# 'CASE REPORT - NERVOUS DISORDER IN STAGS'

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## INTRODUCTION

In July 1983 on a South Canterbury deer farm an outbreak of 'staggers' in two mobs of stags resulted in 35% dying. The problem was mainly in aged stags with few signs being seen in younger stags and none in weaners or hinds. The episode lasted approximately 14 days by which time a total of 23 stags were dead.

This paper reports on the investigation into these deaths and the results.

## BACKGROUND

The property involved is located in the foothills of South Canterbury and is run as a mixed sheep and cattle farm, the deer farm being established in 1978. The present owner took over the property in 1980 and has continued a policy of increasing deer farm size and numbers. The setting of the deer farm is 'ideal' for deer with all paddocks having a number of native trees present and excellent shelter. The water supply is from natural creeks.

At the time of the outbreak the farm was running 350 deer being divided into 4 mobs. Adult hinds, weaners, young stags with several sire stags, and a group of mixed age velvet stags.

The stags had been set-stock for several weeks and were being supplemented with meadow and pea vine hay. The hinds were getting pea vine hay and a fresh 'break' of grass every 3-4 days. The weaners were set stocked and receiving pea vine hay.

## DIARY OF EVENTS:-

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| 26.6.83 | Big wind storm. Trees blown down. |
| 6.7.83 | Sire stag observed with nervous signs 'staggers'. |
| 7.7.83 | Samples taken from one 'staggery' deer. |
| 9.7.83 | Sire stag died, first death. |
| 10.7.83 | Stags roaring and rutting 'again'.<br>A number showing obvious 'staggers'.<br>Three dead stags found in paddock. |
| 11.7.83 | Two more dead. 'Staggers' seen in both mobs of stags. |

12-17.7.83 Clinical picture remained same with several stags dying each day.

17.7.83 Signs in stags started to improve. No new cases of 'staggers'.

18.7.83 All stags moved to new area fenced off.

20.7.83 Last stag died.

TOTAL LOST 23 STAGS

## CLINCIAL EXAMINATION AND NECROPSY FINDINGS

First two stags examined on 7.7.83 were depressed, lethargic and when disturbed became very aggressive. They required sedation to enable blood samples to be taken.

By 11 June 1983 the picture had changed to include more pronounced nervous signs, including violent convulsions, especially when moved. Affected deer would 'stagger' around and then throw themselves on the ground and lie 'paddling ' for some time. If left they would slowly recover (about 10 minutes). These deer would walk around in a depressed state and separate from the main mob. A number of deer exhibited a high stepping stilted gait and nervous twitching of the head and neck. Once disturbed the symptoms became exagerated and some deer in this state did a lot of self inflicted trauma to themselves. The whole mob started roaring again and became quite unsettled.

Necropsy examinations were fairly consistant throughout the whole episode. The alimentary tract showed varying degrees of congestion from the abomasum through to spiral colon. Lymph nodes were enlarged, oedematous and haemorrhagic. Lungs, liver, and kidney were usually congested. The lungs often contained fresh rumen material - presumably inhaled during convulsive state before death. The subcutaneous bruising was substantial and often accompanied by gelatinous oedema and the occasional bone fracture.

## LABORATORY EXAMINATIONS AND RESULTS

An extensive and exhaustive examination and investigation was carried out on a wide range of specimens, a summary of these findings follows.

Blood samples taken 7 July 1983 for lead, haematology, serum magnesium (0.86 mmol/l) and calcium (2.15 mmol/l) were all considered to be within normal ranges.

Histological examination of the many tissues did not confirm or suggest any obvious aetiology.

Lung - frequently contained rumen contents with subagonal alveolar oedema.

Liver - congestion marked cloudy swelling and early hepatocyte dengeneration - both cytoplastic and nuclear vacuolation.

Kidney - glomerulitis and glomerula swelling with mesangial thickening. Some early tubular degeneration with cast formation. NO oxalate crystals seen.

Abomasum - congestion and oedema of the mucosa with some surface erosion.

Intestine - very marked congestion and moderate inflammation (possibly parasitic origin). Superimposed on this were focal heamorrhages scattered throughout the lamina propria.

Lymph node - marked congestion and heamorrhage especially in medulla and around capsule. The germinal centres showed lymphoid depletion.

Brain and Cord - congestion, oedema and some perivascular haemorrhage, mainly around capillaries and arterioles - the haemorrhages being numerous and extensive - probably agonal change.

Culture - examinations did not recover any significant findings.

Toxicology - examination for lead strychine, phosphorus and plant toxins were all negative (rumen contents were indentified for plants by N.Z. Forest Service Research Division).

Plant Specimens - from trees brought down by storm on 26.6.83 and obviously eaten by the stags were identified as all being not known to cause any animal poisonings. (Mainly Hoheria augustifolia). Samples of pea vine and meadow hay were examined for presence of poisonous plants or fungal toxins and found negative.

Animal Feeding Trials - a small trial was conducted at Invermay Nutrition Unit, feeding the peas vine and meadow hay to 2 red deer calves for 2 weeks and they remained normal. Also an extract made from a "canker" or "gall" found in quite large numbers on the branches of the hoheria trees was fed to guinea pigs and 2 red deer calves and they also remained normal.

Grass Samples - collected during the outbreak and examined initially by Department of Scientific and Industrial Research were reported as having no evidence of toxic endophyte in the sample. A sample submitted to Plant Health Section, Ministry of Agriculture and Fisheries, Lincoln, reported Lolium endophyte was detected in quantities sufficient to provide ryegrass staggers symptoms (rgs).

## DISCUSSION

Despite a wide ranging investigation it was not clear at the end of the outbreak as to the precise aetiology. The detailed examinations eliminated a number of possibilities but there remained two most probable causes (Ryegrass Staggers and Hypomagnesaemia).

The conditions considered in the differential diagnoses were, lead, phosphorus and arsenic poisoning, toxicology examinations on a range of tissues all proved negative. Plant poisoning either from the hay being fed or the trees present in the paddocks was ruled out on history and feeding trial. There were no obvious lesions seen on histological examination of a range of tissues for polioencephalomalacia, meningitis, listeriosis, or focal encephalomalacia, and there was no evidence of more subtle changes of phalaris staggers or ryegrass staggers. (It is accepted that the rgs brain changes may not be present in acute cases).

In Australia Annual or Wimmera ryegrass (Lolium rigidum) has poisoned sheep and cattle following grazing of the grass that is mature and is parasitised by a nematode (Anguina funesta) and a bacterium (Corynebacterium rathayi) (1,2,3). Mortality rates of up to 100 percent have been recorded in some sheep flocks. Animals affected with the intoxication show clinical signs similar to those seen in this outbreak (intermittent neurological disturbances characterised by ataxia and collapse with tetanic and clonic convulsions). This condition has also been recognised in the South Island of New Zealand for many years and occurs in sheep fed screenings from grass seed harvesting - with various species especially chewings fescue (Festuca rubra) with the seed head infested with the nematode and bacteria (Hartley pers comm.). While not completely conclusive, this possibility was eliminated on the grounds of history and the feeding trial.

Paspalum staggers with ergot infested hay or grass seed produces a similar clinical entity but with minimal convulsions and seldom death. A tremorgenic fungus growing on the pea vines was also considered but eliminated following careful consideration of the history (the hinds and weaners received the pea vine hay) and in the feeding trial the deer remained normal.

Ryegrass staggers - associated with lolitrem neurotoxins was initially considered to be highly likely as the sole cause of the outbreak. The condition rgs has been described in imported Canadian Wapiti (Cervus elaphus mannitobensis) at the Invermay Agriculture Research Centre, (4) the clinical signs described and those seen in other red herds locally (Beatson pers comm.) were consistent with those seen in this episode. Histopathological findings in the brain in deer (4) and in sheep and cattle (5) which they considered to be characteristic findings in animals with rgs were not seen in any of the brains examined in this outbreak. The negative examination for toxic endophyte reported initially during the outbreak would suggest that rgs was not involved, the confusion arises when the report that Lolium endophyte was detected in quantities sufficient to provide rgs symptoms is comsidered. In this report it was felt by those doing the examination for endophytes that death may have been more of a combination of rgs plus other feed or metabolic factors.

Hypomagnesaemia was considered to be a possible cause from the outset of the outbreak. The initial blood sample analysis showed that animal to be within the normal range as described (McAllum and Wilson 1981 pers comm.). In addition to this normal blood value previous clinical experience (O'Toole pers comm.) suggested that the time of the year (mid winter), the recovery of the affected deer (not normal to expect animals to recover once in convulsions) and the clinical signs showing no major changes in intensity of the heart sounds all tended to lead away from this as a diagnosis. It was perhaps unfortunate that further serum samples were not practical to collect due to the extreme reaction of the affected deer to any forced movement. An attempt was made to collect cerebrospinal fluid from the dead deer but this was not successful. However there are a number of factors which would support hypomagnesaemia as a cause either solely or perhaps in combination with rgs. The deer affected most were the mature stags, particularly the sire stags which has only recently finished mating or the rut period. At this time of the year this class of animal is typically more suspect to change in diet or weather. The deer farm had been topdressed using Potassic super during the autumn. One stag which was that depressed that it could be approached in the paddock was drenched orally with magnesium sulphate recovered fully and this stag is the only one which survived of those which exhibited clinical signs. The shift to another paddock and the oral supplementation with magnesium coincided with the complete cessation of further clinical signs.

From the foregoing it is clear that no one cause for the episode was established, many different possibilities were investigated and the true aetiology probably will not be established.

ACKNOWLEDGEMENTS

We wish to thank Dr M. Orr and Dr C. Mackintosh for examination of specimens and feeding trials and Dr W. Hartley for special examinations and comments on the problem.

REFERENCES

1. McIntosh, G H: Thomas, M (1967): Toxicity of parasitised Wimmera Ryegrass, Lolium rigidum for sheep and cattle. Aust.Vet.J 43:349-53.

2. Berry, P H: Wise, J L (1975): Wimmera ryegrass toxicity in Western Australia. Aust. Vet. J 51:525-30.

3. Bird, A F: Stynes, B A (1977): The morphology of a Corynebacterium sp. parasitic on Annual ryegrass. Phytopathol 67:828.

4. Mackintosh, C G: Orr, M B: Gallagher, R T: Harvey, I C (1982): Ryegrass staggers in Canadian Wapiti Deer New Zealand Vet. J. 30:106-7.

5. Mason, R W (1968): Axis cylinder degeneration associated with ryegrass staggers in cattle and sheep. Aust, Vet. J. 44:428.