STATUS AND REVIEW OF THE VECTOR-BORNE NEMATODE *SETARIA TUNDRA* IN FINNISH CERVIDS

Sauli Laaksonen and Antti Oksanen

Finnish Food Safety Authority Evira, Fish and Wildlife Health Research Unit, P.O. Box 517, FI-90101 Oulu, Finland

ABSTRACT: The filarioid nematode *Setaria tundra* caused an outbreak of peritonitis in Finnish semi-domesticated reindeer in 2003-2006. Our research group studied the invasion and reservoirs of *S. tundra* in Finnish cervid populations and this paper provides an overview of that research. The outbreak had detrimental effects on reindeer health and may, in part, explain the observed decline of the population of wild forest reindeer (*Rangifer tarandus fennicus*). Both range expansion by roe deer, and high summer temperatures that increased vector populations of mosquitoes and gnats and influenced habitat use by reindeer were implicated in the outbreak. We suggest that vector borne parasites will increase in the Arctic owing to the effect of global climate change and have consequences for all cervid populations.

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There is a growing body of literature documenting the expansion of emerging parasites in sub-arctic areas. The potential impact of global warming on shifts in the spatio-temporal distribution and transmission dynamics of vector-borne diseases in domesticated and wild ungulates may be remarkable (Hoberg et al. 2008). Contemporary Finnish studies have revealed an array of filarioid nematodes and associated diseases that appear to be emerging in northern ungulates (Laaksonen et al. 2007, Nikander et al. 2007, Solismaa et al. 2008). For example, members of the genus *Setaria* (Filarioidea: Onchocercidae) are found in the abdominal cavity of artiodactyls (especially Bovidae), equids, and hyracoids. All species produce microfilariae that are present in host blood, and known vectors are haematophagous mosquitoes (*Culicidae* spp.; Anderson 2000) and horn flies (*Haematobia* spp.; Shol and Drobischenko 1973).

The filarioid nematode *Setaria tundra* was first described in semi-domesticated reindeer (*Rangifer tarandus tarandus*) in the Arkhangelsk area of Russia by Rajevsky (1928). *Setaria* sp. infections appear to first emerge in Scandinavian reindeer in the 1960s. *S. tundra* was observed initially in northern Norway in 1973 where there was an outbreak of peritonitis in reindeer. In the same year, tens of thousands of reindeer died in the northern part of the herding area of Finland. Severe peritonitis and large numbers of *Setaria* sp. worms were common. However, the incidence of *Setaria* sp. in Scandinavian reindeer diminished afterward (Laaksonen et al. 2007).

According to meat inspection data and clinical reports from practicing veterinarians in Finland, the latest outbreak of peritonitis in reindeer started in 2003 in the southern and middle parts of the reindeer herding area. In the province of Oulu, the proportion of reindeer viscera condemned due to parasitic lesions identified during meat inspections increased dramatically from 4.9% in 2001 to 47% in 2004; in Lapland the increase was from 1.4% in 2001 to 43% in 2006. These increases caused substantial economic loss and increased workload associated with meat processing. The focus of the outbreak moved northward approximately 100 km/yr, and by 2005 only those reindeer in the small, northernmost part

of Finland (Upper Lapland) were free of lesions. During the same period, the peritonitis outbreak was apparently concentrating in the southern area (Laaksonen et al. 2007). The causative agent was *S. tundra* based on morphologic and molecular data. Samples of DNA sequences of *S. tundra* parasitising reindeer in northern Finland were deposited in GenBank under accession number DQ097309 (Laaksonen et al. 2007, Nikander et al. 2007)

The prevalence and intensity of *S. tundra* microfilariae (smf) were higher in reindeer calves than adults; overall prevalence was 42%. The overall smf-prevalences for moose (*Alces alces*), wild forest reindeer (*Rangifer tarandus fennicus*), and roe deer (*Capreolus capreolus*) were 1.4-1.8%, 23%, and 39%, respectively. The focus of microfilaremia in reindeer moved north as it declined simultaneously in the south as the observed peritonitis outbreak lessened. Experimentally, reindeer calves infected in their first summer of life had peak microfilaremia in their second summer. Captive reindeer were smf positive throughout the year, but smf disappeared from the blood after 2 years. The prepatent period of *S. tundra* was estimated to be about 4 months, with a life span of at least 14 months (Laaksonen et al. 2008a)

Reindeer calves with heavy *S. tundra* infection expressed decreased thriftiness, poor body condition, and undeveloped winter coat. In Kuusamo, 4603 slaughtered reindeer were examined clinically in 2003-04; meat inspections of diseased reindeer carcasses revealed ascites fluid, green fibrin deposits, adhesions, and live and dead *S. tundra* nematodes. Histopathology indicated granulomatous peritonitis with lymphoplasmacytic and eosinophilic infiltration. No specific bacterial growth was found. No significant impact on pH values of meat or on organoleptic evaluation of meat was found. There was a significant positive correlation between worm counts and the degree of peritonitis, and a negative correlation between the degree of peritonitis and back-fat layer (Laaksonen et al. 2007). *Setaria yehi* has been associated with low grade chronic peritonitis in Alaskan reindeer (Dieterich and Luick 1971). *S. tundra*, in combination with *Corynebacterium* sp., has been associated with mild to severe peritonitis in Swedish reindeer (Rehbinder et al. 1975). Based on the evidence in both ante and post-mortem inspections and histological examinations, our studies (Laaksonen et al. 2007, Laaksonen et al. 2008b) and historical data indicate that *S. tundra* can act as a significant pathogen in reindeer.

We collected parasite samples from wild cervids in order to monitor the dynamics of *S. tundra* in nature. About 300 moose, the most abundant wild cervid in the reindeer herding area, were inspected and only a few cases of pre-adult encapsulated *S. tundra* nematodes were found on the surface of livers. However, no peritonitis was identified (Laaksonen et al. 2007), and the prevalence and intensity of smf in 324 moose blood samples within and outside the reindeer herding area were low (1.4% and 1.8%, 1-3 smf/mL blood; Laaksonen et al. 2008a). Because the moose population in northern Finland peaked in 2004-2005, moose are apparently not a suitable reservoir host for the *S. tundra* haplotype occurring in reindeer. There has been one previous report of a peritonitis outbreak in moose associated with *Setaria* sp. nematodes in Finnish Lapland in 1989 (Nygren 1990). Although this earlier outbreak took place within the reindeer herding area, there was no concurrent report of any associated, increased morbidity in reindeer.

It is possible that the high percentage (62%; 21 of 34) of wild forest reindeer with signs of peritonitis caused by *S. tundra* (Laaksonen et al. 2007) may be related to its substantial population decline (1700 to 1000) in 2001-2005 (Kojola 2007). Two roe deer examined fresh in the field had *S. tundra* nematodes in their abdomen and smf in circulating blood, but no peritonitis. According to our studies, roe deer seem to be a capable host and asymptomatic carrier of *S.*

tundra. This conclusion is supported by the simultaneous appearances in the 1960-1970s of *S. tundra* in Scandinavia (Laaksonen et al. 2007) and roe deer in North Scandinavia (Haugerud 1989). Considering the reservoir host capacity of roe deer and the dynamics of *S. tundra,* we suggest that young male roe deer that can disperse many hundred kilometers from their birthplace (Cederlund and Liberg 1995) could be efficient long-distance vectors for *S. tundra*. Further support for this theory is that only minor nucleotide differences exist between the reindeer *S. tundra* sequence (648 bp) and that of specimens from roe deer in Italy (GenBank AJ544874, Casiraghi et al. 2004), indicating that they are the same haplotype.

Mosquitoes, particularly *Aedes* spp. and to a lesser extent *Anopheles* spp., play an important role in the transmission of *S. tundra* in reindeer herding areas in Finland. The prevalence of filariod larvae in Finnish mosquitoes naturally infected with *S. tundra* varied from 0.5-2.5%. However, the rate of development in mosquitoes is temperature dependent; infective larvae were present approximately 14 d after a blood meal in mosquitoes maintained at room temperature (mean 21° C), but did not develop in mosquitoes maintained outdoors for 22 days at a mean temperature of 14.1° C. The third-stage (infective) larvae had a mean length of 1411 μm (SD 207) and width of 28 μm (SD 2) (Laaksonen et al. 2009).

The 1973 *S. tundra* outbreak in Sweden was associated with unusually warm weather and abnormally high numbers of mosquitoes and gnats (Rehbinder et al. 1975). The summers of 1972 and 1973 in Finland were also very warm, as were those in 2002 and 2003 (Finnish Meteorological Institute data, pers. comm., S. Nikander 2004). Warm summers apparently promote transmission and genesis of disease outbreaks by favoring the development of *S. tundra* in its mosquito vectors, by improving the rate of mosquito development and reducing their mortality from frost, and finally, by forcing reindeer to stay in herds on mosquito-rich wetlands (Laaksonen et al. 2009). Mosquito-borne diseases are among those most sensitive to weather and predictably will be influenced by climate change. Climate change can directly affect disease transmission by shifting the vector's geographic range, increasing reproductive and biting rates, and shortening the incubation period of the pathogen (Patz et al. 1996). Thus, we predict that global climate change has the potential to promote the further emergence of filarioid nematodes and diseases caused by them in the subarctic ecosystem.

This study indicated that *S. tundra* likely has an important impact on boreal ecosystems. It also revealed the absence of baseline knowledge concerning temporal parasitic biodiversity in cervids at high latitudes. Therefore it is important to gain knowledge about these parasites, their ecology, transmission dynamics, and their impact on human and animal health. The potential relationship between climate change and a vector-borne disease identified in this paper indicates the potential and obvious threats to the individual and population health of arctic ungulates.

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