AN EVALUATION OF THE EFFECTS OF PARELAPHOSTRONGYLOSIS ON MOOSE POPULATIONS

BY

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ABSTRACT

Long-term historical data were examined for associations between moose (*Alces alces*) population declines, white-tailed deer (*Odocoileus virginianus*) densities, and reports of parelaphostrongylosis as a test of the hypothesis that *Parelaphostrongylus tenuis* causes moose declines. Moose population declines over the past 80 years were associated with deer densities greater than 5/km^2. This observation may be useful to managers but did not allow any effects of *P. tenuis* to be separated from other possible causes of moose declines. Whether moose numbers were stable, increasing, or decreasing, was independent of the occurrence of reports of sick moose. The best test of causality that could be constructed, namely, an increase in the reporting rate of observed sick animals concurrent with moose population declines, did not support the hypothesis. Although there is doubt that reporting rates are representative, a reasonable test of the hypothesis has been possible and the suggestion that *P. tenuis* has caused declines in moose populations is not supported by the historical information available.

Moose populations were also studied in parts of Ontario where they co-habit with white-tailed deer infected with *P. tenuis*. Trend data on changes in cervid numbers for the period 1980-92, current population density estimates and records of moose sickness were obtained from Ontario Ministry of Natural Resources managers; presence and abundance of *P. tenuis* were determined by examining winter deer feces for dorsal-spined larvae. Moose and deer co-exist in 45 of 83 Ontario Wildlife

Management Units (WMU's) surveyed and have persisted there for at least the past 12 years. Cervid populations in most of these WMU's are presently believed stable or increasing. Moose density was inversely related to deer density and was greatest where deer were $<4.0/km^2$. In addition, moose densities were lowest in areas with the highest mean intensity of *P. tenuis* larvae in deer feces. Case studies of moose declines suggest that the effect of this parasite on moose populations is more subtle than previously believed, and further study is required to separate and measure its importance relative to other mortality factors known to act on moose populations.

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INTRODUCTION

In 1964, Anderson (1964) proved experimentally that the parasitic nematode, *Parelaphostrongylus tenuis* acquired from white-tailed deer (*Odocoileus virginianus*), caused a neurologic disease identical to that described historically in moose (*Alces alces*) of eastern North America. Soon thereafter, Anderson (1965) and others (Loken *et al.* 1965, Smith and Archibald 1967) confirmed these results by finding adult worms in wild moose exhibiting signs of moose sickness. However, even before the etiology of this disease was understood, moose sickness (or parelaphostrongylosis) had been associated with observed declines in moose populations by Aldous and Mendall (1941), Lamson (1941), Cameron (1949), Benson (1958), and Dodds (1963). When *P. tenuis* was shown to be the disease agent, the relationship between deer and moose sickness became evident, and the hypothesis that parelaphostrongylosis may have played a role in moose declines emerged (Telfer 1967, Karns 1967, Anderson 1972, Gilbert 1973, Prescott 1974 and reviews by Anderson and Prestwood 1981, Lankester 1987).

In the 1980's, a gradual increase was seen in both moose and deer numbers over parts of their shared range and wildlife biologists and cervid managers began to question the importance of *P. tenuis* as a cause of moose mortality (Cole 1981, Lenarz and Kerr 1987, Thomas and Dodds 1988, Upshall *et al.* 1987). Some even suggested that moose and the nematode may be coevolving toward a more tolerant relationship (Clarke and Bowyer 1986). Subsequently, Nudds (1990) reminded

workers that the idea of *P. tenuis* causing moose declines had never been rigorously tested. He also challenged theories proposed by Telfer (1967), Gilbert (1974), and Kearney and Gilbert (1976) to explain why moose persist in proximity to deer in certain areas (Nudds 1990, Gilbert 1992, Nudds 1992).

Ontario is one of the areas in which moose and deer presently appear to co-exist. They have had a long history in this part of eastern North America and populations have fluctuated greatly. Their ranges reportedly first overlapped along the northshore of Lake Superior and west to Manitoba between 1900 and 1920, concurrent with a climatic warming trend (Peterson 1955, Anderson 1965, Voigt 1991). Along with this northward range expansion, provincial deer numbers increased to the mid-50's but by 1980 had declined by over 50% (Voigt 1991). Ontario's moose population increased to 1920, decreased from the mid 1940's to the early 50's and was considered low enough to close the sport harvest season in 1949-50 (Cumming 1972, Karns 1987). Thereafter, moose increased to the mid 1960's, only to decline again by the mid-70's (Chamberlain *et al.* 1978, Bergerud 1981, Kelsall 1987, Thompson and Euler 1987).

Since 1980, the numbers of both deer and moose have been increasing in much of Ontario (Timmermann 1987, Voigt 1991). Anecdotal information suggests that this has occurred even in areas of the Province where the two species are interspersed or appear to share range. Concurrent increases in both cervids have similarly been suggested in Maine (Clark and Bowyer 1986, Bogacyzk *et al.* 1993), New Brunswick (Upshall *et al.* 1987) and Nova Scotia (Thomas and Dodds 1988).

Such reports were not expected because the deer in all of these areas are infected with *P. tenuis* (Gilbert 1973, Upshall *et al.* 1987, Thomas and Dodds 1988). Because moose sickness has been suggested as a possible cause of moose declines earlier in the century, a logical extension of this paradigm predicts that existing moose populations will decline when sympatric, infected deer increase. However, the parasite's potential role in present day deer-moose population dynamics has not been examined at an appropriate temporal and spatial scale to assess its importance.

Therefore, in an effort to better understand this cervid-parasite relationship, a two-part study was undertaken. The first was to compile and evaluate historical data to examine associations between moose and white-tailed deer densities, and reports of sick moose, as a means of testing the hypothesis that *P. tenuis* causes declines in moose populations. Cervid population and harvest data used in analyses were from 6 areas where moose sickness has been reported over the past 80 years, including northeastern and northwestern Minnesota, northern and southern Maine, New Brunswick, and Nova Scotia. The second part surveyed wildlife managers across much of Ontario for the purpose of obtaining, on a Wildlife Management Unit (WMU) basis, trend data on changes in deer and moose numbers over the past 12 years, current population density estimates, and any records of moose sickness (parelaphostrongylosis). The presence and abundance of *P. tenuis* in each WMU was determined by examining deer feces for dorsal-spined, nematode larvae.

METHODS

Test of hypothesis using historical data

Moose and deer population trends were identified from plots of density or harvest estimates vs. time, for each area. Hunter harvest data were used when population estimates were not consistently available. Density estimates were used directly from the available literature or converted from estimates of total population size, using published land areas. Where data were available from more than one source for a particular year, they were averaged.

For the purposes of further analyses and discussion, plotted harvest data for moose and deer in New Brunswick, Nova Scotia and Maine were converted to pre-harvest population estimates using the DeLury method (Fryxell *et al.* 1991, Roseberry & Woolf 1991). No modifications were made to account for non-hunting mortality. Following Fryxell *et al.* (1991), $N=(\text{harvest})/(1-e^{-HR})$, where N is the estimated autumn population size and HR is the instantaneous rate of harvest. Because rates of white-tailed deer harvest are related to density, harvest rates were set to 0.28 at peak deer harvests and 0.18 with low harvests. The instantaneous rate of harvest of the autumn moose population used here is 0.11 (Dodds 1963, Timmermann 1987, Boer 1991).

Moose and deer population estimates for Minnesota come from published papers and internal Department of Natural Resources reports (Manweiler 1941, Erickson *et al.* 1961, Indstrom 1965, Petraborg and Burcalow 1965, Karns 1967, Berg

1975, Peek *et al.* 1976, Karns 1982, Nelson and Mech 1986, Fuller 1987, Fuller 1989, Lenarz 1991, Lenarz 1992, Fuller *et al.* 1992) (see Appendix 1 for deer and moose density estimates by year and source). Some densities for northeastern Minnesota were calculated using a land area of 14,300 km^2 (Karns 1982).

In Maine, only harvest data were available for white-tailed deer while density estimates were available for moose (Banasiak 1961, Dunn 1966 in Gilbert 1974, Gilbert 1973, Gilbert 1974, Dunn and Morris 1981). The areas of northern and southern Maine used for density calculations were 32,745 km^2 (Dunn & Morris 1981) and 44,855 km^2 (Banasiak 1961) respectively.

Hunter harvest records were summarized for New Brunswick (Squires 1946, Huot *et al.* 1984, Boer 1991, New Brunswick Department of Natural Resources 1991). The provincial area used was 74,736 km^2 (NBDNR 1991).

In Nova Scotia, deer estimates were based on hunter harvest data over the entire province (53,600 km^2) (Benson & Dodds 1977, Brown 1983), as were moose, up to 1938 (Dodds 1963). Moose estimates after 1960, when legal hunting was reinstated, were for Antigonish, Cumberland, Guysborough, Pictou and Colchester counties (area = 16,030 km^2) (Benson & Dodds 1977).

A report of parelaphostrongylosis is defined here as a moose observed showing clinical signs of neurologic disease and/or one in which *P. tenuis* was found in the central nervous system at necropsy. Accounts of moose found dead without a history of neurologic disease and not examined, as well as reports of unidentified dorsal spined larvae in moose feces, were not included. Consecutive years in which

moose with parelaphostrongylosis were seen, in each of the 6 areas studied, were grouped and analyzed as a single reporting interval. A reporting rate (# cases/# of years with reported cases) was calculated for each interval.

Reporting rates were compared between intervals with and without moose population declines (minimum 45% change in population or harvest estimate over one or more years) using a Mann-Whitney U test. Chi-square 2x2 contingency tests and log-liklihood ratio for contingency tables were used to test for associations between moose declines and each of, reports of parelaphostrongylosis, and deer density (> or < 5/km^2). Polynomial regression analysis of deer and moose densities for northeastern Minnesota was performed (Zar 1984). Significance for all analyses was determined at $p<0.05$.

Survey of Ontario Wildlife Management Units

The managers of 83 WMU's were surveyed using data request forms (Appendix 2) and/or telephone interviews. Land areas of those WMU's studied ranged from 326 to 39,593 km^2 (Bisset 1991). Estimates of 1992 moose and deer densities were obtained for 54 of these WMU's. Moose densities were determined from standardized aerial inventories (Bisset 1991). Deer densities were obtained primarily from harvest data and approximations of population size made by experienced local managers. As in the first part of this study, harvest data were converted to density estimates using the DeLury method (Voigt 1991). Inherent limitations in these types of population data, and the existence of variation in

estimating cervid populations between jurisdictions using standardized guidelines have been discussed by Bisset and Rempel (1991) and Voigt (1991). Traditionally, reports of moose exhibiting clinical signs of parelaphostrongylosis have been collected opportunistically by Ontario Ministry of Natural Resources (OMNR) District Offices. These records, in addition to historical trend information on cervid population status, for each WMU during the period 1980-92, were provided by the responsible OMNR District and summarized from their completed questionnaires. Deer pellet groups (16-120 groups/WMU) were collected off snow in areas of winter deer aggregation by OMNR staff from January to March 1992. Three WMU's (8, 36 and 45) were re-sampled in the winter of 1993 and WMU 28 was sampled for the first and only time in 1993. Samples were kept frozen at -18°C for up to 4 months before examination using the Baermann technique. An average of 30g of pellets from each pellet group was suspended over tissue paper (Kimberly-Clark Kimwipes) in stoppered glass funnels (14.5 cm top diameter) filled with water. After 24 hours, 15 ml of water were drawn off each funnel and examined for dorsal-spined larvae using a dissecting microscope at 16X. All glassware were washed with soap and hot water and then rinsed in 95% ethanol to ensure clean glassware (see Appendix 3 for analysis of funnel cleaning protocol).

All dorsal-spined larvae found in feces of white-tailed deer were assumed to be those of *P. tenuis*. The presence of this parasite in deer in Ontario has been confirmed by previous authors (Anderson 1963, Lankester and Anderson 1968, E. M. Addison, personal communication, M. W. Lankester, unpublished data) and this study

(see Results - Survey of Ontario Wildlife Managaement Units). Another elaphostrongyline nematode (*Parelaphostrongylus andersoni*) with first-stage larvae indistinguishable from those of *P. tenuis* has been found in isolated bands of woodland caribou (*Rangifer tarandus caribou*) in Ontario (Lankester and Hauta 1989). This species also matures in white-tailed deer, but its known distribution in this host is spotty across North America. It is not known whether *P. andersoni* occurs in white-tailed deer in Ontario.

Dorsal-spined larvae from each deer pellet group were counted; counts of more than 200 larvae were estimated to the nearest 100. Prevalence (proportion of white-tailed deer infected) and mean intensity (larvae/gram of deer feces) were calculated for each collection area. However, in some instances, deer spend the summer in one WMU, but move to aggregation areas in different WMU's in the winter. In these cases, prevalence and mean intensity estimates for the WMU where deer spend the summer were the average of values for pellet groups collected in WMU's of winter deer use. For example, the deer found in the summer in WMU 53A, traditionally winter in WMU's 49, 50 and 53A. Therefore, prevalence and mean intensity corresponding to summer cervid densities for WMU 53A were the average of the results of pellet groups collected from WMU's 49, 50 and 53A.

Linear, polynomial and stepwise multiple regressions were used to test for relationships between moose density, white-tailed deer density, and prevalence and intensity of dorsal-spined larvae in winter deer feces. Prevalence values were arcsine transformed for all analyses (Zar 1984).

RESULTS

Test of hypothesis using historical data

Minnesota

In northeastern Minnesota there have been 4 moose population declines (D) in the past 80 years (Fig. 1). During this period, deer densities were estimated at 12/km^2 in the early 1930's, but since have ranged from 0.5 to 5.5/km^2. A total of 87 cases of parelaphostrongylosis in moose were reported during 3 time intervals (Thomas and Cahn 1932, Fenstermacher and Jellison 1933, Fenstermacher 1934 a&b, Fenstermacher 1937, Kurtz and Schlotthauer 1966, Peek *et al.* 1976, Karns 1977, Lankester unpubl. data). The reporting rate for each interval was 2.1, 3.8 and 1.8 sick moose/year (reporting rates correspond in chronological order to reporting intervals shown in Figs. 1-6).

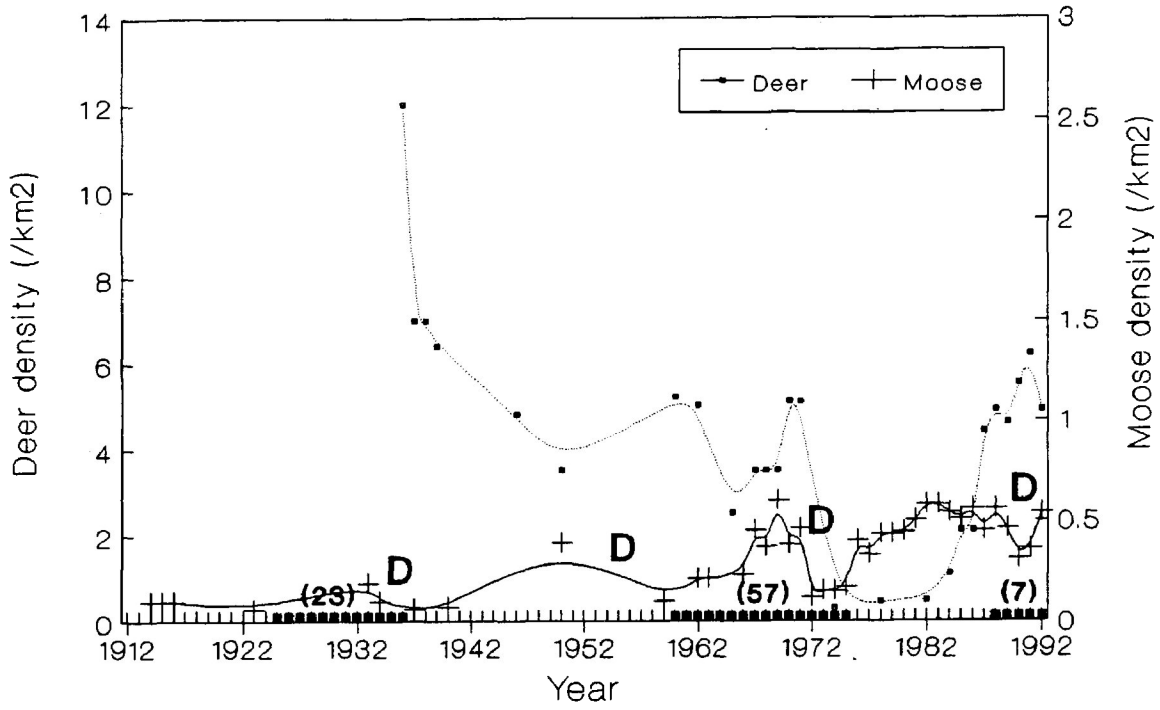
In northwestern Minnesota, 2 moose population declines were recorded, while deer densities rose gradually from 4.0/km^2 in 1935 to 7/km^2 in 1990 (Fig. 2). One reporting interval with 9 cases of moose sickness and a reporting rate of 1.5 cases/year was identified (Berg 1971, Berg 1975, Karns 1972).

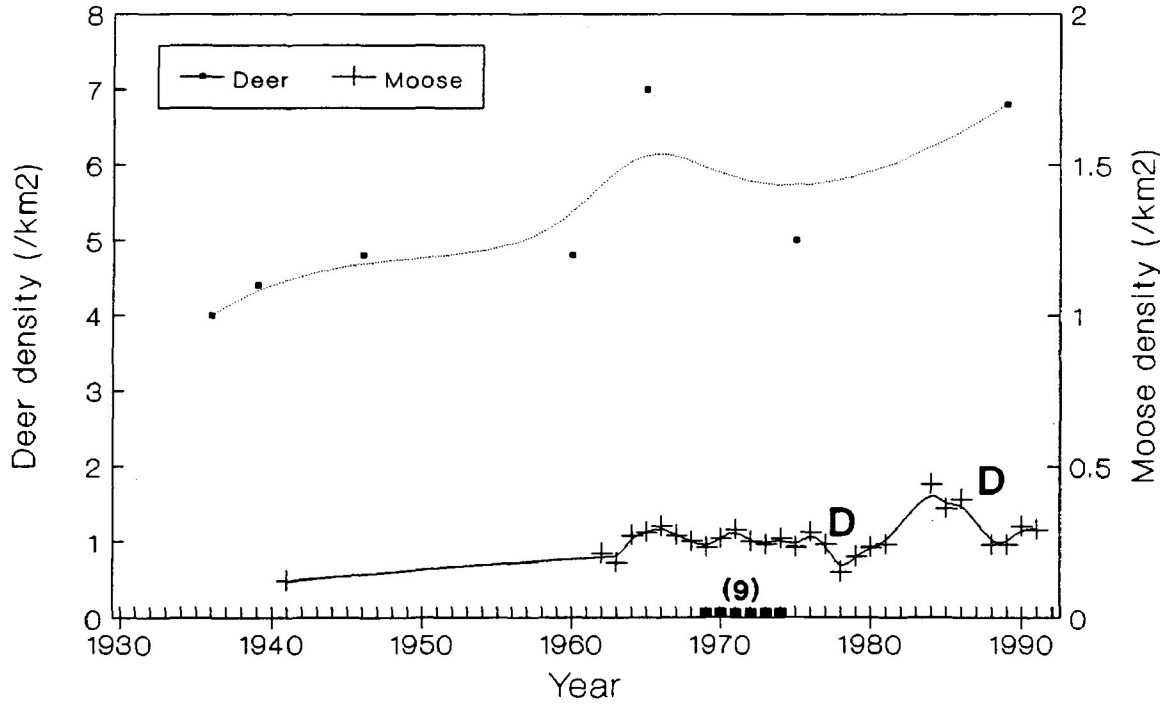
Maine

Historically, moose were common throughout Maine but were at low levels in the first quarter of the century. Since 1935 moose numbers have gradually

Fig. 1. Estimated densities of deer and moose in northeastern Minnesota, 1912-92, indicating intervals in which parelaphostrongylosis was reported (black bars), number of cases in each interval (bracketed numbers) and moose population declines (D).

Fig. 2. Estimated densities of deer and moose in northwestern Minnesota, 1930-92, indicating intervals in which parelaphostrongylosis was reported (black bars), number of cases in each interval (bracketed numbers) and moose population declines (D).


Deer
Moose
Deer density (/km2)
Moose density (/km2)
Year
D
D
D
D
(23)
(57)
(7)
0
2
4
6
8
10
12
14
0
0.5
1
1.5
2
2.5
3
1912
1922
1932
1942
1952
1962
1972
1982
1992


Deer
Moose
Deer density (/km2)
Moose density (/km2)
Year
D
D
D
(9)
0
1
2
3
4
5
6
7
8
0
0.5
1
1.5
2
1930
1940
1950
1960
1970
1980
1990

increased and there have been no reported declines (Figs. 3 & 4). Deer harvests in northern and southern Maine increased to 1960, with the peak values converting to 0.9 and 4.4 deer/km^2, respectively. In 1970, deer densities were estimated at 1.6/km^2 in the north and 3.1/km^2 in the south (Gilbert 1973). There were 2 reporting intervals of moose sickness in each part of the state, with a total of 12 cases in the north and 57 in the south (Lamson 1941, Behrend and Witter 1968, Gilbert 1974). Reporting rates were higher during the 2 intervals in the southern portion (4.0 and 4.6 sick moose/year) than those in the north (1.0 and 1.6 sick moose/year).

New Brunswick

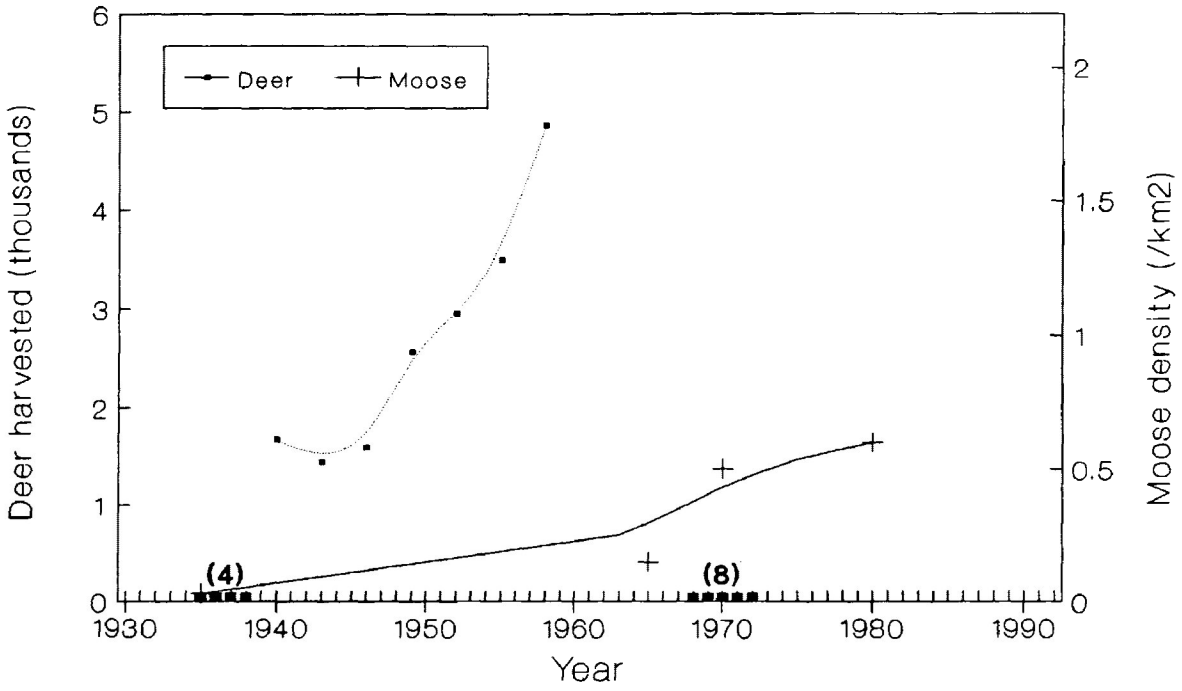
In New Brunswick, the only recorded decline in the provincial moose harvest occurred in the early 1930's, shortly before the hunting season was closed (Fig. 5). Provincial deer harvests peaked in 1960 and again in 1985, and convert to 2.2 and 2.3 deer/km^2, respectively. In the 2 intervals in which sick moose were seen, a total of 27 cases was recorded and reporting rates were 5.0 and 1.0 sick moose/year (Smith *et al.* 1964, Smith and Archibald 1967, Upshall *et al.* 1987, Boer 1988a).

Nova Scotia

In Nova Scotia, there have been 3 declines in the provincial moose harvest (Fig. 6). Deer harvests convert to 5.0, 2.6 and 5.9 deer/km^2 in the 3 reporting intervals. These intervals include a total of 137 reported cases, resulting in reporting rates of 8.5, 6.5 and 10.0 sick moose/year (Cameron 1949, Benson 1958, Smith *et al.*

Fig. 3. Reported harvests of deer and densities of moose in northern Maine, 1930-92, indicating intervals in which parelaphostrongylosis was reported (black bars) and the number of cases in each interval (bracketed numbers).

Fig. 4. Reported harvests of deer and densities of moose in southern Maine, 1930-92, indicating intervals in which parelaphostrongylosis was reported (black bars) and the number of cases in each interval (bracketed numbers).


Deer
Moose
Deer harvested (thousands)
Moose density (/km2)
Year
(4)
(8)
0
1
2
3
4
5
6
0
0.5
1
1.5
2
1930
1940
1950
1960
1970
1980
1990

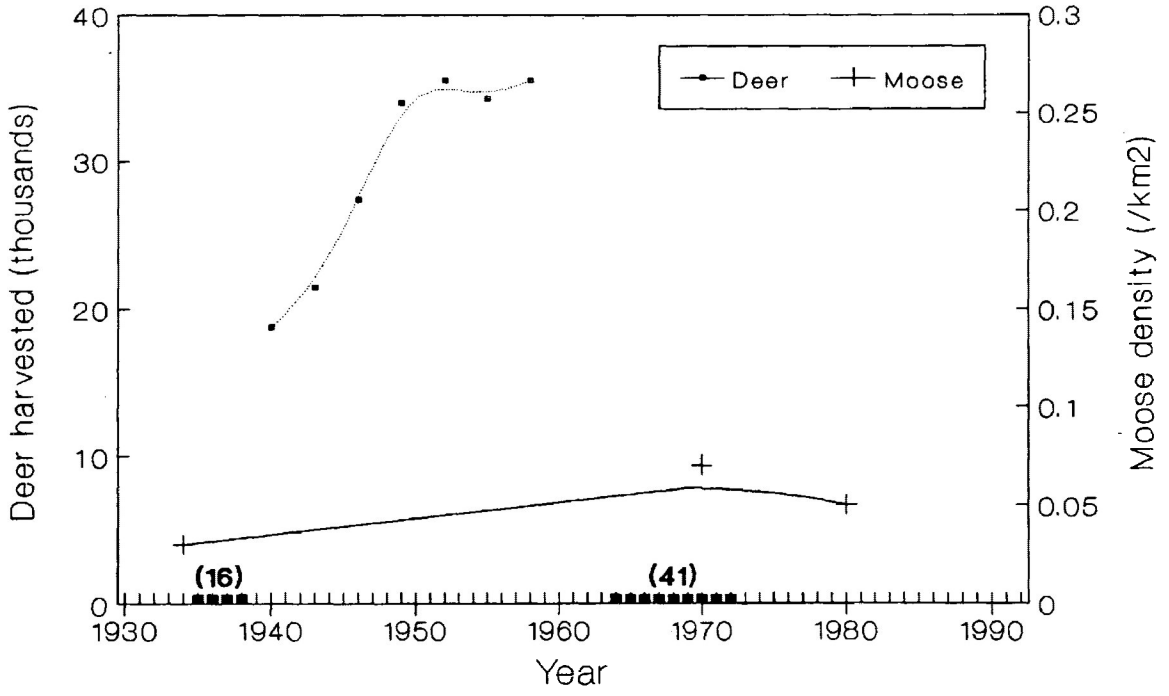
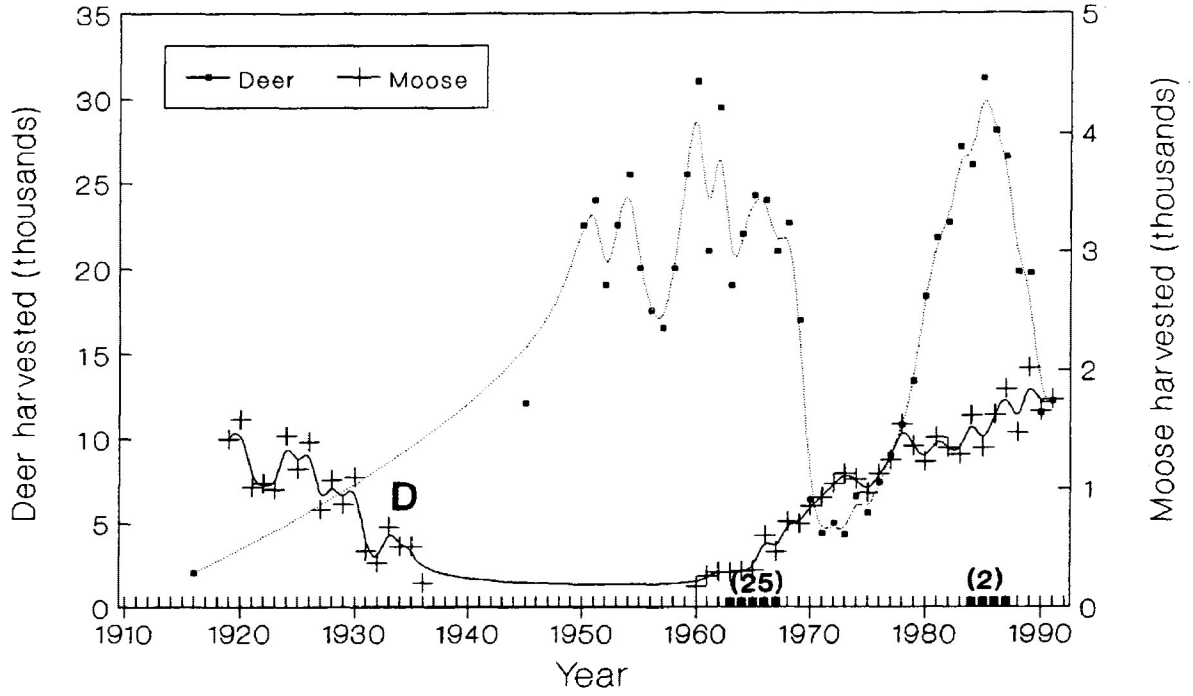
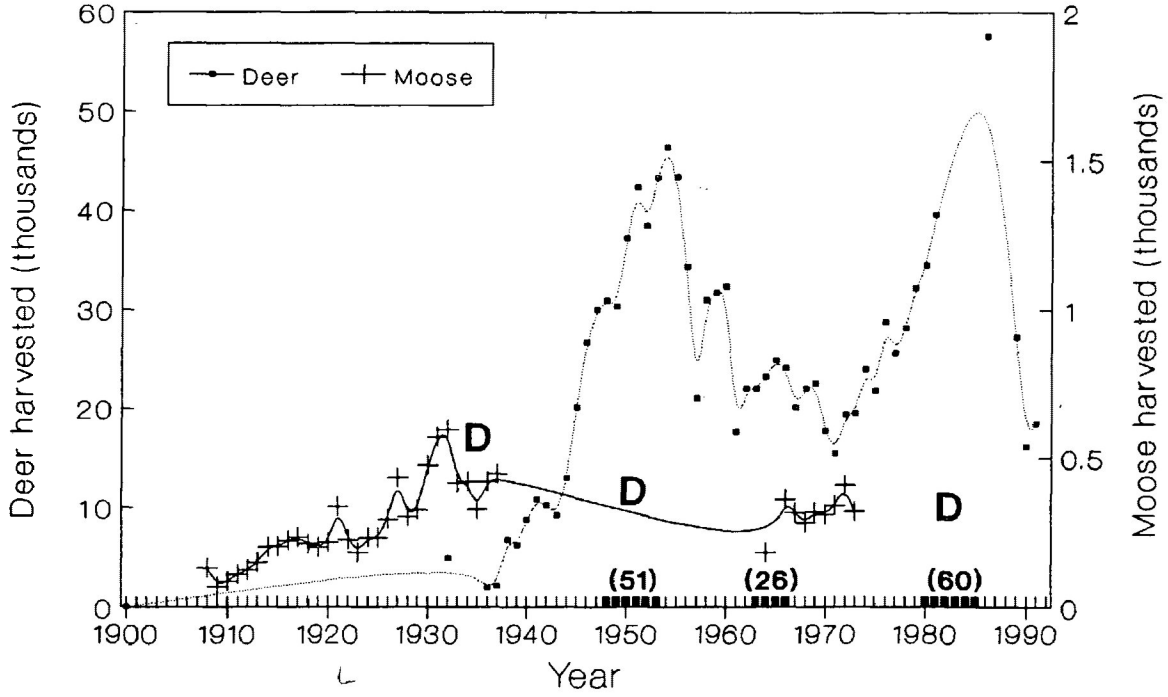

Deer
Moose
Deer harvested (thousands)
Moose density (/km2)
Year
(16)
(41)
0
10
20
30
40
0
0.05
0.1
0.15
0.2
0.25
0.3
1930
1940
1950
1960
1970
1980
1990

Fig. 5. Reported harvests of deer and moose in New Brunswick, 1910-1992, indicating intervals in which parelaphostrongylosis was reported (black bars), number of cases in each interval (bracketed numbers) and moose population declines (D).

Fig. 6. Reported harvests of deer and moose in Nova Scotia, 1900-92, indicating intervals in which parelaphostrongylosis was reported (black bars), number of cases in each interval (bracketed numbers) and moose population declines (D).


Deer
Moose
Deer harvested (thousands)
Moose harvested (thousands)
Year
D
(25)
(2)
0
5
10
15
20
25
30
35
0
1
2
3
4
5
1910
1920
1930
1940
1950
1960
1970
1980
1990


Deer
Moose
Deer harvested (thousands)
Moose harvested (thousands)
Year
D
D
D
(51)
(26)
(60)
0
10
20
30
40
50
60
0
0.5
1
1.5
2
1900
1910
1920
1930
1940
1950
1960
1970
1980
1990

1964, Smith and Archibald 1967, Kelsall and Prescott 1971, Brown 1983, Thomas and Dodds 1988).

Moose declines, parelaphostrongylosis and white-tailed deer densities

A total of 329 moose with parelaphostrongylosis was reported during 13 time intervals, (4 in Minnesota, 2 in New Brunswick, 3 in Nova Scotia, and 4 in Maine); there were 18 intervening intervals with no reports of the disease (Figs. 1-6). Moose population status (stable, increasing or declining) was independent of the occurrence of moose sickness ($X^2=0.1154$, $p=0.734$; $G=0.527$, d.f.$=1$; $n=32$) (Table 1). Reporting rates did not differ between the intervals when moose were declining and when they were not ($U_{5,8}=28$, $p=0.241$, $n=13$).

The historical data, do however, reveal a relationship between white-tailed deer densities and moose population declines. Moose declines occurred when deer densities were high (>5/km^2), under all circumstances ($X^2=4.58$, $p=0.032$; $G=7.037$, d.f.$=1$; $n=22$) (Table 2) and not only when intervals with reports of the disease were considered ($X^2=6.29$, $p=0.012$; $G=11.917$, d.f.$=1$; $n=13$) (Table 3). Polynomial regression analysis also reveals a significant relationship between deer and moose densities ($p=0.007$, $r^2=0.42$) (Fig. 7). Moose densities in northeastern Minnesota decreased to their lowest values when deer densities were greater than 5/km^2.

TABLE 1. Chi-square 2x2 contingency analysis of moose population status (decline or no decline) and the presence of reports of parelaphostrongylosis in moose

	Decline	No decline	Total
Reports	5	8	13
No reports	5	14	19
Total	10	22	32

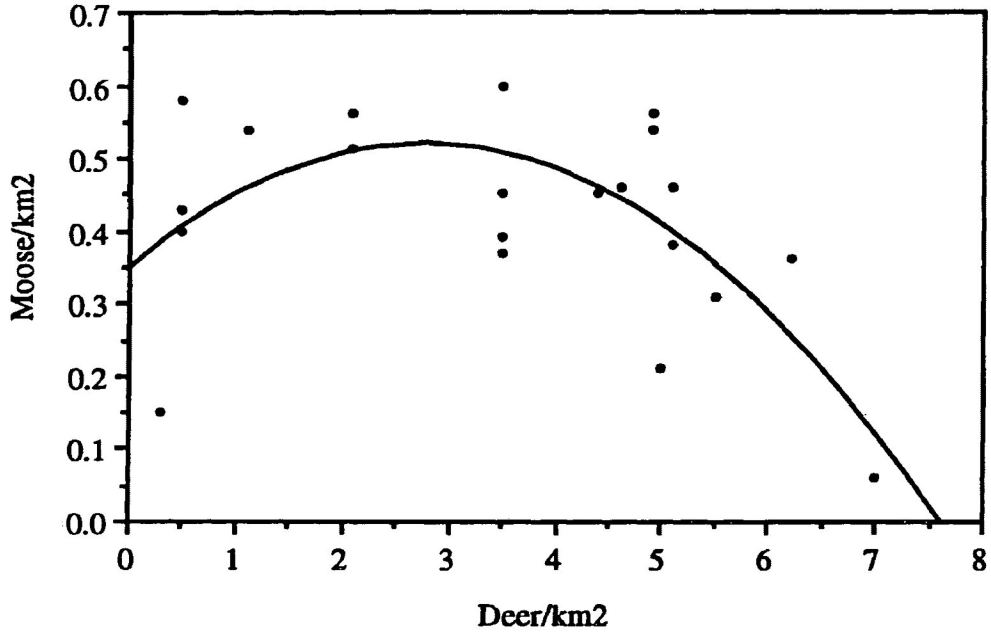
TABLE 2. Chi-square 2x2 contingency analysis of moose population status (decline or no decline) and density of white-tailed deer

	Decline	No decline	Total
Deer > 5/km2	8	3	11
Deer < 5/km2	2	9	11
Total	10	12	22

TABLE 3. Chi-square 2x2 contingency analysis of moose population status (decline or no decline) and density of white-tailed deer, for only those intervals in which parelaphostrongylosis in moose was reported

	Decline	No decline	Total
Deer > 5/km2	5	1	6
Deer < 5/km2	0	7	7
Total	5	8	13

Fig. 7. Polynomial regression analysis of densities of deer and moose in northeastern Minnesota, 1912-1992 ($p = 0.007$, $r^2 = 0.42$).


0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Moose/km2
0
1
2
3
4
5
6
7
8
Deer/km2

Survey of Ontario Wildlife Management Units

From the 83 WMU's studied, 23 reported only moose present, 15, only white-tailed deer and 45, both moose and deer (Fig. 8). In 55 WMU's where cervid densities were estimated, mean deer density in 1992 was 2.7 ± 0.3 deer/km^2 (range 0.01 to 10.0/km^2), and mean moose density, 0.27 ± 0.03/km^2 (0.05 to 1.10/km^2). The majority of WMU's in this study have regulated annual moose hunts. In a few, where moose numbers were above 0.5/km^2 (WMU's 7A, 51), there is limited or no hunting. In the WMU's where moose occurred alone, their densities averaged 0.16 ± 0.03/km^2, yet where they co-existed with deer, moose densities averaged 0.30 ± 0.03/km^2. Generally, however, in the areas where deer densities were high, moose numbers were low ($p=0.021$, $r^2=0.165$). Moose densities were greatest in areas where deer were less than 4/km^2 (Fig. 9). In most of the 45 WMU's where moose and deer occurred together, both species, reportedly, were distributed throughout the entire area. In some, moose or deer occurred in pockets of high density or of restricted distribution (Table 4).

All of the 45 WMU's reporting deer and moose present in 1992 indicated that both species had been present for the entire survey period (1980-1992). In 41 of these WMU's for which appropriate data were available, deer were reported to have been increasing in 34 and stable in 7 (east and west portions of WMU 51 considered separately) (Table 4). Moose were stable or increasing in 36 and declining in 5. During this period, 46 cases of parelaphostrongylosis were reported

Fig. 8. Map of Ontario, illustrating the distribution of white-tailed deer and moose, by Wildlife Management Unit (WMU). Note: in some WMU's distribution is concentrated in pockets, see Table 4.

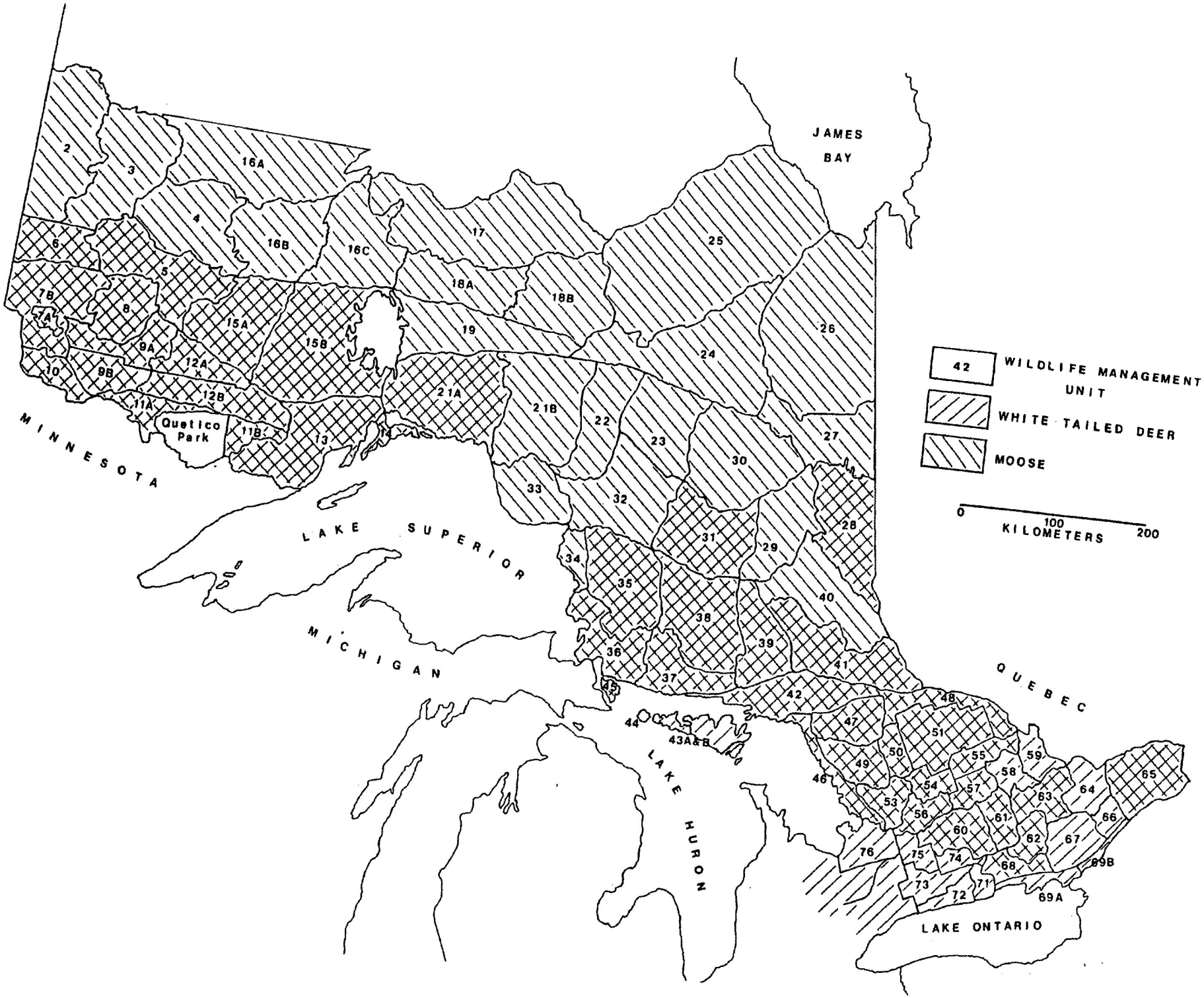

JAMES BAY
MINNESOTA
LAKE SUPERIOR
MICHIGAN
LAKE HURON
QUEBEC
LAKE ONTARIO
Quetico Park
42 WILDLIFE MANAGEMENT UNIT
WHITE TAILED DEER
MOOSE
0 100 200
KILOMETERS

Fig. 9. Polynomial regression analysis of densities of white-tailed deer and moose in Ontario, 1992 ($p=0.021$, $r^2=0.165$).

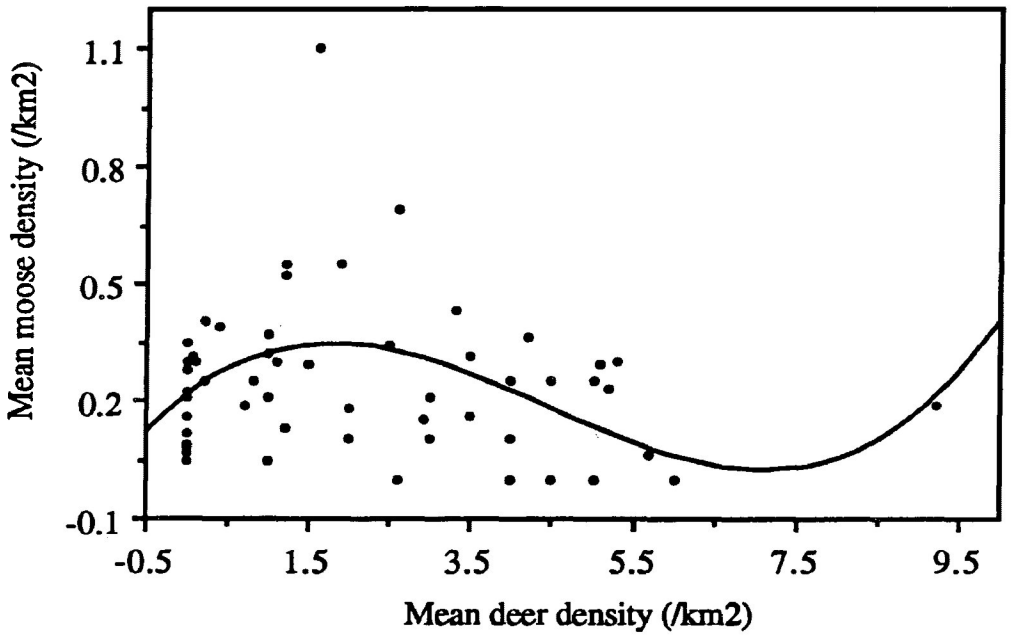

Mean moose density (/km2)
1.1
0.8
0.5
0.2
-0.1
-0.5
1.5
3.5
5.5
7.5
9.5
Mean deer density (/km2)

Table 4: White-tailed deer and moose population densities, prevalence and intensity of dorsal-spined larvae in deer feces and reports of moose sickness in Ontario.

	White-tailed deer					Moose			
	Density (/km2) (1992)		Population trend***	Dorsal-spined larvae		Density (/km2) (1992)		Population trend***	Reported cases of moose sickness
WMU††	Unitwide	Pocket	(1980-1992)	Prevalence (%)	Mean intensity (larvae/gr)	Unitwide	Pocket	(1980-1992)	(1980-1992)
5		4.5	+			0.21		+ / S	
6	0.2	10.0	+			0.29		+ / S	
7A	0.2	3.0	+			1.10		+	
7B	0.4	10.0*	+	74	15.6	0.23		+ / S	
8	1.5	5.0*	+	6	1.6	0.43		+	1
9A		5.3	+			0.30		+	
9B		3.5*	+	65	34.9	0.16		+	3
10	5.7*		+	65	34.9		0.06	S	14
11A	2.5*		+	53	10.8	0.34		+	1
11B	1.0		+			0.37		S	
12A	0.2		+			0.25		+	
12B	0.2		+			0.40		+	
13**	1.0*		+	42	8.4	0.21		- / S	7
14	0.2	2.0*	S / +	94	37.6	0.30		-	
15A		0.1	+			0.28		- / S	
15B	0.1		+			0.30		S	
28		0.4*	S	3	0.2	0.39		S	
31	0.01		S			0.16		S	
35	0.01		S			0.30		+ / S	
36		2.0*	+	38	13.4	0.10	0.26	+	
37	0.7*		+	84	30.6	0.19		S / -	9
38	0.06		+			0.31		S	
41		0.8	+	24	21.0	0.25		S	
45	9.2*		+	58	34.7	0.19		-	3
46	2.0*		+	71	25.2	0.10		+	
47	4.0*		+	77	28.8	0.10		S	
48	1.0*		+ / S	52	9.4	0.32		S	
49	4.2*		+	77 (±1)†	14.3 (±4.6)†	0.36		+	1
50	1.2*		+	62 (±15)†	8.0 (±3.9)†	0.20	0.83	S	
51 (E)	1.9*		S	75 (±4)†	12.9 (±4.4)†	0.55		S	
51 (W)	1.2*		S	62 (±7)†	14.1 (±3.0)†	0.55		S	
53A	2.9*		+	65 (±12)†	11.5 (±4.6)†	0.15		S	
54	1.5		+			0.29		+	
55B	3.0*		S	75 (±4)†	12.9 (±4.4)†	0.10		S	
56	1.2*		+	68 (±6)†	24.7 (±5.5)†	0.13		+	
57	3.5*		+	70 (±2)†	19.2 (±1.1)†	0.23	0.39	S	3
60A	2.6*		+	66	17.0		0.69	+	1
61	4.5*		+	66	17.0		0.25	S	3
62	4.0		+				0.25	S	
63	5.0		+				0.25	S	
68	1.0		+				0.05	S	

* White-tailed deer density associated with prevalence and intensity of infection.

** Sleeping Giant Provincial Park only.

*** Population reported as stable (S), increasing (+) or decreasing (-).

† Mean prevalence and intensity (±SE).

†† Includes only those Wildlife Management Units with both white-tailed deer and moose.

in 11 of the 41 WMU's, for a reporting rate of 3.8 sick moose/year.

A total of 1,027 white-tailed deer pellet groups was examined for dorsal-spined larvae, from 31 locations in 24 WMU's in Ontario. The mean prevalence of infection in all deer populations sampled was 57.5 ± 4.6% and the mean intensity was 17.2 ± 2.1 larvae/g. Interestingly, deer from WMU's 8 and 28 both had a much lower prevalence (≤ 6%) of dorsal-spined larvae in feces than did the rest of the deer populations sampled (Table 4). The deer population at WMU 28 is disjunct from the relatively contiguous distribution of deer observed in the rest of Ontario (Fig. 8). A pocket of deer in WMU 8 was similarly isolated until recently (last 2 years), when numbers in the rest of the WMU began to increase; deer are now distributed throughout the WMU at a low density as well (Table 4).

The presence of *P. tenuis* in the Kirkland Lake area (WMU 28) was confirmed by finding one adult female worm in the head of a hunter harvested white-tailed deer. A total of ten heads were examined, for a prevalence of 10%. Fecal samples were not available for these deer, however throat washes of each head did not reveal any dorsal-spined larvae.

Moose density was inversely related to intensity of dorsal-spined larvae in deer feces ($p=0.018$, $r^2=0.204$) (Fig. 10), but was not dependent on prevalence ($p=0.430$). Prevalence and intensity of dorsal-spined larvae were significantly correlated with each other ($r=0.604$, $n=25$, $p<0.05$) (Fig. 11), but were independent of deer density ($p=0.487$ and $p=0.208$ respectively). Stepwise linear multiple regression of the 3 independent variables (deer density, prevalence and intensity)

Fig. 10. Linear regression analysis of intensity of *Parelaphostrongylus tenuis* larvae in white-tailed deer feces and moose density in Ontario, 1992 ($p=0.018$, $r^2=0.204$).

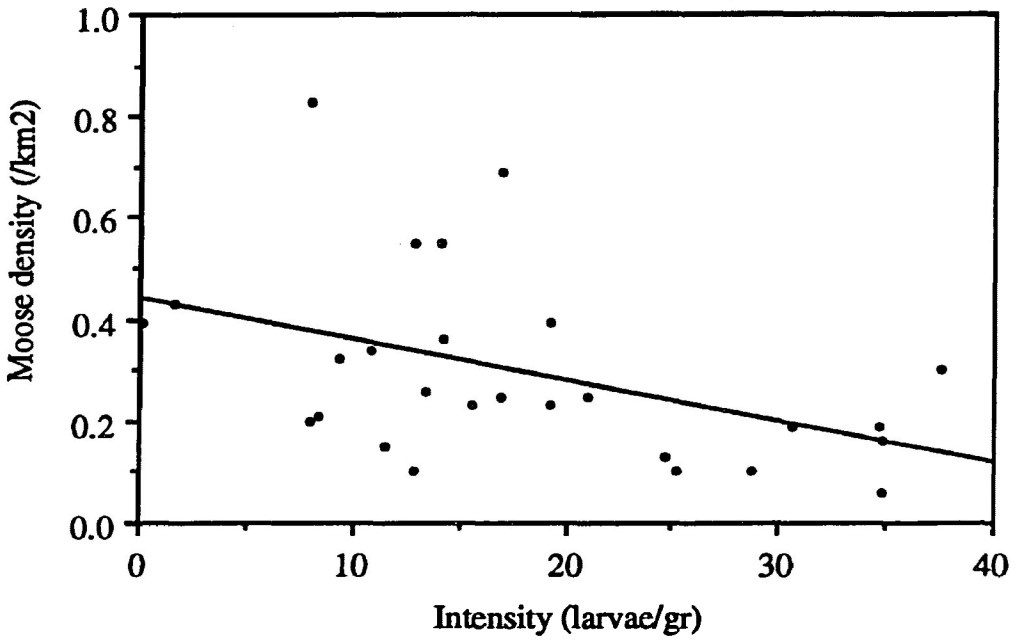
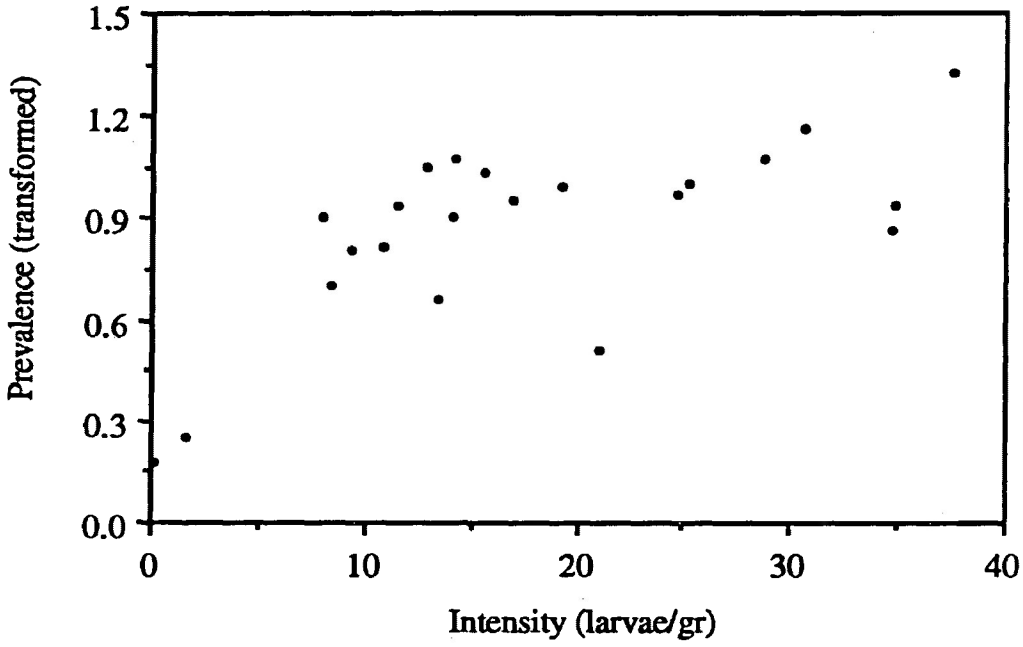

1.0
0.8
0.6
0.4
0.2
0.0
Moose density (/km2)
0
10
20
30
40
Intensity (larvae/gr)

Fig. 11. Correlation analysis of intensity and prevalence of *Parelaphostrongylus tenuis* larvae in white-tailed deer feces in Ontario, 1992 ($r=0.604$, $p<0.05$).


1.5
1.2
0.9
0.6
0.3
0.0
Prevalence (transformed)
0
10
20
30
40
Intensity (larvae/gr)

with the dependent variable (moose density) included only intensity in the significant equation ($F = 6.836$, $r^2 = 0.229$).

DISCUSSION

The two parts of this study, namely, an anlysis of available historical information and a survey of moose and white-tailed deer populations in Ontario, were conducted at quite different temporal and spatial scales. Eighty years of cervid population fluctuations and reports of sick moose from 6 jurisdictions across eastern North America were used to test the hypothesis that *P. tenuis* causes moose population declines. Whereas, changes in moose and white-tailed deer populations in Ontario over the period 1980-92 and current density estimates (1992), allowed an assessment of the parasite's potential role in present day deer-moose population dynamics.

To be able to causally link moose population declines with *P. tenuis* using historical data from the 6 areas examined, the declines should be associated with high white-tailed deer densities and with high reporting rates of sick moose. In fact, moose declines consistently occurred when deer densities were greater than 5/km^2, but even when the disease was not reported. The declines were also independent of the mere occurrence of the disease and of reporting rates. Therefore, the best test of causality that can be constructed using historical data does not support the research hypothesis that *P. tenuis* causes moose population declines. It should be noted, however, that there were 5 periods when higher than average deer densities, reports of sick moose, and moose declines were concurrent. Notwithstanding the lack of significance following contingency analyses, something that is inherently

difficult to attain with co-occurence data (Hastings 1987), *P. tenuis* might still have played a role in these 5 declines.

Whether the above predictions expected of the hypothesis are reasonable, and whether the historical data are of sufficient quality to test them, are valid questions. Firstly, increasing densities of deer may not neccessarily result in increased transmission to moose. It has been proposed that within deer populations, transmissibility is best reflected by the prevalence of *P. tenuis* (i.e. % infected) in the fawn cohort, an easily measured index of the parasite's ability to reach new hosts under prevailing conditions (Lankester 1987, Peterson and Lankester 1991). However, no consistent relationship exists between prevalence and deer density. Instead, prevalence, to a large extent, seems to be affected by changes in weather that alter the survival of first-stage larvae and the number and movement of terrestrial gastropods. No historical information is available on the prevalence of *P. tenuis* in deer where sick moose were reported.

Secondly, reporting rates of sick moose will be influenced by many disparate factors including road access in an area, awareness and compliance of local game officials, and the efficiency of predators in removing sick animals. Additionally, a problem with the broad scale of these historical data limits their interpretation. Moose and deer density estimates averaged over large areas obscure features of local habitats, such as altitude, discrete distribution, and habitat heterogeneity, that may limit direct overlap between the two cervids and reduce rates of transmission to moose (Telfer 1967, Kelsall & Prescott 1971, Kearney and Gilbert 1976), albeit that

the existence of such separating mechanisms is contentious (Nudds 1990, Gilbert 1992). Lastly, converting harvest numbers to density estimates potentially can reduce precision, yet converted data showed reasonable agreement with the few scattered population estimates available for Maine (Peterson 1955), Nova Scotia (Cameron 1949, Thomas and Dodds 1988, Patton 1988 & 1989, ASFWB 1992), and New Brunswick (Boer 1988a & b, 1991).

The inverse relationship confirmed between historical moose and deer densities may be of interest to cervid managers, but any role that *P. tenuis* might have played cannot be separated from other factors known to influence cervid numbers. These include changes in habitat and weather that favour one species over the other (Mech *et al.* 1987), hunting (Boer and Keppie 1988), predation (Mech and Karns 1978, Fuller 1989, Ballard 1992), other diseases such as winter tick (*Dermacentor albipictus*) (see McLaughlin and Addison 1986, Samuel 1991), as well as the question of whether moose and deer compete for space and resources (Telfer and Cairns 1986, Messier 1991, Pruss and Pekins 1992). It has also been suggested that moose populations are cyclic (Peterson *et al.* 1984, Karns 1987, Stewart and Gauthier 1988). Although, retrospectively, we cannot distinguish the individual role or mix of causes involved in moose population dynamics, the historical pattern of moose declines when deer are greater than 5/km^2 is noteworthy. The significant non-linear regression analysis of data for northeastern Minnesota also suggests that moose reach their greatest densities when deer are below 5/km^2. However, the r^2 value indicates that only 42% of the variation in moose densities is explained by deer numbers.

Interestly, Karns (1967) recommended that moose could be managed successfully if deer sharing the area were kept below 5/km^2.

Examination of the literature does not reveal any examples of catastrophic declines or extinctions of moose that can be attributed to *P. tenuis*. Instead, in all of the 6 areas examined, parelaphostrongylosis has been seen repeatedly over at least 80 years, yet moose persist, albeit at low densities. In addition to the areas considered here, moose sickness was reported in the southeastern portion of Manitoba in the early 70's by Lankester (1974) and a number of cases continue to be seen annually (Dr. V.J. Crichton, Man. DNR, pers. comm. 1992). Deer densities in the area have fluctuated widely yet moose still exist (approx. 0.03/km^2). On Anticosti Island in the St. Lawrence River, moose and deer were introduced simultaneously in the late 1890's (Newsom 1937) and almost 100 years later both species are still present; there are no predators (Bertrand 1983, Potvin *et al.* 1991). The meningeal worm is known to exist in deer (Beaulieu-Goudreault 1981) but moose sickness has never been reported, possibly because of the area's inaccessibility (Rau 1984). In these instances it is tempting to suggest that interspecific interactions (including the possible importance of *P. tenuis*) rather than habitat factors are limiting moose numbers (*sensu* Messier 1991) below densities usually seen where deer are absent (*cf.* Timmermann 1987). But removal of deer or their parasite would be the only way to prove it.

Historical data are unlikely to provide further insight into possible effects of *P. tenuis* on moose populations. Experimental field manipulations would be more

decisive although such critical tests may be highly impractical. Progress can nonetheless be made by developing a convenient method of detecting *P. tenuis* infections in moose. Confirmation of the cause of observed neurologic signs and detection of possible subclinical cases among living animals, hunter harvests, and road-kills is essential before the magnitude of this parasite's impact on moose can be understood. More precise knowledge of the susceptibility of moose to *P. tenuis* is also required. It has never been determined whether moose can survive low dosage infections as has been shown recently with wapiti (*Cervus canadensis*, see Samuel *et al.* 1992).

The general observations that moose and deer appear to co-exist in some areas of Ontario and that provincially, their numbers have been increasing since the 1980's provided the opportunity to examine the potential role of *P. tenuis* in present day deer-moose population dynamics. This provincial survey showed that white-tailed deer, moose and *P. tenuis* have existed together in 45 Wildlife Management Units in Ontario for at least the past 12 years. In 36 of 41 WMU's studied in detail, moose numbers were stable or increasing during this period. One of these (WMU 47) includes the Himsworth Game Preserve where deer and moose numbers have not changed appreciably since they were studied by Kearney and Gilbert (1976). And as previously discussed, the two cervids and the parasite have also co-existed in New Brunswick, Nova Scotia, Maine, Minnesota, and on Anticosti Island for nearly 80 years during which time both moose and deer fluctuated greatly, and moose with parelaphostrongylosis were regularly reported. Most importantly, however, in all of

these areas moose never disappeared and still persist to this day.

Deer in all WMU's in Ontario with moose, were at low to moderate densities. Most were well below 6/km^2 with the exception of a few high density pockets at 10/km^2 and a WMU-wide density of 9/km^2 in WMU 45. These moderate densities apparently are typical of present day, hunted, white-tail populations at the northern limits of their distribution where they encounter moose. They can be compared to much higher densities of 15-25/km^2 estimated in more southern areas (Halls 1984) and those that occurred in Minnesota as deer expanded northward into moose range following logging and easier winters in the late 1930's and early 1940's (this study). Results of this Ontario survey illustrate that deer and moose numbers were inversely related, and that moose reached their highest densities where deer were below 4/km^2. These findings support a similar relationship suggested initially by Karns (1967), and later documented with historical data (this study).

It is difficult to argue that *P. tenuis* might have a negative impact on moose numbers when we observed that moose reached higher mean densities in the WMU's they shared with deer, than where they were alone. But, in Ontario, the only areas where moose were allopatric were the more northerly, boreal zone units where winter severity and land capability probably limit their numbers (Timmermann and Whitlaw 1992). Moose would be less limited by these factors in the more southerly, mixed deciduous units where they occur with deer. Moose may be further advantaged when sympatric with deer by being the larger, less vulnerable prey in a multi ungulate-wolf system, as described by Bergerud (1990). Unfortunately, there

are few opportunities in the more temperate, mixed forest zone to study moose exclusive of deer, their parasite, and hunting, to determine what population densities are possible. The closest comparison might be Isle Royale where moose, in the absence of deer and hunting, have fluctuated between 1 and 2/km^2 (Peterson *et al.* 1984)

Even at the intermediate spatial scale investigated here, it was not possible to obtain sufficiently detailed information to reveal the extent to which co-existing deer and moose actually overlapped ranges. It was the opinion of most managers that the summer distributions of deer were fairly even throughout the areas occupied by moose, and in some units, small pockets of higher moose densities occurred in response to patches of habitat most suited to them. Such pockets could also provide a degree of refuge from *P. tenuis* infection in the sense proposed by Gilbert (1974), and thereby result in higher moose densities. Kearney and Gilbert (1976) studied moose and deer seasonal habitat use in a 1,200 ha Ontario forest and concluded that spatial overlap between the two species was reduced by habitat heterogeniety thereby reducing the risk of infection to moose. Nudds (1990) questioned the existence of such refugia, in part because none was totally free of infected deer throughout the year. However, any area with even a lowered rate of *P. tenuis* transmission could provide some advantage to moose resident there. The potential importance of winter deer yards as foci of transmission to moose is unknown.

We report here that moose densities were lowest in areas with the highest mean intensity of *P. tenuis* larvae in deer feces, suggesting that the numbers of larvae

passed by deer may reflect the impact of parelaphostrongylosis on co-habiting moose. In addition, mean intensity explained more of the variation in moose densities than did deer density, possibly allowing an initial separation of the roles of the disease and any interspecific competition that might exist. Saunders (1973) reported that the density of moose in northwestern Ontario was inversely related to the prevalence of *P. tenuis* in the feces of sympatric deer. Our results do not confirm this relationship. Prevalence of infection was proposed by Peterson and Lankester (1991) as an easily measured parameter that reflected the overall suitability of conditions influencing transmission of *P. tenuis* within deer populations. Findings reported here suggest that mean intensity may instead provide a better measure of the risk of disease in moose.

The relationship between intensity and rates of transmission of *P. tenuis* has not been investigated. While prevalence may primarily be dependent upon environmental conditions that determine the availability of gastropod intermediate hosts (Peterson and Lankester 1991), intensity reflects the productivity of adult worms already established in deer. Larval output is probably regulated by many factors, such as the immune response of the host, host age and any density-dependent mechanism at work in the infrapopulation (Peterson and Lankester 1991). Therefore, assuming that these factors are more constant than annual environmental changes, intensity would be a less variable measure of risk of disease in moose than prevalence.

In the present study, moose declined in only 5 WMU's where they existed with deer. In none was it the opinion of managers that *P. tenuis* might have been

primarily responsible and reporting rates of parelaphostrongylosis in all 5 WMU's were less than 1 suspected sick animal per year. Deer densities in 4 of these WMU's were equal to or less than 2/km^2. But in WMU 45 (St. Joseph's Island), where deer and moose share the same range year-round, deer were at 9.2/km^2. In spite of these high numbers it is difficult to implicate deer in the Island's present moose decline (0.44 moose/km^2 in 1985 to 0.19/km^2 in 1992). Rather, the decrease has been attributed to concurrent hunting pressure (30% of adults harvested in each of the first two years of the 1985-1990 harvest) and heavy infection with the moose winter tick, (*Dermacentor albipictus*) (Jones, pers comm 1993). Interestingly, in the early 1950's the unhunted moose were increasing in numbers in WMU 45, while white-tails, for which there was a sport harvest, were considered rare (Medwid 1957).

Moose have recently (1986-92) declined on the Black Bay Peninsula (WMU 14). Several factors may have contributed to the decline, including winter tick, improved hunter access, and predation. Deer have recently increased, although density estimates are generally still low, but they have an unusually high prevalence and mean intensity of *P. tenuis* (Timmermann and Whitlaw 1992, results of this study).

Moose have also declined markedly in Sleeping Giant Provincial Park, the peninsular portion of WMU 13 adjacent to the Black Bay Peninsula (Fig. 8). The research history of the Park, and the resulting large number of reports and publications, allow some insight into possible causes. Prior to the creation of the

Park in 1944 extensive horse logging had been conducted on the peninsula (Cuddy and Norman 1971). This created ideal new edge habitat and the numbers of both deer and moose subsequently increased in the absence of hunting (de Vos 1948, Anonymous 1971, Cuddy and Norman 1971). Deer were described as being stable or increasing slowly until the mid-60's, when a series of severe winters (1968-71) with greater than average snowfall reduced their numbers (Anonymous 1971).

By the early 1970's, an aerial inventory estimated the Park's moose population at approximately 0.8/km^2 (200 moose in the 243km^2 area) (Anonymous 1971). However, habitat quality was estimated to be declining and 90% of the standing timber was classified as mature or overmature (McNicol and Hamilton 1972). By the late 1970's it was estimated that the moose in the Park were as numerous as the food supply would allow and many of the moose were observed to be in poor condition (Fraser 1978, Nisbet 1981). In the early 1980's, a decline in the moose population to 0.52/km^2 was documented (McNicol *et al.* 1985) and by 1985, moose were estimated to have declined to 0.42/km^2 (McNicol *et al.* 1985). Best guess estimates of the 1992 moose population are that they have further declined to about 50 animals (0.21/km^2) (R. Gollat, personal communication 1993).

During the period of moose decline, 10 of the 13 winters between 1972 and 1984 had below average snowfall and increases in white-tailed deer were observed. By the early 1980's deer had become numerous enough that Fraser and Hristienko (1981) included observations of deer in their study of moose using mineral licks in the Park. Currently, deer are estimated at approximately 1.0/km^2 (R. Gollat,

personal communication) and are commonly sighted within the Park boundary.

In addition to changes in habitat and cervid numbers, records of predators and disease agents also exist for the Park. Timber wolf (*Canis lupus*) numbers increased slightly in the 1970's, as the deer increased, yet are currently stable and estimated at approximately 15 animals (Cuddy and Norman 1971, R. Gollat, personal communication 1993, G. Holburn, personal communication 1993). Three cases of moose sickness were reported in the Park in the 9-year period, 1962-71 (0.3 sick moose/year) (Anonymous 1971) and 7 cases were seen in the 12-year period, 1980-92 (0.6 sick moose per year). As well, moose with premature, spring hair-loss characteristic of infection with winter tick were commonly observed by Fraser (1978) in the late 1970's.

In conclusion, large scale historical data from eastern North America do not show a consistent relationship between moose declines and observable sick moose. However, an inverse relationship between moose and white-tailed deer numbers was evident through several cycles, and high deer densities, moose declines and the occurrence of sick moose coincided at least 5 times in the past. Study of current cervid populations in Ontario confirms the inverse relationship between the two cervids and, in addition, more directly implicates the parasite in deer-moose population dynamics, by showing that where the intensity of *P. tenuis* larvae in the feces of co-habiting white-tailed deer was high, moose numbers were low. However, case studies of moose declines suggest that the effect of this parasite on moose populations is more subtle than previously believed, and further study is required to

separate and measure its importance relative to other mortality factors known to act on moose populations.

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Appendix 1

White-tailed deer and moose density estimates by year and source for northeastern and northwestern Minnesota.

Table 1: White-tailed deer and moose density estimates and source by year for northeastern Minnesota.

Deer density (/km^2)	Reference(s)
12.0 (1936)	Erickson *et al* 1961, Karns 1967a
7.0 (1937)	Petraborg & Burcalow 1965
7.0 (1938)	Petraborg & Burcalow 1965
6.4 (1939)	Erickson *et al* 1961, Karns 1967a
4.8 (1946)	Erickson *et al* 1961, Fuller 1987
3.5 (1950)	Fuller 1987
5.2 (1960)	Erickson *et al* 1961
5.0 (1962)	Karns 1967a
2.5 (1965)	Karns 1967a
3.5 (1967)	Peek *et al* 1976, Fuller 1987
3.5 (1968)	Fuller 1987
3.5 (1969)	Fuller 1987
5.1 (1970)	Fuller 1987
5.1 (1971)	Fuller 1987
0.3 (1974)	Nelson & Mech 1986
0.5 (1978)	Nelson & Mech 1986, Fuller 1987
0.5 (1982)	Nelson & Mech 1986, Fuller 1987, Fuller 1989
1.1 (1984)	Nelson & Mech 1986, Fuller 1987, Fuller 1989, Lenarz 1991
2.1 (1985)	Fuller 1989, Lenarz 1991
2.1 (1986)	Fuller 1989, Lenarz 1991
4.4 (1987)	Lenarz 1991
4.9 (1988)	Lenarz 1991
4.6 (1989)	Lenarz 1991, Fuller *et al* 1992
5.5 (1990)	Lenarz 1991
6.2 (1991)	Lenarz 1991
4.9 (1992)	Lenarz 1991

Moose density (/km^2)	Reference(s)
0.10 (1914)	Indstrom 1965
0.10 (1915)	Peek *et al* 1976
0.10 (1916)	Indstrom 1965
0.06 (1923)	Peek *et al* 1976
0.19 (1933)	Peek *et al* 1976
0.10 (1934)	Karns 1967a
0.06 (1937)	Peek *et al* 1976
0.07 (1940)	Peek *et al* 1976
0.39 (1950)	Fuller 1987
0.10 (1959)	Peek *et al* 1976
0.21 (1962)	Karns 1982
0.21 (1963)	Karns 1982

0.23 (1966)	Karns 1982
0.45 (1967)	Peek *et al* 1976, Fuller 1987
0.37 (1968)	Karns 1982, Fuller 1987
0.60 (1969)	Fuller 1987
0.38 (1970)	Peek *et al* 1976, Karns 1982, Fuller 1987
0.46 (1971)	Karns 1982, Fuller 1987
0.12 (1972)	Karns 1982
0.15 (1973)	Karns 1982
0.15 (1974)	Karns 1982
0.17 (1975)	Karns 1982
0.40 (1976)	Karns 1982, Fuller 1987
0.33 (1977)	Karns 1982, Fuller 1987
0.43 (1978)	Karns 1982, Fuller 1987
0.43 (1979)	Karns 1982, Fuller 1987
0.44 (1980)	Karns 1982, Fuller 1987
0.50 (1981)	Karns 1982, Fuller 1987, Fuller 1989
0.58 (1982)	Fuller 1987, Fuller 1989
0.58 (1983)	Fuller 1987, Fuller 1989
0.54 (1984)	Fuller 1987, Fuller 1989, Lenarz 1992
0.51 (1985)	Fuller 1987, Fuller 1989, Lenarz 1992
0.56 (1986)	Fuller 1987, Fuller 1989, Lenarz 1992
0.45 (1987)	Lenarz 1992
0.56 (1988)	Lenarz 1992
0.46 (1989)	Fuller *et al* 1992, Lenarz 1992
0.31 (1990)	Lenarz 1992
0.36 (1991)	Lenarz 1992
0.54 (1992)	Lenarz 1992

Table 2: White-tailed deer and moose density estimates and source by year for northwestern Minnesota.

Deer density (/km^2)	Reference(s)
4.0 (1936)	Erickson *et al* 1961
4.4 (1939)	Erickson *et al* 1961
4.8 (1946)	Erickson *et al* 1961
4.8 (1960)	Erickson *et al* 1961
7.0 (1965)	Karns 1967
5.0 (1975)	Fuller 1987
6.8 (1989)	Fuller *et al* 1992

Moose density (/km^2)	Reference(s)
0.12 (1941)	Manweiler 1941
0.21 (1962)	Karns 1982
0.18 (1963)	Karns 1982
0.27 (1964)	Indstrom 1965, Karns 1982
0.28 (1965)	Karns 1982
0.30 (1966)	Karns 1982
0.27 (1967)	Karns 1982
0.25 (1968)	Karns 1982
0.23 (1969)	Karns 1982
0.26 (1970)	Berg 1975, Karns 1982
0.29 (1971)	Berg 1975, Karns 1982
0.25 (1972)	Berg 1975, Karns 1982, Fuller 1987
0.24 (1973)	Berg 1975, Karns 1982, Fuller 1987
0.26 (1974)	Berg 1975, Karns 1982, Fuller 1987
0.23 (1975)	Karns 1982, Fuller 1987
0.28 (1976)	Karns 1982, Fuller 1987
0.24 (1977)	Karns 1982, Fuller 1987
0.15 (1978)	Karns 1982
0.20 (1979)	Karns 1982
0.23 (1980)	Karns 1982
0.24 (1981)	Karns 1982
0.44 (1984)	Lenarz 1992
0.36 (1985)	Lenarz 1992
0.39 (1986)	Lenarz 1992
0.24 (1988)	Lenarz 1992
0.24 (1989)	Fuller *et al* 1992, Lenarz 1992
0.30 (1990)	Lenarz 1992
0.29 (1991)	Lenarz 1992

Appendix 2

Survey of Ontario Wildlife Mangement Units - Questionnaire

QUESTIONNAIRE - THE GEOGRAPHY OF MOOSE SICKNESS

Thank you for taking the time to complete this survey. I am a Biology MSc student, working for Dr. Murray Lankester at Lakehead University in Thunder Bay, Ont. The purpose of this project is to re-examine the importance of *Parelaphostrongylus tenuis*, the deer brainworm, to moose.

As you may be aware, *P. tenuis* is a nematode parasite that normally occurs without disease in white-tailed deer. In areas where moose and deer ranges overlap, *P. tenuis* becomes a debilitating and deadly pathogen, causing "moose sickness". The discovery of the cause of moose sickness was made by Anderson (1964) and early declines in moose numbers were attributed to the parasite. Moose sickness is still observed in many areas with both moose and deer, yet we are aware that moose populations may not be declining dramatically and moose and deer numbers may even be increasing in certain areas. This leads us to examine the particular set of circumstances that prevail in districts with both moose and deer, hoping to better understand the role played by this parasite.

We are asking for information (hard data and gut feelings) from local managers on what deer and moose populations have been doing over the past 10 years or so and the suspected reasons (ie. in relation to possible changes in hunting, logging, fire, predators, climate etc.). In compiling this information, we are looking to Ministry and resource management personnel most familiar with this kind of data. Knowledge on a broad scale for each management unit would be very useful, as would the specifics of smaller areas of special interest. **Please complete one questionnaire per management unit,** outlining cervid distributions, densities and cases of moose staggering sickness on the map provided. If necessary (ie. areas of interest, particular details etc.) please supplement this with your own more detailed maps (ie. FRI, topographical, WMU, district).

I would expect that information exists in the form of unpublished internal, and some published, reports, maps and recorded inventory data. I have provided lots of room around the WMU ' s map, thinking that the simplest way to convey most of the information is to make notes directly on the map. I would appreciate copies of, or references to, any relevant reports. Of course, all information used in the final paper will be given due credit.

Thank you once again for your interest in this area of research. Please contact me with any comments or questions you may have and feel free to circulate copies of this questionnaire and the accompanying map to those who may be more knowledgable in certain local areas.

Heather A. Whitlaw

The WILDLIFE MANAGEMENT UNIT for which this questionnaire is completed _____

PART A - MAP CHECKLIST

On the map(s) provided, please indicate the following:
(NOTE: When a formal and/or recent population survey is not available, please make your "best guess" and note the estimate as such.)

1. Estimated **deer** densities and their corresponding distributions - including both the most recent and historical estimates. You might outline an area on the accompanying map, NUMBER it and use that number in referring to the area on the Deer Density Summary Table.

2. Estimated **moose** densities and their corresponding distributions - including both the most recent and historical estimates. You might outline an area on the accompanying map, label it with a LETTER and use that letter in referring to the area on the Moose Density Summary Table.

3. Estimated **predator** densities and their corresponding distributions, if available. Indicate these areas with the NAME of the predator species and a number if necessary. These names can be used in referring to the area on the Predator Summary Table.

4. Deer yards/winter concentration areas

PART B - GENERAL QUESTIONS

5. In general, how have numbers of moose and deer changed in this WMU? What are the changes over time and where do you believe the populations are headed in the future?

6. Do you know to what extent *P. tenuis* might exist in deer populations in this WMU? Have pellets

ever been examined for larvae? What were the results? Would you be willing to help collect deer heads and feces in your area in order to learn more about infection rates? (Yes/No)

7. Please map any suspected cases of moose sickness. Include information such as the location, year and time of year reported, sex and age of the sick moose. In your opinion, is there anything that the reported cases of moose sickness may have in common with respect to the factors listed above? Which, if any, of these animals where specifically diagnosed with having *P. tenuis*?

In answering the following questions, we are interested in your thoughts on how these habitat factors might explain or have contributed to changes in deer and moose numbers. Please make use of the map provided in your discussions.

8. What is the hunting history of this unit (ie. changes in season length and area, controlled hunts, featured species)?

9. Generally, what is the forest management history of this unit? Do you believe that these activities or changes (ie. implementation of guidelines) have influenced population numbers?

10. What are the fire and insect histories of this unit? Are there any notable occurrences?

11. Are you aware of any long term climatic changes occurring in this unit (ie. snow course data and winter severity)?

12. Are there any areas of special interest that you could bring to my attention with respect to deer and moose densities and distribution, incidences of moose sickness, significant levels of *P. tenuis* or changing land use patterns, at present and/or historically?

If this study is of interest, and you and/or members of your staff would be willing to talk in more detail about moose and deer numbers, brainworm and moose sickness, please contact me at:

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P7B 5E1

Supervisor: Dr. M. Lankester
Fax (807) 343-8023
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Completed by:_____

District:_____

Tel:_____ Fax:_____

WILDLIFE MANAGEMENT UNIT: _____

DEER DENSITY SUMMARY TABLE

(outlined areas indicated by NUMBERS)

Outlined Area on Provided Map (NUMBERS)	Estimated Deer Density	Year of Survey	Survey Type	Comment

MOOSE DENSITY SUMMARY TABLE

(outlined areas indicated by LETTERS)

Outlined Area on Provided Map (LETTERS)	Estimated Moose Density	Year of Survey	Survey Type	Comment

PREDATOR SUMMARY TABLE

(outlined areas indicated by the species NAME and a number if necessary)

Outlined Area on Provided Map	Predator Species	Estimated Density	Survey Type	Comment

Appendix 3

A practical method for cleaning glassware used in the Baermann technique

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ABSTRACT: When using the Baermann technique to detect larvae of *Parelaphostrongylus tenuis* in deer feces, it is difficult to ensure that no larvae remain on glassware between samples. Of several cleaning methods tested here, emersion in 95% ethanol after flushing with hot or cold water was the most effective and practical.

The need to ensure clean glassware when conducting consecutive examinations of fecal material for nematode larvae using the Baermann technique (Samuel and Gray 1982, Beane and Hobbs 1983) was summarized by MCollough and Pollard (1993). Both the funnels supporting the fecal sample, and the watchglasses into which larvae are drained, must be reliably cleaned and not retain larvae from one sample to the next. McCollough and Pollard (1993) recommend that Baermann glassware be autoclaved prior to each use. However, this cleaning method is not feasible for institutions with limited equipment, nor in the field. Therefore, our objective was to develop an alternative, practical method, suitable for use under a variety of conditions.

The testing procedure was divided into two components. The first involved direct observation of the response of approximately 200 first-stage, *Parelaphostrongylus tenuis* larvae, in 5.5 cm diameter watchglasses, to various cleaning treatments, including 95% ethanol, Javex, Sparkleen dish soap (FisherBrand) mixed with 60°C tap water, and microwave radiation (Kenmore oven, Model 88922, 750W, high power). Nematode larvae used in the experiment were obtained from the feces

of white-tailed deer (*Odocoileus virginianus*) on St. Joseph's Island, in Lake Huron, where the deer are infected with *P. tenuis* (Addison, pers. comm.). Percent larval mortality after various exposure times in the watchglasses was determined by counting dead larvae, using a dissecting microscope at 16X. We determined that ethanol, undiluted Javex, Javex to water solutions of 1:1 and 1:2 and microwave radiation all caused 100% mortality of larvae in less than 5 minutes, whereas, soap and hot water, and a 25% solution of Javex in water did not (Table 1). Further use of Javex solutions was rejected, because despite rinsing, a residue of Javex remained on glassware that killed larvae in subsequent samples.

Secondly, we attempted to determine the efficiency of the various agents and treatments in cleaning glassware (glass funnels that were 14.5 cm in diameter with 3.5 cm of neoprene tubing on the stem, and watchglasses) that had previously contained fecal samples with greater than 15 larvae/gr. Routinely, the funnels each contained approximately 30g of deer feces suspended over porous tissue paper (Kimberly-Clark Kimwipes). After 24 hours, 15 ml were drawn off each sample into watchglasses and examined for larvae. Ninety-five sets of Baermann glassware were cleaned with soap and hot water, scrubbed with a sponge and bottle brush, vigorously rinsed in 95% ethanol for approximately 1 minute, then refilled with water and allowed to stand 24 hours before draining again. Approximately 5L of ethanol was kept in a tightly sealed plastic bucket, into which funnels were dipped. The same ethanol was re-used to rinse several series of funnels. None of the funnels treated in this manner retained live or dead larvae (Table 2). In addition, none of the 32

funnels rinsed in hot water and then in ethanol were contaminated with larvae. However, 2 of the 18 (11.1%) funnels treated only with hot water were contaminated. In addition, one third of the funnels rinsed with only cold water were contaminated (Table 2). Yet, when funnels rinsed only in cold water were followed with a 95% ethanol rinse (n=15), none retained larvae.

Conder and Williams (1983) initially showed that microwave irradiation is effective in killing helminth and protozoan parasites. Our initial tests indicated that it also kills *P. tenuis* larvae if exposed for at least 3 minutes. However, 1 of the 16 funnels (6.3%) that were exposed for only 1 minute to microwave radiation was contaminated with a live first-stage larva (Table 2), and dead larvae were found in a second. After microwaving another 16 funnels for a period of 2 minutes, 3 (18.7%) were contaminated with live larvae (Table 2), and an additional 5 funnels contained dead larvae. Interestingly, dead larvae were only recovered from microwaved funnels. Possibly, larvae that are killed by this method stick to the glass surface or tubing, and with re-filling, are later drained into watchglasses. Whereas, the rinsing action of the ethanol treatments apparently removes all larvae from glassware. In addition, the effects of microwave radiation on the rubber tubing of the funnels prevented us from increasing the time of exposure to ensure death of all larvae. Even after exposure of only 1 minute, the rubber tubing scorched and hardened where it touched surfaces within the oven. Also, the rubber tubing became tacky and the sides stuck together. This prevented the proper and easy drainage of funnels.

The Baermann technique is a widely used to detect dorsal-spined nematode

larvae in feces. When searching for evidence of patent *P. tenuis* infections in hosts other than white-tailed deer the numbers of larvae per sample are likely to be very low (Clark and Bowyer 1986, Lankester 1987, Welch *et al.* 1991) and it is important that the funnels not retain live larvae from one sample to the next. A reliable cleaning method is especially important when examining consecutive fecal samples from white-tails and from other, alternate, cervid hosts. McCollough and Pollard (1993) have demonstrated that larvae will not be retained on autoclaved glassware. However, we have determined that, as a minimum, the same result can be obtained by merely rinsing glassware in cold water, with a subsequent vigourous rinse in 95% ethanol.

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Table 1: Mortality of P. tenuis larvae in various cleaning solutions and following microwave treatment.

Cleaner	Treatment time	% mortality
95% ethanol	2 min	100
Hot water and soap	5 min	0
Javex	30 sec	100
Javex:water - 1:1	45 sec	100
Javex:water - 1:2	3.5 min	100
Javex:water - 1:3	5 min	20
Microwave radiation	15 sec	0
Microwave radiation	1.5 min	80
Microwave radiation	3 min	100

Table 2: Efficacy of cleaning Baermann funnels that contain P. tenuis larvae.

No. of positive funnels examined	Cleaning method	No. of cleaned funnels with live dorsal-spined larvae	No. of live larvae recovered per funnel
95	Hot water, soap & ethanol	0	0
32	Hot water & ethanol	0	0
15	Cold water & ethanol	0	0
18	Hot water only	2	1 to 3
15	Cold water only	5	1
16	Microwave radiation - 1 min	1	1
16	Microwave radiation - 2 min	3	1 to 4