ELAPHOSTRONGYLUS CERVI UPDATE

Paul C. Mason
Invermay Animal Health Laboratory, MAF, Mosgiel

Introduction

In New Zealand, Elaphostrongylus cervi the tissue worm of deer, has achieved prominence through the requirement that it be absent from deer exported to Australia and some other countries.

In this paper, I will briefly review the biology, epidemiology and diagnosis of Elaphostrongylus cervi in deer, and later discuss the requirements concerning E. cervi and the export of deer to Australia.

A more extensive review of E. cervi was published earlier in the year (Mason, 1989) and interested readers are referred to this for more detail.

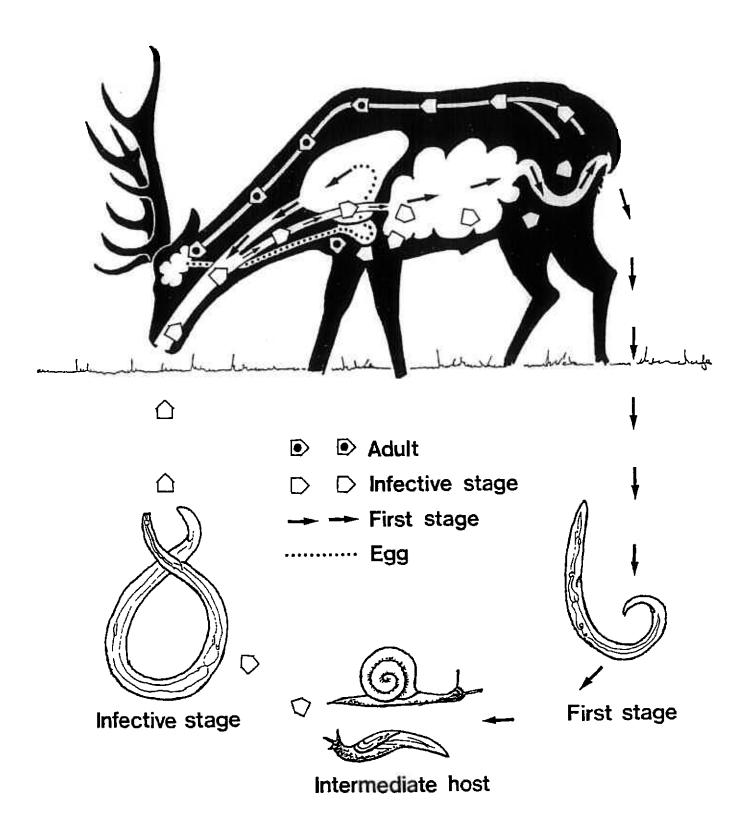
Host Range

Elaphostrongylus cervi is widely distributed in a variety of cervids. It was first described from red deer (Cervus elaphus) in Scotland and has subsequently been identified from C. elaphus in Holland, Czechoslovakia, Austria, New Zealand, Denmark and Poland. It is a common parasite of red deer, marals (Altai wapiti) and sika deer (C. nippon) in the central USSR. It has been reported from wapiti (Cervus elaphus canadensis) in New Zealand and will probably be found in wapiti in North America. It has also been reported from roe deer (Capreolus capreolus) in Sweden, Austria and Scotland. Both wild and domestic reindeer (Rangifer tarandus tarandus) are hosts in Norway, Sweden and northern USSR and caribou (Rangifer tarandus caribou and R. t. groenlandicus) in Canada. Moose (Alces alces) are listed as hosts in Sweden, USSR and Norway. North American moose are susceptible.

Although E. cervi was first described from Scotland in 1931 there have been no confirmed recoveries since then. Protostrongylid larve have however, been seen frequently, but were attributed to another protostrongylid Bicaulus sagittatus. English et al. (1985) examined faeces from Scottish red deer, roe deer and reindeer. They recovered protostrongylid larvae from the faeces of all three deer species and in all cases they were within the size range for E. cervi and outside the size range for B. sagittatus. Three red deer and one roe deer were dissected but no adult E. cervi were found. They conclude that infection with E. cervi is widespread in Scottish deer.

Elaphostrongylus cervi has never been reported from fallow deer (Dama dama). During a survey of New Zealand deer farms (Mason and Gladden, 1983) fallow deer faeces were examined from 12 farms with negative results.

The recovery of <u>E. cervi</u> from roe deer is an unexpected finding. Generally it appears to be a parasite of <u>Cervus</u> spp. I would expect rusa (<u>C. timorensis</u>), sambar (<u>C. unicolor</u>) and Pere David's deer (<u>Elaphurus davidianus</u>) to also be susceptible.



Life Cycle

Adult <u>E. cervi</u> are slender worms, up to 60 mm in length usually found coiled in the connective tissue between muscle blocks or associated with the central nervous system. Females lay eggs which either hatch in situ or are carried to the lungs in the blood and then hatch.

Usually larvae migrate through the lungs to the air passages, up which they pass, are swallowed, and are voided by the host in the mucus coating on the faeces (Figure 1). The first stage larvae are tolerant of climatic conditions and can survive under natural conditions for over two years. Further development does not occur until a larva actively penetrates the foot of a suitable molluscan intermediate host.

In the intermediate host the nematode develops through the second larval stage to the infective third larval stage in 27-50 days, depending on temperature and molluscan species. Infective larvae can survive in molluscs for up to two years and can retain their ability to infect deer for this length of time. A deer becomes infected when it consumes a mollusc containing infective larvae. The larvae are released from the mollusc, by digestion in the stomach, and then burrow through the gut wall of their host. They then travel to their final site and at the same time develop into adults.

In New Zealand, Watson (1983) infected 17 E. cervi naive red deer calves with a single inoculation of 200 third stage E. cervi larvae. The prepatent period ranged between 107 and 125 days. Larval shedding was irregular during the first weeks following patency, rose during the first 10 weeks and then appeared to plateau. In a later paper, Watson (1986) reports that three 18-month-old calves given between 400 and 500 infective larvae reached patency between 86 and 98 days after infection. These results are consistent with the four month prepatent period reported by other authors. Lancaster (1977) however, reports from Canada that the prepatent period in an experimentally infected moose calf was 64 days, and 74 days in an experimentally infected caribou calf.

Adult E. cervi are long lived in deer and can produce eggs for over six years (Watson, 1984).

Intermediate Host Range And Larval Development

Elaphostrongylus cervi is not particular about its choice of intermediate host. Many terrestrial molluscs and even some from fresh water molluscs have acted as suitable hosts. The rate of development appears to be a function of temperature, infection level and intermediate host species.

Immune Response Of Host

Little is known about the immune response of deer to <u>E. cervi</u> infection. Because adult worms appear to live in deer for a long time it would be logical to assume that the host does not mount a significant immune response. Some recent work from Scandinavia suggests however, that some sort of immune response is generated. It is also possible that some species of deer are more capable than others of mounting an immune response.

Gaudernack et al. (1984) report that an indirect immunofluorescent technique demonstrated that sera from two reindeer in Norway infected with E. cervi contained antibodies against antigen(s) on the cuticle of first stage larvae. Output of first stage larvae increased during the rut in male reindeer, but was low outside the rut. The titre of specific antibody decreased during the rut. The authors suggest there is a relationship between stress, immunity and larval output i.e. when animals are stressed their immunity drops and larval output increases. The two periods of stress they identify are rutting and calving.

The results collected by Stuve (1986) suggest that moose can develop immunity to E. cervi. Although he found a high prevalance of infection in the younger age groups, he did not find any larvae in the faeces of animals over 5.5 years old.

Effects Of Infestation

Three types of clinical reaction to $\underline{E.\ cervi}$ infestation have been described:

- an acute disease characterised most commonly by paralysis of the hind limbs, resulting from the association of worms with the brain or spinal cord;
- a chronic disease with signs of ill thrift has been described in reindeer and attributed to connective tissue infestation with <u>E. cervi</u>;
- 3. a type of pneumonia induced by migration of larvae through the lungs.

In addition, adult <u>E. cervi</u> can produce lesions in deer carcasses which require trimming and may result in downgrading or condemnation of carcasses (Mason <u>et al.</u>, 1976; Watson and Charleston, 1985).

In New Zealand all natural infections in farmed deer have been light. Neither the acute nor the chronic diseases listed above have been seen here. Sutherland (1976) described the gross and histological lesions encountered in connective tissue, lymph nodes and lungs of infested red deer in New Zealand. Similar lung lesions attributed to E. cervi have been described from red deer in Scotland.

Elaphostrongylus cervi is a very common parasite of reindeer in Scandinavia and up to 100% of animals over 18 months old can be infected. It is reported to be one of the most significant parasitic diseases among semidomesticated reindeer. The parasite causes lesions in the central nervous system in some reindeer and eggs and larvae produce granulomatous parasitic pneumonias in almost all affected animals. In some heavily infected animals, parts of the carcass have to be trimmed at meat inspection.

A Russian author reported that deer up to 18 months old were most susceptible. The infection was subclinical during the warmer part of the year, but clinical signs appeared in winter.

The same author also reported that $\underline{E.~cervi}$ was the most pathogenic nematode on deer breeding farms in $\overline{eastern}$ Kazakh SSR. Sexually mature nematodes are localised in the brain, spinal column, under the dura mater, in its vessels, in the pia mater, ventricles, eyes, connective tissue of muscles and the peritoneum. They are most frequently found in the CNS of maral deer and in the muscles of $\underline{C.~nippon}$. Because of localisation in the CNS, $\underline{E.~cervi}$ is more pathogenic in maral deer. Clinical signs occur with heavy infections of the CNS - up to 390 worms were recovered from one animal. Molluscs are most heavily infected in autumn.

A survey of moose in southern Norway found a high prevalance of $\underline{E.\ cervi}$, suggesting a well established relationship between $\underline{E.\ cervi}$ and \underline{moose} . Although $\underline{E.\ cervi}$ had a negative influence on the general condition of the moose, the infection was not particularly pathogenic.

Elaphostrongylus cervi is one of the most common endoparasites of red deer in central Europe. It is found in the subdural and subarachnoidal cavities of the brain and spinal cord, yet so far no nervous signs have been reported. Inflammatory proliferative lesions caused by eggs and larvae in the lungs, and minor haemorrhages in the muscles in regions close to the worms, were detected quite often, but no damage was recorded from the cerebral parenchyma. Because <u>E. cervi</u> only causes minor problems, it was concluded that the European red deer <u>Cervus elaphus hippelaphus</u>) is the original host of E. cervi.

Prevalance And Intensity Of Infection

In some areas of the Altai region in USSR, 60-82% of Cervus elaphus and 52% of C. nippon were reported to pass first stage larvae of E. cervi. In C. elaphus the incidence rose from 45%(?) in 4-6 month old animals to 95-100% in 18 month old animals and dropped back to 71-86% in older deer. The respective figures for C. nippon were 10%, 60% and 62%. The numbers of larvae shed increased during the winter, but the porportion of animals shedding remained the same.

In a survey of reindeer faeces collected from six different summer ranges in Norway, <u>Elaphostrongylus cervi</u> was a common parasite in all six ranges. The prevalance of infection increased with increasing age of the reindeer. The highest incidences of infection were found on the ranges in the inner parts of the fiords, and they decreased towards the coast and towards the mountains. The inner part of the fiords contains the highest density of intermediate hosts and has the highest summer temperatures, and therefore the fastest development of the parasite to the infective stage.

In a study of E. cervi in moose in Norway, diagnosis was made on the presence of eggs, first stage larvae and associated histopathological lesions in the lungs, and on the presence of first stage larvae in the faeces. The prevalence was higher among males than females, and higher among yearlings than calves and adults. Carcass weight of affected adults was lower than in noninfected adults. No evidence of infection was found in animals 5.5 years of age or older. Stress associated with rutting may have been responsible for some of the difference in prevalence shown between males and females.

In a study of reindeer in Norway, there was a marked seasonal cycle in the output of E. cervi larvae, and this cycle was dependant on host sex. After an initial phase of logarithmic increase from the onset of patency in late winter/spring, the larval output declined to a minimum in summer in both male and female reindeer. From then onwards a regular cycle was repeated with a maximum density of larvae in autumn/early winter from males, and in late winter/spring from females. This seasonal cycle is probably linked to seasonal changes in the degree of host stress.

Factors Influencing Prevalance And Pathogenicity

The following factors appear to be involved in determining if E. cervi has pathogenic potential in a particular geographical locality.

- 1. Pathogenicity appears to be a function of infection level.
- Infection level depends on the availability of infected snails, therefore must be highest in areas where molluscs will be readily consumed and also where molluscs are easily infected.
- 3. Infection levels and pathogenicity appear to be highest in arctic areas and forested areas.
- 4. Pathogenicity and infection levels are low in New Zealand and Scotland.
- 5. The heaviest natural infections in New Zealand have been seen in deer taken from the wapiti block in Fiordland. Conditions in this part of the country would be similar to those of the inner fiords in Norway where heavily infected reindeer were found.
- 6. Infection levels are unlikely to ever become high in deer that are grazing pasture.
- 7. The larval output of infected deer increases when they are stressed.

In the context of exporting deer from New Zealand to Australia, deer would be under stress when placed in quarantine.

Host Specificity

Elaphostrongylus cervi, unlike the related Parelaphostrongylus tenuis does not appear to constitute a significant threat to noncervid species.

Control

There have been various claims in the literature for successful treatment of E. cervi infection in deer, but I am rather sceptical about the significance of these results. In many cases there is little or no description of an experimental design and where this is provided, success in measured by the cessation of larval shedding following treatment but without any follow-up to ensure that larval shedding did not resume.

Watson (1986) reports on the results of field trials evaluating three different treatment regimes: oxfendazole at 9.0 mg/kg on two occasions 48 hours apart; oxfendazole at 9.0 mg/kg on three consecutive days, and one subcutaneous injection of ivermectin at 200 micrograms/kg. All three treatments reduced larval shedding to very low levels, but did not completely eliminate larval shedding from any group.

Muellerius is related to E. cervi, so the following report may be relevant. McCraw and Menzies (1988) reported that goats stopped shedding Muellerius capillaris larvae after treatment with ivermectin, but that larvae began to reappear in the faeces of treated goats after variable periods of time even when the goats were held in strict isolation with no access to the intermediate host of Muellerius. They suggested that ivermectin was killing the adults, but not (arrested?) immatures which then resumed development to maturity and the production of larvae in faeces. In support of this contention they found dead adult nematodes in lung sections, and some goats resumed larval shedding within the prepatent period of Muellerius. This suggests repeated infection of goats was occurring to provide immatures, and this was supported by their observations that older goats tended to have heavier infections and a greater proportion of older goats resumed shedding.

Diagnosis

Elaphostrongylus cervi infection can be diagnosed by finding either adult worms in the carcass, or larvae in the faeces, tracheal smears or lung washings of the suspected host.

Adult worms are difficult to find in a deer carcass — somewhat akin to finding a needle in a haystack. Bye and Halvorsen (1984) report finding only two worms in a reindeer they believe was killed by E. cervi!

The most usual technique for diagnosing the presence of $\underline{E.\ cervi}$ in live animals is by the examination of faeces for first stage \underline{Iarvae} using modifications of the Baermann technique. Tracheal smears and lung washings are not used in New Zealand – firstly because they cannot be easily standardised or quantified and secondly because we cannot find volunteers to collect the samples!

Basically, the Baermann method consists of suspending the faeces in water in such a way that the \underline{E} . cervi larvae, when they wriggle out of the mucus coating on the faecal pellets, drop to the bottom of a container from where they can be retrieved and counted.

In common with many parasitological techniques, the specificity of these methods is high. When used by qualified staff they should have a false positive rate of zero.

Sensitivity however, is extremely difficult to measure. Theoretically, sensitivity is calculated by dividing the minimum number of larvae that can be recovered (one) by the weight of faecal material examined. In the case of \underline{E} , cervi larvae the theoretical sensitivity is probably greater than the true sensitivity.

Theoretical sensitivities then, are as follows:

Weight Of Faeces (g)	Sensitivity (L1/g)
2	0.5
5	0.2
10	0.1
12	0.08
15	0.07
20	0.05

Optimal sample size is a function of the apparatus used and the weight of faecal sample examined. If the sample weight is too low and sample variability is too high then the sample examined may not be representative for that animal. If sample weight is too high then yield decreases, presumably due to bulk effects resulting in larvae becoming trapped in faeces. With the apparatus generally used, the lower limit is around 8 grams and the upper limit around 20 grams. Allowing for a safety margin either way the N.Z. parasitologists have decided to go for 15 gram faecal samples.

The critical question of course is 'Can it be absolutely guaranteed that a deer is completely free of \underline{E} . cervi'. Unfortunately, the answer to this must be no. Larval output in light infections is very low and variable. The best chance to turn up positive animals would appear to involve sequential sampling from animals under stress. Both quarantine and sequential sampling would provide stress.

Elaphostrongylus cervi In New Zealand

Elaphostrongylus cervi was first identified in New Zealand in 1975 in both red deer (Mason et al., 1976) and wapiti (Mason and McAllum, 1976). In both cases the deer came from Fiordland in the south western corner of the South Island.

In the first case, 21 culled red deer out of a line of 91 were condemned as unfit for human consumption because of marked green discolouration of the muscle fascia around the shoulders, flanks and loins resulting from infestation with $\underline{\text{E. cervi.}}$ Farmed deer do not have such extensive or severe lesions.

In July 1981, deer faecal samples collected from 115 farms from throughout New Zealand were examined for evidence of parasitism (Mason and Gladden, 1983). Elaphostrongylus cervi larvae were recovered from 16 farms in the North Island and 22 farms in the South Island (35% of the farms with red deer and/or wapiti and their hybrids). There was a highly significant relationship between the presence of E. cervi and the introduction of deer from Southland/Fiordland (p < 0.001 by chi-square test). Infection was more prevalent in adults than calves. Mean larval output ranged up to 20.2 L1/g, but on 81% of the properties with E. cervi mean larval count was < 1 L1/g.

The first recovery of <u>Elaphostrongylus cervi</u> in New Zealand was from deer from the Fiordland area. The infection levels in some of these animals were high. The level of infection in farmed deer is generally low. This indicates that conditions are much more suitable for the transmission of <u>E. cervi</u> in Fiordland than they are on farms.

The stocking rate of wild deer is unlikely to be higher than the stocking rate on farms. But the stocking rate in Fiordland now is probably considerably lower than it was in the early 1970's. The most obvious difference between farm land and Fiordland is in the nature of the vegetation. On farmland the predominant vegetation is developed grassland. Fiordland on the other hand is a mountainous area of native vegetation—native bush grading into alpine tussockland—with a high rainfall. In this environment, accidental ingestion of molluscs would be common. On farms by contrast, accidental consumption of molluscs would be a rare event.

Elaphostrongylus cervi In Australia

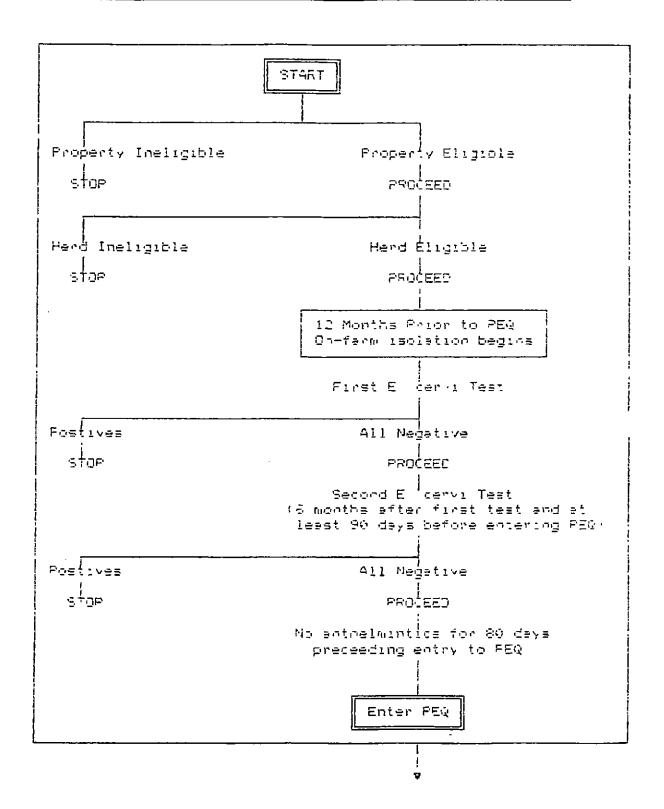
There are no published records of E. cervi being found in deer in Australia, except in imported animals in quarantine (Presidente, 1986).

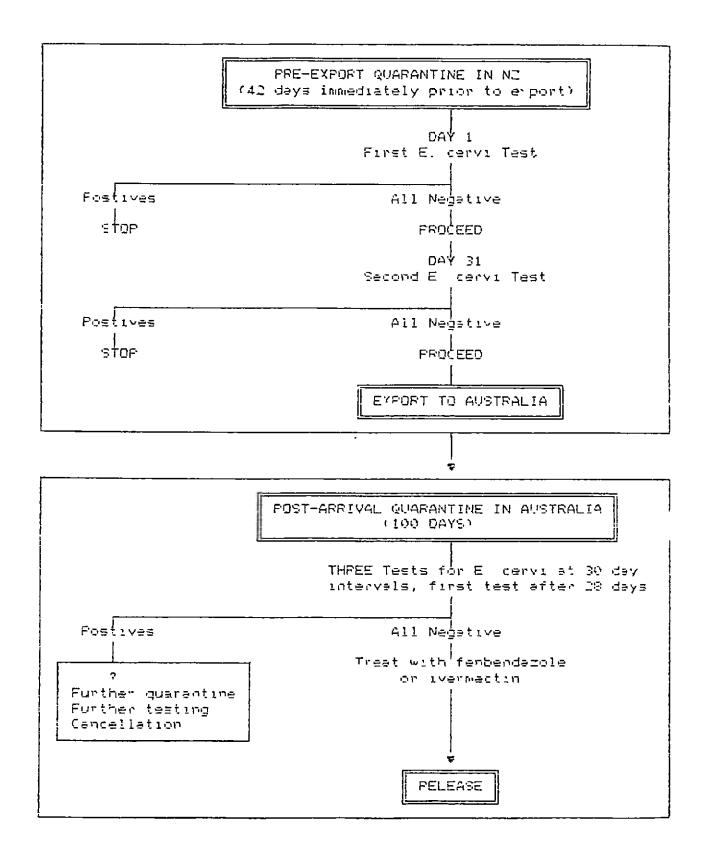
Consequently, the protocol for importation of deer into Australia from New Zealand is designed among other things, to minimise the possibility of importing E. cervi into Australia. The flow diagrams in Figures 2 and 3 set out the procedures designed to minimise the chance of exporting infected deer. These procedures are taken from the April 1989 protocol for export of deer to Australia.

Concluding Remarks

Elaphostrongylus cervi is a parasite of deer which is unlikely to be of any significance in farmed deer on grazing land. It is important only because it can prevent the export of deer from farms where it is present.

ELAPHOSTFONGYLUS CERVI & EXPOPT OF DEER TO AUSTRALIA





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