Experimental infection of reindeer, sheep and goats with *Elaphostrongylus* spp. (Nematoda, Protostrongylidae) from moose and reindeer

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Abstract: Six reindeer (*Rangifer tarandus*), five sheep and six goats (*Ovis ovis* and *Capra hircus*) were experimentally infected with the nematode *Elaphostrongylus alces*. Additionally, one sheep was infected with *E. rangiferi*. Reindeer infected with *E. alces* showed no neurological signs. Sheep and goats infected with the same parasite also remained clinically healthy; however, the sheep infected with *E. rangiferi* showed severe neurological signs and became paralysed. Pathological lesions were minimal in reindeer and domestic ruminants infected with *E. alces*, but were prominent in the lamb infected with *E. rangiferi*. Our results indicate that keeping and transferring sheep and goats into areas inhabited by moose, which is a natural host of *E. alces* may not harm the livestock, while keeping sheep in areas inhabited by reindeer infected with *E. rangiferi* may result in periodic outbreaks of cerebrospinal elaphostrongylosis in sheep.

Key words: Cervidae, small ruminants, *Elaphostrongylus alces, Elaphostrongylus rangiferi*, pathology.

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Introduction

Cerebrospinal nematodiasis caused by *Elaphostrongylus* spp. is a widelly recognised neurological disorder in reindeer (*Rangifer tarandus*), moose (*Alces alces*), and other cervids (Kontrimavichus *et al.*, 1976; Halvorsen, 1986; Stéen & Roepstorpff, 1990; Handeland & Norberg, 1992). Clinical signs of infection are general weakness, poor coordination of hind legs, paresis, paralysis and death. Four species of the genus *Elaphostrongylus* in the Palaearctic region have been reported in connection with disorders, namely *E. cervi* Cameron, 1931; *E. panticola* Lubimov, 1945; *E. rangiferi* Mitskevich, 1960; and *E. alces* Stéen, Chabaud, Rehbinder, 1989. In the last century, two of these nematodes almost certainly would have spread extensively as both reindeer and moose populations have dramatically increased (Cederlund & Markgren, 1987; Statistical Year book of Sweden, 1993). The risk of cross-infection remains where reindeer share habitats with moose. Livestock sharing pastures with reindeer and moose may occasionally acquire *Elaphostrongylus* by ingesting gastropods containing infective larvae (Handeland, 1991).

In North America, *Parelaphostrongylus tenuis*, the meningeal nematode of white-tailed deer (*Odocoileus virginianus*) causes neurological disease in a number of aberrant hosts (Anderson, 1972).

Small domestic ruminants have also been affected where pastures overlap with white-tailed deer (Alden *et al.*, 1975; Guthery *et al*, 1979).

In Norway, similar phenomena have been observed in goats and sheep in regions with reindeer (Handeland & Sparboe, 1991; Handeland, 1991). The objectives of our study were to describe the clinical signs, the gross- and histopathology and to evaluate the ability of *E. alces* to complete development in experimentally infected reindeer, sheep, goats, and one sheep infected with *E. rangiferi.*

Materials and methods

Six Rya sheep lambs and six Swedish dairy goat kids were obtained in the beginning of June 1989. As controls the origin herds of small ruminants were checked for health and parasites during the whole experiment. The lambs and kids were obtained before the herds were let out to the pasture. All had been dewormed orally with Fenbendazol (Axilur® vet 10 mg/kg orally) just prior to the experiment and stabling at the age of two months. Six (three months-old) reindeer also were obtained in August 1989, from a ranched herd where all age-groups receive prophylactic treatment for parasites annually (Ivomec® 0. 2 mg/kg intramuscularly). The origin herd of reindeer was checked for health and parasites during the whole experiment.

Although all animals were negative initially for parasites based on faecal examination, all were treated with mebendazole [Mebenvet®, Telmin® 6 mg/kg orally for 10 days (d)] 30 d after stabling. The use of this drug was based on previous studies demonstrating mebendazole efficacy against *E. rangiferi* (Nordkvist *et al.*, 1983) as reported in Stéen *et al.* (1997). All animals were reared indoors in separate stalls. The animals were maintained for 8 to 12 months. Faeces from animals were examined weekly for protostrongylid larvae and other helminths prior to the experimental infection both before and after treatment with mebendazole.

E. rangiferi L1, were obtained from a reindeer kept as a parasite donor at the University of Tromsø, Norway. *E. alces* LI, were obtained from a necropsied wild moose, originating from Utö island. Utö is not inhabited by either reindeer, red deer (*Cervus elaphus*) or domestic ruminants. To determine the purity of *E. alces* L1 used in the experiment, the morphological criteria have been described in Lankester *et al.* (1998). The infection of the intermediate and final hosts were carried out as reported in Stéen *et al.* (1997). All animals were 3 to 8 months old and infected with approximately 1000 L3 larvae each. The infectivity of *E. alces* and *E. rangiferi* L3 were assessed at the same time by inoculation of moose in a parallel infection study (Stéen *et al.*, 1997). Both experiments conformed to the Swedish regulations on experimental animals and were approved by the regional ethical committee for animal experiments (The National Board of Agriculture, Sweden).

Daily collection of faecal samples from each animal was initiated, 4 to 7 d post-infection (DPI) and continued until animals wete euthanised. Lung tissues were also sampled upon necropsy. Samples were stored at -20 °C until they were analysed. All samples were treated as described in Stéen *et al.* (1997).

Recovered dorsal-spined larvae typical of the family Protostrongylidae were counted. Nematodes were identified to species level (Stéen *et al.*, 1997).

The time of clinical observation, necropsy technique, methods of euthanasia are the same as reported in Stéen *et al.* (1997). The lungs-and liver cut surfaces were scraped and smears were prepared for parasite examination. The brain, spinal cord, muscles and internal organs were fixed in 10% formalin. Tissues were processed, cut to 4 µm thick sections and stained with haematoxylin and eosin.

Results

Reindeer infected with E. alces *L3 (nos. 1 - 6)*

Clinical signs

Reindeer 1, 4, 5 and 6 showed no clinical signs. At 116 DPI, calf 2 limped slightly with his right hind leg, and calf 3 had a persistent cough from 50 d. until it was euthanised at 124 d. Reindeer 1, 4, 5 and 6 were euthanised 75 to 158 DPI. All animals were in a good body condition and showed a normal weight increase throughout the experiment.

Gross pathology and parasites

Multiple small white spots occurred in the liver parenchyma in calf 5. Macroscopic lesions did not occur in the other five. Four adult *E. alces* wotms were found in the epidural space at the position of the 4th. vertebta of the lumbar region in calf 5 (Table 1). Sporadic shedding with low larval concentration occurred in five animals from 39 DPI, throughout the rest of the sampling period. Additionally, larvae were found in the lungs of one reindeer.

Table 1. Experimental infection of sheep, goats and reindeer (*Rangifer tarandus*) with *Elaphostrongylus*[1] spp. from moose (*Alces alces*) and reindeer respectively. All animals received an estimated dose of about a thousand L3 larvae.

Animal no.	Parasite	Animal terminated (DPI)	First neurological signs appeared (DPI)	Inflammatory lesions[2] in the CNS.
Reindeer				
1	*E. a.*	75	-	-
2	*E. a.*	124	-	-
3	*E. a.*	124	-	++
4	*E. a.*	133	-	-
5	*E. a.*	158	-	++
6	*E. a.*	158	-	-
Sheep				
1	*E. a.*	126	-	-
2	*E. a.*	126	-	-
3	*E. a.*	126	-	-
4	*E. r.*	24	21	+++
5	*E. a.*	126	-	-
6	*E. a.*	126	-	-
Goats				
1	*E. a.*	129	-	-
2	*E. a.*	125	-	-
3	*E. a.*	129	-	-
4	*E. a.*	125	-	-
5	*E. a.*	125	-	-
6	*E. a.*	125	-	-

[1] *E. a.* = *E. alces; E. r.* = *E. rangiferi.*

[2] Inflammatory lesions were graded in to the following: - = no lesions; + = mild; ++ = moderate; +++ = severe.

Histopathology

In the *cerebrum* of teindeer 1 and 3, slight to moderate hyperaemia, oedema and haemorrhages was observed. Moderate cellular infiltration of eosinophils, neutrophils, macrophages and plasma cells were also observed in the epidural side of the *dura mater* in calves 3 and 5. Inflammatory cells such as, plasma cells, macrophages, eosinophils and neutrophils were also present around the lateral nerves of the spinal cord in the same cases. In the other cases, microscopic lesions were not observed in the brain, in the spinal cord or along the *dura mater.* The lungs exhibited moderate hyperaemia and oedema in all cases. The white spots described macroscopically in the livers of case 5 were not visible histologically in the parenchyma. Lymphadenitis and perilymphadenitis characterised by hyperaemia and heavy granulocytic exudate of mainly eosinophils wete visible in sinuses, in capsule's and in the surrounding adipose tissue in reindeer 1, 2 and 5. Lymphoid-follicular hyperplasia was present in reindeer 1 and severe hemosiderosis appeared in case 5.

Sheep infected with E. rangiferi *L3 (no. 4)*

Clinical signs

Twenty-one DPI, the lamb limped slightly and had an uncoordinated locomotion which became progressively worse. It was lethargic and unwilling to rise.

On twenty-three DPI, the sheep showed complete paralysis of all four limbs, it was unable to hold its head up, even when placed in an upright position. No righting and placing reflex could be elicited, which caused the animal to lie in a supine position. The eye expressions were noticeably bright and alert.

The sheep was euthanised 24 DPI. The sheep was in good body condition and had normal weight increase before it was sacrificed.

Gross pathology and parasites

Haemorrhages were prominent in *cavum epidurale* and *cavum subdurale* close to 7th. *thoracic vertebrae* and 4th. *lumbar vertebrae.* Petechial bleedings occurred close to *n. branchialis.* Ecchymotic haemorrhages appeared close to *n. ischiaticus* in the pelvis. The lungs were filled with fresh and old nodules with yellow/teddish discoloration. A small number of elaphostrongyline larvae were found in the lungs at the day of necropsy.

Histopathology

A moderate hyperaemia and oedema were prominent in the meninges and in the brain parenchyma. In the meninges and in the grey matter of the spinal cord, severe myelomeningitis with mononuclear cells, reticulo-endothelial cells, perivascular cuffing, hyperaemia and haemorrhages were present (Fig. 1).

Similar lesions also occurred in the subdural side of the *dura mater* and around nerves (Fig. 2). The lungs were slightly hyperaemic, oedematous and emphysematous. In addition there were few focal parasitic granulomas with degenerated eosinophils surrounded by mononuclear cellular infiltration. The liver, kidney and spleen were hyperaemic.

Sheep infected with E. alces *L3 (nos. 1, 2, 3, 5, 6)*

Clinical signs

The sheep showed no clinical signs during the experiment and they were euthanised at 126 DPI. All animals were in a good state of nutrition and had normal weight increase.

Gross pathology and parasites

A few nodules appeared in the lungs of each animal. Some few white spots also were seen in the livers. No adult *E. alces* or larvae were found in all animals (Table 1).

Histopathology

A mild to moderate hyperaemia and oedema occurred in the cerebral meninges and in the brain parenchyma. In all cases, the lungs had a moderate hypetaemia and oedema. Nonpurulent bronchointetstitial pneumonia characterised by mononuclear cell infiltration, slight to moderate hyperplastic alveolar septa and desquamated epithelial cells appeared. The liver of sheep 1 and 5 had granulomatous lesions with temaining parasitic structures and central necrosis surrounded by mono- and polymorphonuclear cells with a few foreign body giant

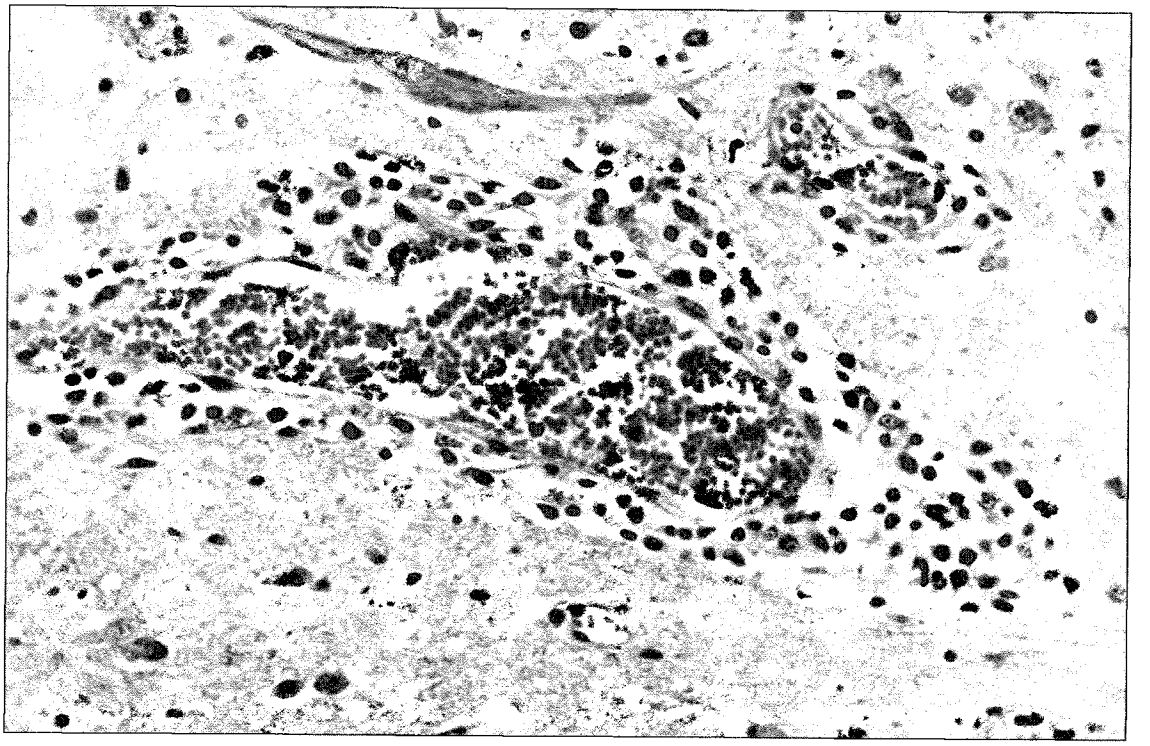

Fig. 1. In the spinal cord, myelomeningitis characterised with perivascular cuffs of mononuclear cells were seen in the sheep infected with *Elaphostrongylus rangiferi* (x 400; H & E).

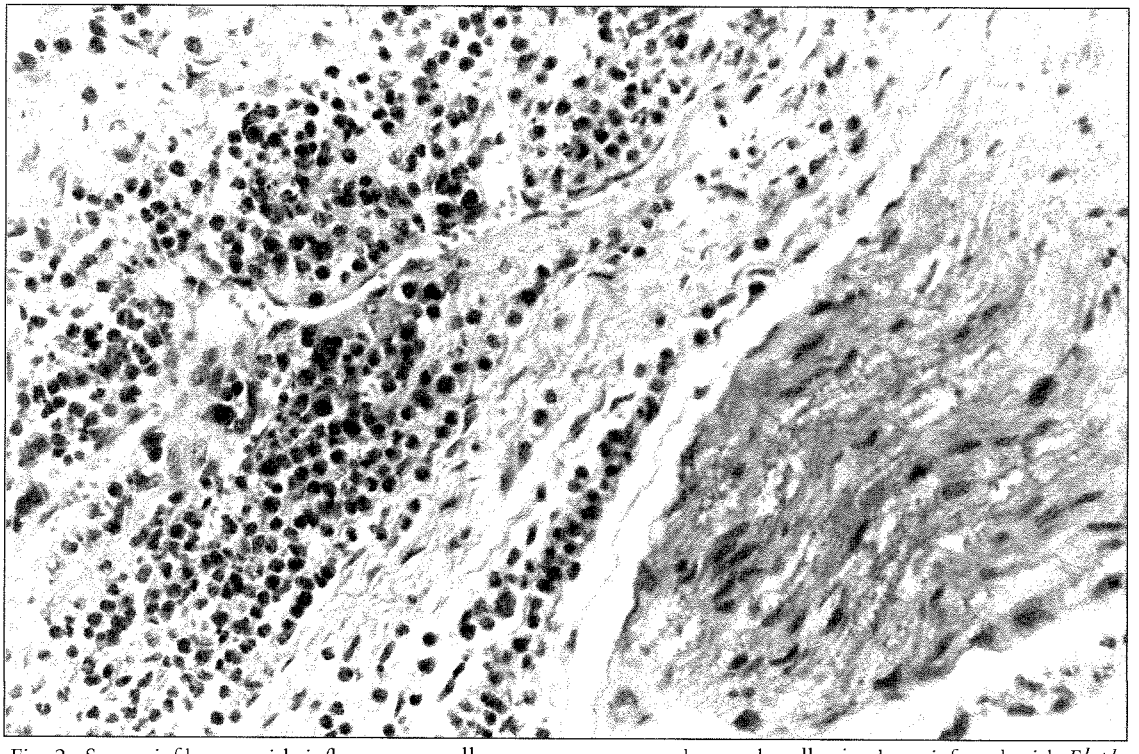

Fig. 2. Severe infiltrates with inflammatory cells were present around nerve bundles in sheep infected with *Elaphostrongylus rangiferi* (x 400; H & E).

cells. These granulomas were encapsulated by fibrotic tissues. A slight oedema, hyperaemia and an increased number of Kupffer cells were noted in sheep 1.

Goats infected with E. alces *L3 (nos. 1 - 6)*

Clinical signs

The goats showed no clinical signs during the experiment. All animals were in a good body conditions and showed a normal weight increases. They were euthanised at 125 and 129 DPI.

Gross pathology and parasites

A few nodules appeared in the lungs of each animal. Goat no. 6, had inflammatory changes in the right knee which was not related to elaphostrongylosis. No adult *E. alces* or larvae were found in all animals (Table 1).

Histopathology

A mild to moderate hyperaemia and oedema were present in the *cerebrum,* in the spinal cord along the *dura mater,* and around nerves.

In goat no. 5, a few parasite eggs were present in the lung alveolar wall close to the blood vessels. These eggs were looked eosinophilic and seem to have been degenerating. In goat no. 6, a focal mononuclear cellular reaction appeared in the *dura mater.* The lungs were moderately congested, oedematous, and emphysematous, with occasional haemorrhages. In 3 animals (nos. 2, 4, 5) interstitial pneumonia characterised by thickened alveolar septa and moderate lymphocytic cellular reaction was visible. Increased number of peribronchial lymphoid tissue occurred in goat 3. The liver of animal 3, had a few clear vacuoles indicating fat infiltration. Goat 5, had a subacute purulent hepatitis with mainly eosinophils and neutrophils. Small necrotic foci with cell debris also occurred in the same goat. The livers of the other cases (nos. 1, 2, 4, 6) had multi focal mononuclear cellular infiltrations in the interstitial tissues. Goat no. 2 exhibited lymphadenitis characterised by a moderate granulocytes in the sinuses.

Discussion

The recovery of adult *E. alces,* in one reindeer and the presence of larvae in reindeer (Stéen *et al.,* 1997), demonstrates that the parasite can complete its life cycle in reindeer. The sparse shedding of lar-

vae in all animals, and the only four adults recovered from a total infective dose of about 6000 L3 (Stéen *et al.*, 1997) shows that a very low percentage managed to develop to maturity.

Despite the heavy infective doses of *E. alces,* histopathological lesions were rare and clinical signs were absent, or very mild in all cases. The only histopathological obsetvations were inflammatory responses along the epidural side of the *dura mater,* around the nerves and in the lymph nodes. These findings are in contrast to the observations in reindeer infected with similar doses of *E. rangiferi* (Handeland & Skorping, 1994).

In sheep and goats, infected with *E. alces* no progressive infections or clinical signs of neurologic disease could be observed in our study. In addition, they did not excrete any larvae, and no larvae were found at necropsy. This is in agreement with the results of Stuve & Skotping (1990). However, in our study, we found macro- and microscopical lesions both in the viscera and in the preferred tissues of *E. alces,* not noted by Stuve & Skorping. Protosttongylid eggs in lungs were seen here in one goat infected with *E. alces* suggesting that the parasite can mature and produce eggs in goats. These eggs appeared to have been unable to develop further.

The observations in sheep, goats and reindeer infected with *E. alces* contrasts dramatically with the results of infection of one lamb with *E. rangiferi.* This animal showed severe neurological signs already after 3 weeks and became completely paralysed. Prominent macro- and microscopical lesions were found. In addition, *E. rangiferi* larvae without dorsal spines were found in the lungs at necropsy. These larvae must have otiginated from the inoculum, since L3 lacks dorsal spines. First-stage larvae of elaphostrongylines loose dorsal spines during their development to infective larvae in the intermediate host (Mitskevich, 1964; Panin, 1964; Lankester *et al.*, 1998). The finding of the larvae and the pathological lesions shows that *E. rangiferi* invaded the host. From an infection study with *E. rangiferi* in sheep and goats, Handeland & Skorping (1992a; 1992b; 1993) and Handeland *et al.* (1993) reported similar clinical signs and pathological lesions in goats, but fewer clinical signs in sheep than described here.

Bakken *et al.* (1975), using a much lower infection dose of *E. rangiferi* in lambs and calves (*Bos taurus*) , observed no clinical signs, but found typical histological lesions in CNS. The observed inflammatory response in the sheep infected with *E. rangiferi* in our study indicates that the patasite migrated along ot through the spinal parenchyma. The observed paralysis is also consistent with migrating larvae within CNS or inflammatory reaction resulted from the parasite infection.

In reindeer and small ruminants, experimentally infected with *E. rangiferi,* traumatic lesions from migrating worms and presence of parasites in the CNS parenchyma were described by Handeland & Skorping (1992a; 1992b), Handeland *et al.* (1993) and Handeland (1994). These shows that this species grows within the CNS tissue. In contrast to that, neither traumatic lesions nor migrating parasites in the CNS were observed here in the animals infected with *E. alces.*

The lesions with remaining parasitic structures in the liver of two sheep infected with *E. alces,* the findings of larvae without dorsal spines in the lungs and the haemorrhages found along the nerves in sheep infected with *E. rangiferi,* indicate a direct migration of the infective larvae. These findings supports the results from experimentally infected guinea pigs (*Cavia porcellus*) with *E. cervi* described by Olsson *et al.* (1998).

Handeland & Skorping (1992a), studying the early migration route of *E. rangiferi* in goats and Handeland (1994) in reindeer, suggested that infective larvae migrated to the lungs via a porto-hepatic route. Additionally, they noted lesions in the mesenteric lymph nodes points out a complimentary, or alternative route. The observations in our study such as inflammation in the body lymph nodes and in the surrounding adipose tissue, suggests that developing larvae of *E.* spp. may use lymph system as an additional way of migration.

The documented knowledge from studies of reindeer meningeal worm *E. rangiferi* and the related species of white-tailed deer *P. tenius* (Platt, 1984) shows that, they both invade the CNS parenchymal tissues. However, the so far reported studies in both natural and experimentally infected animals with *E. alces,* does not show presence of adult worms nor larvae or remnants of parasites in the CNS parenchyma of alternate and definitive hosts.

Adult *E. rangiferi* in reindeer is usually found at the subdural side of the spinal canal and in the cranial caviry (Ronéus & Nordkvist, 1962; Hemmingsen *et al.*, 1993; Stéen *et al.*, 1997). The four *E. alces* recovered in reindeer were all situated epidurally. This is the same location where *E. alces* are observed in moose (Stéen & Rehbinder, 1986; Stuve & Skotping, 1987). These differences in loca-

tion seems to be species-specific to the parasites (Stéen *et al.*, 1997).

In all experimental studies with *E. rangiferi* in aberrant hosts, the histopathological lesions were more prominent than in animals infected with *E. alces*. The lack of significant lesions in *E. alces* infected reindeer suggests that reindeer is an unsuitable host for *E. alces*. The lower virulence of *E. alces* in reindeer and domestic ruminants could be due to a biological character of the parasite, or indicate a reduction in host-parasite compatibility as reported in American cervids infected with *P. odocoilei* (Platt & Samuel, 1978).

Considering differences within species of Elaphostrongylinae, we are suggesting that *E. alces* has a different migration path way and developmental sites than *E. rangiferi* and *P. tenius* (Stéen *et al.*, 1997). *E. rangiferi* also appears to develop further than *E. alces* within domestic ruminants. It seems therefore, that *E. rangiferi* is more virulent to domestic ruminants and reindeer than *E. alces*.

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