High winter loads of Oestrid larvae and *Elaphostrongylus rangiferi* are associated with emaciation in wild reindeer calves

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**ABSTRACT**

The Oestrid flies *Cephenemyia trompe* and *Hypoderma tarandi* and the nematode *Elaphostrongylus rangiferi* are important parasites of *Rangifer* spp. The larvae of Oestrid flies develop in the throat (*C. trompe*) and skin (*H. tarandi*) of their host during winter while *E. rangiferi* develop in the CNS. Oestrid pupation, and development of *E. rangiferi* larvae from first- (L1) to infective third-stage in the environment during summer is highly temperature dependent. We investigated the possible negative effects of these parasites on the winter body-condition of wild reindeer calves. Two year-classes (generations) of calf, born in a warm (2014) and cold (2015) summer respectively, were examined for changes in body condition between autumn and spring, in relation to the parasite load determined in the spring. The body condition in the autumn was assessed as carcass weight, while the body condition in the spring was assessed as carcass weight, supplemented by an evaluation of fat reserves in various bodily locations. Oestrids were counted directly whereas the *E. rangiferi* quantification was based on faecal counts of L1 larvae. The abundance of infections for Oestrids and *E. rangiferi* were significantly greater in the 2014 generation than in the 2015 generation. The mean carcass weight decreased between autumn and spring for the 2014 generation but increased in the 2015 generation. Emaciation in the spring was documented (fat reserve evaluation) in 42% and 7% of calves in the 2014 and 2015 cohorts, respectively. There was a significant correlation between high parasite load and the probability of emaciation. The mean summer temperature in 2014 was 2.6 °C higher than the mean for 2015, and 1.0 °C higher than the mean for the last 30 years. Our findings suggest that following a warm summer, high loads of Oestrids and *E. rangiferi* may cause emaciation and potentially deaths among the calves.

1. Introduction

The Norwegian wild tundra reindeer (*Rangifer tarandus tarandus*) populations live in a harsh environment in the mountain areas of southern Norway (Fig. 1). During a short summer, the reindeer must build up body reserves to survive a long winter on poor food resources (lichen) covered in snow. Wild reindeer also face challenges related to climate change and human disturbance in their habitats (decades of hunting have led to high wariness of humans). Human disturbance (e.g., hiking trails, cabins, roads) triggers evasive behavior, flock aggregation and reduced pasture utilization (Strand et al., 2006, 2010; Gundersen et al., 2020). Animal aggregation in smaller areas may also increase the infection pressure of important reindeer parasites, such as the Oestridae *Cephenemyia trompe* (throat bots) and *Hypoderma tarandi* (warbles) and the nematode *Elaphostrongylus rangiferi* (brain worm). These parasites develop in the reindeer during winter and may cause loss of body condition and neurological disorders (*E. rangiferi*), especially among calves (Roneus and Nordkvist, 1962; Skjenneberg and Slagsvold, 1968; Cuyler et al., 2012). The development of the larval stages of Oestrids and *E. rangiferi* in the environment requires extended periods of relatively...
Fig. 1. Map of southern Norway showing the location of the 24 Norwegian wild tundra reindeer populations. The Hardangervidda population (No. 16) is marked with brighter tan.
high temperature (Halvorsen and Skorping, 1982; Nilssen, 2006) and in semi-domesticated reindeer flocks in subarctic northern Norway, summer temperature is considered a limiting factor for infection (Halvorsen et al., 1980; Nilssen and Haugerud, 1995). In these areas, outbreaks of clinical elaphostrongylosis occur in autumn and winter, following hot summers (Handeland and Sletbak, 1994). Oestrid L3 larvae (L1) towards the reindeer nostrils (Anderson and Nilssen, 1990) whereupon the larvae enter the nasal cavities. Development to second (L2) and third (L3) larval stages takes place in the retro-pharynx in late winter and spring. Hypoderma tarandi lay eggs in the animal’s coat and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin.

Cephenemyia trompe and H. tarandi are large flies that have obligatory larval development in Raniger spp. Cephenemyia trompe eject first stage larvae (L1) towards the reindeer’s nostrils (Anderson and Nilssen, 1990) whereupon the larvae enter the nasal cavities. Development to second (L2) and third (L3) larval stages takes place in the retro-pharynx in late winter and spring. Hypoderma tarandi lay eggs in the animal’s coat and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin. Development to second (L2) and third (L3) larval stages takes place in the dorsal subcutis between late autumn and following hatching, L1 larvae penetrate the skin.
Fig. 2. Kernel Density Analysis, visualizing the main pasture area of radio-collared females in the wild reindeer population in Hardangervidda during June, July, and August 2001–2017. The darker the color, the larger number of GPS positions recorded. Calculated center at UTM 32V: 426706-6650377 and average altitude at 1283m. The center in the previously used summer area in the west (the mountain of Hårteigen) is marked with an asterisk. The red lines represent summer trails for hikers marked by the Norwegian Tourist Association. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)
Fig. 3. Visualization of fat reserves in the bone marrow of femur (row 1), knee joint (row 2), vertebral canal (row 3), orbital cavity (row 4), and heart surface (row 5). The three categories used are illustrated from left to right: clearly visible white and stiff fat (+); low amounts of visible fat with a soft consistency (+÷); absence of visible fat (serous adipose atrophy) (÷). All figures are from wild reindeer calves killed on Hardangervidda in spring 2015, except for the heart to the bottom right which is from a moose that died from winter starvation.
calculated for the summers 2014 and 2015 and compared with the calculated summer temperatures for other years in the last 30-year-period (1989–2018). The calculations were based on interpolated long-term datasets from all weather stations in Norway (Lussana et al., 2019a, 2019b). These datasets have a 1 × 1km spatial resolution and a daily temporal resolution. Temperature was calculated as the mean temperature for several points in the terrain around the center of the main summer grazing area, adjusted to the average grazing altitude (1283m) (see p. 2.1 and Fig. 2).

Large amounts of snow and temperatures above freezing, which may compact the snow and cause pasture icing problems, can have a negative effect on the body condition in winter (Strand, 2008). To investigate any differences in the snow conditions between the two calf generations, the mean daily temperature and total amount of precipitation (snow) (December–March) were calculated for the winters 2014/15 and 2015/16 (precipitation data was based on in situ observations from Møgen hydrological station on Hardangervidda).

2.5. Statistical analyses

Carcass weights for each calf generation were compared between autumn and spring by a t-test. A wilcoxon-test was used to compare the intensity of Hypoderma and Cephenemyia larval infection (summed) in the two calf generations. Spring carcass weights were then analyzed in a regression analysis as a function of larval intensity. By this analysis, the sum of larvae in each calf was log-transformed to normalize the variance. We also compared carcass weights by testing a two-level parameter of larval infection, high versus less-than high, defining a high level as those calves with a total number of larvae above or equal to the 0.75 quantile. In addition, we used a logistic regression model to analyze the number of emaciated versus non-emaciated calves in the spring. This model was run as a function of year or log intensity of Hypoderma and Cephenemyia infection. The prevalences of E. rangiferi infection in 2015 and 2016 were compared by a logistic regression analysis while abundance of infection (a measure of the level of infection in all hosts, including non-infected individuals) was compared using a wilcoxon test. We also tested all regression models by including potential covariates (sex, Julian day) and compared the models by AIC (Burnham and Anderson, 2002). The potential covariates did not reduce the AIC-value of the basic regression and no significant effects were found. Statistical analyses were performed in R version 4.0.3 (R Core Team, 2020).

3. Results

3.1. Body condition

In total, 50 calves fulfilled the sampling criteria. The number, sex and carcass weights for calves examined in autumn and spring from each calf generation are given in Table 1. The table also shows the results of the fat score evaluation (see Fig. 3) for the calves examined in spring. Fat reserves on the heart appeared last to disappear, with all but one calf displaying intact (white and stiff) fat reserves on the heart surface. For the 2014 generation, there was no significant difference in carcass weight between autumn and spring (p = 0.60), while there was a tendency towards higher spring than autumn carcass weights in the 2015 generation (p = 0.13). The number and percentage of calves classified as emaciated was markedly higher (p = 0.06) in the spring of 2015 (42%), compared to spring 2016 (7%).

3.2. Parasitological findings

3.2.1. Cephenemyia trompe and Hypoderma tarandi

Eighty-three % (10/12) and 75% (9/12) of the calves shot during the autumn hunt in 2014 and 2015, respectively, displayed L1 larvae of C. trompe in the nasal mucosa (Fig. 4). The number of larvae was generally higher in the autumn of 2014, with six calves having >50 larvae, whereas all calves examined in the autumn of 2015 had <30 larvae.

All calves examined in the spring of 2015 and 2016 carried C. trompe larvae in the pharynx (Fig. 5) and H. tarandi larvae in the subcutis of the skin (Fig. 6). The results are summarized in Table 2. The intensity of Oestrid infection (both species together) was significantly higher in the spring of 2015, compared to spring 2016 (p = 0.02). There was a trend towards an association between increased Oestrid larval infection and low carcass weight (p = 0.09). The quarter of calves with the highest intensities of infection had significantly lower carcass weights (−1.7 kg (SE = 0.75), p = 0.03), compared to the calves with lower infection intensities. Similarly, the probability of an animal being classified as emaciated increased with the intensity of larval infection (p = 0.03): The average sum of larvae found in emaciated calves, versus calves with a fair body condition were 308 and 143, respectively.

3.2.2. Elaphostrongylus rangiferi

As expected, none of the calves shot in the autumns of 2014 and 2015 excreted Elaphostrongylus L1 in faeces (calves normally do not develop patent infection until after the hunting season). The prevalence, intensity, and abundance of Elaphostrongylus larval faecal excretion in the calves killed in spring are shown in Table 2. The prevalence and abundance of infection were significantly higher (p = 0.003; p < 0.001) in the spring of 2015, compared to the spring of 2016. Histological examination of lung tissues from calves excreting E. rangiferi L1 larvae in faeces, revealed disseminated microgranulomas containing Elaphostrongylus eggs and hatching L1 larvae. Half of the calves displayed light-, and the remainder moderate to heavy- presences of microgranulomas. A massive and confluent presence of microgranulomas with extensive destruction of respiratory tissues was observed in the lungs of the most severely infected calf (Fig. 7).

No adult Elaphostrongylus nematodes were detected in the CNS of any of the calves examined in spring, whereas 1–22 nematodes were successfully identified in the skeletal muscles (Fig. 8) of five of the calves

| Season   | No. | Females | Males | Carcass weight Mean | Range  | SD   | Bodily condition |
|----------|-----|---------|-------|---------------------|--------|------|-----------------|
| Autumn 2014 | 12  | 6       | 6     | 13.4                | 10.2–19.0 | 2.3 |               |
| Spring 2015 | 12  | 1       | 11    | 12.9                | 10.1–16.2 | 2.0 | Fair           |
| Autumn 2015 | 12  | 1       | 10    | 11.8                | 6.5–16.0 | 2.5 | Emaciated       |
| Spring 2016 | 14  | 6       | 8     | 13.2                | 9.8–16.0 | 1.8 | Fair           |

a) Live weight minus head, skin, viscera, blood and mandibulopatals.

b) One calf was of unknown sex.
excreting L1 larvae in faeces (detection of this hair-thin organism is challenging in skeletal muscle tissues). The calf with the highest number of recovered nematodes also excreted the most L1 larvae in faeces (LPG: 1632). Isolated nematodes were identified morphologically to the genus *Elaphostrongylus* (Cameron, 1931). Although not speciated, we conclude, based on the host species and fecal excretion of typical L1 larvae, that

Fig. 4. Translucent white first stage *Cephenemyia trompe* larvae (1 mm) in the nasal mucosa of a calf killed on Hardangervidda in autumn 2014.

Fig. 5. *Cephenemyia trompe* larvae in the pharynx/nasopharynx of a calf killed on Hardangervidda in spring 2015. Note the paired, swollen retropharyngeal lymph nodes (Ln).

Fig. 6. Photograph from the inside of the skin showing *Hypoderma tarandi* larvae removed from the lower part of the back of a calf killed on Hardangervidda in spring 2015.
these nematodes were indeed *E. rangiferi*.

### 3.3. Other findings

The reindeer sinus parasite, *Linguatula arctica* (Riley et al., 1987) was detected in the maxillary sinuses of three calves. Six calves displayed eye lesions compatible with chronic keratitis.

### 3.4. Meteorological data

The calculated mean summer temperatures in the pasture area for 2014 and 2015 were 1.0 °C above and 1.7 °C below the average (7.4 °C) for the last 30-year-period (Fig. 9). Fig. 10 shows the number of summer days with a mean air temperature ≥12 °C for this period, and for the years 2014 and 2015, as well as for the five individual years with a higher mean temperature than in 2014.

The total snow precipitation (measured as mm water) for the winters 2014/15 and 2015/16 was high, but similar (284–296 mm). The number of days with a calculated mean air temperature >0 °C was however higher in the winter of 2015/21016 (11 days), compared to winter 2014/15 (3 days). Based on these results, it could be suggested that winter conditions were in favor of the 2014 generation.

### Discussion

This study found a significant relationship between high winter loads of Oestrids and *E. rangiferi*, low carcass weights and cases of emaciation in wild reindeer calves on Hardangervidda. The parasite loads were

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Table 2

| Season     | No. | Hypoderma tarandi |            | Cephenemyia trompe |            |
|------------|-----|-------------------|------------|-------------------|------------|
|            |     | Prevalence | Intensity of infection | Prevalence | Intensity of infection |
|            |     | Mean  | Range  | SD         | Mean  | Range  | SD         |
| Spring 2015| 12  | 100%   | 170    | 66–371     | 101   | 100%   | 64        | 25–196     |
| Spring 2016| 14  | 100%   | 119    | 21–422     | 135   | 100%   | 17\(^a\)   | 1–48       |

\(^a\) 32% of the *C. trompe* larvae found in spring 2016 were detected after rinsing with water. This complementary examination was not conducted in spring 2015.

Table 3

| Season     | No. examined | No. positive | Prevalence | Intensity\(^a\) | Abundance\(^b\) |
|------------|--------------|--------------|------------|-----------------|-----------------|
|            |              |              |            | Mean | Range  | SD         | Mean | Range  | SD         |
| Spring 2015| 12           | 11           | 92%        | 347  | 24–1632 | 452        | 452  | 119     | 27         |
| Spring 2016| 14           | 3            | 21%        | 124  | 5–242   | 119        | 119  | 27      |             |

\(^a\) Number of larvae in infected individuals.

\(^b\) A measure of the mean level of infection in all hosts, including non-infected individuals.

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Fig. 7. Verminous pneumonia in a calf killed on Hardangervidda in spring 2015. Numerous confluent granulomas containing *Elaphostrongylus rangiferi* eggs and hatched first stage larvae. Haematoxylin and eosin stain. Bar, 100 μm.
significantly higher for the calves born in the summer of 2014, compared to those born in 2015. We consider these differences in parasite loads to be closely related to differences in summer temperature and possibility of successful Oestrid pupation and development of *E. rangiferi* L3 larvae in the environment. The mean summer temperature in 2014 was 2.6 °C higher than the mean for 2015.

The developmental time of Oestrid pupae and L3 larvae of *E. rangiferi* in gastropods decreases proportionally with temperatures above 10 °C and at 12 °C the minimum developmental period for Oestrids and *E. rangiferi* is about 1.5 and 2 months, respectively (Halvorsen and Skorping 1982; Nilsen, 1997). Based on our calculations, these temperature requirements were not met at the average summer pasture altitude (1283m) for either of the two summers (see Fig. 10). As air temperatures increase in mountain areas by almost 1 °C per hundred meters decrease in elevation, it is considered likely that fulfilled development of Oestrid pupae and *Elaphostrongylus* L3 larvae could be accomplished at a few hundred meters below the average summer pasture altitude in 2014, and many hundred meters below this level in 2015. However, it must be emphasized that in sites with a favourable microclimate, suitable temperatures may be reached at higher altitudes. Moreover, for *E. rangiferi*, it has been suggested that the parasite may compensate for low temperatures by fulfilling its larval development over two summers, by overwintering in gastropods (Halvorsen and Skorping, 1982).

Fig. 8. Group of adult hair-thin, about 3–5 cm long *Elaphostrongylus rangiferi* nematodes in the muscle fascia of a calf killed on Hardangervidda in spring 2015.

Fig. 9. Calculated mean air temperature (°C) in the main summer pasture area of the Hardangervidda wild reindeer population for individual summers (June–August) 1989–2018. The summers of 2014 and 2015 are marked with a black circle. The dotted horizontal line shows the mean (7.4 °C) for the 30-year-period.

Fig. 10. Calculated number of summer days (June–August) with a mean air temperature ≥12 °C in the main summer pasture area of the wild reindeer population on Hardangervidda. The figure shows the mean and range for the 30-year period 1989–2018, and the mean for individual years 2014 and 2015 as well as for the five years with a higher mean temperature than in 2014.
We consider the prevalence (100%) and intensity of *H. tarandus* larval infection (mean 170 larvae) and *C. trompe* (mean 64 larvae) identified in the 2014 calf generation to be high. The real intensity of *C. trompe* larvae was obviously higher since an additional (water flushing) count, identifying 32% of the larvae found in the 2015 generation, was not performed for the 2014 generation. In comparison, studies carried out in semi-domesticated reindeer flocks in northern Norway reported mean intensities of 60 *H. tarandus* and 18 C. trompe larvae (Foistad, 1991; Nilsen and Haugerud, 1995). Oestrid larvae are rich in protein and fat, which originates from the host. The host’s loss of energy to the larvae will peak in late winter and spring when the larvae grow to full size (1.5 g). Calves are especially vulnerable since they are frequently infected with more larvae and have lower fat reserves, than adults (Skjenneberg and Slagsvold, 1968). Cuyler et al. (2012) examined 10-month-old Greenland caribou calves with heavy Oestrid larval infections and estimated the energy costs for the heaviest infected calves to be equivalent to 12 days basal metabolic rate, or forgone fattening equivalent to 1.2 kg fat. They concluded that high larval burdens could negatively affect winter calf survival. We suggest that high and even moderate Oestrid larval infection levels may have fatal energy costs for calves balancing on a nutritional knife-edge during a long and harsh alpine winter and spring on Hardangervidda.

The prevalence of *E. rangiferi* infection in the 2014 calf generation was high (92%) whereas the intensity of infection, with reference to earlier studies in reindeer (Halvorsen et al., 1985) can be considered moderate to high. The calf with the highest intensity of infection (LPG 1632) was in an advanced stage of emaciation and 22 adult *E. rangiferi* were isolated from its skeletal muscles. This was similarly to the number of nematodes recovered from two experimentally infected reindeer calves that showed signs of ataxia and posterior paresis of several months’ duration (Handeland, 1994; Handeland et al., 1994). On this basis, we consider it likely that the most heavily infected calf in our study would have suffered locomotory disturbances during the winter. The same calf also had a comprehensive verminous pneumonia caused by *Elaphostrongylus* eggs and L1 larvae, which may have hampered lung function. Verminous pneumonia can also create beneficial conditions for opportunistic pathogens e.g., *Pasteurella multocida*. This bacterium is commonly carried in the upper airways of reindeer and is a well-known cause of pleuropneumonia and septicaemia in semi-domesticated reindeer in northern Norway (Kummeeneje, 1976). Pasteurellosis has also been diagnosed in wild reindeer populations (Handeland, 2016).

Although *E. rangiferi* infection is common in wild reindeer populations (Handeland et al., 2019) and may cause relatively heavy infestations, as demonstrated in the present study, no outbreaks of clinical elaphostrongyllosis have so far been recognized. This contrasts to semi-domesticated reindeer herds in northern Scandinavia where epidemics and severe losses, primarily in calves, have been reported (Rones and Nordkvist, 1962; Bakken and Sparboe, 1973; Kummeeneje, 1974). The apparent differences in clinical significance between semi-domesticated and wild reindeer could be related to differences in the probability of detecting disease. Semi-domesticated flocks are herded and thus more or less continuously monitored, whereas the wild reindeer populations, with the exception of the hunting season, are left to themselves in remote areas. Given a probable main time of infection in late summer or autumn (Mitsgevich, 1964; Halvorsen et al., 1980), and neurological signs normally starting 1–2 months after infection (Handeland et al., 1994), clinical disease in wild reindeer will not occur until the following hunting season, with small chances of detection. It should also be emphasized that even moderate infections with long-lasting slight or subtle locomotory disturbances may be fatal during the harsh alpine winter.

We assume that the high winter loads of Oestrids and *E. rangiferi* in wild reindeer calves on the Hardangervidda are related to the population’s occupation of a limited area in the south during the summer (Fig. 2). This aggregation in the south in summer leads to higher concentrations of parasites on the ground, compared to previous years when the parasites were spread along a migratory route towards, and in larger summer pastures, in the west. The fact that Oestrid larvae are dropped and developed into adult flies within the same area, also probably increases Oestrid fly harassment. Harassment by Oestrid flies in summer is associated with reduced grazing time and low carcass weights in the autumn (Skjenneberg and Slagsvold, 1968; Colman et al., 2003). A gradual increase in the level of infection with Oestrids and *E. rangiferi* can also be expected in the face of climate warming. It has been shown that the average summer temperature in the Norwegian wild reindeer areas has increased by 0.6 ◦C from the previous, to the last, 30-year period (Handeland et al., 2019).

In conclusion, this study provided strong indications that Oestrid and *E. rangiferi* infections contribute to low body condition and cases of emaciation among wild reindeer calves in winter, particularly after a warm summer. We anticipate an increased future significance of these infections in wild reindeer populations due to climate warming and animal aggregation, following human pressure on their living areas.

Declaration of competing interest

None.

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