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LOW-DOSE MENINGEAL WORM (*PARELAPHOSTRONGYLUS TENUIS)* INFECTIONS IN MOOSE (*ALCES ALCES*)

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ABSTRACT: Parelaphostrongylosis has a rapid onset and is lethal in neonatal moose (*Alces alces*) when large numbers of third-stage *Parelaphostrongylus tenuis* larvae (L3) are given experimentally. Little is known, however, about the severity and prognosis of infections acquired naturally by accidentally ingesting terrestrial gastropods which are rarely infected and have few larvae. To investigate the relationship between infecting dose, age of moose, and severity of disease, five calves were given low doses of three to 10 L3 when five (n=2) or 9.5 mo old (n=3). Each of two animals initially given low doses were later challenged with a dose of 15 L3. As positive controls, two calves were given doses of 15 and 30 L3, considered to be high. All five calves given low doses showed abnormal locomotory signs at 20–28 days postinoculation (DPI) that progressively became more pronounced with hind quarter weakness and front lameness. However, after 77–130 DPI, signs diminished markedly in two of these animals and disappeared in another two. Challenge infections of 15 L3 given 199 days after initial infections had no noticeable effects although an immature worm, probably resulting from the challenge, was found in the spinal cord of one animal killed 51 days later. Two positive control animals given the high doses of 15 and 30 L3 showed moderate to severe, non-resolving, locomotory signs and had to be euthanized. Results demonstrate that single, low doses of three to10 *P. tenuis* L3 cause moderate disease in moose calves but over time, some worms die and animals can recover. A degree of protection may develop against future infection.

Key words: *Alces alces*, meningeal worm, moose, moose sickness, *Parelaphostrongylus tenuis.*

INTRODUCTION

Moose sickness (parelaphostrongylosis) is caused by the nematode *Parelaphostrongylus tenuis* acquired from cohabiting white-tailed deer (WTD; *Odocoileus virginianus*; Anderson, 1964). The moose (*Alces alces*) is an abnormal host in which infections seldom become patent. Worms developing in the spinal cord and in the cranial subdural space cause neuromotor ataxia (Anderson, 1965a). The disease syndrome was described almost 70 years ago (Thomas and Cahn, 1932) and nearly 500 cases have since been reported in the literature (Whitlaw and Lankester, 1994a).

It was hypothesized that parelaphostrongylosis contributed to historical declines in moose numbers in eastern North America as WTD increased following logging (Anderson, 1972) yet unequivocal proof of a causal relationship between infection and past changes in moose numbers has been elusive. Nonetheless, historical moose numbers have varied inversely with densities of WTD and with the intensity of *P. tenuis* infections in them (Whitlaw and Lankester, 1994a, b). The frequency of disease in moose is also reasonably well correlated with WTD densities (Gilbert, 1974; Dumont and Créte, 1996).

Pronounced locomotory signs are seen in naturally infected moose when as few as one nematode is detected in the central nervous system (CNS) (Lankester, 2001). This may have fostered a belief that even light infections with *P. tenuis* lead ultimately to death, a view that is inconsistent, however, with occasional reports of apparently normal moose with adult worms in the cranium (Smith and Archibald, 1967; Gilbert, 1974; Thomas and Dodds, 1988). More recently it has also become apparent that moose can persist in *P. tenuis* enzootic areas, provided that WTD densities remain below about 6/km^2 (Whitlaw and Lankester, 1994b). Clearly, much remains to be learned about the pathogenesis and prognosis of naturally acquired infections in moose.

The pathogenesis of infection was stud-

ied experimentally by Anderson (1964) who gave two neonatal moose calves 164 and 200 third-stage, infective larvae (L3) of *P. tenuis* when calves were about 1 mo old. Both developed debilitating neuromuscular signs and had to be euthanized within 2 mo. Up to 41 sub-adult worms were recovered in the CNS of one animal. Such heavy infections, however, are unknown in nature, probably because naturally infected intermediate hosts (terrestrial snails and slugs) seldom contain more than a few L3 (means of 1.5–6) and only 0.1% to 0.8% have been found infected in northern regions where moose co-occur with WTD (Lankester, 2001). For these reasons the present experiment was designed to determine the effects of meningeal worm on moose when low doses approximating those expected in nature are given to calves of the age at which they are first likely to encounter infected gastropods.

MATERIALS AND METHODS

Seven moose calves were collected in May 1992, when 1–3 wk old at locations in central (45°35′N, 78°30′W) (n=4) and northwestern Ontario, Canada (48°20′N, 85°10′W) (n=3) where WTD were either absent or at very low densities. Their assumed birth date was May 15. The calves were housed with shelter in a 40×40 m fenced enclosure that had not previously held cervids at the Lakehead University Large Animal Research Facility, Kakabeka Falls, Ontario, Canada (48°00′N, 89°43′W). They were bottle-fed a milk formula and provided with alfalfa hay and dairy ration mix (Lankester et al., 1993). The animals were trained during bottle-feeding to mount a platform scale and to allow the taking of body measurements and blood from the saphenous vein without the need for restraints or tranquillizers. After weaning, animals were enticed with sliced apples and given water in nursing bottles while data were collected. Protocols followed were approved by the Lakehead University Animal Care Committee.

First-stage *P. tenuis* larvae (L1) used in experiments were obtained by washing the cranial meninges of a WTD killed near Grand Marais, Minnesota, USA (47°45′N, 90°30′W). Larval identification was confirmed by finding adult *P. tenuis* in the subdural space and intercavernous blood sinuses. Laboratory-reared terrestrial gastropods (*Triodopsis albolabris*) were exposed to larvae on filter paper in Petri dishes and kept 6 wk at 20 C before being digested in pepsin solution to release L3.

Low doses of three to 10 L3 were given to five calves when they were 5 or 9.5 mo old; a challenge dose of 15 L3 was given 199 days later to two of the three older calves. High doses of 15 and 30 L3 were given as positive controls to a 9.5 mo old and 5 mo old calf, respectively. Larvae were pipetted into nursing bottles with milk or water from which animals, both 5 and 9.5 mo olds, sucked vigorously when their weights were 85–153 kg, and 130–187 kg, respectively. Bottles were repeatedly rinsed and the animals allowed to suck again. Challenge infections were given by pipetting larvae into the back of the pharynx of calves #6 and #7 (170 and 296 kg, respectively) following light anesthesia (0.3 mg/kg) with xylazine hydrochloride (Xylamax, Bimeda/MTC, Cambridge, Ontario, Canada).

Animals were bled prior to infection and biweekly thereafter, except #7 who was intractable. Serum was separated, frozen within 1.5 hr at −18 C, and later tested for anti-*P. tenuis* antibodies using an enzyme-linked immunosorbent assay (ELISA) (Ogunremi et al., 2002a). The moose were observed daily for changes in gait and behavior and were euthanized 61 to 250 days postinoculation (DPI). Fecal samples were examined periodically for helminth eggs using sugar flotation and weekly (after 90 DPI) for dorsal-spined nematode larvae using the Baermann funnel technique (Lankester, 1974).

At necropsy, brain and spinal cord were removed and the surface of the meninges and the cranial venous sinuses examined for worms following the procedure of Slomke et al. (1995). The cranium and vertebral canal were flushed with saline and the washings examined. The entire brain and spinal cord were teased apart under a dissecting microscope and then pressed between glass plates and examined at 6.4–10 x using transmitted and reflected light simultaneously. Worms recovered were drawn and measured using a compound microscope and drawing tube.

RESULTS

All calves given low doses of three to 10 L3, whether at 5 or 9.5 mo old, first showed abnormal signs 20 to 28 DPI (Table 1). Initial signs included lethargy, diminished activity, splay-legged stance of hind legs (hocks together), slight posterior ataxia, lowered dew-claws, hind toe dragging, and spreading of the toes. By 46–53

TABLE 1. Experimental *P. tenuis* infections in moose.

Animal number	Age at infection (mo)	L3s[a] given	Signs first observed (DPI)[b]	Signs pronounced (DPI)	Signs prior to necropsy	Euthanized (DPI)	Worms in CNS[c]
1	5	30[d]	28	43–61	marked ataxia	61	9[e]
2	5	3	25	53–77	subtle[f]	89	1
3	5	3	28	53–86	subtle	96	1
4	9.5	15[d]	18	42–95	front lameness, post. weakness	95	3
5	9.5	5	21	46–186	post. weakness	186	0
6	9.5	5 (+15)[g]	20	46–130	normal	243	0
7	9.5	10 (+15)[g]	21	50–104	normal	250	1[h]

[a] Third-stage larvae.
[b] Days post-inoculation.
[c] Central nervous system.
[d] Positive control animal given relatively high dose for comparison.
[e] Seven of nine worms still beneath pia of spinal cord.
[f] Signs slight, intermittent, and barely detectable.
[g] Challenge infection given 199 days after initial dose.
[h] Immature worm in spinal cord probably from challenge infection 51 days earlier.

DPI all animals showed more pronounced signs including stiffness or lameness in one or both forelegs and hind-quarter weakness. By 70 DPI, the two animals each given three larvae showed improvement in the foreleg lameness and by 86 DPI showed improved hock posture, although slightly lowered dew-claws and spread toes were still seen intermittently in calves #2 and #3. Of those given five or 10 L3 when 9.5 mo old, calf #5 still showed weakness in the hind quarters and splay-legged posture of the pelvic limbs at 186 DPI when it was euthanized. Calf #6, however, showed improved gait with no detectable signs after 130 DPI while calf #7 appeared normal after 104 DPI. Challenge doses of 15 L3 given to calves #6 and #7 199 days after initial infections had no noticeable effects during the 51 days until necropsy.

Positive control animals given high doses of 15 and 30 L3 first showed signs 18 and 28 DPI, respectively. Calf #1, given 30 larvae, became particularly weak in the hind quarters and swayed, knuckled, and dragged the hind toes. Between 56 DPI and necropsy at 61 DPI, marked ataxia was evident. When standing, the hocks were angulated and the animal collapsed when pressure was applied over its pelvis. Prior to necropsy at 95 DPI, calf #4 given 15 L3 still showed right foreleg lameness and occasionally stumbled on the left front. Some weakness was apparent in both hind legs.

Up to one-third of the L3 given to calves were later recovered from the CNS as developing adult worms (Table 1). Most worms found in calf #1 killed 61 DPI were still in the spinal cord. Seven of the nine were visible beneath the leptomeninges ($n=5$) or deep in gray matter ($n=2$) at the level of the cervical and lumbar enlargements; five females were 4.1–7.1 cm long and two males were 4.2 and 4.9 cm long. The other two worms (both female, 5.8 cm long and 6.5 cm with head missing) were in the subdural space in the anterior region of the vertebral canal and on the cerebellum, respectively. All worms found in calves killed 89 DPI and later were in the subdural space. Single female worms found at 89 and 96 DPI in calves #2 and #3 were in the subdural space of the cranium and anterior vertebral canal and were 4.9 and 7.0 cm long, respectively.

The three worms in calf #4 killed at 95 DPI were all in the subdural space of the cranium; a male was 5.9 cm long and only pieces of two females were recovered. The anterior end of one penetrated about 1 cm into a deep sulcus over the left cerebral hemisphere. No adult worms could be

found in the remaining calves necropsied later than 185 DPI (calves #5, #6, and #7), except one immature female (4.2 cm long) that was still deep in the spinal cord at the level of the fourth cervical nerve of calf #7 when killed 250 DPI but 51 days after a challenge infection.

Lesions grossly visible at necropsy included areas of hemorrhage up to 1 cm long in the leptomeninges of the spinal cord, often removed 0.5–1 cm from where developing worms were located within the parenchyma of the cord (calves #1 and #7). Three small (1 mm diameter) lymphoid nodules were seen on the leptomeninges near lateral spinal nerves of only one animal (calf #2). A film or patches (up to 1.5 cm^2) of viscous, red or green-yellow exudate were visible on the dura in the floor of the cranium near the optic chiasma and beneath the occipital lobes of the cerebellum in three animals (#2, #3, and #7). Cerebrospinal fluid appeared clear in all animals. No dead worms or their remains were visible in any calf.

The feces of all animals were negative for dorsal-spined nematode larvae but most passed low numbers of helminth eggs including those of *Trichuris* sp., *Capillaria* sp., *Ostertagia* sp., and *Oesophagostomum* sp.

DISCUSSION

Although this experiment involved several variables and only a small number of animals, certain limited conclusions still seem justified. There was no evidence that the age of moose calves appreciably affected the success of worms reaching the spinal cord. One-third of the L3 given to three 5 mo old calves and one-fifth of those given to a 9.5 mo old were recovered as maturing adult worms within 2–3 mo of infection. This was similar to the recovery reported by Anderson (1964) who was able to account for 41 of 200 L3 given to a 1 mo old moose calf when necropsied 2 mo after infection.

The delayed movement of *P. tenuis* from neural tissue of moose compared to that observed in WTD was first noted by Anderson (1965b). While most worms given to WTD had left the spinal neural parenchyma by 40 DPI, a large number were still present in the cord of a moose calf killed at 60 DPI (Anderson, 1964). The more severe neurologic signs observed in moose were attributed to the greater tissue damage resulting from the delayed presence of developing worms in the cord and their larger size and coiled configuration. Seven of nine worms recovered here from a calf killed 61 DPI were still in the cord. Worms recovered later than 88 DPI had all left the cord and were in the subdural space of the anterior cord or in the cranium.

The progression of neurologic disease in moose coincides roughly with the known migration and development of *P. tenuis*. Signs of neurologic disease were first seen in moose calves at 18–28 DPI. Migrating L3 are known to reach the spinal cord of neonatal white-tailed deer in about 10 days (Anderson and Strelive, 1967). They may take slightly longer in the larger and older moose calves and likely some development of L3 is required before animals are noticeably affected. Signs became more pronounced in moose calves after about 6 wk, presumably as worms moved and grew in nerve tissue. Signs subsided in some animals after 87 to 95 DPI, by which time worms had left the spinal cord. However, noticeable improvement in gait took up to 130 DPI in one animal and deficits persisted in another, despite the apparent absence of worms.

The number of L3 given to moose clearly determines the outcome of infection. Severe and persistent disease signs resulted from doses of 15 or more while some moose receiving doses of 10 or less recovered. Improvements in the gait of these animals occurred after worms had left the spinal cord but may also have been due in part to the death of worms. The two calves showing complete recovery had no worms in the cranium when killed after 6–8 mo, despite having shown earlier locomotory

signs of infection. Worms presumed to have been present may have been overcome in the spinal neural tissue since remains were not visible on the meninges or in the subdural space. Doses received by moose in the wild are unknown but likely are low and infrequent. The majority of infected gastropods have only one or two L3 but some are known to have as many as 97 (Lankester, 2001). As well, the frequency of infection in gastropods in northern areas where moose occur is often much less than 1%. Slomke et al. (1995) demonstrated that a WTD fawn in northeastern Minnesota may only ingest one infected gastropod in its first and second summer of life. Prestwood and Nettles (1977) suggested that WTD may similarly become infected with *P. andersoni* by isolated encounters with gastropods containing low numbers of L3.

Results reported here suggest that low-dose *P. tenuis* exposure may induce a degree of protective immunity in moose. Only one immature worm was recovered from the spinal neural parenchyma of one of two calves challenged with 15 L3. This can be compared numerically to three worms recovered from another calf given a single dose of 15 L3. It is recognized, however, that the reduced number of worms found in the challenged moose could in part be due to the mode of infection. Larvae were squirted into the back of the pharynx of the challenged animals instead of being sucked from feeding bottles as in the case of all earlier infections. Sucking stimulates the esophageal groove reflex directing liquids past the rumen and more quickly into the abomasum where larvae are thought to penetrate (Anderson, 1963). Although having to pass through the rumen may have reduced the success of L3, the penetration of at least some from the challenge infection was indicated by an increase in the anti-*P. tenuis* antibody titer in calf #6 (Ogunremi et al., 2002b); blood was not available from the second challenged animal (#7).

Experimental moose had reached the age of 16 mo when challenge doses were given but their increased age is not likely to have played a role in reduced worm recovery. Although parelaphostrongylosis tends to be seen more in younger animals (Anderson and Prestwood, 1981) older moose clearly are vulnerable. Meningeal worm accounted for 38% of the mortalities seen over a 4 yr period following the reintroduction of moose into northern Michigan and the majority of cases (10/13) were seen in animals translocated as adults (Aho and Hendrickson, 1989).

A strong protective response is thought to develop in WTD after they acquire an initial *P. tenuis* infection as fawns (Slomke et al., 1995). Davidson et al. (1985) hypothesized that fallow deer may also have some innate resistance to *P. tenuis* but free-ranging animals probably survive an initial low-level infection and subsequently develop immunity to reinfection. Evidence supporting this view included inflammatory lesions indicative of *P. tenuis* infection in the CNS of several adult animals that were otherwise normal and in good condition. If moose similarly develop a degree of protection after surviving an initial low-dose infection, knowledge of its duration, individual variability, and whether it requires continual boosting will be required to thoroughly understand the population effects of *P. tenuis* on moose coexisting with WTD.

The relative susceptibility of North American ungulates to *P. tenuis* can best be judged on the basis of low and medium dose infections. A number of such studies now exist. Most elk (*Cervus elaphus*) given 25–75 L3 developed neurologic disease and two died yet none of five given 15 L3 showed signs, despite finding worms in the cranium of one killed after 158 DPI (Samuel et al., 1992). None of two elk given six L3 and two given 20 showed signs although as many as two adult worms were still present in the cranium after 243 days (Ogunremi et al., 2002). All of six fallow deer (*Dama dama*) given 25–150 L3 died (Pybus et al., 1992). Most llamas devel-

oped fatal infections after receiving 5–6 L3 (Foreyt et al., 1992; Rickard et al., 1994). Both domestic sheep and wild bighorn sheep (*Ovis canadensis*) generally resist doses of less than 100 L3 but transient ataxia and mild paresis were seen in one adult bighorn given 25 (Pybus et al., 1996).

Moose can therefore be considered less susceptible than llamas but more so than elk, fallow deer, and sheep. Reindeer (*Rangifer tarandus tarandus*) and caribou (*R. tarandus caribou*) are judged to be particularly susceptible based on the rapid failure of numerous herds either released or held captive on ground previously occupied by WTD (Lankester, 2001). Disease progressed rapidly in mule deer (*Odocoileus hemionus hemionus*) given more than 75 L3 (Anderson et al., 1966; Tyler et al., 1980) but the extent to which this species might tolerate low experimental or natural doses has not been studied. Black-tailed deer (*O. hemionus columbianus*) which were considered to be more tolerant than mule deer (Tyler et al., 1980), steadily declined in numbers after being released onto WTD range in Tennessee and parelaphostrongylosis was considered a major contributing factor (Nettles et al., 1977).

Although innate susceptibility must play a role in determining the success of certain ungulates cohabiting with WTD, other important factors include the density of infected WTD and whether they are young demographically and passing large numbers of larvae (Slomke et al., 1995). Also important is the degree of spatial overlap between WTD and the susceptible species, feeding habits, climatic conditions that determine the level of infection in terrestrial gastropods, and the initial infecting dose and the rate of subsequent exposure by animals capable of developing some protective immunity.

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