Parelaphostrongylus tenuis (FRYADKO AND BOEV) IN THE MOOSE AND WHITE-TAILED DEER OF NOVA SCOTIA

by

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ABSTRACT

Research was initiated in 1980 to survey the frequency of occurrence and the distribution of *Paralephostrongylus tenuis* in white-tailed deer and moose in Nova Scotia. From 1980 through 1982, 305 moose and deer heads were collected from cooperating hunters, road and miscellaneous kills and examined for the presence of the adult nematode. A total of 853 feces collections from these sources and six study areas were examined for first stage larvae using the Baermann technique. The six study areas were established in areas of varying moose and deer densities and were sampled seasonally for two years. In deer 50.7% of the heads and 64.7% of the feces were infected. In moose 6.5% of the heads and 12.7% of the feces were infected. The parasite is probably not the controlling factor on the moose population. The occurrence of the parasite in moose was related to the frequency of occurrence in deer. Moose appear to be surviving with the parasite.

SYMBOLS USED IN TABLES

N - number of heads/feces examined

I - number of heads/feces infected

% - percent infected

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INTRODUCTION

In recent years there has been intensive research on *Parelaphostrongylus tenuis*, a nematode parasite of white-tailed deer. *P. tenuis* has been suggested as a possible factor in the declines of moose and other cervids in areas where their ranges overlap white-tailed deer range (Anderson, 1971a).

Based upon an analysis of the sex and age distribution of moose harvested in 1980 in Nova Scotia, Patton (1980) estimated mortality for that year of 35.6±6.6% for females 1½ years and older and 38.2±6% for males 1½ years and older. Using an estimate by Scott (1976) of 1,200 moose in Nova Scotia and the 1980 legal harvest of 14%, Patton estimated that other factors accounted for 16-26% of the mortality of the moose population in that year.

Research was initiated in Nova Scotia in 1980 to survey the frequency of occurrence and the distribution of *P. tenuis* in the moose and white-tailed deer populations. The research extended through 1982 and included the analysis of both heads and feces of moose and white-tailed deer from throughout the Province.

This study was established to investigate the possible effect of *P. tenuis* on mortality of Nova Scotia's moose and deer.

LITERATURE REVIEW

HISTORICAL REVIEW OF THE MOOSE AND DEER

General

Four sub-species of moose are found in North America. *Alces alces americana* occurs from Ontario eastward, *A. a. andersoni* occurs from Ontario to British Columbia, *A. a. shirasi* occurs in the mountains of Wyoming, Idaho, Montana and southeast British Columbia and *A. a. gigas* occurs in Alaska, western Yukon, and northwestern British Columbia (Coady, 1982).

Moose are considered to be relatively recent arrivals to North America from Eurasia (Anderson, 1965b). They came across the Bering Land Bridge to unglaciated refugia in Alaska during the Illinoian glaciation (Péwé and Hopkins, in Coady, 1982). During interglacial intervals, moose dispersed from Alaska to Canada and the continental United States (Coady, 1982).

In the United States, up to the nineteenth century, moose occurred over much of New England, New York, northern Pennsylvania and northern Michigan and Wisconsin (Parker, 1966). During the 1800's many major changes in moose populations occurred. DeVos (1964) attributed the change in moose distribution and population density to a warming phase after the last glacial period, plus to a

lesser extent, man-made changes to habitat. As the glacial ice melted, moose dispersed from Wisconsin and Pennsylvania northward. Prior to 1870 moose were rarely found on the southern or northern shores of Lake Superior or north of Lake Huron. For the next 75 years, range expansion and increases in population occurred. Although there was range expansion northward, the moose population declined along its southern boundary (deVos, 1964).

By the twentieth century moose were absent from Pennsylvania and only wandered occasionally into Vermont and New York. New Hampshire had a small population of from one to several hundred. In New Brunswick, Nova Scotia and in Maine moose occurred as fluctuating local populations. Mose were successfully introduced into Newfoundland in 1878 and 1904, and into Newfoundland-Labrador in 1953 (Dodds, 1974). In the midwest, *A. a. andersoni* formerly occurred in northern Wisconsin and the peninsula of Michigan but the population is now virtually absent from this area (Krefting, in Coady, 1982).

Originally, it was believed that there was little contact between moose and deer as deer did not range as far north as moose (Anderson, 1965b). Later, the deer followed a similar pattern of northward range extension as the moose. Shortly after the turn of the century much of the country north of Lake

Superior was occupied by deer. Probably, the most effective restriction to further northward expansion and survival is winter snow depth (deVos, 1964).

Coady (1982) in assessing the current status of moose in North America reported densities of *A. a. americana* increasing in many areas. *A. a. andersoni* and *A. a. shirasi* appear stable or slowly increasing except in Manitoba and some parts of Saskatchewan (which he suggested may be due to habitat loss from timber harvests). *A. a. gigas* has fairly high densities and is stable in western and northern parts of its range, but less dense or declining from interior and southern Alaska eastward. Overall, he found moose populations relatively high along the western edge of its range but decreased in most central and eastern regions due to habitat loss. The following is a brief summary of moose and deer in the northeast:

In eastern North America moose occasionally wander into the Adirondacks of New York from Vermont and Canada (Hicks and Peterson, 1980). An estimated 5-12 moose are reported to exist in the Adirondacks of northern New York. The state deer harvest of both sexes exceeded 150,000 in 1981. In the Adirondacks, a buck-only harvest area, the harvest of bucks was below 10,000 (M. K. Brown, pers. comm. 1982).

In New Hampshire the moose population has increased to several thousand animals (J. E. Wiley, pers. comm., 1982). Wiley suggested that this may be due to a

combination of factors such as reduced deer populations (estimated at 30,000-40,000) and a change in the habitat caused by intensive logging.

In Maine, moose have also increased to the extent that a 1980 moose season was initiated. Seven hundred permits were issued with 636 moose harvested. There was no season in 1981 and in 1982, 1,000 permits were issued (K. Morris, pers. comm., 1981). Morris estimated moose densities in the eight wildlife management units to range from 0.1 to 1.6 animals per square mile. Surveys in 1978 estimated the population statewide at over 20,000 animals (Morris and Dunn, 1981). In 1981, 37,255 deer were harvested. The densest deer populations are found in the southern portions of the state.

In Canada, Quebec's moose populations are stable and their range has expanded near the United States border in the past decade. The deer population decreased in the mid-1960's to mid-1970's due to overharvest and severe winters but have recovered rapidly since 1979 (F. Potvin, pers. comm., 1982).

Ontario's moose populations have declined and are now 35% lower than 15 years ago. This decline has not occurred in the more inaccessible areas where the population is stable (Ontario Ministry of Natural Resources, 1980). Since the mid-1950's the number of

deer has decreased by almost 70%. Several factors may be causing this decline: more severe winters, over-harvesting, poaching, wolf and dog predation in winter and deteriorating habitat conditions (Ontario Ministry of Natural Resources, no date).

In New Brunswick moose have apparently remained stable as reflected by harvests of 1,043 in 1972 and 1,434 in 1981. The deer have increased rapidly from 1972 through 1981 from a harvest of 4,955 to 21,772 in 1981 (A. H. Boer, pers. comm., 1982). Boer noted that deer and moose populations co-exist throughout the province, although levels of the two species vary widely from one area to another.

For more information on the range of moose and deer see Figure 1.

Nova Scotia

Deer. Excavations by Erskine (1957-1958) at Indian campsites indicated that white-tailed deer (*Odocoileus virginianus*) had been abundant until about AD 500. The climate then became more rigorous and deer were probably extinct by the 1600's throughout the Province (Benson and Dodds, 1980).

Deer were later recorded in Nova Scotia in the late 1800's. Some deer probably came into Cumberland and Colchester counties prior to 1894 from New Brunswick by

way of the Isthmus of Chignecto (Figure 2). Introductions took place in 1894 in Digby County and in 1910 in Annapolis and Yarmouth Counties. A possible introduction may also have taken place in 1864 in Halifax county. The deer spread from three main areas: The Isthmus of Chignecto, Cumberland County; Lake Jolly, Digby County; and Halifax County (Benson and Dodds, 1980).

By 1916 deer were common and an open hunting season was declared on the mainland. Deer crossed the Canso Strait to Cape Breton Island around 1911 and increased rapidly. A hunting season was opened in 1922 for Richmond County and in 1928 for the entire island.

Smith (1940) reported deer in all parts of the Province with the population increasing. Hunter kills rose from several hundred in 1916 to over 20,000 in the mid-1940's. The 10 year annual average kill steadily rose from 183 from 1916-1926, to 2,004 from 1927-1937, to 15,694 from 1938-1948. In 1957, however, it became evident that deer were declining. Kills of over 43,000 in 1955 dropped under 30,000 in the late 1950's and early 1960's. Key factors included range deterioration, winter snow conditions and changes in land use patterns (Benson and Dodds, 1980).

Since the late 1950's deer have once again been increasing, especially throughout the western counties.

The 10 year annual average kill since 1971 was 26,253 and winter kill has been minimal (Benson and Dodds, 1980) (Table 1).

Moose. Moose bone fragments were also found frequently in excavations by Erskine (1957-1958). Peterson (1955) reported moose common throughout the Province during the early settlement period. In Cape Breton the moose were gradually exterminated, mainly due to the unregulated harvest by early settlers (Telfer, 1965).

In the early 1800's moose became relatively scarce throughout the Province, mainly because of the extensive hunting (Benson and Dodds, 1980).

In 1843 local governments were given the authority by the Provincial government to control the taking of moose and in 1844 this power was expanded to hunting seasons. However, it was not until 1856 that the first regulated moose season was declared Province-wide from September 1 until February 1.

Conflicting reports exist in regard to the moose population from around 1825 until 1879. The moose season was closed for the period 1874-1877, but was reopened in 1878 (Benson and Dodds, 1980).

Early in the 1900's mainland moose populations fluctuated until the season was again closed in 1937.

Cameron (1949) reported that moose had been declining since 1930 and were still declining in 1948 except in Cumberland, Antigonish and Guysborough counties where they were relatively abundant.

Moose were re-introduced into Inverness County, Cape Breton in 1928 and 1929. A second release took place in 1947 with 18 animals from Alberta (subspecies *andersoni*) released into the Cape Breton Highlands National Park (Dodds, 1974). These successful introductions and subsequent increases led to an open season in Inverness and Victoria counties in 1980 (Patton, 1980, 1981).

On the mainland, between 1938 and 1948 the moose population increased and then dropped to their lowest numbers from 1948-1951. These declines tended to coincide with deer population increases (Benson and Dodds, 1980).

Ground and air surveys began in the 1950's to determine moose numbers, herd structure, range conditions and the interrelationships with deer. In 1963 Dodds estimated the population at a maximum of 4,000 animals located mainly in Cumberland, Colchester, Pictou and Antigonish counties. An aerial survey in 1963-64 estimated the population at around 3,600 (Benson and Dodds, 1980).

In 1964 an experimental ten day hunting season was opened in Cumberland, Colchester, Pictou and Antigonish counties with 183 moose harvested. In 1966 the season

was opened on a regular basis except for closures in 1975 and 1976 (Benson and Dodds, 1980).

In 1965 Telfer estimated moose populations in these counties at about 2,886. Prescott estimated 3,072 in 1968 and Scott estimated 1,200 animals in 1976, reflecting a possible decline (Prescott, 1968; Scott, 1976).

Other sources also showed a decline in moose populations. A 1977 reproduction study by Vukelich (1977) reflected a declining population. From 1966 to 1974 the average annual moose harvest was 315, falling from 1977-1981 to 201 (Table 2).

HISTORICAL REVIEW OF MOOSE DISEASE

General

The moose in eastern North America are frequently afflicted with a disorder characterized by locomotor ataxia, general and lumbar weakness, apparent deafness and blindness or impaired vision, fearlessness, listlessness, circling (sometimes associated with a peculiar position of the head) and paraplegia (Anderson, 1964).

Sick and dead moose have been reported in northern Minnesota since 1912. Fenstermacher and Jellison (1933) reported that the most distinctive feature common to all sick moose was their fearlessness of humans. From other areas came more reports of sick moose. Cahn *et al.* (1932) reported a "new disease among moose" in northeastern Minnesota and Ontario that appeared in the spring. Fenstermacher described sick moose which he autopsied from 1933-1942 (Fenstermacher and Jellison, 1933; Fenstermacher, 1934b, 1937; Fenstermacher and Olsen, 1942). Lamson (1941) observed a sick moose in captivity in Maine and Benson (1952, 1958) ran histopathological tests on moose in Nova Scotia.

Theories

There were many theories for the cause of moose sickness, although few researchers accumulated sufficient evidence to support them.

Cahn *et al.* (1932) attributed the disease to a bacterium, *Klebsiella paralytica*, transmitted by the winter tick, *Dermacentor albipictus* in the early spring. Their theory helped to explain their finding high numbers of sick moose in the spring. The isolated bacterium was injected into guinea pigs and rabbits which died after exhibiting similar symptoms to those exhibited by moose.

Fenstermacher and Jellison (1933) autopsied sick moose but found no further possible cause. Although they recognized that ticks could be a vector of some bacterial or protozoan disease, their transmission of *K. paralytica* to guinea pigs and rabbits did not produce disease symptoms. Fenstermacher (1934b) later reported that moose disease was not a seasonal occurrence, contrary to Cahn *et al.* (1932).

Nematodes were found in the eyes of two sick moose in 1937 by Fenstermacher and were assumed to be members of either the family Metastrongylidae or Protostrongylidae, however, no relationship between the nematodes and moose sickness was suspected. Lamson (1941) also found nematodes in the brains of two Maine moose and also failed to recognize their significance. He thought that the disease was either "the result of some obscure pathogen . . . or a combination of factors which greatly lower the resistance of the animals, making them more susceptible to other infections." He also considered the possibility of nutritional deficiencies causing mortality.

Fenstermacher and Olsen (1942) continued their autopsies, not ruling out a neurotropic virus.

Cameron (1949) and Benson (1952) in separate studies attributed mortality of the moose to a disease of the brain and spinal cord.

Benson (1952) referred to a sick captive moose treated with cobaltous chloride which recovered. He suggested that abnormal moose in Nova Scotia were suffering from a cobalt deficiency. However, Beeler *et al.* (1959) found higher concentrations of cobalt in the livers of sick moose from Nova Scotia than from Newfoundland where neurologic disease is unknown.

Pneumostrongylus tenuis was first described in a white-tailed deer in New York by Dougherty in 1945 on the basis of a male lungworm. This parasite was further investigated by Anderson in experimental infections of white-tailed deer fawns. He established details of its life cycle and intermediate hosts (Anderson, 1956, 1963).

In 1963, Anderson (1964) experimentally infected two moose calves with third stage *P. tenuis* larvae. Both calves exhibited the symptoms of moose sickness. After autopsies, fifth stage *P. tenuis* were found in the central nervous system in one calf and in the cranium of the second. Anderson was the first to verify that *P. tenuis* was the causative agent of moose sickness.

DISTRIBUTION OF *P. tenuis*

P. tenuis is common in white-tailed deer throughout the deciduous forest biome and the deciduous coniferous ecotone in eastern North America (Anderson and Prestwood,

1981). Anderson (1974) also found the parasite present in some southern parts of the coniferous biome and in aspen parkland. Prestwood and Smith (1969) found that even when deer densities were high, the parasite was not reported from regions with sandy soil and predominantly pine forest. There are also no reports of meningeal worms in the grasslands biome (Anderson, 1972) (Figure 2).

In Canada *P. tenuis* has been found in Nova Scotia (Smith *et al.*, 1964; Parker, 1966; Hansen, 1975), New Brunswick (Smith *et al.*, 1964; Steventon, 1977), Quebec (Bindernagel and Anderson, 1972), Ontario (Anderson, 1963, 1965c; Lankester and Anderson, 1968; Bindernagel and Anderson, 1972; Saunders, 1973), Manitoba (Bindernagel and Anderson, 1972; Lankester, 1972, 1974), and Saskatchewan (Bindernagel and Anderson, 1972).

In the United States *P. tenuis* has been reported in Maine (Behrend and Witter, 1968; Gilbert, 1973, 1974; Gleich *et al.*, 1977), New Hampshire (Thurston and Strout, 1978), Vermont (L. Garland, pers. comm., 1981), New York (Behrend, 1970; Brown and Brown, 1982), Pennsylvania (Alibasoglu *et al.*, 1961; Samuel and Beaudoin, 1966; Beaudoin *et al.*, 1970), New Jersey (Pursglove, 1977), Connecticut (Nielson and Aftosmis, 1964), Wisconsin (Samuel and Trainer, 1969), Minnesota (Kurtz *et al.*, 1966; Karns, 1967), Alabama, Arkansas, Florida, Georgia,

Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Tennessee, and West Virginia (Prestwood and Smith, 1969), Virginia (Dudak, 1964), Oklahoma (Carpenter *et al.*, 1972) and eastern Texas (Anderson and Prestwood, 1981).

NOMENCLATURE OF *Parelaphostrongylus tenuis*

There is a certain amount of confusion in the literature relative to the correct name of the meningeal worm. Dougherty in 1945 named it *Pneumostrongylus tenuis*, which was the most widely accepted name until Pryadko and Boev in 1971 transferred the parasite to the genus *Parelaphostrongylus*.

The following names have been applied to the meningeal worm (from Anderson and Prestwood, 1981):

Pneumostrongylus tenuis	Dougherty, 1945
Odocoileostrongylus tenuis	(Dougherty) Schulz, 1951 (refer to Skrjabin ., 1952)
Neuroflaria cornellensis	Whitlock, 1952
Elaphostrongylus tenuis	(Dougherty) Whitlock, 1959
Elaphostrongylus odocoilei	Anderson, 1956
Parelaphostrongylus tenuis	Pryadko and Boev, 1971

CLASSIFICATION OF *Parelaphostrongylus tenuis*

Following Pryadko and Boev, 1971:

Phylum	Aschelminthes
Subclass	Phasmidia
Suborder	Strongylina
Family	Metastrongylidae
Subfamily	Protostrongylinae
Genus	*Parelaphostrongylus*
Species	*tenuis*

OTHER SPECIES OF *Parelaphostrongylus*

There are two other species in the genus *Parelaphostrongylus: P. odocoilei* and *P. andersoni*. Both are found in white-tailed deer. Comparative measurements of the first stage larvae reveal few morphological differences between the three species. *P. andersoni* has a tail which appears to be more sharply pointed than that of the other two species (Prestwood, 1972).

In the adult stage, the morphological determination of the *Parelaphostrongylus* species is not possible in females. Males of the three species can be identified (Table 3). The *P. tenuis* male has a characteristic gubernaculum, a sclerotized thickening of the cuticle, with a prominent crura, the two parts of which have outer borders with several tooth-like projections. *P. tenuis*

has a split in the distal portion of the first spicule (Platt and Samuel, 1978).

In *P. andersoni* the crura is reduced and apparently fused to the corpus. The spicules are also shorter than those of *P. tenuis* and have a different shape, being knoblike proximally, lacking a foramen and are bifurcate at the distal tip (Platt and Samuel, 1978).

P. odocoilei is similar to *P. tenuis* but is smaller in size with shorter spicules (Platt and Samuel, 1978; Anderson and Prestwood, 1981) (Figure 3).

Besides morphological differences, *P. andersoni* and *P. odocoilei* adults are found in the loin and thigh muscles of deer, while *P. tenuis* is associated with the subdural spaces and venous sinuses of the cerebral meninges (Nettles and Prestwood, 1976).

P. odocoilei has been reported in black-tailed deer and mule deer in California and in mule deer in Alberta (Brunetti, 1969; Platt and Samuel, 1978). *P. andersoni* has been reported in white-tailed deer in Georgia, South Carolina, Louisiana, Florida and North Carolina in southern flood plain habitat, southern mixed and oak-hickory pine vegetative types. It also has been reported in New Jersey (Prestwood and Kellogg, 1974; Pybus and Samuel, 1981). *P. tenuis* and *P. andersoni* have been found concommitantly in one white-tail from North

Carolina (Prestwood and Kellogg, 1974). In Canada *P. andersoni* has been reported from British Columbia (Pybus and Samuel, 1981).

LIFE CYCLE

P. tenuis develops in the white-tailed deer's central nervous system. Disease normally does not result in infected white-tails, although a temporary lameness and weakness of the limbs has been found in deer with heavy experimental infections (Anderson, 1968; Lankester, 1976). Eckroade *et al.* (1970) provided one of the few reports of a naturally infected white-tailed deer exhibiting symptoms of neurologic disease. A later autopsy revealed ten adult *P. tenuis* in the cerebrum, an unusual area of invasion in deer.

Adult worms are commonly found in the cranial subdural space and cranial venous sinuses, and occasionally in the spinal subdural space. Eggs are deposited in the venous circulation and carried to the heart via the jugular veins and then to the lungs. Occasionally females deposit eggs which develop to the first stage on the meninges or in chronically inflamed tissue of the venous sinuses. Undeveloped eggs in the lungs eventually block minute vessels, become surrounded by a delicate fibrous capsule and develop normally. Eggs develop to the first larval

stage and hatch, move into the air spaces and pass up the bronchi. Then they are swallowed and passed in the feces (Anderson, 1968, 1971a; Anderson and Prestwood, 1981).

First stage larvae passed in the feces penetrate into the foot of gastropods where they molt twice and give rise to the third and infective stage. A film of moisture is required for penetration of the foot, but total immersion prevents larval penetration. Experimentally, the infective stage is reached in about three weeks at 20-25°C. A longer time period is probably required under field conditions (Lankester and Anderson, 1968; Anderson, 1972; Anderson and Prestwood, 1981).

Deer and other cervids become infected when they accidentally ingest infected gastropods. In the alimentary tract, larvae leave tissues of the gastropod, penetrate the gastrointestinal wall, cross the peritoneal cavity and follow the lumbar and other nerves to the vertebral canal. About ten days are required before larvae reach the central nervous system from the time of ingestion (Anderson and Strelive, 1966; Anderson, 1968). Eckroade *et al.* (1970) suggested that the arteries were used as a guidance system towards the brain surface as the worms approach the cortex.

Migrating third stage larvae invade the dorsal horns of gray matter of the spinal cord where they develop for about one month. Between 30 and 40 days after infection most worms, by now subadult, have left the neural parenchyma and entered the spinal subdural space. Here they mature and migrate anteriorly to the cranium. Some worms remain in the subdural space while others invade cranial venous sinuses by penetrating the dura mater (Anderson, 1964, 1972) (Figure 4).

The prepatent period is reported to be 82-91 days but may be longer in individuals (Anderson and Prestwood, 1981).

STAGES

Larval and adult stages have been studied in detail by Anderson (1956, 1963). Here we are concerned with the first stage larvae and the adult worm.

First Stage Larvae

The following description of first stage larvae is from Anderson (1963):

"The first stage larvae was rather stout with slightly tapering extremities. When inactive it invariably assumed the position as illustrated [Figure 5]. The cuticle was thick and smooth in the living worm although striations were present in preserved specimens.

Wide, lateral alae extended from about 15μ behind the cephalic extremity to within 7-8μ of the caudal extremity. The cuticle on the dorsal side of the posterior part of the tail had a triangular-shaped spine. The minute oral opening was surrounded by six papillae of the internal circle and four submedian elevations representing the external circle, in addition to lateral, clearly defined amphids. The slender oesophagus was dilated in front of the nerve ring at the point where subventral oesophageal glands emptied. The oesophagus gradually expanded behind the nerve ring before joining the intestine. Nuclei of oesophageal glands as well as granules associated with them were easily seen. The oesophago-intestinal valve was highly developed. Delicate cells forming the intestinal wall contained numerous refractory inclusions. Cuticle lining the short rectum was clearly defined. Phasmids were not located but their presence was indicated by cuticular linings left on the shed cuticle of the first molt. The prominent excretory pore led into a long, terminal, excretory duct lined with cuticle. The genital premordium was obscure and spindle-shaped."

Adult Stage

The following description of the adult stage is from Anderson (1956):

"Long slender nematodes with thin, delicate cuticle. Teguminal sheath absent. Oral opening circular, bordered

by a delicate, transparent, cuticular ring, leading into weakly cuticularized, cone-shaped, buccal capsule that is surrounded by esophageal tissue. Esophagus broad, muscular and club-shaped. Excretory pore prominent, leading into elongated terminal duct lined with cuticle. Terminal duct leading into short, ill-defined excretory sinus, the latter dividing posteriorly into two greatly elongated, faintly granular, excretory gland cells each of which contains a large nucleus towards its distal end. Posterior end of excretory gland cells rounded. Six perityls present of equal size each bearing a papilla (internal circle). Four submedian groups of two papillae present; each group bordered by slightly raised cuticular ridge. Ventrolateral papillae lateral to pore-like amphids, exceedingly small, and difficult to locate" (Figures 6 and 7).

INTERMEDIATE HOSTS

First stage *P. tenuis* larvae are found in the mucous coat of the feces of its host. Terrestrial gastropods probably acquire larvae while crawling over the feces (Anderson and Prestwood, 1981).

Experimentally, a variety of terrestrial gastropods can be infected; however, only a few species may be important in natural transmission. Lankester and

Anderson (1968) found seven naturally infected gastropods in Ontario: *Deroceras laeve, D. reticulatum, Arion circumscriptus, Zonitoides nitidus, Anguispera alternata, Cionella lubrica,* and *Succinea ovalis.* The most commonly found gastropods, *D. laeve* and *Z. nitidus,* were the gastropods most commonly infected.

In Maine, Gleich (1972) also found these two snail species and two slug species, *Discus cronkhitei* and *Pallifera dorsalis,* to be the most common. However, he only found *P. tenuis* in *P. dorsalis.* He noted that this may be due to the location of his study area in central Maine where terrestrial gastropods were not abundant.

In Nova Scotia, Parker (1966) found *Z. arboreus* and *Arion* sp. to be most abundant. The following species were found to be naturally infected: *D. reticulatum, D. cronkhitei, Z. arboreus, Striatura exigua,* and *Philomycus carolinianus.* No aquatic snails in any of these studies were found to be infected.

Other species in which first stage larvae have been found to develop to the third stage experimentally are: *Haplotrema concavum, Mesodon thyroidus, Stenotrema fraternum, Triodopsis albolabris* and *T. notata* (Lankester and Anderson, 1968).

Seasonal variations in the infection of gastropods were noted. In *D. laeve* the prevalence of *P. tenuis* larvae was highest in the spring and probably an important source of infection until mid-July when adults die. *Z. nitidus* infection did not fluctuate greatly (Lankester and Anderson, 1968). Parker (1966) also noted a seasonal decline in overall infection rates from early spring through fall. He suggested that this was directly correlated with the seasonal decline of moisture on the forest floor.

Snails already infected with *P. tenuis* can be reinfected, without any noticeable effect on the rate of development of either group of larvae. Development rates do vary, though, between different gastropod species (Lankester and Anderson, 1968).

Larvae can survive in gastropods under severe environmental conditions. Larvae cease to develop in estivating snails but will develop normally when conditions improve. Larvae also were found to be fairly resistant to freezing and can winter in the foot of snails and slugs (Lankester and Anderson, 1968).

Larvae in the soil may survive for a longer period of time than in the feces. Lankester and Anderson (1968) suggested that infection from the soil may explain some naturally infected gastropods.

Habitat type affects gastropod abundance and variety. Kearney (1975) found a fairly even distribution of gastropods in three habitat types: softwoods, mixed woods and hardwoods. In Nova Scotia, Parker (1966) also found gastropods in these types of habitats, but found that the proportion was greater in hardwoods (51%) as opposed to mixed woods (34%) and softwoods (15%). Steventon (1977) noted that heavy deciduous leaf litter, moisture and rotting wood all contribute towards good conditions for gastropods.

Parker (1966) also suggested that high snail densities are found wherever calcium is high, supporting his finding that a greater abundance of gastropods was in hardwoods. Steventon (1977) found less available calcium in coniferous stands than in adjacent hardwoods.

Transmission to deer may occur without high prevalences and densities of gastropods as the large food consumption of deer increase the chances of it acquiring infected snails or slugs. Experimentally, patent infections have been produced in deer by extremely small numbers of larvae (Anderson and Prestwood, 1981).

PATHOLOGY IN DEER

As mentioned previously, *P. tenuis* develops in the central nervous system of white-tailed deer, though

they rarely show obvious symptoms of neurologic disease.

Anderson (1965a) noted two sites in white-tailed deer where important pathological changes occur in *P. tenuis* infections, the lungs and central nervous system. In the central nervous system, Gilbert (1973) found that adult nematodes were commonly found lying on the dura mater and diaphragm sellae. Worms on the dura mater were usually associated with yellowish plaques of udate streaked with blood. Small hematomas were occasionally noted in the dura mater (Anderson and Prestwood, 1981).

In the lungs, lesions consisting of small discolored spots uniformly distributed throughout the parenchyma are noticeable (Anderson and Prestwood, 1981).

Third stage larvae migrating from the gut to the central nervous system leave few detectable lesions except in heavy infections (Anderson and Prestwood, 1981). While developing in the neural parenchyma of the spinal cord worms are usually confined to the dorsal horns lying longitudinally to the cord (Anderson, 1968).

For a more detailed examination of *P. tenuis* pathology see Anderson (1963, 1965a), Anderson and Strelive (1967a), and Anderson and Prestwood (1981).

OTHER HOSTS

P. tenuis has been naturally found in a variety of ungulates and is believed to be an important factor in reducing or eliminating certain populations of wild ungulates in eastern North America (Anderson and Prestwood, 1981).

Livestock

Domestic livestock in pastures frequented by deer have been reported to be infected with meningeal worm in New York, New Hampshire, Connecticut and West Virginia (Nielson and Aftosmis, 1964).

In experimental studies, sheep were found to be fairly resistant to *P. tenuis* and rarely showed neurologic signs unless heavily infected (Anderson, 1965b; Anderson and Strelive, 1966). Alden *et al.* (1975) in West Virginia did observe locomotor deficits. They observed that larvae developed as in deer until reaching the spinal cord where, after an initial inflammatory response, the larvae were eventually destroyed.

Isolated incidents of parelaphostrongylosis have been reported in goats in New York (Mayhew *et al.*, 1976) and in angora goats in Texas (Guthrey and Beasom, 1979).

Caribou

Woodland caribou (*Rangifer tarandus*) have declined over much of their southern range in North America. In some areas declines have been associated with the arrival of white-tailed deer, although habitat destruction may be the causative agent (Behrend and Witter, 1968; Anderson, 1971b; Trainer, 1973; Dauphine. 1975).

Experimental studies reported the characteristic clinical and histological signs attributed to *P. tenuis* (Anderson and Strelive, 1967b).

Wapiti

Present populations of wapiti or elk (*Cervus elaphus*) in eastern North America are the result of introductions of western races. *P. tenuis* has been suspected as a factor in the relative lack of success of these introductions (Karns, 1966; Anderson, 1972; Olsen and Woolf, 1979). Experimental infections of *P. tenuis* in wapiti were pathogenic at levels of infection that normally do not produce marked or lasting clinical signs in white-tailed deer (Anderson *et al.*, 1966).

Black-tailed Deer

In 1973 the first instance of neurologic disease was found in a female black-tailed deer (*O. hemionus columbianus*). Experimental infections produced symptoms

of neurologic disease. It is interesting to note that only immature fifth-stage worms were found when fatalities occurred, rather than adult worms (Nettles and Prestwood, 1977b).

Fallow Deer

Early reports of neurologic disease were reported in fallow deer (*Cervus dama*) in Kentucky (Kistner *et al.*, 1977; Nettles and Prestwood, 1977a). As in black-tailed deer, no adult worms were found nor were larvae or eggs found in the meninges, lungs or feces. Clinical, pathogenic and parasitological features of *P. tenuis* infection were present as in other susceptible ruminants (Nettles and Prestwood, 1977a).

Mule Deer

Mule deer (*O. hemionus hemionus*) have been experimentally infected with *P. tenuis* and have exhibited the characteristic symptoms of neurologic disease. Little is known about the relationship of white-tailed deer, meningeal worm and mule deer. There are no reports of naturally infected animals (Anderson *et al.*, 1966; Tyler and Hibler, 1980).

Others

Guinea pigs have been experimentally infected with *P. tenuis*. These did not exhibit neurologic disease and only a small number of worms were found in the

central nervous system, suggesting that guinea pigs are a poor host (Anderson and Strelive, 1965; Spratt and Anderson, 1968).

Nettles and Prestwood (1979) found cottontail rabbits (*Sylvilagus floridanus*) and laboratory rabbits (*Oryctolagus cuniculi*) to be relatively resistant to infection. Their results suggested that domestic rabbits were midly susceptible to infection while cottontails were resistant.

Other species that have been found naturally infected are: captive llamas (*Lama guanicoe*) in Texas (Brown *et al.*, 1978), a captive eland (*Taurotagus oryx*) in Georgia and a pronghorn antelope (*Antilocapra americana*) (Anderson and Prestwood, 1981).

Anderson and Prestwood (1981) cite several possible reasons explaining the severity of neurologic signs in hosts other than white-tails:

1. Worms seem to be unusually active in neural tissue and coil upon themselves, causing considerable damage to surrounding tissue,

2. Some worms do not leave the neural parenchyma before 40 days and, as normal growth continues, considerable tissue damage is caused by the large worms,

3. Worms tend to invade and damage the ependymal canal, the layer of epidermis lining the brain and the spinal cord,

4. Many ungulates seem unusually susceptible to neural invasion and ingestion of even small numbers of infective larvae can bring about a fatal neurologic condition, and

5. Worms that have left the spinal neural parenchyma and matured in the subdural space may invade the brain or spinal cord and deposit eggs.

STUDY

OBJECTIVES

1. To determine the distribution of *P. tenuis* and its incidence of occurrence in moose and deer in Nova Scotia relative to age and sex of moose and deer, method of kill, county, habitat, month, season and year.

2. To determine sex, location and number of adult *P. tenuis* in the cranial cavity of moose and deer.

3. To determine the frequency of occurrence and distribution of *P. tenuis* in relation to density of deer and moose.

4. To compare *P. tenuis* data from Nova Scotia with those from other areas.

METHODS

The prevalence of *P. tenuis* in Nova Scotia's moose and deer populations was based upon frequency of infection in a sample of heads and feces of these two species.

Most of the moose heads examined were collected from cooperating hunters during the 1980 and 1981 moose seasons. Members of the Nova Scotia Department of Lands and Forests were asked to bring in heads of accidentally killed moose and moose killed when behaving abnormally

from September 1980 through March 1982. Heads were stored in walk-in freezers at the regional offices in Nova Scotia and brought to the Kentville Wildlife Division Laboratory for autopsy.

Deer heads were collected by regional biologists through the same time period. These were mainly from road kills and accidental deaths. All heads were frozen and brought to Kentville for autopsy.

Previous to autopsy, deer heads were thawed for at least 24 hours and moose heads for at least 48 hours. Heads that were damaged from bullets, skull fractures or were decomposed were not examined. Each head was partially skinned and the cranium was opened by four cuts with a bone saw. The skull was cut horizontally across the frontal area posterior to the orbits and once across the parietal bone at the external occipital protuberance with care taken not to damage the meninges. Two cuts were made through the lateral aspect of the frontal and parietal bones.

The top of the cranium was removed and the exposed portions of the meninges and the brain surface were examined for *P. tenuis* adults under magnification. The meningeal blood vessel was slit and the lumen examined. The entire brain was removed gradually as the cranial nerves were severed and examined. The cranium and all of the brain stem present were also examined.

Location of adult nematodes was noted as: 1) falx cerebri, the dura mater between the two cerebral hemispheres; 2) tentorium cerebelli, the dura mater forming the septum between the cerebrum and cerebellum; 3) dorsal dura mater-cerebrum, the dorsal subdural space between the dura mater and cerebrum; 4) ventral dura mater-cerebrum, the ventral subdural space between the dura mater and cerebrum; 5) dura mater-cerebellum, the subdural space between the dura mater and cerebellum; 6) eye orbit; and 7) pia mater, the vascular meninges covering the surface of the brain (Figure 8).

The number and location of adult *P. tenuis* were recorded. Brainworms were preserved in 10% formalin and were later sexed at 100X under a compound microscope. They were sexed as male based upon the presence of spicules or female based upon the presence of a vulva.

Sex, county of origin and date of kill were determined by biologist reports for both deer and moose. Age was determined by the biologist by tooth eruption, replacement and wear as described by Severinghaus (1949) and Passmore *et al.* (1955).

Moose feces were obtained from hunters during the 1981 moose season. Hunters were sent plastic bags and asked to slit the rectal portion of the large intestine and collect any feces found there. Regional biologists also

collected feces from miscellaneous kills from September 1980 through October 1982.

Deer feces were obtained by regional biologists from miscellaneous kills through the same time period. All feces were frozen as soon as possible.

In addition six study areas of varying moose and deer densities were set up to sample feces. These were: low deer:no moose, low deer:low moose, low deer:high moose, high deer:no moose, high deer:low moose, and high deer: high moose (Figure 9). Regional biologists were asked to collect from each area seasonally beginning in September 1981. Due to difficulty in finding fresh feces during the summer and fall months, collections were continued through October 1982. Feces were not collected from wet areas where larvae could have been washed out. Each sample was individually bagged and tagged with the location and date and frozen as soon as possible.

Feces were examined using the Baermann technique for first stage larvae. Frozen fecal pellets were wrapped in a single layer of cheesecloth and placed in plastic funnels supported by ring stands. Rubber tubing was attached to the end of each funnel and tube clamps sealed the ends of the tubing. The feces were covered with warm water. Deer feces were left for at least eight hours and

moose feces for 24 hours. A small amount of filtrate was then drawn off through the rubber tubing into disposable petri dishes and examined at 40X under a Wild binocular microscope for first stage larvae. Feces were recorded as either positive or negative based upon the presence or absence of larvae similar to *P. tenuis*. Other parasites were either preserved in formalin or a slide was made with hot glycerol. These parasites were later identified by Mr. Ward Stone, a pathologist for the New York State Department of Environmental Conservation.

All data from heads and feces were summarized by animal, kill type or collection area, sex, age, month, season, year, number of adult worms, sex and location of adult worms. Data were analysed in the computer subprogram Crosstabs of the Statistical Package for the Social Sciences. Tests for significance ($P < .05$ or probability of occurrence by chance $< .05$) in the data were done using chi-quare to determine whether or not data variables were statistically independent. When a variable such as sex or kill type was not known for an animal, the animal was not included in the analysis for that particular variable.

STUDY AREAS

In order to examine the occurrence of *P. tenuis* as it relates to ungulate density and habitat type, six areas were chosen for study.

Percentage of forest type was determined in each region using a planimeter and Nova Scotia Provincial Forest Inventory maps.

Area 1 - Halifax County

Study area 1 had a low deer:low moose density. An estimated population of 10+ moose is found in this area (A. McInnis, pers. comm., 1982). In 1981 and 1982, 55 samples of deer feces and 80 samples of moose feces were collected in the Grover Lake area of Halifax County. An additional 9 deer and 5 moose feces collections were made in the spring of 1981 in the Mooseland area of Halifax County.

MacDougall *et al.* (1963) classified the soils in this area as derived from the Gibraltar Series. Soils are developed from a pale yellowish brown sandy loam till derived from granite. The soil is shallow and extremely stony except on the ridges which are likely eskers of glacial origin. The open porous soil allows rapid drainage and a low moisture-holding capacity. The topography is undulating.

Loucks (1962) classified the forest cover as belonging to the Eastern Shore District of the Atlantic Shore Ecoregion. This is characterized by an abundance of black spruce (*Picea mariana*) and balsam fir (*Abies balsamea*) with local area of red maple (*Acer rubrum*) and yellow birch (*Betula lutea*). On barren areas witherod

(*Viburnum cassinoides*) and alder (*Alnus rugosa*) were common (A. McInnis, pers. comm., 1982) (Table 5 and Figures 10 and 11).

Area 2 - Kings County

This area had a low deer:no moose density. A total of 187 deer feces was collected in Kings County around Black River Lake, the Huntingdon Brook area, and on the south slope of North Mountain.

Black River Lake soils are of the Morristown Series. Soils are derived from reddish-brown shaly loam till. The soil is often shallow and well drained. The topography is rolling with some steep slopes. Collections were made along power lines and the numerous logging roads found in this area.

The Huntingdon Brook soil belongs to the Annapolis Series. This is derived from reddish-brown or dark reddish brown sandy loam. Drainage is moderately good although internal drainage is imperfect due to compact subsoil. The surface is moderately to very stony (Cann *et al.*, 1965).

Two forest types make up the areas of feces collection. The North Mountain District is part of the Fundy Bay Eco-region. Red spruce (*P. rubens*) is moderately abundant. Sugar maple (*A. saccharum*), beech (*Fagus grandifolia*) and mountain ash (*Sorbus americana*) are found at higher elevations (Loucks, 1962). The soils from the collection area on the south slope of North Mountain belong to the Somerset

Series. The bedrock is usually a fine conglomerate breaking down to a reddish-brown or dark-red coarse sandy loam. Both surface and internal soil drainage is good.

The Black River Lake collection area falls within Loucks' Annapolis District of the Clyde River-Halifax Ecoregion. Sugar maple, red spruce and hemlock (*Tsuga canadensis*) are found with tolerant hardwoods on dry exposed locations. Other common species present are white pine (*Pinus strobus*), balsam fir and red maple interspersed with red oak (*Quercus rubra*) (Loucks, 1962) (Table 6 and Figures 12-14).

Area 3 - Shelburne County

This area had a low deer:high moose density. Moose and deer feces were collected near Little Port Hebert, Shelburne County. Thirty samples of deer feces and 39 samples of moose feces were collected.

The soils in the area belong to the Lydgate Series. The surface and subsoil consist of very dark brown sandy loam over dark yellowish brown sandy loam. The high organic matter content of the surface layer retains moisture over long periods and internal drainage is restricted. The topography is undulating and very stony (MacDougall *et al.*, 1961).

The forest type is characteristic of the Eastern Shore District of the Atlantic Shore Ecoregion (Loucks, 1962). This was previously described in Area 1 (Table 7 and Figure 15).

Area 4 - Colchester County

This region had a high deer:low moose density. Deer and moose feces were collectedon the south facing slope of the Cobequid Mountains in areas of major winter deer concentrations. Based on a 1979 pellet count moose were estimated at 0.4 per square mile and deer at 7.8 per square mile (R. Hall, pers. comm., 1982). Eighty samples of deer feces and 38 samples of moose feces were collected from this area.

The soil in the Belmont Mountain feces collection area is part of the Cobequid Association (Wicklund and Smith, 1948). Soils are derived from igneous rocks consisting of granite, syenite, diorite and felsite giving a stony character to the soil on a brownish gray gravelly sandy loam till. The topography is varied from rolling and hilly to roughly undulating. Drainage is good externally and internally.

The soil from the Delaney Settlement feces collection area belongs to the Folly Association. It consists of a light brown gravelly loam on a light brown gravelly sandy loam till derived from a hard gray conglomerate and brown and gray sandstone. The topography is moderately undulating and drainage is good. Stones and boulders are plentiful (Wicklund and Smith, 1948).

According to Loucks (1962), these areas lie in the Cobequid Mountain District of the Maritime Upland Ecoregion.

Vegetation includes variable amounts of red and black spruce, balsam fir, sugar maple and yellow birch. A considerable amount of forest harvesting has been done throughout the area (Table 8 and Figures 16 and 17).

Area 5 - Richmond County

This area was considered one of high deer:no moose. Twenty-six samples of deer feces were collected along a lake edge, apple orchard and field edge near Capelin Cove, Richmond County, Cape Breton Island. In this area deer winter concentrations were heavy (D. Harris, pers. comm., 1982).

The soil is derived from the Gibraltar Series which has been described in Area 1 (Cann *et al.*, 1963).

The forest type belongs to the Eastern Shore District of the Atlantic Shore Ecoregion, described in Area 1 (Table 9 and Figures 18 and 19).

Area 6 - Pictou County

This area represented a high deer:high moose density. Deer and moose feces were collected from an area bounded on the north and east by clearcuts near MacPherson Lake, Pictou County. Forty-two samples of deer feces and 15 samples of moose feces were collected.

Soils are in the Barney Soil Association. The surface and subsoil are grayish brown loam over

yellowish brown shaly loam derived from shale. Under virgin conditions the surface soil consists of several inches of semi-decomposed leaves and forest litter permeated by matted roots. The topography is undulating with marshy sites, brooks and gullies (Cann and Wicklund, 1950).

The forest cover is representative of the East River-Antigonish District of the Magaquadavic-Hillsborough Ecoregion (Loucks, 1962). Beech and sugar maple predominate along with a wide distribution of white pine, spruce and balsam fir (Table 10 and Figure 20).

RESULTS AND DISCUSSION

P. tenuis has been reported in moose only in areas where its range overlaps with that of white-tailed deer (Anderson and Prestwood, 1981). A report of *P. tenuis* in moose on a deer-free island was later discounted due to possible contamination of funnels in the Baermann Technique used to analyse the deer feces (Karns and Jordan, 1969; M. W. Lankester, pers. comm., 1982).

The meningeal worm is found in white-tailed deer throughout the Province. In this study 213 deer heads were examined with 50.7% being infected with adult *P. tenuis* nematodes. Deer feces were analysed and 346 of 535 samples (64.7%) were positive with first stage larvae similar to *P. tenuis*. Totals of 50.7% infected heads and 64.7% infected feces from this study are lower than a previous study by Hansen (1975) who found an infection rate of 62.5% in deer heads and 67.5% in feces. Gilbert (1973) found an annual prevalence from 63-80% in deer heads from 1968-1970 in Maine and Behrend and Witter (1968) found 84% of Maine deer heads infected. In northern New York I found the infection rates ranging from 65.8-75% in deer feces (Brown and Brown, 1982). Preliminary figures in Vermont showed an infection rate of 32.9% in deer heads (L. E. Garland, pers. comm., 1981).

Ninety-two moose heads had a 6.5% infection rate in this study. In moose feces 12.7% (41 of 318 samples) were positive for first stage larvae similar to *P. tenuis*. In this study the overall infection rate found in moose was higher than that found earlier by Hansen (1975), who found an infection rate of 4.2% in heads and 5.8% in feces. Gilbert (1974) found 28.2% of moose heads infected in a Maine study. Smith *et al.* (1964) found four of nine moose examined to be infected in heads from New Brunswick and Nova Scotia.

There are significant differences in rate of infection in deer heads from the various methods of kill (Table 11). Since there were no mandatory deer check stations during the study, it was not possible to obtain heads from hunter shot deer. Although illegally killed deer and dog kills had a greater prevalence of infection than other kill types (87.5% in illegal killed deer and 66.6% in dog kills), it is unlikely that infected deer are more susceptible to any specific type of kill. For example, road killed deer exhibited lower infection rates than other types of kill. Illegally killed deer were obtained from September through December and deer from other types of kills were obtained year round. The period October through December was one of the peaks of *P. tenuis* infection. All collection types were found

to be more heavily infected during these months which probably explains why illegally killed deer have a higher infection rate.

Moose also showed a significant difference in the rate of *P. tenuis* infection by collection type in the feces and in the heads (Table 12). Moose exhibiting signs of sickness (referred to in the tables as biologist collections) exhibited a large number of infected animals. No larvae were recorded in feces from biologist collections, indicating that adults had not yet begun reproduction. Symptoms are usually observed when *P. tenuis* adult nematodes are present and have done damage to the neural tissue in the spinal cord and cranium.

Rau and Caron (1979) suggested that moose more heavily parasitised with *Echinococcus granulosus* were more susceptible to being shot by hunters and were the first to be killed. Gilbert (1974) had suggested that hunting (legal and illegal) tended to remove diseased animals from the population. He noted that the opening of a moose season in 1960 resulted in a lowering of the percentage of infected moose found in subsequent years in New Brunswick. In Nova Scotia the moose season reopened in 1964. Heads from Nova Scotia hunter killed moose examined by the Canada Department of Agriculture from 1964-1968 revealed higher infection rates than in

Hansen's 1975 study and this study. From 1964-1968 infection varied from 3-25% (10.0% in 1964, 3% in 1966, 25% in 1967 and 15.4% in 1968). Hansen found 0.9% of hunter killed moose heads infected and I did not find any infected hunter killed moose heads, reflecting a decline since 1964 in hunter killed moose infection rates. However, if Gilbert's hypothesis is true, the overall infection rate should also decrease which it did not in Hansen's or my studies.

Neither age class or sex in deer showed a significant difference in infection rates (Table 13 and 14; Figure 21). Conflicting reports on infection rates by age and sex are found in the literature. Thurston and Strout (1978) and Karns (1967) found males to be infected more than females. Hansen (1975), Behrend and Witter (1968) and Dudak (1964) found females to be infected more often than males. There appears to be no consistency regarding infection rates by sex. There are also conflicting reports regarding infection and age. Dudak (1964) found 1½ year old females to have a greater infection rate than males of the same age. Animals less than 1½ years were infected at similar rates. Behrend and Witter (1968) found no difference in infection rates between age classes. Hansen (1975) found 2 and 3 year old deer had the highest infection rates while

Karns (1967) found the lowest rates in deer of this age. Behrend and Witter (1968) suggested that their results might have differed due to different environmental conditions between years in the several areas studied.

Infection rates in male and female moose did not differ in the heads or feces (Tables 15 and 16). There was also no significant difference in infection rates by age (Figure 22). Gilbert (1974) found *P. tenuis* infection greatest in moose from 1 to 4 years and Hansen (1975) reported infected moose ranged from 1 year to 3½ years.

The number of worms per infected deer head in this study ranged from 1-9 with a mean of 1.9 in females and 2.3 in males (Table 13). No significant differences were found between the number of worms found and either sex or any age class. Hansen (1975) found a range from 1-8 (mean 2.6), Anderson (1963) found a range from 1-20 (mean 3.9) and Dudak (1964) reported a range from 1-13 with an average of 3.4 per infected head.

The number of worms per infected moose head was similar in the different age classes (Table 15). Only one moose had more than one adult nematode and this female had three adult worms. Hansen (1975) found similar results with a mean of 1.8 and range of 1-4 per

moose. Gilbert (1974) found an average of 1.9 worms per infected head with a range of 1-7. Smith *et al.* (1964) reported an average of 2.5 worms per infected head. The higher average found by Smith *et al.* (1964) may be due to the methods employed and more complete histological sectioning.

The location of adult nematodes in deer heads was most commonly the ventral subdural space between the cerebrum and dura mater (Table 17). Nematodes were usually found lying on the dura mater and in only four occurrences were they found penetrating the pia mater. Dudak (1964) and Behrend and Witter (1968) also found the ventral area of the cranium to be the most common location for *P. tenuis* adults. Thurston and Strout (1978) found that fawns were commonly infected in the falx cerebri and adults were most commonly infected on the tentorium cerebelli. Gilbert (1973) found adult worms in fawns most commonly in the ventral subdural space and in adults in the dorsal subdural space. No difference in location of infection between age classes or sexes was found in this study.

Adult nematodes were commonly found in the pia mater or partially penetrating the ventral dura mater in moose heads. Of eight adult *P. tenuis* in the craniums, three

were partially or wholly within the pia mater, three were in the ventral subdural space, one was in the dorsal subdural space and one was from an unknown location. Hansen (1975), Gilbert (1974) and Anderson (1964) all reported similar results. Gilbert (1974) found that penetration of the nervous parenchyma of the moose brain was normal, rather than the exception. He found a higher number of worms in the spinal cord than within the cranial cavity. One moose had ten nematodes in the lumbar region while in the heads one moose had seven worms but 24.4% had only one *P. tenuis* present.

Anderson (1964) reported that many worms may not reach the brain and remain in the spinal cord. In this study, only heads were examined, therefore, the incidence of *P. tenuis* must be considered a minimum figure.

In deer heads I found female adult *P. tenuis* to be almost twice as common as adult *P. tenuis* males (59 males, 108 females and 53 unknown sex). In moose heads, females were six times as common as males (one male, six females and one unknown. The difference in sex ratio between moose and deer was not significant. Little work has been done by other researchers to explain this result.

The seasonal prevalence of *P. tenuis* fluctuated in deer and was significantly higher in the spring and fall (Table 18 and Figure 23). Peak infection rates in deer heads overlapped with peaks shown from feces data, but often appeared somewhat later. During the winter when

snow depths increase deer may concentrate in certain yarding areas. As the snow settles and melts deer move out into open areas consuming gastropods found on or in newly exposed vegetation. Gastropods are most abundant in the spring and early summer. Parker (1966) suggested that this was a time of maximum infection.

As the summer progresses conditions become drier causing feces to dry out more readily, killing the larvae (Parker, 1966). In addition to this factor, fewer species of gastropods are present at this time of year. Kearney (1975) also found fewer gastropods to be infected in the summer. Transmission to deer, however, can occur even without high numbers of gastropods. Lankester and Anderson (1968) also found relatively few gastropods infected , as did Parker (1966), but the large quantity of food easten by deer increases the probability that deer will eventually acquire infected gastropods (Anderson and Prestwood, 1981).

Lankester and Anderson (1968) found that in hot dry weather, snails may withdraw into their shells and remain inactive, retarding the development of *P. tenuis* larvae until conditions are more favourable.

First stage larvae develop in gastropods about a month before developing into the third and infective stage. Following ingestion, a few larvae are passed in the feces after three months. The number steadily

increases until about two months after the first larvae were passed or about five months after the initial ingestion (Anderson, 1965b).

A peak in deer infection rates was found in March and April. Allowing four to six months for larval development in the intermediate host, for ingestion and finally, passing in the feces, another peak would be expected within the period July through October. A peak is found from September through November. The lower infection rates in July and August may be due to drying out of the feces or a delayed larval development in the gastropod during hot weather. Another peak of infection would be expected four to six months after the peak from September through November, or within the period February through June (Table 19 and Figure 24).

In heads and feces collected at the same time in this study, over 50% of the heads with adult *P. tenuis* also had first stage larvae present in the feces.

Little is known about how long adults can survive in the cranium and how many times they reproduce or how long they live.

In the moose feces the heaviest seasonal infections occur in the winter and spring. Anderson (1964) reported that the life cycle of *P. tenuis* is similar in moose and in deer. Thus, infected gastropods would have been

ingested three to five months earlier, in the summer and fall.

Monthly infection rates show peaks in December and January. Allowing four to six months for larval development in the gastropod, ingestion of the infected gastropod and larvae being passed in the feces, other monthly peaks would be expected during the period April-July. As mentioned previously, hot or cold weather may interrupt larval development and delay the cycle (Table 19 and Figure 25).

Gilbert (1973) found a positive correlation between prevalence of infection in deer and precipitation in Maine. He suggested that a relationship could exist between gastropod population size and the amount of ground moisture. Parker (1966) and Steventon (1977) also suggested that precipitation is an important factor affecting gastropod abundance and distribution.

Yearly infection rates increased in both moose and deer (Table 20 and Figure 26) during the study and 1980 to 1982 meteorological summaries from Environment Canada were examined for the January through August periods (Table 21). Total precipitation increased from 631.5 mm in 1980 to 857.2 mm in 1981 to 929.3 mm in 1982. Precipitation levels from 1980-1982 were positively correlated with yearly infection (Figure 27). The increase

in infection may be a result of a change in moisture improving gastropod habitat and hence gastropod distribution. No gastropod collections were made in this study to demonstrate this occurrence.

Telfer (1965) in Nova Scotia found that in some areas moose were ecologically separated from deer in the winter. Few deer are found above 400-600 feet while moose were found at high elevations year round. Kelsall and Prescott (1971) in New Brunswick found that when snow depths were greater than 20 cm deer moved to lower altitudes. Deer become more restricted in movement at about 40 cm of snow and moose at 70 cm. Moose can remain at higher elevations. Little contact occurs until late spring and early summer. Irwin (1975) in Minnesota found that the greatest overlap of moose and deer occurred in June.

P. tenuis prevalence is not always related to abundance of gastropods. Gleich and Gilbert (1976) in central Maine and Kearney (1975) in central Ontario found gastropods most common in mixed woods, followed by coniferous and finally deciduous woods. However, Kearney found *P. tenuis* infection in gastropods greatest in deciduous woods.

Steventon (1977) in New Brunswick and Parker (1966) in Nova Scotia found gastropods most abundant in hardwoods followed by mixed woods and softwoods. Parker (1966)

suggested that the leaf litter in hardwoods may provide food and moisture for snails and slugs. Calcium in the soil also seems to be an indicator of snail abundance since calcium is essential for shell formation. Steventon (1977) found calcium levels greatest in hardwoods and lowest in softwoods. Parker (1966) found variations, but generally found calcium levels were greater in hardwoods. He found, however, that infection of gastropods by *P. tenuis* was greatest in mixed woods, followed by softwoods and hardwoods. The total number of infected gastropods out of a sample of over 3,000 gastropods from these forest types was ten, two, and one, respectively. In my study, the proportion of infected feces was also most common in mixed woods followed by hardwoods and softwoods (Table 22).

Kearney (1975) found balsam fir and white birch habitat types to have the greatest number of deer days of use. Mixed and coniferous forests supplied the greatest shelter which is a major factor in deer use. When climatic conditions were favourable he found deer using deciduous areas. Kearney also found that moose used deciduous forest types more than other types. Alder areas exhibited the greatest degree of deer and moose overlap in both summer and fall and he suggested that this was an important transmission site.

Irwin (1975) found burn areas in Minnesota were used by both moose and deer. Deer preferred the periphery and unburned forest in winter and spring and burn areas in summer and fall. Moose selected the periphery areas during winter and the open areas from May through September. The species were found to be highly associated in June and separated in the winter.

Peek *et al.* (1976) found that moose used coniferous areas to a great extent in the fall and upland spruce-fir habitat in winter in Minnesota. In early summer aquatic plants were an important food source, but were used less later in the summer as their palatability decreased. Upland aspen and white birch areas and lowland deciduous areas increased in usage.

The literature then suggests that in any given area local conditions such as elevation, snow depths, burn areas or cutovers will influence the degree of overlap between moose and deer and, in turn, affect transmission of *P. tenuis*.

P. tenuis infection rates in deer feces in the study areas were significantly different in each of the forest types (Tables 22-28). In deer, infected feces were mainly found in winter in mixed and softwood areas, since, as mentioned previously, softwood areas provide shelter. In spring all habitat types held infected feces suggesting that deer were leaving yard

areas. There are little data for summer and fall for mixed wood and hardwood areas, however, an apparent decrease in deer feces in softwood areas was probably due to greater use of other habitat types. In the fall the prevalence increases again in the softwoods.

No significant difference in infection rates in moose feces among the study areas was found (Tables 29-33). Complete seasonal samples from mixed and hardwood forest types were not available for moose and, therefore, not compared.

Behrend and Witter (1968) and Karns (1967) found a positive relationship between the incidence of *P. tenuis* in white-tailed deer and deer density. Gilbert (1973), Saunders (1973) and Lankester (1974) did not find prevalence related to deer density and suggested that other factors were important, such as abundance and distribution of suitable hosts, precipitation and habitat types.

The rate of infection in my six study areas was not related to deer density in either deer or moose (Table 33 and Figure 29). From the 1981 deer harvest figures a rough population density per county was estimated using an approximation of 20% of the population harvested annually. Although infection rates by county varied significantly, comparing population estimates per county to rates of infection per

county revealed no relationship between infection rate and density (Tables 34 and 35).

Karns (1967), Gilbert (1974) and Telfer (1965) reported that moose densities were highest in areas of low deer densities. Sauders (1973) found that the highest prevalence of *P. tenuis* in moose correlated with deer infection. In this study the infection rate in moose was found to be related to the occurrence of *P. tenuis* in deer rather than deer density. This correlation was present in both the study areas and the counties.

In both moose and deer, *P. tenuis* occurrence is higher in the feces than in the heads. This is similar to results found by Anderson (1964) and Hansen (1975). There are several possibilities which may influence the higher number of feces infected among which two are considered here. Another parasite of the genus *Parelaphostrongylus* was suggested by Hansen (1975) as possibly occurring and being mistaken for *P. tenuis*. Anderson (1963) also considered this but rejected the idea due to the failure to find any adults of the other two species in deer he examined. *P. andersoni* has been reported in Canada in British Columbia (Pybus and Samuel, 1981). Thus it would seem that larvae in the feces found in Nova Scotia are most likely *P. tenuis*.

Anderson (1963) reported *P. tenuis* adults and eggs between the meninges of the spinal cord in deer and in

moose, confirming that adult *P. tenuis* can occur elsewhere than in the cranium.

Although there is no evidence that the parasite can establish itself in moose populations without the presence of deer, Anderson (1964) noted that the comparable rate of development of *P. tenuis* in moose as compared with deer and the large numbers of worms recovered suggested that moose might serve as suitable definitive hosts if they survive the neurologic damage that may result from infection. There is certainly evidence that some moose are surviving with *P. tenuis* (this study; Anderson and Prestwood, 1981). There is, however, no information as to whether moose can survive indefinitely with *P. tenuis*.

As has been noted, moose are increasing throughout portions of the northeastern United States despite areas of relatively dense deer population. This fact and the relatively high number of moose feces infected in this study suggest that moose may be surviving with the parasite. Further, in Nova Scotia, *P. tenuis* does not appear to be a major mortality factor in moose. Other factors such as illegal kill and habitat changes need to be carefully assessed to understand their roles in the apparent changes in moose numbers.

It would, in any case, be extremely difficult and uneconomical to attempt to control *P. tenuis* in moose through gastropod control. This study and others have shown that the prevalence of *P. tenuis* is related to a number of factors including survival of first stage larvae, availability, abundance and survival of gastropod intermediate hosts, habitat preferences and behaviour and density of the definitive host.

Further studies on the behaviour and physiology of *P. tenuis* within its hosts, especially in the area of its reproductive rate, life span and host tolerance are needed to answer remaining questions concerning *P. tenuis* and its role in moose and other cervid populations.

CONCLUSIONS

1. There appears to be an equal likelihood of infection by *P. tenuis* in deer of either sex and any age. There is also no difference in severity of infection.

2. The most common location of adult *P. tenuis* in deer is in the ventral subdural space between the dura mater and the cerebrum.

3. Fewer *P. tenuis* are found in moose than in deer. The number of worms per infected head is similar in all age classes and in both sexes.

4. Penetration of the meninges by adult *P. tenuis* is common in moose.

5. The rate of infection in deer was greatest during the spring and fall. Transmission to moose probably occurred to the greatest extent in the summer and fall.

6. *P. tenuis* infection rates in moose and deer increased from 1980 to 1982. This is positively correlated with an increase in precipitation which may affect gastropod abundance and distribution.

7. The proportion of infected deer feces was greatest in mixed woods. Habitat used by deer is one

factor affecting infection rates in moose in any one area.

8. Many moose appear to be surviving with *P. tenuis*.

9. *P. tenuis* infection is found throughout the Province. In moose the infection rate is related to the percent of infected deer rather than deer density.

10. *P. tenuis* is not a major mortality factor in Nova Scotian moose.

LITERATURE CITED

LITERATURE CITED

Alden, C., Woodson, F., Mohan, R. and Miller, S. 1975. Cerebrospinal nematodiasis in sheep. J. Am. Vet. Med. Ass. 166(8): 784-786.

Alibasoglu, M., Kradel, D. C. and Dunne, M. W. 1961. Cerebral nematodiasis in Pennsylvania deer (*Odocoileus virginianus*). Cornell Vet. 51(3): 431-441.

Anderson, R. C. 1956. *Elaphostrongylus odocoilei* Hobmaier and Hobmaier, 1934 in the cranial case of *Odocoileus virginianus borealis* Miller. Can. J. Zool. 34(3): 167-173.

_____. 1963. The incidence, development, and experimental transmission of *Pneumostrongylus tenuis* Dougherty (Metastrongyloidea: Protostrongylidae) of the meninges of the white-tailed deer (*Odocoileus virginianus borealis*) in Ontario. Can. J. Zool. 41: 775-792.

_____. 1964. Neurologic disease in moose infected experimentally with *Pneumostrongylus tenuis* from white-tailed deer. Pathl. Vet. 1: 289-322.

_____. 1965a. The development of *Pneumostrongylus tenuis* in the central nervous system of white-tailed deer. Path. Vet. 2: 360-379.

_____. 1965b. Cerebrospinal nematodiasis (*Pneumostrongylus*) in North American cervids. Thirtieth North American Wildlife National Resource Conference. Washington, D. C. Mimeo. 15pp.

_____. 1965c. An examination of wild moose exhibiting neurologic signs, in Ontario. Can. J. Zool. 43: 635-639.

_____. 1968. The pathogenesis and transmission of neurotropic and accidental nematode parasites of the central nervous system of mammals and birds. Helminthological Abstracts 37(3): 191-210.

_____. 1971a. Lungworms. Chapter 3 in Parasitic Diseases of Wild Mammals. J. W. Davis and R. C. Anderson (eds.). Iowa State University Press. pp. 81-126.

Anderson, R. C. 1971b. Neurologic disease in reindeer (*Rangifer tarandus tarandus*) introduced in Ontario. Can. J. Zool. 49(2): 159-166.

_____. 1972. The ecological relationships of meningeal worm and native cervids in North America. J. Wildl. Dis. 8: 304-310.

_____. 1974. Cerebrospinal nematodiasis (*Pneumostrongylus tenuis*) in North American ruminants. First International Congress of Parasitology. Mimeo. 8pp.

Anderson, R. C. and Lankester, M. W. 1974. Parasites and diseases of moose. Nat. Can. 101: 23-50.

Anderson, R. C., Lankester, M. W. and Strelive, U. R. 1966. Further experimental studies of *Pneumostrongylus tenuis* in cervids. Can. J. Zool. 44(5): 851-861.

Anderson, R. C. and Prestwood, A. K. 1981. Lungworms. Chapter 21 in Diseases and Parasites of White-tailed Deer. Davidson, W. R., Hayes, F. A., Nettles, V. F. and Kellogg, F. E. (eds.). Compiled and expanded from the Second International White-tailed Deer Disease Symposium. pp. 274-301.

Anderson, R. C. and Strelive, U. R. 1965. The transmission of *Pneumostrongylus tenuis* to guinea pigs. Can. J. Zool. 44: 533-540.

_____. 1966. Experimental cerebrospinal nematodiasis (*Pneumostrongylus tenuis*) in sheep. Can. J. Zool. 44(5): 889-894.

_____. 1967a. The penetration of *Pneumostrongylus tenuis* into the tissues of white-tailed deer. Can. J. Zool. 45: 285-289.

_____. 1967b. The experimental transmission of *Pneumostrongylus tenuis* to caribou *(Rangifer tarandus terraenovae)*. Can. J. Zool. 46(3): 503-510.

Beaudoin, R. L., Samuel, W. M. and Strome, C. P. A. 1970. A comparative study of the parasites in two populations of white-tailed deer. J. Wildl. Dis. 6: 56-63.

Behrend, D. F. 1970. The nematode, *Pneumostrongylus tenuis* in white-tailed deer in the Adirondacks. New York Fish and Game Journal 17(1): 45-49.

Behrend, D. F. and Witter, J. F. 1968. *Pneumostrongylus tenuis* in white-tailed deer in Maine. J. Wildl. Mange. 32(4): 963-966.

Beeler, D. A., Benson, D. A. and Langille, W. M. 1959. Mineral analysis of livers and kidneys of moose (*Alces americana*). J. Wildl. Manage. 23(3): 356-358.

Benson, D. A. 1952. Treatment of a sick moose with cobaltous chloride. J. Wildl. Manage. 16(1): 110-111.

_____. 1958. Moose "sickness" in Nova Scotia - II. Can. J. Comp. Med. 22(8): 282-286.

Benson, D. A. and Dodds, D. G. 1980. The Deer of Nova Scotia. Nova Scotia Department of Lands and Forests. Halifax. 92pp.

Bindernagel, J. A. and Anderson, R. C. 1972. Newer aspects of the problem of meningeal worm (*Pneumostrongylus tenuis*) in North America: Distribution in white-tailed deer and moose. Trans. Eighth North American Moose Conference, Thunder Bay, Ontario. pp. 1-17.

Brown, M. K. and Brown, J. E. 1982. Preliminary analysis of *Parelaphostrongylus tenuis* (brainworm) in white-tailed deer in northern New York. Unpub. Manuscript. 7pp.

Brown, T. T., Jordan, H. E. and Demorest, C. N. 1978. Cerebrospinal parelaphostrongylosis in llamas. J. Wildl. Dis. 14: 441-444.

Brunetti, O. A. 1969. Redescription of *Parelaphostrongylus* (Boev and Schuls, 1950) in California deer, with studies on its life history and pathology. Cal. Fish and Game J. 55(4): 307-316.

Cahn, A. R., Wallace, G. I. and Thomas, W. 1932. A new disease of moose III. Science 76 (1974): 385-386.

Cameron, A. W. 1949. Report on biological investigations of game and fur-bearing animals in Nova Scotia, 1948. Nova Scotia Dept. of Lands and Forests. 28pp.

Cann, D. B., MacDougall, J. I. and Hilchey, J. D. 1963. Soil Survey of Cape Breton Island, Nova Scotia. Can. Dept. of Agr., Truro. Report No. 12.

Cann, D. B., MacDougall, J. I. and Hilchey, J. D. 1965. Soil Survey of Kings County, Nova Scotia. Can. Dept. of Agr., Truro. Report No. 15.

Cann, D. B., and Wicklund, R. E. 1950. Soil Survey of Pictou County, Nova Scotia. Can. Dept. of Agr., Truro. Report No. 4.

Carpenter, J. W., Jordan, H. E. and Morrison, J. A. 1972. Meningeal worm (*Parelaphostrongylus tenuis*) infection in white-tailed deer in Oklahoma. J. Wildl. Dis. 8: 381-383.

Coady, J. W. 1982. Moose. Chapter 46 in Wild Mammals of North America. Biology, Management and Economics. Chapman, T. A. and Tedhamar, G. A. (eds.). Johns Hopkins University Press. pp. 902-922.

Dauphine, T. C. 1975. The disappearance of Caribou reintroduced to Cape Breton Highlands National Park. Can. Field Nat. 89(3): 299-310.

deVos, A. 1964. Range changes of mammals in the Great Lake region. Am. Mid. Nat. 71(1): 210-231.

Dodds, D. G. 1974. Distribution, habitat and status of moose in the Atlantic provinces of Canada and north-eastern United States. Nat. Can. 19: 51-65.

Dougherty, E. C. 1945. The nematode lungworms (suborder strongylina) of North American deer of the genus *Odocoileus*. Parasit. 36: 199-208.

Dudak, D. 1964. The incidence and degree of infection of *Pneumostrongylus tenuis* in the white-tailed deer of western Virginia. M.Sc. thesis, Virginia Polytechnic Institute, Blacksburg. 75pp.

Eckroade, R. J. ZuRhein, G. M. and Foreyt, W. 1970. Meningeal worm invasion of the brain of a naturally infected white-tailed deer. J. Wildl. Dis. 6: 430-436.

Environment Canada. 1980-1982. Annual Meterological Summary. Atmospheric Environment Service. Halifax-Dartmouth.

Erskine, J. A. 1957-58. Shell-heap archaeology of south-western Nova Scotia. Proc. Nova Scotia Inst. of Sci. 24(4): 339-375.

Fenstermacher, R. 1934b. Further studies of diseases affecting moose. Univ. Minn. Agr. Exp. Sta. Bull. 308. 26pp.

_____. 1937. Further studies of diseases affecting moose. II. Cornell Vet. 27: 25-37.

Fenstermacher, R. and Jellison, W. L. 1933. Diseases affecting moose. Univ. Minn. Agr. Exp. Sta. Bull. 294. 20pp.

Fenstermacher, R. and Olsen, O. W. 1942. Further studies of disease affecting moose. III. Cornell Vet. 32: 241-254.

Gilbert, F. F. 1973. *Parelaphostrongylus tenuis* (Dougherty) in Maine: I. The parasite in white-tailed deer (*Odocoileus virginianus*, Zimmerman). J. Wildl. Dis. 9: 136-143.

_____. 1974. *Parelaphostrongylus tenuis* in Maine. II. Prevalence in moose. J. Wildl. Manage. 38(1): 42-46.

Gilbert, S. G. 1976. Pictorial Anatomy of the Cat. University of Toronto Press, Toronto. 81pp.

Gleich, J. G. 1972. Terrestrial gastropods from central Maine: Distribution, relative abundance, and relationship to parasitic nematodes, especially *Pneumostrongylus tenuis*. M.Sc. thesis, University of Maine, Orono. 176pp.

Gleich, J. G. and Gilbert, F. F. 1976. A survey of terrestrial gastropods from central Maine. Can. J. Zool. 54: 620-627.

Gleich, J. G., Gilbert, F. F. and Kutscha, N. F. 1977. Nematodes in terrestrial gastropods from central Maine. J. Wildl. Dis. 13: 43-46.

Guthrey, F. S. and Beasom, S. L. 1979. Cerebrospinal nematodiasis caused by *Parelaphostrongylus tenuis* in angora goats in Texas. J. Wildl. Dis. 15: 37-42.

Hansen, G. L. 1975. The incidence of the nematode *Parelaphostrongylus tenuis* Dougherty in Nova Scotian moose and deer. Honours thesis, Acadia University, Wolfville. 51pp.

Hicks, A. and Peterson, A. 1980. The history of the moose (*Alces alces*) in New York. Mimeo. 12pp.

Irwin, L. L. 1975. Deer-moose relationships on a burn in northwestern Minnesota. J. Wildl. Manage. 39(4): 653-662.

Karns, P. D. 1966. *Pneumostrongylus tenuis* from elk (*Cervus canadensis*) in Minnesota. Bull. Wildl. Dis. Ass. 2: 79.

_____. 1967. *Pneumostrongylus tenuis* in deer in Minnesota and implications for moose. J. Wildl. Manage. 31(2): 299-303.

Karns, P. D. and Jordan, P. A. 1969. *Pneumostrongylus tenuis* in moose on a deer free island. J. Wildl. Manage. 33(2): 431-433.

Kearney, S. R. 1975. Habitat use by white-tailed deer and moose in an area enzootic for *Parelaphostrongylus tenuis*. M.Sc. thesis, University of Guelph, Ontario. 138pp.

Kelsall, J. F. and Prescott, W. 1971. Moose and deer behavior in snow in Fundy National Park, New Brunswick. Can. Wildl. Ser., Report Series No. 15. 27pp.

Kistner, T. P., Johnson, G. R. and Rilling, G. A. 1977. Naturally occurring neurologic disease in a fallow deer infected with meningeal worms. J. Wildl. Dis. 13: 55-58.

Krefting, L. 1974. The ecology of the Isle Royale moose. Tech. Bull. No. 297, For. Ser. No. 15. Agric. Exp. Stn., Univ. Minnesota. 75pp.

Kurtz, H. J., Loken, K. and Schlolthauer, J. C. 1966. Histopathologic studies on cerebrospinal nematodiasis of moose in Minnesota naturally infected with *Pneumostrongylus tenuis*. Am. J. Vet. Res. 27(117): 548-557.

Lamson, A. L. 1941. Maine moose disease studies. M.Sc. thesis, University of Maine, Orono. 61pp.

Lankester, M. W. 1972. Diseases of moose in southeastern Manitoba. Trans. Eighth North American Moose Conference. Thunder Bay, Ontario. pp. 42-59.

_____. 1974. *Parelaphostrongylus tenuis* (Nematoda) and *Fascioloides magna* (Trematoda) in moose of southeastern Manitoba. Can. J. Zool. 52: 235-239.

Lankester, M. W. 1976. A protostrongylid nematode of woodland caribou and implications in moose management. Trans. Twelfth North American Moose Conference. pp. 173-190.

Lankester, M. W. and Anderson, R. C. 1968. Gastropods as intermediate hosts of *Pneumostrongylus tenuis* Dougherty of white-tailed deer. Can. J. Zool. 46(3): 373-383.

Loucks, O. L. 1962. A forest classification for the Maritime provinces. Proc. Nova Scotia Inst. Soc. 25(2): 86-166.

MacDougall, J. I., Cann, D. B. and Hichley, J. D. 1961. Soil Survey of Shelburne County, Nova Scotia. Can. Dept. Agr., Truro. Report No. 10.

_____. 1963. Soil Survey of Halifax County, Nova Scotia. Can. Dept. Agr., Truro. Report No. 13.

Mayhew, I. G., de Lahunta, A., Georgi, J. R. and Aspros, D. G. 1976. Naturally occurring cerebrospinal parelaphostrongylosis. Cornell Vet. 66(1): 56-72.

Morris, K. M. and F. D. Dunn. 1981. Moose management in Maine in the 1980's. Mimeo. 7pp.

Nettles, V. F. and Prestwood, A. K. 1976. Experimental *Parelaphostrongylus andersoni* infections in white-tailed deer. Vet. Pathol. 13: 381-393.

_____. 1977a. Cerebrospinal parelaphostrongylosis in fallow deer. J. Wildl. Dis. 13: 440-444.

_____. 1977b. Meningeal worm-induced neurologic disease in black-tailed deer. J. Wildl. Dis. 13: 137-143.

_____. 1979. Experimental infection of rabbits with meningeal worm. J. Parasit. 65(2): 327-328.

Nielson, S. W. and S. Aftosmis. 1964. Spinal nematodiasis in two sheep. J. Am. Vet. Med. Ass. 144: 155-158.

Nova Scotia Department of Lands and Forests. 1981. White-tailed deer harvests by county. Mimeo. 1pp.

Olsen, A. and Woolf, A. 1979. A summary of the prevalence of *Parelaphostrongylus tenuis* in a captive wapiti population. J. Wildl. Dis. 15: 33-35.

Ontario Ministry of Natural Resources. No date. Deer management in Ontario--the future. Pamphlet No. 5831.

_____. 1980. Moose management in Ontario--a report of open house public meetings. Mimeo. 14pp.

Parker, G. R. 1966. Moose disease in Nova Scotia: Gastropod-nematode relationship. M.Sc. thesis, Acadia University, Wolfville. 126pp.

Passmore, R. C., Peterson, R. L. and Cringan, A. T. 1955. A study of mandibular tooth wear as an index to age of moose. In North American Moose, Peterson, R. L. University of Toronto Press, Toronto. pp. 223-238.

Patton, A. E. 1980. Report on the 1980 moose season. Nova Scotia Dept. of Lands and Forests, Truro. Mimeo. 13pp.

_____. 1981. Report on the 1981 moose season. Nova Scotia Dept. of Lands and Forests, Truro. Mimeo. 7pp.

Peek, J. W., Urich, D. L. and Mackie, R. J. 1976. Moose habitat selection and relationships to forest management in northeastern Minnesota. Wildl. Monogr. No. 48. 65pp.

Peterson, R. L. 1955. North American Moose. University of Toronto Press, Toronto. 280pp.

Péwé, T. L. and Hopkins, D. M. 1967. Mammal remains of pre-Wisconsin age in Alaska. In The Bering Land Bridge, Hopkins, D. M. (ed.). Stanford University Press, Stanford. pp. 226-270.

Platt, T. R. and Samuel, W. M. 1978. A redescription and neotype designation for *Parelaphostrongylus odocoilei* (Nematoda: Metastrongyloidea). J. Parasit. 64(2): 226-232.

Prescott, W. H. 1968. A study of winter concentration areas and food habits of moose in Nova Scotia. M.Sc. thesis, Acadia University, Wolfville. 194pp.

Prestwood, A. K. 1972. *Parelaphostrongylus andersoni* sp. n. (Metastrongyloidea: Protostrongylidae) from the musculature of white-tailed deer (*Odocoileus virginianus*). J. Parasit. 58(5): 897-902.

Prestwood, A. K. and Kellogg, F. E. 1974. Distribution of muscleworm, *Parelaphostrongylus andersoni* among white-tailed deer of the southeastern United States. J. Wildl. Dis. 10: 404-409.

Prestwood, A. K. and Smith, J. F. 1969. Distribution of meningeal worm (*Pneumostrongylus tenuis*) in deer in the southeastern United States. J. Parasit. 55(4): 720-725.

Pryadko, E. J. and Boev, S. N. 1971. (Systematics, phylogeny, and elaphostrongyloid nematodes of deer). Izdatelstvo Akad. Nauk. Kaza Khskoi SSR, Alma-Ata. 5: 41-48 (in Russian).

Pursglove, S. R. 1977. Helminth parasites of white-tailed deer (*Odocoileus virginianus*) from New Jersey and Oklahoma. Proc. Helmint. Soc. Washington, D. C. 44(1): 107-108.

Pybus, M. J. and Samuel, W. M. 1981. Nematode muscleworm from white-tailed deer of southeastern British Columbia. J. Wildl. Manage. 45(2): 1981.

Rau, M. E. and Caron, F. R. 1979. Parasite induced susceptibility of moose to hunting. Can. J. Zool. 57(12): 2466-2468.

Samuel, W. M. and Beaudoin, R. L. 1966. Evaluation of two survey methods for detection of helminth infections in white-tailed deer (*Odocoileus virginianus*). Bull. Wildl. Dis. Ass. 2: 100-107.

Samuel, W. M. and Trainer, D. O. 1969. A technique for survey of some helminth and protozoan infections of white-tailed deer. J. Wildl. Manage. 33(4): 584-588.

Saunders, B. P. 1973. Meningeal worm in white-tailed deer in northwestern Ontario and moose population densities. J. Wildl. Manage. 37(3): 327-330.

Severinghaus, C. W. 1949. Tooth development and wear as a criteria of age in white-tailed deer. J. Wildl. Manage. 13: 195-216.

Scott, C. J. 1976. Nova Scotia moose: A new inventory technique. M.Sc. thesis, Acadia University, Wolfville. 111pp.

Skrjabin, K. I., Shilhobalova, N. P., Schulz, R. S., Popova, T. J., Boev, S. N. and Delyamure, S. L. 1952. (Descriptive Catalogue of parasitic nematodes: Vol. 3. Strongylata). Moscow. Izdatelstvo Akademii Nauk. SSSR. (in Russian).

Smith, R. W. 1940. The land mammals of Nova Scotia. Am. Mid. Nat. 24: 213-241.

Smith, H. J., Archibald, R. G. and Corner, A. H. 1964. Elaphostrongylosis in maritime moose and deer. Can. Vet. J. 5(11): 287-296.

Spratt, D. M. and Anderson, R. C. 1968. The guinea pig as an experimental host of the meningeal worm (*Pneumostrongylus tenuis* Dougherty). J. Helm. 42: 139-156.

Statistics Canada. 1981. Census of Canada. Cat. 93-903. Vol. 2. Provincial Series.

Steventon, J. D. 1977. Influence of habitat on the incidence of *Parelaphostrongylus tenuis* in white-tailed deer and possible consequences for moose. B.Sc. thesis, University of New Brunswick, Fredericton. 24pp.

Telfer, E. S. 1965. Studies of moose and white-tail deer ecology in northern Nova Scotia. M.Sc. thesis, Acadia University, Wolfville. 88pp.

Thurston, D. R. and Strout, R. G. 1978. Prevalence of meningeal worm (*Parelaphostrongylus tenuis*) in white-tailed deer from New Hampshire. J. Wildl. Dis. 14: 89-96.

Trainer, D. O. 1973. Caribou mortality due to the meningeal worm (*Parelaphostrongylus tenuis*). J. Wildl. Dis. 9: 376-378.

Tyler, G. V. and S. P. Hibler. 1980. Experimental infection of mule deer with *Parelaphostrongylus tenuis*. J. Wildl. Dis. 16(4): 533-540.

Vukelich, M. F. 1977. Reproduction and productivity of moose in Nova Scotia. M.Sc. thesis, Acadia University, Wolfville. 109pp.

Whitlock, J. H. 1952. Neurofilariosis, a paralytic disease of sheep. II. *Neurofilaria cornellensis* n.g.n.sp. (Nematoda, Filarioides), a new nematode parasite from the spinal cord of sheep. Cornell Vet. 42: 125-132.

_____. 1959. *Elaphostrongylus*, the proper designation of *Neurofilaria*. Cornell Vet. 49: 3-14.

Wicklund, R. E. and Smith, G. R. 1948. Soil Survey of Colchester County, Nova Scotia. Can. Dept. Agr., Truro. Report No. 3.

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TABLES

Table 1. Nova Scotia deer harvest 1916-1981 (from Benson and Dodds (1980) and Nova Scotia Department of Lands and Forests 1980 and 1981 kill statistics)

Year	Season length (days)	Number of licenses	Total kill	Kills per licenses	Kill per square mile
1916[a]	10	--	154	--	.007
1917	10	--	101	--	.004
1918	10	--	69	--	.003
1919	15	--	198	--	.009
1920	15	--	125	--	.006
1921	15	9,301	255	.03	.014
1922[b]	15	6,279	232	.04	.013
1923	15	5,781	147	.03	.008
1924	15	5,513	174	.03	.009
1925	15	5,714	280	.05	.015
1926	15	7,569	281	.04	.015
1927	40	10,830	635	.06	.035
1928[c]	30	8,818	987	.11	.046
1929	30	11,160	1,316	.12	.061
1930	30	14,102	1,886	.13	.088
1931	30	16,823	4,685	.28	.218
1932	30	14,764	4,866	.33	.227
1933	20	9,560	1,216	.13	.047
1934	20	10,694	1,327	.12	.062
1935	19	8,270	996	.12	.046
1936	10	10,639	1,950	.18	.091
1937	10	12,709	2,185	.17	.102
1938	30	17,191	6,727	.39	.314
1939	30	17,621	6,224	.35	.291
1940	45	17,941	8,717	.49	.407
1941	45	21,525	10,784	.50	.503
1942	45	25,492	10,233	.40	.478
1943	45	20,786	9,203	.44	.430
1944	45	20,582	12,939	.63	.604
1945	45	29,327	20,124	.69	.939
1946	45	33,948	26,750	.79	1.25
1947	45	38,681	30,007	.78	1.40
1948	60	43,882	30,934	.70	1.44
1949	45	43,012	30,318	.70	1.42
1950	45	49,656	37,176	.75	1.74
1951	45	55,785	42,343	.76	1.98
1952	45	58,576	38,481	.66	1.80
1953	35	62,201	43,330	.70	2.02
1954	45	63,025	46,389	.74	2.19
1955	45	58,215	43,400	.75	2.03
1956	45	56,232	34,332	.61	1.62
1957	45	48,491	21,065	.44	.98
1958	45	54,905	30,951	.56	1.44
1959	45	57,647	31,701	.56	1.47
1960	45	62,602	32,329	.52	1.51
1961	45	49,480	17,682	.36	.83
1962[d]	45	47,410	22,036	.46	1.07
1963	45	47,756	22,071	.46	1.03
1964[c]	45	52,341	23,259	.44	1.08
1965	45	57,569	24,945	.43	1.16
1966	45	57,616	24,103	.42	1.12
1967	45	52,842	20,133	.38	.94
1968	45	57,809	22,079	.38	1.03
1969	45	57,620	22,574	.39	1.05
1970	30	51,573	17,725	.34	.83
1971	30	56,333	15,491	.28	.72
1972	30	58,406	19,385	.33	.91
1973	30	67,136	19,567	.29	.91
1974	30	75,975	24,015	.33	1.12
1975	30	66,151	21,860	.34	1.02
1976	30	65,350	28,794	.44	1.34
1977	30	83,479	25,676	.31	1.19
1978	30	82,443	28,201	.34	1.31
1979	45	81,606	32,215	.41	1.50
1980	30	81,801	34,470	.42	1.61
1981	30	83,959	39,558	.47	1.85

[a] Mainland only

[b] Mainland and Richmond County

[c] Province

[d] Province except Yarmouth County

Table 2. Nova Scotia moose harvest 1964-1981 from Benson and Dodds (1980) and Patton (1980, 1981).

Year	Season length (days)	Number of licenses	Total kill	Kills per license	Kill per square mile
1964[a]	10	400	183	.46	.04
1965	--	--	--	--	--
1966[b]	10	800	361	.41	.06
1967	10	1,000	316	.32	.05
1968	10	1,000	282	.28	.04
1969	10	1,000	318	.32	.05
1970	10	1,000	310	.31	.05
1971	10	1,000	340	.34	.05
1972	10	1,000	409	.41	.06
1973	10	1,000	321	.32	.05
1974	10	1,000	319	.32	.05
1975	--	--	--	--	--
1976	--	--	--	--	--
1977	10	650	229	.35	.04
1978	10	605	226	.37	.04
1979	10	600	182	.30	.03
1980[c]	10	660	209	.32	.03
1981	10	440	161	.37	.02

[a]1964 - Cumberland, Colchester, Pictou, Antigonish Counties open

[b]1966-1979 - as in 1964 plus Guysborough County

[c]1980-1981 - as in 1966 plus Inverness and Victoria Counties

Table 3. Key to males of the genus *Parelaphostrongylus* (Platt and Samuel, 1978)

1(2)	Gubernaculum reduced, less than 52 in total length; corpus undivided, crura chitinized, lacking large, lateral projections. Spicules divided distally to form bifid tip. Parasites of the dorsal musculature of *O. virginianus*	*P. andersoni*
2(1)	Gubernaculum large, greater than 70 in total length; corpus divided or undivided. Crura lateral to corpus, possessing 4 to 6 lateral projections. Spicules not divided at distal tip.....	2
3(4)	Corpus of gubernaculum undivided. Spicules with a foramen in the mid-region of the shaft. Parasites in central nervous system of cervids.....	*P. tenuis*
4(3)	Corpus of gubernaculum divided along the proximal 1/3. Spicules lacking a foramen. Parasites of the dorsal musculature of the mule deer, *O. hemionus* subspp.	*P. odocoilei*

Table 4. Reference for study area maps.

FOREST REFERENCE

HEIGHT CLASS

UP TO 16 FEET - A
16 TO 30 FEET - B
31 TO 50 FEET - C
OVER 50 FEET - D
ALL HEIGHTS - E

DENSITY CLASS

UP TO 40% - 1
41 TO 60% - 2
61 TO 100% - 3

SITE CLASS

III - GOOD
V - POOR
ALL OTHERS - AVERAGE

SPECIES COMPOSITION CLASS

UP TO 25 % HARDWOOD - S SOFTWOOD
26 TO 75 % HARDWOOD - M MIXEDWOOD
76 TO 100 % HARDWOOD - H HARDWOOD

RECENT CLEAR CUT - CC
RECENT BURN - ++
FLOWAGE:
SEASONAL - F
OTHER - ω ω ω

NON-FORESTED LAND

BRUSHLAND & ALDERS - (a) ROCK BARREN - VV BOG OR OPEN MUSKEG -
MUSKEG & STUNTED TREES - AGRICULTURAL - A URBAN - U
TIDAL FLATS & MARSH - TF

MAP REFERENCE

ROADS:
HARD SURFACE
LOOSE SURFACE, ALL WEATHER
DRY WEATHER
CROWN LAND ACESS
WOODS
TRAILS
RAILWAYS:
SINGLE TRACK, OVERPASS
DOUBLE, UNDERPASS
ABANDONED
POWER LINES:
STEEL TOWERS
WOODEN POLES
TELEPHONE LINES
HORIZONTAL CONTROL:
TRIANGULATION, TRAVERSE
BENCH MARK
LIGHTHOUSE, TOWER

BOUNDARIES:
PROVINCIAL
COUNTY
MUNICIPAL
GAME SANCTUARY
CROWN, SURVEYED
NOT SURVEYED
APPROXIMATE DIVISION OF LARGE LANDOWNERS
MILITARY RESERVATIONS AND PARKS
FOREST TYPE
RIVERS:
RAPIDS, FALLS, BRIDGES.
STREAM: PERENNIAL
POSITION DOUBTFUL
DIVIDED
WHARF, BREAKWATER
BEACH, CLIFF
AERIAL PHOTO. CENTER

CROWN LANDS - 1 LARGE OWNERSHIP (1000 ACRES PLUS) - 2
SMALL OWNERSHIP (1000 ACRES OR LESS) - 3
MILITARY RESERVATIONS AND FEDERAL PARKS - 5

ONE INCH EQUALS .25 MILES OR .4 KILOMETERS

1000 0 1000 2000 3000 4000 5000 FEET
300 0 300 600 900 1200 1500 METERS

Table 5. Study Area 1 forest types.

Area	Forest type	Square kilometers	Percent of cover
Grover Lake	Softwood	1.15	56.65*
	Hardwood	0.41	20.19
	Mixed wood	0.26	12.81
	Non-forested	0.21	10.34
	Overall	2.03	99.99
Mooseland	Softwood	0.10	41.67
	Hardwood	--	--
	Mixed wood	0.14	58.33*
	Overall	0.24	100.00

*Predominant forest type

Table 6. Study Area 2 forest types.

Area	Forest types	Square kilometers	Percent of cover
Black River Lake	Softwood	--	--
	Hardwood	0.01	11.11
	Mixed wood	0.08	88.89*
	Overall	0.09	100.00
	Softwood	--	--
	Hardwood	0.05	100.00*
	Mixed wood	--	--
	Overall	0.05	100.00
	Softwood	0.02	28.57
	Hardwood	0.04	57.14*
	Mixed wood	0.01	14.29
	Overall	0.07	100.00
Huntingdon Brook	Softwood	0.10	100.00*
	Hardwood	--	--
	Mixed wood	--	--
	Overall	0.10	100.00
North Mountain	Softwood	--	--
	Hardwood	--	--
	Mixed wood	0.05	71.43*
	Non-forested	0.02	28.57
	Overall	0.07	100.00

*Predominant forest type

Table 7. Study Area 3 forest type.

Area	Forest types	Square kilometers	Percent of cover
Little Port Hebert	Softwood	1.33	40.30*
	Hardwood	0.68	20.61
	Mixed wood	1.12	33.94
	Non-forested	0.17	5.15
	Overall	3.30	100.00

*Predominant forest type

Table 8. Study Area 4 forest types.

Area	Forest types	Square kilometers	Percent of cover
Belmont Mountain	Softwood	0.37	64.91*
	Hardwood	--	--
	Mixed wood	0.12	21.05
	Non-forested	0.08	14.03
	Overall	0.57	99.99
Delaney Settlement	Softwood	0.01	4.00
	Hardwood	0.16	64.00*
	Mixed wood	0.06	24.00
	Non-forested	0.02	8.00
	Overall	0.25	100.00

*Predominant forest type

Table 9. Study Area 5 forest types.

Area		Forest types	Square kilometers	Percent of cover
Capelin Cove	(a)	Softwood	0.01	100.00*
		Hardwood	--	--
		Mixed wood	--	--
		Overall	0.01	100.00
	(b)	Softwood	0.02	40.00
		Hardwood	--	--
		Mixed wood	0.03	60.00*
		Overall	0.05	100.00

*Predominant forest type

Table 10. Study Area 6 forest types.

Area	Forest types	Square kilometers	Percent of cover
MacPherson's Lake	Softwood	1.25	41.25*
	Hardwood	0.24	7.92
	Mixed wood	1.24	40.92
	Non-forested	0.30	9.90
	Overall	3.03	99.99

*Predominant forest type

Table 11. *Parelaphostrongylus tenuis* in deer by collection type.

Collection type	Heads			Feces		
	N	I	%	N	I	%
Road kill	162	77	47.5	52	21	40.4
Illegal kill	16	14	87.5	--	--	--
Dog kill	9	6	66.6	8	3	37.5
Hunter kill	--	--	--	1	1	100.0
Garden kill	--	--	--	1	0	0.0
Found on ground	--	--	--	34	27	79.4
Miscellaneous	9	5	55.5	2	1	50.0
Total	196	102	52.0	98	53	54.1

χ^2 for heads = 9.85 3 DF $P < .025$

χ^2 for feces = 15.64 5 DF $P = .0080$

Table 12. *P. tenuis* in moose by collection type.

Collection type	Heads			Feces		
	N	I	%	N	I	%
Road kill	5	1	20.0	3	0	0.0
Biologist coll.	18	4	22.2	10	0	0.0
Hunter kill	49	0	0.0	110	5	4.5
Illegal kill	6	1	16.7	2	0	0.0
Found on ground	--	--	--	15	4	26.7
Miscellaneous	3	0	0.0	1	0	0.0
Total	81	6	7.4	141	9	6.4

χ^2 for heads = 11.88 3 DF $P < 0.025$

χ^2 for feces = 12.04 5 DF $P = 0.0342$

Table 13. *P. tenuis* infection and number of worms in deer heads by age and sex.

Age	Sex	N	I	%	Worms	Range	Mean
0-½	Male	5	3	60.0	14	1-5	4.7
	Female	3	1	33.3	4	1-4	4.0
	Total	8	4	50.0	18	1-5	4.5
½-1	Male	21	13	61.9	31	1-9	2.4
	Female	16	6	37.5	13	1-3	2.2
	Total	37	19	51.4	44	1-9	2.3
1-1½	Male	17	7	41.2	10	1-2	1.4
	Female	16	7	43.8	11	1-3	1.6
	Total	33	14	42.4	21	1-3	1.5
1½-2	Male	22	11	50.0	18	1-6	1.6
	Female	13	8	61.5	13	1-4	1.6
	Total	35	19	54.3	31	1-6	1.6
2-2½	Male	5	0	0.0	0	0	0.0
	Female	5	1	10.0	3	1-3	3.0
	Total	10	1	10.0	3	1-3	3.0
2½-3	Male	11	5	45.5	12	1-5	2.4
	Female	11	10	90.9	24	1-7	2.4
	Total	21	16	76.2	36	1-7	2.3
3-3½	Male	--	--	--	--	--	--
	Female	1	0	0.0	0	0	0.0
	Total	1	0	0.0	0	0	0.0
3½+	Male	22	11	50.0	30	1-7	2.7
	Female	25	15	60.0	26	1-4	1.7
	Total	47	26	55.3	56	1-7	2.2
Total	Male	103	50	48.5	115	1-9	2.3
	Female	90	48	53.3	94	1-7	1.9
	Total	193	98	50.8	209	1-9	2.1

χ^2 for sex = 0.19 1 DF P = 0.6642
χ^2 for age = 11.84 7 DF P = 0.1061
χ^2 for sex and age = 20.41 14 DF P = 0.1178

Table 14. *P. tenuis* infection in deer feces by age and sex.

Age	Sex	N	I	%
0-½	Male	--	--	--
	Female	--	--	--
	Total	--	--	--
½-1	Male	9	6	66.7
	Female	9	5	55.6
	Total	18	11	61.1
1-1½	Male	8	3	37.5
	Female	5	2	40.0
	Total	13	5	38.5
1½-2	Male	3	0	0.0
	Female	--	--	--
	Total	3	0	0.0
2-2½	Male	2	0	0.0
	Female	1	0	0.0
	Total	3	0	0.0
2½-3	Male	2	1	50.0
	Female	4	1	25.0
	Total	6	2	33.3
3-3½	Male	--	--	--
	Female	--	--	--
	Total	--	--	--
3½+	Male	11	4	36.4
	Female	6	2	33.3
	Total	17	6	35.3
Total	Male	35	14	40.0
	Female	25	10	40.0
	Total	60	24	40.0

χ^2 for sex = 0 1 DF P = 1.0000
χ^2 for age = 12.30 6 DF P = 0.0555
χ^2 for sex and age = 8.22 10 DF P = 0.6069

Table 15. *P. tenuis* infection and number of worms in moose heads by age and sex.

Age	Sex	N	I	%	Worms	Range	Mean
0-½	Male	-	-	-	-	-	-
	Female	-	-	-	-	-	-
	Total	-	-	-	-	-	-
½-1	Male	4	0	0.0	0	0	0.0
	Female	8	1	12.5	3	3	3.0
	Total	12	1	8.3	3	3	3.0
1-1½	Male	1	1	100.0	1	1	1.0
	Female	1	0	0.0	0	0	0.0
	Total	2	1	50.0	1	1	1.0
1½-2	Male	9	1	11.1	1	1	1.0
	Female	13	1	7.7	1	1	1.0
	Total	22	2	9.1	2	1	1.0
2-2½	Male	-	-	-	-	-	-
	Female	-	-	-	-	-	-
	Total	-	-	-	-	-	-
2½-3	Male	5	1	20.0	1	1	1.0
	Female	8	0	0.0	0	0	0.0
	Total	13	1	7.7	1	1	1.0
3-3½	Male	-	-	-	-	-	-
	Female	-	-	-	-	-	-
	Total	-	-	-	-	-	-
3½+	Male	6	1	16.7	1	1	1.0
	Female	26	0	0.0	0	0	0.0
	Total	32	1	3.1	1	1	1.0
Total	Male	25	4	16.0	4	1	1.0
	Female	56	2	3.6	4	1-3	2.0
	Total	81	6	7.4	8	1-3	1.3

χ^2 for sex = 2.29 1 DF P = 0.1301
χ^2 for age = 2.61 4 DF P = 0.6254
χ^2 for sex and age = 7.08 9 DF P = 0.6293

Table 16. *P. tenuis* infection in moose feces by age and sex.

Age	Sex	N	I	%
0-½	Male	-	-	-
	Female	-	-	-
	Total	-	-	-
½-1	Male	3	0	0.0
	Female	6	0	0.0
	Total.	9	0	0.0
1-1½	Male	1	0	0.0
	Female	1	0	0.0
	Total	2	0	0.0
1½-2	Male	11	1	9.1
	Female	11	0	0.0
	Total	22	1	4.5
2-2½	Male	1	0	0.0
	Female	1	0	0.0
	Total	2	0	0.0
2½-3	Male	12	0	0.0
	Female	22	2	9.1
	Total	34	2	5.9
3-3½	Male	1	0	0.0
	Female	2	0	0.0
	Total	3	0	0.0
3½+	Male	27	1	3.7
	Female	4	0	0.0
	Total	31	1	3.2
Total	Male	56	2	3.6
	Female	47	2	4.3
	Total	103	4	3.9

χ^2 for sex = 0.12 1 DF P = 0.7419
χ^2 for age = 1.07 6 DF P = 0.9827
χ^2 for sex and age = 4.14 14 DF P = 0.9896

Table 17. *P. tenuis* location in deer heads by age and sex.

Age	Sex	Eye orbit	Falx cerebri	Tentorium	Dorsal dura mater	Ventral dura mater	Pia mater	Cerebellum
0-1	Male	6	2	1	17	11	3	-
	Female	1	-	-	22	4	-	1
	Total	7	2	1	29	15	3	1
1-1½	Male	1	-	1	5	3	-	-
	Female	-	-	-	7	3	-	1
	Total	1	-	1	12	6	-	1
1½-3½	Male	3	6	-	4	15	-	2
	Female	2	3	3	7	24	1	-
	Total	5	9	3	11	39	1	2
3½+	Male	1	-	1	15	9	-	2
	Female	6	-	2	7	8	-	-
	Total	7	-	3	22	17	-	2
Total	Male	11	8	3	41	38	3	4
	Female	9	3	5	33	39	1	2
	Total	20	11	8	74	77	4	6

Table 18. *P. tenuis* infection in deer and moose by season.

Season[a]	Deer						Moose					
	Heads			Feces			Heads			Feces		
	N	I	%	N	I	%	N	I	%	N	I	%
Winter	60	28	46.7	307	213	69.4	7	2	28.6	110	21	19.1
Spring	14	8	57.1	72	52	72.2	1	0	0.0	34	7	20.6
Summer	52	18	34.6	95	40	42.1	5	1	20.0	44	5	11.4
Fall	71	46	64.8	51	33	64.7	66	3	4.5	129	8	6.2
Total	197	100	50.8	525	338	64.4	79	6	7.6	317	41	12.9

χ^2 for deer heads = 11.64 3DF P = 0.0081
χ^2 for deer feces = 25.84 3DF P = 0.0000
χ^2 for moose heads = 6.44 3DF P = 0.0920
χ^2 for moose feces = 10.76 3DF P = 0.0131

[a]Seasons: Winter = December, January, February, March
Spring = April, May
Summer = June, July, August
Fall = September, October, November

Table 19. *P. tenuis* infection in deer and moose by month.

Month	Deer						Moose					
	Heads			Feces			Heads			Feces		
	N	I	%	N	I	%	N	I	%	N	I	%
January	18	7	38.9	28	13	46.4	4	1	25.0	10	3	30.0
February	12	4	33.3	31	23	74.2	1	0	0.0	21	2	9.5
March	9	5	55.6	219	168	76.7	1	1	100.0	66	11	16.7
April	9	7	77.8	69	52	75.4	-	-	-	32	7	21.9
May	5	1	20.0	3	0	0.0	1	0	0.0	2	0	0.0
June	25	8	32.0	27	15	55.6	2	0	0.0	10	1	10.0
July	18	6	33.3	60	22	36.7	-	-	-	24	4	16.7
August	9	4	44.4	8	3	37.5	3	1	33.3	10	0	0.0
September	21	10	47.6	49	31	63.3	2	1	50.0	18	3	16.7
October	25	16	64.0	1	1	100.0	56	1	1.8	110	5	4.5
November	25	20	80.0	1	1	100.0	8	1	12.5	1	0	0.0
December	21	12	57.1	29	9	31.0	1	0	0.0	13	5	38.5
Total	197	100	50.8	525	338	64.4	79	6	7.6	317	14	12.9

χ^2 for deer heads = 23.66 11DF P = 0.0143
χ^2 for deer feces = 67.54 11DF P = 0.0000
χ^2 for moose heads = 25.23 9DF P = 0.0027
χ^2 for moose feces = 22.82 11DF P = 0.0188

Table 20. *P. tenuis* infection in deer and moose by year.

Year	Deer						Moose					
	Heads			Feces			Heads			Feces		
	N	I	%	N	I	%	N	I	%	N	I	%
1980	17	8	47.1	7	1	14.3	46	1	2.2	1	0	0.0
1981	160	81	50.6	202	111	55.0	32	3	9.4	219	24	11.0
1982	20	11	55.0	317	227	71.6	5	2	40.0	98	17	17.3
Total	197	100	50.8	526	339	64.4	84	6	7.1	318	41	12.9

χ^2 for deer heads = 0.24 2DF P = 0.8877
χ^2 for deer feces = 22.73 2DF P = 0.0000
χ^2 for moose heads = 10.17 2DF P = 0.0172
χ^2 for moose feces = 2.61 2DF P = 0.2714

Table 21. Precipitation totals for January through August 1980-1982 (from Environment Canada, 1980-1982).

	Total precipitation (mm)		
	1980	1981	1982
January	73.4	124.9	152.4
February	28.2	53.8	79.8
March	124.1	88.3	84.8
April	139.2	55.5	180.6
May	71.1	178.5	53.8
June	110.1	153.4	137.4
July	55.0	128.6	133.3
August	30.4	74.2	107.2
Total	631.5	857.2	929.3

Table 22. *P. tenuis* infection in deer and moose feces seasonally by habitat (from Study Areas).

Forest type	Season	Deer			Moose		
		N	I	%	N	I	%
Softwood	Winter	87	60	69.0	88	16	18.2
	Spring	25	18	72.0	23	6	26.1
	Summer	71	32	45.1	34	5	14.7
	Fall	14	10	71.4	17	3	17.6
Mixed wood	Winter	112	88	78.6	4	0	0.0
	Spring	35	26	74.3	1	0	0.0
	Summer	--	--	--	--	--	--
	Fall	--	--	--	--	--	--
Hardwood	Winter	39	20	51.3	8	2	25.0
	Spring	12	12	100.0	2	0	0.0
	Summer	--	--	--	--	--	--
	Fall	34	20	58.8	--	--	--
Softwood Total		197	120	60.9	162	30	18.5
Mixed wood Total		147	114	77.6	5	0	0.0
Hardwood Total		85	52	61.2	10	2	20.0
		429	286	66.7	177	32	18.1

χ^2 for deer = 11.19 2DF $P < 0.005$
χ^2 for moose = 1.15 2DF $P > 0.25$

Table 23. *P. tenuis* infection in deer feces from Study Area 1.

Month	Year	Grover Lake			Mooseland		
		N	I	%	N	I	%
March	1981	4	1	25.0	9	3	33.3
April	1981	6	1	16.7	-	-	-
July	1981	6	0	0.0	-	-	-
December	1981	10	7	70.0	-	-	-
March	1982	12	9	75.0	-	-	-
June	1982	8	4	50.0	-	-	-
September	1982	9	9	100.0	-	-	-
Total		55	31	56.4	9	3	33.3
Combined Total		64	34	53.1			

Table 24. *P. tenuis* infection in deer feces from Study Area 2.

Month	Year	North Mountain			Huntingdon Brook			Black River Lake								
		N	I	%	N	I	%	N	I	%	N	I	%	N	I	%
January	1981	-	-	-	-	-	-	6	0	0.0	-	-	-	-	-	-
February	1981	-	-	-	-	-	-	-	-	-	13	12	92.3	-	-	-
April	1981	-	-	-	1	1	100.0	-	-	-	-	-	-	-	-	-
September	1981	-	-	-	-	-	-	-	-	-	-	-	-	6	3	50.0
February	1982	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
March	1982	77	71	92.2	-	-	-	-	-	-	-	-	-	-	-	-
April	1982	28	20	71.4	-	-	-	-	-	-	-	-	-	-	-	-
June	1982	-	-	-	4	4	100.0	-	-	-	-	-	-	-	-	-
July	1982	-	-	-	21	12	57.1	-	-	-	-	-	-	-	-	-
August	1982	-	-	-	3	0	0.0	-	-	-	-	-	-	-	-	-
September	1982	-	-	-	-	-	-	-	-	-	-	-	-	28	17	60.7
Total		105	91	86.7	29	17	58.6	6	0	0.0	13	12	92.3	34	20	58.8
Combined Total		187	140	74.9												

Table 25. *P. tenuis* infection in deer feces from Study Area 3.

Month	Year	Little Port Herbert		
		N	I	%
February	1981	11	11	100.0
April	1981	8	7	87.5
March	1982	11	7	63.6
Total		30	25	83.3

Table 26. *P.tenuis* infection in deer feces from Study Area 4.

Month	Year	Belmont Mountain			Delaney Settlement		
		N	I	%	N	I	%
March	1981	13	10	76.9	22	18	81.8
April	1981	-	-	-	12	12	100.0
December	1981	-	-	-	11	2	18.2
January	1982	7	3	42.9	-	-	-
March	1982	11	10	90.9	-	-	-
July	1982	1	1	100.0	-	-	-
August	1982	1	1	100.0	-	-	-
September	1982	1	0	0.0	-	-	-
October	1982	1	1	100.0	-	-	-
Total		35	26	74.3	45	32	71.1
Combined Total		80	58	72.5			

Table 27. *P. tenuis* infection in deer feces from Study Area 5.

Month	Year	Capelin Cove					
		N	I	%	N	I	%
March	1981	-	-	-	10	2	20.0
December	1981	1	0	0.0	-	-	-
March	1982	-	-	-	10	6	60.0
April	1982	5	5	80.0	-	-	-
Total		6	4	66.7	20	8	40.0
Combined Total		26	12	46.2			

Table 28. *P. tenuis* infection in deer feces from Study Area 6.

Month	Year	MacPherson's Lake		
		N	I	%
February	1981	7	2	28.6
April	1981	5	5	100.0
July	1982	29	9	31.0
August	1982	1	1	100.0
Total		42	17	40.5

Table 29. *P. tenuis* infection in moose feces from Study Area 1.

Month	Year	Grover Lake			Mooseland		
		N	I	%	N	I	%
March	1981	9	1	11.1	4	0	0.0
April	1981	10	2	20.0	1	0	0.0
July	1981	10	0	0.0	-	-	-
December	1981	12	5	41.7	-	-	-
March	1982	13	2	15.4	-	-	-
June	1982	9	0	0.0	-	-	-
September	1982	17	3	17.6	-	-	-
Total		80	13	16.3	5	0	0.0
Combined Total		85	13	15.3			

Table 30. *P. tenuis* infection in moose feces from Study Area 3.

Month	Year	Little Port Hebert		
		N	I	%
February	1981	9	2	22.2
April	1981	6	3	50.0
June	1981	1	1	100.0
March	1982	5	1	20.0
April	1982	4	0	0.0
July	1982	14	4	28.6
Total		39	11	28.2

Table 31. *P. tenuis* infection in moose feces from Study Area 4.

Month	Year	Belmont Mountain			Delaney Settlement		
		N	I	%	N	I	%
March	1981	7	2	28.6	8	2	25.0
April	1981	-	-	-	2	0	0.0
January	1982	9	3	33.3	-	-	-
March	1982	12	0	0.0	-	-	-
Total		28	5	17.9	10	2	20.0
Combined Total		38	7	18.4			

Table 32. *P. tenuis* infection in moose feces from Study Area 6.

Month	Year	MacPherson's Lake		
		N	I	%
February	1981	12	0	0.0
April	1981	3	1	33.3
Total		15	1	6.7

Table 33. *P. tenuis* infection in moose and deer feces by density (from Study Areas).

Study area	Density	County	Deer			Moose		
			N	I	%	N	I	%
1	Low deer:low moose	Halifax	64	34	53.1	85	13	15.3
2	Low deer:no moose	Kings	187	140	74.9	-	-	-
3	Low deer:high moose	Shelburne	30	25	83.3	39	11	28.2
4	High deer:low moose	Colchester	80	58	72.5	38	7	18.4
5	High deer:no moose	Richmond	26	12	46.2	-	-	-
6	High deer:high moose	Pictou	42	17	40.5	15	1	6.7
Total			429	286	66.7	177	32	18.1

χ^2 for deer = 33.80 5DF P = 0.0000
χ^2 for moose = 4.47 3DF P = 0.2152

Table 34. *P. tenuis* infection in deer and moose by county.

County	Deer						Moose					
	Heads			Feces			Heads			Feces		
	N	I	%	N	I	%	N	I	%	N	I	%
Annapolis	2	0	0.0	-	-	-	-	-	-	-	-	-
Antigonish	-	-	-	-	-	-	13	0	0.0	7	1	14.3
Cape Breton	9	1	11.1	9	5	55.6	-	-	-	-	-	-
Colchester	18	12	66.7	82	58	70.7	9	2	22.2	57	7	12.3
Cumberland	7	2	28.6	32	26	81.3	23	0	0.0	49	5	10.2
Digby	2	1	50.0	2	0	0.0	-	-	-	-	-	-
Guysborough	-	-	-	-	-	-	8	2	25.0	14	1	7.1
Halifax	52	19	36.5	73	37	50.7	3	0	0.0	91	13	14.3
Hants	10	6	60.0	7	3	42.9	1	0	0.0	-	-	-
Inverness	7	4	57.1	7	3	42.9	3	0	0.0	5	2	40.0
Kings	-	-	-	188	141	75.0	-	-	-	-	-	-
Lunenburg	1	0	0.0	-	-	-	-	-	-	-	-	-
Pictou	6	2	33.3	42	17	40.5	13	0	0.0	39	1	2.6
Queens	8	6	75.0	3	2	66.7	2	1	50.0	1	0	0.0
Richmond	12	6	50.0	34	16	47.1	-	-	-	-	-	-
Shelburne	11	6	54.5	32	25	78.1	4	0	0.0	47	11	23.4
Victoria	10	5	50.0	12	7	58.3	3	0	0.0	7	0	0.0
Yarmouth	50	34	68.0	6	1	16.7	1	1	100.0	-	-	-
Total	205	104	50.7	529	341	64.5	83	6	7.2	317	41	12.9

χ^2 for deer heads = 25.24 16DF $P < 0.05$
χ^2 for deer feces = 51.13 13DF $P = 0.0000$
χ^2 for moose heads = 30.0 11DF $P < 0.01$
χ^2 for moose feces = 13.66 9DF $P = 0.1348$

Table 35. White-tailed deer kill by county, 1981 (from Statistics Canada, 1981 and Nova Scotia Dept. of Lands and Forests, 1981).

County	Total harvest	Estimated population[a]	Land area (sq. km.)	Kills per sq. km.
Annapolis	1,908	9,540	3,203.7	0.6
Antigonish	1,034	5,170	1,461.4	0.7
Cape Breton	1,284	6,420	2,472.9	0.5
Colchester	3,801	19,005	3,622.3	1.0
Cumberland	3,924	19,620	4,288.2	0.9
Digby	1,937	9,685	2,472.5	0.8
Guysborough	3,497	17,485	4,380.8	0.8
Halifax	3,165	15,825	5,557.3	0.6
Hants	2,811	14,055	3,054.7	0.9
Inverness	1,397	6,985	3,696.9	0.4
Kings	1,140	5,700	2,182.2	0.5
Lunenburg	3,252	16,260	2,880.4	1.1
Pictou	2,605	13,025	2,774.4	0.9
Queens	1,867	9,335	2,367.7	0.8
Richmond	980	4,900	1,230.2	0.8
Shelburne	1,843	9,215	2,356.5	0.8
Victoria	611	3,055	2,767.9	0.2
Yarmouth	2,502	12,510	2,070.7	1.2
Total	39,558	197,790	52,840.8	0.7

[a]Population estimated using 20% of total population harvested

FIGURES

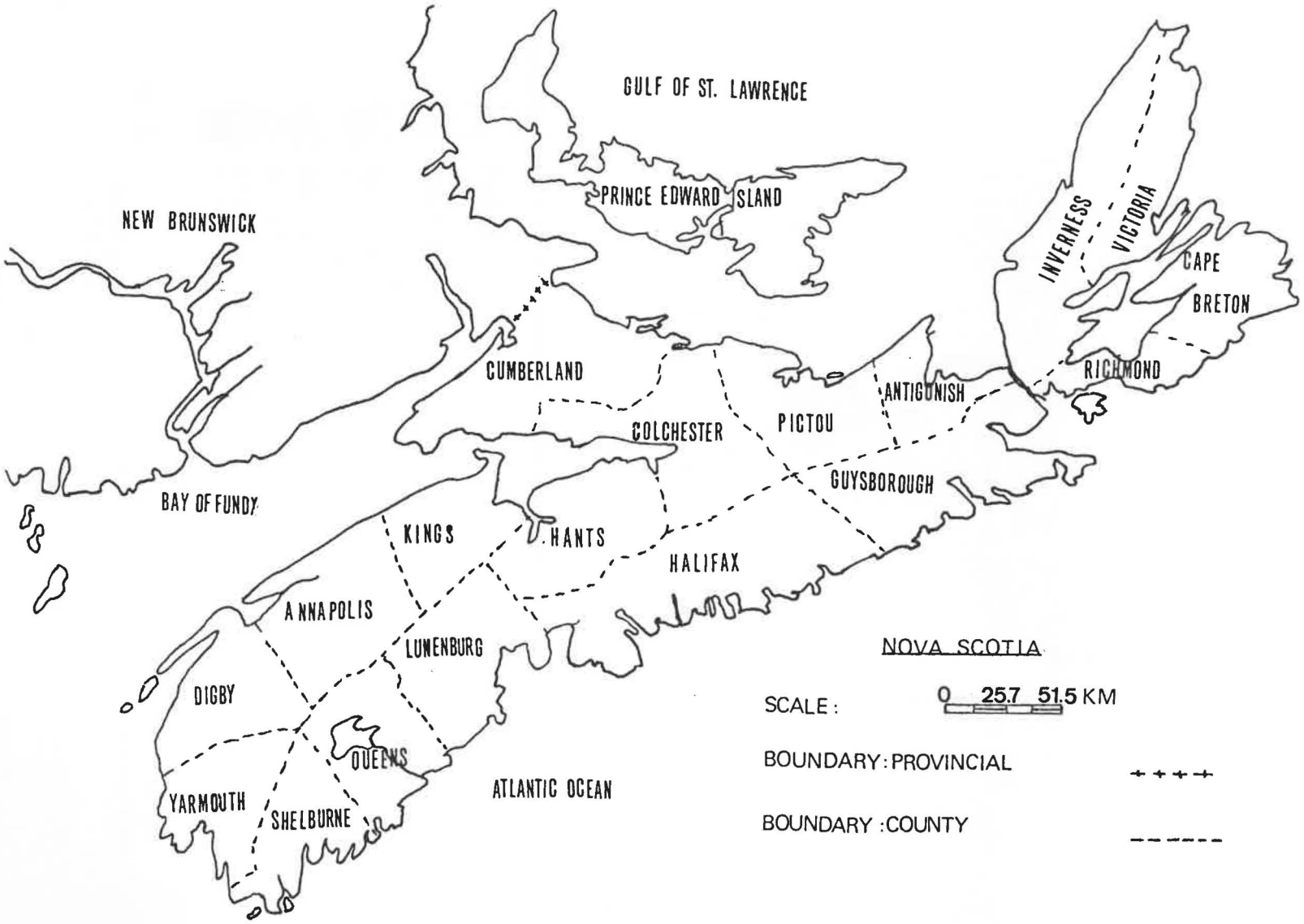


Figure 1. Map of Nova Scotia showing counties.

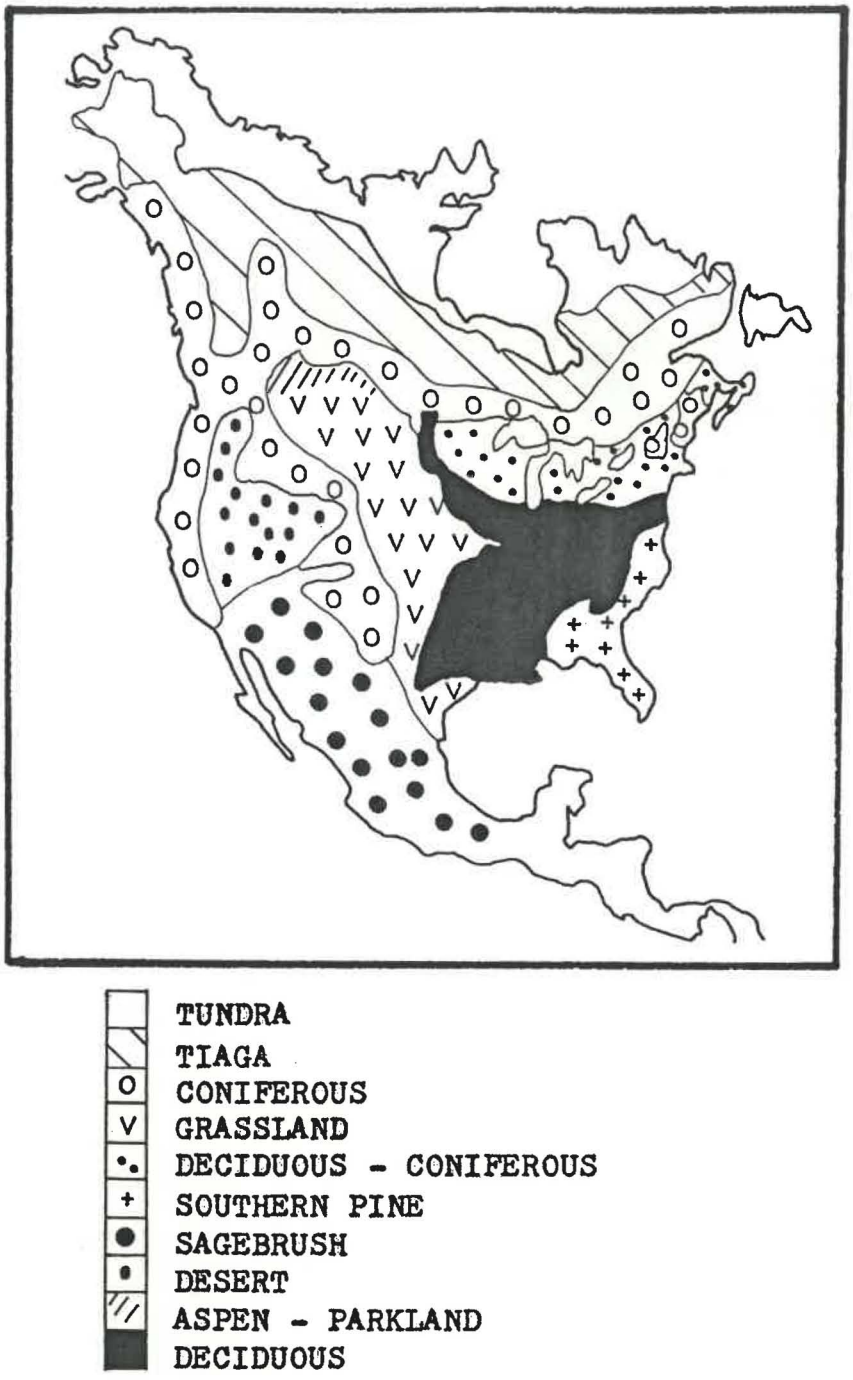


Figure 2. Schematic map of the major biomes of North America. Adapted from Anderson & Prestwood (1981).

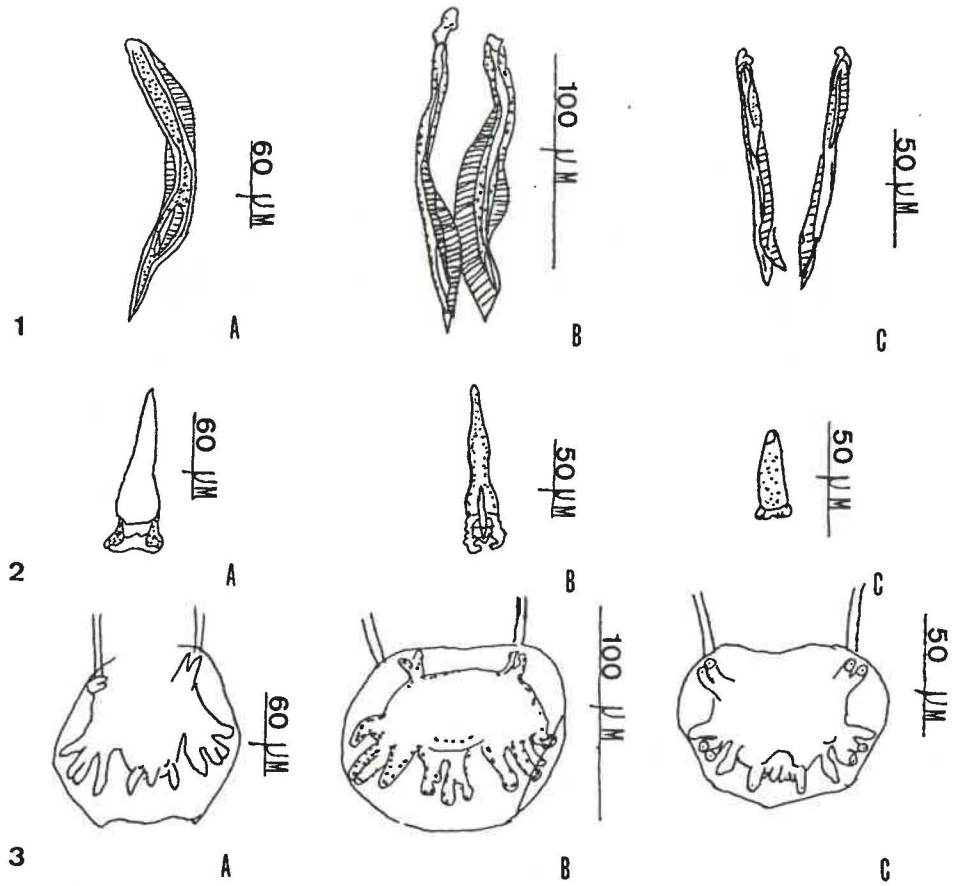


Figure 3. Comparison of male reproductive structures of *Parelaphostrongylus*. Adapted from Platt and Samuel (1978).

1. Spicules
2. Gubernaculum
3. Bursa
A. *P. tenuis*
B. *P. odocoilei*
C. *P. andersoni*

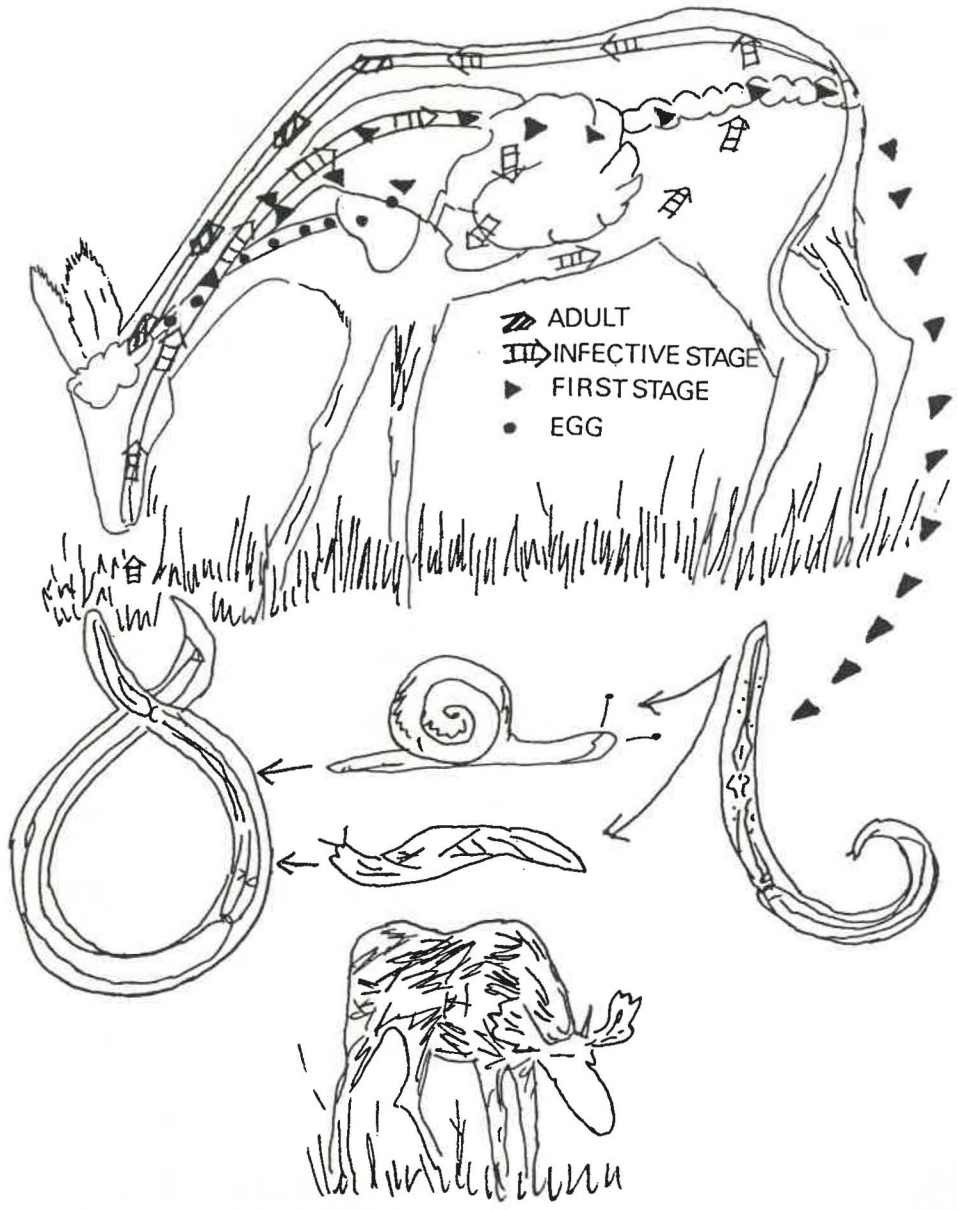


Figure 4. *P. tenuis* life cycle. Adapted from Anderson and Lankester (1974).

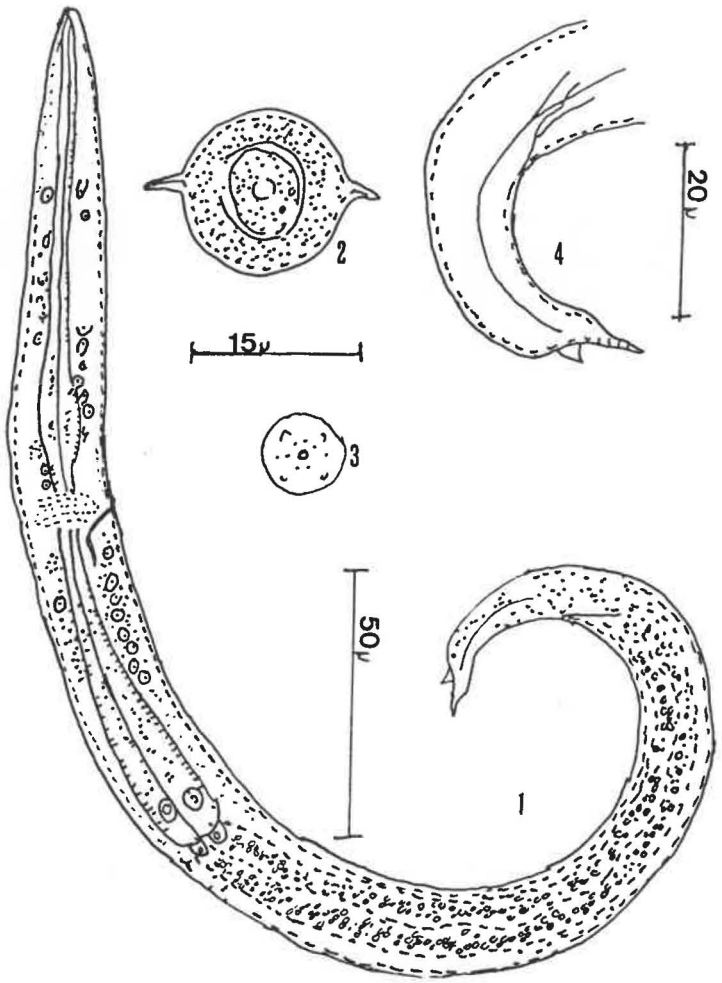


Figure 5. First stage *P. tenuis* larvae. From Anderson (1963).

1. First stage larva of *P. tenuis*, lateral view.
2. First stage larva, cross-section through middle of body showing lateral alae.
3. First stage larva, en face view.
4. First stage larva, lateral view of caudal extremity showing dorsal spine and lateral ala.

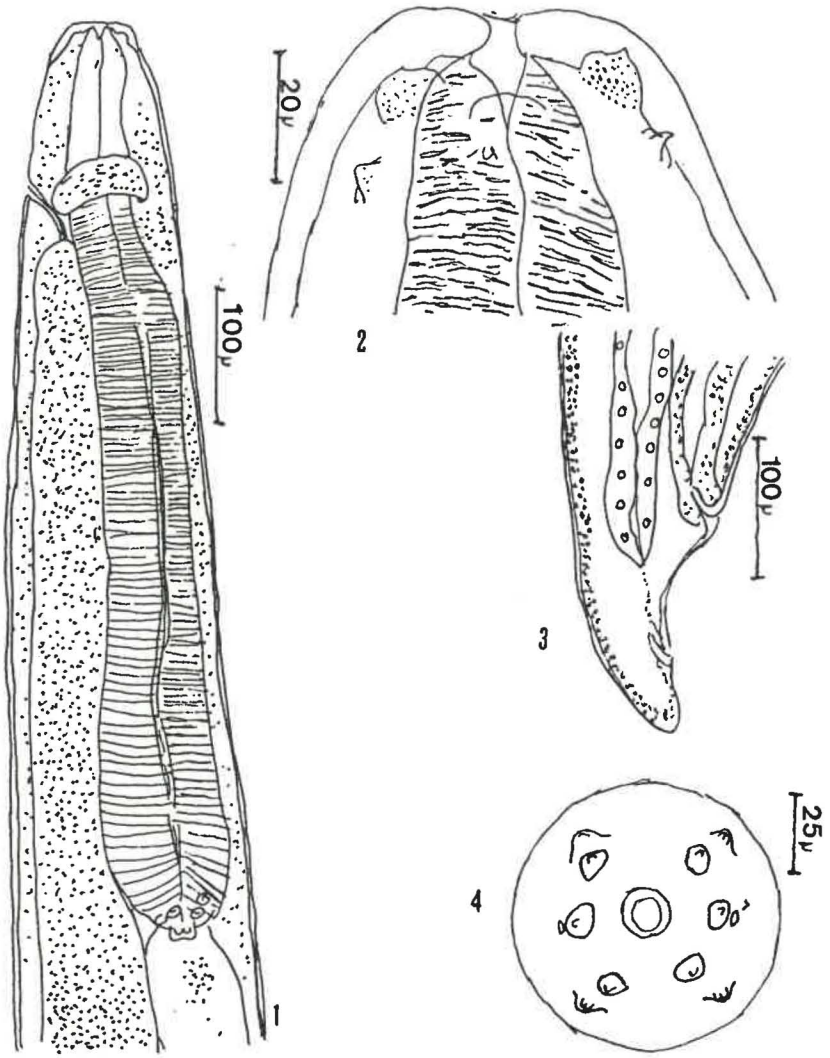


Figure 6. Adult *P. tenuis*. From Anderson (1956).

1. Anterior end male, lateral view, showing nerve ring, esophagus, excretory pores, terminal excretory duct and one excretory gland cell.

2. Anterior end male, lateral view.

3. Caudal end female, lateral view, showing anus, rectum, intestine, vulva and posterior part of ovijector.

4. Anterior end female, en face view.

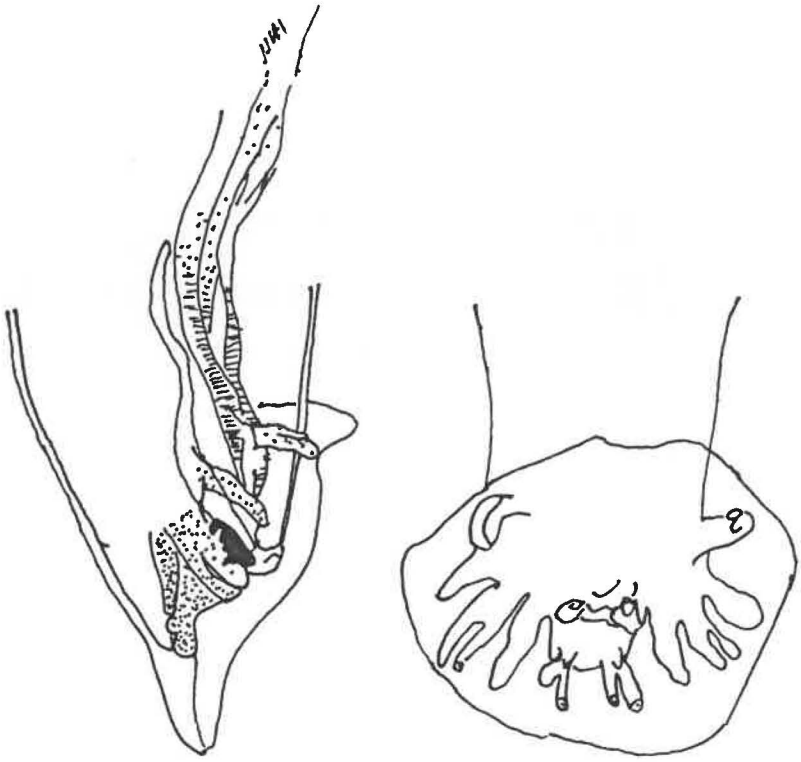


Figure 7. Adult male *P. tenuis*. From Anderson and Prestwood (1981).

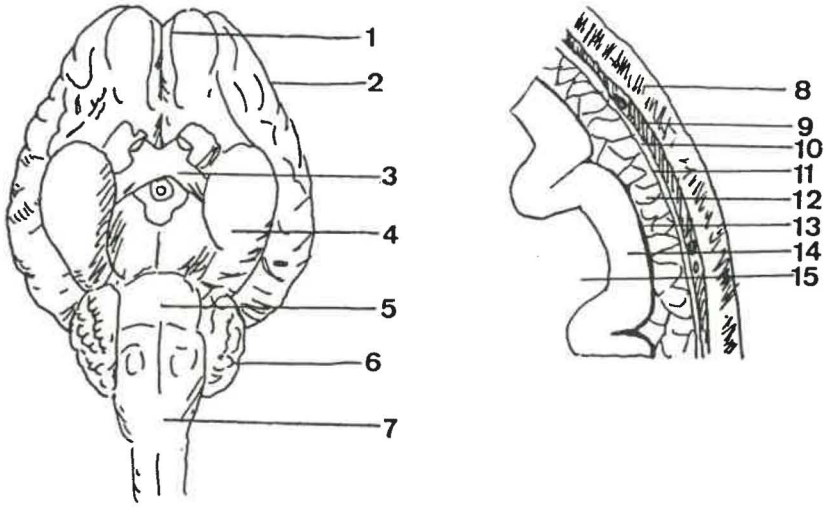


Figure 8. Ventral view of brain and cross section of meninges. Adapted from Gilbert (1968).

1. Olfactory bulb
2. Cerebrum
3. Optic chiasm
4. Piriform lobe
5. Pons
6. Cerebellum
7. Medulla oblongata
8. Bone
9. Dura mater
10. Subdural space
11. Arachnoid
12. Subarachnoid space
13. Pia mater
14. Gray matter
15. White matter

Figure 9. Map of Nova Scotia showing six study areas.

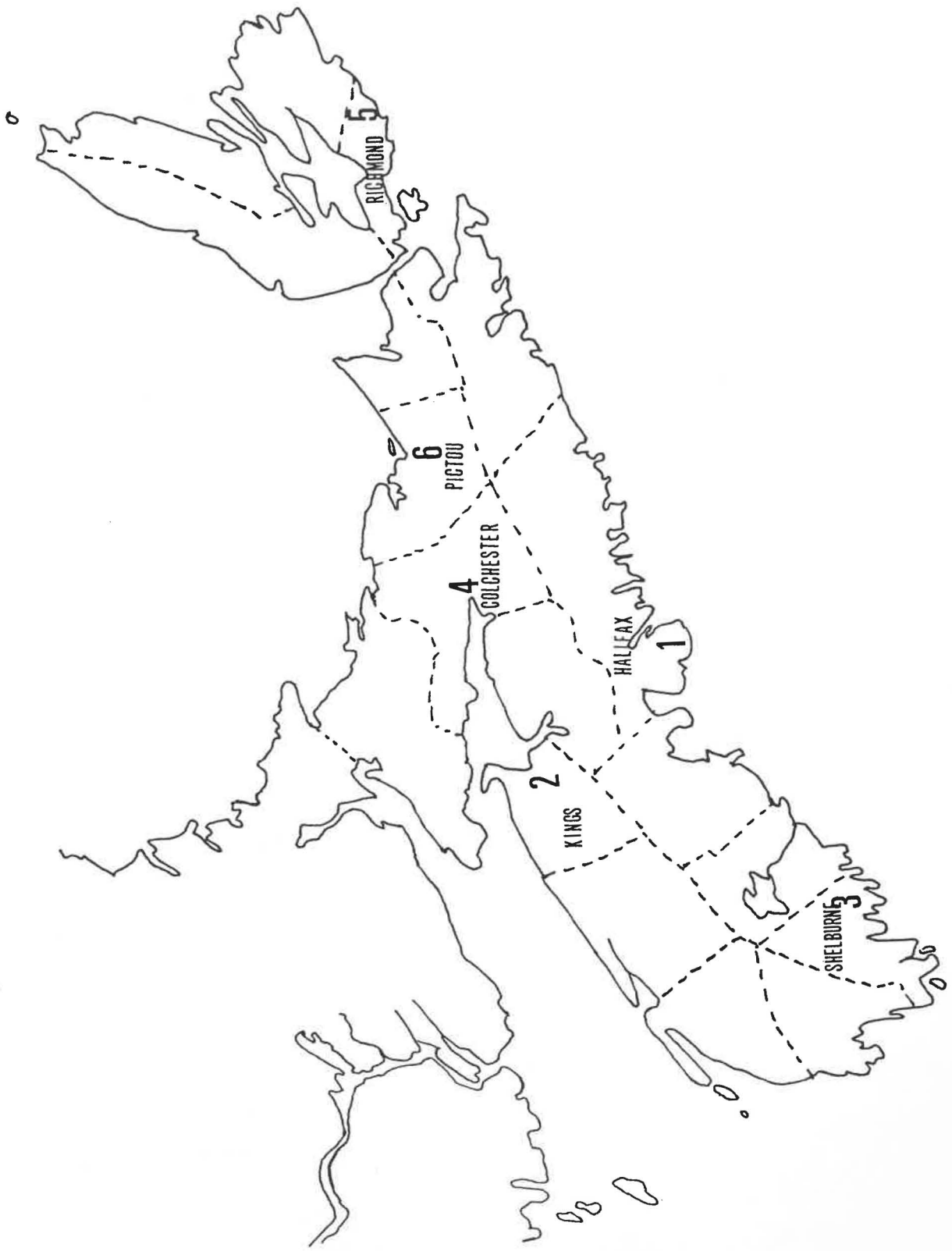

RICHMOND
5
6
PICTOU
4
COLCHESTER
HALIFAX
1
2
KINGS
3
SHELBURNE

Figure 10. Grover Lake, Halifax County, Study Area 1.

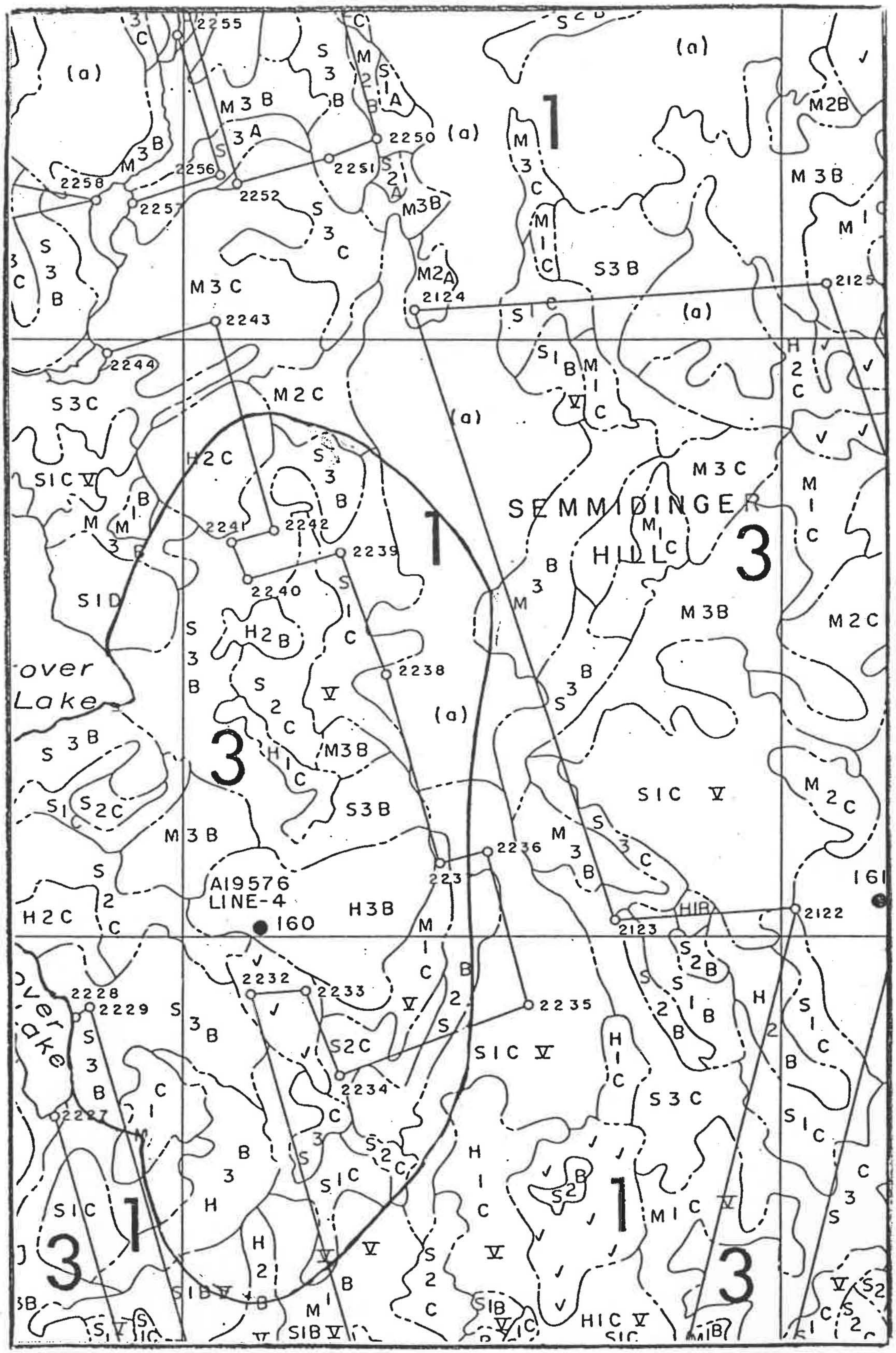

SEMMIDINGER
HILL
A19576
LINE-4
160
161
Lake

Figure 11. Mooseland, Halifax County, Study Area 1.

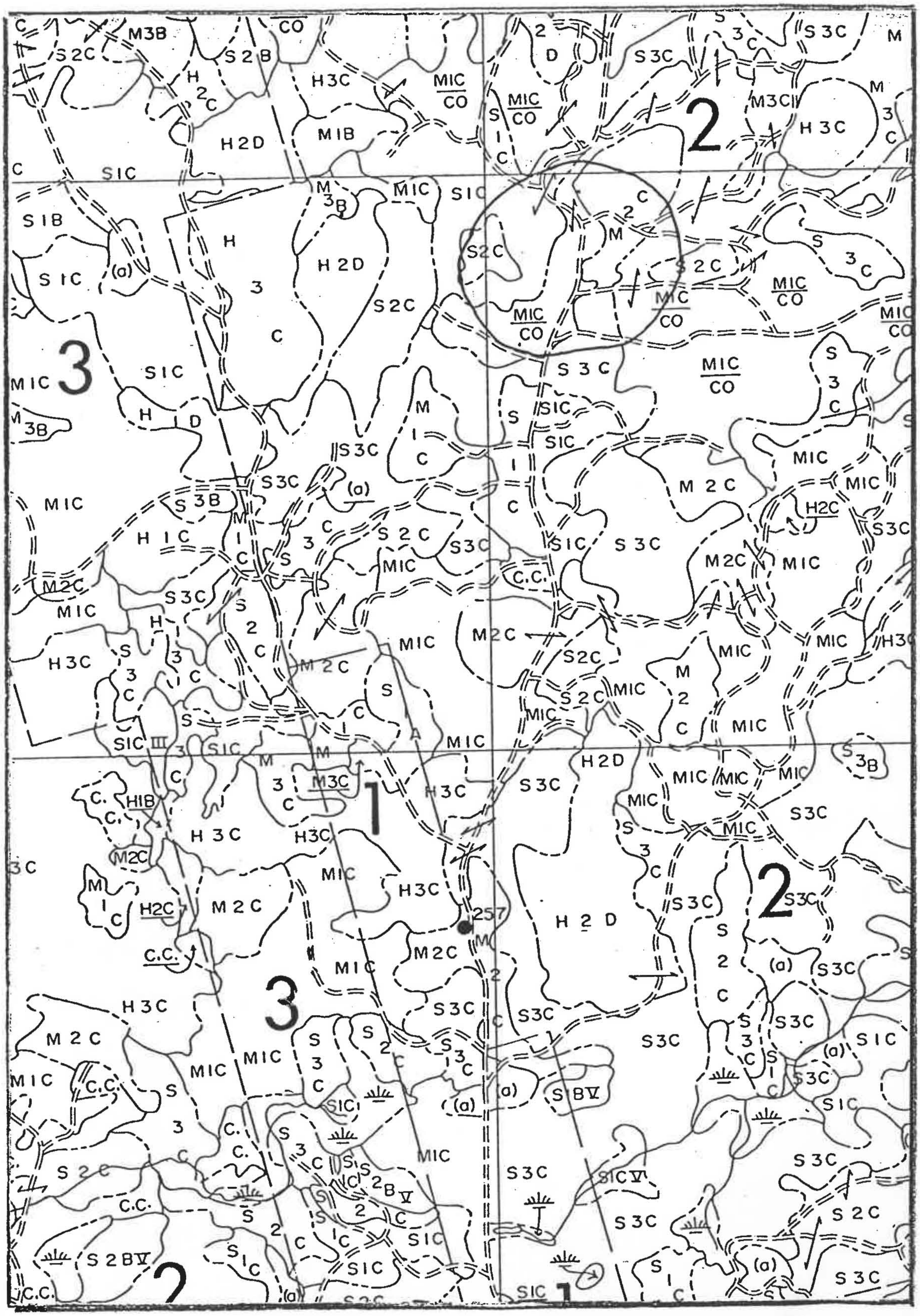

M3B
S2C
S2B
CO
H3C
MIC
CO
H2D
MIB
2
M3C
H3C
SIC
SIB
H
3
C
H2D
S2C
M
2
C
MIC
CO
S3C
3
MIC
S3C
SIC
S3B
H1C
M2C
MIC
M2C
S3C
H3C
M2C
H2C
SIC
MIC
H2D
S3C
MIC
1
H3C
H3C
M2C
C.C.
257
H2D
2
3
M2C
S3C
SBV
S2BV
SICV
S2C
S3C
2
1

Figure 12. Black River Lake, Kings County, Study Area 2.

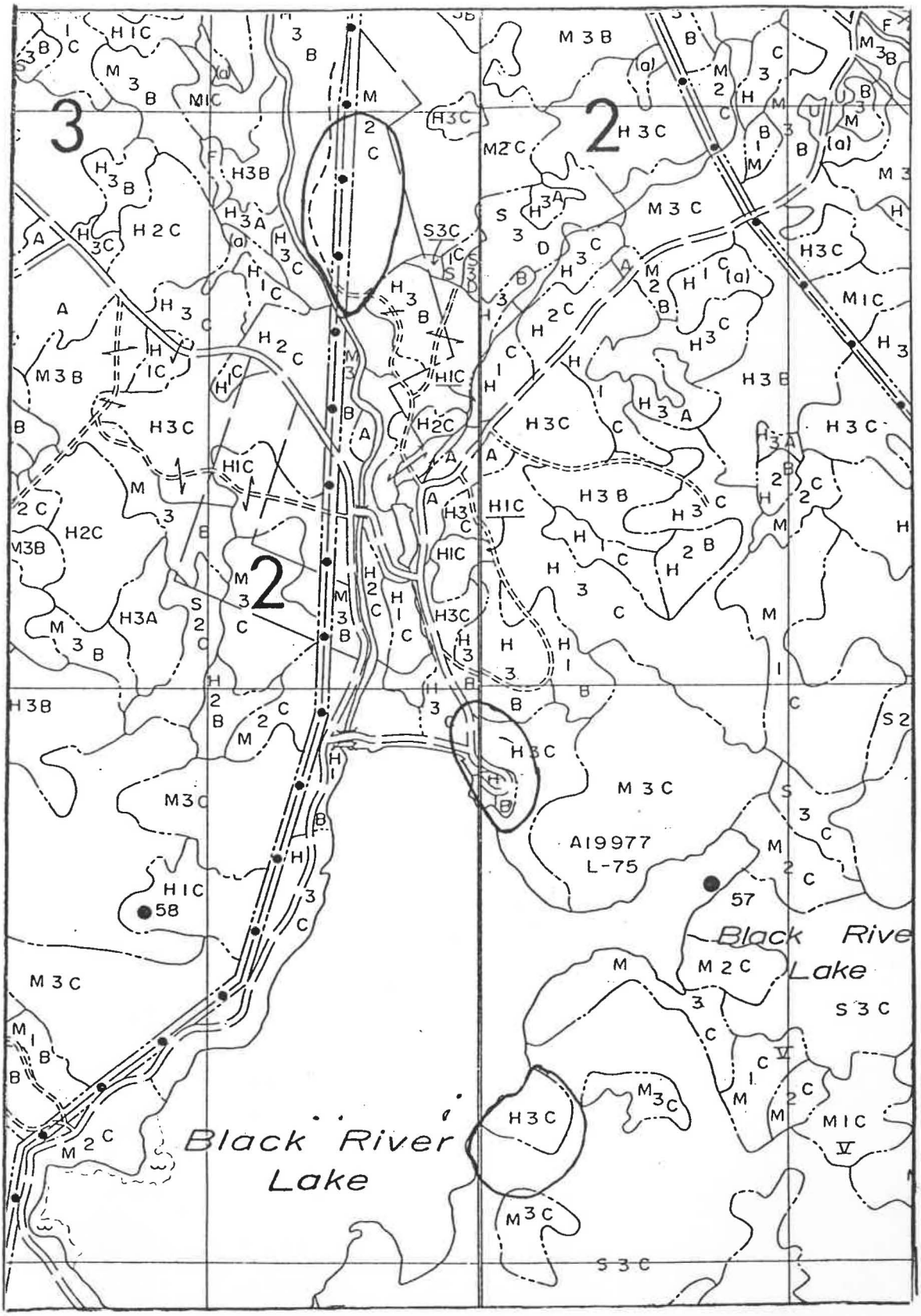

3
2
2
Black River Lake
Black River Lake
A19977
L-75
57
58
H3C
M3C
M2C
S3C
H2C
H3B
M3B
H1C
M1C

Figure 13. Huntingdon Brook, Kings County, Study Area 2.

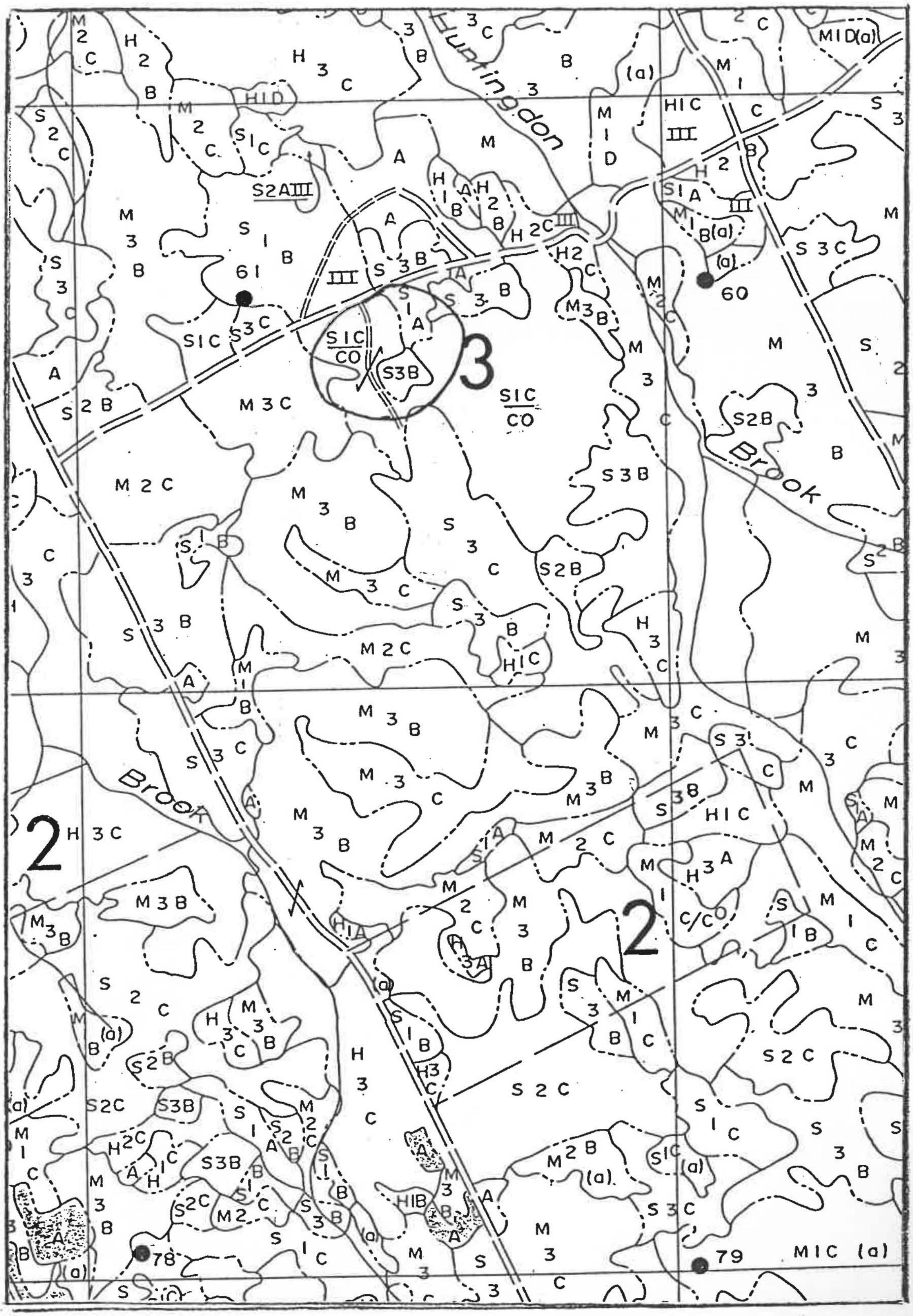

Huntingdon
Brook
Brook
61
60
78
79
3
2
2

Figure 14. North Mountain, Kings County, Study Area 2.

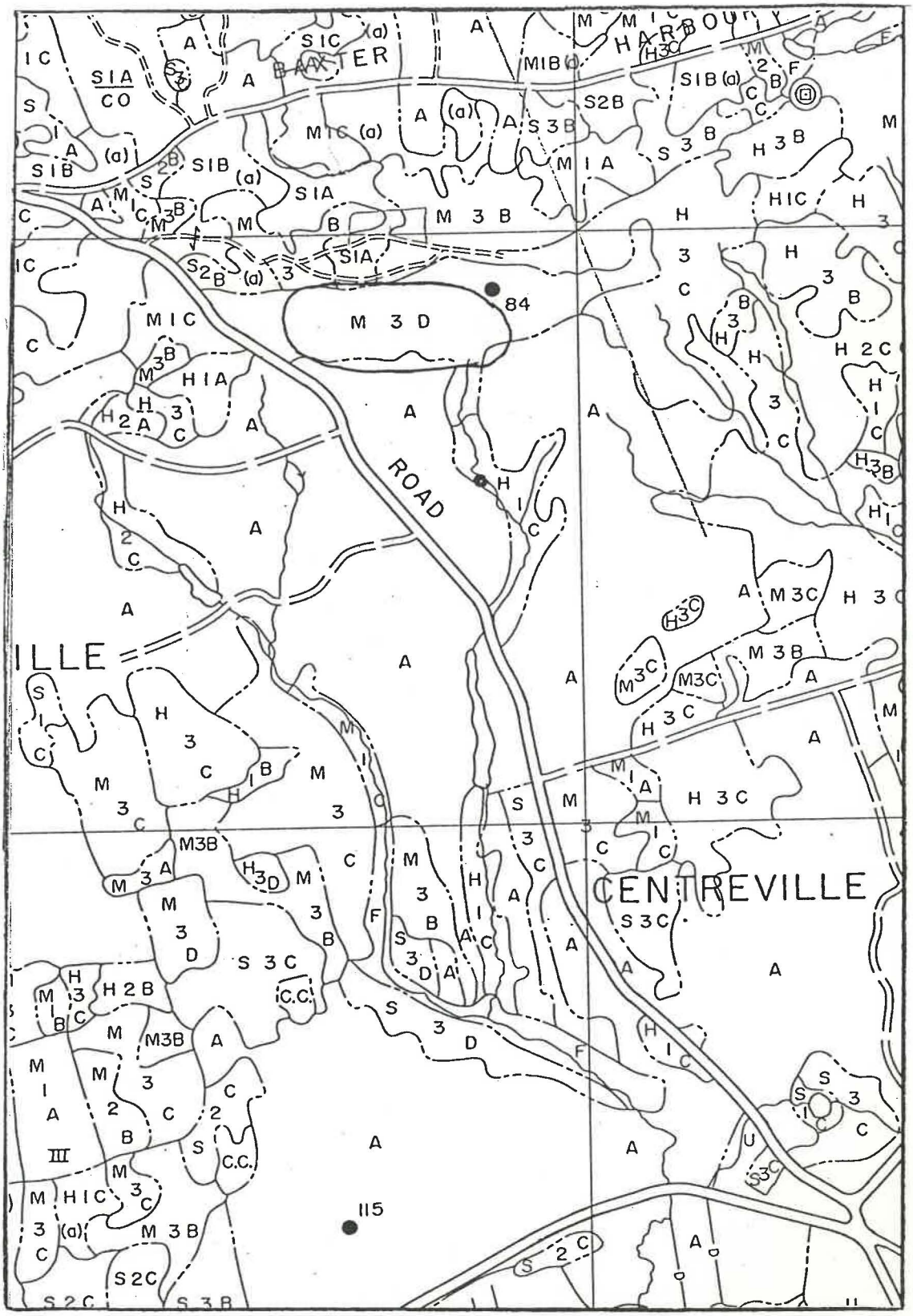

ROAD
CENTREVILLE
84
115

Figure 15. Little Port Hebert, Shelburne County, Study Area 3.

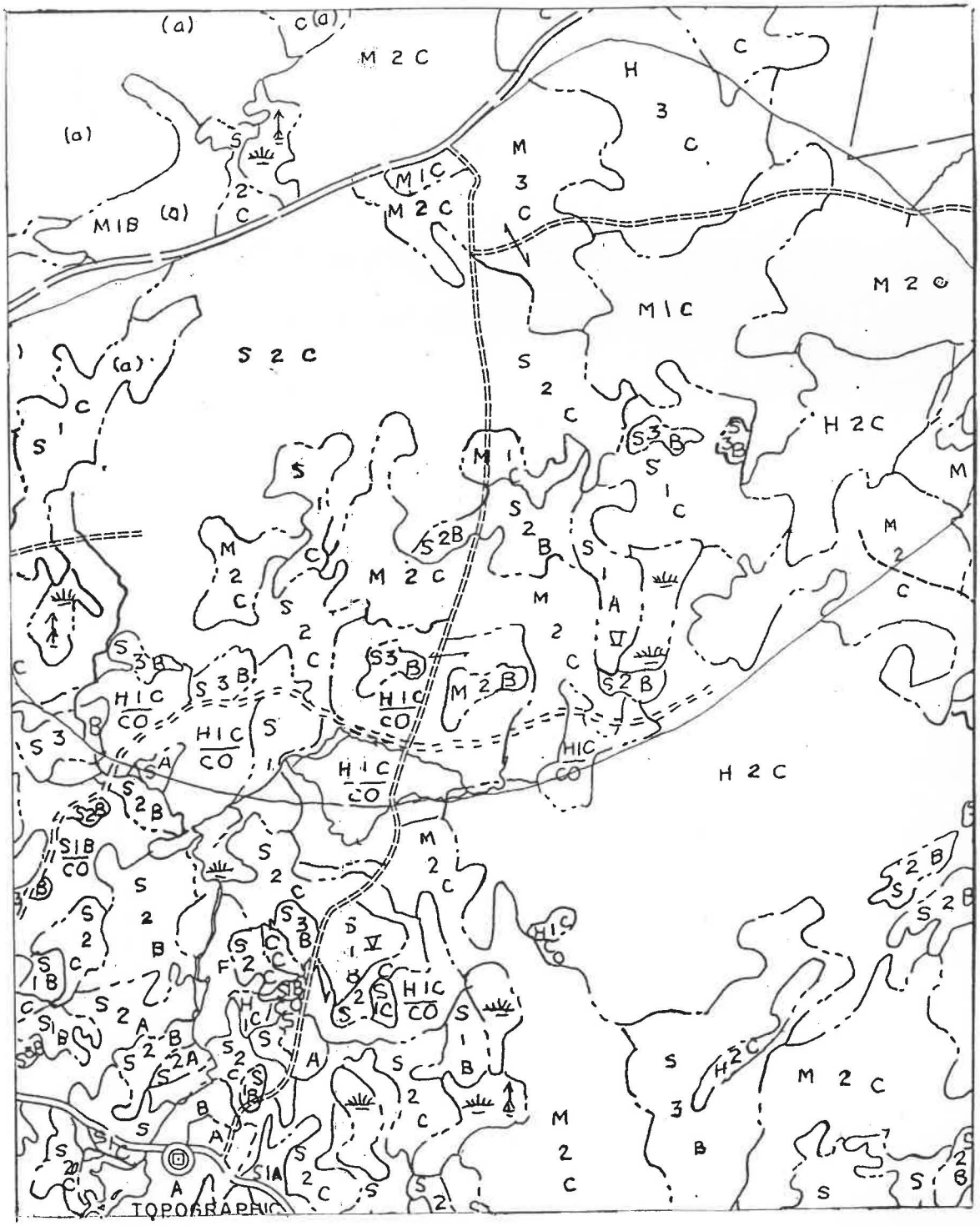

(a)
M 2 C
H 3 C
M 3 C
M 1 C
M 2 C
M 1 B
S 2 C
H 2 C
H 1 C / C O
S 1 B / C O
S 3 B
S 2 B
M 2 B
TOPOGRAPHIC

Figure 16. Belmont Mountain, Colchester County, Study Area 4.

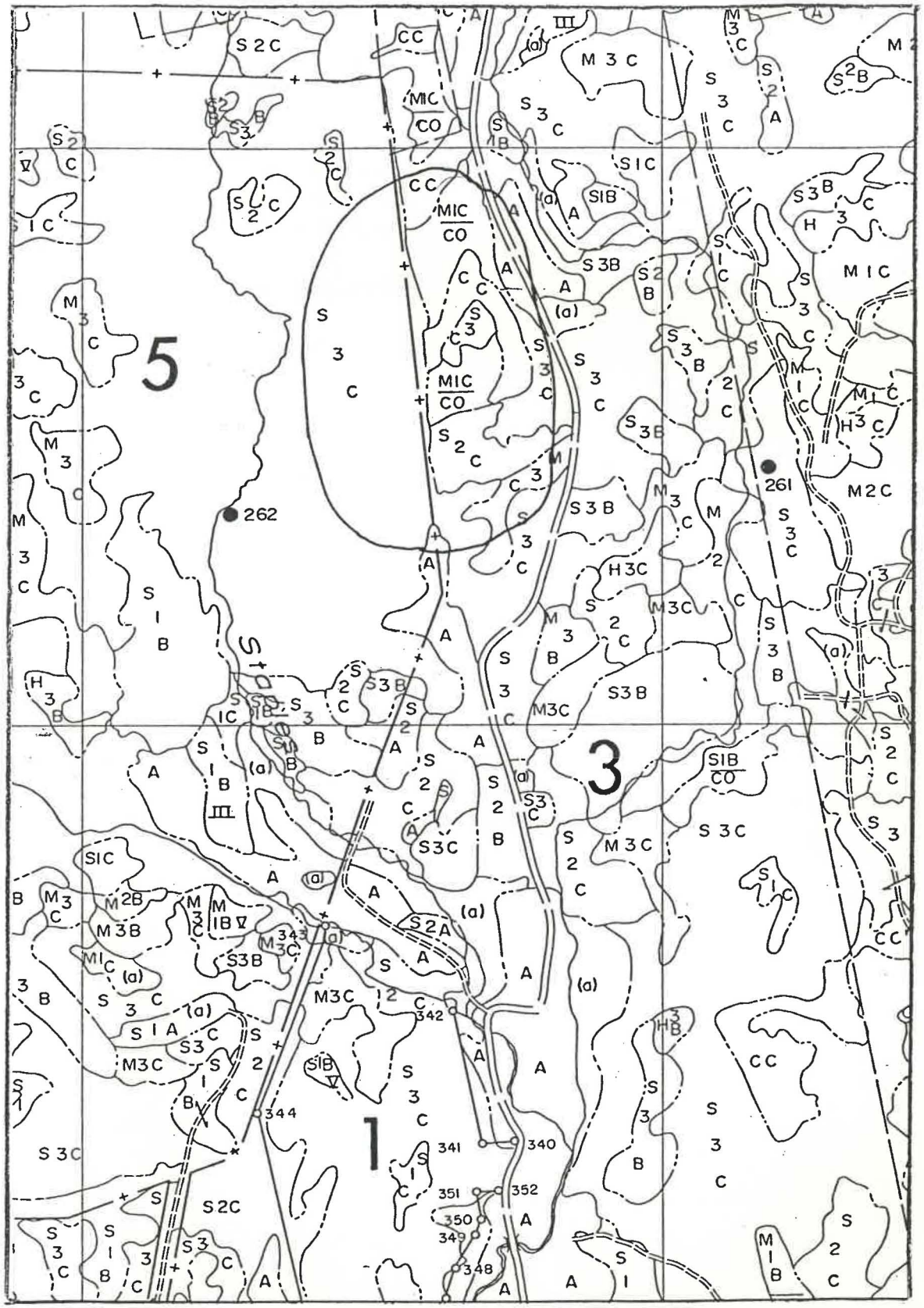

5
3
1
262
261
344
343
342
341
340
351
352
350
349
348

Figure 17. Delaney Settlement, Colchester County, Study Area 4.

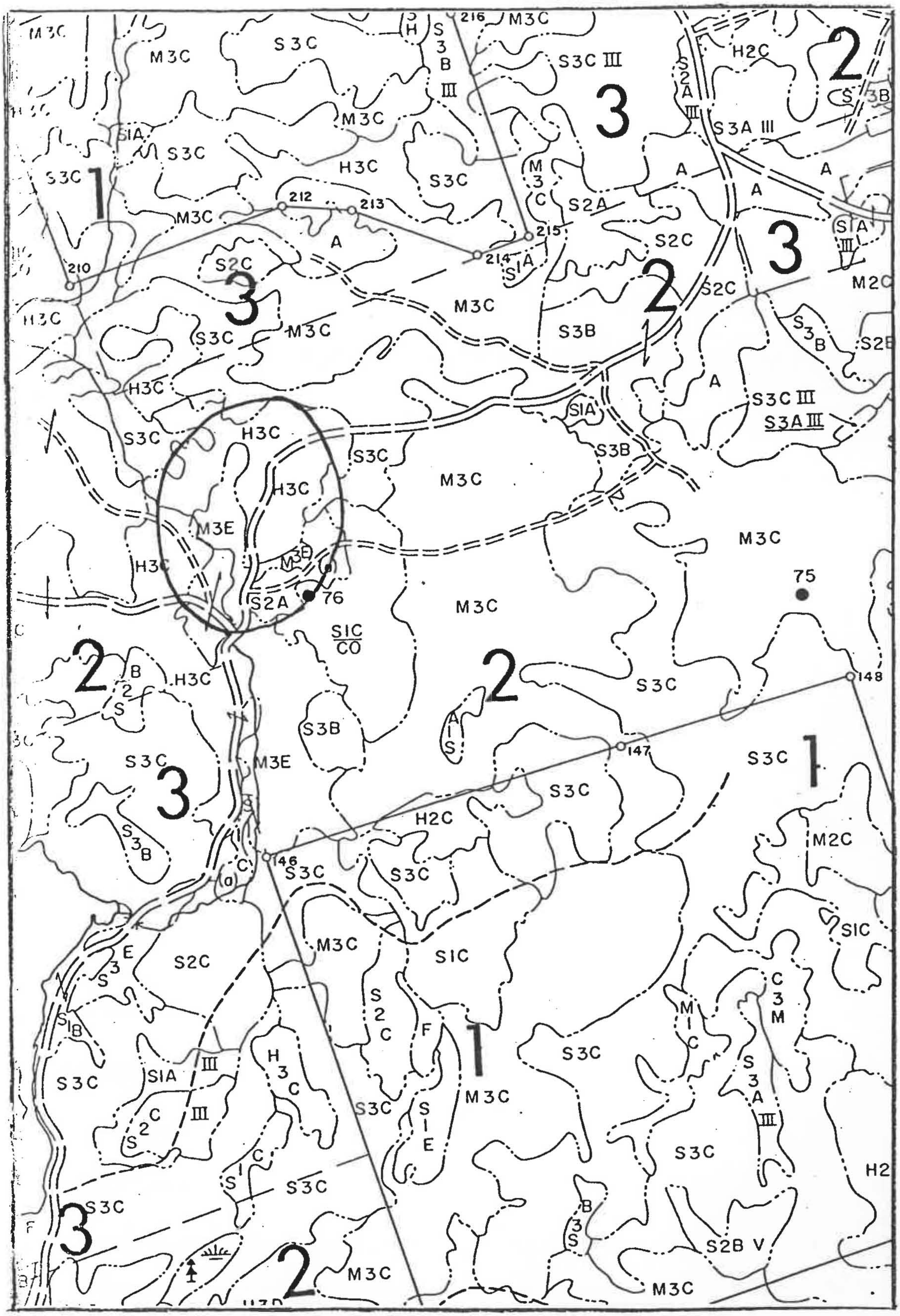

M3C
S3C
S3B III
216
S3C III
H2C
S3B
S2A III
S3A III
SIA
H3C
212
213
A
214
215
S2A
S2C
210
M2C
S2C
M3C
S3B
S3C III
S3A III
SIA
S3B
M3E
S2A
76
SIC
CO
75
148
147
146
H2C
M2C
S2C
SIC
S3C
S2B V
H2
1
2
3

Figure 18. Capelin Cove (a), Richmond County, Study Area 5.

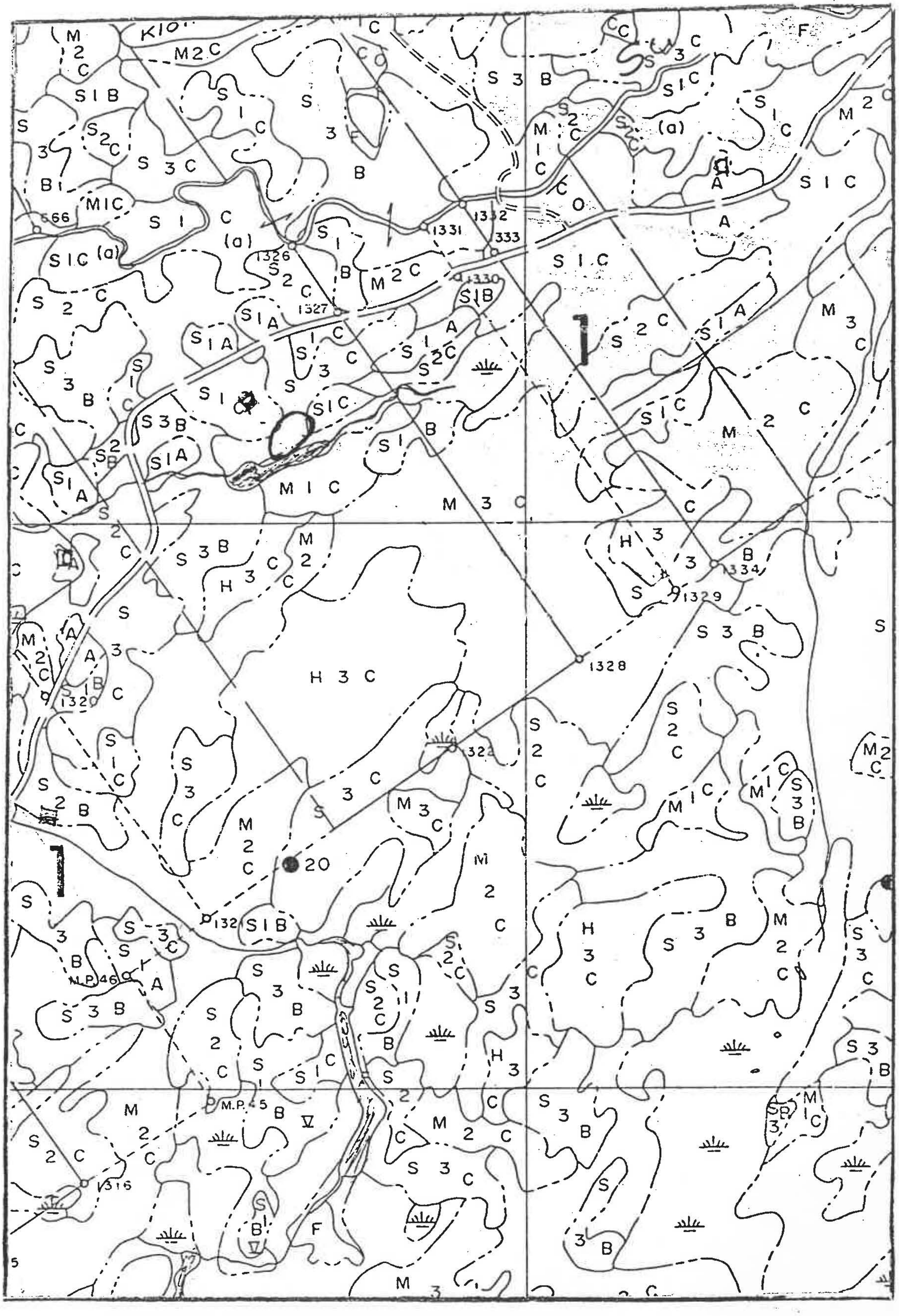

M2C
S1B
S2C
S3C
MIC
S1C (a)
S3B
S1C
M2C
S1B
1326
1327
1331
1332
333
1330
S1C
S1A
M3C
S2C
M2
S1A
S3B
MIC
M3C
H3C
S3B
1334
1329
1328
S3B
S2C
1320
S3C
M2C
20
132
S1B
M.P.46
S3B
H3C
M.P.45
1316
M2C
S3C
F
M3

Figure 19. Capelin Cove (b), Richmond County, Study Area 5.

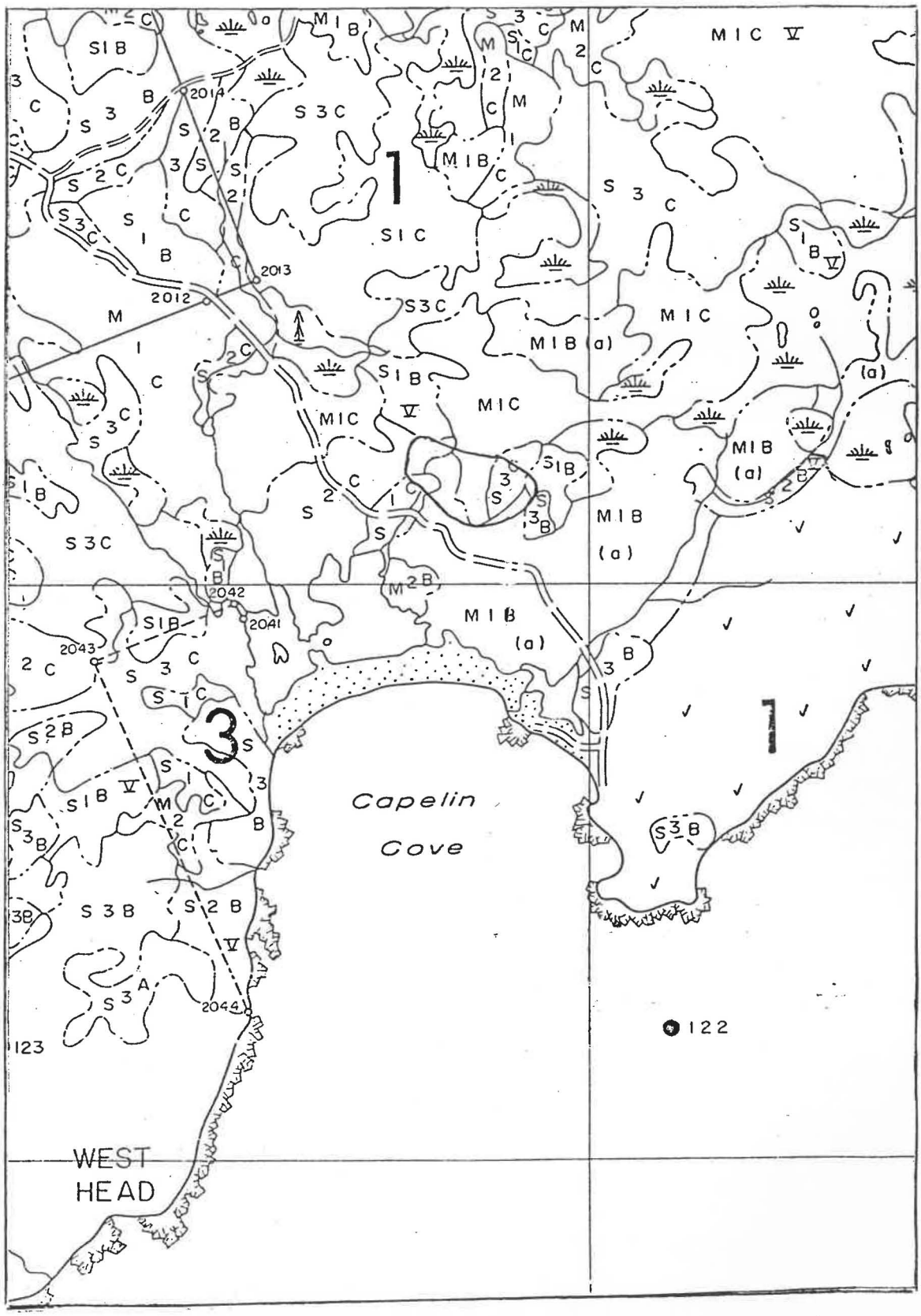
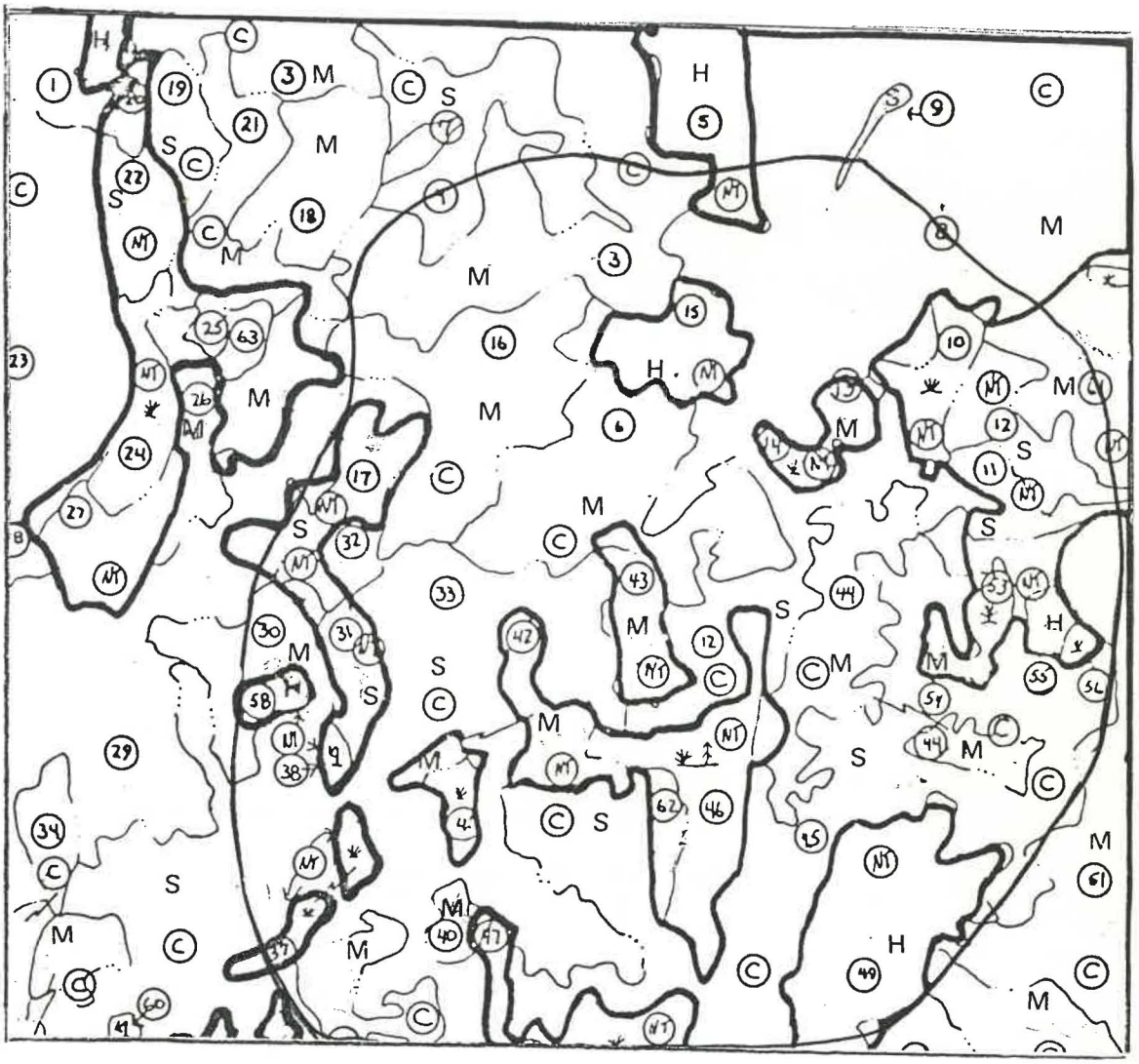

Capelin
Cove
WEST
HEAD
122
123
2012
2013
2014
2041
2042
2043
2044
MIC V
S3C
MIB (a)
S3B

Figure 20. MacPherson's Lake, Pictou County, Study Area 6.


H
C
M
S
NT

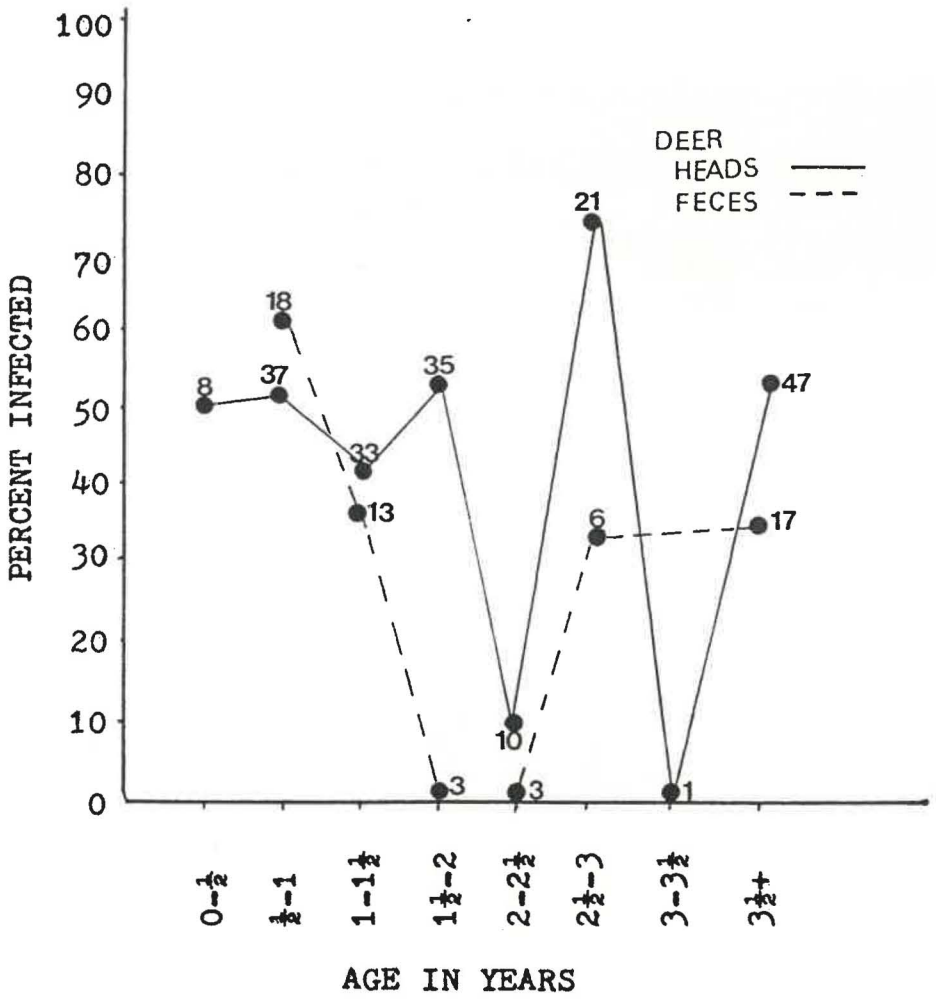


Figure 21. *P. tenuis* infection in deer by age. (sample size given)

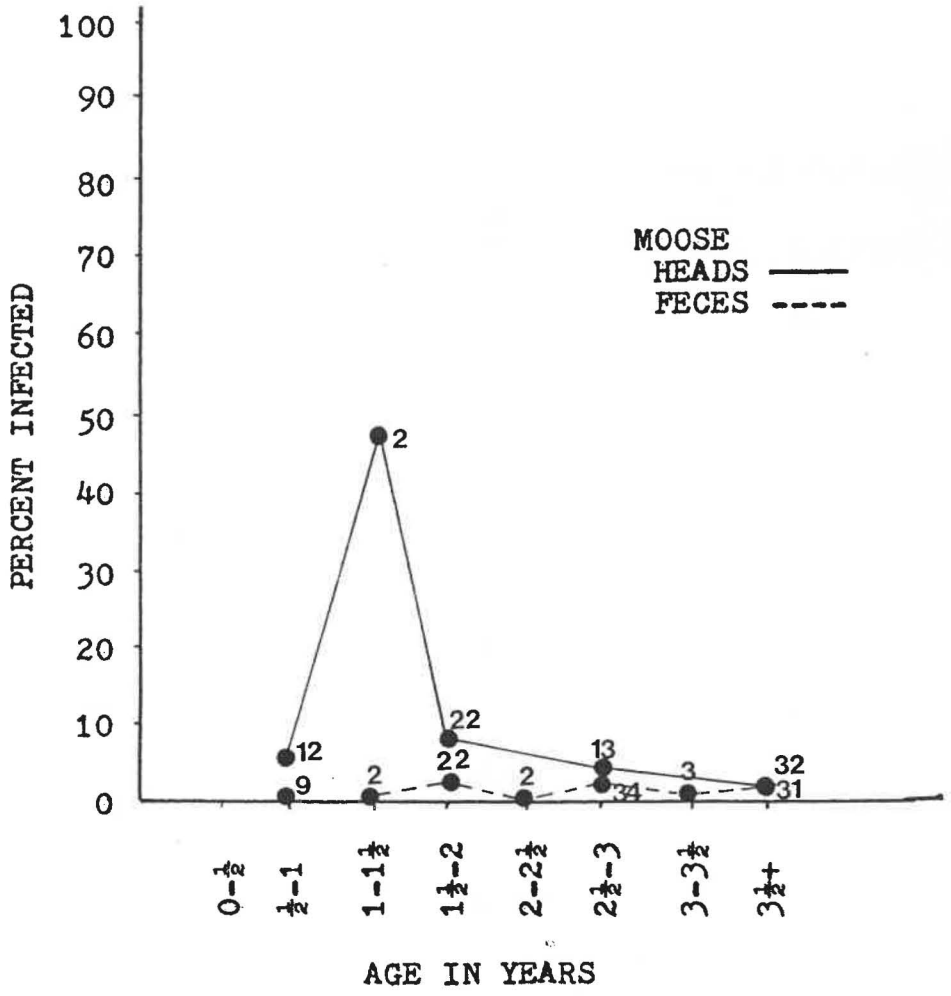


Figure 22. *P. tenuis* infection in moose by age.

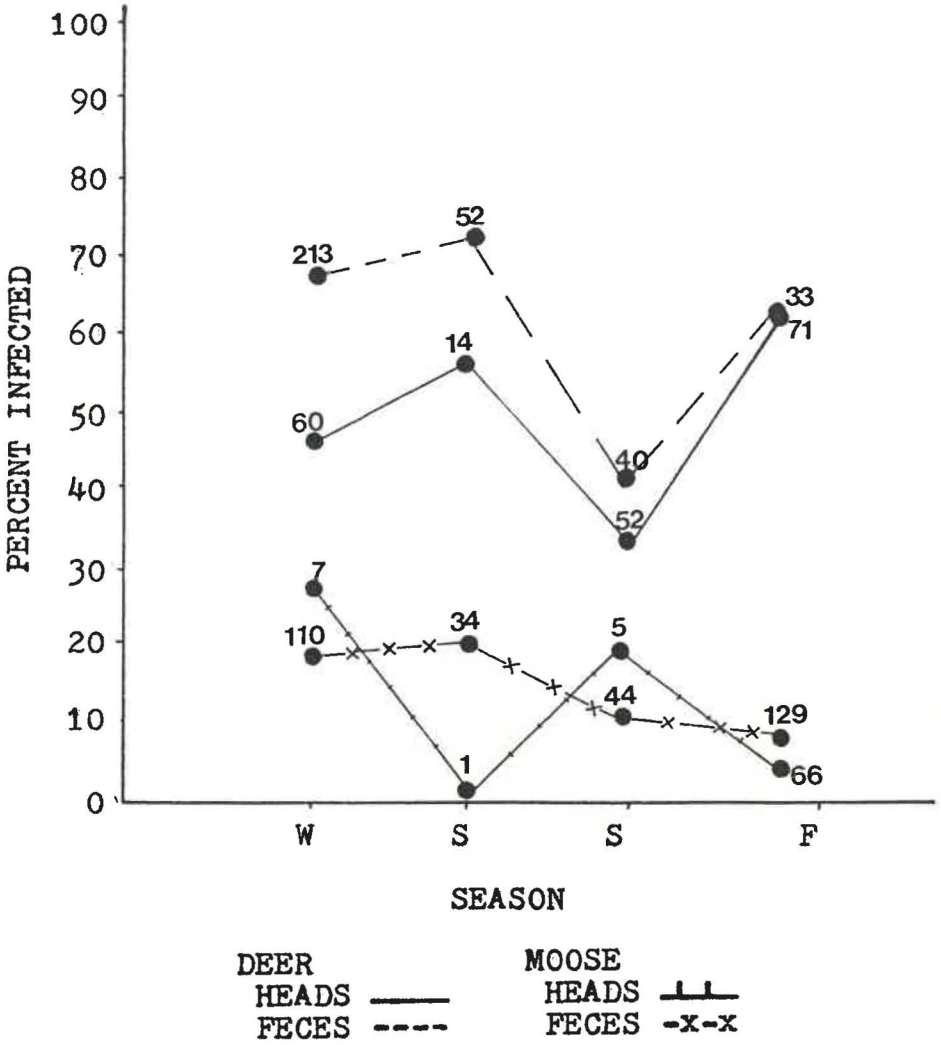


Figure 23. *P. tenuis* infection in deer and moose by season.

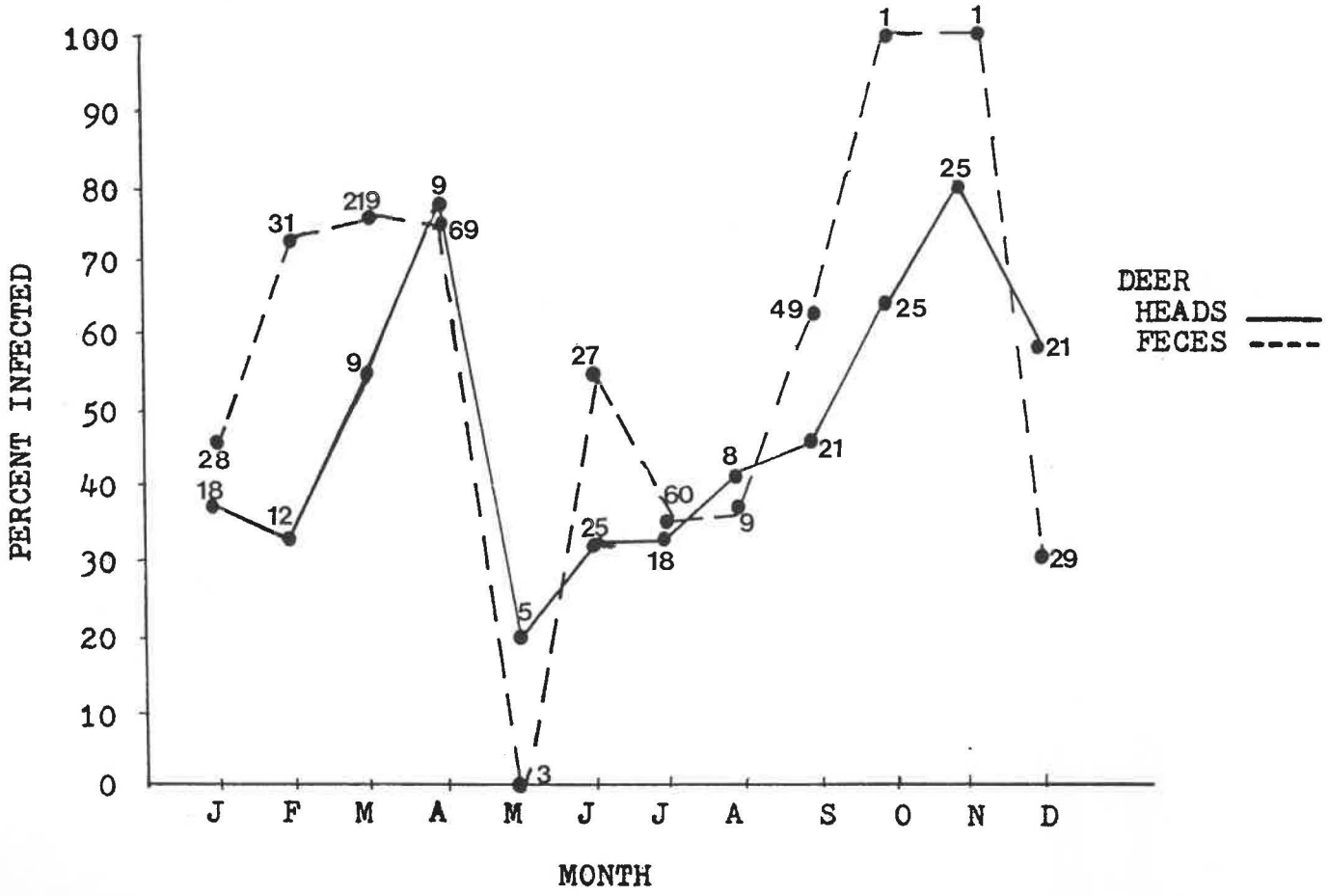


Figure 24. *P. tenuis* infection in deer by month.

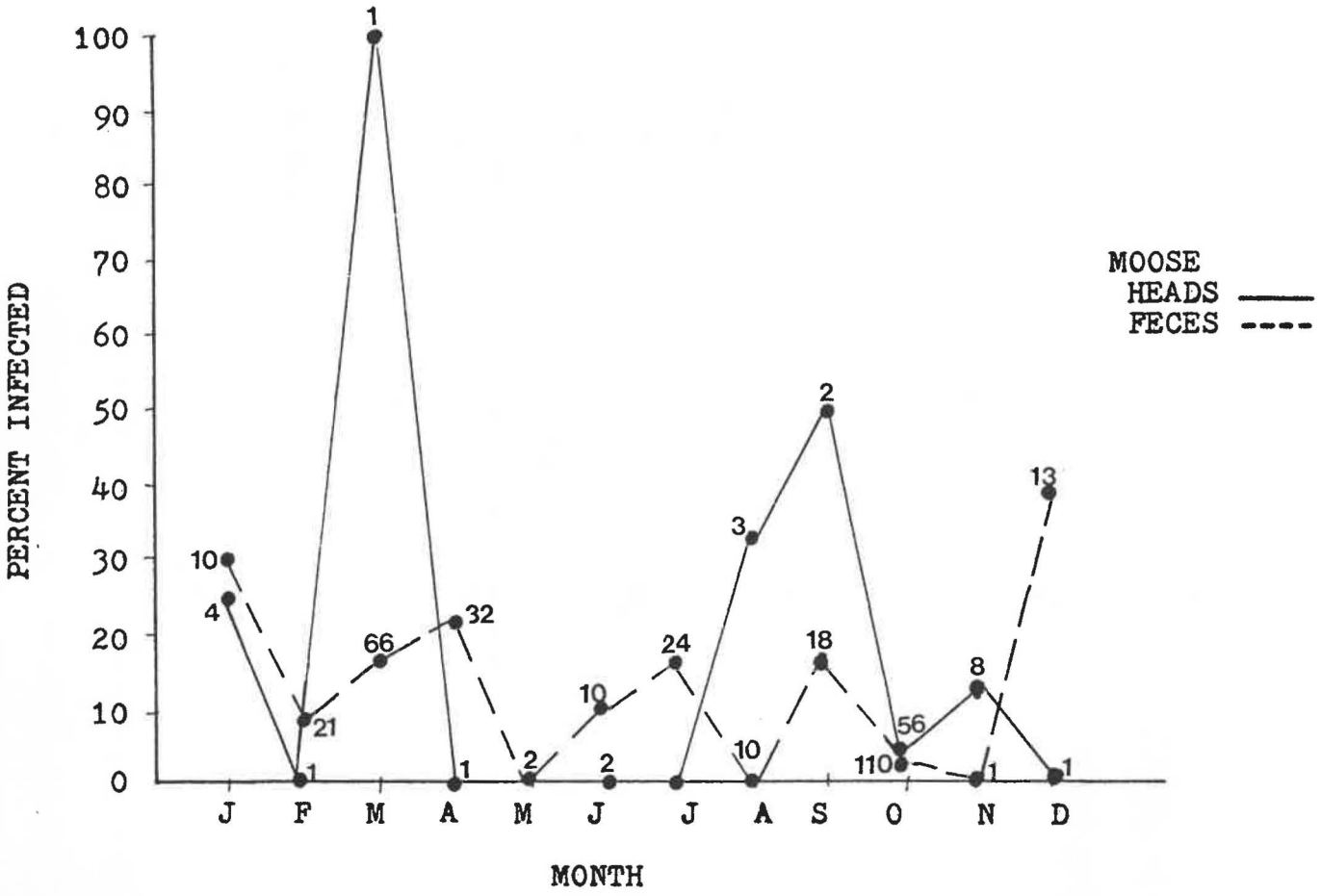


Figure 25. *P. tenuis* infection in moose by month.

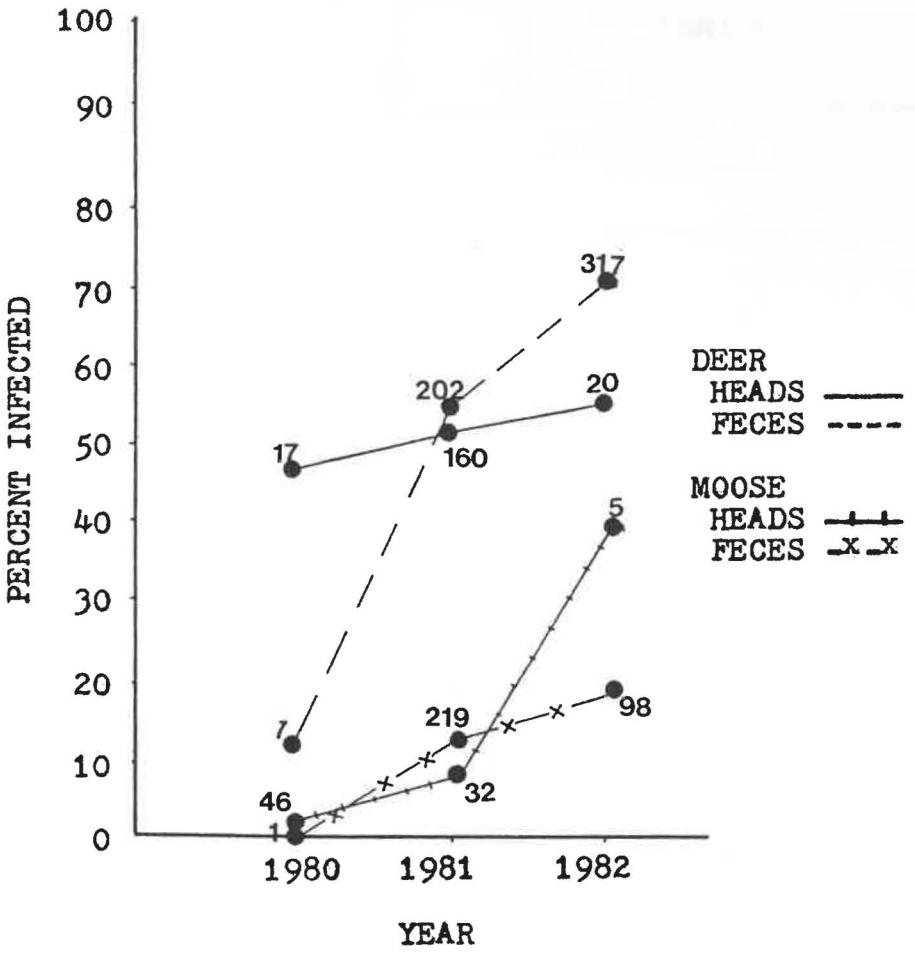


Figure 26. *P. tenuis* infection in deer and moose by year.

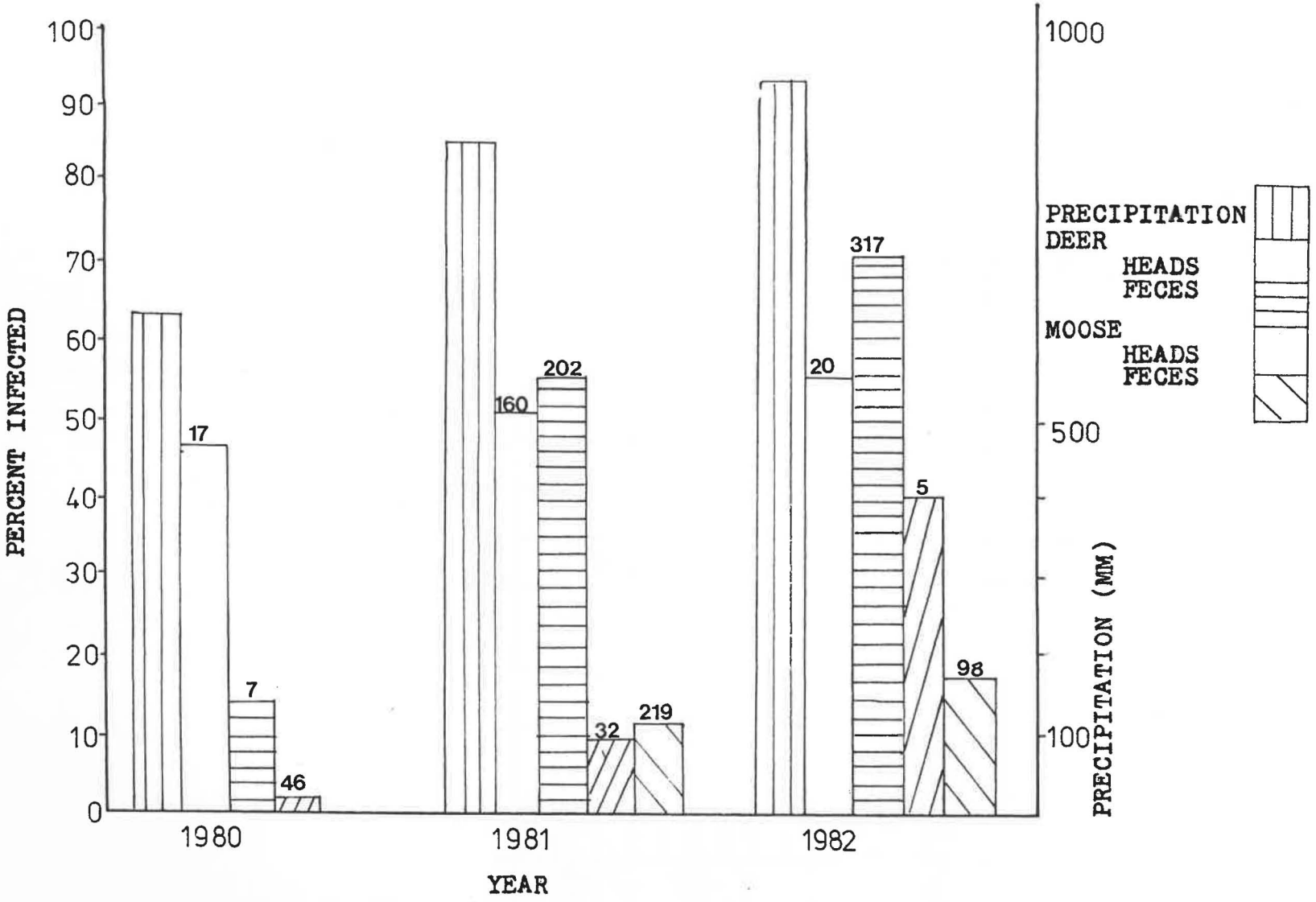


Figure 27. *P. tenuis* infection in deer and moose yearly with annual precipitation.

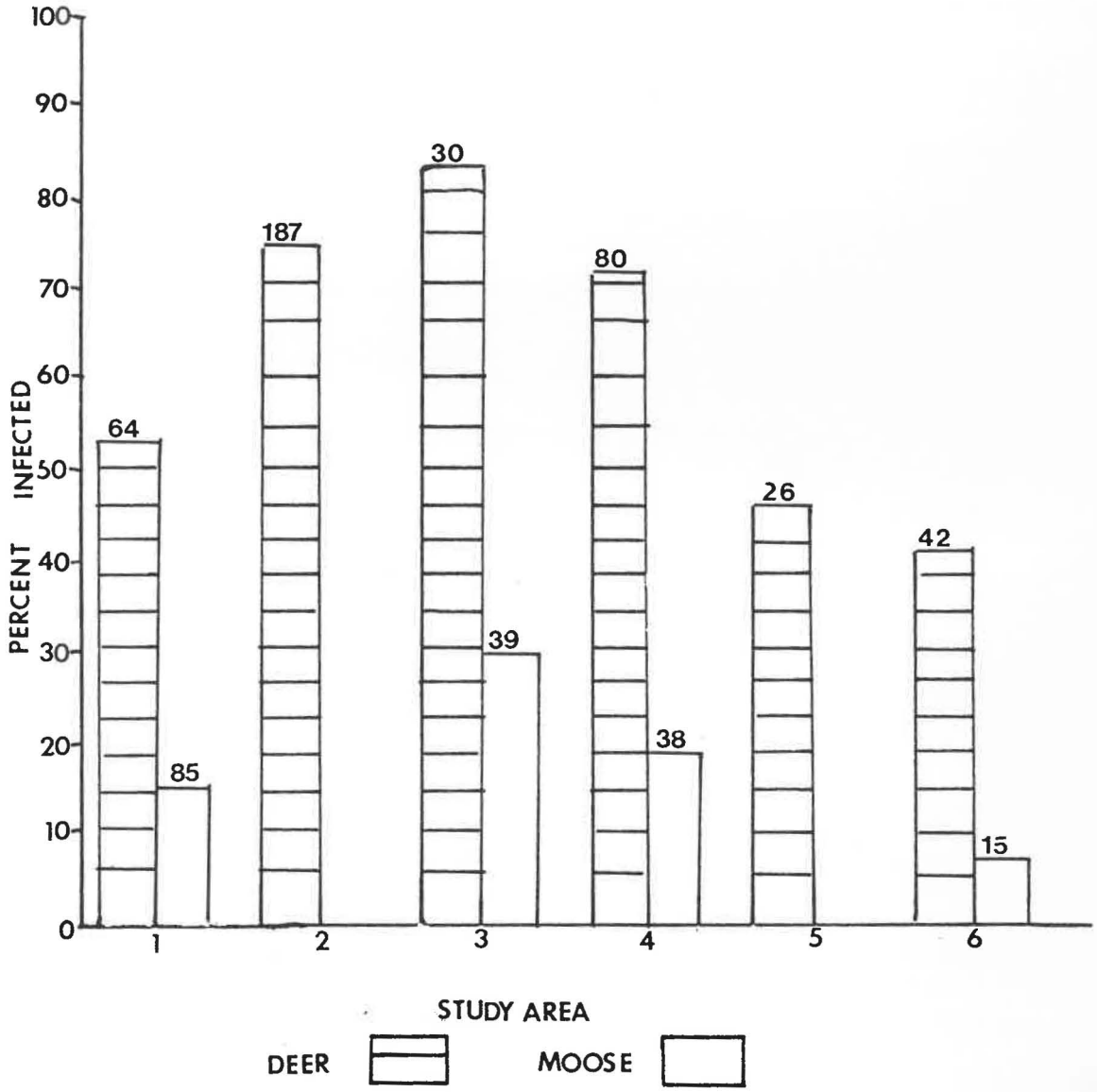


Figure 28. *P. tenuis* infection in deer and moose feces from Study Areas.