The Seventh North American Caribou Conference,
Thunder Bay, Ontario, Canada,
19–21 August, 1996.

Protostrongylid nematodes in caribou (*Rangifer tarandus caribou*) and moose (*Alces alces*) of Newfoundland

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Abstract: Two species of protostrongylid nematodes with dorsal-spined, first-stage larvae, are present in caribou and moose of Newfoundland. *Elaphostrongylus rangiferi* Mitskevich, 1958, a parasite introduced from Scandinavia, causes periodic epizootics of a severe neurological disease in caribou. Sick animals exhibiting signs of cerebrospinal elaphostrongylosis (CSE) were particularly noticeable in central Newfoundland each winter between 1981 and 1985. Those collected for examinarion were mostly male calves. The disease again became prominent in caribou on the Avalon Peninsula in the winters of 1996 and 1997; it may have spread to that isolated part of the province as recently as 1990. *E. rangiferi* was also found in moose but no cases of neurologic disease have been reported in this host. *Parelaphostrongylus andersoni* Prestwood, 1972, was found in caribou, both in central Newfoundland and on the Avalon Peninsula. Moose may also be infected. Of 1407 terrestrial gastropod intermediate hosts examined, 9 (0.6%) contained infective, third-stage, protostrongylid larvae resembling those of *E. rangiferi* and *P. andersoni* which are indistinguishable. The small dark slug, *Deroceras laeve*, dominated gastropod collections and was the only species infected.

Key words: cerebrospinal elaphostrongylosis, muscle worms, lungworms, cervidae, gastropod intermediate hosts.

Rangifer, Special Issue No. 10, 73–83

Introduction

Elaphostrongylus rangiferi has been known for some time from caribou (*Rangifer tarandus caribou*) in Newfoundland (Lankester, 1976; 1977; Lankester & Northcott, 1979) and was probably introduced with reindeer (*R. t. tarandus*) brought from Norway in 1908 (Lankester & Fong, 1989). In earlier publications, we followed European authors who recommended that this nematode found in the central nervous system and musculature of *Rangifer* be considered a synonym of *E. cervi* (see Kutzer & Prosol, 1975) or be referred to as *E. cervi rangiferi* (Pryadko & Boev, 1971; Kontrimavichus *et al.*, 1976). However, we now defer to Scandinavian workers (Stéen *et al.*, 1989; Halvorsen *et al.*, 1989; Gibbons *et al.*, 1991) who have provided new biological and morphological data supporting distinct species status for *E. rangiferi*.

In Scandinavia and northern Russia, the parasite causes a disease called cerebrospinal elaphostrongylosis (CSE) which is characterized by a lack of fear, ataxia, and posterior paralysis (Polyanskaya, 1965; Bakken & Sparboe, 1973; Handeland & Norberg, 1992). Heavy losses of young animals less than one year old periodically occur in late winter. Domestic sheep and goats that share range with infected reindeer may also succumb to the disease (Handeland, 1991; Handeland & Sparboe, 1991). In Newfoundland, *E. rangiferi* was prevalent in caribou of the Middle Ridge area in the mid 1970s when the first case of CSE was diagnosed (Lankester & Northcott, 1979). There have been opportunities for *E. rangiferi* to spread with translocated reindeer and caribou from Newfoundland to mainland Canada but there is as yet no conclusive evidence that it has become established anywhere outside of Newfoundland (Lankester & Fong, 1989).

Parelaphostrongylus andersoni, another protostrongylid nematode, is widely distributed in woodland and barrenground caribou of mainland Canada

(Lankester & Hauta, 1989). This slender nematode is easily overlooked because of its location deep within muscles of the back and hind limbs. If, as Lankester & Hauta (1989) suggested, caribou are the original host of *P. andersoni,* rather than white-tailed deer (the type host, see Prestwood, 1972), we predict that it should also occur in caribou of Newfoundland along with *E. rangiferi.* Because *P. andersoni* probably is not neurotropic (Pybus & Samuel, 1984), it is not thought to cause neurologic disease in wild cervids. However, its eggs and larvae, like those of *E. rangiferi,* develop in the lungs where an intense granulomatous inflammatory reaction contributes to verminous pneumonia (Lankester & Northcott, 1979, Anderson & Prestwood, 1981).

The first indication that caribou in Newfoundland might have both *E. rangiferi* and *P. andersoni* was provided by Lankester *et al.* (1990) following experimental infection of fallow deer (*Dama dama*) with larvae collected off Newfoundland caribou range. First-stage larvae were first passed 69 days after infection which is consistent with the shorter prepatent period of *P. andersoni,* and fragments of worms resembling both species were recovered at necropsy. Lankester & Fong (1989) mentioned finding *P. andersoni* in naturally infected caribou from Newfoundland but specimens were not described.

The purpose of this paper is to document the extent of *E. rangiferi* infection in cervids and its role in an epizootic of neurologic disease seen in the early 1980s in caribou of central Newfoundland. We also provide dimensions of *E. rangiferi* collected from moose and of *P. andersoni* from caribou in Newfoundland, and investigate the role of terrestrial molluscs in the field transmission of these parasites.

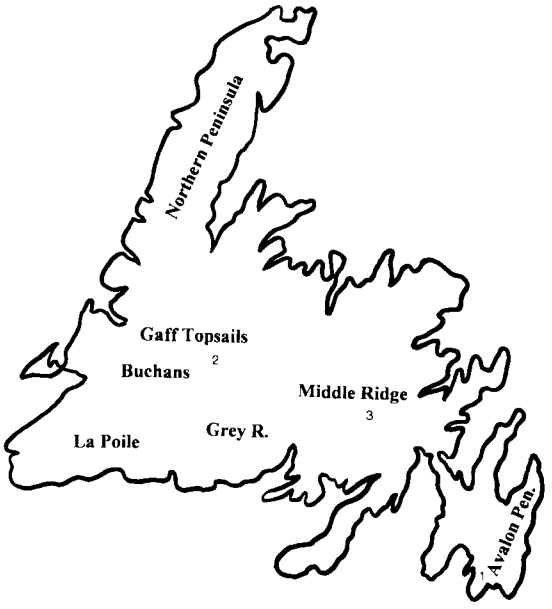

Fig. 1. Map of Newfoundland indicating the general areas occupied by major caribou herds from which sick caribou, moose, and fecal samples were collecred. Locations 1- 3 are sites where rerrestrial gastropods were collected: site 1 - Avalon Peninsula (Peter's River Rd., 46°47'N 54°10'W, 24 - 29 May, 1984); sites 2 & 3 - Central Newfoundland (sire 2., adjacent to the Buchans Hwy #370 between Buchans and Buchans Junction, 48°37'N 57°26'W, 30 May - 3 June, 1984; and site 3., Sandy Pond, 48°05'N 55°42'W, 7 - 20 July, 1987).

Materials and methods

Examination of caribou exhibiting neurological signs

Animals behaving in an abnormal way were collected opportunistically and the body musculature of the chest and limbs was inspected visually for nematodes in the field. The head and vertebral column were removed and frozen along with a fecal sample, until examined later in the laboratory. The tops of the cranium and vertebrae were removed using a Stryker surgical saw. The brain and spinal cord were removed, and the surface and surrounding meninges checked for nematodes using a ring-lamp magnifier. Feces were examined for nematode larvae using the Baermann funnel technique.

Herd infection levels determined by fecal examination

Caribou feces were periodically collected off snow over several years (1982-90) from the traditional wintering areas of 7 major caribou herds in Newfoundland (Fig. 1). Samples were kept frozen at -15 °C until examined using the Baermann funnel technique. Pellets were floated over Kimwipe tissue (Kimberly-Clarke, Mississauga, Ontario) in stoppered, water-filled funnels (15 cm top diameter) for 24 hr, after which time 20 ml were drained into a Syracuse watch glass and examined for protostrongylid larvae at 20X using a stereoscopic microscope. First-stage larvae were pippetted onto a slide, heat relaxed on a hot-plate, covered with a cover slip and drawn and measured using a Wild drawing tube at 400-1000X.

Fig. 2. Male caribou calf with posterior paresis caused by *Elaphostrongylus rangiferi* appeared unafraid of humans and could be easily approached and restrained.

Examination of wild moose

Eight moose were shot and field examined, May 7-10, 1990; 7 (2-10 yr old) in the area occupied by the Middle Ridge caribou herd and a male calf (11.5 mo) in the area of the Gaff Topsails herd (Fig. 1). The fascia and surface of muscles beneath the shoulders were examined for nematodes. Representative nematode specimens were fixed in glycerin-alcohol and later drawn and measured. Fecal samples (22-30 pellets) taken from the rectum were frozen and later examined for larvae. Additional moose feces were collected off range in the Gaff Topsails area, March 15, 1989, frozen, and examined later for nematode larvae using the Baermann funnel technique.

Searching for P. andersoni

Two caribou were shot on the Avalon Peninsula and 2 in the Gaff Topsails area, June 23-30, 1987. Eight more were examined from the Avalon Peninsula in the vicinity of Peter's River Road, December 9-13, 1989. The cranium and shoulder muscles were examined in the laboratory for *E. rangiferi* according to Lankester & Northcott (1979) and the longissimus dorsi muscles of the back for *P. andersoni* according to Lankester & Hauta (1989). Feces were collected from the rectum of each animal.

Detecting infections in intermediate hosts

Terrestrial gastropods were collected from beneath cardboard sheets and off vegetation at one location on the Avalon Peninsula (Peter's River Rd., 46°47'N 54°10'W, 24-29 May, 1984), and at 2 sites in central Newfoundland (adjacent to the Buchans Hwy #370 between Buchans and Buchans Junction, 48°37'N 57°26'W, 30 May-3 June, 1984; and Sandy Pond, 48°05'N 55°42'W, 7-20 July, 1987) (Fig. 1). Snails and slugs were identified with the aid of Pilsbury (1939-1948), Burch (1962), and Maunder (1985), digested in artificial pepsin solution, and examined for protostrongylid nematode lar-

vae (Lankester & Peterson, 1995). Larvae were heat-relaxed, stored in 10% glycerin in 70% alcohol, and later drawn and measured.

Differences in larval dimensions were tested using ANOVA and Duncan's Multiple Range test according to the Statistical Package for the Social Sciences (SPSS, Inc., Chicago, Illinois, USA).

Results

Examination of caribou exhibiting neurological signs

A total of 17 caribou exhibiting abnormal neurologic signs was examined from 1981 to 1985 (Table 1). Most were from the Buchans and Gaff Topsails areas of central Newfoundland, and were reported between January and April. All but one were calves (<1 yr) and 12 of 15 sexed animals were males. Affected animals were described as appearing "tame" or "stunned". They could be approached closely (Fig. 2). Some stood for long periods with the head held low and back arched. Others exhibited marked weakness of the hind limbs, sometimes dragging one or both legs (Fig. 3). Two animals were unable to stand when collected.

At necropsy, all animals (excluding nos. 4 and 5 for which the brain and spinal cord were not available) had adult *E. rangiferi* associated with the central nervous system (CNS) (Table 1). The majority of worms in the CNS (75%) were females. Many were free in the subdural space or were weakly attached by strands of connective tissue to the overlying dura or underlying pia-arachnoid. Six were lying partially or completely beneath the pia-arachnoid with up to 1 cm of their body length penetrating into brain tissue (in caribou nos. 7, 11, 15, and 16). The dura adhered firmly to the pia over much of the brain in caribou nos. 11, 14, and 16. Adhesions and membranes near worms were yellowish to pink in colour. Worms in the vertebral canal were all in the subdural space over the cervical or thoracic regions of the cord. Worms outside of the CNS were mostly found among the muscles of the chest, forelimbs, and hindlimbs. The number of first-stage larvae found in the feces of 9 animals showing signs ranged from 2 to 277/g of fresh feces (Table 1).

Wildlife protection officers reported a number of additional animals with signs characteristic of elaphostrongylosis. In the winter of 1981, 10 were

Table 1. *Elaphostrongylus rangiferi* in caribou exhibiting neurologic signs in Central Newfoundland, 1981-85

No.	Date	Location	Sex	Age[a]	Number of *E. rangiferi*			Sex of worms in CNS	Larvae/g[c] feces
					Cranium	Vertebral[b] Canal	Muscle		
1.	3 Jan 81	Buchans	M	calf	2	1	?	?	?
2.	22 Jan 81	Buchans	M	calf	24	7	15	?	?
3.	27 Mar 81	Buchans	?	calf	1	1	20	?	?
4.	21 Jan 82	Buchans	M	calf	?	?	20	?	?
5.	Feb 82	Buchans	M	calf	?	?	21	?	?
6.	28 Jan 84	Gaff Topsails	M	calf	5	0	5	3F, 2M	6
7.	30 Jan 84	Gaff Topsails	F	yrlg	5	?	6	3F, 2M	8
8.	30 Jan 84	Grey River	M	calf	5	?	?	4F, 1M	277
9.	21 Feb 84	Gaff Topsails	?	calf	4	?	?	3F, 1M	?
10.	21 Mar 84	Gaff Topsails	F	calf	4	4	?	8F, 0M	35
11.	20 Mar 84	Gaff Topsails	M	calf	2	1	7	1F, 2M	2
12.	21 Mar 84	Gaff Topsails	F	calf	8	0	14	6F, 1M, 1?	104
13.	29 Mar 84	Gaff Topsails	M	calf	6	1	20	6F, 1M	?
14.	11 Apr 84	Middle Ridge	M	calf	2	?	0	2F, 0M	126
15.	24 Apr 84	Gaff Topsails	M	calf	5	0	21	4F, 1M	267
16.	Mar 85	Middle Ridge	M	calf	8	1	?	6F, 2M, 1?	?
17.	23 Apr 85	Gaff Topsails	M	calf	14	?	?	8F, 5M, 1?	58

[a] A calf is <1 yr old, assuming a birth date of 1 June.

[b] Worms in subdural space, beneath the pia-arachnoid or in nerve parenchyma.

[c] First-stage, dorsal-spined larvae.

? Not available for examinarion.

Table 2. Prevalence and mean intensity of first-stage prorostrongylid larvae in caribou feces from Newfoundland[a]

Location	Prevalence					
	1982	1983	1984	1985	1989	1990
Northern Peninsula	---	50 (24) M[b](1±1)[c]	---	---	---	---
La Poile R.	30 (60) M	---	---	---	38 (50) J (2±4)	---
Grey R.	70 (56) M	60 (42) A (19±29)	---	---	---	---
Gaff Topsails	50 (81) M	---	86 (29) A (24±37)	74 (27) J (8±9)	---	---
Middle Ridge	(40 (95) M	48 (27) M (3±4)	---	45 (49) J	50 (50) J (14±23)	---
Avalon Peninsula	---		---	33((40) M (94±150)	35 (40) J	31 (26)

[a] All samples collected January – April.
[b] Percent passing larvae, followed in brackets by sample size (*n*).
[c] Month of collection and, when aviable, mean no. larvae/g wet feces ± S.D. in brackets.

seen in the Buchans-Buchans Jct. area; 5 were seen in the winter of 1982, and 3 in 1983. In the winter of 1984, 17 of 28 cases reported from across the central part of Newfoundland were seen in the Gaff Topsails area as were 11 of 18 animals sighted in winter of 1985. We were not aware of any sick animals being seen in other parts of the Province during this period.

Two apparently healthy caribou were collected from the Gaff Topsails area in late June, 1987. The 2-3-yr-old male had a total of 16 adult *E. rangiferi* among muscles of the chest and legs and a 13-mth-old male had 22; no worms were found in the cranium of either animal. Neither showed neurologic signs but the gross pathology seen at necropsy was unusual. Considerable yellowish-red, subcutaneous edema and caseous exudate were visible over all large muscles of the chest, lateral abdomen, and lower limbs. Such extensive subcutaneous reaction was not seen in infected caribou examined during winter and early spring.

Herd infection level 1982-90

The prevalence of dorsal-spined, protostrongylid nematode larvae in caribou feces was lowest in samples from the La Poile and Avalon Peninsula herds and highest in the Grey River and Gaff Topsails herds; it varied little between years in any particular herd (Table 2). The intensity of larval output ranged from 11 larvae/g (mean S.D.) in samples from the Northern Peninsula to 94±150 larvae/g on the Avalon Peninsula (collected May, 1985). At the latter site, at least 4 animals, assumed to be calves because of their small pellets, were passing up to 580 larvae/g of fresh feces.

Protostrongylid larvae collected from caribou on the Avalon Peninsula in 1985 and 1989 were shorter (366±3, 310-385 µm, n=30; and 359±4, 320-392 µm, n=30, respectively) than those from caribou at Middle Ridge in 1985 (424±3, 385-470 µm, n=30) and the Gaff Topsails areas in 1985 (439±3, 405-457 µm, n=30) (P=0.04) but those collected from the Avalon Peninsula in 1990 were not (395±5, 352-445 µm, n=30).

Examination of wild moose

Four of the 7 adult moose from Middle Ridge had *E. rangiferi* on the surface of the latissimus dorsi muscle and associated fascia beneath the shoulder (Table 3). Dorsal-spined larvae (n=9, mean length 348±8; 295-372 µm) were present in the feces of only one of the four moose with adult *E. rangiferi.* Longissimus dorsi muscles were not examined for *P. andersoni.*

One of 28 moose fecal samples collected off snow in the Gaff Topsails area (April, 1989) contained 20 dorsal-spined larvae in 15 pellets. The larvae were 388±7 µm; 340-420 long. Although the mean length of larvae from moose feces in both the Gaff Topsails and Middle Ridge areas were shorter than

Table 3. Dimensions (μm) of *Elaphostrongylus rangiferi* on chest and shoulder muscles of moose from Middle Ridge, Newfoundland

	No. measured	Mean ± S.E.	Range
Males			
Length (mm)	6	35± 1	31– 38
Width	6	199± 7	175–220
Esophagus	6	681±14	650–740
Nerve ring	6	132±11	100–170
Excretory pore	4	153±13	115–175
Spicules	7	220± 4	205–232
Gubernaculum	7	75± 3	63– 85
Bursa (length)	7	174± 8	173–195
(width)	7	139± 5	128–157
Females			
Length (mm)	1	47	47
Width	3	223± 7	220–240
Esophagus	4	698±30	635–770
Nerve ring	4	131± 7	120–150
Excretory pore	4	145±13	118–170
Vulva	1	300	300
Anus	1	68	68

larvae from caribou at those locations (P=0.05), it is noteworthy that some larvae in the sample from the Gaff Topsails exceeded 400 μm in length.

Searching for P. andersoni

Only 2 of 12 caribou had *P. andersoni* (Tables 4 and 5). Both were young animals (7 and 13 mo old). In one from the Gaff Topsails area of central Newfoundland, a portion of a male nematode (160 μm wide) resembling *P. andersoni* was found deep in the longissimus dorsi muscle. This animal also had numerous *E. rangiferi* in muscles of the shoulder and chest. A second, from near Peter's R. Rd., Avalon Peninsula, had 22 *P. andersoni* in the longissimus dorsi muscles of the back and 7 in the neck, rump, and thigh muscles.

All specimens of *P. andersoni* were found loosely coiled, deep within muscles. A dark red area of haemorrhage (0.5-1 cm diam.) was associated with about one-half of the specimens, and helped in locating them. Petechial haemorrhages (1-3 mm diam.) were visible across the entire surface of the lungs of the infected calf from the Avalon Peninsula.

Natural infections in intermediate hosts

Of 1407 terrestrial gastropods collected, 9 (0.6%) were infected with protostrongylid larvae (Table 6). A small, dark slug, *Deroceras laeve,* dominated collections in all 3 areas and only this slug was infected. Two slugs had 1 and 3 recently-penetrated, first-stage larvae (397-415 μm long, n=4); 7 had 1-15 third-stage larvae (800-1002 μm long, n=12). All measurements are of alcohol-fixed specimens.

Table 4. Examination of normal caribou from central Newfoundland (Gaff Topsails) and from the Avalon Peninsula for *Elaphostrongylus rangiferi* and *Parelaphostrongylus andersoni,* 1987-89.

No.	Date	Location	Age	Sex	*E. rangiferi* cranium/ muscle	*P. andersoni* long. dorsi muscle	Larvae/g feces
1.	30/06/87	Gaff Topsails	2-3 yr	♂	0/2 ♂♂ & 14 ♀♀	0	0.2
2.	30/06/87	Gaff Topsails	13 mo.	♂	0/5 ♂♂ &, 17 ♀♀	1♂[3]	0.2
3.	23/06/87	Avalon Pen.[1]	2-3 yr	♂	0/0	0	0
4.	23/06/87	Avalon Pen.	3-4 yr.	♂	0/0	0	0
5.	9/12/89	Avalon[1] Pen.	1.5 yr.	♂	0/0	0	0
6.	9/12/89	Avalon[1] Pen.	adult	♀	0/0	0	0
7.	9/12/89	Avalon[1] Pen.	adult	♂	0/0	0	0
8.	9/12/89	Avalon[1] Pen.	1.5 yr.	♀	0/0	0	0
9.	12/12/89	Avalon[2] Pen.	1.5 yr.	♂	0/0	0	0
10.	12/12/89	Avalon[2] Pen.	1.5 yr.	♂	0/0	0	0
11.	13/12/89	Avalon[1] Pen.	1.5 yr.	♂	0/0	0	0
12.	13/12/89	Avalon[1] Pen.	7 mo.	♂	0/0	15 ♂♂ & 14 ♀♀[4]	many

[1] Peter's River Road.
[2] Mr. Misery, S. Avalon.
[3] Partial worm, 160 μ wide (no spicules).
[4] 2/29 in neck, rump, and high muscles.

Table 5. Dimensions (μm) of adult *Parelaphostrongylus andersoni* in longissimus dorsi muscle of caribou from the Avalon Peninsula, Newfoundland

	No. measured	Mean ± S.E.	Range
Males			
Length (mm)	3	21± 2	18– 23
Width	6	102± 5	90– 125
Esophagus	6	853±50	680–1000
Spicules	6	118± 2	115– 128
Gubernaculum	5	64± 3	53– 72
Females			
Length (mm)	2	34± 0	34, 34
Width	6	125± 3	110– 132
Esophagus	4	895±37	830–1000
Vulva[1]	5	165± 8	140– 180
Anus[1]	5	56± 3	51– 65

[1] Position measured from posterior end.

Discussion

An outbreak of CSE involving calves such as that seen in the Buchans-Gaff Topsails areas between 1981 and 1985 has not been reported previously in Newfoundland, despite *E. rangiferi* having been introduced into the province over 70 yr ago (Lankester & Fong, 1989). Although Bergerud (1971) reported emaciated calves standing and feeding for long periods in central Newfoundland in March-April of 1959 and 1961, their condition was attributed to the difficulty in getting food during these two exceptionally severe winters. High mortality, also particularly involving male calves, was seen during several summers but lynx (*Lynx canadensis*) attack and subsequent *Pasturella* infection was proven to be the cause (Bergerud, 1971).

In Norway where *E. rangiferi* originated, epizootics of CSE involving the loss of many animals occur sporadically, principally in domesticated reindeer (Halvorsen *et al.*, 1980) but the disease has also been reported in wild reindeer (Bye & Halvorsen, 1984). The cause of epizootics has previously been attributed largely to conditions associated with reindeer domestication. But Halvorsen *et al.* (1980) demonstrated that the level of *E. rangiferi* infection in herds was correlated with summer temperatures. An epizootic in northern Norway around 1970 was preceded by a series of unusually warm summers. It subsided as summers cooled. Elevated mean summer temperatures at this subarctic location (above 70°N latitude) were thought to increase the rate at which larvae developed in gastropods, resulting in more infective larvae being available to reindeer before freeze-up in the fall. The likelihood of this being the principal cause of epizootics at a more southerly, maritime location like central Newfoundland (48°37'N) remains to be tested.

At the time of writing this manuscript, we became aware of another cluster of cases of CSE occurring in caribou on the Avalon Penininsula of Newfoundland (McBurney *et al.*, 1996; Shane Mahoney & Con Finlay, pers. comm.). Sick animals were reported from January to March of 1996 and 1997. Over 100 were seen in the vicinity of Cape Race, at the southern tip of the peninsula in 1997 (Con Finlay, pers. comm.). *E. rangiferi* was recovered from animals that separated from the herd, stay-

Table 6. Numbers of terrestrial gastropods examined for prorosrrongylid nematode larvae in Newfoundland.

	Avalon Peninsula		Central Newfoundland				
	Peter's River Rd.[1]		Buchans Hwy.[2]		Sandy Pond[3]		
Species	No. exam.	No. infected	No. exam.	No. infected	No. exam.	No. infected	Total infected/ exam.
Deroceras laeve	321	2	675	2	294	5	9/1290
Zonitoides arboreus	11	0	2	0	65	0	0/78
Succinea ovalis	15	0	1	0	9	0	0/25
Arion sp.	2	0	9	0	---	---	0/11
Euconulus fulvus	1	0	---	---	2	0	0/3
Total	350	2	687	2	370	5	9/1407

[1] Collected at site 1 (Fig. 1) 24 May - 3 June, 1984.
[2] Collected at site 2 (Fig. 1) 7 - 20 July, 1987.
[3] Collected at site 3 (Fig. 1) 7 - 20 July, 1987.

ed for days in the same general location, were unafraid of humans approaching, and had mild to marked hind-limb paresis. Many of these animals were under-weight, despite appearing to spend considerable time eating . Both calves and older animals were showing clinical signs. It is probably worth noting that the Avalon Peninsula experienced milder than usual temperatures and an absence of snow during much of the winters of 1995-96 and 1996-97. As a result, the period during which infected gastropods remained accessible to grazing caribou probably was extended considerably. *D. laeve,* which is very abundant over much of the caribou range in Newfoundland, can remain active on ground vegetation at temperatures close to 0 °C (Lankester & Peterson, 1996).

Caribou showing neurological signs from central Newfoundland were almost exclusively young (< 1 yr) and male. Halvorsen (1986) made a similar observation in Norway. Although no signs of disease were apparent during his study, infection with *E. rangiferi* was most prevalent in the heaviest calves. These, in fact, were mostly males but larger female calves also were more frequently infected than smaller ones. This was attributed to the larger amount of food likely eaten by the largest individuals and the attendant increased risk of ingesting infected gastropods.

It has been suspected that animals showing signs of CSE are likely those with the most worms (Halvorsen, 1986). Yet, in our sample, neither the number of *E. rangiferi* present in the CNS, total numbers of worms recovered, nor the numbers of larvae being passed in feces, was correlated with the severity of neurologic signs observed. However, this may not be a valid test of the hypothesis since all animals were examined during winter and early spring when some worms were probably immature and difficult to find. Also, only sick animals were examined and counts of worms in muscles may have been underestimated working under field conditions. As well, the Baermann funnel technique has recently been shown to be unreliable for accurately estimating the numbers of protostrongylid larvae in ungulate feces (Forrester & Lankester, 1997) .

The preponderance of female worms found in the CNS of sick caribou may be noteworthy. Because they are longer and wider than males, they may experience more difficulty leaving the CNS and thereby play a greater role in the pathogenesis of infection. However, satisfactory interpretation of this observation requires more complete knowledge of the migration route taken by *Elaphostrongylus* spp. in the course of their normal development within cervid hosts. Although the route is not completely understood, developing worms initially migrate into the CNS to moult and grow in nerve tissue. After about 90 days, they begin to leave the CNS via cranial and spinal nerves to reach their definitive site among skeletal muscles (Lankester, 1977; Hemmingsen *et al.* 1993; Handeland, 1994). Infection in late summer and autumn would explain why most developing worms are in the CNS over winter but increasingly in muscles in spring and summer as observed here.

Among the herds in central Newfoundland, the prevalence and intensity of infection, as indicated by protostrongylid larvae in feces, were greatest in animals of the Topsails area, where sick animals were most commonly seen in 1981-85. However, caution must be exercised in comparing levels of herd infection measured in this way unless fecal samples are collected at similar times of the year and proportionately, from animals of similar age and sex. Most protostrongylid nematodes of cervids show marked seasonal variation in larval output and young animals typically produce the greatest numbers (Slomke *et al.*, 1995). As well, the output of *E. rangiferi* larvae is known to vary with season of the year and with sex of the host; the greatest numbers of larvae apparently being passed by male reindeer in fall during the rut and by females in spring after parturition (Halvorsen *et al.,* 1985).

Data reported here suggests that *E. rangiferi* did not spread to caribou of the Avalon Peninsula until the late 1980s. Only *P. andersoni,* was found at necropsy of Avalon caribou in 1989. And, up to this time, all protostrongylid larvae found in caribou feces were less than 400 µm long as is characteristic of *P. andersoni* (see Lankester & Hauta, 1989). However, first-stage larvae from caribou feces collected on the Avalon in the winter of 1990 were up to 445 µm long, suggesting that some caribou had by that time become infected with *E. rangiferi.* Its presence in the Avalon caribou and its involvement in an outbreak of CSE in late winter of 1996 was confirmed by McBurney *et al.* (1996).

The Avalon Peninsula is connected to the central part of Newfoundland by a narrow isthmus of land only a few kilometres wide at its narrowest point (Fig. 1). This has probably limited the movement of caribou between the central and Avalon herds. The warble fly, *Hypoderma (Oedemagena) tarandi,* another parasite of caribou in central Newfoundland, also

appeared in caribou for the first time on the Avalon Peninsula around 1990 (Shane Mahoney, pers. comm.), supporting the suggestion that caribou with *E. rangiferi* from one of the expanding herds of central Newfoundland may have crossed the isthmus and joined animals of the isolated Avalon herd at about that time. It may also be interesting to note that the Avalon herd has, in past at least, consistently shown a greater rate of increase than any of the central herds of Newfoundland (Bergerud, 1971; Bergerud *et al.*, 1983) that have long been infected with *E. rangiferi.*

Gastropods collected on the Avalon Peninsula in 1984 presumably had larvae of only *P. andersoni* while those from central Newfoundland could have had both *P. andersoni* and *E. rangiferi.* Nonetheless, the prevalence of protostrongylid larvae in gastropods from both parts of the Province was the same (0.6%). Such a low prevalence in gastropods is typical of this group of parasites. For example, in northern Minnesota where almost 80% of white-tailed deer become infected with *P. tenuis* before they are one year old (Slomke *et al.*, 1995), the highest rate of infection in snails and slugs was only 0.2% (Lankester & Peterson, 1996). A high prevalence of infection by these parasites are likely achieved in cervids, despite low levels in gastropods, because of the large volumes of food consumed. Caribou in particular take much of their food close to the ground where conramination by gastropods is impossible to avoid.

The dimensions and morphology of third-stage larvae recovered from gastropods collected in all localities in Newfoundland were similar to those of *E. rangiferi* and *P. andersoni* but larvae of this group cannot be specifically identified (Pybus & Samuel, 1981). The slug, *D. laeve,* is ubiquitous and abundant in Newfoundland (pers. observ.) and probably is the principal source of infection to caribou. The same species is widely distributed in North America (Pilsbry, 1939-1948) and, because it is highly mobile and active from early spring until late fall, is similarly important in the transmission of other protostrongylids of cervids (Lankester & Anderson, 1968; Samuel *et al* , 1985; Lankester & Peterson, 1996).

In summary, *E. rangiferi* was introduced into Newfoundland with reindeer landed at St. Anthony, on the tip of the Northern Peninsula in 1908 (Lankester & Fong, 1989). Cerebrospinal elaphostrongylosis (CSE), primarily affecting young caribou, was firsr reported in the Buchans and Gaff Topsails herds of central Newfoundland in the 1970s (Lankester & Northcott, 1979); a larger than usual number of sick animals were collected in the period 1981-85 and are described here. Evidence also suggests that the parasite finally spread to the Avalon Peninsula caribou herd around 1990 with a large number of cases of CSE being seen in calves, as well as in older animals, in the latter part of wintets of 1996 and 1997. Moose in central Newfoundland were also found infected with *E. rangiferi* but clinical signs of CSE have not been reported in this host. Only 6% of moose passed dorsal-spined larvae in their feces but the mean lengths of these larvae were slightly shorter than that expected of *E. rangiferi* larvae.

The presence of the muscle worm, *P. andersoni* is confirmed in caribou of both central Newfoundland and of the Avalon Peninsula but adult worms could only be found in young animals (< 1.5 yr). The muscles of moose were not examined for *P. andersoni* but the shorter, dorsal-spined larvae (mean < 400 µm) found in feces suggests that this parasite might become patent in moose.

In conclusion, this paper provides a biological and historical basis for further study of CSE, a significant disease of native caribou that is caused by an introduced parasite, *E. rangiferi.* Future work will be complicared by the presence of *P. andersoni* that does not cause neurologic signs but contributes to verminous pneumonia and produces similar dorsal-spined larvae in cervid feces.

Acknowledgements

We wish to thank Hayward White, Dave Slade, and Baxter Slade who helped with field work in central Newfoundland. Con Finlay who collected caribou for examination and generously contributed his expertise and familiarity with animals of the Avalon caribou herd. Lana Foster (nee Bresele) helped with gastropod collections as did Carleen, Connie, and Cindy Finlay and Mrs. Lynn Ruxton helped with statistical analyses. The study was made possible by the encouragement and support of Gene Mercer and Shane Mahoney of the Newfoundland and Labrador Wildlife Division and by funding to MWL from the Natural Sciences and Engineering Research Council of Canada.

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