The moose (Alces alces) population in Scandinavia began to rise in the 1970s, peaking in the mid-1980s in Sweden. With few large predators at that time, it was not unusual to find dead or sick animals (Hörnberg 2001, Stéen et al. 2005), and in the 1980–1990s, high mortality was noted in both Swedish and Norwegian moose, as well as in semi-domestic reindeer (Rangifer tarandus). A previously unknown disease, elaphostrongylosis (Stéen and Rehbinder 1986, Stuve 1986), was reported in the 1980s and sick animals were characterized by locomotive abnormalities such as ataxia, incoordination, swaying of the hindquarters, broad and stamping gait, and a certain way of hypermetria that suggested paralysis of ascending proprioceptive nerve fibers (Stéen and Roepstorff 1990). A previously undescribed species of elaphostrongyline nematode with a dorsal-spine larva, Elaphostrongylus alces (Stéen et al. 1989) was invariably associated with sick and dead moose (Stéen and Rehbinder 1986).

Parasites of the genera Parelaphostrongylus and Elaphostrongylus belong to the subfamily Elaphostrongylinae (Protostrongylidae, Metastrongyloidea, Nematoda). Species of the genus Parelaphostrongylus (P. tenuis, P. odocoilei, P. andersoni) affect the central nervous system (CNS) and skeletal muscle
fasciae of Nearctic cervids in North America including white-tailed deer (*Odocoileus virginianus*), black-tailed deer (*O. hemionus hemionus*), mule deer (*O. h. columbianus*), and occasionally wapiti (*Cervus canadensis*) and moose (*Alces alces* spp.). Species of *Elaphostrongylus* (*E. alces, E. cervi, E. panticola, E. rangiferi*) affect the CNS, the peripheral nerve system (PNS), and the skeletal muscle fasciae in Eurasian cervids including moose, red deer (*Cervus elaphus*), maral deer (*C. e. sibiricus*), roe deer (*Capreolus capreolus*), and reindeer (Lankester 2001). In the New World, as in the Old World, central nervous disorders and mortality occur in wild cervids infected with elaphostrongyline nematodes (Anderson 1964, Lankester 2001, 2010). Representatives of the genera *Elaphostrongylus* and *Parelaophostrongylus* are also harmful to domestic ruminants (Lankester 2001).

Both *Elaphostrongylus* spp. and *Parelaophostrongylus* spp. develop from the first to third larval stage (L₁–L₃) in their gastropod intermediate host, and develop from the L₃ to the adult (L₅) stage in their cervid (final) host (Olsson et al. 1998, Olsson 2001). Specific identification of adult protostrongylids and first-stage larvae (L₁) in feces of Swedish moose was a result of multiple studies. The morphology of *E. alces* was initially described by Stéen et al. (1989) and (Stéen and Johansson 1990), and subsequent comparison of specific proteins in protostrongylid L₁ indicated that L₁ and adult *E. alces* had the same protein pattern in moose, but differed from the L₁s and adult protostrongylid parasites in other wild ruminants (Stéen et al. 1993). Experimental infection of captive moose indicated that L₁ collected from wild moose caused elaphostrongylosis, and L₁ excreted from infected and sick moose and transmitted to terrestrial snails (*Arianta arbustorum*) in which larvae develop (Lankester et al. 1998), were identified as *E. alces* using genomic DNA (Gajadhar et al. 2000) and single-strand conformation polymorphism (SSCP) analysis (Chilton et al. 2005, Huby-Chilton et al. 2006). Collectively, these studies indicate that protostrongylid larvae in Swedish moose are *E. alces*. Given the prevalence and deleterious effect of this disease, our objective was to determine if *E. alces* is related to age, sex, condition, and geographic distribution of moose in Sweden.

**STUDY AREA**

Sweden was divided into 6 regions from the far north (69°03′36″N 20°32′55″E) to the far south (55°20′13″N 13°21′34″E) to determine the distribution and prevalence of elaphostrongylosis (Fig. 1). Sweden is sheltered by the Scandinavian mountains and has a continental climate with large differences in temperature and precipitation between summer and winter, and a relatively small amount of precipitation (Swedish Meteorological and Hydrological Institute [SMHI]). Summer temperatures are similar to those in North America and Asia at similar latitude, although due to the Gulf Stream, winter in Sweden is typically milder (SMHI 2015).

**METHODS**

Hunting begins on the first Monday of September in northern Sweden, on the second Monday of October in the south, and seasons end in December or January (Swedish Association for Hunting and Wildlife Management 2015). Prior to the hunting seasons (1986 and 1987), we sent hunters report cards (hunting site, sex, and approximate age) and wrapping materials to pack body parts (i.e., lungs, feces, spinal cords, and mandibles). Moose carcasses and parts (n = 1137) were examined in 5 consecutive years (1985–1989); 896 (79%) were associated with harvested moose (1986, 1987, 1989) and 241 (21%) were from non-harvested animals (i.e., euthanized or found dead; 1985–1989).
We used 1020 lungs and 1084 fecal samples to identify presence of elaphostrongylid L1, 655 spinal cords (membranes) to identify presence of adult worms, and 636 mandibles to measure fat content (Table 1).

Age was determined by dental wear (Gasaway et al. 1978) or from information provided by hunters; 151 animals were not aged due to lack of information. Five age classes were established: 1) calves were ≤12 months), 2) yearlings were >12 and ≤24 months, 3) young animals were >24 months and ≤5 years, 4) middle-aged animals were >5 and ≤9 years, and 5) old animals were >9 years. Sex was determined from the whole carcass or hunter information.

Evaluation of body condition was by visual inspection, location, and appearance of body fat (n = 948), and/or by measuring fat content (%) in mandible bone marrow (n = 636; Engelsen Etterlin et al. 2009). Three categories of condition were established:
normal, poor (below normal), and emaciated (lack of adipose tissue). The fat content in bone marrow was measured with standard techniques under specified assay conditions and techniques (NMKL No 131, Nordic Committee on Food Analysis 1989) and also used to assign condition: normal = 75–94%, poor = 16–<75%, and emaciated = 0.4–<16% fat content. Assigning condition from visual inspection (without measuring fat content) was considered reliable because of the strong correlation between the condition category assigned from fat measurements and visual inspection of the same animals ($R_s = -0.801, P < 0.001, n = 592$).

Bodies/parts were inspected for adult *E. alces* worms and L₁ with necropsy procedures described previously (Stéen and Rehbinder 1986, Stéen et al. 1997, 1998) and included examination of muscle fasciae, the cranial cavity, brain, and spinal cord membranes and epidural space of the spinal cord (Stéen and Rehbinder 1986, Stéen et al. 1997). Lungs from all animals were palpated and inspected for nodules, and 20 g samples of minced lungs and feces were processed to detect L₁ (Baermann 1917). L₁s were identified as protostrongylids quantified in a counting chamber under a stereo microscope, expressed as larvae per gram of wet feces (lpg), and classified into 7 levels of relative abundance ranging from none (0) to heavy (6) (National Veterinary Institute, Sweden). There were 4 categories of infection: 0 = uninfected, 1 = in the epidural space but not in lungs or feces, 2 = in lungs but not feces, and 3 = in feces. Animals were categorized as either infected or uninfected (presence or absence of L₁ and/or *E. alces* worms) for certain statistical comparisons (e.g., sex or age groups, prevalence in population or region).

### Data management and statistics

Data were tested for normal distribution and seasonal variation, and if not normally distributed, normality was achieved with log-transformation. A peak function analysis was used to identify the best fit to the relationship between bone marrow fat and season (TableCurve software, Systat 2002). A mean value was calculated for harvested animals and this value was applied together with the individual values for remaining
animals. The residuals for all animals were calculated (harvested moose were not combined as above), and adjusted values were calculated by adding the residual to the common mean. This produced a few values >100% that were not further corrected in subsequent analyses. Bone marrow fat (adjusted for seasonal variation) was subsequently analysed using generalized linear models. Body condition was also analysed with generalized linear models, modeling the probability of being in normal condition (see above) assuming a binary distribution of the response variable. The total parasite infection or parasites found in either the lungs, feces, or in the epidural space were similarly corrected, and the probability of being infected was tested with respect to 3 predictors (age, sex, region).

The mean category of infection (0–3) in each age group was calculated to illustrate the relationships among age (mean age of group), category of infection, and body condition. These values were used to develop a contour graph using SigmaPlot software (Systat 2008) where body condition, age group, and infection category were interpolated.

**RESULTS**

**Infection, age and sex**

Age of moose was skewed towards young animals (Table 1), and age in the two groups (harvested and unharvested) was not distributed evenly. Unharvested moose were older than those harvested for combined age classes, calves, and by sex (Table 3). The average age of harvested animals (n = 761) was 10.4 months (95% CI = 9.6–10.4; range = 0–15 years), and 22.3 months (CI = 17.4–28.4; range = 0–20 years) for unharvested animals (n = 227). Females were older in the yearling, middle-aged, and combined age groups.

A slight majority (57%) of the harvested sample (n = 896) was infected with L1 and/or adult *E. alces* worms. The prevalence was similar between sexes in each age class for L1 in lungs, L1 in feces, and adult worms in the epidural space of the spinal cord (Fig. 2). There was a tendency (*P* = 0.074) toward higher prevalence in males than females in the young age class. Worms were found in the epidural space of the spinal cord in animals 3 months to 2 years old, but not in animals 3 to 9 years old; worms were found in a single 10-year old moose. The abundance of L1 in lungs (n = 784) was high in calves and yearlings, lower at 3–4 years of age, and minimal in adults.

Nearly the entire sample (98%) of unharvested moose (n = 241) was infected with *E. alces* (Fig. 3). Worms were found in the epidural space of the spinal cord in 3 month to 4 year-old animals. The average age of
infected calves was 4.8 months (95% CI = 4.7–4.9). No worms were found in the epidural space of the spinal cord in 5–9 year-old moose, but worms reappeared at 10–16 years of age.

The prevalence of adult worms in the epidural space of the spinal cord was 36% in the combined data (harvested and unharvested, n = 655); the prevalence of L1 in lungs (n = 1020) and feces (n = 1084) was 64 and 53%, respectively. The prevalence (worms/L1) was 66% overall; 88% in old moose, 74% in yearlings, 67% in calves, 55% in young, and 48% in middle-aged animals. There were differences (P < 0.001) in frequency of infection among age groups; the oldest animals had the highest frequency of infection (L1) and the middle-aged the lowest.

The frequency of worms in the epidural space of the spinal cord was high in calves/yearlings, leveled out at 4 years, and then was not identified until 10–16 years at low frequency. The abundance of L1 in lungs of old animals was at the highest level (6).

**Body condition**

Body condition of harvested animals (n = 981) was either normal (40% overall, 24% calves) or poor (59%, 75% calves). In unharvested moose (n = 227), body condition was normal in 38% overall, with calves and old animals lower; 25% calves, 45% young, and 29% old animals were in normal condition. For all moose, body condition and category of infection were correlated (R = 0.215, P < 0.001). In separate age classes, this
correlation was found in yearlings ($R_s = 0.262$, $P < 0.001$, $n = 239$), young ($R_s = 0.463$, $P < 0.001$, $n = 67$), middle-aged ($R_s = 0.441$, $P = 0.002$, $n = 47$), and old animals ($R_s = 0.456$, $P = 0.003$, $n = 40$), but not in calves ($R_s = 0.054$, $P = 0.239$, $n = 471$). For all moose, body condition was correlated inversely with category of infection ($R_s = 0.084$, $P = 0.025$, $n = 721$); separate correlations were found in yearlings ($R_s = 0.213$, $P = 0.002$, $n = 227$) and young animals ($R_s = 0.398$, $P = 0.011$, $n = 40$). Figure 4 illustrates the probability of normal body condition relative to age and category of infection, indicating that calves have poor body condition regardless of category of infection, and that some middle-aged animals have normal body condition despite high abundance of $L_1$ in feces. Old individuals were generally in normal body condition if not infected, although few were without infection.

Bone marrow fat content ($n = 615$) varied annually (Table 4, Fig. 5). On average, harvested animals had higher fat content (93%, $95\%$ CI = 91 – 96) than unharvested animals (70%, CI = 66 – 75) with values corrected for time of year, sex, and age class (Table 4). In a combined sample, a negative correlation was found between bone marrow fat content and category of infection ($R_s = -0.212$, $P < 0.001$, $n = 635$). This negative correlation was found in calves ($R_s = -0.131$, $P = 0.020$, $n = 319$), yearlings ($R_s = -0.223$, $P = 0.002$, $n = 193$), young ($R_s =

*Sexes differ by age class (all causes of death included).
†Includes individuals not sexed.
§Sexes differ with all age classes combined.

Table 3. Age in months (mean and 95% CI) of Swedish moose examined for *Elaphostrongylus alces*, 1985–1989. The column to the far right gives level of significance between harvested and euthanized + dead moose (for the last three rows the t-tests are performed on log transformed data; the data presented in the table are back-transformed values). If all sexed animals are combined, the sexes differed in age ($P < 0.05$).
−0.618, \( P < 0.001, n = 46 \), and old (\( R_s = -0.736, P < 0.001, n = 21 \)), but not middle-aged moose (\( R_s = -0.319, P = 0.062, n = 35 \)).

**Time of infection**

The earliest identification of a calf diagnosed with elaphostrongylosis was at \( \sim 1.5 \) months on 21 July in Region 3, County of...
The abundance of $L_1$ was category 6 in the lungs and 4 in feces, and the calf was in normal body condition. The earliest calf death where worms were found in the epidural in the spinal cord was on 10 October (~4 months old) in Region 2, County of Värmland; the abundance of $L_1$ was category 6 in the lungs and 3 in feces. Worms were first found in harvested calves on 15 September (~3 months old).
Table 4. Analysis of bone marrow fat (%) and body condition of harvested and unharvested moose, Sweden, 1985–1989. Values are mean ± SE with sample size in parentheses. Pair-wise comparisons (t-values) of harvested and unharvested animals are provided in each age group (raw). Means denoted by the same letter in each column (percent fat and body condition separately) were not different (P < 0.05). The values for percent fat are adjusted for time of year causing certain values to be >100% (see Data management and Statistics).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Age class</th>
<th>Harvested</th>
<th>Euthanized or dead</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone marrow fat (%)</td>
<td>Calves</td>
<td>83.6 ± 0.8a (270)</td>
<td>60.5 ± 1.8a (48)</td>
<td>t = 11.6 P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Yearlings</td>
<td>97.1 ± 0.9b (182)</td>
<td>69.9 ± 3.8b (11)</td>
<td>t = 6.88 P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Young</td>
<td>98.7 ± 2.1b (36)</td>
<td>83.6 ± 4.0c (10)</td>
<td>t = 3.32 P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Middle aged</td>
<td>99.6 ± 2.3b (30)</td>
<td>77.4 ± 5.7bc (5)</td>
<td>t = 3.62 P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Old</td>
<td>104.6 ± 5.7b (5)</td>
<td>63.3 ± 3.2ab (16)</td>
<td>t = 6.34 P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>All animals</td>
<td>96.7 ± 1.3 (523)</td>
<td>70.9 ± 1.7 (92)</td>
<td>t = 11.6 P &lt; 0.001</td>
</tr>
<tr>
<td>Probability of normal condition</td>
<td>Calves</td>
<td>0.17 ± 0.03a (368)</td>
<td>0.25 ± 0.06a (102)</td>
<td>z = 0.81 P = 0.420</td>
</tr>
<tr>
<td></td>
<td>Yearlings</td>
<td>0.48 ± 0.04b (204)</td>
<td>0.30 ± 0.12c (35)</td>
<td>z = 1.19 P = 0.235</td>
</tr>
<tr>
<td></td>
<td>Young</td>
<td>0.50 ± 0.11bc (40)</td>
<td>0.47 ± 0.15b (27)</td>
<td>z = 0.25 P = 0.800</td>
</tr>
<tr>
<td></td>
<td>Middle aged</td>
<td>0.74 ± 0.12c (30)</td>
<td>0.64 ± 0.21b (17)</td>
<td>z = 0.34 P = 0.734</td>
</tr>
<tr>
<td></td>
<td>Old</td>
<td>0.84 ± 0.17abc (6)</td>
<td>0.12 ± 0.06ab (34)</td>
<td>z = 2.53 P = 0.012</td>
</tr>
<tr>
<td></td>
<td>All animals</td>
<td>0.33 ± 0.02 (648)</td>
<td>0.29 ± 0.05 (215)</td>
<td>z = 0.56 P = 0.578</td>
</tr>
</tbody>
</table>

Fig. 5. The dependence of moose body condition (expressed as percent fat) on day of the year (Julian date). In this analysis harvested moose are represented by a single value (Julian date = 292.80; percent fat = 66.05). The line in the figure is the estimated peak function; a Lorentzian peak function; \( y = 45.58 + \left[ -34.65/(1 + (((X - 115.05)/62.57)^2)) \right] \). \( F_{3,89} = 6.23, r^2 = 0.174, P < 0.001. \) The peak are estimate to Julian date = 115.05, which is 25 April.
The abundance of L1 was category 0 in the lungs and 6 in feces. First stage larvae (infection intensity = 6) were found in lungs from 14 August (~2 months old) to 4 June the following year (~12 months old). Abundance of L1 in calves ranged from categories 1–6 by 2 months old, and the lung infection remained high; 81% had an L1 abundance category of 4–6 in the first year. The excretion of larvae began at a low level (2) on 14 August, and calves continued to excrete larvae throughout the first year at all levels of abundance (1–6).

The prevalence of infection in harvested moose (n = 896) differed among regions (Fig. 6), ranging from 13% in southernmost Region 6 to 56% in Region 3 (Fig. 1 and Table 2). Infection was most prevalent in central Sweden, least prevalent in southern Sweden, and similar (P < 0.05) in southern and northern Sweden.

**DISCUSSION**

Although parasites at low abundance are generally less harmful to their host, when the host population increases rapidly, as with Swedish moose in the 1970–80s (Hörnberg 2001, Stéen et al. 2005), an increasing risk to the individual and host population is possible (Toft 1991). The proportion of elaphostrongylosis (symptoms of nervous disorder and/or emaciation) varies among age-classes in moose, with young animals more prone to illness (Stéen et al. 2005). Similarly, we found that *E. alces* worms located in the epidural space of the spinal cord were more prevalent in calves and yearlings, and only occasionally found in adults. The high abundance measured in young animals may simply reflect that the Swedish moose population is skewed towards young animals (Sand et al. 2011). Conversely, abundance of L1 in lungs and feces was highest in old moose, and lowest in young and middle-aged moose.

Both Stuve (1986) and Stéen et al. (2005) suggested that *E. alces* most frequently infects males and young animals; however, we found no difference in the abundance within the epidural space, lungs, or feces between sexes or age groups of harvested moose, only a tendency toward males in the young age group. Similarly, male reindeer calves with dominant mothers had higher abundance of *E. rangiferi* than female calves, and it was suggested that because these calves had better access to forage, they were at greater risk of ingesting infected gastropods (Halvorsen 1986a). Calf weight is dependent on summer browse availability in a

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<table>
<thead>
<tr>
<th>Parasite location</th>
<th>N</th>
<th>Mean age ± SD</th>
<th>Min age</th>
<th>Max age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidural</td>
<td>281</td>
<td>140.8 ± 20.0</td>
<td>50</td>
<td>215</td>
</tr>
<tr>
<td>Lung</td>
<td>348</td>
<td>140.2 ± 20.8</td>
<td>50</td>
<td>215</td>
</tr>
<tr>
<td>Feces</td>
<td>416</td>
<td>141.0 ± 17.6</td>
<td>101</td>
<td>215</td>
</tr>
</tbody>
</table>
Cow’s home range, with access to and quality of forage related to its relative status (Saether and Heim 1993). Stuve (1986) attributed the difference in infection rate between sexes in older moose to physiological changes associated with the rut, as suggested with reindeer (Halvorsen 1986b).

A novel finding of our study was that L1 were found in lungs and feces of calves by 21 July, and adult worms in the epidural space by 15 September, or ~50–100 days after birth (Broberg 2004, Malmsten 2014). This prepatent period aligns with experimental infections of E. alces in moose in which patent infection was realized 39–73 days post-infection (Stéen et al. 1997). Because calves sample vegetation in the first days of life to promote development of rumen microbes (Syroechkovsky et al. 1989), their potential to exposure to E. alces L3 is almost immediate. Not surprisingly, adult E. alces were identified in the epidural space of the caudal vertebral canal in 2 other calves harvested in September (Handeland and Gibbons 2001). Further, calves and yearlings were most frequently infected in the epidural space of the spinal cord which seemingly corroborates that moose shed most E. alces L1 during their early years, after which a sharp drop in larval shedding and low numbers of adult worms in older animals occur (Stuve 1986, Stéen et al. 2005).

In both harvested and unharvested moose, E. alces worms were found in the epidural space of the spinal cord of animals aged 3–4 months to 4 years, not in middle-aged animals, and again at 10–16 years. Conversely, high levels of larvae were found in lungs and feces irrespective of age. We believe that the low frequency of worms in older animals, despite having L1 in lungs and shed larva, is due to migration from the CNS/PNS into the muscle fasciae, as with some other elaphostrongylins (Lankester 2001).

The pattern of E. alces adults migrating out to the muscle fasciae, presumably due to an immune response in the epidural space (Stéen et al. 1997, 1998), differs somewhat from that of E. rangiferi, E. cervi, and P. tenuis. The latter are believed to remain in the CNS as adult worms during their entire life (in the subdural or subarachnoid space, inside the meninges), although E. rangiferi also migrates to the muscle fasciae (Hemmingsen et al. 1993). E. rangiferi, E. cervi, and P. tenuis may realize an immunological harbor within the CNS, as might P. andersoni that is associated with blood vessels and connective tissues where females deposit eggs (Lankester 2001). We hypothesize that E. alces worms are attacked by the immune system in the epidural space, and they migrate to the muscle fasciae where, with lower immunological defense, they deliver most of their larvae.

After ingestion, L3 migrate from the gastrointestinal (GI) tract to the perineal cavity along the mesenchyme nerves, and into the abdominal wall associated with the more posterior lateral nerves. It is likely that E. alces does not need to enter the CNS parenchyma to develop to the 5th stage (adult), as other Elaphostrongylus spp., but remains epidurally-associated with lateral nerves of the PNS and finally migrates to the muscle fasciae (Olsson et al. 1998). The lack of worms in the epidural space of the spinal cord in moose during their prime could be explained by this migration; however, it could also reflect an immune response to prevent reinfection as described for P. andersoni that realizes declining larval output as deer age with few adult worms in deer >1 year old. Further, repeated infection in white-tailed deer resulted in sharp decline in larval numbers and a strong cellular response to adult worms (Lankester 2001). Worms in the epidural space of older moose could simply be a reinfection associated with a weaker immune system, or an initial infection. Whether some L3s migrate directly to muscle...
fasciae without being associated with neural tissue is unknown.

Infected animals, on average, had lower body condition than uninfected animals except for middle-aged animals in their prime. Calves were in poorer condition regardless of category of infection (as expected for young, growing animals), middle-aged were likely in normal condition despite high shedding rate of L1 in feces, and old individuals were in normal condition if uninfected. Thus, infection, not age per se, seemed to reflect relative body condition. However, individual variation of immunological response to the parasite presumably exists because some individuals die young, others remain in normal condition through prime, and old animals are increasingly susceptible.

In contrast with E. alces, no protostrongylid L1 of E. cervi were recovered from Iberian red deer fawns (Cervus elaphus hispanicus) (Vicente and Gortázar 2001). Prevalence of E. cervi L1 increased with age of deer (Vicente et al. 2006) which is opposite to our findings with E. alces in moose; both had higher infection rates in young males than females. The E. cervi pattern corresponds with that in reindeer in which E. rangiferi infects the host late in the season, remaining at the same intensity for at least 3 years (Halvorsen et al. 1985).

It appears that E. cervi and E. rangiferi have more similar and longer evolutionary relationships to each other and their respective hosts than E. alces. Moose have a long, independent evolutionary history from the Alceini and the Plio-Pleistocene, suggesting a peculiar adaption and habitat restriction of the species (Niedziałkowska et al. 2014), and presumably, a relatively short evolutionary period with E. alces that could be less adapted with its host than E. cervi and E. rangiferi. It is possible that E. alces is more pathogenic to its host because both harvested and unharvested moose of below normal or emaciated body condition were infected with E. alces. In 2-year old moose, Stuve (1986) found that infected moose were lighter (carcass weight) than uninfected moose, yet conversely, Stéen et al. (1997) found that moose experimentally infected with E. alces retained normal weight when fed ad libitum. It remains unclear, however, if poor body condition is an indirect or direct effect of the parasite, that emaciation is either directly caused by an inflammatory response due to an epidural localization, or that elaphostrongylosis causes locomotor disorders making it difficult to move and feed (Stéen and Rehbinder 1986, Stéen and Roepstorff 1990, Stéen et al. 2005).

In summary, different morphology (Stéen et al. 1989, Stéen and Johansson 1990, Gibbons et al. 1991, Lankester et al. 1998), genetics (Gajadhar et al. 2000, Chilton et al. 2005, Huby-Chilton et al. 2006), location (Stéen et al. 1997, 1998) (epidural for E. alces, subdural/subarachnoid for E. rangiferi), and life span and host age relationships with infection (Lankester 2001) suggest different, and perhaps, ongoing evolutionary adaption in Elaphostrongylus species with their hosts. Of further consequence is that rising temperatures, and a warmer and wetter climate are predicted to increase habitat, distribution, and abundance of mollusk hosts (Halvorsen and Skorping 1982, Halvorsen et al. 1985), which in turn could lead to higher infection rates in cervids (Handeland and Slettback 1994, Halvorsen 2012). Although moose are not necessarily in poor condition when infected with E. alces, condition and parasite abundance were correlated. We therefore suggest continued surveillance of this disease and its specific consideration in management of moose in Sweden.

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