Moose Parasites in Relation to Supplementary Feeding

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Master Thesis at Faculty of Forestry and Wildlife Management

HEDMARK UNIVERSITY COLLEGE

2011

Contents

Abstract

In this study, I tested the effect of winter supplemental feeding of moose (*Alces alces*) on gastrointestinal parasite infection in two counties in Norway, by comparing faecal egg counts of moose which used and did not use feeding stations. I identified three different GI nematodes based on egg morphology; *Trichostrongylidae spp.*, *Nematodirus spp.* and *Trichuris spp.* All species were found in Hedmark while in Telemark I found only *Trichuris spp.* Prevalence of *Trichostrogylidae spp*. and *Nematodirus spp.* varied significantly depending on year sampled, and age class and month, respectively. Age class, year and body mass significantly affected intensity of *Trichostrogylidae spp*. infection. *Trichostrogylidae spp*. abundance was higher in 2009 when weather conditions were more challenging, and decreased with increasing body mass. Adult moose had a higher intensity of infection than juvenile moose and female juvenile moose had lower abundances than male juvenile moose. Use of feeding stations did not affect prevalence of any parasite species or intensity of infection of *Trichostrogylidae spp*. Due to the low prevalence and high number of zeros, I was unable to model intensity of *Nematodirus spp.* and *Trichuris spp.* infection. Possible explanations for my findings and future research prospects are discussed.

Keywords: *Alces alces; moose; supplementary feeding; GI nematodes; Trichostrogylidae; Nematodirus; Trichuris; negative binomial distribution; parasite aggregation*

Sammendrag

I denne undersøkelsen analyserte jeg påvirkning av fôringsstajoner på elgens infeksjoner med gastrointestinale nematoder i to fylker i Norge ved å sammenligne mengden av parasittegg i avføring hos elg som brukte og ikke brukte fôringsstasjoner. Jeg identifiserte tre slekter tilhørende gastrointestinale nematoder basert på egg morfologi, i. e. *Trichostrongylidae spp.*, *Nematodirus spp.* og *Trichuris spp.* Alle tre slekter ble funnet i Hedmark mens i Telemark fant jeg kun *Trichuris spp.* Prevalens av *Trichostrongylidae spp.* og *Nematodirus spp.* varierte signifikant med henholdsvis år, og aldersklasse og måned. Aldersklasse, år og kroppsvekt påvirket signifikant på grad av *Trichostrogylidae spp*. infeksjon. Graden av infeksjon var høyre i 2009 når værforholdene var mer utfordrende og den minket med økende kroppsvekt. Voksen elg hadde høyre grad av infeksjon enn kalv og hunnkalv hadde lavere grad av infeksjon enn hannkalv. Bruk av fôringsstasjoner påvirket ikke prevalensen av de tre nematode slektene eller graden av *Trichostrogylidae spp*. infeksjon. På grunn av lav prevalens og høy antall nuller klarte jeg ikke å tilpasse negativ binomial regresjon for graden av *Nematodirus spp.* og *Trichuris spp.* infeksjon. Det diskuteres om mulige forklaringer for mine funn og utsikter for fremtidig forskning.

Nøkkelord: *Alces alces; elg; fôringsstasjon; GI nematoder; Trichostrogylidae; Nematodirus; Trichuris; negativ binomial regresjon; parasitt aggregasjon*

Introduction

Parasite infections in wildlife, as in domestic animals, seldom lead to mortality but are rather characterized by morbidity and chronic course (Tompkins et al. 2002, Gunn and Irvine 2003, Irvine et al. 2006, Lankester and Samuel 2007). The sub-lethal effects of parasites on a host animal include general reduced fitness because of reduced appetite and food assimilation, leading to poorer competitive ability, reduced resistance against other pathogens, impaired growth, poorer reproductive success and changes in behavior (Bye 1987, Gulland 1995, Hudson and Dobson 1995, Arneberg et al. 1996, Stien et al. 2002b, Gunn and Irvine 2003, Newey and Thirgood 2004, Newey et al. 2004, 2005, Irvine 2006, Hughes et al. 2009). In turn, these effects can impact on host population dynamics, as well as the dynamics of the parasite population.

Previously it was common to assume that parasites played a minor role in the dynamics of their host population, living in a more or less sensitive balance with their hosts (Dobson and Grenfell 1995, Tompkins et al. 2002). It was argued that parasites could not regulate their host population because by causing the death of their host they would also die off. But as the lifetime reproductive success of a parasite depends on several processes including transmission, reproduction and survival, they can nonetheless have serious effects on their hosts (Tompkins et al. 2002). Roy Anderson and Robert May (Anderson and May 1978, May and Anderson 1978) developed the first theoretical models showing that parasites can indeed regulate host populations if they reduce the fecundity and/or survival of their hosts in a density dependent manner. When regulating the host population they reduce the tendency for uncontrollable growth or variation in host numbers. The regulative effect of parasites is highly dependent on the degree of, so called, parasite aggregation; some individual hosts have high parasite abundances, while others have none or a few parasites (Pacala and Dobson 1988, Shaw and Dobson 1995). With increasing aggregation, the stability of the host-parasite interaction is enhanced (Anderson and May 1978, May and Anderson 1978, Wilson et al. 2002). In contrast, time delays in parasite recruitment and a random distribution of parasites in the host population could destabilize populations. Depending on the nature of density dependent processes in the host-parasite interaction and the degree of parasite aggregation, either regulative or destabilizing processes will predominate (Roberts et al. 1995, Newey et al. 2005).

Host population regulation by parasites can be difficult to detect in populations that are in equilibrium, so a disturbance may be necessary. Field studies with anthelminthic experiments by Hudson et al. (1985, 1992, 1998) have demonstrated the regulatory effect of parasites in a red grouse population and by Albon et al. (2002), showing evidence of parasites regulating a Svalbard reindeer (*Rangifer tarandus platyrhynchus*) population in its natural environment through decreasing fecundity. Soay sheep (*Ovis aries*) and the gastrointestinal nematode *Teladorsagia circumcincta* (Gulland 1992, Tompkins et al. 2002), and mountain hare (*Lepus timidus*) infected with gastrointestinal nematodes *Graphidium strigosum* and *Trichostrongylus retortaeformis* (Newey and Thirgood 2004, Newey et al. 2005) are examples of other systems in which parasites play a role in regulating their host populations.

Host-parasite systems are influenced by several heterogeneities leading to individual variation in the degree of infection. These heterogeneities can be classified into four groups: immunological, spatial, genetic and ecological (Dobson and Grenfell 1995, Wilson et al. 2002). The grouping is clear but not without overlaps, as heterogeneities can occur simultaneously. Some of the individual variation in parasite abundances can be explained by differences within host population, like sex and age (Hudson and Dobson 1995, Schalk and Forbes 1997, Boag et al. 2001, Isomursu et al. 2006, Wirsing et al. 2007, Davies et al. 2008, Hillegass et al. 2008). An individual's fitness (Halvorsen et al. 1999, Ezenwa 2004a) and behavior, including feeding behavior (Ezenwa 2004b, Apio et al. 2006), can also explain parasite aggregation, as can host population genetics (Galvani 2003). We also have to consider parasite genetics, seasonal variation in infection levels and spatial distribution of the parasite's infective stages in the environment (Bordes et al. 2009). Still, it is unclear how important these different mechanisms are, either on their own or when possibly working together (Wilson et al. 2002).

Parasite infections are transferred by a contact process between hosts. Increases in population density have been suggested as a possible reason for increases in parasitic diseases (Aguirre et al. 1999, Arneberg 2001, Albon et al. 2002) through increased contact between host animals and increased host aggregation (Gortázar et al. 2006). Supplementary feeding of wildlife can lead to unnaturally high local densities and levels of contact between hosts (Putman and Staines 2004, Hines et al. 2007) resulting in an increase in the basic reproductive rate of the parasite (R_0). R_0 is a measure of parasite fitness as it describes the number of female worms that result from one female worm in a population of fully susceptible hosts, when there are no density dependent constrains operating (Hudson et al. 2002).

There are several reasons for supplementary feeding of game animals during winter months, as practiced throughout Europe and parts of North America (Putman and Staines 2004). Reasons for supplementary feeding include reducing or preventing agricultural or forest damage, improving body weights or trophy size, increasing reproductive performance and fertility, improving the annual yield through hunting so benefiting the landowners and indirectly the hunting community, and for other recreational opportunities like viewing and photographing. In addition, diversionary feeding may help prevent traffic accidents on main highways and railways (Andreassen et al. 2005).

There is an ongoing debate about the benefits and possible negative consequences of supplementary feeding among landowners, managers and researchers (Putman and Staines 2004, van Beest 2010 a, b). Effects on body condition and reproductive performance vary depending on feeding regime, type of supplementary feed provided, and location of the feeding site. Effects on fecundity are equally equivocal; indeed feeding could maintain artificially high densities depleting summer and spring resources causing a decline in fecundity. In addition there is the potential for increased disease transmission as animals gather into unnaturally dense winter populations around feeding stations (Hines et al. 2007). This includes pathogens like bacteria and viruses, and parasites like gastrointestinal (GI) nematodes and other internal parasites (endoparasites).

GI nematodes are abundant in wild ruminants (Hoberg et al. 2001), but the parasite status of Norwegian moose is mostly unknown. Therefore how subclinical endoparasite infections affect growth and reproductive success of moose and thus population dynamics is also unknown. Supplementary feeding of moose with silage (bales of mixed graminoids) has been practised in Norway for two main reasons. One is to reduce train and vehicle collisions caused by seasonal migration of moose across the main highways and railways located in the valleys (Gundersen et al. 1998). The other important reason is the severe damage moose cause to forestry during winter months (Storaas et al. 2001). Moose can gather in groups in their winter habitat, feeding mainly on young pine. Supplementary feeding with silage is thought to keep the moose off young pine plantations and decrease the browsing damage.

In this thesis I investigated the GI parasites of moose and compared moose that use supplementary forage with moose feeding on natural browse. I tested two alternative hypotheses: H1) Winter supplementary feeding enhances parasite (and disease) transmission by aggregating moose at feeding grounds leading to higher parasite abundances in feeding site

users, or H2) supplementary feeding may improve body condition enabling moose to better combat parasite infection leading to lower parasite abundances in fed moose. I investigated qualitatively and quantitatively whether adult and juvenile moose shared the same parasite species and whether they had different parasite species at the start compared with the end of the feeding season, at two study sites in southern Norway.

Material and Methods

Study areas

Two separate areas were included in the study, one located in southern Norway, within parts of Telemark, Buskerud and Vestfold counties and the other in southeastern Norway, in Hedmark county, Stor-Elvdal municipality (fig. 1). Both areas harbored large moose populations. The landscape-scale winter density of moose was >1 moose/km^2 in both study areas (Gundersen et al. 2008; Solberg et al. 2003), with local wintering area densities far exceeding this. In Telemark, hunting was the main cause of moose mortality as large predators were absent. Hedmark county has large predators, but hunting still remains the main cause of mortality. In Stor-Elvdal municipality in Hedmark the supplementary feeding of moose started in the late 1980s (Andreassen et al. 2005) while in Telemark the feeding stations had been used for ≤ 6 years (van Beest et al. 2010a). 182 and ~1700 tons of forage was consumed by moose during the 4 - 6 month long winter in Telemark and Hedmark, respectively (van Beest 2010a, b).

The southern location was in the boreonemoral zone with mostly coniferous forest dominated by Norway spruce (P*icea abies*; 72%) and Scots pine (*Pinus sylvestris;* 17%) with some mixed deciduous stands of birch (*Betula spp.*), mountain ash (*Sorbus aucuparia*), willow (*Salix spp.*) and aspen (*Populus tremula*). Altitude ranged from 20 m to 800 m. Mean snow depth in January and March was 27 and 65 cm, respectively using averages from two weather stations inside the study area (Mykle, altitude 430 m and Godal 475 m) (Norwegian Meteorological Institute 2011). The mean monthly winter temperature (January - April 2007-08) was 1.9 °C (min: -0.6 °C in February, max: 6.6 °C in April; Siljan weather station at 100 m altitude, The Norwegian Meteorological Institute). The Hedmark study area ranged in elevation from 250 m to 1100 m and consisted mainly of boreal forest with pure or mixed stands of Scots pine and Norway spruce, with more pine and less spruce than in the Telemark study area. Throughout the whole area minor stands of deciduous forest were found. Average temperatures and snow depths at Haugedalen weather station (approx 35 km to south of the

study area, altitude 240 m) in January and March 2009 were -8.2° C and 52 cm and -2.1°C and 74 cm, respectively, and in January and March 2010 -15.7° C and 46 cm and -3.74° C and 54 cm, respectively (Norwegian Meteorological Institute 2011).

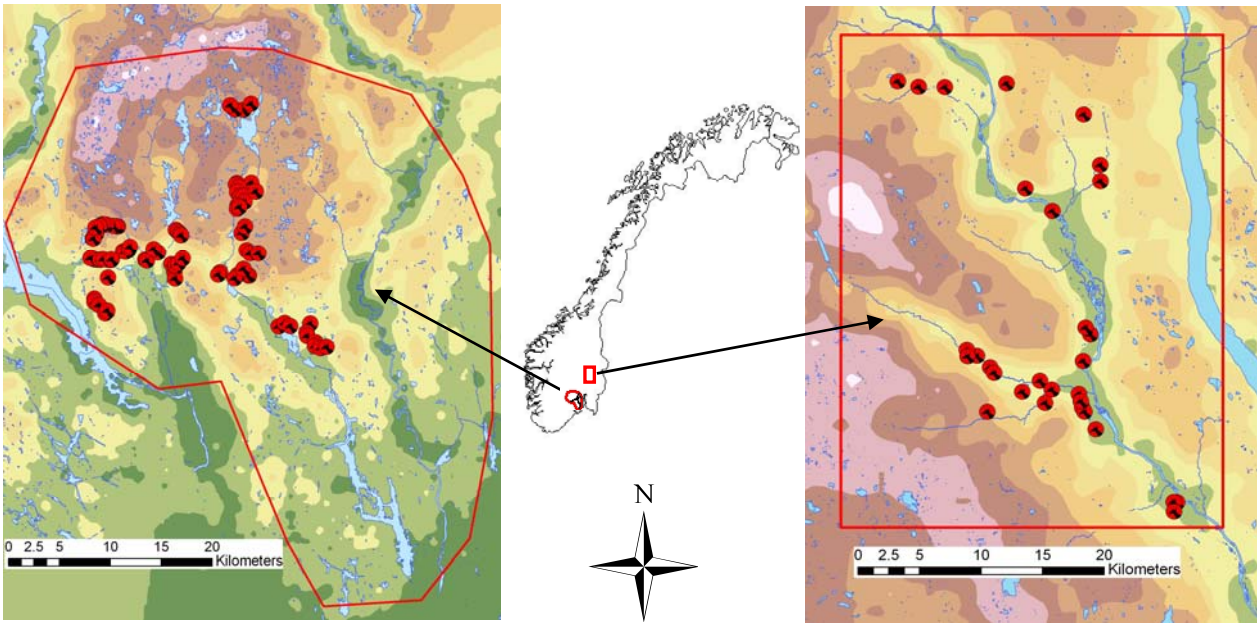

Fig. 1. Feeding stations (red circles) in the two study sites: Telemark (on the left) in the southern Norway and Hedmark (on the right) in the southeast Norway (by courtesy of F. M. van Beest).

Parasitological sampling

Faecal and blood samples were collected in conjunction with the "Elgfôringsprosjekt" (Milner 2010). Faecal samples were collected in Telemark in January 2007 and in Hedmark in January and March 2009 and 2010 from immobilized moose. Blood samples (n=40) were also collected in Hedmark in January 2009. Adult female moose with calves were immobilized from a helicopter with a dart gun (Arnemo et al. 2003) and adult moose were collared with a GPS-collar. In January 2009, the calves got a VHF-collar. The moose were also weighed using a net and helicopter. Moose were recaptured in March and all procedures were repeated. Some of the calves were not with their mothers in March 2009 and could not be found, or they were in too poor condition for recapturing (four calves). Because of the possible disturbance from capturing in January and subsequent separation of mother and calf, only adult moose were captured in January 2010, while both adult and juvenile moose were captured in March 2010.

Faecal and blood samples were frozen at -20° C until analyzed at the laboratory of Finnish Food Safety Authority, Evira in Oulu, Finland. Downloaded GPS positions were used to

assign feeding status of the moose dividing them into users and non-users. Moose spending 10% or more of their total GPS locations within 100 m of a feeding station were defined as users while others were defined as non-users.

EDTA (VenoSafe™, Terumo Europe) blood samples were examined for the presence of the filarial nematode, *Setaria* species (sp.). All *Setaria sp.* produce larval stages, microfilariae, which can be found in the blood over one year after initial infection (Laaksonen et al. 2009). In the late 1960s, infections with *Setaria sp*. emerged in Scandinavian cervids and peritonitis caused by *Setaria tundra* was diagnosed for the first time in Swedish reindeer in 1973 (Rehbinder et al. 1975). In Norway, *Setaria tundra* was isolated for the first time in outbreak of peritonitis and perihepatitis in reindeer in 1973 (Kummeneje 1980). Filarial nematodes are transmitted by vectors, and known vectors are haematophagous mosquitoes (*Culicidea* species) and horn flies (*Haematobia* species). In moose, *Setaria sp.* can cause a mild, chronic peritonitis (Lankester and Samuel 2007) with highest prevalence in young animals (<2 years old) (Laaksonen et al. 2007, 2009).

Each blood sample was examined for the presence of microfilariae with an oil immersion objective using modified Knott's technique (Georgi 1985). One ml EDTA blood was added into a centrifuge tube containing 10 ml of 2% formalin. After mixing the blood by inverting the centrifuge tube, it was centrifuged at 1300 G for 12 minutes. The supernatant was discarded and the sediment was stained with 20 µl methylen blue. The mixture was put on a glass slide and examined for microfilariae. To make the identification of *Setaria sp.* reliable and making sure of having a correct search image, I used a reference sample which I repeatedly examined with the microscope in-between examining the blood samples. Each sample was examined with one prepared slide.

Faecal samples were analyzed for parasite eggs using standardized flotation techniques for parasite investigations (Evira LAB 5614/1). I estimated eggs per gram faeces (EPG) by weighing and homogenizing 3 g of faeces in 42 ml lukewarm tap water. The mixture was sieved with a tea sieve and 12.5 ml of the solution was centrifuged at 300 G for 3 minutes. The supernatant was discarded and saturated saccharose was added to the sediment to total volume of 2.5 ml. After carefully mixing, the solution was pipetted into a chambered McMaster slide. Parasite eggs were counted using a microscope with 100x magnification and categorized based on morphological features.

Faecal samples were also examined for *Salmonella spp.*, a group of zoonotic bacteria causing serious diarrhea in animals as well as humans, using an established culture technique (Evira LAB 5201/1). I used pooled samples of 10 animals instead of individual faecal samples. After a pre-enrichment process of 24 h in buffered peptone water at 37 ° C, 3 drops of pre-enrichment media were transferred with Pasteur-pipette on MSRV (modified semisolid Rappaport Vassiliadis) agar and incubated at 41.5 ° C for 24 h. If present, bacteria growth was further cultured on XLD (Xylose-Lysine_Desoxycholate) agar at 37° C for 24 h.

Assessment of physical condition

I used body mass (kg) to estimate physical condition in both study areas.

Statistical Analysis

I analyzed a set of common parasitological parameters in connection to my parasite epidemiological data to explain the patterns of parasite distribution within the moose populations. These were the parasite prevalence, intensity of parasite infection (abundance), and the degree of aggregation. When the variance to mean ratio (s^2/m) of parasite numbers per host is significantly greater than 1, there is an aggregated distribution (Wilson et al. 2002). The *corrected moment estimate of k* from the negative binomial distribution is best suited as an index of this aggregation (Gregory and Woolhouse 1993). It inversely measures the degree of aggregation in the host population (Anderson and May 1978). Fisher (1941) and Bliss & Fisher (1953) explained the negative binomial distribution as $s^2 = m + m^2/k$. As k decreases the aggregation increases. Parasite infections in wildlife hosts often have $k < 1$ meaning there is a high degree of aggregation (Shaw and Dobson 1995) and the negative binomial distribution has been found to provide a statistically satisfactory fit in wildlife host-parasite systems (Shaw et al. 1998).

My sample size was the number of faecal or blood samples. Prevalence was the number of moose infected / number of moose examined. Intensity of infection (abundance) was the total number of eggs per gram faeces of a particular parasite species /total number of examined host-animal (i.e. infected /(infected + uninfected)) (Margolis et al. 1982). The index of aggregation (k) was *corrected moment estimate of k* [$k=m^2-(s^2/n)/(s^2-m)$] (Gregory and Woolhouse 1993, Wilson et al. 2002).

Statistical analyses were carried out in SAS (SAS v. 9.2 SAS Institute Inc., Cary, USA). I tested for differences in *Trichostrongylidae spp., Nematodirus spp.* and pooled parasite prevalence using generalized linear mixed models (GLMM) with binomial errors and logit

link (Glimmix). My explanatory variables in Hedmark were age class, feeding status, month, body mass, year and their 2-way interactions. I fitted moose identification as a random factor to avoid pseudo-replication. In Telemark I only had one sample per individual so I tested for differences in *Trichuris spp*. prevalence with generalized linear models (GLM) with binomial errors and logit link (Glimmix). My explanatory variables were age class, body mass and age class*body mass.

I investigated the frequency distribution of *Trichostrogylidae spp.* and *Nematodirus spp.* in Hedmark and *Trichuris spp.* in Telemark in adult and juvenile moose using histograms. Based on these results, I was able to understand the difficulties in trying to get statistically satisfactory fits with negative binomial distribution when running *Nematodirus spp.* and *Trichuris spp.* (Telemark) abundance models.

I investigated the possibility of co-infection with the two parasite species found in Hedmark by correlation, but found it non-significant (correlation coefficient 0.024). Furthermore, the frequency distributions of *Trichostrogylidae spp.* and *Nematodirus spp.* were so dissimilar that fitting models of pooled parasite abundance was not warranted (Grafen and Woolhouse 1993).

In addition, I ran models of factors affecting the intensity of *Trichostrogylidae spp*. infection in Hedmark with all moose, adults and calves. I investigated the effect of age class, feeding status, body mass, and month and year (both fitted as a two level factors), and their 2-way interactions using GENMOD procedure and negative binomial distribution with log link function. Again, I fitted moose identification as a random effect. When running models for calves only, I tested the effect of sex. My response variable was intensity of *Trichostrongylidae spp.* infection. Due to the very low prevalence of *Tricuris spp*. in Hedmark (one adult infected in January 2009) I did not make a separate model for *Trichuris spp.*

I used backward stepwise selection from a starting model including all variables and their interactions. In all analyses the significance level was set to $p \leq 0.05$.

Results

FAECAL SAMPLES

I found three morphologically different types of GI nematode eggs in faeces; *Trichostrogylidae spp*., *Nematodirus spp.* and *Trichuris spp.* All three types were found in Hedmark while in Telemark I only found *Trichuris spp.* No *Salmonella spp.* was found in any faecal samples.

Prevalence

Parasite prevalence varied from 0% to 80% depending on study site, month and year sampled, age class and parasite species (table 1). In Hedmark, 51 (78%) of 65 and 20 (49%) of 41 samples in 2009 and 2010, respectively, were infected with at least one GI parasite. Year was the single significant factor for *Trichostrongylidae spp.* and pooled parasite prevalence ($F_{1,103}$ = 13.84 and 9.36, p = 0.0003 and 0.003, respectively). Moose were more likely to be infected with *Trichostrongylidae spp*. in 2009 than in 2010. *Nematodirus spp.* prevalence was affected by month ($F_{1, 101}$ = 4.60, p = 0.034) and age class ($F_{1,101}$ = 4.60, p = 0.034). Moose were more likely to be infected with *Nematodirus spp.* in January (i.e. early winter) and juvenile moose had a higher likelihood of *Nematodirus spp.* infection than adult moose. Neither weight, feeding status, year nor sex (in calves) affected prevalence of *Nematodirus spp.* (p > 0.05). *Trichuris spp.* only occurred in one sample from an adult female in Hedmark. In Telemark, neither *Trichostrongylidae spp.* nor *Nematodirus spp.* were found. The prevalence of *Trichuris spp.* was not significantly affected by body mass, age class or their interaction (p > 0.05).

Table 1. Prevalence (%) of gastrointestinal nematodes in moose in Hedmark and Telemark.

				Prevalence (%)			
Study site	**Age class**	**Month**	**N**	***Trichostrongylidae spp.***	***Nematodirus spp.***	***Trichuris spp.***	***Pooled***
HEDMARK	**Adult**	**January 2009**	19	74	11	5	79
		2010	14	29	29	0	50
		March 2009	16	75	6	0	55
		2010	18	39	11	0	50
	Calf	**January 2009**	20	65	45	0	80
		March 2009	10	80	20	0	80
		2010	9	33	11	0	44
TELEMARK	**Adult**	**January 2007**	15	0	0	27	-
	Calf	**2007**	9	0	0	44	-

Intensity of infection

Frequency distribution

Intensity of *Nematodirus spp.* infection varied from 0 to 15.6 (±SE 8.7) EPG depending on age class, feeding status, and month and year sampled (table 2). The large number of zeros and the very low prevalence of *Nematodirus spp.* in adult (58 (87%) out of 67 faecal samples uninfected) and juvenile (26 (68%) out of 38 faecal samples uninfected) moose in Hedmark meant that negative binomial distribution models were not suitable for analyzing intensity of infection in these species (fig. 2). Instead I would have liked to fit zero-altered negative binomial models (ZANB or Hurdle models; Zuur et al. 2009), but unfortunately such models are not yet available in SAS. Factors affecting the intensity of infection with *Nematodirus spp.* have therefore not been modeled.

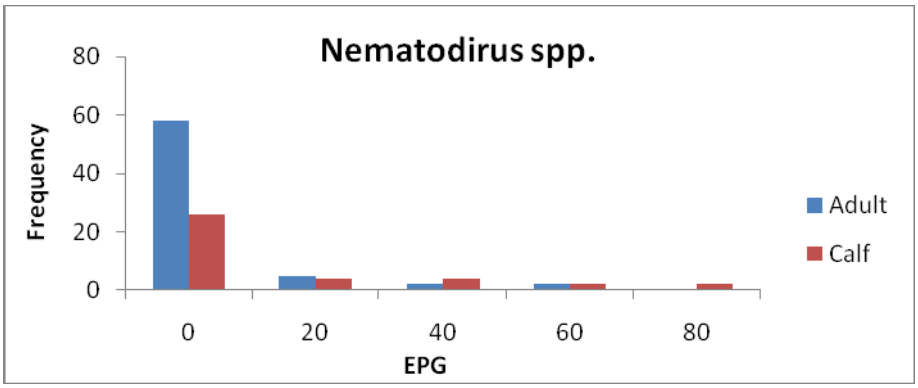

Fig. 2. *Nematodirus spp.* frequency in Hedmark did not fit to negative binomial distribution models due too many zeros.

The intensity of *Trichostrongylidae spp*. infection ranged from 4.4(±2.9) to 60.0 (±0) depending on age class, feeding status, and month and year sampled (table 2). The frequency distribution of *Trichostrongylidae spp*. abundance fitted the negative binomial distribution well (see modeling below). 45% of adult and 40% of juvenile moose faecal samples were not infected with *Trichostrongylidae spp*. (fig.3).

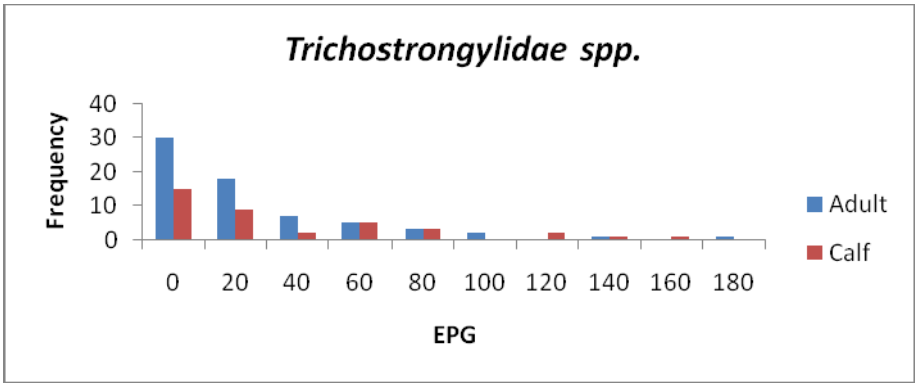

Fig. 3. Both adults and calves showed fairly similar frequency distributions of *Trichostrongylidae spp*. in Hedmark.

73% of adult moose and 56% of juvenile moose had no *Trichuris spp.* in faecal samples from Telemark. The intensity of *Trichuris spp.* infection varied from 31.4 (±24.4) to 86.7 (58.8) depending on age class (table 2). The low prevalence and large number of zeros again made it inappropriate to model *Trichuris spp.* intensity with negative binomial models (Fig. 4), and also here I would have liked to fit zero-altered negative binomial models (ZANB or Hurdle models).

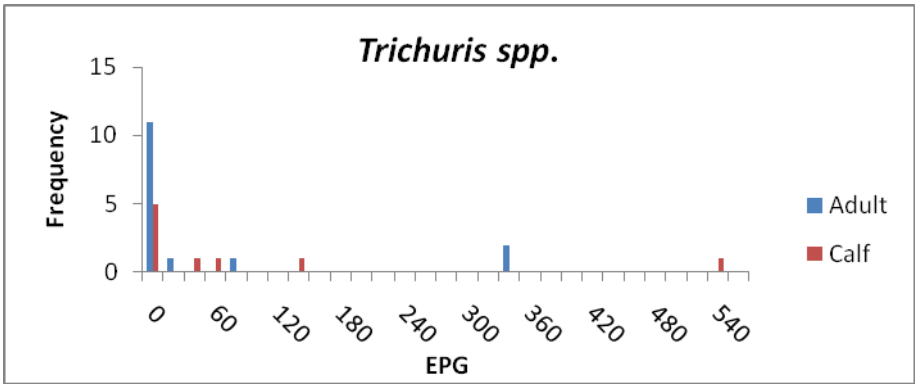

Fig. 4. 11 out of 15 faecal samples in adult moose and 5 out of 9 faecal samples in juvenile moose had no *Trichuris spp.* eggs in Telemark.

Intensity of Trichostrogylidae spp. infection

Modeling adult and juvenile moose together, age class, year and body mass significantly affected intensity of *Trichostrogylidae spp.* infection in Hedmark (χ^2 = 4.22, 14.80 and 6.26, p= 0.04, 0.0001 and 0.012, respectively; fig. 5 and table 2). Adult and juvenile moose had a lower intensity of *Trichostrogylidae spp.* infection in 2010 than 2009 and intensity of infection decreased with increasing body mass. Adult moose had a higher intensity of *Trichostrogylidae spp.* infection than juvenile moose. Neither feeding status nor month were influential after accounting for body mass (p = 0.479 and 0.138, respectively).

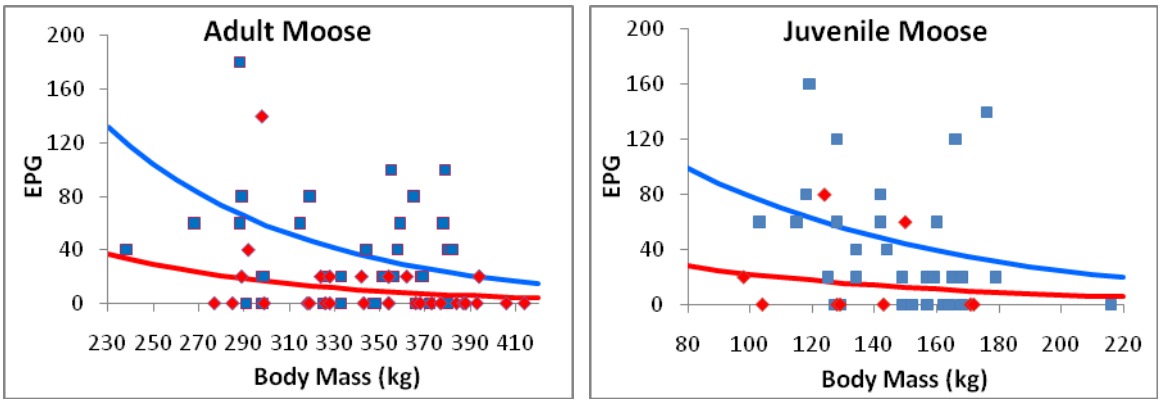

Fig. 5. Predicted intensity of *Trichostrogylidae spp*. infection for adult and juvenile moose in 2009 (blue line) and in 2010 (red line) and observed values (2009 blue and 2010 red) in Hedmark (EPG = eggs per gram faeces). Notice the different scales for body mass.

Within adults only, moose had a higher intensity of *Trichostrogylidae spp*. infection in 2009 ($\chi^2 = 11.39$, $p = 0.0007$) and intensity of infection decreased with increasing body mass ($\chi^2 = 4.02$, $p = 0.045$, fig. 5). Within juveniles, female moose had a lower intensity of *Trichostrogylidae spp.* infection than male juvenile moose ($\chi^2 = 6.79$, $p = 0.047$) and the intensity of infection decreased with increasing body mass ($\chi^2 = 10.30$, $p = 0.03$; fig. 6).

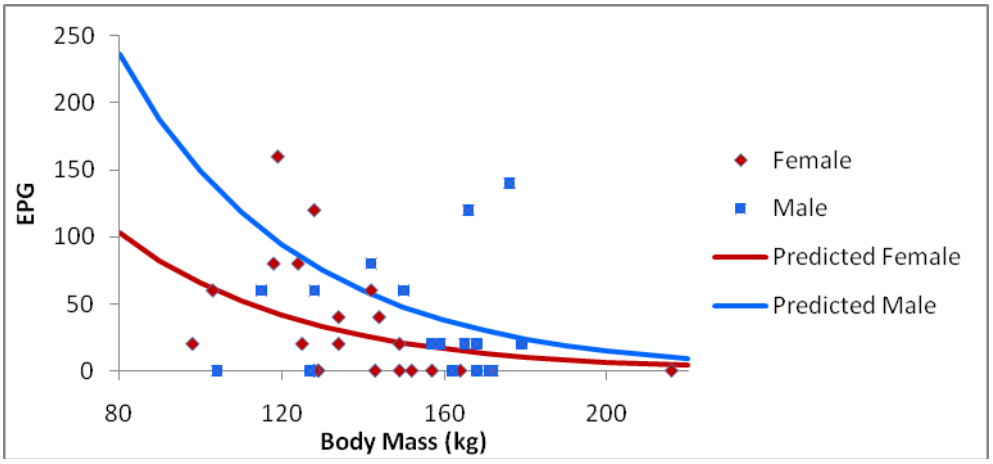

Fig. 6. Intensity of *Trichostrogylidae spp*. infection in calves was affected by sex and body mass.

Table 2. Intensity of GI nematode infection (±standard error) (eggs per gram faeces) of feeding station user and non-user moose in two age classes in Hedmark (2009 and 2010) and Telemark (2007).

Age class	Month and Year	*Trichostrongylidae spp.*		*Nematodirus spp.*		*Trichuris spp.*	
		User	**Non-user**	**User**	**Non user**	**User**	**Non-user**
Adult	**January 2007**	0	0	0	0	340 (0)	31.4 (24.4)
	2009	30.0 (7.6)	20.0 (8.9)	0	10.0 (6.8)	0	0
	2010	4.4 (2.9)	8.0 (4.9)	15.6 (8.7)	4.0 (4.0)	0	0
	March 2009	52.0 (11.2)	56.7 (27.0)	2.0 (2.0)	0	0	0
	2010	16.9 (10.6)	12.0 (8.0)	1.5 (1.5)	8.0 (8.0)	0	0
Calf	**January 2007**	0	0	0	0	0	86.7 (58.8)
	2009	48.9(17.7)	25.5 (12.6)	11.1 (4.8)	15.0 (15.0)	0	0
	March 2009	51.1 (18.6)	60.0 (0)	8.9 (5.9)	0	0	0
	2010	10.0 (10.0)	33.3 (24.0)	0	13.3 (13.3)	0	0

Parasite aggregation

Both feeding site users and non-users had aggregated or highly aggregated parasite distributions as shown in Fig. 1-3 and indicated by low k values at both study sites (table 3).

Table 3. Estimates of parasite aggregation parameter *k* (*corrected moment estimate*) for feeding station users and non-users in Telemark (2007) and Hedmark (2009 and 2010).

Age class	Month and Year	*Trichostrogylidae spp.*		*Nematodirus spp.*		*Trichuris spp.*
		User	**Non-user**	**User**	**Non-user**	**Non-user**
Adult	**January 2007**					0.047
	2009	1.28	0.70	-[1]	0.20	
	2010	0.15	0.36	0.25	0	
	March 2009	2.13	0.57	0	-[1]	
	2010	0.12	0.26	0	0	
Calf	**January 2007**					0.13
	2009	0.81	0.77	0.50	0	
	March 2009	0.74	-[2]	0.15	-[1]	
	2010	0	0.31	-[1]	-[2]	

[1]None infected.

[2]No *k* estimate possible as no positive values

BLOOD SAMPLES

The prevalence for *Setaria sp.* in the 40 individuals examined from Hedmark was 0 %.

Discussion

In this first study of gastrointestinal parasites in moose and supplementary feeding in Scandinavia, I found three gastrointestinal nematode species based on egg morphology, i.e. *Trichostrongylidae spp.*, *Nematodirus spp.* and *Trichuris spp*. All three species were found in Hedmark while in Telemark I only found *Trichuris spp.* which occurred at low prevalence and intensity. Contrary to my two hypotheses, supplementary feeding had no effect on the prevalence of any of the three parasite species or the intensity of *Trichostrogylidae spp*. infection. I was not able to fit models for intensity of *Nematodirus spp.* or *Trichuris spp.* infection because of the low prevalence and large number of zeros. I found no evidence of co-infection between *Trichostrogylidae spp.* and *Nematodirus spp.*

I found significantly higher prevalence and intensity of *Trichostrogylidae spp.* infection in Hedmark moose in 2009 compared to 2010 and the intensity of infection decreased with increasing body mass. Other studies support my findings; weight gains in semi-domesticated reindeer in northern Norway correlated negatively with nematode intensity (Arneberg et al. 1996), poor physical condition together with high density was linked to high intensity of abomasal nematodes in wild reindeer (Bye 1987), female adult caribou (*Rangifer tarandus)* showed significant decrease in body weight with increasing nematode abundance (Hughes et al. 2009), and wild bovid species unable to maintain adequate nutrition were less able to manage GI parasite infections (Ezenwa 2004a). Animals in poorer condition are less resilient and resistant to infection while those able to maintain adequate nutrition manage to cope with GI parasite infections. In 2009 the snow cover had several layers caused by periodically milder weather during winter months making it more strenuous for moose to move around. This could have increased stress levels, resulting in a higher intensity of *Trichostrogylidae spp.* infection, especially by late winter. Both prevalence and intensity of *Trichostrogylidae spp*. increased from January to March, but month was not significant in my models, any effect being masked by a decrease in body mass between January and March; i.e. body mass was on average 7% higher in January than March. *Trichostrogylidae spp*. causes enteritis in young domestic animals with heavy infections, but no disease is known in moose (Hoberg et al. 2001, Lankester and Samuel 2007). In North American moose, *Trichostrogylidae spp.* are generally uncommon and prevalence and intensity of infection are low (Lankester and Samuel 2007).

As with most parasitic nematodes (Newey et al. 2005), GI nematodes have direct life cycles. Eggs are defecated with faecal pellets, they go through several larval stages before maturing and they have no intermediate host. Infective larvae are incidentally ingested by susceptible host animals while they feed on vegetation (Soulsby 1983, Lankester and Samuel 2007). Before maturing into egg-producing adults and depending upon environmental stimuli, GI nematode larvae of ruminants may undergo arrested development in abomasal mucosa (i.e. hypobiosis) during winter months (Armour and Duncan et al. 1987, Halvorsen et al. 1999). They first mature in late winter and spring following the seasonality of GI nematode biology. Such a lifecycle might explain the lack of difference between feeding station users and non-users as infection occurs after the end of the winter feeding season. Halvorsen et al.'s (1999) study on parasitic nematodes in Svalbard reindeer provides evidence for arrested development as well as continued transmission under arctic winter conditions. The continued transmission was believed to be a result of reindeer ingesting larvae that had already developed to the infective stage during the summer and autumn rather than developing at below 0° C temperatures. They thought the temperature-dependent development of trichostrongyle nematodes was an adaptive trait allowing transmission through cold winters as long as host animals were available. Continued transmission could explain the increase in prevalence of *Trichostrogylidae spp.* and *Nematodirus spp.* and intensity of *Trichostrogylidae spp.* as winter proceeded. Clearly, if moose in Hedmark continued to ingest infective larvae during winter, my findings suggest that transmission did not occur exclusively at feeding stations as both feeding station user and non-user moose had higher intensity of *Trichostrongylidae spp.* in March than in January.

Behavioral patterns like selective defecation and selective foraging could further explain why I found no support for my hypotheses if moose, like other ungulates (Booth 1981, Murray 1991, Quale and Kershaw 1996, Hester et al. 1999), actually do not defecate while feeding but have spatial separation of foraging and ruminations bouts and dung deposition (van der Wal et al. 2000, Ezenwa 2004b). This could reduce or even prevent contamination of feeding stations as infective larvae cannot relocate far (Soulsby 1983). However, faecal pellets are found close to the silage bales (K.M. Mathisen, pers. comm.) and pellet abundance was extremely high at 12.5m from feeding sites (van Beest et al. 2010).

Adult moose had a higher intensity of *Trichostrogylidae spp.* infection than calves. I anticipated the opposite if the immune response of moose can be expected to develop as that of sheep and cattle. However, it has been suggested that the high intensity of GI nematode

infection in adult Svalbard reindeer illustrates a weak immune response (Halvorsen et al. 1999, Irvine et al. 2000). Nutrition is important for a proper immune response (Murray et al. 1998, Coop and Kyriszakis 2001, Ezenwa 2004a) and as ungulates can experience malnutrition during or at the end of the winter season (Aguirre et al. 1999, Halvorsen et al. 1999, Stien et al. 2002b, Hughes et al. 2009) their acquired immunity could simply take a longer time to develop than that of domestic animals. Indeed, allocating resources for building up immunity against parasites has a second priority after maintenance of body protein and insurance of growth and reproduction, which ensure animal's short-term survival and long-term genetic success (Coop and Kyriszakis 2001).

When running models for calves only, sex in addition to body mass had a significant effect on intensity of *Trichostrogylidae spp*. infection. No year effect, as observed in adult moose, was found. A reasonable explanation for this was the exclusion of calves in very poor condition from recapturing in March 2009 and capturing calves only in March 2010. Other studies support my findings of female calves having lower intensity of *Trichostrogylidae spp*. infection than male calves (Folstad et al. 1989, Isomursu et al. 2006, Wirsing et al.2002). The immunosuppressive effect of male sex hormones is one possible explanation for the male bias in parasitism.

I found *Nematodirus spp.* both in adult and juvenile moose at low prevalence in Hedmark. Prevalence was significantly higher in January than in March and juvenile moose were more likely to be infected with *Nematodirus spp.* than adults. Low prevalence of *Nematodirus spp.* is consistent with reports from North America (Stock and Barrett, 1983, Hoeve et al. 1988, Lankester and Samuel 2007). Fruetel and Lankester's (1988) findings suggested that *Nematodirella alcidis*, a fairly specific nematode to moose found throughout the circumpolar region, has a lifecycle strategy ensuring production of parasite eggs throughout the year and that those eggs are resistant to freezing and desiccation. If the species I discovered in moose in Hedmark has a similar lifecycle strategy, high prevalence is probably unnecessary for its survival in a moose population. On the other hand, *Nematodirus spp.* could simply have low egg production in moose in winter when egg development and survival of larvae are likely to be reduced due to snow cover and temperatures below 0° C (Stien et al. 2002a). Furthermore, if *Nematodirus spp.* infection epidemiology and the parasite's lifecycle in moose is similar to that in sheep (Soulsby 1982), infection in juvenile moose is short-lived as they develop immunity becoming highly resistant to re-infection. My findings were consistent with this.

I found *Trichuris spp.* only in one adult moose in Hedmark in January 2009. On the contrary, in Telemark, it was the only GI parasite species discovered. I have no clear explanation why only one moose had the parasite in Hedmark. If inter-specific competition exists, certain parasite species could thrive better than others and over time predominate. This could also explain the high prevalence of *Trichostrogylidae spp*. in Hedmark and its nonexistence in Telemark. *Trichuris spp.* is not commonly reported in moose and is normally of no concern in wild moose (Lankester and Samuel 2007). It can cause bloody diarrhea, especially in young animals (Hoeve et al. 1988) and is likely to increase in intensity in captive or farming situation (Clauss et al. 2002, Lankester and Samuel 2007). In Europe, captive moose with *Trichuris spp.* infections have been connected to grazing on pasture and Wasting Syndrome Complex, a condition of chronic diarrhea and body mass loss (Clauss et al. 2002). Faecal eggs are long-lived and resistant to a range of weather conditions. Sheep over eight months of age demonstrate an age resistance to infection and acquired immunity develops quickly after infection (Soulsby 1983).

In my study the negative binomial dispersion factor *k* (corrected moment estimate) was low for *Trichostrogylidae spp*., *Nematodirus spp.* and *Trichuris spp.* indicating a high degree of aggregation. This is a common and expected phenomenon for parasite infections in both wild and domestic animals (Barger 1985, Shaw et al. 1998). While a high degree of aggregation is one of the suggested criteria for stable parasite host population dynamics (Anderson and May 1978, May and Anderson 1978, Tompkins et al. 2002), a random (non-aggregated) distribution of parasites within the host population and negative effect of parasites on host fecundity without direct effect on mortality, could on the contrary, lead to instability in host population dynamics. This further emphasizes the importance of better knowledge on parasite lifecycle and epidemiology in moose.

In this investigation, I did not found *Setaria spp*. in the 20 moose sampled in January 2009. The sample size was probably too small to say anything conclusive, but it is possible that the parasite is nonexistent in Hedmark. In Finland the prevalence of *Setaria tundra* was 1.4 – 1.8% in a sample of 324 moose. 212 of the samples were collected from reindeer herding areas, commonly harboring *Setaria tundra* (Laaksonen et al. 2007, 2009) and 112 outside the southern border of the reindeer herding area. The sampling was done in a follow-up period (2004-2006) after a peritonitis outbreak in semi-domestic reindeer (*Rangifer tarandus tarandus*) caused by *S. tundra* in northern Finland in 2003.

I found no *Salmonella spp*. bacteria in Hedmark or Telemark faecal samples. This group of bacteria is uncommon in domestic animals in Norway (Veterinærinstituttet 2008) making it even more unlikely for game animals to harbor it. Salmonella spp. bacteria are zoonotic bacteria causing serious enteritis in humans (Folkehelseinstituttet 2010). If Salmonella spp. was in the silage moose fed on during winter, moose could get infected and the bacteria could be transferred to meat and again to humans during the slaughtering process. This does not appear to be the case.

It would have been interesting to further investigate the annual cycle of GI nematode abundance in adult and juvenile moose. Spring is believed to be the most important time period for parasite transmission (Soulsby 1983), but GI nematode life-histories in moose could also differ from each other, as in the life-histories of the two most abundant trichostrongyle species in Svalbard reindeer (Irvine et al. 2000). Depending on the nematode species, there were seasonal differences in egg output and adult worm abundance, age-related differences in rate of infection of hosts, and spatial differences in species profile. The prevalence and abundance ought to be related to habitat (habitat cover), seasonal ranges (Bordes et al. 2009) and host age, condition and weight (Dobson and Grenfell 1992, Aguirre et al. 1999, Halvorsen et al. 1999) as these heterogeneities are known to affect parasite aggregation. Such factors have yet to be investigated in the parasites of Scandinavian moose. Increasing temperature due to climate change has also been indicated as a reasonable cause for increase in parasite and other pathogen related diseases (Lenarz et al. 2009). A good understanding of parasites' annual lifecycle and epidemiology in wildlife species may therefore become even more important in the future.

Acknowledgements

I would like to thank Jos Milner, my supervisor, for her advice, support and comments while going through the drafts of my thesis: KIITOS. I would also like to thank my co-supervisors Sauli Laaksonen and Antti Oksanen (DVM, PhD, Finnish Food Safety Authority (Evira) at Oulu Research Unit, Fish and Wildlife Research Unit) and the staff at Evira for helping me with the parasite analyses and making my stay in Finland, my home country, pleasant and comfortable. This study was financially supported by Hedmark county governor.

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