

HAIR-LOSS EPIZOOTIC IN MOOSE (*ALCES ALCES*) ASSOCIATED WITH MASSIVE DEER KED (*LIPOPTENA CERVI*) INFESTATION

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ABSTRACT: Deer keds (*Lipoptena cervi*) are blood-sucking flies in the family *Hippoboscidae*; moose (*Alces alces*) are their main host in Scandinavia. There are no detailed reports of the negative impacts of deer keds on moose. In 2006 and 2007, hunters in southeastern Norway and midwestern Sweden found several moose cadavers with severe alopecia; numerous moose had extensive hair loss. Between February 2006 and June 2007, materials from 23 moose were submitted for laboratory examination and large numbers of deer keds were found in the coat of most animals. The body condition of the moose varied but was poor in animals with severe alopecia. The findings of enormous numbers of deer keds in the coat of the majority of the affected animals and a consistent histologic image (acute to chronic, multifocal to coalescing, eosinophilic to lymphocytic dermatitis), concurrent with the absence of any other lesions, trace element deficiencies, or dermal infections which are known to cause alopecia, suggest that the hair-loss epizootic was linked to massive infestations with deer keds. The emergence of this hair-loss syndrome implies that the dynamics between parasite and host have been disrupted by a currently unknown environmental or ecological factor. A high moose density, combined with extraordinarily mild weather June 2006–June 2007 and a particularly long period with the absence of night-frost in autumn of 2006, may have been ideal for deer ked development, survival, and optimal host acquisition.

Key words: *Alces alces*, climate, deer ked, dermatitis, hair loss, *Lipoptena cervi*, population density, pathology.

INTRODUCTION

The deer ked (*Lipoptena cervi*) is a blood-feeding ectoparasite that infests several cervid host species (Hackman et al., 1983). Deer ked females are viviparous and produce new prepupae throughout the year. Fully developed pupae passively drop out of the coat to the ground where they develop into winged imagoes in late summer and early autumn. When the imagoes reach their hosts, they lose their wings and remain on the same host for the rest of their lives (Haarløv, 1964). Keds have been present in continental Europe since the stone age (Gothe and Schöl, 1994) and were described in southern Sweden as early as 1758 (Linné, 1758). This parasite has expanded its distribution west and northward in Scandinavia over the last few decades and is now abundant in southeastern Norway and central Sweden (Fig. 1) (Välimäki et al., 2010). Factors

contributing to the increase in deer ked distribution and abundance are not fully elucidated, although increases in host animal populations and changes in climate have been suggested (Välimäki et al., 2010).

The deer ked is not reported to cause disease in the host (Allan, 2001), although Ströse (1916) blamed the parasite for causing focal hair loss and eczema in red deer (*Cervus elaphus*). Darling (1963) reported that red deer seemed not to react strongly to the deer ked but argued that the effect on the host must be considerable, without further describing the cases. Fatigue and growth retardation have been seen in fallow deer (*Dama dama*) experiencing massive infestations of deer keds (Ivanov, 1974). Laaksonen (2009) reported that semidomesticated reindeer (*Rangifer tarandus*) showed pruritus and developed focal alopecia after infestation with deer keds.

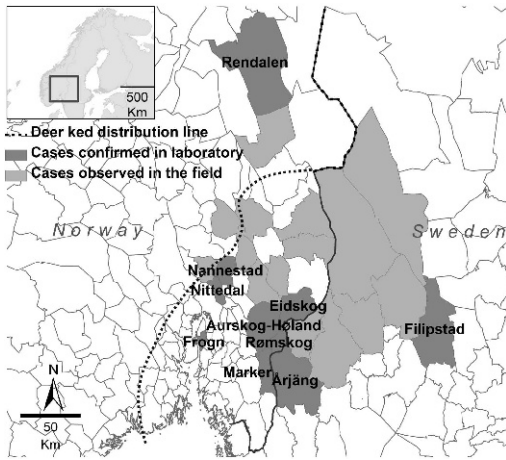


FIGURE 1. A map of southeastern Norway and midwestern Sweden. Dark grey color represents municipalities with laboratory-confirmed cases of alopecia caused by deer keds (*Lipoptena cervi*) in moose (*Alces alces*), 2006–2007. Light grey represents municipalities with observed severe alopecic moose in the field. The known distribution of deer ked in Norway is east of the dotted line.

We describe the clinical and pathologic findings associated with epizootic hair loss among moose in areas of southeastern Norway and midwestern Sweden where the deer ked is abundant. We discuss to what degree the hair-loss syndrome was associated with deer ked infestation and to what extent local moose population densities and weather conditions were factors in the outbreak.

MATERIALS AND METHODS

Study area

The study area was in southcentral Scandinavia (59°06'–62°16'N, 10°27'–15°13'E; Fig. 1). The elevation ranges from sea level in the southwest to about 600 m in the north, covering both the middle boreal, southern boreal, and boreonemoral vegetation zones (Moen, 1998). The landscape is undulating with forest-covered hills and some farmland in intersecting valleys. Forests are dominated by Scots pine (*Pinus sylvestris*) and Norway spruce (*Picea abies*) and, to some extent, birch (*Betula* spp.).

Affected population

The moose population of the study area is mainly limited (and regulated) by hunting. Predation by wolves (*Canis lupus*) and a few

brown bears (*Ursus arctos*) also may have some impact on the population (Sand et al., 2008).

Body mass and reproductive rates of moose are relatively high, but variable, throughout the study area. Observations by moose hunters suggest a decline in body condition during the last 15 yr, particularly in the north (Solberg et al., 2010). The decline is associated with increasing population density and is thought to be caused by food limitation (Lavsund et al., 2003). Data from radio-collared moose indicate that moose in this area are sedentary (Lavsund et al., 2003). Moose population density in the study area is higher than the average for Scandinavia. On a regional (county) scale, hunters harvested, on average, about 0.3–0.6 moose per km² of forest and bogs in 2000–2007 (Solberg et al., 2010). During the same period, we estimated the preharvest moose density was about 1.0–1.4 adult (≥ 1 yr old) moose per km² (E.J. Solberg, unpubl. data).

Data collection

Nonconfirmed observations of alopecic moose were based on phone and email correspondence with the National Veterinary Institutes in Oslo, Norway (NVI) and Uppsala, Sweden (SVA). To estimate the prevalence of alopecia in the moose population in September and October 2007 and 2008, moose hunters in five Norwegian counties, representing areas within and outside the deer ked distribution range, were asked to complete a questionnaire during the moose hunting season. They were asked to note if they had observed moose with hair loss and to classify the pattern of alopecia on a scale from 0 (no hair loss) to 5 (complete hair loss) according to a figure (Fig. 2). In March 2007, two cows and one subadult with severe hair loss were fitted with radio collars in Årjäng, Sweden (Fig. 3a). The development of hair loss was monitored in these three animals; blood samples for hematology were drawn from the jugular vein at the time radio collars were fitted.

Specimen analysis

The patho-anatomical part of the study was based on an examination of material from carcasses and biopsy material from alopecic moose submitted to NVI and SVA from the study area from February 2006–June 2007 (Fig. 1, Table 1). In the laboratory, whole carcasses were carefully inspected and described before the hide was removed and examined. A full necropsy was performed and samples of lung, heart, liver, kidney, brain, lymph nodes, thyroid gland, adrenal gland, and skin were collected and fixed in 10% neutral buffered formalin, dehydrated and

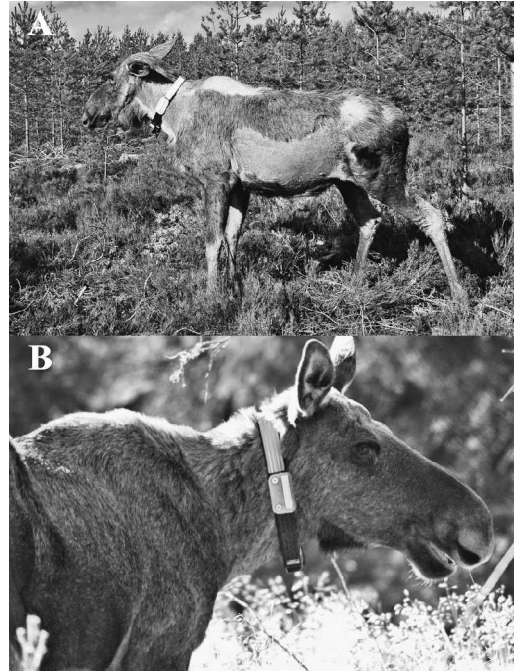
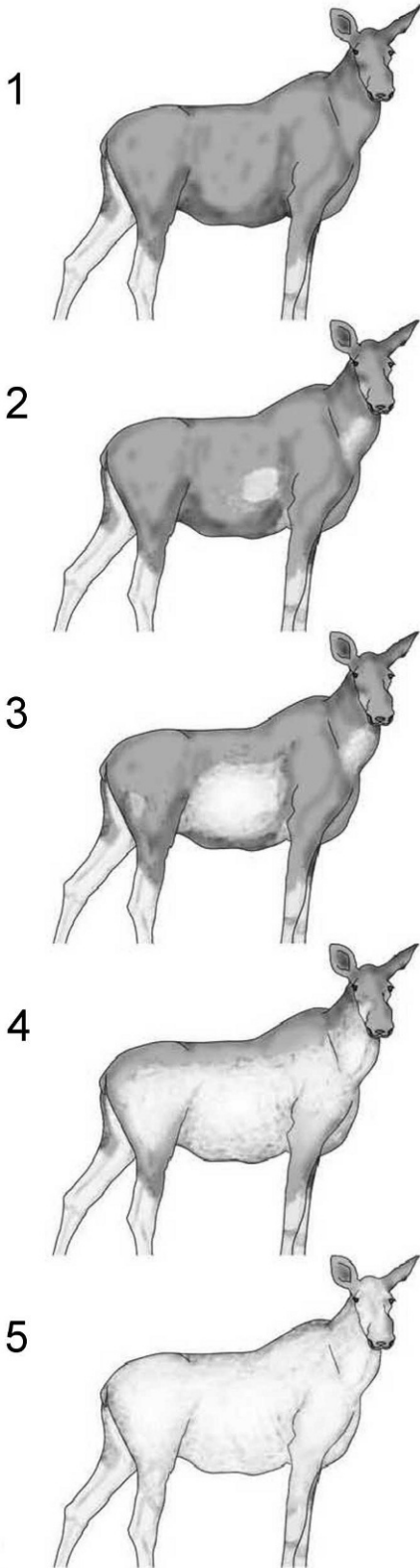


FIGURE 3. (A) Adult, female moose (*Alces alces*) with typical pattern of alopecia, radio collared in March 2007 in Värmland county, Sweden. (B) The same moose, in September 2007, showing regrowth of the coat on neck and withers. Photo: Gunnar Glöersen, Swedish Hunters Association.

embedded in paraffin, sectioned at 5 μm , and stained with hematoxylin and eosin and van Gieson for histologic examination. Standard bacteriologic examination on calf blood agar plates was performed on samples from liver ($n=12$), spleen ($n=9$), and skin ($n=14$). The plates were incubated aerobically at 37 C and examined after 24–48 hr. Biopsies from both affected and normal skin were collected for routine parasitologic ($n=11$) and mycologic ($n=15$) examination. All visible deer keds and pupae in the coat of four animals were collected and counted. Liver samples from

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FIGURE 2. Alopecia in moose (*Alces alces*), as assessed by hunters in the field during the 2007 and 2008 hunting seasons in Norway and Sweden: 1) No visible hair-loss, 2) slight hair loss; spots of alopecia affecting about 5 to 20%, especially thorax and ventral neck, 3) moderate hair loss; about 30 to 40% of coat lost and remaining coat sparse, 4) severe hair loss, about 40 to 80% of coat lost, 5) naked moose; more than 80% hair loss. Figure inspired by figure 1 in Samuel (1989).

TABLE 1. Chronologic listing of moose (*Alces alces*) received for laboratory examination during an outbreak of alopecia in southeastern Norway and midwestern Sweden in 2006–2007.

No.	Collection date	Location ^a	Sex ^b	Age ^c	Euthanized	Weight ^d	Amount of deer keds ^e	Alopecia ^f	Gross lesions
1	2/13/2006	Marker	F	Subadult (1,5)	Yes	210	Large	3	None
2	10/25/2006	Rømskog	F	Adult	Yes	—	Large	3	None
3	12/7/2006	Rømskog	F	Calf (0,5)	No	—	Large	3	None
4	12/15/2006	Filipstad	F	Adult	Yes	—	Moderate	3	None
5	12/20/2006	Årjäng	M	Subadult (1,5)	Yes	—	Large	3	None
6	1/8/2007	Rømskog	F	Adult	Yes	365	Large (11,014)	4	None
7	1/22/2007	Aurskog-Høland	F	Adult (18)	Yes	290	Large (16,496)	4	Solitary fibropapillomas
8	1/29/2007	Rømskog	F	Adult	Yes	390	Large (5,938)	3	None
9	2/12/2007	Aurskog-Høland	F	Subadult (2)	Yes	240	Large	3	None
10	2/12/2007	Aurskog-Høland	F	Adult	Yes	350	Large	3	Solitary kidney cysts
11	2/15/2007	Rendalen	M	Subadult (2)	Yes	—	None	3	None
12	2/26/2007	Rendalen	M	Subadult (2)	Yes	290	None	2	None
13	3/1/2007	Aurskog-Høland	F	Adult (5)	Yes	330	Large	3	None
14	3/1/2007	Aurskog-Høland	F	Subadult	Yes	110	Large (10,687)	1	None
15	3/2/2007	Nannestad	F	Adult(9)	No	170	None	3	None
16	3/12/2007	Eidskog	F	Adult	Yes	—	Large	2	None
17	5/2/2007	Aurskog-Høland	M	Adult	Yes	350	Moderate	3	Generalized fibropapillomatosis
18	5/11/2007	Aurskog-Høland	F	Adult (12)	Yes	360	Small	4	Front leg fracture (hit by car)
19	5/21/2007	Nittedal	M	Subadult (2)	Yes	180	Small	4	None
20	5/21/2007	Aurskog-Høland	F	Adult (22)	Yes	275	Large	3	Malignant etmoidal tumor
21	6/8/2007	Frogn	M	Adult (10)	Yes	330	None	4	Peruke antlers
22	6/20/2007	Aurskog-Høland	F	Adult	Yes	325	None	3	None
23	6/28/2007	Aurskog-Høland	M	Adult (5)	Yes	350	None	3	Solitary fibropapillomas

^a Municipality in Norway and Sweden.^b M=male; F=female.^c In brackets, estimated age based on dental cementum annuli counts.^d In kilograms, dash indicates weight was not measured.^e In brackets, total count of deer keds.^f Degree of alopecia by range; 1 (normal coated) to 5 (naked); see Figure 2 for details.

12 moose were frozen at -20°C prior to carrying out trace element analysis for copper, selenium, and cobalt (Vikøren et al., 2005).

Meteorologic data collection

We used meteorologic data from Gardermoen (elevation: 202 m) weather station, which is representative for the inland areas of southeastern Norway and adjacent areas of Sweden. Gardermoen is an automated weather station that provides hourly measurements of precipitation, temperature, humidity, wind, and pressure. Prior to 2009, these variables were measured manually every 3–6 hr, depending on weather and time of day. We wanted to elucidate factors that may influence off-host survival and development of deer keds, as well as host acquisition of winged keds. Temperature influences deer ked development (Härkönen et al., 2010) and repeated freezing and thawing may be unfavorable for parasite survival (Davidson et al., 2008), with the pupal stage on the ground being most vulnerable. Hence, we looked at the depth and duration of snow cover and temperature fluctuations around zero degrees C. Warm and sunny weather may facilitate host acquisition by winged keds, while frost is probably less beneficial; therefore, we also included degree-days (daily mean temperature multiplied by the number of days), length of growing season (sum of days with daily mean temperature $>5^{\circ}\text{C}$), precipitation, and first frost in the autumn.

RESULTS

Field observations

Earlier reports of alopecia in moose in the study area are scarce. On February 13, 2006, wildlife managers in Marker municipality, Norway (Fig. 1), reported a subadult, female moose with severe hair loss, emaciation, and abnormal behavior. The animal was euthanized after seeking shelter in a farm building and submitted to NVI for necropsy (moose 1, Table 1). In October 2006, wildlife managers in the neighboring municipality of Rømskog (Fig. 1) reported that five of 60 hunted moose had severe hair loss and that moose, in general, were severely infested with deer keds. Skin samples from one of the alopecic moose were submitted to NVI (moose 2, Table 1). In November and

December 2006, 11 alopecic moose were reported in the same area; three were found dead and eight were euthanized (J. Moen, pers. comm.). In Sweden, between Årjäng and Filipstad (Fig. 1), hunters euthanized 12 and observed another 50 moose with severe hair loss during the autumn of 2006 and winter of 2007 (G. Glörsen, pers. comm.). From December 2006 to June 2007, skin samples or whole carcasses from an additional 21 moose (moose 3–23), representing seven different municipalities in the study area in Norway and two in Sweden, were submitted to NVI and SVA (Table 1). During this period, wildlife managers and laymen also reported observations of alopecic moose in another nine municipalities; two in February, one in March, three in April, and three in May (Fig. 1). According to the reports, severely alopecic moose seemed to be in poor condition and did not respond to human activity. Pruritus, however, was not observed.

Necropsy findings

Seventeen whole carcasses and biopsy material from six animals were submitted to NVI ($n=21$) and SVA ($n=2$) from the study area from February 2006–June 2007 (Fig. 1). Twenty of the animals were euthanized because of severe alopecia, two were found dead, and one (moose 14) was a normally coated calf, shot simultaneously with a female alopecic moose.

The necropsy of the index case (moose 1, Table 1) revealed severe, bilateral alopecia and moderate, crusting dermatitis involving the cheeks, ventral neck, lateral thorax, abdomen, and haunch. A large number of deer keds were observed on the carcass, most in areas that were still covered with hair. The lymph nodes of the body were generally enlarged, but no other specific gross lesions were noted. Histologic examination of alopecic areas revealed moderate and diffuse orthokeratotic hyperkeratosis and diffuse, moderate hyperplasia of the epidermis, with moderate formation of rete ridges (Fig. 4).

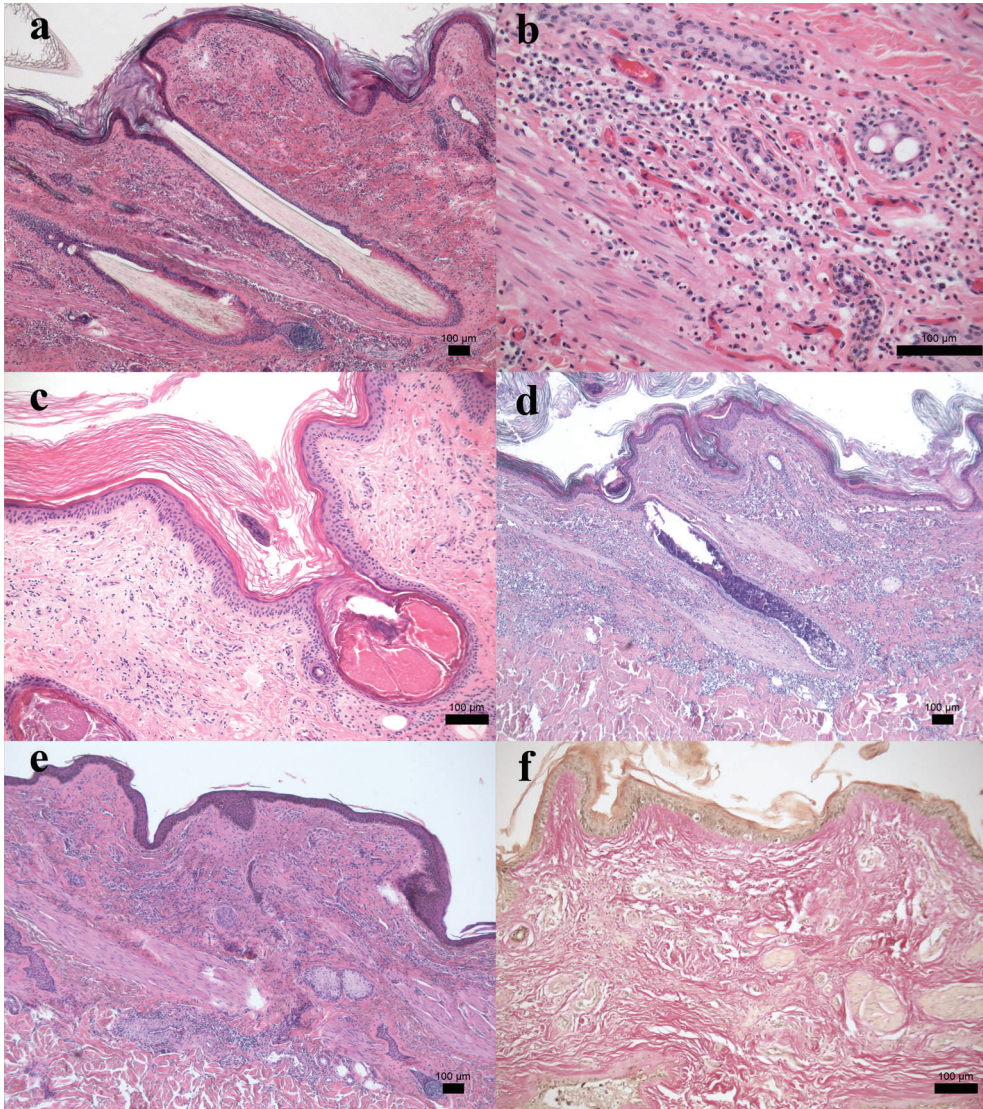


FIGURE 4. Skin histology of alopecic moose (*Alces alces*). (a) Sparse coat; inflammatory infiltrates in dermis, flame follicles, and follicular keratosis. (b) Sparse coat; perivascular infiltration of eosinophils, macrophages, and plasma cells. (c) Alopecic skin, orthokeratotic hyperkeratosis, acanthosis, intracorneal pustule, and follicular keratosis. (d) Sparse coat; orthokeratotic hyperkeratosis, perivascular infiltration of eosinophils, macrophages, and plasma cells and a follicle filled with these cells. (e) Alopecic skin; acanthosis, dermal fibrosis, and perivascular infiltration of macrophages and lymphocytes. (f) Sparse coat; acanthosis, and fibrosis. a, b, c=moose 6 (see Table 1), d=moose 16, e=moose 18, f=moose 20. All are hematoxylin and eosin-stained sections, except for f, which is van Gieson stained. Bar=100 µm in all sections.

The hair follicles showed moderate keratosis, with the infundibulum of the majority of the follicles dilated with keratin plugs of exfoliated corneum and debris. The hair bulbs had the appearance of “flame follicles” (i.e., follicles morphologically characterized by excessive trichilem-

mal keratinization, giving the impression of a flickering flame). Sebaceous and sweat glands appeared relatively normal but had prominent, thickened basal membranes. The dermis showed increased cellularity consisting of diffuse, mild to moderate infiltrates of eosinophilic granu-

locytes, macrophages, plasma cells, and lymphocytes. A diffuse, mild edema and a diffuse, moderate increase in the amount of regularly organized connective tissue in the dermis were also observed. The vessels of the dermis appeared contorted and were characterized by hypertrophic endothelium with prominent nuclei and a thickened basal membrane. In areas where some hair was still present, the skin was characterized by mild, diffuse orthokeratotic hyperkeratosis, a mild focal hyperplasia of the epidermis, and occasional intraepidermal pustules. The changes in the dermis were similar to those in alopecic areas, but the increase in connective tissue was less prominent and characterized by irregularly organized fibers.

The macroscopic changes in animal numbers 2–23 showed large similarities with each other and with the case described above. The general pattern was bilateral, fairly symmetric alopecia on the ventral neck, sides, and abdomen while the dorsal neck, back, and limbs showed less-prominent hair loss. Moose 14 did not show alopecia but had skin changes as described below. In the alopecic animals, the naked skin showed varying degrees of hyperpigmentation (increasing with day length) and was diffusely thickened and covered by light, removable crusts but appeared otherwise relatively smooth and did not show obvious papules, erythema, or erosive ulcerative lesions. Although single hairs were broken, the general impression was that hairs were either present at full length or not present. Self trauma, consistent with scratching, was not a common finding. The remaining hair coat appeared dull and the hairs were easily epilated. Ked infestation was confirmed in the majority of cases (17 of 23; Table 1). Keds were distributed throughout the animal, except distally on the limbs, but only a few specimens were found in alopecic areas. The keds aggregated in the axillae, neck, groin, and perineal region. In fresh carcasses, the

insects were located on the surface of the skin, their head oriented towards the skin, while they crawled out on the protective hairs as the carcass cooled. The woolen hairs of severely infested animals were smeared with a reddish-brown, dust-like material, probably deer ked feces mixed with moose blood. The infestation intensity was highest in the animals examined in December–February (2006–2007), while few deer keds were found in May and June 2007. The body condition of the moose varied, although it was poor in the animals with severe alopecia, and there were no consistent changes in the internal organs.

Histologic presentation varied depending on the month of examination. In general, histologic examination of the skin revealed a mild to moderate diffuse orthokeratotic hyperkeratosis and mild to moderate multifocal to diffuse hyperplasia of the epidermis (acanthosis). In some cases, affected epidermis showed multifocal small ulcerations, micropustules, and serocellular crusts. The dermis was characterized by multifocal to coalescing, mild to moderate infiltrates of inflammatory cells. The infiltrates had a perivascular distribution and were found in both the superficial and the deep dermis. During the first half of the epizootic, they were dominated by eosinophilic granulocytes while lymphocytes, macrophages, and plasma cells were more prominent in the spring and early summer. Blood vessels adjacent to inflammatory infiltrates were contorted and showed endothelial hypertrophy. Inflammatory changes were most severe and acute in areas with a remaining coat, while alopecic areas were often characterized by milder, mononuclear infiltrates. Increased amounts of irregularly organized connective tissue (fibrosis) were most pronounced in chronic cases (i.e., in late winter and spring) and in denuded areas. During autumn, winter, and early spring the hair follicles appeared as flame follicles. However, in May–June, a gradual shift to anagen follicles was observed (Table 2). The density of follicles and dermal papillae did

TABLE 2. Summary of histologic evaluation of skin samples from moose (*Alces alces*) received for laboratory examination during an outbreak of alopecia in southeastern Norway and midwestern Sweden in 2006–2007.

Epidermis			Dermis				Hair follicles ^b
No.	Hyperkeratosis ^a	Acanthosis	Fibrosis	Inflammatory cell infiltration			
				Distribution	Severity	Dominating cells	
1	Diffuse, mod ^c	Diffuse, mod ^c	Mod ^c	Multifocal to coalescing	Mild to mod ^c	Eosinophils, macrophages, lymphocytes, plasma cells	Flame
2	Diffuse, mod ^c	Diffuse, mod ^c	Mild	Multifocal to coalescing	Mild to mod ^c	Eosinophils, macrophages, lymphocytes, plasma cells	Flame
3	Diffuse, mild	Multifocal, mild	Mild	Multifocal, perivascular	Mild	Eosinophils, macrophages, plasma cells	Flame
4	Diffuse, mild	No	Mild	Multifocal, perivascular	Mild	Eosinophils, macrophages, plasma cells	Flame
5	Diffuse, mod ^c	Diffuse, mild	Mild	Multifocal to coalescing, perivascular	Mod	Eosinophils, macrophages, plasma cells	Flame
6	Diffuse, mild	No	Mild	Multifocal to coalescing, perivascular	Mod	Eosinophils, macrophages, plasma cells	Flame
7	Diffuse, mild	No	Mild	Multifocal to coalescing, perivascular	Mod	Eosinophils, macrophages, plasma cells	Flame
8	Diffuse, mod ^c	Diffuse, mild	Mild	Multifocal to coalescing, perivascular	Mod	Eosinophils, macrophages, plasma cells	Flame
9	Diffuse, severe	Diffuse, mild	Mild	Multifocal to coalescing, perivascular	Mod	Eosinophils, macrophages, plasma cells	Flame
10	Diffuse, mod ^c	Diffuse, mild	Mild	Multifocal to coalescing, perivascular	Mod	Lymphocytes, plasma cells	Flame
11	Diffuse, mod ^c	Multifocal, mild	Mild	Insufficient inflammatory infiltration	Mod	Lymphocytes, plasma cells	Flame
12	Diffuse, mod ^c	Multifocal, mod ^c	Mild	Insufficient inflammatory infiltration			Flame
13	Diffuse, mod ^c	Multifocal, mod ^c	Mod ^c	Multifocal to coalescing, perivascular	Mild	Lymphocytes, plasma cells	Flame
14	Diffuse, mod ^c	Multifocal, mod ^c	Mild	Multifocal to coalescing	Mod	Eosinophils	Flame
15	Diffuse, mild	Multifocal, mild	No	Insufficient inflammatory infiltration			Flame
16	Diffuse, mod ^c	Multifocal, mod ^c	Mild	Multifocal to coalescing	Mod	Eosinophils, macrophages, plasma cells	Flame
17	Diffuse, mild	Diffuse, mild	Mod ^c	Multifocal, perivascular	Mild	Macrophages, lymphocytes, plasmacells	Flame and anagen
18	Diffuse, mild	Diffuse, mod ^c	Mod ^c	Multifocal, perivascular	Mod	Macrophages, lymphocytes, plasmacells	Flame and anagen
19	Diffuse, mild	Diffuse, mod ^c	Mod ^c	Multifocal, perivascular	Mild	Lymphocytes, plasma cells, neutrophils	Flame and anagen
20	Diffuse, mod ^c	Multifocal, mild	Mild	Multifocal, perivascular	Mild	Lymphocytes, plasma cells	Flame and anagen
21	No	Diffuse, mod ^c	Mod ^c	Multifocal, perivascular	Mild	Lymphocytes, plasma cells, neutrophils	Anagen
22	No	Diffuse, mod ^c	Mild	Multifocal, perivascular	Mod	Lymphocytes, plasma cells, neutrophils	Anagen
23	No	Diffuse, mod ^c	Mild	Multifocal, perivascular	Mild	Lymphocytes, plasma cells, neutrophils	Anagen

^a Orthokeratotic hyperkeratosis.

^b Dominating hair cycle stage in contour hairs.

^c Mod=moderate.

not show any obvious association with alopecia, deer ked infestation, or time of year.

Parasitologic examination

Seven of the 11 skin samples examined for ectoparasites other than deer keds were negative, while low numbers of *Choriotopes* spp. were found in four animals.

Microbiologic culture results

Most skin samples had negative bacterial cultures, although *Staphylococcus aureus* ($n=6$), *Streptococcus agalactiae* ($n=2$), *Pasteurella multocida multocida* ($n=1$) and *Clostridium perfringens* ($n=1$) were isolated from some skin samples. Mycologic examinations were negative except for one moose on which *Scopulariopsis brevicaulis* was isolated.

Trace elements

Liver concentrations ($\mu\text{g/g}$ wet weight) of trace elements were (mean $\pm$ SEM): cobalt 0.11 ± 0.01 , selenium 0.97 ± 0.20 , and copper 74.8 ± 7.98 .

Observations and blood analysis of captured moose

Hemoglobin values from the captured animals in March 2007 were 124, 144, and 145 g/l (normal range: mean $\pm$ SD 125.5 ± 9.9 ; Adolfsson, 1993). In September 2007, both radio-collared cows were observed and appeared healthy with a coat, although slightly sparse, covering former denuded areas (Fig. 3b).

Observations during hunting seasons 2007 and 2008

In Norway, 16,845 moose were observed and reported in the questionnaire answered by moose hunters in autumn 2007. Twenty (0.12%) animals had alopecia (nine adult females, eight adult males, two calves, and one animal with age unclassified). Of these, 15 were scored as having grade 2 alopecia while the remaining five were scored as grade 3. In 2008, only one (subadult female) of 15,136 (0.007%) was alopecic (grade 2).

Weather

Weather conditions for winter 2005–2006 were fairly normal, but summer and autumn 2006 were milder than normal (Table 3). The 365-day running mean air temperature for the period from 11 June 2006–10 June 2007 was by far the highest since records at Gardermoen began in 1940 (Fig. 5a). The temperature was three standard deviations above the corresponding mean temperature of the normal period (1961–1990). Furthermore, autumn 2006 was characterized by an extraordinarily late arrival of the first frost night after August 1 (87 days, average is 47; Fig. 5b).

DISCUSSION

Based on the pathologic skin changes of alopecic moose, the massive deer ked infestation in affected animals, and the absence of findings indicative of any other consistent factor (other ectoparasites, infectious agents, endocrine disorders, or deficiencies) that could be a plausible cause of such a condition, we conclude that severe deer ked infestation was a key factor in the current outbreak. However, deer keds were not found on two alopecic bulls from Rendalen, north of the deer ked distribution range (Fig. 1) nor on four (one in March, three in June) alopecic animals inside the deer ked area. Young moose bulls can disperse considerable distances (up to 250 km) from their birth area before they establish a more permanent home range (Hundertmark, 2007). Hence, the two bulls from Rendalen may have been born within the deer ked range and may have experienced massive deer ked infestation as calves before dispersal. The hair loss in these two animals may, as such, represent chronic, partially healed lesions. The absence of the parasite in the last three cases may be explained by the deer ked life cycle; the adult ectoparasites are thought to die during spring and summer (Haarløv, 1964).

In Canada, premature winter hair loss in moose associated with the presence of

TABLE 3. Selected meteorologic data for 2000–2010 from Gardmoen weather station (elevation: 202 m), Norway, representative for the inland areas of southeastern Norway and adjacent areas of Sweden. The autumn of 2006 (bold type) was characterized by high numbers of deer keds in the study area.

Year	Previous winter ^a — pupal survival			Summer ^b — pupal development			Autumn ^c — swarming of adults					
	Days with snowfall ^d	Snowsum ^e	Melting-freezing episodes	Length of growth season ^f	Mean temperature in growth season	Degree-days ^g in above 5 C in growth season	Degree-days above 5 C in growth season	Mean temperature in autumn	Degree-days in autumn	Degree-days above 5 C in autumn	Precipitation ^h	Days before frost ⁱ
2000	17	1,068	72	209	13.0	1,546.2	951.2	9.9	1,056.9	535.5	455.9	123
2001	27	3,151	57	179	13.1	1,508.0	933.5	9.3	998.6	543.5	320.7	56
2002	20	1,641	64	174	13.9	1,714.3	1,100.4	8.7	928.8	636.3	193.5	54
2003	32	5,070	58	181	13.5	1,655.1	1,057.6	8.6	920.9	535.4	176.4	33
2004	6	1,586	63	200	12.8	1,662.2	1,016.0	9.8	1,048.2	580.2	273.9	62
2005	11	819	73	188	12.3	1,567.0	940.0	10.0	1,045.0	585.1	307.8	48
2006	22	3,871	64	185	14.6	1,678.2	1,106.1	10.7	1,140.7	703.3	392.3	87
2007	20	1,168	71	217	12.1	1,717.7	1,036.0	8.8	945.5	511.1	161.3	47
2008	22	751	78	192	13.6	1,633.4	1,036.9	8.4	901.3	462.4	318.6	61
2009	25	3,816	60	173	12.9	1,641.3	1,007.7	8.3	889.4	500.2	265.5	60
2010	NA ^j	NA ^j	43	167	13.4	1,500.9	950.5	8.0	857.5	469.9	250.5	59
Normal ^k	27	5,970	63	170	13.0	1,438.9	890.0	8.0	860.3	453.9	298.0	46.9
SD ^l	8	3,022	15	17	1.0	105.0	90.5	0.9	95.4	63.7	106.5	11

^a From November (year before) to April.
^b From onset of growth season to 15 August.
^c 1 August–15 November.
^d Snowfall=both increased snow depth and precipitation the same day.
^e Snowsum=daily snow depth multiplied by days (e.g., 10 cm snow depth for 10 days=100-cm days).
^f Growth season=sum of days with daily mean temperature >5 C.
^g Degree-days=the daily mean temperature multiplied by days (e.g., 10 C for 10 days=100-degree days).
^h Precipitation=daily precipitation in millimeters.
ⁱ Days from 1 August to first episode with temperature below 0 C.
^j NA=not available.
^k Mean value in the normal period 1961–1990.
^l SD = standard deviation.

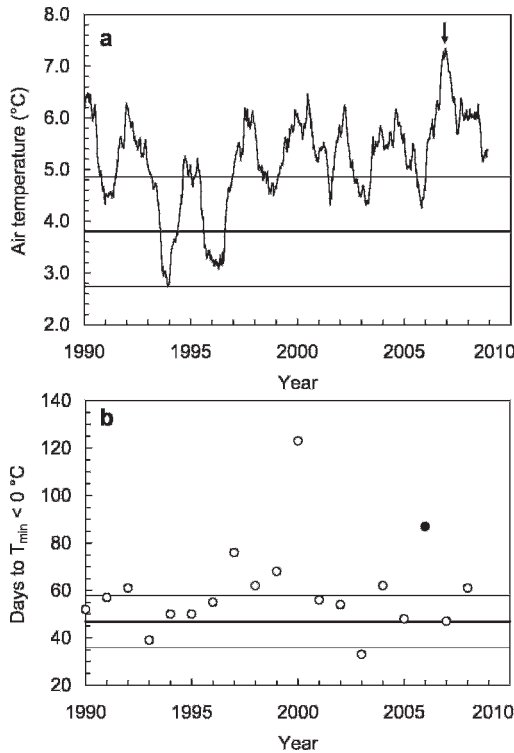


FIGURE 5. Meteorologic data from the Gardermoen weather station (elevation: 202 m), which is representative for inland areas of eastern Norway and midwestern Sweden (see www.met.no). The thick black line and the thin grey lines display, respectively, the mean and 30-yr standard deviations for the normal period 1961–1990. (a) Series of 365-day running mean air temperature for 1990–2009. The black arrow marks the maximum 365-day mean temperature in the series corresponding to the period 6 November 2006 to 6 October 2007. (b) Number of days after 1 August before the minimum temperature drops below 0 °C for 1990–2008. The black-filled circle marks the year 2006.

the winter tick (*Dermacentor albipictus*) has been described (McLaughlin and Addison, 1986). The winter tick was reported once in Norway, found on an imported horse from the USA, but the parasite is currently not present (Lillehaug et al., 2002).

Chorioptes spp. may cause skin lesions in the outer ear canal of moose (Hestvik et al., 2007) and have also been associated with single cases of generalized alopecia in moose in Norway and Sweden. Histologically, the ear skin lesions caused by this

mite seem quite similar to those seen in our study. However, moose with massive *Chorioptes* infestation typically show a patchy pattern of alopecia, compared to the confluent pattern seen in the current outbreak. Also, *Chorioptes* was not found in the majority of our cases. Other ectoparasites known to cause alopecia in cervids, including chewing lice (*Cervicola* sp.; Bildfell et al., 2004), sarcoptic mange (*Sarcoptes scabiei*; Bornstein et al., 2001), and mites of the genus *Demodex* (Gentes et al., 2007) were not found in our study.

Several endocrine dermatopathies cause alopecia in cattle, among them hypovitaminosis A and hypothyroidism (Ginn et al., 2007). Blood plasma levels of vitamin A and thyroxin were not examined in our material, but the histologic changes were not consistent with an endocrine dermatopathy. Deficiencies of copper (Frank, 1998) and cobalt (Kennedy et al., 1997), and selenium intoxication (Kaur et al., 2003), are reported to be associated with alopecia in animals. In our study, these trace elements were not at toxic or deficient levels based on the NVT's criteria set for cattle.

In Belarus, Ivanov (1974) reported that all moose hunted during the autumn harbored deer keds in their coat with an average of about 1,000 keds per moose. In Finland, average numbers of keds in normally coated cows and calves were 3,549 and 1,730, respectively (Paakkonen et al., 2010). We found 16,496, 11,014, and 5,938 keds in the coats of three cows with severe hair loss and 10,687 in the coat of a calf with a normal coat (Table 1). We consider these numbers to be very high, taking into account that keds prefer habitats with hair-covered skin (Haarløv, 1964), and it can be presumed that a major proportion of the initial ked population may have been lost together with the hair. Consequently, perhaps the high density of deer keds, biting and feeding 15 to 20 times per day (Ivanov, 1974), exceeded a threshold of what moose skin could tolerate, thus triggering a cascade of

skin inflammatory reactions as seen in the majority of our skin samples. Why this inflammatory reaction should induce hair loss without causing simultaneous pruritus is not clear. In experiments with sheep, Nelson (1963) suggested that the skin inflammatory response to the sheep keds (*Melophagus ovinus*) resulted in reduced dermal blood supply. Hence, it is possible that changes in blood supply, induced by deer ked bites, could affect the hair papillae and thereby cause hair loss. Further research is needed.

The prevalence and extent of the hair loss in the moose population in 2006–2007 is difficult to estimate due to the lack of surveillance data. Finding diseased or dead animals is the most direct evidence that disease is occurring in an area or population, but this technique has limited application and, invariably, results in an underestimation of the occurrence of disease (Wobeser, 2006). The number of submitted and reported animals is, therefore, not a good measurement of prevalence of disease. However, during the attempt to immobilize moose in March 2007, three of 15 moose (20%) observed from a helicopter had severe hair loss (Gunnar Glöersen, pers. comm.). Also, based on the pattern and severity of the alopecia seen in the moose and the assumption that the hair loss was progressive, it is plausible that a major proportion of the population experienced significant, but not clinically obvious, hair loss and, therefore, were not observed. We found a low prevalence of alopecia among moose in autumn 2007 (0.12%) and 2008 (0.007%), indicating a termination of the hair loss epizootic in spring 2007. We cannot rule out cases of deer ked-related hair loss prior to 2006, given the potentially low detection rate for the condition and the fact that moose alopecia was not of public concern prior to the hair-loss epizootic.

Deer keds have been established in the study area for 15–20 yr (Välimäki et al., 2010). It is curious that hair-loss epidemics have not occurred previously. Gener-

ally, parasite abundance increases with increasing host population density (Arneberg et al., 1998). Balashov (1996) reported a strong correlation between high density of moose populations and abundance of deer keds. Moose population density in the study area is high and, thus, favorable for deer ked survival and reproduction. However, high density alone cannot explain the epizootic in 2006–2007, as moose density has been high and stable in the study area for the last decade.

Balashov (1996) argues that mild and dry summers are important for the development of pupae and winged keds. On the other hand, Kadulski (1974) proposes that a long, severe winter with heavy snowfall is linked to high deer ked infestation intensity on cervids the following autumn and winter, due to weakening of the host. An alternative explanation for Kadulski's observations might be that frequent snowfalls will rapidly cover the pupae and protect them from potential predators (birds and rodents), thus creating stable storage conditions ideal for pupal survival during winter (Härkönen et al., 2010).

We consider the extraordinarily high summer and autumn temperatures in 2006 to be the most important factor for maximum pupal development and survival. Additionally, in 2006, the first frost night occurred late in autumn (Fig. 5b), possibly enabling winged keds to search for a host during a longer window of time than normal. Therefore, more swarming deer keds might have managed to find a host, causing unusually high deer ked densities on the moose in 2006–2007.

Independent of the cause of alopecia, moose lacking large proportions of hair coat will need to increase their metabolic rate to maintain body temperature (McLaughlin and Addison, 1986). Notwithstanding metabolic and behavioral adaptations, the basic metabolic energy requirements of a moose losing 30% of its winter coat may double at ambient temperatures of -20°C (McLaughlin and Addison, 1986). Hence, hypothermia or

starvation might have been the proximate cause of the winter mortality of alopecic moose in the study area in 2006–2007. Additionally, a transient negative effect of blood loss cannot be excluded, even though hemoglobin levels from the alopecic captured animals were normal and postmortems were not consistent with anemia. No decrease in moose population density or recruitment rates were reported from the study area in 2007 compared to the previous years, indicating that the outbreak did not have substantial effects on the local moose population (Solberg et al., 2010). There is still insufficient knowledge of the effects of deer ked harassment and infestation on the health and fitness of wildlife.

ACKNOWLEDGMENTS

We thank Marthe Opland, Nina Brekke Tvedt, and Oddgeir Rissa Jacobsen at NVI for the excellent technical assistance in the laboratory. We are grateful to Rebecca K. Davidson, NVI, Professor Jon Teige, Norwegian School of Veterinary Science and Preben Ottesen, and The Norwegian Institute of Public Health for helpful comments and Kjell Handeland, NVI, and Karin Bernodt, SVA, for performing some of the necropsies. Attila Tarpai, NVI, kindly prepared the maps. We thank Jørn Daltorp, wildlife manager, for the excellent work with the hair-loss questionnaire. We also thank Gunnar Gløersen (Swedish Hunters Association), John Sigmund Moen (wildlife manager in Rømskog), Atle Løvland (Indre Østfold Food and Animal Health Authority), and all the other helpful wildlife managers and hunters for providing important field reports and pictures of alopecic moose, submitting moose, and reporting alopecia. This work was supported by the municipalities of Aurskog-Høland, Eidsberg, Eidskog, Elverum, Grue, Halden, Kongsvinger, Lørenskog, Marker, Nannestad, Sør-Odal, Trøgstad, Vestby, and Åmot, the County Governor of Hedmark, the Norwegian Forest Owners' Federation, the Norwegian Directorate for Nature Management, and the National Health Surveillance Program for Cervids (HOP) in Norway.

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Submitted for publication 23 December 2010.

Accepted 9 April 2011.