THE RUB POST

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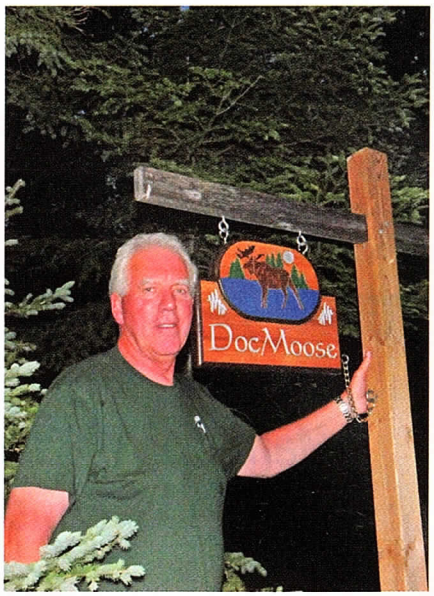

Brain worm! What is it? Can we consume venison from infected deer? All great questions that I have been asked over the years and for those who have not heard my presentation on diseases entitled "The Good, The Bad and The Ugly", below is a summary of the biology of this parasite.

This parasite is a nematode or roundworm and is common in white-tailed deer throughout the deciduous mixed wood forests of eastern Canada. In 1972, it was reported as far as western Manitoba. When doing my doctorate thesis at the University of Guelph, I was most fortunate to have the late Dr. Roy Anderson on my research committee. It was Roy who first described this parasite as the causative agent of "moose sickness" in moose from NW Ontario. The parasite is referred to by most as brain worm or meningeal worm. The latter name refers to the location of adult worms on/in the meninges, membranes that cover the brain and spinal column. Thus began my education about brain worm.

Being a nematode, there are male and female worms. Adults reside in the cranium of white tailed deer, and females deposit eggs into small blood vessels which travel the circulatory system until they reach the lungs. After hatching, larvae enter the air passages, eventually coughed up and passed to the exterior in faeces – they are in the mucous surrounding the outside of deer pellets. Snails and slugs (terrestrial gastropods) in spring and summer crawl over the surface of pellets, enabling the larvae to penetrate into the snail or slug, and in a short period of time, develop to what we call the infective stage. Feeding deer ingest these gastropods which are on vegetation, and once in the stomach, the snail is digested. However, the larvae escape and penetrate the stomach wall and migrate via the nerves in the rib area to the spinal column and brain. For deer no problem! However, when these gastropods are eaten by moose or caribou, which are nontypical hosts, problems ensue for the infected animal. Once they reach the spinal column, they enter tissue and often cause severe damage to the spinal column and other nerve tissues (e.g. nerves to the eye). The damage often results in moose circling, blindness, and eventually death will follow. However, some moose will overcome the infection and survive. I have found this parasite in Manitoba's moose, deer and elk and on one occasion, in domestic goats in the Pansy area of SE Manitoba.

As there are male and female worms, if an animal only acquires a single worm of either sex, obviously larvae will not be produced, but the host animal may develop an immunity to future infections - this is likely what has happened in cases where a single worm is found in the aforementioned animals.

Dr. Bill Samuel from the University of Alberta and I had a student review the distribution of this parasite in deer in Manitoba in the late 1980s. Results showed that 60% + of the deer examined in Game Hunting Areas (GHA) 35 and 36 were positive and 40-60% of those in GHA's 19, 14 and 12 were positive. In contrast, in southwestern GHAs only 0-20% was positive. The lower prevalence of the parasite in the southwest is attributed to the lack of suitable habitat for gastropods which are essential to complete the life cycle – the drier prairie areas are not suitable gastropod habitat which accounts for the lower prevalence. In GHAs to the north of Winnipeg, the prevalence varied from 20-40% and the prevalence in GHAs further north in the Interlake was greater than 0 but less than 20%. In summary, our student found the highest prevalence in to be in the southeast and decreasing with distance northwest, west and southwest.

Higher temperatures in fall, good precipitation, higher winter and spring temperatures, high deer densities and moderately forested areas make for high prevalence of this parasite in deer. As a point of interest, virtually every deer in the City of Winnipeg is infected.

High densities of infected deer and gastropods enhance transmission of the parasite. Forest cover also plays a significant role in moderating microclimate near the forest floor and hence habitat and movement of terrestrial gastropods. The latter require moisture as do the larvae passed by deer. Gastropods become relatively inactive when moisture is low and when deer faeces and surface soils become dry, there is increased mortality of the larvae and transmission is severely restricted. The positive correlation with the presence of brain worm and precipitation suggests that lower rainfall and lack of gastropod habitat, as seen in Canada's prairies, makes this region a natural ecological barrier for the natural westward spread of the parasite. To prevent it from accidentally moving west via movement of game ranched animals, Alberta enacted legislation years ago making it illegal to import deer or elk (some elk can act like deer and be carriers of the parasite, pass larvae and not show any symptoms of the disease) to Alberta from east of the Manitoba/Saskatchewan border.

When the student did his study, heads from hunter harvested deer were collected and examined (Note: this study would not have been possible without the co-operation of Manitoba's deer hunters). As this sample included fawns, it resulted in the prevalence being lower than it actually is, as worms in fawns taken during the fall deer hunting season have not yet migrated to the cranium where they are readily visible. When the Manitoba study was done in the late 1980s, the prevalence was 60+% and involved only heads collected during the hunting season. Including fawns in the sample will lower the apparent prevalence of the parasite. The issue is that juvenile deer (young-of- the-year) do not have patent infections (adult worms passing larvae) as the worms have not yet matured, thus being able to find them in the nervous tissue versus the cranial cavity is impossible. Any future study on prevalence from hunter killed animals must involve only heads from adults which will give a more accurate evaluation of prevalence.

It is the professional opinion of myself and others who have studied this parasite that the prevalence of brain worm in eastern Manitoba has not decreased over the last 25 or so years. It has been suggested that brain worm evolved in whitetails in the southern U.S. and when they arrived back in most of Manitoba about the turn of the 20th century, the parasite came with them. Another reason to expect the continued presence of the parasite in Manitoba is that brain worm is long-lived in deer. Thus, annual fluctuations in climate do not impact the prevalence of this parasite in the deer population as a whole. Once infected, a deer remains infected for life. The earliest officer reports that I can find describing sick moose with symptoms suggestive of brain worm are from 1949 east of Winnipeg. At that time, no one knew what was causing this anomaly.

Those familiar with the parasite contend that climate/weather will exacerbate the impact of brain worm on moose. For example, warmer winters may see less snow and generally shorter winters. This means less mortality of deer fawns and increasing deer numbers in areas previously inhabited mainly by moose (or caribou). Another important aspect is the length and wetness of the green season. For example, a "summer" like 2009 in Manitoba was excellent for gastropod survival, reproduction and mobility as well as for survival of brain worm larvae in deer faeces; all are significant factors enhancing transmission to deer fawns, moose and caribou. The bottom line is that easy winters result in increased deer numbers in moose habitat (which we have in the east) while wet summers enhance transmission. These are the two most important factors affecting the impact of this parasite on moose (easy winter for deer, wet summer for transmission and snail survival). The over-all result is the cumulative effect of both. It must be noted that new born moose and caribou are more susceptible to acquiring the infection than adults, thus the impact of this parasite on their survivability can be significant which impacts herd productivity.

To fully understand this parasite, it should be pointed out that young deer pass larvae at 3X the rate seen in older adults, and animals of all ages pass the greatest number in the spring. Research in northern Minnesota in spring found that 10-month-old fawns passed twice as many larvae as older animals. Deer pellets collected randomly in late winter had from 80 to 1,374 brain worm larvae each (mean = 439)! If in winter, a deer produces at least 50 pellets per sample and may defecate 15x per 24 hour period, the mean number of larvae released per day per deer is 329, well over a quarter of a million per day. The number passed by a fawn could approach one million larvae per day. With a high number of deer and many females, this leads to bumper fawn crops which in turn results in many young deer passing elevated numbers of larvae, which in turn leads to greater risk for species such as moose and caribou.

Approximately 70% of larvae in deer pellets survive freezing at -20C for up to 6 months. Only by examining deer at the right time of year can the true level of infection with brain worm be determined. For example, by examining both heads and pellets, mostly in mid- to late winter, researchers in northern Minnesota found 91% of yearlings and 96% of older deer to be infected.

The advantage of collecting pellets in late winter is that fawns that became infected the previous summer and fall will now be passing larvae. Plus, the larvae freeze as soon as the pellets are expelled and larvae cannot leave the pellets. They survive freezing and can be detected alive when pellets are later thawed. The ideal time to collect pellets is in March/April when most fawns have developed patent infections (eggs being passed by females and subsequently larvae in faeces). Pellet samples must be collected off snow to avoid contamination by free living soil nematodes. Larvae are only found in the mucous surrounding each pellet.

The high prevalence in deer is interesting in light of the fact the prevalence of infective larvae in gastropods has been shown to only be about 2-3%.

Recent studies have shown that moose exposed to low dose infections can recover but in the early stages, show some signs of infection. Studies suggest that the parasite is not necessarily lethal to moose but rather the outcome is dose dependent. It likely has a greater range of effects on moose than previously suspected with some signs being difficult to detect.

Will moose ever recover in SE Manitoba? The prognosis is not good as long as there are deer present carrying brain worm - do not bet your bank account that they will recover! While I was with Manitoba Conservation and Water Stewardship, we initiated more liberal hunting seasons for deer in GHA 26, which at one time had a substantial moose population. Such liberal deer seasons must continue in an effort to keep deer in this GHA at low densities to assist in recovery of moose to sustainable levels. The parasite likely contributed to some of the moose declines reported in eastern North America in the first half of the last century, but whether it was a major factor in the declines is debatable. The bottom line is it can kill moose and several other cervids native to North America, and it is hoped that ongoing research can clarify some of the relationships.

And, in answer to the question about the edibility of venison from infected deer – it's not a problem!

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