

Enzootic ataxia, caused by copper deficiency, was diagnosed in deer in New Zealand in 1978. It probably occurs throughout the country, particularly where soil is deficient or marginal in copper, or where pastures are irrigated.

It is most common in late autumn, and can at times affect, to varying degrees, up to 20% of the young adult deer in a herd.

Symptoms

The condition mainly appears in young adult hinds and stags and seldom affects older deer.

Affected young adults, aged from 6 months–2 years, develop an unsteady swaying gait, but appear healthy and continue to eat. If untreated, the condition progresses through squatting to complete paralysis and eventual death in about 6 weeks.

Deer younger than 6 months may develop sore muscles. In one group of 6-week-old deer calves, some showed varying degrees of lameness and some died suddenly. The only liver analysed had an abnormally low copper level (Table 1).

Once deer develop enzootic ataxia, they cannot be cured, although the condition can be halted.

Cause

A deficiency of copper brings about a demyelination of the nerves, with a consequent disruption of nervous impulses, and later a degeneration of the nerve axons (Fig. 1).

Although the whole of the spinal cord shows bilaterally symmetrical demyelination, the areas most severely affected are the medulla of the brain and the cervical nerve cord. The nerve axons and sheaths cannot be renewed once they have been destroyed.

Diagnosis

Copper deficiency must be distinguished from the effects of the tissue worm, *Elaphostrongylus cervi* (see AgLink FPP 249) which may migrate through the spinal

cord causing damage to the tissue, partial paralysis and death. Animal Health laboratory staff can distinguish between the conditions by examining post-mortem specimens of brain, spinal cord, and liver.

Table 1: Copper levels in the blood and liver of deer

Tissue	Age of animal	Normal Cu level	Deficient Cu level
blood serum	> 6 months	0.66 ± 0.22 mg/litre	< 0.4 mg/litre
fresh liver	< 6 months	23 mg/kg	—
" "	> 6 months	10.5 mg/kg	< 5 mg/kg

There are no relevant post-mortem findings in animals which have enzootic ataxia, and diagnosis is made upon:

- Analyses of copper levels in blood and fresh liver.
- Analysis of copper levels in pasture.
- Analysis of deer faeces for the larvae of tissue worms.

Prevention and treatment

Injections of copper products, marketed for sheep and cattle, have prevented the condition occurring in deer on properties deficient or marginal in copper, and have halted it in affected animals.

If there is likely to be copper deficiency, copper should be administered by injection to all the deer in a herd every 6 months. Copperised licks can be provided instead of, but not in addition to, injections, as there is a chance of poisoning from too much copper.

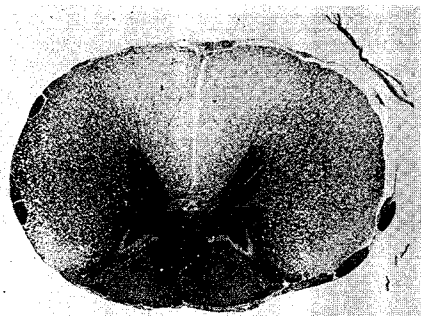


Fig. 1: Cross section of cervical spinal cord showing areas of demyelination.



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Enzootic Ataxia In Deer

Copper Deficiency