs while on club grounds and must accept any liability for animals in their care at all times.
Any dog that is sick WILL NOT be permitted onto the Club Grounds.
Dogs who have suffered a CONTAGIOUS DISEASE WILL NOT be allowed on to the Grounds until 5 weeks after being cleared by a veterinarian (a vets certificate may be required). Puppies should not be brought out onto the Club Grounds under the age of 8 weeks or at least 2 weeks after they have been inoculated for HEPATITIS, DISTEMPER AND PARVOVIRUS (vaccination certificate may be required).
The Head Instructor has the authority to permit the use of a muzzle or training devices, as required in special cases. The use of the equipment is to be under supervision, and its use on the Grounds, other than above, is STRICTLY FORBIDDEN.
The use of switches, spiked collars or whips is STRICTLY FORBIDDEN ON THE CLUB GROUNDS.
Dogs are NOT to be taken off lead or left unattended, except on the directives, or under the supervision of a Class Instructor.
Bitches in season are not permitted on the grounds unless at a recognised conformation show.
Dogs under the age of 12 months ARE NOT permitted to use jump equipment higher than 400 mm, or longer than 800 mm.
The handlers of dogs that foul the Club Grounds will be responsible for cleaning up same, with the equipment provided.
Pursuant of the Health act “Dogs” ARE NOT permitted inside the Club Rooms.
In the interest of safety and health, and in consideration of others, parents are required to supervise the behaviour of their children. The Club Rooms and Toilets MUST NOT be used as playing areas.
In the interests of public health the Club Rooms and the veranda area are NON-SMOKING areas.
Appropriate foot wear must be worn at ALL times when handling / double handling dogs on the Club Grounds. Bare feet, thongs, slip on shoes, high heels, sandals, crocs and shoes with metal-spiked soles (covered or uncovered) are not suitable foot wear and members wearing any of the above will be asked to leave the Club grounds.