Attachment 1

From Parasitic Diseases of Wild Mammals, Second Edition

9

EXTRAPULMONARY LUNGWORMS OF CERVIDS

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INTRODUCTION. Of mammalian lungworms, none has attracted as much attention as members of the genera *Parelaphostrongylus* and *Elaphostrongylus*. These genera comprise a small but important group of parasites found in ruminants, notably members of the Cervidae. This chapter reviews the six known species in these genera, with emphasis on recent literature.

PARELAPHOSTRONGYLUS TENUIS (DOUGHERTY 1945)

Classification: Nematoda: Metastrongyloidea: Protostrongylidae.

Synonyms: *Odocoileostrongylus tenuis* (Dougherty) Schulz, 1951; *Elaphostrongylus odocoilei* Anderson, 1956 (not Hobmaier and Hobmaier, 1934); *Neurofilaria cornellensis* Whitlock, 1952; *Elaphostrongylus tenuis* (Dougherty) Whitlock, 1959, Smith and Archibald, 1967.

Common Names: Cerebrospinal nematodiasis, parelaphostrongylosis, meningeal worm, brain worm, moose sickness, moose disease.

Parelaphostrongylus tenuis is common almost everywhere white-tailed deer (WTD) (*Odocoileus virginianus*) occur in eastern North America (Fig. 9.1). Little or no disease is apparent in white-tails, but when other native cervids, and some bovids and camelids, encounter the parasite, debilitating neurological signs may result. Since the discovery that *P. tenuis* was the causative agent of "moose sickness" (Anderson 1964a,b), considerable knowledge about this parasite has accumulated in the literature. Yet our understanding of its past and present impact on populations of wild moose (*Alces alces*) and other native cervids remains incomplete. In certain areas, parelaphostrongylosis causes financial loss to owners of llamas, sheep, and goats that share range with white-tailed deer, and it is an important concern in zoos and game farm settings. Fear of spreading this parasite to western North America has led to legislation restricting the translocation of white-tails and other hosts in which the parasite occasionally matures.

Previous reviews of *P. tenuis* include Anderson (1968, 1971a), Anderson and Prestwood (1981), Lankester (1987), Anderson (1992), Lankester and Samuel (1998) (also, see annotated bibliography on members of *Parelaphostrongylus* and *Elaphostrongylus* by Samuel 1991). The present account defers to Anderson (1971a) and Anderson and Prestwood (1981) for some earlier references, but attempts to cite most recent literature, particularly that on the biology and epizootiology of *P. tenuis* in white-tailed deer, the impact of this parasite on other species, and advancements in diagnostic methods. For information on the parasite's morphology and phylogeny, readers are referred to Anderson (1963a,b), Platt (1984), Carreno and Lankester (1993, 1994), and Carreno and Hoberg (1999).

Life History. Adult *P. tenuis* are long and thread-like. Males are up to 6.2 cm long x 0.2 mm wide and greenish-yellow to brown in color (Table 9.1, Fig. 9.2). Females are up to 9 cm x 0.25 mm and coloured darker brown to black by the contents of their intestine (Carreno and Lankester 1993). In white-tailed deer, adult worms are found most frequently in the veins and venous sinuses of the cranial meninges. These include the cavernous and intercavernous blood sinuses in the floor of the cranium, as well as the connecting sagittal and transverse venous sinuses in the overlying dura membrane (Anderson 1963a; Slomke et al. 1995). Worms also occur free in the cranial subdural space, where they are easily detected on the surface of the brain or on the inner surface of the dura (Gilbert 1973). Few worms are found adhering to or beneath the pia in white-tailed deer (Slomke et al. 1995). In abnormal hosts such as moose, worms may be associated with the cranial nerves, and their eggs and larvae have been found in the eyes (Anderson 1965a; Kurtz et al. 1966). Rarely, worms are swept from the blood sinuses to other locations in the body. This probably explains the recovery of the holotype specimen of *P. tenuis* from the lung of a white-tailed deer and its original assignment to *Pneumostrongylus* by Dougherty (1945).

Unembryonated eggs released by females into the venous blood are carried to the heart and then to the lungs, where they lodge in alveolar capillaries and complete their development to the first larval stage (L_1) (Table 9.2). Eggs laid by females extravascularly within the cranium develop and hatch, but whether these larvae can enter the circulation and reach the lungs is unknown. L_1's move into the alveolar air space and are propelled out of the lungs in the layer of mucous that moves upwards on the so-called ciliary escalator lining most of the respiratory tree. Upon

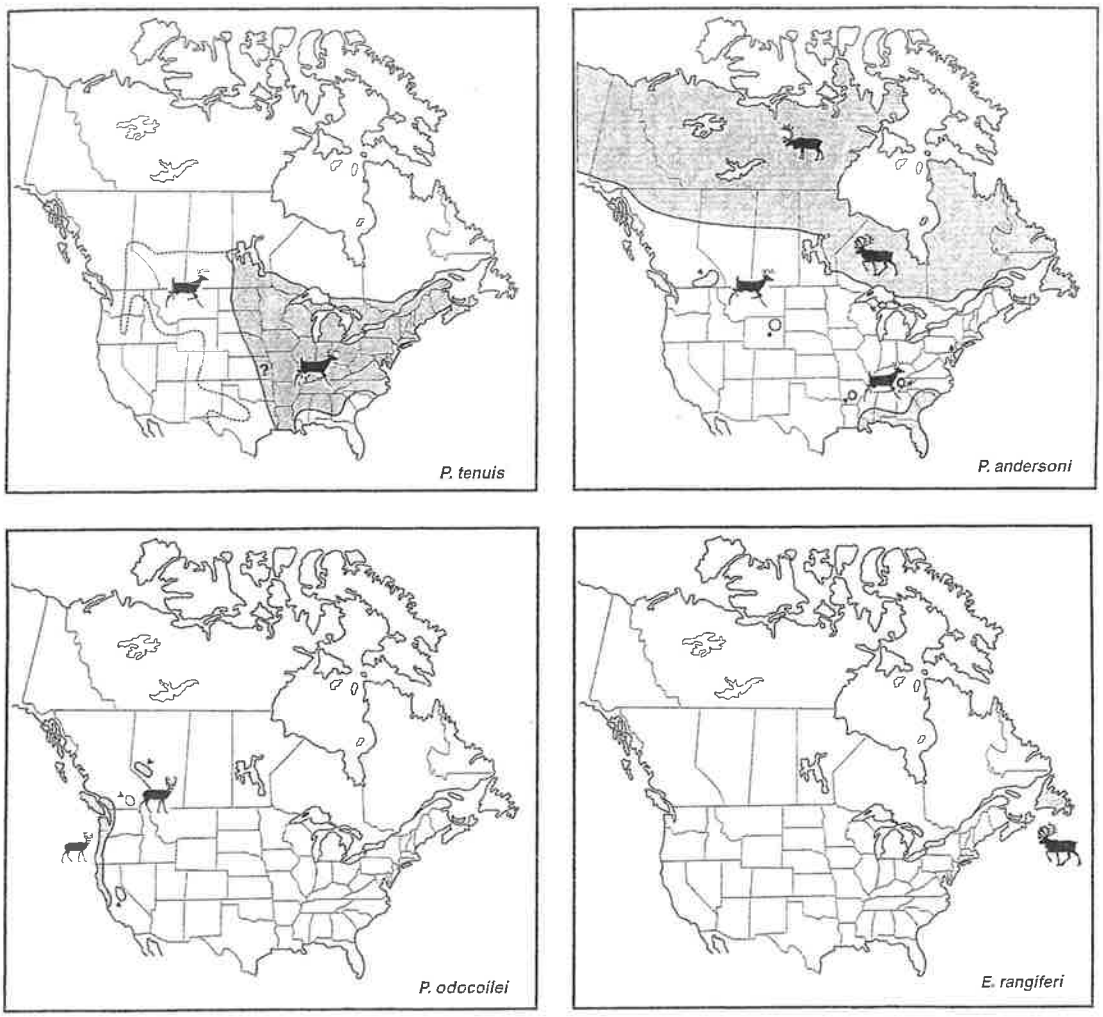

FIG. 9.1—Distribution of the four species of elaphostrongyline nematodes of cervids in North America. Shaded areas indicate the known distribution of the parasite in the cervid hosts illustrated (*P. tenuis* in white-tailed deer, *P. andersoni* with a disjunct distribution in white-tailed deer and more continuous distribution in woodland and barrenground caribou, *P. odocoilei* in mule deer [larger silhouette] and black-tailed deer [smaller silhouette], and *E. rangiferi* in woodland caribou). There are no published reports of *P. tenuis* in Kansas (?). The dotted line approximates the western limits of the distribution of white-tailed deer. Arrowheads accentuate the location of isolated reports.

reaching the pharynx, larvae are swallowed and pass unharmed through the digestive tract and out with the feces (Anderson 1963a).

Most L_1's are located on the surface of fecal pellets in a thin layer of mucous (Lankester and Anderson 1968; Forrester and Lankester 1997a). This contrasts, for example, with the location of larval *Protostrongylus* spp. of bighorn sheep (*Ovis canadensis*) that are most numerous toward the center of pellets (Forrester and Lankester 1997b). L_1's of *P. tenuis* readily leave pellets immersed in water and presumably are also removed by rain and melting snow. To develop further, they must penetrate, or be eaten by, a terrestrial snail or slug. A large variety of species are capable of serving as intermediate hosts (Anderson and Prestwood 1981) (Table 9.3). Terrestrial gastropods may become infected most frequently when they encounter larvae dispersed in the soil. This method of infection has been demonstrated to occur under laboratory conditions (Lankester and Anderson 1968) and might explain why most naturally infected gastropods contain few larvae. Natural infections have not been found in aquatic snails but *Lymnea* sp. has been infected experimentally (Anderson 1963a).

TABLE 9.1—Adult dimensions of elaphostrongyline nematodes in North American cervids (μm), unless otherwise indicated

	P. tenuis[a]		*P. andersoni*[b]		*P. odocoilei*[c]		*E. rangiferi*[d]	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Males								
Length (mm)	55	(31–62)	20	(19–23)	23	(18–26)	35	(31–38)
Width	162	(92–200)	111	(87–140)	147	(138–156)	199	(175–220)
Espohagus length	640	(562–770)	726	(670–770)	653	(565–717)	681	(650–740)
Nerve ring[f]	—	(110–150)[e]	85	(80–100)	88	(68–94)	132	(100–170)
Excretory pore[f]	—	(100–140)[e]	103	(87–120)	75	(56–94)	153	(115–175)
Spicule length	223	(202–249)	104	(87–115)	149	(132–170)	220	(205–232)
Gubernaculum length	109	(89–137)	47	(42–52)	93	(73–112)	75	(63–85)
Females								
Length (mm)	79	(66–90)	31	(30–35)	44	(39–48)	47	(47)
Width	209	(120–250)	113	(95–130)	163	(141–179)	223	(220–240)
Espohagus length	694	(623–796)	747	(670–900)	627	(588–658)	698	(635–770)
Nerve ring[f]	104	(90–126)	90	(70–100)	92	(79–106)	131	(120–150)
Excretory pore[f]	139	(109–164)	—	(67–130)	78	(71–82)	145	(118–170)
Vulva[g]	181	(138–233)	122	(97–170)	178	(161–194)	300	(300)
Tail	53	(35–62)	53	(40–75)	48	(44–65)	68	(68)

[a]Measurements according to Carreno and Lankester (1993). Others available from Anderson (1956).
[b]Measurements according to Prestwood (1972). Others available from Pybus and Samuel (1981), Lankester and Hauta (1989), Carreno and Lankester (1993), Lankester and Fong (1998).
[c]Measurements according to Platt and Samuel (1978b). Others available from Hobmaier and Hobmaier (1934), Brunetti (1969).
[d]Measurements according to Lankester and Fong (1998). Others available from Lankester and Northcott (1979), Carreno and Lankester (1993).
[e]From Anderson (1956).
[f]Position measured from anterior end.
[g]Position measured from posterior end.

In the foot tissue of gastropods, L_1's molt to the L_2 and then to the L_3, or infective stage. The rate of development is temperature dependent. Almost 4 weeks were required to reach the infective stage at temperatures fluctuating between 18° C and 30° C (Anderson 1963a). It probably takes 2–3 times as long at lower field temperatures likely experienced by terrestrial gastropods, although this has never been investigated. Development is slowed or stopped in snails that estivate to avoid dessication, but resumes with the return of favorable conditions. L_3's can survive freezing temperatures over winter in gastropods and probably remain viable for the life of the intermediate host (Lankester and Anderson 1968).

White-tailed deer become infected when they accidentally ingest terrestrial gastropods along with vegetation. L_3's released by digestion from gastropod tissues penetrate through the gastrointestinal wall (particularly of the abomasum) and reach the peritoneal cavity (Anderson 1963a, 1965b,c). Their migration to the central nervous system is thought to be direct. Migrating dorsally in the abdominal cavity and following lateral spinal nerves, mostly in the lumbar region, larvae reach the vertebral canal in about 10 days (Anderson and Strelive 1967, 1969). Still in the third larval stage, they enter tissue of the spinal cord (dorsal horns of grey matter) and molt twice to the fourth and then to the fifth, or subadult stage. By 40 days after infection, most worms have left the spinal cord, apparently via dorsal nerve roots. They move anteriorly in the spinal subdural space to reach the cranium and enter the venous sinuses. The presence of developing worms in the neural parenchyma of the brain of white-tailed deer apparently is rare (Anderson 1968).

The prepatent period of *P. tenuis* in white-tailed deer varies from 82 to 137 days (Anderson and Prestwood 1981; Rickard et al. 1994). It apparently varies inversely with the infecting dose (Rickard et al. 1994), but white-tailed deer age at the time of infection may also be important (M.W. Lankester and A.A. Gajadhar, unpublished). Fawns born in early June become naturally infected and pass larvae as early as mid-October (Peterson and Lankester 1991), but most do not become patent until mid-December or January, a prepatent period estimated to be ~4.5 months (Slomke et al. 1995). The ensuing production of larvae by newly infected white-tailed deer has not been studied thoroughly. In one experimentally infected fawn, larval output increased rapidly following patency, peaked 1 month later, and then declined (Samuel et al. 1992).

Most white-tailed deer acquire a small number of worms within the first or second summer of their life, and intensity does not increase appreciably thereafter (Slomke et al. 1995). Up to 71% of fawns were infected within 5–6 months of birth, and 91% by the time they were 17–18 months old. Average intensities by host age were 2.7 (fawns), 3.0 (yearlings), 3.5 (2 to 6 years) and 4.1 (7 to 15 years). Similar observations were made by Anderson and Prestwood (1981), who first suggested that white-tailed deer acquire a protective immune response against repeated infection. The high proportion of unisexual *P. tenuis* infections in white-tailed

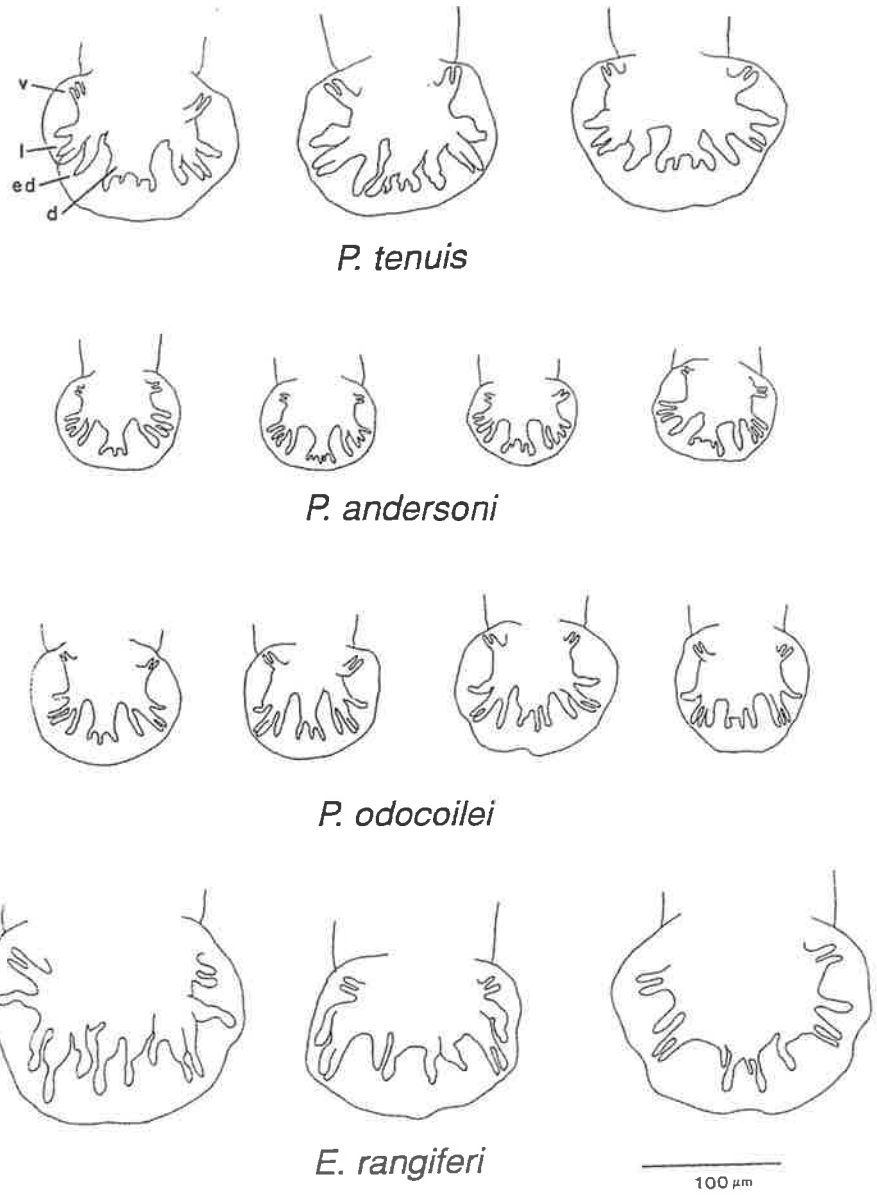

FIG. 9.2—Bursae of North American elaphostrongyline nematodes, ventral view: ***v***, ventral ray; ***l***, lateral ray; ***ed***, externaldorsal ray; ***d***, dorsal ray. *Parelaphostrongylus tenuis* from white-tailed deer; *P. andersoni* from white-tailed deer and barren ground caribou; *P. odocoilei* from black-tailed deer and mule deer; *Elaphostrongylus rangiferi* from caribou (bottom right specimen) and moose (After Carreno and Lankester 1993; used with permission of NRC Research Press.).

deer provides additional evidence of an acquired immunity. As many as one-third of infected white-tailed deer pass no larvae in their feces because adult worms of only one sex are present (Slomke et al. 1995). If repeated infections were possible, the proportion of these unisexual infections would decrease in older animals, but this was not observed. As well, since the mean number of worms acquired by fawns during their first summer and fall was unchanged during their second summer as yearlings, this protection must develop during winter. Variations in the length of the frost-free period when transmission to fawns is possible will, therefore, influence both the mean intensity and the proportion of patent infections. White-tailed deer in

TABLE 9.2—Larval dimensions of elaphostrongyline nematodes in North American cervids (μm)

Larva	*P. tenuis*[a]	*P.andersoni*[b]	*P. odocoilei*	*E. rangiferi*[c]
First Stage				
Length	348(310–380)	351(308–382)	378[d]	426(381–490)
Width	18(16–19)	17(17–18)	17	20(17–24)
Nerve ring	94(80–112)	94(66–109)	—	110(95–130)
Excretory pore	94(80–112)	94(66–109)	98	109(97–125)
Esophagus	165(132–181)	175(163–183)	166	191(163–230)
Genital prim.	224(210–246)	234(216–249)	—	267(245–325)
Tail	32(29–41)	32(27–36)	40	44(32–53)
Third Stage				
Length	971(900–1080)	1019(966–1200)	890(738–977)[e]	1004(937–1041)
Width	42(36–45)	36(33–50)	44(36–52)	46(42–49)
Nerve ring	114(100–125)	117(100–133)	141(135–154)	139(120–150)
Excretory pore	133(122–149)	117(100–133)	—	153(138–163)
Esophagus	352(300–400)	358(300–420)	323(282–399)	381(338–421)
Genital prim.	629(569–707)	681(600–800)	561(521–586)	615(574–648)
Tail	35(31–45)	35(33–45)	47(45–50)	52(40–70)

[a]Anderson (1963).
[b]Prestwood (1972).
[c]Lankester and Northcott (1979).
[d]Hobmaier and Hobmaier (1934).
[e]Ballantyne and Samuel (1984).

some southern areas reportedly have slightly greater numbers of worms than deer near the northern limit of their range (Anderson and Prestwood 1981).

Field evidence provided by Slomke et al. (1995) supports the hypothesis that *P. tenuis* is long-lived in white-tailed deer and that worms acquired by fawns may persist for life. This is consistent with observation by others on the related nematodes, *E. cervi* and *P. odocoilei* that are known to live at least 6 and 8.5 years, respectively (Watson 1984; W.M. Samuel, personal communication).

Epizootiology

DISTRIBUTION. Meningeal worm is common in white-tailed deer of the deciduous forest biome and deciduous-coniferous ecotone of eastern and central North America (Fig. 9.1). It is rare or absent in the coastal plains region of the southeastern United States and is not documented in western North America. New distribution records not listed by Anderson and Prestwood (1981) include Foreyt and Trainer (1980) (Wisconsin), Beaulieu-Goudreault (1981) and Claveau and Fillion (1984) (Quebec), Garrison et al. (1987) (Missouri), Platt (1989) (Indiana), Comer et al. (1991) (South Carolina), Jarvinen and Hedberg (1993) (Iowa), Wasel (1995) (North Dakota, Saskatchewan), Davidson et al. (1996) (Wassaw Island, Geogia), and Oates et al. (1999) (South Dakota and Nebraska).

In 1968, *P. tenuis* was recovered from one white-tailed deer in Collier Co., Florida, where it was probably introduced with white-tailed deer from Wisconsin (Prestwood and Smith 1969). However, it has not been found there since, despite large numbers of white-tailed deer having been examined (Comer et al. 1991). Its occurrence on Wassaw Island off the coast of Georgia was also attributed to an introduction of white-tailed deer from Pennsylvania, further adding credence to concerns that *P. tenuis* can be spread through the translocation of infected hosts (Davidson et al. 1996).

It is not known what limits the natural spread of the parasite westward. The drier central grasslands of North America are presumed to be a barrier by being less hospitable for gastropods, but it is more difficult to imagine why the more northerly aspen parklands did not provide a corridor (Anderson 1972). It is equally unclear why *P. tenuis* is rare or absent in white-tailed deer of the coastal plains portion of the Atlantic and Gulf coast states of Alabama, Georgia, Mississippi, South Carolina, and Florida. Prestwood and Smith (1969) suggested that this might be due to a scarcity of suitable species of gastropod intermediate hosts or to other factors associated with the predominantly sandy soil, pine forest habitat. However, Davidson et al. (1996) rejected this hypothesis on finding a high prevalence of *P. tenuis* in white-tailed deer occupying similar habitat on Wassaw Island, Georgia, where the worm appears to have persisted for almost 90 years. Since the related nematode, *P. andersoni,* is successfully transmitted among white-tailed deer in the coastal plains region (Forrester 1992), yet concurrent infections of *P. andersoni* and *P. tenuis* are rare (Prestwood et al. 1974), some form of host cross-immune response might prevent the geographical overlap of the two parasites (Lankester and Hauta 1989).

HOST RANGE. White-tailed deer is the normal definitive host in which *P. tenuis* becomes patent without causing disease. However, the parasite develops to varying degrees in a variety of abnormal hosts that have contracted infection on natural white-tailed deer range, in proximity to white-tailed deer in zoos or game

TABLE 9.3—Terrestrial gastropods found naturally infected with *Parelaphostrongylus tenuis*

Reference	Location	Deer Density/km² (% Infected)	Overall Prevalence of *P. tenuis* % (Number of Gastropods Examined)	Mean Number Larvae/Gastropod (Range)	Species of Gastropods Infected[a]
Beach (1992)	New Brunswick, Canada	1.2–3.6 (50%–60%)	0.2%–2.0% (1960)	NR[b] (1–28)	*Zonitoides arboreus* (0.5% of 635), *Deroceras laeve* (1.5% of 589), *Discus cronkhitei* (1.6% of 249), *Pallifera dorsalis* (0.8% of 118), *Triodopsis albolabris* (9.5% of 21), *Strobilops labyrinthica* (5.3% of 38)
Gleich et al. (1977)	Central Maine (USA)	1.6–3.1[c] (63%–80%)	0.1% (1700)	2.0 (2)	*P. dorsalis* (0.8% of 225)
Kearney and Gilbert (1978)	Near North Bay, Ontario, Canada	2.0–6.4[d]	0.05%–0.12% (16,450)	2.4	NR
Lankester (1967)	Algonquin Park, Ontario, Canada	< 4[e] (41%)	1.0% (1540)	NR	*T. albolabris* (18% of 39), *D. cronkhitei* (2% of 256), *Z. arboreus* (0.4% of 562), *Succinea ovalis* (1.5% of 66), *D. laeve* (0.3% of 385)
Lankester and Anderson (1968)	Navy Island near Niagara Falls, Ontario, Canada	94 (61%)	4.2% (9940)	2.9 (1–97[f])	*D. laeve* (8.2% of 2434), *Zonitoides nitidus* (4.3% of 4719), *Anguispera alternata* (2.5% of 121), *S. ovalis* (4% of 195), *Cochlicopa lubrica* (0.4% of 249), *Deroceras reticulatum* (0.3% of 1,097), *Arion circumscriptus* (0.1% of 1064)
Lankester and Peterson (1996)	Near Grand Marais, Minnesota (USA)	19 (for 5 months) 4 (for 7 months) (80%)	0.1% (12,095)	3.2 ± 2.5 (1–7)	*A. alternata* (1.2% of 81), *D.cronkhitei* (0.4% of 968), *S. ovalis* (0.3% of 393), *D. laeve* (0.2% of 1212), *D. reticulatum* (0.03% of 6949)
Maze and Johnstone (1986)	Northcentral Pennsylvania (Elk State Forest) (USA)	12–15 (54%)	9.0% (808)	3.0 (1-44)	*T. albolabris* (20% of 70), *Triodopsis tridentata* (12% of 17), *Ventridens intertextus* (20% of 118), *D. laeve* (1.6% of 61), *Z. arboreus* (12% of 41), *D. cronkhitei* (15% of 143), *Stenotrema fraturnum* (12% of 50)

(*continued*)

TABLE 9.3 ***(continued)***

Reference	Location	Deer Density/km^2 (% Infected)	Overall Prevalence of *P. tenuis* % (Number of Gastropods Examined)	Mean Number Larvae/Gastropod (Range)	Species of Gastropods Infected[a]
Parker (1966)	Nova Scotia, Canada	2.6[g] (63%)	2.6% (509)	NR	*D. cronkhitei, Z.arboreus, D. reticulatum, Striatura exigua, Phylomycus carolinianus*
Pitt and Jordan (1994)	Northeastern Minnesota (USA)	4–5 (57%)	0.8% (744)	NR	NR
Platt (1989)	Northwestern Indiana (USA)	40 (over winter) (38%)	1.9% (736)	2.5-3.8 (1–26[f])	*Cochlicopa* spp (5.5% of 163), *D. cronkhitei* (1.2% of 252), *D. laeve* (2.8% of 71)
Raskevtiz et al. (1991)	Eastern Oklahoma (USA)	17 (39%)	8.0% (959)	65 larvae in 8/15 species	*Discus patulus* (0.8% of 129), *Helicina orbicula* (3.1% of 293),*Mesopmphix cupreus* (10% of 10), *Mesodon inflectus* (24% of 33), *Triodopsis divesta* (30% of 83), *Strenotrema strenotrema* (4.3% of 70), *Polygyra dorenilliana* (10% of 113), *Polygyra jacksoni* (8.8% of 194)
Rowley et al. (1987)	National Zoological Park near Front Royal, Virginia (USA)	NR	2.2% (670)	2.5 ± 2.9 (1–10)	*D. laeve* (1.5% of 338), *T. tridentata* (33% of 9), *Z. arboreus* (4.3% of 69), *P. carolinianus* (9% of 31), *Vallonia collisella* (20% of 5)
Upshall et al. (1987)	Near Fredericton, New Brunswick, Canada	2.3[g] (78%)	2.5% (569)	6.0	*D. laeve* (5.3% of 225), *Z. arboreus* (0.4% of 229), *D. cronkhitei* (2.2% of 45)
Whitlaw et al. (1996)	Northcentral New Brunswick, Canada	12 (in winter) 2-3 (in summer) (67%)	0.4% (10,343)	1.5 (1–3)	*Arion* spp. (0.04% of 2377), *D. laeve* (0.5% of 438), *D. cronkhetei* (0.02% of 4671)

[a]Number gastropods infected/number examined (%).
[b]NR, not reported.
[c]Gilbert (1973).
[d]Kearney and Gilbert (1976).
[e]Wilton (1987).
[f]In three different studies individual *D. laeve* had high numbers of larvae (97, 26).
[g]Whitlaw and Lankester (1994b).

farms, and by experimental infection. These include moose, wapiti (*Cervus elaphus canadensis*), red deer (*C. e. elaphus*), woodland caribou (*Rangifer tarandus caribou*), reindeer (*R. t. tarandus*), mule-deer (*Odocoileus hemionus hemionus*), black-tailed deer (*O. h. columbianus*) and black-tails × white-tailed deer hybrids, fallow deer (*Dama dama*), and the non-cervids, bighorn sheep (*Ovis canadensis*), domestic sheep and goats, llamas, guanocos, alpacas, camels, pronghorns (*Antilocapra americana*), eland (*Taurotragus oryx*), sable antelope (*Hippotragus niger*), and possibly bongo antelope (*Tragelaphus eurycercus*), scimitar-horned oryx (*Oryx dammah*), blackbuck antelope (*Antilope cervicapra*), and domestic cattle (see Anderson 1992; Rickard et al. 1994; Oliver et al. 1996; Yamini et al. 1997). The parasite causes neurologic disease in most of these, and occasionally becomes patent, passing small numbers of larvae, as in moose and wapiti.

Guinea pigs have proven to be useful laboratory hosts for investigating the hematological response to infection and the parasite's tissue migration route; a few L_3's may reach the central nervous system, develop to the subadult stage, and produce paresis (Anderson and Strelive 1966b; Spratt and Anderson 1968; Bresele 1990). A single worm was found in the brainstem of a domestic rabbit (*Oryctolagus cuniculus*) given 100 L_3's, but none was recovered from cotton-tails (*Sylvilagus floridanus*) or a swamp rabbit (*S. aquaticus*), each given 25 L3's (Nettles and Prestwood 1979).

PREVALENCE AND INTENSITY IN WHITE-TAILED DEER AND GASTROPODS. The reported prevalence of meningeal worm in white-tails varies widely (1%–94%) (see review by Anderson and Prestwood 1981 and additional reports by Foreyt and Trainer 1980; Beaulieu-Goudreault 1981; Kocan et al. 1982; Rau 1984; Upshall et al. 1987; Thomas and Dodds 1988; Dew 1988; Comer et al. 1991; Foreyt and Crompton 1991; Garner and Porter 1991; Bogaczyk et al. 1993; Jarvinen and Hedberg 1993; Pitt and Jordan 1994; Slomke et al. 1995; Davidson et al. 1996; Peterson et al. 1996; Gogan et al. 1997). This wide variation in prevalence may, however, be due mostly to sampling error. Instead, most or all white-tailed deer in a population may eventually become infected as was suggested several years ago by Karns (1967) and Behrend and Witter (1968). This was recently confirmed by Slomke et al. (1995) for a low density white-tailed deer population in northern Minnesota. Ninety-one percent of yearlings and 96% of animals 7–15 years old were infected. Lower percent infection has been observed in habitats near the extreme limits of the parasite's distribution (Kocan et al. 1982; Comer et al. 1991; Whitlaw and Lankester 1994b; Wasel 1995).

Underestimates of prevalence may be due to a lack of care and skill required to find adult worms in venous sinuses, the inclusion of traumatized heads that cannot be reliably examined, or sampling at the wrong time of the year. Young, infected animals available from hunters in autumn may not yet have either adults in head or first-stage larvae in feces. Their inclusio calculations may grossly underestimate the ov population prevalence. Considerable discrepancy occurs between prevalence determined by exami heads and that from feces (Anderson and Prestw 1981; Thomas and Dodds 1988; Bogaczyk 1990; ner and Porter 1991). Much of this is due to unisex occult infections that are not identified by fecal ex nation (Slomke et al. 1995). In addition, preval may be underestimated by examining feces in fall early winter when larval output by older animals its lowest and may have fallen below levels detec using the Baermann technique (Peterson et al. 1 White-tailed deer heads and feces are best collecte examination from February to April, and only standardized for age and season should be compar

The prevalence of infection does not appear to with host sex (Garner and Porter 1991; Slomke 1995; Peterson et al. 1996), although some e authors suspected this to be the case (Gilbert 1 Thurston and Strout 1978). There is some evidence overall sample prevalence in white-tailed deer vary annually in relation to summer precipit (Gilbert 1973; Bogaczyk 1990; Peterson and Lank 1991; Bogaczyk et al. 1993), but workers have fail find a consistent relationship between prevalence white-tailed deer population density (Karns Behrend and Witter 1968; Gilbert 1973; Brown Thomas and Dodds 1988; Bogaczyk 1990; Garne Porter 1991; Peterson and Lankester 1991; Boga et al. 1993).

Only the white-tailed deer fawn cohort is usef investigating environmental factors that may alter of transmission of the parasite. Significant annual ation in the prevalence of first-stage larvae measu fawns in late winter correlated best with the numl days prior to snow accumulation the previou (Peterson et al. 1996). Extended periods for tran sion in the fall were thought to increase the numl animals becoming infected and to reduce the num unisexual infections.

The intensity of adult *P. tenuis* in white-tailed usually is quite low. Anderson and Prestwood (reviewed the accumulated literature and reported sities of 1–20 worms with mean intensity ranging 1.5 to 8.7 worms. Mean intensity changes littl deer density or with age after 1 year (Behrend Gilbert 1973; Slomke et al. 1995). In fact, a thr number of adult worms may be reached that exceeded appreciably as white-tail densities a probability of infection increase. Slomke et al. found the same mean intensity of adult wor white-tailed deer confined year-round in a fence at a density of 30/km^2 (3.5 ± 1.8 worms) as in t a summer density of about 2/km^2 (3.0 ± 2 w Occasionally, individual white-tailed deer acquir numbers of worms (Prestwood 1970).

Although the number of adult worms change with white-tail age and time of year, the number

detectable in feces varies considerably. Young, recently infected white-tailed deer pass more larvae than older animals, and animals of all ages pass the greatest numbers of larvae in spring (Anderson 1963a; Anderson and Prestwood 1981; Slomke et al. 1995; Peterson et al. 1996; Forrester and Lankester 1998). The number of larvae passed in feces cannot be correlated, however, with the total number of adult worms in the cranium (Bogaczyk 1990) or with the total number of female worms present (Slomke et al. 1995). Although expected intuitively, such a relationship may be masked by the combined effects of infection age, season, location of female worms, and possibly, individual host immune response. For example, naturally infected fawns approaching 1 year old passed 132 ± 133 larvae/g of fresh feces and had 2.0 ± 1.2 adult worms in the cranium (Slomke et al. 1995), while an experimentally infected fawn with 3 females and 1 male worm passed 4800 larvae/g at 200 days postinfection (M.W. Lankester, unpublished).

Mean intensity of larvae in white-tailed deer feces provides a useful measure of the productivity of the parasite suprapopulation in an area and may be the best estimator of disease risk to cohabiting susceptible animals (Whitlaw and Lankester 1994b). However, of the many factors known to affect larval numbers, the most important is probably the method used to extract them from feces (Welch et al. 1991). Forrester and Lankester (1997a) demonstrated that the traditional Baermann technique may recover as few as 13% of the *P. tenuis* larvae actually present and does not provide a repeatable result (see Diagnosis section of this chapter).

The prevalence of *P. tenuis* in gastropods is probably determined primarily by white-tailed deer density, as well as by microclimatic conditions that favor snails and slugs. A large number of studies conducted throughout the range of *P. tenuis,* report an overall prevalence of infection in gastropods ranging from 0.01% to 9.0% and reaching 33% in certain species (Table 9.1). Generally, prevalences of < 1% are reported near the northern limits of white-tail distribution, while values of 1.9%–2.6% found in the Canadian maritime provinces and eastern United States may reflect greater white-tailed deer densities as well as a slightly warmer climate with longer seasons for gastropod activity. Higher values of 4.2%–9.0% are unusual and are reported only where white-tailed deer may be at exceptionally high densities or restricted in their seasonal movements (Lankester and Peterson 1996).

High prevalence in *Triodopsis* spp. suggests that this snail is attracted to fresh fecal material, although no such attraction was found for the closely related *Mesodon* sp. (McCoy 1997). Whether white-tailed deer actually ingest snails as large as mature adult *Triodopsis* spp., *Anguispira alternata,* and *Mesodon* spp., either for extra protein or mistakenly as mast, remains to be investigated. Infective larvae are thought to survive as long as their gastropod host. Consequently, more seasonal and annual fluctuation in prevalence can be expected in shorter-lived species like *D. laeve* than in *Zonitoides* and *Discus* spp. that live 2–3 years (Lankester and Anderson 1968).

Most snails and slugs contain only a few infective larvae (means of 2–6) (Table 9.1). Since the mean number of adult worms in white-tailed deer (2.8 ± 1.8) (Slomke et al. 1995) can be similar to the mean number of larvae per infected gastropod in the same area (3.2 ± 2.5) (Lankester and Peterson 1996), many infections in white-tailed deer may be the result of ingesting a single infected snail or slug. Prestwood and Nettles (1977) hypothesized that white-tailed deer may similarly acquire *P. andersoni* as a result of a single exposure. In three separate reports (Table 9.1), individual *D. laeve* were found to contain unusually large numbers of larvae (26, 75, and 97). These individuals probably lingered on fresh feces. Possibly the ingestion of such a heavily infected slug accounts for the rare reports of massive infection in white-tailed deer and some of the more severe cases of parelaphostrongylosis in susceptible hosts.

Although a large variety of wild gastropods is found naturally infected with *P. tenuis,* important species in field transmission will be those most frequently infected and most abundant in areas used by susceptible age classes of white-tailed deer. In the northern parts of the white-tail range, these species include the snails *Zonitoides* spp. and *Discus cronkhitei* and the slugs *Deroceras* spp. (Lankester and Anderson 1968; Lankester and Peterson 1996; Whitlaw et al. 1996). In addition, marked seasonal changes in the abundance of terrestrial gastropods will affect the relative importance of different species (Lankester and Peterson 1996). Reasonable estimates of the numbers and kinds of terrestrial gastropods encountered by deer can be obtained by sampling during damp weather conditions using corrugated cardboard sheets (Hawkins et al. 1998).

It is important to recognize in future studies that wild gastropods frequently contain a variety of larval and adult nematodes, some of which are easily mistaken for those of *P. tenuis.* For example, Gleich et al. (1977) found nematodes in 19% of *Pallifera* spp. and in 7% of gastropods overall, but only 0.1% had larvae of *P. tenuis.* Similarly, Lankester and Peterson (1996) found larvae of other nematodes species in 4% of a sample of over 12,000 gastropods, while only 0.1% had developing *P. tenuis* larvae.

TRANSMISSION AND ENVIRONMENTAL LIMITATIONS. The ability of L_1's to survive adverse natural conditions and remain infective to gastropods has not been thoroughly studied. In the laboratory L_1's survive constant, subzero temperatures several months (Lankester and Anderson 1968), but repeated freezing and thawing greatly reduces survival (Shostak and Samuel 1984), as does repeated drying and wetting at room temperature. The latter authors cautioned that some survivors that regain motility may have lost their ability to infect gastropods.

Survival of larvae beneath snow in northeastern Minnesota was relatively low (16%), despite moderated

and stable subnivean temperatures (-0.2° C to -2.5° C compared to ambient air temperatures of 6.5° C to –24.0° C) (Forrester and Lankester 1998). Even those larvae produced during the "spring rise" in mid-March experienced high mortality (70%), since winter conditions at this northern location continued until late April. Nothing is known of the ability of L_1's to survive summer or winter conditions in the soil. This information, along with more knowledge of the life span of various gastropods, is needed to determine when an area, previously occupied by white-tails, would be free of risk to other susceptible host species.

Transmission may be more likely in particular areas within white-tailed deer range, but whether distinct foci of infection exist is uncertain. Such areas might have a higher than usual density of infected gastropods and be used by young, susceptible white-tailed deer. Small foci were reported by Lankester and Anderson (1968) and Maze and Johnstone (1986), but none could be clearly identified in a study by Platt (1989). Based solely on the availability of intermediate hosts, Kearney and Gilbert (1978) concluded that all forested habitats in central Ontario had approximately equal potential to serve as transmission sites, while open areas have a lower potential except during late summer and fall. The use of open fields and meadows where gastropods were less numerous was thought to explain the persistence of wapiti in an area with infected white-tailed (Raskevitz et al. 1991). Rather than the existence of particular foci of infection, Anderson and Prestwood (1981) suggested that the large volume of vegetation eaten daily by ungulates probably explains the high prevalence of infection in white-tailed deer, even in areas with few infected gastropods. Lankester and Peterson (1996) examined this hypothesis in an area where most fawns became infected within 6 months of birth, yet fewer than 0.1% of gastropods were infected. Fawns were estimated to consume at least one infected gastropod within 51 days, even when infected gastropods were assumed to be distributed randomly and ingested accidentally. White-tailed deer wintering yards were not thought to be especially important in transmission, despite higher densities of infected gastropods (Lankester and Peterson 1996). Deer arrive in yards after snowfall, and by early spring, when gastropods become available, many are immune to reinfection.

Clinical Signs and Pathology in White-tailed Deer. Signs of parelaphostrongylosis are rare in white-tailed deer. Circling and progressive loss of motor function were described in a wild doe with 30–40 adult worms in the cranium (Prestwood 1970), and in an animal raised on a game farm that had 10 worms in the subdural space and others deep in the cerebral cortex (Eckroade et al. 1970). Even large experimental doses of *P. tenuis* in white-tailed deer resulting in as many as 65 adult worms in the cranium produce only transitory lameness or limb weakness (Anderson 1968; Pybus et al. 1989; M.W. Lankester and A.A. Gajadhar, unpublished data). These experimental results are remarkable in indicating that white-tails generally show no ill effects of the spinal cord tissue damage associated with the presence of many more developing worms than are ever likely to be encountered in nature. They also raise the possibility that susceptible species such as moose and wapiti may be less affected by the physical trauma caused by a few worms in the spinal cord than by the meningoencephalitis and perineuritis resulting from infection.

In the spinal cord of experimentally infected white-tailed deer fawns, worms develop in the dorsal horns of grey matter (Anderson 1965b,c). They usually are found in cell-free tunnels surrounded by compressed neural tissue. Malacia is absent except for tiny areas occasionally seen in white matter. The central canal remains undamaged. In white matter, scattered, single myelin sheath degeneration as well as degeneration and disappearance of axis cylinders are common. Infiltrations of eosinophils, lymphocytes, and plasma cells are observed in and on the dura mater, the epineurium, ganglion capsules, and other tissues of the epidural space. Mature worms accumulate in the subdural space over the brain where they are found free or partially embedded in the dura (Anderson 1963a). Areas on the surface of the dura are covered by yellowish exudate unevenly colored by blood. The dura may be thickened and inflamed with patches of eggs and larvae surrounded by giant cells and fibrous tissue visible in sections. Eggs are disseminated to all regions of the lung and found in all stages of development, usually singly or in groups of two or three (Anderson 1963a). Larvae are numerous in alveoli. Heavily infected areas of the lung are considerably altered with congested vessels, collapsed alveoli, fibrosed alveolar walls, petechiae, and collateral vessel formation. Numerous agranulocytes and giant cells are invariably applied to the remains of hatched eggs and clumps of eosinophils and macrophages with hemosiderin-like material are common.

Epizootiology in Abnormal Cervid Hosts. The severity of infection in hosts other than white-tailed deer generally is thought to be due to the higher proportion of invading worms that reach the central nervous system, their longer developmental period in the spinal cord, their resulting larger size and coiling behaviour, and frequent invasion of the ependymal canal (Anderson 1968). Occasionally, naturally infected moose and wapiti pass larvae, but in most abnormal hosts either the worms die or the host dies before infections become patent. Pathogenesis and clinical signs are known from infections produced experimentally as well as those acquired naturally.

MOOSE. Nearly 500 cases of moose sickness have been reported in the literature since the syndrome was first described by Thomas and Cahn (1932). The disease has been reported only in the Canadian provinces of New Brunswick (n = 27), Nova Scotia

(137), Quebec (84), Ontario (50), and Manitoba (12) and the northern states of Maine (69), Minnesota (97), and Michigan (13) where moose share range with infected white-tails (Anderson 1965a,b; Aho and Hendrickson 1989; Whitlaw and Lankester 1994a,b, Dumont and Créte 1996; M.W. Lankester and W.J. Peterson, unpublished). The frequency of the disease seems reasonably well correlated with the density of cohabiting white-tailed deer (Karns 1967; Gilbert 1974; Dumont and Créte 1996).

Despite a relatively large number of opportunistic reports of sick moose, only a few studies provide estimates of *P. tenuis* prevalence in wild moose populations. Smith and Archibald (1967) found adult worms in the crania of 5% of 115 clinically normal moose examined over a 4-year period in Nova Scotia and New Brunswick, while 80% of 45 moose showing clinical signs had worms. Similarly, of 153 moose examined in Maine over a 4-year period by Gilbert (1974), *P. tenuis* could be recovered from the cranium of 25% of those killed by poachers, 10%–15% of those killed by vehicles and other miscellaneous causes, and in 80% of those showing signs of parelaphostrongylosis. Thomas and Dodds (1988) found worms in the head of 6.5% of moose shot by hunters and dying of other causes.

In Minnesota, larvae presumed to be those of *P. tenuis* were found in 0.6% of 361 moose fecal samples (Karns 1977), in 0.3% of feces from 617 hunter-killed moose (M.S. Lenarz, personal communication: cited in Gogan et al. 1997), and in 5% of 22 field-collected, moose fecal samples from Voyageurs National Park (Gogan et al. 1997). Higher prevalences reported by Clark and Bowyer (1986) in moose feces in Maine (up to 31%) and by Thomas and Dodds (1988) in Nova Scotia (13%) could not be confirmed in subsequent studies (Upshall et al. 1987; McCollough and Pollard 1993). Estimating the prevalence of *P. tenuis* by examining fecal samples for larvae has serious limitations. Dorsal-spined larvae cannot be identified with certainty, and those of both *P. tenuis* and *P. andersoni* may be passed by moose (Lankester and Fong 1998). As well, the proportion of infected moose that pass larvae can vary. Karns (1977) found larvae in feces of 29% of moose diagnosed as being sick. In a sample of 27 sick moose examined by M.K. Lankester and W.J. Peterson (unpublished) in Minnesota, 15% were passing larvae. How many clinically normal moose, if any, pass larvae is unknown. Lastly, the habit of sick moose remaining for extended periods in the same area makes it difficult to avoid overrepresenting them in fecal collections.

The intensity of adult *P. tenuis* in naturally infected moose usually is very low (mean of ~2; range 1–10) (Anderson 1965a; Smith and Archibald 1967; Gilbert 1974; Thomas and Dodds 1988; M.W. Lankester and W.J. Peterson, unpublished). Animals showing severe signs may have as few as one grossly visible worm. Others may have none. Adult worms were found in the heads of only one-third of sick moose examined by Lankester (1974) and a presumptive diagnosis of parelaphostrongylosis was made based on scattered inflammatory and degenerative lesions in the meninges and parenchyma of the brain and spinal cord.

Moose of all ages can be affected, but reports of younger animals have tended to predominate (Anderson and Prestwood 1981; M.W. Lankester and W.J. Peterson, unpublished data). In this regard, the overhanging muzzle that becomes accentuated in older moose may reduce their feeding low to the ground where there is a greater likelihood of ingesting gastropods. On the other hand, Dumont and Créte (1996) noted that cases in calves were lower, proportionately (3%), than would be expected from their percentage in the population (28%).

Wild moose may show any or all of the following signs: swaying and weakness in the hindquarters, wide base stance of the legs, standing with weight forward on the front legs, tilting or turning of the head and neck to one side (torticollis), knuckling, overextension of the rear fetlock joints and spreading of the toes, circling, fearlessness, depression, rapid eye movements (nystagmus), apparent blindness, ataxia, paresis, difficulty in rising, inability to stand, and weight loss. Peterson (1989) noted the presence of abnormal antlers and kidney stones in moose displaying signs of moose sickness. Worms in moose are frequently found within or beneath the pia-arachnoid (Smith et al. 1964; Lankester 1974). In this location, they may more easily reenter nerve tissue of the brain causing clinical signs. This may explain the slight preponderance of clinical cases in mid- to late winter, several months after gastropods were available (Anderson 1965a,b).

Histopathological lesions in experimentally infected moose killed within 60 days of infection included focal traumatic malacia caused by developing nematodes in dorsal horns of the spinal cord, gliosis and giant cell response, disruption of the ependyma, neuronal loss and single-fiber myelin degeneration, and perivascular infiltrations primarily of lyphocyctes, plasma cells, and eosinophils (Anderson 1964a,b). In wild moose with parelaphostrongylosis, the brain is more extensively involved than the spinal cord (Smith and Archibald 1967; Anderson 1965a; Kurtz et al. 1966). Lesions in the brain parenchyma include cuffing with round cells; disrupted areas or tracts with swollen axis cylinders; gitter cells; congestion; infiltrations of eosinophils, lymphocytes, and plasma cells; and calcified remains of worms. Eggs and larvae may be found associated with the eyes or the roots of cranial nerves, on the leptomeninges, and in brain tissue. Only small glial scars and scattered areas of malacia, degenerating axis cylinders, and microcavitation occur in the spinal cord.

Historically, many authors have associated marked declines in moose populations and reports of sick moose with incursions by white-tailed deer (see Anderson 1972; Lankester and Samuel 1998). However, the implicit hypothesis that *P. tenuis* was the major cause of the declines was never tested (Nudds 1990) until Whitlaw and Lankester (1994a) attempted a retrospective study using published historical data from six jurisdictions where moose sickness has been repeatedly

seen. An inverse relationship between moose and white-tailed deer numbers was evident, with moose declining when white-tailed deer exceeded 5/km^2. However, despite a coincidence of relatively high white-tailed deer densities, moose declines, and reports of sick moose in at least 5 of 13 population cycles examined, these factors were not consistently related. Although the test was probably weakened by the poor reliability of opportunistic reporting of sick animals, the hypothesis could not be supported by available historical data. They concluded that the precise role of *P. tenuis* in past declines of moose may never be known.

In present times, white-tailed deer densities are relatively low in most areas shared with moose because of hunting and winter snow depths (Whitlaw and Lankester 1994b). Throughout much of Ontario where moose and white-tailed deer coexist, white-tailed deer numbers seldom exceeded 6/km^2 throughout the 1980s, and populations of both cervids were either stable or increasing moderately with only sporadic reports of neurologic disease in moose (Whitlaw and Lankester 1994b). However, moose densities were greatest when white-tailed deer were < 4/km^2 and varied inversely with the mean numbers of first-stage larvae being passed by white-tailed deer. In Voyageurs National Park, Minnesota, where no hunting occurs, white-tailed deer reached densities of 8/km^2 during the 1980s, yet no cases of parelaphostrongylosis in sympatric moose were reported (Gogan et al. 1997). At white-tailed deer densities approaching 13/km^2 in southern Quebec, the annual mortality rate of sympatric moose due to meningeal worm was estimated to be < 1% (Dumont and Créte 1996). Although moose still persisted, the disease was considered a limiting factor, diminishing their demographic vigour. Similar low estimates of moose mortality were made by Lenarz and Kerr (1987) in Minnesota. In 1985, moose were reintroduced into Michigan. Despite *P. tenuis* initially causing 38% of the observed mortality, the moose population continued to grow in the presence of infected white-tailed deer at 5/km^2 (Aho and Hendrickson 1989). Introduced moose experienced high twinning rates, and no wolf or bear predation was suspected. No hunting or poaching occurred. This experiment has demonstrated that a moose population coexisting at moderate white-tailed deer densities can increase, despite some mortality due to meningeal worm, at least while other factors are exceptionally favourable.

A belief that *P. tenuis* was invariably lethal to moose, probably led earlier authors to reason that moose appearing to cohabit successfully with white-tailed deer must be isolated spatially or temporally from infection. Rather compelling evidence thought to support this view included areas where both cervids existed but were separated at different altitudes during winter in response to snow depths (Telfer 1967; Kelsall and Prescott 1971), the existence of refugia where moose were thought to experience lower rates of infection (Telfer 1967; Gilbert 1974), and areas with considerable habitat heterogeniety thought to reduce overlap between moose and white-tailed deer (Kearney and Gilbert 1976). Nudds (1990) and Gilbert (1992) debated the relative strengths of these data, while more recent moose workers have failed to find strong evidence for the existence of such isolating mechanisms (Whitlaw and Lankester 1994b; Dumont and Créte 1996; Gogan et al. 1997).

If moose are not separated spatially from white-tailed deer and if their feeding habits do not differ substantially, particularly when young, they likely ingest similar numbers of infected gastropods when cohabiting. The number of larvae consumed would be low (Lankester and Peterson 1996), but the effects of such low doses on moose have only recently been investigated (M.W. Lankester, unpublished data). Each of two 5-month-old calves infected with 3 L_3's developed some lameness and hindquarter weakness after 6 weeks, but signs were hardly noticeable at 3 months when a single adult worm was found subdurally in each animal. Two moose infected at 9.5 months with 5 and 10 L_3's, respectively, showed no lasting locomotory signs, and only a single worm was found in one moose after 8 months, despite each having been challenged with 15 L_3's, 199 days after the initial infection. Two other moose given 5 and 15 larvae, respectively, showed persistent lameness and hindquarter weakness and had zero and three worms in the cranium when killed after 3 months. Apparently the severity of parelaphostrongylosis in moose is dose and age dependant. In addition, infection with low numbers of larvae, approximating those found in naturally infected gastropods, is not immediately lethal. Results also suggest that some moose can overcome such infections and that an acquired immunity may protect surviving individuals. Unfortunately, this experiment had to be terminated before the ultimate fate of animals with live worms still in the cranium could be determined with more certainty (M.W. Lankester, unpublished).

In light of this study, it is not altogether clear why some wild moose develop terminal neurologic disease with only one or two worms apparent in the cranium. An additional worm in a vital area within the brain or cord could be responsible, but experiments indicate that the immediate effects of small numbers of worms developing in the spinal cord can sometimes be overcome. Of greater consequence may be the trauma and inflammation caused by persistent adult worms in the subdural space or by those that reenter and oviposit in tissues of the brain as reported by Anderson (1965a). The outcome of infection may also be determined by an individual's innate and acquired immune response to larvae ingested throughout its life.

Overall, the impact of meningeal worm on moose in an area may in large part be a function of dose and age at first exposure for individuals as well as prior experience of older animals with the parasite. The density and age composition of the cohabiting white-tailed deer population will, in turn, determine the numbers of *P. tenuis* larvae being produced, provided that conditions are suitable for terrestrial gastropods. Since the

frequency of the disease is independent of moose numbers, the parasite cannot regulate moose populations in the strict sense of this word, but it may be an important limiting factor (Whitlaw and Lankester 1994a; Dumont and Crete 1996). Whether the parasite plays a significant role in the observed inverse relationship between moose and white-tailed deer numbers is still unclear. Moose numbers are also affected by changes in habitat and weather, hunting, predation, and other parasites such as winter tick (*Dermacentor albipictus*) (Lankester and Samuel 1998). The extent to which moose are limited by *P. tenuis* can only be determined by measuring survival and reproductive rates of individuals in relation to their experience with the parasite under various conditions. Although relatively low rates of overt disease are observed in moose at moderate white-tailed deer densities, the possible importance of subclinical effects cannot be discounted. For example, an interesting modeling exercise by Ives and Murray (1997) demonstrated that sublethal effects of a parasite on snowshoe hare can have a destabilizing effect through increased vulnerability to predation, making population cycles more likely.

WAPITI/RED DEER. Meningeal worm can cause debilitating neurologic disease and death in free-ranging wapiti, and it has probably limited the success of past wapiti reintroductions into eastern North America (Anderson and Prestwood 1981; Raskevitz et al. 1991). Nonetheless, despite sporadic cases of parelaphostrongylosis, a few native populations and some introduced herds do persist on range with infected white-tails (Samuel et al. 1992). Infected wapiti or red deer have been reported in eastern Oklahoma (Carpenter et al. 1973; Raskevitz et al. 1991), Pennsylvania (Woolf et al. 1977; Olsen and Woolf 1978, 1979), northcentral Pennsylvania (Devlin and Drake 1989), Michigan and Virginia (Anderson and Prestwood 1981), and Manitoba (Pybus et al. 1989).

Prevalence and intensity of infection in wapiti in areas with sympatric infected white-tailed deer are not well documented. However, contact rates with the parasite can be fairly high while cases of overt disease are less frequent. Histological lesions suggestive of infection were seen in 34% of clinically normal wapiti sampled over a 5-year period in Pennsylvania, but only 11 cases of neurologic disease were recorded (Olsen and Woolf 1979). Infection was most frequent in 1.5 to 2.5-yr-old animals. Four sick wapiti with a history of circling, ataxia, adipsia, or vision impairment were seen within a year or two of being released in eastern Oklahoma (Carpenter et al. 1973). Most were yearlings, and each had 1–3 adult *P. tenuis* in tissues of the brain. The parasite is presumed to behave similarly in red deer.

Experimentally, the development of worms in wapiti is similar to that in white-tailed deer (Anderson et al. 1966). Worms and microcavitations were seen mostly in dorsal horns of grey matter along the entire length of the spinal cord; some worms entered the central canal. Although a few lesions were found in the medulla, choroid plexus, and cerebellum, their relative scarcity in the brain, compared to that in the spinal cord, is in accordance with results seen in experimentally infected moose (Anderson 1964b). As in moose, developing worms stayed an abnormally long time in the spinal cord, but tissue invasion and heavy infiltrations of lymphocytes, eosinophils, and plasma cells in the epineurium and connective tissue surrounding spinal nerve roots were more marked than in moose (Anderson et al. 1966).

Lesions seen in naturally infected wapiti with adult worms in the cranium consisted mostly of meningitis with focal, disseminated areas of lymphocytes, macrophages, eosinophils, and some giant cells (Carpenter et al. 1973). Adult nematodes were found only in the meninges and elicited little inflammatory response. Lesions in the brain and spinal cord included mild cuffing and gliosis with little reaction visible around clumps of nematode eggs and larvae in brain parenchyma. There was no evidence of nematode-induced trauma as seen in the cord of experimental animals by Anderson et al. (1966).

The severity and outcome of infection in wapiti is dose dependent (Samuel et al. 1992). All animals (2 or 7 months old) given 125 or more L_3's died, while only six of eight given 25 or 75 L_3's showed neurologic signs (two died). Several elk shed L_1's in their feces 78–165 days postinfection. Five given 15 larvae showed no clinical signs nor shed larvae, even though two animals had 2 and 3 adult worms in the cranium when killed up to 158 days postinfection. Clearly, some wapiti can resist or recover from doses of infective larvae (Anderson et al. 1966; Samuel et al. 1992) that are much greater than those likely to be encountered in nature. Nonetheless, mortality of wapiti is probably related to the number of infective larvae ingested, the age at infection, and possibly the specific damage caused by worms within the central nervous system. It has yet to be demonstrated whether a degree of acquired immunity will, in time, reduce observed herd mortality following an introduction.

Potentially, wapiti could introduce *P. tenuis* to areas where white-tailed deer are presently free of infection (Samuel et al. 1992). Although only a few larvae appear intermittently in the feces of experimentally infected elk (Anderson et al. 1966; Welch et al. 1991), both the worm and the host are long-lived, thereby increasing the potential for the parasite to become established. In nature, the presence of dorsal-spined larvae of *P. tenuis* have been presumed in wapiti feces in Minnesota (Karns 1966) and proven in samples from central and southwestern Manitoba (Pybus et al. 1989). There is, however, no evidence that *P. tenuis* can persist in wapiti populations without the continued presence of white-tailed deer.

CARIBOU/REINDEER. There are no reports of *P. tenuis* in free-ranging caribou but there is considerable evidence that caribou and reindeer are particularly suscep-

tible to meningeal worm. The parasite has been suspected of being a factor in the failure of several caribou introductions in areas with white-tailed deer, including the Cape Breton Highlands, Nova Scotia (Dauphiné 1975); Red Lake Refuge, Minnesota (Karns 1979); Liscombe Game Sanctuary, Nova Scotia (Benson and Dodds 1977); and Baxter State Park, Maine (McCollough and Connery 1990). After reviewing 33 reintroduction attempts in eastern North America, Bergerud and Mercer (1989) concluded that caribou cannot be reintroduced to ranges where white-tailed deer have a high frequency of meningeal worm infection. Presently, there are few places in eastern North America, with the exception of eastern Quebec, where infected white-tails even threaten to encroach on caribou habitat.

Even holding caribou or reindeer in enclosures in areas occupied by white-tailed deer has had dire consequences in Ontario (Anderson 1971b), central Wisconsin (Trainer 1973), Virginia (Nichols et al. 1986), and Maine (McCollough and Connery 1990). Anderson (1971b) provided a detailed account of the fate of a shipment of reindeer from Norway placed in an enclosure that had been recently constructed on white-tailed deer range. Neurologic disease was first seen 3 months after their release, and within 5 months, 8 of the 12 were showing signs. In Wisconsin, all of 14 woodland caribou (including 10 adults) released into a 2640 ha enclosure with 600 white-tails, died within 6 months. Trainer (1973). Typically, caribou that were otherwise in good condition showed lumbar weakness, posterior ataxia, circling, severe torticollis, and bulging eyes.

Anderson and Strelive (1968) experimentally infected each of two woodland caribou calves with 200 L3's of *P. tenuis*. Slight neurological signs began 5–7 days postinfection. One died shortly thereafter of a mycotic infection while the second showed progressively severe signs including severe ataxia with knuckling and posterior weakness and was euthanised 29 days postinfection. Developing worms were in dorsal horns of grey matter of the spinal cord and in the medulla oblongata and brain stem. Traumatic lesions and worms were unusually numerous in lateral and dorsal funiculi of white matter, compared to other experimentally infected cervids.

The feasibility of reintroducing caribou into parts of their former habitat now occupied by white-tails has been examined more recently by Gogan et al. (1990) and Pitt and Jordan (1994), but no such introductions have been attempted. This is probably contraindicated unless white-tailed deer can be kept at extremely low densities and caribou can be protected from most other causes of mortality.

MULE DEER/BLACK-TAILED DEER. There are no reports of parelaphostrongylosis in wild mule deer, despite their proximity to infected white-tailed deer in areas such as southwestern Manitoba. Nonetheless, their susceptibility, as well as that of black-tail χ white-tail hybrids, has been demonstrated experimentally (Anderson et al. 1966; Nettles et al. 1977a; Tyler et al. 1980). Mule deer given 75–200 larvae showed neurologic signs after 35 days that progressed rapidly to paralysis within 80 days postinfecton (Tyler et al. 1980). All died or had to be euthanised, except one adult that showed only slight signs before recovering. Tyler et al. (1980) suggested that mule deer show a weaker cellular response to *P. tenuis* than black-tailed deer as described by Nettles et al. (1977a). Anderson et al. (1966) noted that worms from an experimentally infected mule deer were fertilized, suggesting that *P. tenuis* might become patent in this host if individuals survived long enough. Nematode eggs were found in the cranial dura of a mule deer killed at 87 days postinfection by Tyler et al. (1980), but no larvae were found in lungs or feces.

Histological findings in an experimentally infected mule deer fawn were considered noteworthy since some worms were still in nerve tissue 62 days postinfection (Anderson et al. 1966). Traumatic lesions were intermediate in size and number between moose and wapiti, and white-tailed deer. Cellular infiltration of the neural parenchyma was slight or absent, but worm and tracks left by worms were relatively numerous in the brain. Lesions found in fawns were also most severe in the brain, while those in adult mule deer were more marked in the spinal cord (Tyler et al. 1980). These authors concluded that adult mule deer are more likely to succumb within 40 days to the initial effect of the parasite developing in the spinal cord, whereas fawns may survive this phase, only to have signs reappear later, possibly when large adult worms reenter brain tissues.

Black-tailed deer and their hybrids will not prosper on range with appreciable numbers of infected white-tailed deer (Nettles et al. 1977a). A herd brought to Tennessee grew in number and rarely had sick animals as long as they were held in an enclosure with relatively few white-tailed deer. When some were released into an area where white-tails were increasing, neurologic disease was more frequent, and numbers steadily declined. Black-tails found dead or unable to stand had up to three adult *P. tenuis* in the cranial and spinal subdural space, in the lateral ventricle, or associated with the optic nerve. Multiple foci of malacia, gliosis, and microhemorrhage were seen mostly in white matter of the brain and spinal cord. No eggs or larvae were detected in lungs or feces.

FALLOW DEER. Parelaphostrongylosis has been reported in fallow deer on a game ranch in Georgia (Kistner et al. 1977) and in the Land between the Lakes area bordering Kentucky and Tennessee (Nettles et al. 1977b). In one instance the rapid onset of hindquarter weakness and paresis was seen in adult deer following strenuous capture efforts. Up to four adult worms were found in the cranial and spinal subdural space of other animals found with advanced neurologic impairment but no eggs or larvae were observed in lungs or feces. The persistence of fallow deer in the Land between

Lakes area with white-tailed deer at 13/km² later led Davidson et al. (1985) to hypothesize that fallow deer may have a degree of innate resistance to *P. tenuis* and that lightly infected individuals may acquire protective immunity against reinfection. Evidence supporting this idea included mild degenerative and inflammatory central nervous system lesions (considered indicative of prior *P. tenuis* infection) in several adult animals that were otherwise normal and in good physical condition.

Histological lesions in fallow deer showing neurologic signs include thickening and chronic lymphocytic inflammation with mineralization of the dura and microcavitations, and lymphocytic and eosinophillic cuffing, within the cervical and lumber cord (Kistner et al. 1977). Scattered foci of malacia, gliosis, microhemorrhage, and mononuclear cuffing are evident in brains (Nettles et al. 1977b). Small round nodules (2–3 mm diameter) visible on the surface of the cord represent granulomatous accumulations of mononuclear cells and often surround cross sections of dead nematodes.

Pybus et al. (1992) infected six fallow deer fawns with 25 or 150 L_3's, and all died. The three fawns given the higher dose died of peritonitis 6–23 days postinfection. Those given lower doses showed progressive paralysis and had to be euthanized 54–67 days postinfection; a mean of ~20 adult worms was recovered from the nerve tissue and subdural space of the central nervous system. A strong lymphoid response and the presence of dead worms were considered evidence of some innate immunity. Small, fleshy lymphoid nodules were seen along the thoracic cord and epidurally around nerve roots, as were widespread, multifocal meningitis and myelitis of the central nervous system. Adult worms remained in the spinal grey matter and cerebral white matter well after 40 days when they leave the cord of white-tailed deer.

Sporadic mortality can be expected in fallow deer held on farms with infected white-tails. Survival of individuals will probably depend on the number of infective larvae ingested, possibly the age at first exposure, and the time elapsing between reinfection. Although fallow deer have never been known to pass *P. tenuis* larvae, the feces of few survivors of infection have been examined. Caution is urged in translocating fallow deer from enzootic areas (Pybus et al. 1992).

Diagnosis. Recovering adult worms from the central nervous system is presently the only way to confirm infection with *P. tenuis*. Dimensions, particularly of the male spicules and gubernaculum, will distinguish *P. tenuis* from close relatives (Carreno and Lankester 1993). Clinical neurological signs in susceptible species held near white-tails are suggestive of infection, as is the presence of nematode eggs (50 (m diameter) and larvae in washings of the cranium or in histological sections of central nervous system tissues.

Dorsal-spined larvae in cervid lungs or feces are not diagnostic of *P. tenuis* infection. The dimensions of the first-stage larvae of several closely related species are similar (Prestwood 1972; Pybus and Shave 1984; Lankester and Hauta 1989). The Baermann funnel technique is unreliable for detecting and quantifying larvae in feces, and a more sensitive method using fecal pellets held in screen envelopes and submerged in water-filled, straight-sided beakers has been described (Forrester and Lankester 1997a). Forrester and Lankester (1997a) also emphasized the importance of expressing numbers of larvae on a dry weight basis since the weight of "fresh" feces changes rapidly in air. Even this improved methods has limitations. A fecal test cannot be relied upon to identify those animals that pass larvae in very low numbers, or only intermittently (Welch et al. 1991). In addition, a fecal test is of no diagnostic value in a case of clinical illness due to unisexual infection. Apparatus used in fecal examinations is easily contaminated. Larvae from previous samples can remain viable on glassware, but a hot soapy wash and a vigorous alcohol rinse will effectively remove them (Whitlaw and Lankester 1995). In the absence of fecal samples, washes of the oral cavity can be used to detect white-tailed deer passing larvae (Slomke et al. 1995).

Larvae from feces can be used to infect gastropods and to produce L_3's. The distinctive C- or J-shape assumed by lungworm larvae when they are heat-relaxed helps to distinguish L_1's and L_3's from other nematode larvae that occur commonly in fecal material and in gastropods, respectively (Anderson 1963a). The dimensions of L_3's, however, also overlap with those of closely related species, and the size and position of a dorsal bump near the tip of the tail, considered to be diagnostic (Ballantyne and Samuel 1984), may be too variable in this and other species to be useful (Lankester and Hauta 1989). Many of the problems associated with the identification of larvae may be superseded by the application of molecular techniques.

Progress has been made using polymerase chain reaction (PCR) to identify elaphostrongyline larvae in feces (Gajadhar et al. 2000). Amplification of ITS-2 DNA of both L_1's and L_3's, as well as adult worms, allowed the separation of *Parelaphostrongylus* spp. from closely related genera. Available primers also distinguished all three species of the genus: *P. tenuis, P. odocoilei,* and *P. andersoni.*

Hematology and blood chemistry are of limited value in detecting infection. Eosinophilic pleocytosis of the cerebrospinal fluid was used in conjunction with clinical signs to make a presumptive antemortem diagnosis of meningeal worm infection in llamas in an endemic area (Lunn and Hinchcliff 1989). However, Rickard et al. (1994) concluded that cerbrospinal fluid and serum chemistry values were too variable to be of diagnostic value in llamas, as was concluded for goats and white-tailed deer (Dew et al. 1992).

The lack of a reliable conventional test for *P. tenuis* infection in cervids has stimulated considerable interest in the development of a blood test using immunological and molecular techniques. Dew et al. (1992), using antigen extracts from adult *P. tenuis* in an enzyme-linked immunosorbent assay (ELISA), demonstrated antibodies in both serum and cerebrospinal fluid of two

goats, but only in cerebrospinal fluid of two white-tail fawns, 4–8 weeks after infection. Using similar methods, Duffy et al. (1993) detected a serum antibody response in two experimentally infected white-tail fawns 75 days after they received 20 *P. tenuis* L_3's, and in nine naturally infected white-tailed deer.

Using sera obtained from rabbits immunized with *P. tenuis* soluble extracts, Neumann et al. (1994) identified two larval (L_3) and seven adult somatic antigens of *P. tenuis* that differed from those in *Dictyocaulus viviparus* and *Trichinella spiralis*. A continuation of this work led to detection of serum antibodies to *P. tenuis* antigens in wapiti (Bienek et al. 1998). When reactivity of sera was tested using an ELISA, larval and adult antigens were consistently recognized by serum from wapiti given 300 L_3's, but only larval antigens were recognized by those given 15. When these sera were further tested by immunoblot analysis, samples (collected from elk with adult worms in the central nervous system) consistently recognized the 25–27, 28–30, and 34–35 kDa antigens of infective larvae after 83 days. However, several *D. viviparus* molecules also were recognized by antibodies directed at *P. tenuis*.

Recent studies show continued progress toward a more sensitive and specific blood test for *P. tenuis* in white-tailed deer using excretory-secretory and somatic antigen preparations from L_3's and somatic antigens from adult worms (Ogunremi et al. 1999a,b). Larval preparations, particularly excretory-secretory antigens, were superior in that they detected infections earlier and more consistently, while somatic antigens prepared from adult worms failed to detect all *P. tenuis* infected animals. This work also revealed considerable cross-reactivity between unfractionated antigen preparations of *P. tenuis* and sera from other cervids infected with the related nematodes *P. andersoni* and *E. rangiferi*. Anti-*P. tenuis* L_3 antibodies were detected as early as 21 days after infection of white-tailed deer given as few as six infective larvae (and later found to have only three adult worms in the cranium). Immunoblotting demonstrated that a total of six *P. tenuis* antigens were recognized, but only one, a 37 kDa protein present in both larval and adult antigen preparations, reacted specifically with serum from infected white-tailed deer. This antigen may be indistinguishable from the 36 kDa protein identified by Neumann et al. (1994) and may be unique to *P. tenuis*. Its reliability in a routine serological test is being examined more closely (O. Ogunremi et al., unpublished).

A satisfactory blood test for *P. tenuis* in white-tailed deer and wapiti requires a high level of sensitivity, allowing early detection of lightly infected and prepatent animals, and a degree of specificity that will not produce false positives in animals infected with other parasites. With helminth infections these standards are difficult to meet and will require rigorous field validation. Yet, such a test will be of great value in veterinary practice and wildlife management.

Immunity. An acquired or concomitant immunity following low-dose infections in white-tailed deer (Slomke et al. 1995) and fallow deer (Davidson et al. 1985) is suggested by field studies but has not been confirmed experimentally. Protection against a challenge infection with *P. tenuis* may also occur in moose (M.W. Lankester, unpublished). The nature of this apparent protection is just beginning to be understood. Antibody titers against larval *P. tenuis* antigens continued to increase in some infected white-tailed deer throughout 147 days of experimentation but declined in others after 2 months (Ogunremi et al. 1999a). This decline might be expected as worms mature to the adult stage. However, if the 37 kDa antigen found in both L3's and adult *P. tenuis* are similar, either adult worms or repeated exposure to L_3's in nature may maintain or continually boost the antibody response in many animals (Ogunremi et al. 1999b).

Innate differences in the susceptibility of various cervids to *P. tenuis* apparently exist. In part this is reflected by the relative success of larvae in reaching the central nervous system. For example, at least when relatively high doses are given (> 150 L_3) about 1 of every 5 larvae given to moose reaches the spinal cord to begin development while only 1 in 20 do so in white-tailed deer (Anderson 1963a, 1964a, 1965c; M.W. Lankester, unpublished). Host species with the least innate defense against migrating larvae can be expected to succumb most frequently to low-level, natural infection when sharing range with infected white-tailed deer. Rickard et al. (1994) recognized that resistance to the parasite, whether innate or acquired, appears to be more effective when animals are exposed to few infective larvae. Although the minimum dose required to produce sustained neurologic disease is unknown for most susceptible hosts, available field and experimental data suggest that caribou, mule deer, and black-tailed deer are the most likely to exhibit signs of parelaphostrongylosis following exposure to low-level infection under field conditions. The next most susceptible hosts are moose, followed by fallow deer and wapiti and red deer. Similarly for domestic species, llamas are more susceptible than goats and goats more than sheep. A dearth of cases in domestic cattle suggests that they may be the least susceptible, yet exotic bovids housed near white-tails clearly are vulnerable.

Evidence of age immunity is equivocal. Although parelaphostrongylosis tends to be seen more frequently in young moose, older animals also become infected (Whitlaw and Lankester 1994a; Dumont and Créte 1996). When adult moose were introduced into Michigan, 38% of mortality seen over the first few years was due to *P. tenuis* (Aho and Hendrickson 1989). Thereafter, fewer sick animals were reported, and the herd continued to grow. Dispersal of animals and decreased surveillance could have accounted for fewer reports of disease, but some of the surviving animals may have acquired a degree of protection. Disease is reportedly seen most frequently in younger wapiti (Olsen and Woolf 1979).

A strong immune response by white-tailed deer might also explain in part why *P. tenuis* has not spread

westward. As prevalence and intensity drop in drier grassland habitat that is marginal for transmission, an increasing number of white-tailed deer may acquire only a single worm and become immune. A high proportion of single-sex infections producing no larvae would depress parasite productivity and contribute to the low prevalence often seen near the parasite's distributional limits (Kocan et al. 1982; Whitlaw and Lankester 1994b; Wasel 1995).

Control and Treatment. Rates of disease may be controlled in cohabiting wild cervids by reducing white-tailed deer numbers, through liberal hunting, for example. Risk of infection by captive stock can be reduced by the use of white-tailed deer–proof fencing and gravel or paved barriers treated with molluscicides. Zoos should choose neonatal white-tails when acquiring new stock. Otherwise, susceptible species should be separated from white-tailed deer by mollusc barriers. It is not known how much time must elapse before ground that has previously held infected white-tailed deer is safe. However, some *P. tenuis* larvae probably live in the soil at least 1 year, and some snail hosts live 2 or 3 years. Small enclosures with little or no ground vegetation can probably be freed of risk sooner by the replacement of soil or by tilling to promote drying.

Considerable effort has been made by owners and clinicians to save valuable exotic and domestic species, particularly llamas. Treatments have included various anthelminthics (levamisole, albendazole, diethylcarbamazine, subcutaneous ivermectin, oral fenbendazole, and intramuscular flunixine meglumine) as well as steroids and anti-inflammatory agents. None of these has been tested in controlled studies, but when used with good supportive care, they may contribute toward recovery, at least of lightly infected animals (Krogdahl et al. 1987; Lunn and Hinchcliff 1989; Rickard et al. 1994). Kocan (1985) demonstrated that ivermectin (at 0.1–0.4 mg/kg) will protect white-tailed deer and fallow deer if given 24 hours after experimental infection with *P. tenuis*. If not given until 10 days after infection, worms develop normally. By 10 days, migrating larvae have entered the spinal cord and appear to be protected by the so-called blood-brain barrier. Treatment has no effect on adult worms already in the central nervous system but depresses the number of larvae developing in the lungs and being passed in feces. Larvae reappear in feces, however, within a month of treatment (Kocan 1985). Ivermectin combined with banamine shows some promise in stopping the progression of signs in sick llama, although controlled studies have yet to be conducted (A. Kocan, personal communication).

Domestic Animal Health Concerns. Sheep have some innate resistance to infection. Reports of parelaphostrongylosis are infrequent, although some may go unrecognized or be misdiagnosed (Anderson 1965b). Sporadic cases of neurologic disease in sheep attributed to *P. tenuis* have been reported in New Hampshire, Connecticut, West Virginia, and Minnesota (Anderson and Prestwood 1981; Jortner et al. 1985; O'Brien et al. 1986). Morbidity in infected flocks has ranged from 2% (Alden et al. 1975) to 59% (Jortner et al. 1985). The worm does not mature in sheep, and spontaneous recovery from clinical signs has been observed (Alden et al. 1975). Progressive hind limb weakness leading to total paresis has been produced experimentally in lambs given ≥ 150 larvae (Anderson and Strelive 1966a). Cross sections of worms or their remains were seen in dorsal and ventral horns of grey matter with microcavitations, swollen axis cylinders, demyelination, giant cells, and gliosis in lateral and ventral funiculi of white matter. The amount of trauma to the central nervous system was surprisingly slight in view of the severity of signs, even in animals receiving 1000 L_3's. The authors suggested that worm secretions, excretions, or breakdown products of moribund and dead worms might account for some of the signs observed. In a study by Pybus et al. (1996), each of 12 domestic sheep lambs received 15–300 larvae; only 1 lamb (given 125 L_3's) showed mild transitory signs.

The response of bighorn sheep to *P. tenuis* is similar to that of domestic sheep (Pybus et al. 1996). Bighorns resist light infections but show neurologic signs or die if exposed to high numbers of larvae. In both domestic and bighorn sheep, most migrating larvae seem to be killed before reaching the central nervous system, thereby avoiding fatal damage to the host (Pybus et al. 1996).

Goats are somewhat more susceptible to meningeal worm infection than sheep. Neurologic disturbance caused by *P. tenuis* in naturally infected goats has been reported in New York, Texas, and Michigan (Anderson and Prestwood 1981; Kopcha et al. 1989). Infected animals usually were in good condition but frequently became separated from the flock (Guthery and Beasom 1979). They often stood in a "humped up" position and exhibited posterior weakness or ataxia that predisposed them to accidental death and coyote predation. Central nervous system lesions consisted of scattered malacic areas with adjacent clusters of gitter cells and blood vessels cuffed with lymphocytes and occasional eosinophils and plasma cells. One kid given only 50 larvae developed progressive hind limb weakness after about 40 days and died (Anderson and Strelive 1969, 1972). Goats given doses of 200 larvae or more, developed fatal necrotizing colitis and bacterial peritonitis within about a week. Worms can reach the adult stage in the central nervous system of this host. Similar results were reported by Dew et al. (1992).

Parelaphostrongylosis is either rare or largely overlooked in domestic cattle. A 3-month-old calf off pasture in Michigan was recumbent with a suspected thoracolumbar spinal cord lesion (Yamini et al. 1997). A coiled worm seen in histological sections of the lumbar region was associated with extensive vacuolation, necrosis, disintegration and swelling of axons in the funiculi, gitter cells in grey matter, and multifocal lymphoplasmacytic and eosinophilic, perivascular cuffing. In Virginia, a 7-month-old heifer presented with acute-

onset, rear-limb ataxia that progressed over 10 days to sternal recumbency (Duncan and Patton 1998). Sections of coiled nematodes resembling *P. tenuis* were present in nerve parenchyma of cervical and lumbar regions of the spinal cord. Perivascular, eosinophilic, and lymphoplasmacytic infiltrates were seen in the meninges and in white and gray matter, as were tracts and varying degrees of axonal degeneration at all levels of the cord. Grayish-white nodules (up to 7 mm diameter) visible grossly at the surface and within cervical, thoracic, and lumbar regions were characterised microscopically as nodular lymphoid hyperplasia.

Exotic Bovids and Camelids. Meningeal worm infection was confirmed in one, and suspected in a second, adult sable antelope in Virginia where white-tailed deer frequented the fence line of a zoological park (Nichols et al. 1986). Both animals showed a rapidly progressing hind limb ataxia. Hemorrhage and perivasculitis were seen in the dura over the brain and spinal cord as well as tracts, cuffing with lymphocytes, plasma cells, and eosinophils in nerve tissue; remains of a nematode were seen in the medulla of one animal. Oliver et al. (1996) reported a cluster of cases of neurologic disease in blackbuck antelope on two game farms in southwestern Louisiana that also held white-tailed deer. Clinical signs included a protracted course of weakness, staggering, trembling, torticollis, and eventual recumbency. Adult nematodes identified as *P. tenuis* and nematode larvae were found in the meninges and neural parenchyma of some animals; others were diagnosed on the basis of clinical signs and histological examination. Lesions in the meninges were remarkably slight, with perivascular cuffing and a few foci of granulocytic and lymphocytic infiltrates surrounding larvae. Foci of necrotic cells, glial cells, and areas of swollen axons were seen in the cerebral hemispheres. Sections of a worm were seen in the dorsal horn of gray matter of the spinal cord. Blackbuck antelope are a commonly raised exotic species in southwestern Louisiana and are often allowed to range freely with white-tailed deer on game farms (Oliver et al. 1996). Either infection is not widespread in white-tailed deer, or neurologic disease caused by *P. tenuis* may until recently have gone unnoticed.

Llamas and their relatives are susceptible to *P. tenuis* at doses that can be acquired on pastures frequented by white-tailed deer. Reports in New York (Brown et al. 1978), Ohio (Baumgartner et al. 1985), Minnesota (O'Brien et al. 1986), Virginia (Krogdahl et al. 1987), and Wisconsin (Lunn and Hinchcliff 1989) may underrepresent the frequency of cases in routine veterinary practice. The variety of camelids frequently held in zoological parks should be considered at risk unless isolated from infected white-tails.

The disease progresses rapidly and is often fatal. Signs include head tilting, arching of the neck, incoordination, difficulty rising, posterior paresis, and gradual loss of weight (Brown et al. 1978; O'Brien et al. 1986). Adult nematodes may be found associated with hemorrhage in the cranial meninges. Microscopi lesions in the brain and along much of the spinal cor include swollen and demyelinated axons, necroti tracts with debris, perivascular cuffing, and small cav tations in white matter surrounded by macrophages an glial cells (O'Brien et al. 1986). Experimentally, adu llamas given 5–7 infective larvae develop signs of neu rologic deficit with incoordination and hypermetr about 50 days after infection (Foreyt et al. 199 Rickard et al. 1994). Younger animals were affecte first. Two of six animals survived after showing onl slight neurologic signs; a dead nematode was found the central nervous system of one when the experime was terminated at 146 days postinfection (Rickard et a 1994). The presence of adult nematodes was associate with severe meningoencephalomyelitis and eosin philia of cerebralspinal fluids. Histological lesio were found primarily in the cervical spinal cord ar consisted of nonsymmetrical microcavitations of gre matter, and spongiosis of white matter accompanied b gliosis, infiltrates of lymphocyctes, and some plasn cells, histiocytes, and eosinophils. Llamas are consi ered to pose little risk of spreading meningeal worm nonendemic areas, since either they or the worms us ally die before infections are patent (Foreyt et al. 199 Rickard et al. 1994).

Management Implications. Every effort should b made by government regulation and game ranchi industry practice to prevent the introduction of *P. tenu* into western North America. The highly adaptab white-tailed deer presently flourishes in a variety habitats throughout the western United States a Canada and shares range with mule deer, black-tail deer, moose, wapiti, woodland caribou, and pron horns, all of which are susceptible to par laphostrongylosis. Currently, meningeal worm absent from western North America, but there is no re son to believe that conditions there are unsuitable f transmission if it were to arrive there with infect cervids. White-tails from enzootic areas represent t greatest threat of accidental introduction, but wapi and possibly other cervids, could be responsib (Samuel et al. 1992). To prevent such an occurrenc strict interstate/interprovincial and international mo toring of all ungulate translocations in conjunction w a reliable test are needed to exclude *P. tenuis*–infect animals (de With et al. 1998).

Ecosystyem restoration projects that involve t reintroduction of extirpated species are highly pub cized events and normally are not undertaken light Their failure can have high economic as well as poli cal costs. The complete failure of past attempts to intr duce caribou, reindeer, and black-tailed deer i enzootic areas should clearly discourage any futu efforts, unless white-tails are virtually absent and a guaranteed to remain so. A definitive assessment h yet to be made on the advisability of reintroduci moose into areas where *P. tenuis* occurs. The curre experiment in upper Michigan will provide valual

information if the interest and financial support needed to monitor white-tailed deer density and the growth of the moose herd can be sustained. The persistence of a few localized wapiti herds within white-tailed deer range has recently encouraged new introductions of several hundred wapiti from Alberta into Ontario and Kentucky. Introduced moose and wapiti will experience some initial mortality that may later diminish and involve mostly immunologically naive recruits to the population. Long-term monitoring of white-tail densities, intensity of *P. tenuis*, growth of the introduced population, and serological evidence of contact with the parasite will help determine the likelihood of success for future reintroductions.

The sizes of indigenous moose populations historically have varied inversely with white-tailed deer over the medium to long term, but the role of *P. tenuis* in these fluctuations still is not fully understood. The impact of the parasite may be relatively low in areas of eastern North America where range of moose and white-tailed deer overlap. Here, white-tailed deer numbers are periodically reduced by severe winters, and most populations are hunted. However, white-tail numbers are less restricted in parks and areas with extensive secondary forest succession following commercial harvesting or fire. Local conditions allowing increased white-tailed deer densities predictably will increase the number of sick moose. Possible subclinical effects of *P. tenuis* ultimately may prove to be important in understanding the long-term interaction between moose and infected white-tailed deer.

PARELAPHOSTRONGYLUS ANDERSONI PRESTWOOD, 1972

Classification: Nematoda: Metastrongyloidea: Protostrongylidae.

Common Name: muscleworm, parelaphostrongylosis.

Parelaphostrongylus andersoni is a widely distributed muscleworm of caribou (*Rangifer tarandus* var.) in North America and may also occur in reindeer in Eurasia (Fig. 9.1). Its occurrence in white-tailed deer, the host in which it was originally found (Prestwood 1972), probably is incompletely known. Infection runs a rapid course in young animals; first-stage larval production is high for several weeks and then subsides to low levels in older animals. Clinical disease has not been reported in naturally infected caribou or deer but a resulting interstitial pneumonia may compromise normal respiratory function. This parasite is also of interest because it shares cervid hosts with more pathogenic protostrongylids, namely *P. tenuis* and *E. rangiferi*, from which it must be distinguished.

Life History. Adult *P. andersoni* are delicate, thread-like nematodes (Table 9.1, Fig. 9.2) associated with blood vessels and connective tissue deep within loin (longissimus dorsi and psoas) and thigh muscles (Prestwood 1972; Pybus 1983; Pybus and Samuel 1984a; Lankester and Hauta 1989). A few may be seen on the surface of the lateral abdominal and intercostal muscles, but those located within larger muscles are only visible upon teasing muscle samples apart under a stereomicroscope. Adult worms are relatively short (females 23–36 mm long, males 17–23 mm) and only about 100 µm wide. Males and females are often paired. They may be loosely coiled or outstretched, with much of the body length oriented parallel to adjacent muscle fibers. Female worms are commonly seen lying partially within small veins where they deposit eggs. Eggs are carried as emboli to the lungs, where they lodge in alveolar capillaries and later hatch. First-stage larvae (L_1's) emerge into the alveolar spaces, move up the bronchial escalator, and are swallowed and passed in feces.

Larvae must penetrate the foot of a terrestrial gastropod in order to molt twice and develop to the L_3 or infective stage (Table 9.2). Natural infections have been found in the snail, *Mesodon* sp. (Anderson and Prestwood 1981), and the slug, *D. laeve* (Lankester and Fong 1998), but other species likely become infected as well. Experimentally, larvae developed to the infective stage within 3–4 weeks in *Mesodon* spp. and *Triodopsis* spp. held at 20° C–26° C (Prestwood 1972; Prestwood and Nettles 1977; Pybus and Samuel 1981). Snails probably remain infected for life, but intensity decreases with time (Anderson and Prestwood 1981). Cervids are infected upon accidentally ingesting gastropods with vegetation.

The migration and development of adult *P. andersoni* within cervid hosts is incompletely known. The best information comes from Pybus (1983) and Pybus and Samuel (1984a), who studied both *P. andersoni* and the related nematode *P. odocoilei* and concluded that both species behave similarly. An impressive 54% of infective larvae given to deer were recovered during necropsies. At 46 days postinfection, when animals were first examined, most worms were found in the backstrap muscles. They had already reached the fifth stage, but none was gravid. In animals examined at later intervals, some worms appeared to move away from this location and were found in a variety of skeletal muscles (hind legs, abdominal wall, thorax, and neck), in epidural fat within the lumbar and sacral regions of the spinal canal, and in an enlarged spinal lymph node in the cauda equina. Curiously, some adults were found in abdominal fat deposits immediately ventral to the sacral vertebrae and overlying the ventral curvature of the abomasum (Pybus and Samuel 1984a). Prestwood (1972) also mentioned finding a fragment of an adult worm in washings of the abomasum. These results suggest that migrating L_3's of *P. andersoni* and *P. odocoilei* do not have to reach a particular site or tissue in which to molt, as has been demonstrated for related neurotropic forms (namely *P. tenuis* and *Elaphostrongylus* spp.). Studies in guinea pigs and rabbits likewise suggest that L_3's migrate in the body cavity and penetrate tissues directly, but some