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Source: Journal of Wildlife Diseases, 9(4) : 376-378

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-9.4.376>

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CARIBOU MORTALITY DUE TO THE MENINGEAL WORM (*Parelapostromylus tenuis*)

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Abstract: Fourteen woodland caribou (*Rangifer tarandus terraenovae*) were released into a game reserve occupied by white-tailed deer (*Odocoiles virginianus*) with a high prevalence of the meningeal worm (*Parelapostromylus tenuis*). Within 6 months all of the caribou had succumbed to this parasite. The clinical, pathological, and epidemiological findings associated with this die-off are described.

The role and significance of the meningeal worm in the biology of native cervids of North America has received special attention in recent years and an excellent review of the subject was presented in 1972 by Anderson.¹

The current status of knowledge regarding the significance of the meningeal worm to the woodland caribou is limited to experimental studies. Based on laboratory findings this parasite is highly pathogenic to caribou.² It has also been documented that reindeer (*Rangifer tarandus tarandus*) are highly susceptible to the parasite and cannot be introduced into an area with meningeal worms.²

This is a report concerning the catastrophic introduction of woodland caribou onto a range occupied by a dense population of white-tail deer with a high prevalence of the meningeal worm.

CASE REPORT

During a 5-year period, a commercial game reserve in Wisconsin successfully raised woodland caribou. The animals were maintained in holding pens (ca. 15 x 30 m) and fed artificially. Caribou raised in this manner appeared to be in good physical condition and reproduced successfully. The caribou propagation procedure provided isolation from other wild ruminants of the reserve, including white-tailed deer.

In an attempt to provide a more "wild" environment, the caribou herd of 14 animals was released in mid-June into a 2640 hectare fenced area. This fenced compound contained approximately 600 white-tailed deer and 250 elk (*Cervus canadensis*).

During late August and September, half of the 14 caribou died and were necropsied. All but one of the seven animals were adults ranging in age from 3-6 years and all had been on the premises from 1 to 5 years. Each caribou was ill prior to death and all had signs characteristic of neurologic disease.

Typically, these caribou had lumbar weakness and locomotor abnormalities, including posterior ataxia. Circling was reported in many of the caribou. Severe torticollis existed in several instances and bulging eyes were detected in all sick animals. Adults characteristically were in good flesh and continued to eat until moribund. In terminal stages of the disease, posterior paralysis developed and the animals usually died within 2 to 4 days.

A typical case history involved a 3 year old female caribou reported sick on August 23. She was prostrate, paralyzed, and ataxic, had a body temperature of 40.3°C, labored breathing, bulging eyes, and extreme torticollis. The animal had been observed in this condition for 48 hours. The caribou died during the examination and a necropsy was performed.

The animal was in good flesh; its liver was infected with liver flukes (*Fascioloides magna*); pneumonia and heavy infections of lungworms (*Dictyocaulus viviparus*) were detected in the lungs; and large numbers of stomach worms, including *Ostertagia* spp. and *Hemonchus contortus*, were found in the abomasum. The brain was examined and several areas of petechial hemorrhage were noted; two adult meningeal worms were detected in the neural parenchyma of the cerebellum. The spinal chord was surrounded by hemorrhagic gelatinous fluid.

Histologically there was rarefaction and degeneration of the white matter. Vascular cuffing with eosinophils and lymphocytes was evident, especially in the white matter. The leptomeninges and the dura mater were infiltrated with eosinophils and lymphocytes. There was degeneration in the brain stem.

Another caribou which died during this outbreak was examined at the Wisconsin Department of Agriculture Regional Laboratory. It was reported that the cause of death was due to a nematode larvae identified as *Pneumoststrongylus tenius*. Parasites were detected in the spinal cord parenchyma and within meningeal vessels; there was rarefaction of the white matter and two small foci of gliosis and inflammatory cells.

Hematology was done on two sick animals. The white blood cell counts exceeded 15,400 and high eosinophil counts (10%) were recorded.

Results of bacterial and toxicological studies, including *Listeria* cultures, were all negative. Virus isolation attempts resulted in an isolation of a Bynyamwera group arbovirus,⁴ but based on the serologic and disease history of the herd and the geographic area, as well as the timing and duration of the die-off, it was not considered important in the caribou losses.

By January, all of the caribou in this herd were dead. Although none of these

later carcasses was examined in the laboratory, reported signs were similar to those recorded previously and it is suspected that these animals, including five young of the year, also succumbed to meningeal worm infection.

DISCUSSION

The reported susceptibility of caribou and reindeer to the meningeal worm, the fact that meningeal worms existed in deer on the reserve (50% prevalence), the history of the herd prior to and after release, the appropriate incubation period for the meningeal worm to develop following caribou release and exposure, the fact that adults were affected first and young only after nursing ceased, and the necropsy results, all suggest that the cause of mortality and extermination of this caribou herd was the meningeal worm.

It is appreciated that only 7 of the 14 dead animals were examined in the laboratory and the meningeal worm confirmed as the cause of death; however, consistent field and gross observations suggest that a similar and probably identical disease syndrome was involved in all of the losses.

It is of interest that several attempts to introduce moose (*Alces alces*) into the reserve resulted in failure. The role of the meningeal worm in these failures was not established, but gross observations and reports of the moose behaviour and losses suggest involvement of this parasite.

Obviously this was not a natural environment or setting for caribou; however, the outbreak illustrates the importance of diseases and disease control as essential game management tools. It further suggests that the integration of caribou into white-tailed deer range (with meningeal worm infections) is not feasible and provides additional evidence for the suggestions of Anderson¹ that the caribou decline in its southern range was due to this parasite.

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Received for publication 18 June 1973
