DISEASES IN A MOOSE POPULATION SUBJECTED TO LOW PREDATION

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ABSTRACT: Well-publicized studies on two moose diseases, elaphostrongylosis and moose wasting syndrome, conducted across Sweden from 1985 to 1994 resulted in an increased number of reports of sick and dead moose. A sample of 724 moose were investigated, including 426 females, 208 males, and 90 of unknown sex with an average age of 3.7 years (SD = 4.9, range 0-20). Prominent diagnoses were elaphostrongylosis (18%), moose wasting syndrome (11%), and accidental death (11%). Other important diagnoses were neoplasm (5%), parasitic (6%), nervous system (5%), infectious (4%), eye and ear diseases (4%), and predation (3%). From the beginning to the middle of the 20th century approximately 10 wolves, 130 bears, 175 lynx, and 100 wolverines were present in Sweden (449,000 km^2). Currently, the scene is quite different with wolf, bear, lynx, and wolverine populations all increasing. The total number of large predators and scavengers is estimated at 2,500-3,000. We believe that the diversity of moose diseases seen in the future will differ from that observed during the 1980s and 1990s by being less visible due to increasing predation.

ALCES VOL. 41: 37-48 (2005)

Key words: *Alces alces*, bear, *Canis lupus*, diseases, elaphostrongylosis, moose wasting syndrome, predation, *Ursus ursus*, wolf

Statistics on regulated hunting (culled) of moose (*Alces alces*) have been recorded in Sweden since 1881, mirroring the population density. The population began to increase in the late 1920s, rose rapidly in the 1970s, and peaked in the 1980s (Cederlund and Markgren 1987, Cederlund and Bergström 1996). Today, the summer population is estimated to be 300,000-400,000 animals (Stéen et al. 1998b) resulting in an approximate density of 1.0 – 1.5 moose/km^2.

The moose harvest in Sweden is substantial with approximately 100,000 animals being culled each year and the meat annually comprising 4-5% of the country's total meat production (Stéen et al. 1998b). Because moose are an important natural resource of considerable economic value to tourism, hunting, and meat production, a long-term overview of diseases affecting their health is of great societal interest.

Routine investigations of wildlife diseases have a long history in Sweden (Stéen et al. 1997, 1998b). Diseases in moose have mostly been described as single or clusters of cases (Borg 1975, 1987; Stéen et al. 1998b). Climate, especially snow depth, and nutritional stress due to limited food resources have been regarded as the main causes of natural (i.e., culling and traffic excluded) mortalities. During the 1980s another picture emerged, when large numbers of sick or dead moose were found throughout Sweden. Two diseases, elaphostrongylosis (ELA), caused by the parasite *Elaphostrongylus alces*, and moose wasting syndrome (MWS), the etiology of which is still unknown, gave rise to public concern and interest. In 1985, two projects were initiated at the Swedish University of Agricultural Sciences (SLU) to investigate both diseases and a number of reports and papers have subsequently been published (Stéen and

Rehbinder 1986; Feinstein et al. 1987; Stéen and Diaz 1988; Stéen et al. 1989; Stéen and Johansson 1990; Stéen and Roepstorff 1990; Rehbinder et al. 1991; Stéen 1991; Olsson et al. 1993; Stéen et al. 1993; Frank et al. 1994; Merza et al. 1994; Stéen et al. 1994; Olsson et al. 1995; Stéen et al. 1997; Frank 1998; Lankester et al.1998; Olsson et al. 1998; Stéen et al. 1998a; Frank et al. 2000a, 2000b, 2000c, 2000d; Gajadhar et al. 2000; Olsson 2001; Broman et al. 2002a, 2002b). Our definition of disease in wild animals is in accordance with that of Wobeser (1981) who described diseases as any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, climate, infectious agents, inherent or congenital defects, or combinations of these factors.

An overview of the diseases seen in moose examined from 1985 to 1989, when non-human predators, including wolf (*Canis lupus*), brown bear (*Ursus arctos*), wolverine (*Gulo gulo*), and lynx (*Lynx lynx*), were few is presented in this paper. Diseases and other causes of morbidity and mortality were grouped into 19 diagnostic categories. The results are compared to previous reports of disease and mortality in moose.

METHODS

Study Period and Material

Whole carcasses or organs from approximately 1,000 moose were examined from 1985 to 1989. In this paper we describe moose examined by Stéen (Table 1).

Data Collection and Necropsy

Data describing where and how each animal was found or killed and the circumstances surrounding the case accompanied each sample. Post-mortem and follow-up investigations were performed as described in Stéen et al. (1997, 1998a). Evaluation of physical condition was done (n = 642) by visual inspection of the body fat, its location, and appearance. Condition categories included normal condition, below normal (poor), serous atrophy, or absence of adipose tissue (emaciated). Moose were aged by tooth wear and eruption (Skunke 1949, Reimers and Nordby 1968). Diagnosed causes of disease were categorized (Table 2).

Table 1. Summary of moose samples examined in the study.

Parameters		Number of animals
Total		724
Type of sample	Carcass	315
	Organ	405
	Unknown	4
Sex	Females	426
	Males	208
	Sex unknown	90
Season	Fall	294
	Winter	154
	Spring	200
	Summer	76
Manner of Death	Euthanized	230
	Found dead	304
	Culling	171
	Unkown	19

Statistics

Inferential statistics were performed using SAS®. Analyses were considered statistically significant when $P < 0.05$.

RESULTS

Samples originated from across Sweden, with the majority of cases being from north of Stockholm (59°N). Samples of sick or dead moose were submitted year-round, although the finds were most frequent in spring, followed by fall, winter, and summer (Table 1). Samples from hunter harvested animals were taken in the fall.

Age and Sex

The average age of moose examined was

Table 2. Number of animals diagnosed per disease category based on age classes.

Disease category	Calves	Yearlings	Adults (2-11 years)	Seniors (12-20 years)	Unknown	Total
Blood, lymphatic and cardiovascular systems (BLC)	1	1	3	2	1	8
Digestive system (DIG)	7	1	6	1	1	16
Endocrine system (END)	-	-	3	-	1	4
Eye and ear (EE)	5	2	14	5	-	26
Infectious diseases (INF)	7	-	20	3	2	32
Metabolic disturbances (MET)	-	-	1	-	-	1
Muscle-skeletal system (MUS)	3	1	13	1	2	20
Neoplasm (NEO)	1	3	24	6	5	39
Nervous system (NER)	11	-	24	3	-	38
Parasitic diseases (PAR)	11	12	16	1	3	43
Physical influences (PHY)	25	12	36	3	2	78
Reproductive and urinary systems (REP)	2	-	4	1	-	7
Respiratory system (RES)	5	-	11	2	2	20
Skin and connective tissue (SKN)	1	-	1	-	-	2
Malformation (MAL)	4	-	8	1	-	13
Elaphostrongylosis (ELA)	94	15	16	2	5	132
Moose wasting syndrome (MWS)	16	2	50	13	2	83
Predation (PRED)	11	3	7	3	1	25
Miscellaneous causes (MIS)	7	4	8	2	1	22
Total	211	57	265	48	28	609

3.7 years (SD = 4.9, range 0-20, n = 617). Grouped into 4 age classes, the distribution was: calf (41%), yearling (11%), adults 2-11 years (49%), and seniors 12-20 years (9%). The overall sex ratio was 1 bull/2cows, a female-biased adult ratio which makes cows more numerous than bulls (Fig. 1). The sex was unknown in 12% of the cases.

Diagnoses

Diagnoses were grouped into: blood, lymphatic and cardiovascular systems (BLC), digestive system (DIG), endocrine system (END), eye and ear (EE), infectious diseases (INF), metabolic disturbances (MET), muscle-skeletal system (MUS), neoplasm (NEO), nervous system (NER), parasitic diseases (PAR), physical influences (PHY), reproductive and urinary systems (REP), respiratory system (RES), and skin and connective tissue (SKN) (Merck Veterinary Manual 1979). Additional diagnoses were malformation (MAL), elaphostrongylosis (ELA), moose wasting syndrome (MWS), predation (PRED), and miscellaneous causes (MIS). No pathological findings were made in 155 cases (15%).

Of the 609 diagnosed cases, the most frequently diagnosed condition was ELA (22%), followed by MWS (14%), and accidental death (PHY, 13%) (Table 2). Other noticeable diagnoses were PAR, NEO, NER, INF, and EE. Predation was seen in 3% of cases and was comprised of 11 calves, 3 yearlings, 7 adults, 3 seniors, and 1 of unknown age. The age distribution of the predated cases did not differ from that of cases with other diagnoses (χ^2 = 2.5280, 3 df, P = 0.4703).

The relative risk (proportion of diagnosis

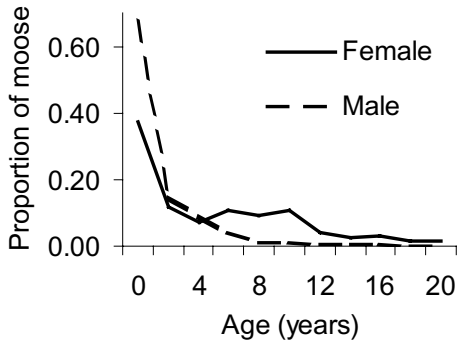

Fig. 1. Age distribution of examined moose (female $n = 426$; males $n = 208$).

among necropsied moose) from ELA was greater in calf/yearlings than in adults/seniors in both females ($\chi^2 = 73.4419$, 1 df, $P < 0.0001$, Fig. 2a) and males ($\chi^2 = 18.8946$, 1 df, $P < 0.0001$, Fig. 2b). For MWS the opposite pattern was seen in respect to age classes (Fig. 2a,b), however differences were not statistically significant for males ($\chi^2 = 14.1872$, 1 df, $P = 0.0002$; $\chi^2 = 0.6691$, 1 df, $P = 0.4134$, females and males, respectively). Animals with NEO and INF were over-represented in older animals ($\chi^2 = 17.0127$, 1 df, $P < 0.0001$, $\chi^2 = 7.6095$, 1 df, $P = 0.0058$, NEO and INF, respectively).

Among calves, sex was not related to occurrence of ELA nor MWS ($\chi^2 = 0.1374$, 1 df, $P = 0.7109$, $\chi^2 = 0.2476$, 1 df, $P = 0.6188$) (Fig. 2a, b). Two yearlings, 1 male and 1 female, were diagnosed with MWS and the relative risk did not differ between the sexes ($\chi^2 = 0.0037$, 1 df, $P = 0.9516$, Fig. 2a, b). Among older animals, adults and seniors together, bulls appeared to be more prone to ELA ($\chi^2 = 4.253$, 1 df, $P = 0.0392$, Fig. 2a, b) but the opposite pattern was seen for MWS; i.e., the relative risk was higher for cows ($\chi^2 = 6.3486$, 1 df, $P = 0.0117$, Fig. 2a, b).

The frequency of both ELA and MWS differed among seasons ($\chi^2 = 33.1174$, 3 df, $P < 0.0001$, $\chi^2 = 13.699$, 3 df, $P = 0.0033$, ELA and MWS, respectively). Moose with ELA were over-represented in springtime while cases with MWS were most prevalent in winter (Fig. 3). Predated carcasses were found most frequently in spring and in areas along the Norwegian border.

The occurrence of animals with ELA and MWS differed geographically. The relative risk of ELA was greatest in northern Sweden (Fig. 4a) while the relative risk of MWS was

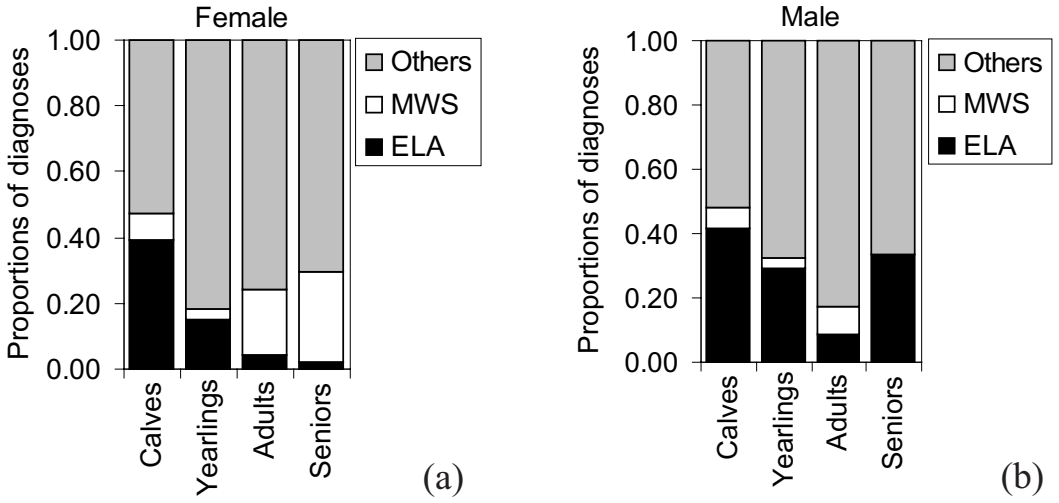

Fig. 2. Proportion of moose diagnosed as ELA, MWS, and 'Others' based on age class and sex; (a) female (calves $n = 125$, yearlings $n = 27$, adults $n = 220$, and seniors $n = 47$) and (b) males (calves $n = 96$, yearlings $n = 34$, adults $n = 69$, and seniors $n = 3$).

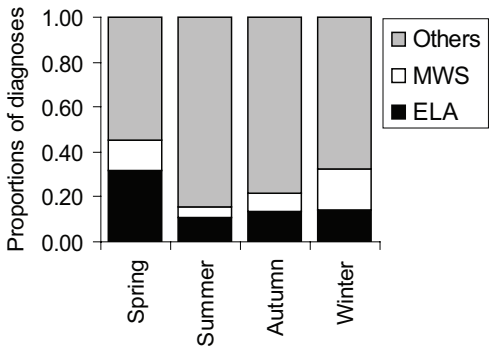

Fig. 3. Proportion of moose diagnosed as ELA, MWS, and 'Others' based on season (spring n = 200, summer n = 76, autumn n = 294, and winter n = 154).

highest in the south (Fig. 4b).

Condition

A disease was not always manifested by diminished body condition. Emaciation, poor, and normal condition were almost equally represented among the cases (33%, 31%, and 36%, respectively). Condition was related to season; poor/emaciated being most prevalent in spring, followed by winter, summer, and autumn. Excluding animals culled in the fall, this pattern did not change (Fig. 5). The poor/emaciated categories were over-represented in moose showing ELA and MWS (χ^2 = 28.0245, 1 df, $P < 0.05$, χ^2 = 10.237, 1 df, $P < 0.05$, respectively), but there was a tendency for INF to be under-represented (χ^2 = 3.7057, 1 df, P = 0.0542). About 50% of animals with INF were in normal body condition, compared to other disease diagnoses where the corresponding value was < 40% (Fig. 6). Moose with tumors (NEO), as well as predated animals, were in all categories of body condition.

DISCUSSION

Diagnoses

The proportion of different diagnoses (i.e., relative risks) varied between age-class and sex. MWS is more common among older animals and more common among cows than bulls. For ELA, the opposite pattern was observed. It appears that both age and sex can explain some of the variance in susceptibility to mortality from MWS or ELA. However, such conclusions are equivocal, as we do not know the size and structure of the population from which the dead moose came, which is essential to estimate the absolute risk of death. Therefore, one can only estimate the relative risk of death.

With reservations about discrepancies between relative and absolute risks, this study indicates that adult bulls were more prone to ELA than adult cows, but no differences were found between male and female calves and yearlings. Infections with *Elaphostrongylus* spp. normally occur in summer and fall, with clinical disease appearing in spring. Halvorsen (1986) studied ELA in reindeer and concluded that male calves belonging to dominant mothers are more heavily infected with *E. rangiferi* than females. The largest calves eat more and therefore experience a higher risk of ingesting gastropods with *E. rangiferi*. Stuve (1986) found a higher prevalence of ELA in male than female moose calves, also suggesting that males were more likely to be infected. Saether and Heim (1993) demonstrated that moose calf weights were dependent on summer browse in the cows' home range, the quality of which is related to the cows' status. For older animals, Stuve (1986) attributed the difference in infection between the sexes to physiological changes during the rut in accordance with Halvorsen's (1986) studies on reindeer.

Age is related to ELA, with yearling and calves being most frequently infected. Earlier studies have shown that moose shed most *E. alces* larvae during their first year of life followed by a sharp drop in larval shedding and reduced level of adult worms in older animals (Stuve 1986, Stéen 1991, Olsson et al.1995, Stéen et al. 1997).

Our results show that emaciation is associated with ELA. This could of course be a spurious correlation or a cause/effect of

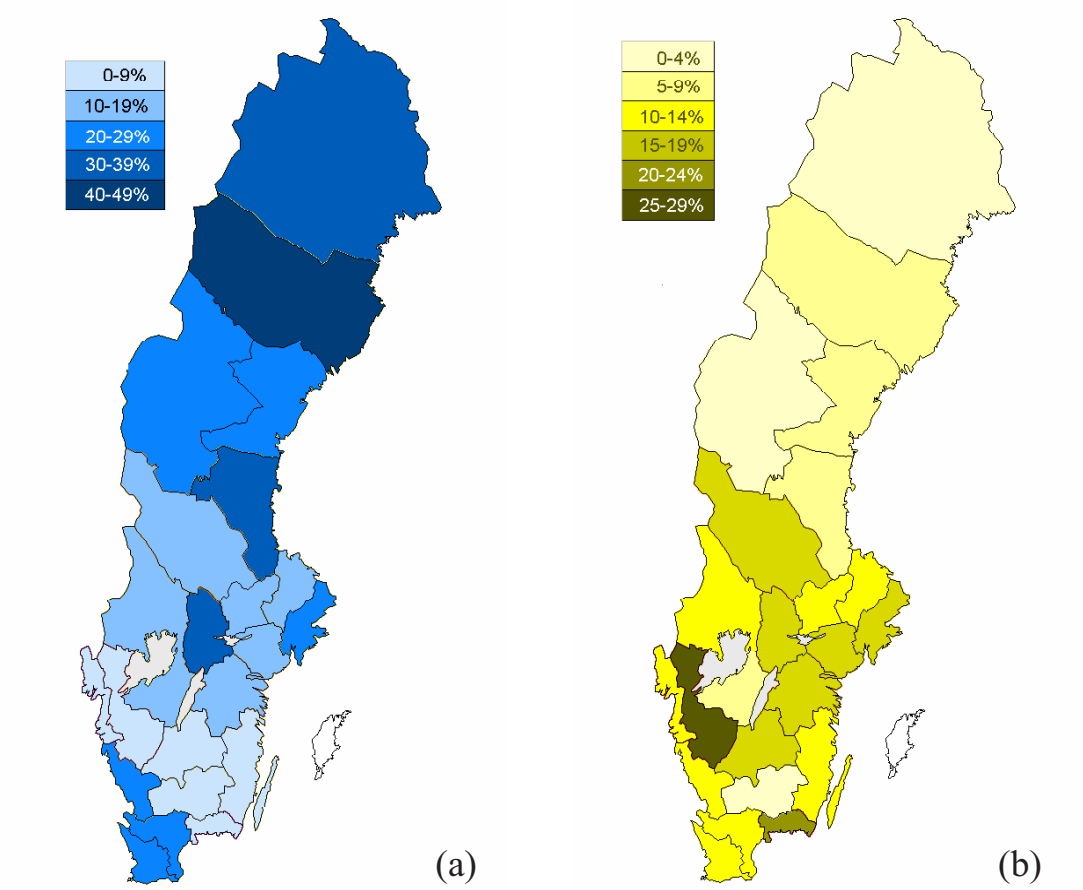

Fig. 4. Proportion of moose examined between 1985 and 1989 with signs of (a) ELA and (b) MWS for each Swedish County.

ELA. That emaciation might be an effect is supported by the fact that *E. alces* can cause a nervous disorder, with lack of co-ordination, making it difficult to forage (Stéen and Rehbinder 1986, Stéen et al. 1989, Stéen and Roepstorff 1990). Stuve (1986) found that the parasite has a negative influence on the general condition and he found that the difference in carcass weights between infected and non-infected animals increased with age. Conversely, moose experimentally infected with *E. alces* retained their normal weight when fed ad libitum (Stéen et al. 1998a).

INF differed from other diseases in being positively related to condition. Animals with INF probably die acutely before they loose condition or become emaciated, which is in contrast to animals with ELA and MWS.

Health Status

The disease pattern, including deaths caused by wild predators seen in our sample differs from that seen elsewhere (Lankester 1987, Guilazov 1998, van Ballenberghe and Ballard 1998), suggesting that moose populations in Sweden might be different from other populations. For example, densities and human harvest rates are higher in Sweden than in Russia and North America, but predation is lower. We believe that the disease patterns observed are masked worldwide by predation. The scenario of the Swedish moose loss, with the exception of hunting, will probably change over time and become more similar to the causes of deaths (e.g., predation) observed in other countries.

Another difference between the Swedish

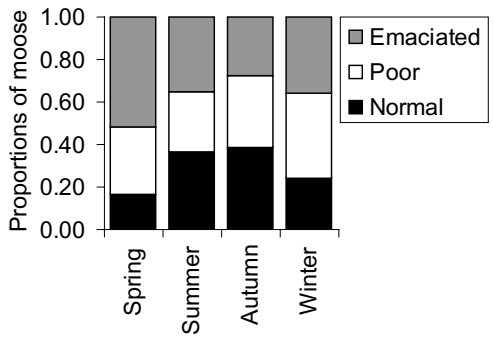

Fig. 5. Proportion of moose in normal condition, poor condition, or emaciated split by season (spring n=183, summer n=73, autumn n=166, and winter n=131). Moose culled during regular sports hunting excluded.

and North American moose populations is the origin of the diseases (Borg 1956, Nilsson 1971, Borg and Nilsson 1985, Borg 1987, Lankester 1987, Lankester and Samuel 1998, Stéen et al. 1998b). An interesting and notable observation is that moose in North America have few Eurasian parasites but have acquired new parasites and diseases from indigenous wild ungulates and livestock (Lankester 1987, Lankester and Samuel 1998). The diseases observed in Sweden are, as far as we can evaluate, specific to moose and not transmitted from livestock or deer, with the exception of malignant catarrhal fever (Warsame and Stéen 1989). Most of the diseases in American moose have not been diagnosed in Swedish moose. The viroses epizootic haemorrhagic disease, bluetongue, western equine and St. Louis encephalitis, Norway virus, California encephalitis virus, and contagious ecthyma have not been diagnosed in Swedish moose or livestock. Parainfluenza type 3 and infectious bovine rhinotracheitis are, however, known in Swedish livestock (Moreno-Lopéz 1979, SJV 1994) and in reindeer (Rockborn et al. 1990) but not moose. Further, the bacterial and parasitic diseases in American moose (leptospirosis, brucellosis, necrobacillosis, *Toxoplasma gondii*, *Entamoeba bovis*, *Paramphistomum* spp., *Fascioloides magna*, *Taenia ovis*, *T. krabbei*, *Echinoccocus granulosus*, *Thysanosoma actinioides*, *Orthostrongylus macrotis*, *Parelaphostrongylus tenuis*, *Elaeophora schneideri*, *Onchocerca cervipedis*, *Setaria yehi*, *Rumenfilaria andersoni*, *Dermacentor albipictus*, *Cephenemyia jellisoni*, *C. phobifera*, and *Haematobosca alcis*), have not been found nor reported for Swedish moose (Nilsson 1971, Stéen et al.1998b).

Despite all the diseases and parasites enumerated, Lankester (1987) and Lankester and Samuel (1998) proclaim American moose to be generally healthy. They explain this status of health partly by the fact that American moose occur at a low densities (0.1-0.6/km^2), which reduces the transmission rate of parasites and diseases. Also van Ballenberghe and Ballard (1998) state that moose host a variety of diseases and parasites that are seldom a major limiting factor for population growth. On the other hand, Wobeser (1994; 3) declared: "Although most infectious agents do not result in obvious disease, the host must pay a price for harboring parasites that live, grow, and reproduce at expense of the host. Interactions between parasites and other stress factors can be important". He also stated that diseases in wild animals are often considered only in terms of death or obvious physical disability, probably because these are readily identified parameters. In other words, the effect of diseases on wild populations may be much greater than is evident by simply counting the dead or

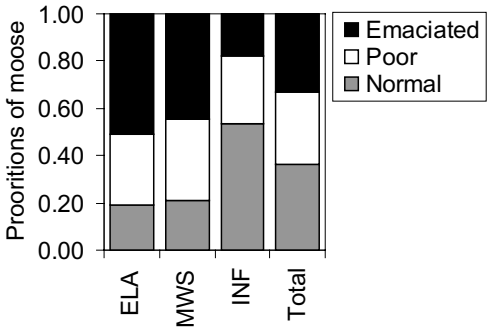

Fig. 6. Proportions of moose in normal condition, poor condition, or emaciated split by diagnosis (ELA n=126, MWS n=81, INF n=28, and 'Total' n=642).

maimed. The Swedish moose population is dense, with up to 1.0-1.5 moose/km^2, which may increase the risk of disease and parasite transmission as discussed by Lankester and Samuel (1998). It is unknown how our data relate to density, but for MWS there are indications that density dependence might be the ultimate reason for its appearance (Broman et al. 2002a).

Does the large number of diseased moose seen in Sweden during the latest 2 decades indicate anything about the health status of Swedish moose? The opportunity to see and report abnormal moose is greater in the intensely managed forests of Sweden than in the wilderness of North America and Russia. From 1985 to 1989, approximately 200 moose per year were necropsied and diagnosed in Sweden, compared to a total of 420 between 1947 and 1982 (Borg 1987, 1991). Currently, up to 100 moose per year are examined at the National Veterinary Institute, Uppsala, Sweden (Mörner 2001). If the necropsies reflect number of deaths, the impression is an increased mortality during the 1980s. However, the moose population increased sharply in the 1980s (Cederlund and Markgren 1987, Cederlund and Bergström 1996), making it more likely to find diseased or sick animals. Thus, increased observations do not necessarily indicate a higher absolute mortality risk. Broman et al. (2002b) estimated natural death risks (i.e., culling and traffic excluded) to be < 4% for adults in the area where the highest incidences of natural moose mortalities were recorded (community of Mark) between 1991 and 1998. There were no wild predators in Mark during this period implying that natural mortalities were synonymous with mortality caused by disease. Without predators it appears that the mortality risk due to disease has been quite low in the 1980s and 1990s, but the relative risk of ELA and MWS was high. Our description of diseases and natural mortalities differs from that of Guilazov (1998) who described predation as the primary mortality factor in moose of Northern Russia. Based on our results, the risk of being killed by predators was low in the late 1980s. Only 25 of the moose in the entire sample were killed by wolves suggesting that predation was not common.

Future Scenario

Currently, winter populations of moose and roe deer in Sweden are approximately 250,000 and 1.5 million, respectively (Stéen et al. 1998b). Predators have increased substantially in recent decades. Wolves were protected in Sweden when estimated numbers were ≤ 10 individuals; today there are approximately 67-81 (Aronsson et al. 2001). In the 1930s, 130 brown bears were known, and in 60 years (1996) they had increased to 800-1,300 (SOU 1999). It is realistic to believe that, despite future increase in numbers and range expansion, harvesting of predators will remain banned or be highly regulated by the Swedish government. Higher moose mortality from predation will no doubt result.

Interactions between risk of disease and predation may result in compensatory rather than additive death. For moose, the risk of being killed by wolves or bears depends on age (e.g., van Ballenberghe 1987, Ballard and van Ballenberghe 1998, SOU 1999, Wikenros 2001). Ballard and van Ballenberghe (1998) showed that calves and old cows are the primary target of wolves. Predation by bears was the most frequent cause of early calf mortality (Franzmann and Schwartz 1986; Boertje et al. 1987, 1988). Franzmann and Schwartz (1986) estimated bear density in Alaska to be 19.0/100 km^2. This compares to a desirable density of at least 0.7/100 km^2 anticipated outside the Swedish reindeer husbandry area (61°N to 69°N) (SOU 1999). Swedish studies of bear predation on moose calves indicate a 20-25% loss, while bears accounted for 0.5-1.5% of adult mortalities (SOU 1999). Studies in both Sweden and North America indicate that bear predation on calves is additive, at approxi-

mately 3 calves per bear/year (SOU 1999). SOU (1999) reports that natural mortality of adults was approximately 5% in an area with no predation and that the additive loss of adults by bear predation was 0.5-1.5% per bear/year. While predation on calves is mostly likely additive, van Ballenberghe (1987) stated that predation on adults was mostly compensatory, with the various mortality factors tending to substitute more for each other. Ballard and van Ballenberghe (1998) cite Mech et al. (1995) whose results show that wolf-predated calves and adults during winter have low marrow fat values, indicating poor condition. Also, Peterson (1977) reported that wolves from Isle Royale prey on heavily parasitized, diseased, or otherwise inferior moose. This information suggests that in the future, the weak, vulnerable, sick, and old moose will be preyed upon before dying from a disease. We suggest that the panorama of moose diseases seen in the future will differ from that seen during the 1980s and 1990s by being less visible due to increasing predation.

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