EXPERIMENTAL INFECTION OF FALLOW DEER (*DAMA DAMA*) WITH ELAPHOSTRONGYLINE NEMATODES (NEMATODA: PROTOSTRONGYLIDAE) FROM CARIBOU (*RANGIFER TARANDUS CARIBOU*) IN NEWFOUNDLAND

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ABSTRACT: Five fallow deer fawns (*Dama dama*) were given 25 to 150 infective larvae of elaphostrongyline nematodes originating from wild caribou (*Rangifer tarandus caribou*) in Newfoundland. The inoculum contained infective larvae of both *Elaphostrongylus cervi* (*E. c. rangiferi* in the sense of Pryadko and Boev 1971) and *Parelaphostrongylus andersoni* Prestwood, 1972. No animal showed clinical signs of disease.

At necropsy, all exposed deer exhibited a mild, to focally intense, eosinophilic meningitis indicating helminthic invasion of the CNS. A male *E. cervi* and fragments of a female nematode were recovered from the brain and spinal cord, respectively, of one animal. Fragments of nematodes identified as *P. andersoni* were found in the longissimus dorsi muscles of this same animal as well as in one other. Three deer, including the two in which worms were found, passed dorsal-spined larvae in their feces, 69-75 days after infection. Larvae were 331-410μ long ($\bar{x}$=378μ).

Apparently *E. cervi* can migrate as far as the CNS of fallow deer. Results also reveal, for the first time, that fallow deer are suitable hosts of *P. andersoni*, a muscleworm which is widely distributed in wild cervids of North America.

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Fallow deer (*Dama dama*) have been raised for many years in Canada in private and municipal zoos and game parks. Most of the original stock probably came from the United Kingdom. During the past few years there has been considerable renewed interest in obtaining large numbers of fallow deer as commercial livestock. Several thousand animals have recently been imported from New Zealand, mainly into the western Canadian Provinces of Saskatchewan and British Columbia, by the rapidly growing game ranching industry.

The importation of cervids from New Zealand raises concerns about the possibility of introducing parasites, one of which is *Elaphostrongylus cervi*. This nematode migrates into the central nervous system and can be found in the subdural space surrounding the spinal cord and brain. Severe neurologic disease with paralysis can result. *Elaphostrongylus cervi* occurs in red deer (*Cervus elaphus elaphus*) and wapiti (*C. e. canadensis*) in New Zealand (Mason *et al.* 1976, Mason and McAllum 1976) but, according to Mason and Gladden (1983), fallow deer have not been found to pass larvae. Sugar (1978), on the other hand, reportedly found *E. cervi* in fallow deer in Hungary. In contrast, *E. cervi* was not listed as a parasite of fallow deer in the USSR and Czechoslovakia where other deer species are infected (Pryadko 1976, Kotrla and Kotrly 1977). It is not clear from the literature, therefore, whether fallow deer from New Zealand can potentially introduce *E. cervi* into western Canada.

Elaphostrongylus cervi (*E. c. rangiferi* in the sense of Pryadko and Boev 1971) is already known from caribou (*Rangifer tarandus caribou*) in Newfoundland (Lankester and Northcott 1979). The worm probably was introduced there with infected reindeer (*R. t. tarandus*) brought from Norway in 1908 (Lankester and Fong 1989). It has not been found anywhere else in Canada. In Newfoundland, *E. cervi* causes severe neurologic disease in wild caribou (Lankester and Northcott 1979, Lankester and Fong, unpubl.). It also has been shown to cause posterior paralysis in an experimentally infected moose calf (*Alces alces*) (Lankester 1977) but natural infections in moose of Newfoundland have not been reported.

Larvae used to infect fallow deer in the present study originated from caribou feces collected off range in central Newfoundland. Larvae from the same source were used previously to produce experimental infections of *E. cervi* in caribou and moose (Lankester 1977). Recently, however, Lankester and Fong (1989) found adult specimens of a second elaphostrongyline nematode, *Parelaphostrongylus andersoni*, in the back and limb muscles of caribou of Newfoundland. The wide distribution of this rather inconspicuous parasite is only recently being recognized. It was initially reported as a parasite of white-tailed deer (Prestwood *et al.* 1974, Pursglove 1977, Pybus and Samuel 1981, Pybus *et al.* 1990). Now, it also is known to occur widely in caribou of central and northern Canada (Lankester and Hauta 1989). Adult worms cause haemorrhage and myositis in muscles of the back and upper hind legs (Pybus 1983, Pybus and Samuel 1984). Most importantly, with respect to the present experiment, the first-stage larvae of *P. andersoni* cannot be reliably distinguished from those of *E. cervi*. It was recognized, therefore, that larvae used to infect fallow deer might include both species.

METHODS

The Baermann technique was used to isolate dorsal-spined nematode larvae from caribou feces collected off range in central Newfoundland (Topsails and Middle Ridge herds). Laboratory-reared snails (*Triodopsis albolabris* and *Mesodon thyroidus*) were allowed to crawl over filter paper inoculated with the larvae. Snails were fed lettuce and chalk and held in terraria at 20°C for 3 to 9 months before being digested in artificial pepsin solution to recover third-stage, infective larvae.

Fallow deer fawns were given infective larvae in saline by using a nursing bottle or by squirting larvae from a pipette into the back of the pharynx. They were approximately 6 weeks old and weighed 7.7-11.8 kg when infected. Five fawns were each given from 25 to 150 larvae; a sixth was retained as a control. Fawns were separated from their dams a few days after birth and bottle-fed for about 12 weeks before being weaned onto pelleted alfalfa rations and hay.

The fawns were observed daily for signs of disease. Feces collected daily, beginning 50 days post-exposure (DPE), were examined for larvae using the Baermann technique. Larvae were heat-fixed and measured using a drawing-tube. At various intervals after exposure, animals were sedated with xylazine hydrochloride (Rompun) and euthanized with sodium pentobarbitol (Euthanyl).

Necropsy procedure included skinning the animal and examining the underside of the skin and the surface of the skeletal musculature for worms. All major muscles were separated and the surface examined. Nerves, fascia and muscles in the axillary and inguinal regions were examined. The longissimus dorsi and psoas muscles were sliced at a thickness of 5-8 mm and the cut surfaces of each piece examined and teased apart with forceps under a stereoscopic microscope at 10X. The larger muscles of the thighs were examined in the same manner. The brain and

spinal cord were removed. The dura was resected and the subdural surfaces were examined at 6X. The entire brain and spinal cord was cut into several sections and each teased apart at 6X in search of nematodes. The vertebral canal was examined at 6X while teasing away accumulations of epidural fat around intervertebral fossae. Selected tissues were fixed and prepared for histological examination using standard procedures.

RESULTS

No clinical disease was observed in any of the fallow deer involved in the experiment. Animals reached weights of 20-24 kg at 16 to 18 weeks of age. The control deer had no visible lesions.

Recovery of Larvae and Worms in Tissue

Three of the 5 exposed animals passed dorsal-spined larvae in their feces, beginning at 69, 69, and 75 days post exposure (Table 1). Larvae passed by two of the patent deer measured 365-400μ long ($\bar{x}$=382±11, n=15) and 331-410μ long ($\bar{x}$=373±22, n=15). The number of larvae in feces of patent animals rose gradually, reaching 0.5 - 7.3 larvae per g (wet weight) at the time deer were killed (Table 1).

Nematodes were recovered from the tissues of only two deer (Nos. 1 and 2). In one, a worm measuring 130 to 143μ in diameter was seen in histological sections of the right longissimus dorsi muscle (Fig.1). An immature adult male worm, 14.1 mm long and 122μ wide, was found in the subdural space over the right olfactory lobe of the second deer. The worm moved slowly in saline and cells adhered to the cuticle. The spicules were only lightly sclerotized and were 155μ long; the gubernaculum was not visible. The esophagus was 530μ long. In the same deer the posterior end of an immature adult female worm was found near the base of the ventral nerve root at T_3 (3rd thoracic vertebra). As well, deep in the longissimus dorsi muscle, broken pieces of one or two more nematodes were recovered. The fragments were 110-140μ wide and the esophagus was 910μ long.

Gross and histopathology

Deer No.1 - At necropsy, numerous pin-point and ecchymotic haemorrhages were seen in the fascia over both longissimus dorsi muscles. In the CNS, an enlarged, reddish lymphoid nodule was visible beneath the dura, mid-ventrally on the cord at C_7 (7th cervical vertebra). A small amount of yellowish, subdural exudate was present at T_1.

In histological sections of the longissimus dorsi muscle, accumulations of leukocytes, primarily eosinophils, were seen in inter-

Table 1. Nematodes recovered from fallow deer infected with elaphostrongyline larvae originating from caribou feces collected in central Newfoundland.

Deer no.	Killed (DPE)[1]	No. infective	Prepatent period	LPG*	Muscle haemor.	Inflam. CNS	Worms
1	78	55	69	2.44	+	+	worms(s) in muscle
2	108	75	69	7.32	+	+	2 frag. in muscle, 2 worms in CNS
3	109	125	-	-		+	none found
4	109	150	75	0.50	+	+	none found
5	223	25	-	-		+	none found
6	78	control	-	-		-	none found

[1]Days post-exposure

*Larvae per gram of wet feces on day of necropoy. Larvae passed were 311-410μ long.

muscular fascia along with lymphatic dilatation and diffuse haemorrhage. Small numbers of eosinophils were seen around a nematode deep in the muscle mass (Fig. 1) and around a nerve. Moderate, multifocal, lobular congestion along with focal atelectasis and numerous nematode larvae were seen in random lung samples (Fig. 2). In the CNS there was focal, mild eosinophilic and lymphocytic meningitis with haemorrhage at the base of the optic nerves. Around the ventral nerve root at C_7, there were focal accumulations of lymphocytes and axonal degeneration evident in one nerve bundle. Perineural haemorrhage was seen at T_1. Active haemorrhagic lymphoid follicles at T_9 and T_{10} had intense accumulations of eosinophils at their periphery.

Deer No.2 - A dark red area of haemorrhage (0.5x0.5 cm) occurred deep in the left longissimus dorsi near the dorsal process of the 3rd lumbar vertebra; a larger haemorrhagic area (1.5x3.0 cm) was present in the mid-region of the right longissimus dorsi. In the CNS, small amounts of greenish-yellow, subdural exudate were present over the right olfactory lobe (where the male nematode was found) and along the spinal cord near ventral root ganglia at C_4 and T_1-T_5. A portion of an immature female worm was found at T_3 near the ventral nerve root. There was extensive epidural haemorrhage ventral and lateral to the cord in the region of L_4-L_6 and white granular lesions throughout the epidural adipose tissue at L_6.

Histologically, there were focal areas of intense eosinophilic and lymphocytic inflammation with haemorrhage and myonecrosis, and granuloma formation around larval nematodes in the trapezius, psoas, and longissimus dorsi muscles (Fig. 3). Lung lesions were small and multifocal with some lobular atelectasis, perivascular haemorrhage and vasculitis, and lymphocytic aggregations with focal eosinophilic interstitial pneumonia.

A mild meningitis was seen over the brain with eosinophils in areas of diffuse haemorrhage. Focal encephalomalacia with brown pigment-filled macrophages was seen in the anterior cerebral cortex. Mild diffuse, to focally intense eosinophilic and lymphocytic infiltration of the meninges and perineuronal tissue occurred near the base of nerves C_1, T_1, and along the brachial plexus in the subscapular region. Epidural fat at the level of L_6 had moderate to intense haemorrhage with eosinophilic and lymphocytic inflammation, and areas of necrosis (saponification).

Deer No.3 - In the cranium, a small area (3 mm diam.) of yellowish exudate was visible on the ventral surface of the right and left pyriform lobes. Creamy, white to yellow accumulations were present over the pons and on the ventral surface of the medulla. Individual lymphoid nodules adhered to the spinal cord, mid-dorsally at C_5, mid-ventrally at C_6, and laterally near the ventral nerve root at T_5. Gelatinous, pinkish-yellow, epidural accumulations occurred ventrally along the cord and around all nerve roots from C_7 to T_2. Yellowish fluid and discrete, bright yellow spots (1-2 mm) were present on the ventral surface of the cord in this region. There was diffuse, greenish-yellow inflammation of the perineural tissues of the sacral spinal cord and cauda equina.

There was a mild to moderate meningeal inflammation with eosinophils, some hemosiderin-ladened macrophages and free red blood cells over the pons and medulla. No tissue damage or inflammation was evident in brain parenchyma. Along the spinal cord at C_5, T_2, and T_5, discrete lymphoid follicles occurred in areas with moderate to intense eosinophilic accumulations in the meninges and between the nerve root bundles. Subdural, lymphoid follicular development and a diffuse, moderate to severe, eosinophilic meningitis and perineuritis was seen over much of the cauda equina (Fig. 4).

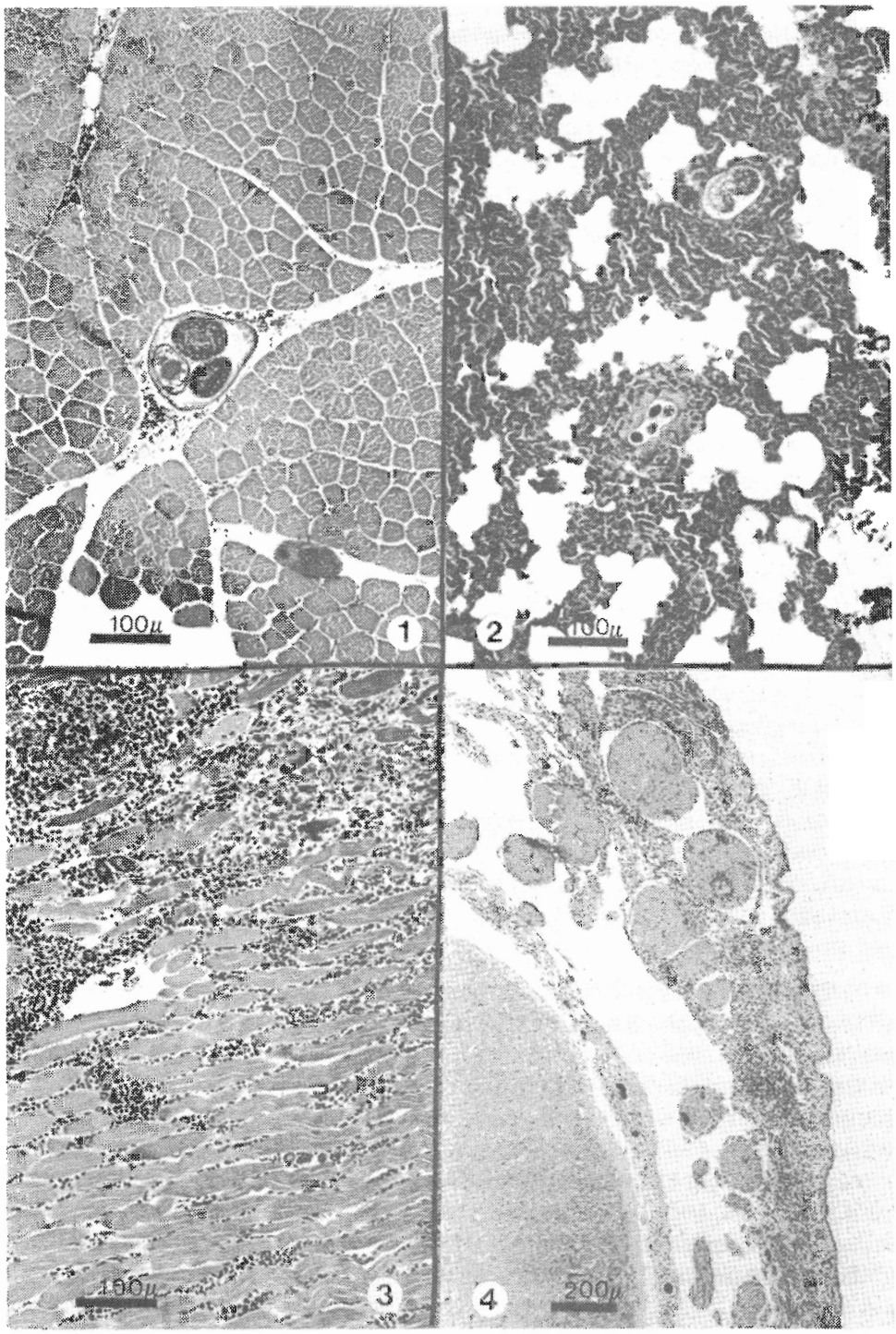

Fig. 1. Mild infiltration of eosinophils around a nematode deep within the longissimus dorsi muscle.

Fig. 2. Nematode larvae within lung associated with areas of congestion and atelectasis.

Fig. 3. Intense eosinophilic and lymphocytic accumulation within muscle tissue.

Fig. 4. An intense eosinophilic meningitis and perineuritis surrounding the cauda equina.

Deer No.4 - A focal area of haemorrhage (1x2 cm) was seen deep in the left longissimus dorsi muscle at the level of vertebrae 10 and 11 with smaller haemorrhages in the left quadriceps and semimembranosus. Numerous petechiae (0.5-1.5 mm) were visible on the fascia over the right and left longissimus dorsi muscles. In the CNS, there were 2 subdural lymphoid nodules (2 mm diam.) at the level of C_1 and yellowish subdural exudate at C_4. Small, firm, whitish epidural fat nodules were seen dorsally in the spinal canal at the level of C_5. There were single to multiple, small (2-3mm) subdural lymphoid nodules at C_1, C_2, C_7, T_7, and L_7 and greenish yellow meningeal inflammation at C_4, C_7, T_1, and T_8. Accumulations of greenish-yellow subdural exudate and a cluster of 5 small lymphoid nodules were seen among terminal filaments of the cauda equina.

Tissue samples from superficial muscle adjacent to S_2 and from the lower right leg showed moderate to severe eosinophilic and lymphocytic myositis. Along the cord at C_4, C_7, and T_1, there was mild neural root infiltration and severe meningeal and interstitial eosinophilic inflammation. There was also an intense eosinophilic and lymphocytic inflammation around active lymphoid follicles and around the lateral nerve roots.

Deer No.5 - In the CNS, a large, yellowish-pink lymphoid nodule (12x5 mm) was found at the base of the nerve root at C_6 with numerous smaller nodules (2-3 mm) in a similar location at T_1 where the pia-arachnoid was also thickened and opaque. At the posterior extremity from L_7 to S_6, three small (2 mm), subdural lymphoid nodules occurred near the base of nerve roots and golden, granular accumulations were visible in the pia-arachnoid.

Along the spinal cord at C_6, intensive lymphoid follicular activity was organized around 2 central foci of mineralized debris. At T_1 there was an eosinophilic meningitis with marked congestion of lymphoid follicles and a diffuse eosinophilic and lymphocytic perineuritis of the lateral nerves.

DISCUSSION

Results indicate that some nematodes reached the central nervous system (CNS) of all 5 experimentally infected fawns. The morphometrics of 2 worms recovered from the CNS of one animal were consistent with an identification as *E. cervi*. The male worm was similar in size to a male *E. cervi* recovered by Lankester (1977) from the CNS of an experimentally infected caribou killed after 113 days. The spicules were only lightly sclerotized making their total length difficult to discern. Nonetheless, they were only slightly shorter (155μ) than those of mature *E. cervi* (187-231μ) and larger than those of *P. andersoni* (87-130μ) (Lankester and Hauta 1989).

It is believed that *E. cervi* undergoes its initial development in the CNS of cervids, migrating later into skeletal muscles of the chest and limbs (Anderson 1968, Lankester1977). In an experimentally infected caribou calf, 1 of 5 worms recovered after 113 days was found along brachial nerves in the axilla while the remainder were still in the cranium or vertebral canal. In an experimentally infected moose calf killed after 130 days, 18 worms were in the axillae while 18 were still in the CNS. In fallow deer killed 78-223 days after infection, there was no indication that any *E. cervi* had migrated to the axillary region.

Histopathological lesions seen in the CNS of fallow deer were similar to those in reindeer and caribou in which *E. cervi* matures (Roneus and Nordkvist 1962, Lankester and Northcott 1979). Similar lesions were described in calves and lambs given *E. cervi* (see Bakken *et al* 1975). All calves and lambs had eosinophilic leptomeningitis but none showed neurologic disease and no mature worms or larvae in feces were found.

Fragments of nematodes found deep in

the longissimus dorsi muscles of two of the three patent fallow deer resembled *P. andersoni*. In particular, the esophagus was 910μ long, similar to that of female *P. andersoni* (640-882μ for males, 625-1062μ for females)(max. range, several sources, see Lankester and Hauta 1989) and considerably longer than the esophagus of *E. cervi* (420-577μ for males, 492-610μ for females). All three patent deer with larvae in their feces had haemorrhage and eosinophilc myositis in loin muscles.

It can be concluded that *P. andersoni* larvae were passed by infected fallow deer but whether some *E. cervi* larvae might also have been passed cannot be determined. The prepatent period of *P. andersoni* is 51-69 days in white-tailed deer (Pybus and Samuel 1981, 1984). Fallow deer in this study first passed larvae 69, 69 and 75 days after infection. *Elaphostrongylus cervi* requires a longer period before larvae appear in feces (86-125 days, Watson 1983, 1986; 3 to 4 months, Panin 1964, Mitskevich 1964). The longest surviving deer was not killed until 223 days after infection but it never passed larvae. Two of the patent deer were killed 108 and 109 days after infection. They lived long enough that some of the larvae in their feces could conceivably have been *E. cervi* but larvae of the two species are not distinctive. The first-stage larvae passed by fallow deer (331-410μ long) were most similar in length to those of *P. andersoni* (308-382μ, Prestwood 1972; 319-412μ, Lankester and Hauta 1989). Larvae of *E. cervi* tend to be longer (mean length >400μ) than those of *P. andersoni* (see Lankester and Hauta 1989) but the ranges of measurements overlap considerably.

In summary, results indicate that *Elaphostrongylus cervi* and *Parelaphostrongylus andersoni* were both present in the infecting doses and both developed to varying degrees in fallow deer. *Parelaphostrongylus andersoni* caused haemorrhage and myositis and probably became patent in 3 deer. Knowing that *P. andersoni* can mature in fallow deer is important to wildlife managers and game ranchers. The larvae could be mistaken for those of *P. tenuis*. Fallow deer contracting *P. tenuis* from white-tailed deer in eastern North America exihibit fatal neurologic disease (Kistner *et al.* 1977, Nettles *et al.* 1977). However, some fallow deer, possibly those ingesting only small numbers of larvae, survive infection and may become immune (Davidson *et al.* 1985). Dorsal-spined larvae have not been found in feces of fallow deer infected with *P. tenuis*.

All 5 fallow deer infected in the present study had eosinophilic meningitis indicating helminth invasion of the CNS but *E. cervi* could only be found in the CNS of one. Results of the experiment do not permit a conclusion as to whether or not *E.cervi* can reach patency in fallow deer. For these reasons, further study is required to evaluate the likelihood of fallow deer introducing the parasite into western Canada.

Recently, Steen *et al.*(1989) described morphological differences in the bursa of *Elaphostrongylus* which they believe distinguish three distinct species in cervids of Sweden; *E. cervi* Cameron,1931 in red deer, *E. rangiferi* Mitskevitch, 1958 in reindeer, and *E. alces* Steen *et al.*, 1989 in moose. The form of *E. cervi* used here from caribou in central Newfoundland originated in reindeer from Norway while the form present in New Zealand cervids originated in red deer from the United Kingdom. Future experiments should therefore expose fallow deer to both forms of *E. cervi* and precautions should be taken to avoid using infective material contaminated with *P. andersoni*.

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