WHITE TAILED DEER

AND

BRAINWORM IN MANITOBA

(A Summary - October 2009)

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Based on available evidence, in contemporary times, white tailed appeared in Manitoba in the late 1800's/early 1900's and have dispersed across a significant part of the southern portion of the province. Albeit in lower numbers, deer also spread to more northern locations. The late John Kjartanson shot such an animal on Hecla Island in 1922 and did not know what it was. Geiri Johnson (pers. comm.) has advised that when he trapped along the Berens and Pigeon Rivers in the 30's he saw white-tailed deer at these sites. In more recent time they have been seen east of Lake Winnipeg at such places as Sasaginnigak lake in the 70's and citizens of Little Grand Rapids have noted increasing number of deer in recent years (Steve Taylor pers. comm.). Deer have also been seen at the north end of South Indian Lake in the 80's and in 1997 on Highrock Lake. Thus, based on the aforementioned, white-tails are widely distributed across Manitoba but with decreasing densities as one heads north in the boreal zone.

A major natural mortality factor facing deer is severe winters with some years such as in the mid 70's when deer died by the hundreds across southern Manitoba. This resulted in closure of the deer hunting season for approximately 3 years. Since then populations have recovered and in recent years there has been an opportunity for hunters to harvest a second deer in many game hunting areas (GHAs). This opportunity will be curtailed beginning in 2009 in western Manitoba because of perceived lower deer numbers and, following the precautionary principle. However, availability of a second tag will continue in the central and eastern portions of Manitoba where deer numbers are more abundant. Other factors which can and have contributed to lower deer densities are predation and the uncontrolled harvest. With changes to rules regarding hunting by Métis peoples, this will be a factor to be considered in ongoing management programs.

When managing for deer in a mixed cervid community, other considerations arise. Both moose and caribou are susceptible (elk less so) to a parasitic nematode (see below) carried by deer. And the impact on these species increases with increased deer densities.

Brain or meningeal worm (*Parelaphostrongylus tenuis*) is a common parasite of white-tailed deer throughout the deciduous mixed woods forest of eastern Canada (Lankester 2001) and was reported in 1972 (Bindernagel and Anderson 1972) as far west as western Manitoba. Bindernagel and Anderson (op cit) stated that it has not been found in boreal mixed wood forests of north central and western Canada characterized by low densities of white tailed deer. Various authors (Bindernagel and Anderson 1972; Samuel and Holmes 1974) have expressed concern that the parasite might be expanding its range westward through the Aspen Parkland Ecoregion and may eventually reach western Canada (e.g. Alberta) where white tailed deer occur sympatrically with other ungulates which are susceptible to the disease (parelaphostrongylosis). A more recent study of the distribution of meningeal worm in Manitoba (Wasel et al 2003) found that 60% + of the deer examined in GHA's 35 and 36 were positive and 40-60% of those in GHA's 19, 14 and 12 were positive. In contrast, in southwestern MB only 0-20% were positive. The lower prevalence of the parasite in the southwest is attributed to the lack of suitable habitat for snails and slugs which are the intermediate host and to generally drier conditions that reduce snail activity and opportunities for transmission. In GHA 25B the prevalence was 20-40% and in those GHA's to the north of this GHA it was greater than 0 but less than 20%.

Wasel et al (2003) found the highest prevalence in MB to be in the southeast and decreasing with distance northwest, west and southwest. Only 3 of 126 deer from the extreme southwestern GHA's in MB were positive for brainworm.

Correlation of higher summer temperatures through fall, good precipitation, higher winter and spring temperatures, higher deer densities and moderately forested areas with the presence of brain worm makes biological sense (Wasel et al 2003). Terrestrial gastropods "require moisture" as do the free living first stage larvae of meningeal worm. Gastropods become relatively inactive when moisture is low (Burch 1956) and deer faeces and surface soils become dry with the likely results that mortality of larvae is increased and transmission is severely restricted (Lankester and Anderson 1968). Conversely, extremes of temperature, short periods between killing frosts and little moisture likely contribute to fewer gastropods (Beetle 1989).

Gastropod intermediate hosts must have spatial and temporal overlap with infected deer faeces (and therefore first stage larvae). High densities of 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 positive correlation with the presence of *P. tenuis* and precipitation suggests that lower rainfall associated with the prairie biome likely acts as a natural ecological barrier for the natural westward spread of the parasite.

A proposal to collect deer heads from hunters this coming hunting season and deer feces later in winter from GHA's 26, 20 and 21 is intended to ascertain what the prevalence is in these areas. Speculation is it will be as high (likely higher) as those reported by Wasel et al (2003) but sample sizes previously were not that large. I

have developed a protocol for such collections and am prepared to do the examinations on heads and feces collected. This issue has been discussed with Dr. Murray Lankester (a noted authority on brain worm) who is still very much interested in brain worm and its impacts on moose. It is our professional opinion based on our familiarity with this parasite that there is no way the prevalence of brain worm in the eastern part of Manitoba has decreased over the last 15-20 years. It has been suggested by others that brain worm evolved in whitetails in the southern U.S. and when they arrived back in MB about the turn of the 20th century the parasite came with them. It is well established in southern MB where habitat and moisture for snails are adequate but, because of its life cycle through snails, its western distribution is spotty – snail habitat is limited in the "great plains". Another reason to expect the continued presence of the parasite in the Province is knowledge that meningeal worm is long-lived in deer (Duffy et al. 2002). 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.

Both Murray and I are becoming more convinced of the general importance of climate/weather to the expected impact of brain worm on moose. For example, warmer winters may see less snow and generally shorter winters (the prediction for the coming winter is it will be an "el nino" one). This means less mortality of deer fawns and increasing deer numbers in areas previously inhabited mainly by moose (or caribou). Another very important aspect of climate/weather is the length and wetness of the green season. For example, a "summer" like 2009 in MB has been excellent for snail/slug survival, reproduction and mobility as well as for survival of brain worm larvae in deer feces. All are significant factors enhancing transmission to new deer fawns, and to 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 we believe are the 2 most important factors affecting parasite impact on moose populations (easy winter for deer, wet summer for transmission and snail survival). The over-all result is the cumulative effect of both. Although the past two winters have been a little more harsh than in the recent past we have had no reports of significant deer mortality and the wet summers, based on what we know about snails, have ensured that the prevalence of brain worm remains high (conditions for transmission are ideal)

I also draw attention to other aspects of brain worm biology. When the MB study was done in the late 80's, the prevalence was 60+% but this was only in adult deer and involved only heads collected during the hunting season. Although fawns make up a significant portion of the harvest, having become infected for the first time in autumn they would not yet have detectable mature worms in their head. Including them in the sample would lower the apparent prevalence of the parasite. The issue is that juvenile deer (young-of- the-year) do not have patent infections as the worms have not yet matured thus being able to find them in the nervous tissue versus the cranial cavity is impossible.

Further, young deer pass larvae at 3X the rate seen in older adults. With a high number of deer and ostensibly many females, this leads to a bumper fawn crop which in turn leads to a lot of young deer in the population which are passing elevated number of larvae which in turn leads to greater risk for species such as moose and caribou.

Over the last 15 years the deer population has expanded significantly in the southeast and this is based on reports from field staff and the general public. And, we still have a second deer licence in this area of the province!

Lankester and Anderson (1968) showed that 70% of larvae in deer pellets survive freeing at -20C for up to 6 months.

The advantage of collecting pellets in late winter is that fawns becoming infected in autumn 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 March-April when most fawns will have become patent. It is also important that samples of smaller-sized pellets, likely from fawns, should not be omitted. As well, pellet samples must be collected off snow to avoid contamination by soil nematode larvae.

Bindernagel and Anderson collected 305 deer pellet groups in Manitoba with larvae being found in 49% of the samples. Prevalence ranged from 81% in southeastern MB to 31% in the aspen parkland of the southwest. To confirm that larvae recovered from deer pellets were in fact meningeal worm, they examined heads from hunter killed animals and road kills and of 59 deer heads from the southwest found 10% positive

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, Slomke et al (1995) in northern Minnesota found 91% of yearlings and 96% of 7-15 year old deer to be infected.

Young, recently infected deer pass more larvae than older animals and animals of all ages pass the greatest number in the spring (Slomke et al 1995; Anderson et al 1963; Anderson and Prestwood 1981). For example, in northern MN, in spring 10-month-old fawns passed twice as many larvae as all older animals (mean = 132/gm vs. 57/gm (dry weight)). By season, all animals passed a mean of 10 larvae/gm in fall rising to a mean

of 57/gm in late-winter/spring. At an average of 10 pellet depositions per day by deer, one can speculate how many larvae will be deposited per day or annually. An example of what these numbers mean in general terms comes from studies of Forrester et al. (1997, JWD 33:511-516) in northern Minnesota. They found that deer pellets collected randomly in late winter had from 80 to 1374 *P. tenuis* larvae each (mean = 439)! If one estimates that, in winter, a deer produces at least 50 pellets per sample and may defecate 15 times per 24 hr, the mean number of larvae released onto range per day, per deer, is 329,250; ie. on average, well over a quarter of a million per day. The number passed by a fawn could approach one million larvae per day.

Forrester S.G. and M.W. Lankester. 1997. Extracting protostrongylid nematode larvae from ungulate feces. J. Wildlife Diseases. 33:511-516.

Slomke, A.M., M.W. Lankester and W.J. Peterson. 1995. Infrapopulation dynamics of *Parelaphostrongylus tenuis* in white-tailed deer. J. Wildlife. Diseases. 31:125-135.