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Authors: Broman, Emil, Wallin, Kjell, Steén, Margareta, and Cederlund, Göran

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'Mass' deaths of moose *Alces alces* in southern Sweden: population level characterisation

Emil Broman, Kjell Wallin, Margareta Steén & Göran Cederlund

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Moose *Alces alces* suffering from Moose Wasting Syndrome (MWS) have been observed in Sweden since 1985. In this study a population characterisation of MWS is done based on moose delivered to the National Veterinary Institute of Sweden during 1985-1989, public reports of non-traumatic moose deaths during 1991-1998, and radio-collared moose in one high- and one low-report frequency area. Moose suffering from MWS have been observed nationwide, but deaths from non-traumatic causes, including MWS, are most frequently reported from the county of Älvsborg. Nationwide reports of non-traumatic deaths peaked in 1992, but no peak was observed in Älvsborg. We suspect that the temporal as well as the spatial patterns observed were largely due to information about the MWS by the media, research activities and local concern. The age-specific mortality pattern of non-traumatic deaths did not differ, in either sex, between one high- and one low-report frequency area. The risk of dying from non-traumatic causes increased with moose age. Reports of female moose either suffering from MWS or having died from other non-traumatic causes largely outnumbered reports of males (1,349 and 387, respectively). Our results of age-specific risks suggest, however, that females are less vulnerable to non-traumatic mortality than males. The discrepancy between report frequencies and death risks is explained by the fact that the population sex ratio is female-biased (by sport harvest), and few males reach the age at which the risk of dying from non-traumatic causes increases. In the area from which most of the reports came the non-age-specific yearly risk of dying from non-traumatic causes, including MWS, was estimated to be 3.5% for females and 3.0% for males. Because there are other non-traumatic causes of death, the risk of dying from MWS exclusively is lower than our risk estimates indicate. There is no immediate risk of local extinction of moose due to MWS, as we estimate that the population would increase if culling was stopped.

Key words: Älvsborg, death risk, disease, moose, Moose Wasting Syndrome (MWS), mortality, Sweden

Emil Broman & Kjell Wallin, Department of Applied Environmental Science, Göteborg University, P.O. Box 464, SE-405 30 Göteborg, Sweden - e-mail addresses: emil.broman@miljo.gu.se (Emil Broman); kjell.wallin@miljo.gu.se (Kjell Wallin)
Margareta Steén, Department of Veterinary Microbiology, Swedish University of Agricultural Sciences, P.O. Box 7070, SE-750 07 Uppsala, Sweden - e-mail: Margareta.Steen@x.lst.se*
Göran Cederlund, Department of Conservation Biology, Swedish University of Agricultural Sciences, Grimsö Wildlife Research Station, SE-730 91 Riddarhyttan, Sweden - e-mail: Goran.Cederlund@nvb.slu.se

*Present address: County Administrative Board of Gävleborg, SE-801 70 Gävle, Sweden

Corresponding author: Emil Broman

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Beginning in the mid-1980s, reports of dead or sick moose *Alces alces* in Sweden gave rise to public concern about the moose population and were thought to indicate some sort of environmental hazard in terrestrial habitats, such as mineral leakage in the soil due to acid rain (Gadolin 1993, Frank, Galgan & Petersson 1994). Aware of the ongoing acidification of lakes in large parts of Sweden, hunters in the county of Älvsborg (hereafter referred to as 'Älvsborg'; Fig. 1) were especially anxious since most reports of moose found dead appeared to come from Älvsborg. This concern should be understood particularly in light of the mass death at the time of other species, such as the harbor seal *Phoca vitulina* (Dietz, Heide-Jørgensen & Härkönen 1989) and the red fox *Vulpes vulpes* (Heide-Jørgensen, Härkönen, Dietz & Thompson 1992, Lindström, Andrén, Angelstam, Cederlund, Hörnfeldt, Jäderberg, Lemnell, Martinsson, Sköld & Swenson 1994) observed in different parts of Europe. Veterinary analyses of dead moose showed signs of a previously unknown, complex syndrome (Stéen, Diaz & Faber 1993). In this paper we will use the term 'Moose Wasting Syndrome' (MWS) to refer to the condition (Stéen, Faber & Oksanen 1998) although the literature offers many other names such as 'Älvsborg disease' (Anon. 1995), 'erosive/ulcerative alimentary disease' (Stéen et al. 1993), 'wasting disease' (Merza, Larsson, Stéen & Morein 1994) and 'bovine viral diarrhoea/mucosal disease-like syndrome' (Rehbinder, Gimeno, Belak, Belak, Stéen, Rivera & Nikkilä 1991). The term 'Moose Wasting Syndrome' (MWS) should not be confused with Chronic Wasting Disease (CWD) of North American deer (Gross & Miller 2001).

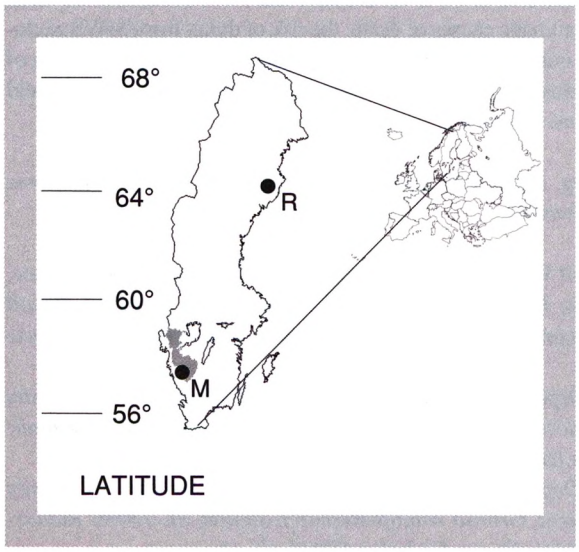

Figure 1. Location of sites in Sweden mentioned in the text; the grey area indicates the county of Älvsborg (Älvsborg) covering 11,395 km^2. The capital letters M and R indicate the communities of Mark and Robertsfors covering 940 and 1,298 km^2, respectively.

Although there is still no clear answer to what causes the syndrome (Broman, Wallin, Stéen & Cederlund 2002), two broad groups of hypotheses have been suggested: food related and host-parasite related. In the food-related hypotheses, the syndrome is thought to have one of three ultimate causes: acidification/liming, browser density/food production or pollution.

At an individual level MWS is characterised by catarrhal to haemorrhagic enteritis, atrophied lymphoid organs and/or numerous erosive necrotising lesions of the digestive mucosa (Stéen et al. 1993). Histopathologically the mucous membranes in animals with MWS reveal inter- and intracellular oedema, erosions and ulcers (Stéen et al. 1993). Sick moose display symptoms including anorexia, emaciation, weakness, lack of wariness towards humans, impaired vision, diarrhoea and circling (Stéen et al. 1993).

At a population level no thorough characterisation of MWS has been done. However, it has been noted that MWS preferentially affects middle-aged females (Sand & Cederlund 1992) and Älvsborg is supposed to be the most affected area (Cederlund, Stéen & Wallin 1993, Frank et al. 1994). In Älvsborg the syndrome has been thought to be a serious threat to the moose population (Frank 1994, Merza et al. 1994).

Moose are the most important game species in Sweden, and to maintain sustainable harvest quantitative information about the extent of different causes of death are needed. Here we describe the spatial and temporal pattern of MWS incidence, compare the sex- and age-

specific demography and seasonal deaths of affected and unaffected moose and estimate the maximal death risk due to MWS. We hope that observations at the population level, including spatial distribution and frequency of moose deaths according to age and sex class, may shed light on the cause of MWS.

Methods

Animal sample

We considered four different data sets (samples A-D), separated according to method of collection and moose health status.

Sample A

Sample A consisted of moose found dead or euthanised and brought to the National Veterinary Institute of Sweden (SVA) during 1985-1989 (N = 692; Table 1). Age was determined when the sample submitted allowed it, then the carcass was necropsied and a diagnosis of MWS (or not) was made (Stéen et al. 1993). The diagnosis was somewhat arbitrary because no single diagnostic criterion was exclusive (Stéen et al. 1993). We did, however, exclude moose killed by vehicles or hunters. Thus, this sample consists of moose in which the cause of death was not traumatic (hereafter referred to as 'non-traumatic deaths').

The reliance on reports from the general public means that the data may be biased. For instance, moose conspicuous by their behaviour may have been euthanised and sent to the SVA for necropsy more often than moose found dead. When we tested whether MWS was more frequent among euthanised animals than among those found dead, however, we found no difference (Fisher exact test: P = 0.722).

Table 1. Frequency of reported and diagnosed deaths from non-traumatic causes (any dead moose without any sign of injury from vehicles or sports hunting) and fraction of diagnosed moose with signs of Moose Wasting Syndrome (MWS). See the 'Methods' section for a description of Samples A and B.

Sample	Sex	Period	Reports	Diagnosis	MWS Fraction
A	♀	1985-1989	404	400	0.21
B	♀	1991-1998	945	86[2]	0.64[3]
A+B	♀	1985-1998	1349	486	-
A	♂	1985-1989	188	186	0.07
B	♂	1991-1998	199	17[2]	0.25[3]
A+B	♂	1985-1998	387	203	-
A	All[1]	1985-1989	692	677	0.12
B	All[1]	1991-1998	1361	105[2]	0.53[3]
A+B	All[1]	1985-1998	2053	782	-

[1] Including moose of unregistered sex
[2] Diagnosed only during 1991-1994
[3] Moose for necropsy not randomly sampled

Sample B

Sample B consisted of reports, by the public, of non-traumatic moose deaths during 1991-1998 (N = 1361; see Table 1). In most cases we registered the animal's sex (84%), and whether it was a juvenile (<1 year) or not (43%), as well as the date of discovery and/or death (81%) and approximate location (93%; within a radius of 1 km). For 76% of adults (≥1 year), we determined their exact age by counting cementum annuli in the first molar (Sergeant & Pimlott 1959).

At the beginning of the period 1991-1998 the public was informed about the research in the media and asked to report sick and dead moose. Hunting authorities were informed verbally (Cederlund et al. 1993).

Between 1991 and 1994, 3% of the moose reported dead and 64% of the euthanised moose were necropsied and clinically investigated. For clinical examination purposes, moose with signs of MWS were preferred; therefore necropsied moose were not randomly selected. Consequently, MWS was more common among euthanised animals than among those found dead (Fisher exact test: P = 0.002). That sampling bias meant that we could not assess whether MWS was a more common cause of non-traumatic moose deaths (referred to as the MWS fraction) during the early years (1985-1989) than during the later years of the survey (1991-1994). Assuming that the bias was independent of area and time of year, we used the MWS fraction to examine the spatial and seasonal patterns of MWS.

Sample C

Sample C consisted of moose culled or killed by vehicles in Älvsborg during 1991-1998. Both culling and traffic collisions with moose have to be reported by law. We determined the age of 1,758 adults by counting cementum annuli in the first molar (Sergeant et al. 1959).

Sample D

Sample D consisted of radio-collared moose from a high-frequency report area ('Mark' in Fig. 1) and a low-frequency report area ('Robertsfors' in Fig. 1). During 1994-1998 we radio-marked adult moose in Mark (N_{female} = 128, N_{male} = 99) and during 1991-1998 in Robertsfors (N_{female} = 216, N_{male} = 152). During January-March each year we immobilised adult moose with Ethorphine and Xylazine (Sandegren, Pettersson, Ahlqvist & Röken 1987) using a dart gun from a helicopter. Each moose was fitted with a radio collar and uniquely numbered ear tags. After marking, we located the animals by triangulation at least once a month. If a moose had not moved since the previous triangulation, we checked by visual contact whether or not the

moose was dead. If the date of the last observation and that of the documented death were several months apart, we used the median date as approximate death date. Swedish law ensured that most deaths by hunting or traffic were reported.

Frequency of moose deaths

From Samples A and B we constructed age, and spatial, temporal and seasonal frequency distributions of all non-traumatic deaths as well as deaths of moose with signs of MWS. For comparison, we constructed the age distribution of moose that died from hunting and traffic accidents, using data from Sample C. From Samples A and B we estimated the fraction of deaths from MWS to other non-traumatic moose deaths.

Carcass report rate and death-to-discovery time lag

To estimate the rate at which the public reported moose deaths from non-traumatic causes, we left *in situ* radio-marked moose that had died from non-traumatic causes. We registered whether, and when, the public reported these carcasses. The method was applied in Mark and enabled us to also estimate the mean, median and maximum time lag between death of the moose and report by the public.

Mortality rate

We made two independent estimates of mortality from non-traumatic causes, using a multiple decrement life table with a synthetic cohort and the Kaplan-Meir method with a staggered entry design.

Multiple decrement life table with a synthetic cohort

When individuals are subject to a number of mutually exclusive hazards two probabilities can be estimated: the probability of dying from a specific cause in the presence of other causes, and secondly, the probability of dying from a specific cause in the absence of other causes of death. We constructed a multiple decrement life table as suggested by Carey (1989) using the known-age moose from Samples B and C and the fraction of deaths from different causes. We compared the probability of dying from non-traumatic causes in the presence of hunting and traffic with the probability of dying from non-traumatic causes in the absence of other causes.

Kaplan-Meir method with a staggered entry design

We performed age-specific, seasonal and population average survival analyses based on radio-marked individuals from Sample D. To estimate death rates and to do inferential statistics we applied the method suggested by Pollock, Winterstein, Bunck & Curtis (1989).

Statistical analyses

Except for the Wilcoxon signed-ranks test (Siegel & Castellan 1988) all statistical tests were performed using the SAS package (SAS 1999).

Results

Spatial distribution

The first incidence of MWS was observed in 1985 in the county of Dalarna in central Sweden (Feinstein, Rehbinder, Rivera, Nikkilä & Stéen 1987). Later, moose showing similar clinical signs were observed throughout most of Sweden (Stéen et al. 1993).

The national distribution of non-traumatic moose deaths from 1985 to 1998 showed concentrations in southwestern and eastern Sweden (Fig. 2). The frequency of reports of MWS in Älvsborg was higher than in other parts of Sweden (see Fig. 2), but the MWS fraction was not significantly different between counties (Sample A: $\chi^2 = 22.677$, df = 21, P = 0.361; Sample B: $\chi^2 = 15.908$, df = 14, P = 0.319; Fig. 3).

Temporal distribution

From 1985 onwards, except for 1990 when no survey was conducted, the public in Sweden reported annually about one hundred non-traumatic moose deaths. Reports routinely collected at the SVA in Uppsala (1985-

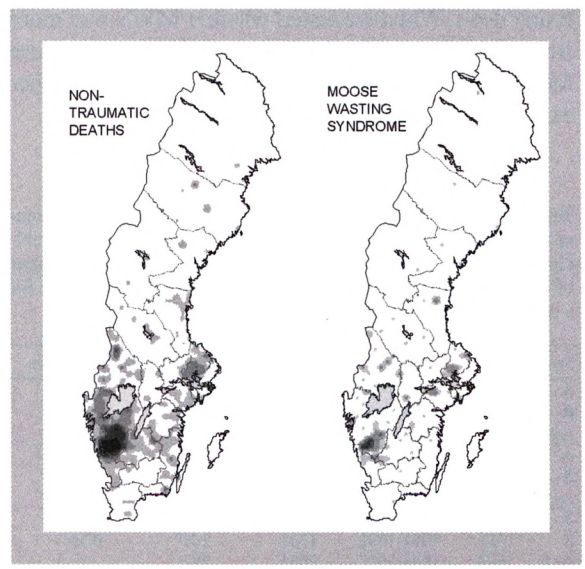

Figure 2. Reports of non-traumatic moose deaths during 1985-1998 (left) and incidences of Moose Wasting Syndrome (MWS) during 1985-1994 (right). The darker the colour the higher the density of reported animals.

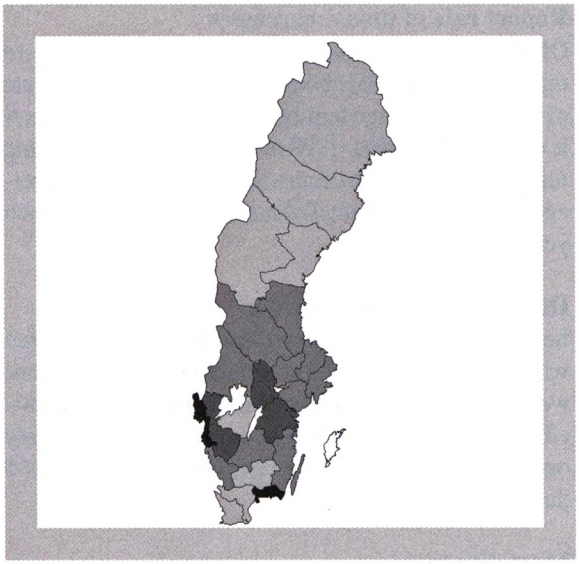

Figure 3. Fractions of 677 moose exhibiting signs of MWS routinely examined by the National Veterinary Institute (excluding animals killed by vehicles or sports hunting) during 1985-1989. Black indicates a fraction of 30-39%, dark grey 20-29%, grey 10-19% and light grey 0-9%.

1989) included few reports from Älvsborg and none from Mark. In 1992, after appeals were made to the public to report dead or sick moose, there was a strong peak nationwide in reports of non-traumatic deaths (Fig. 4). No peak was observed in either Älvsborg or Mark, but the largest number of moose deaths was reported in these counties in 1992 (see Fig. 4).

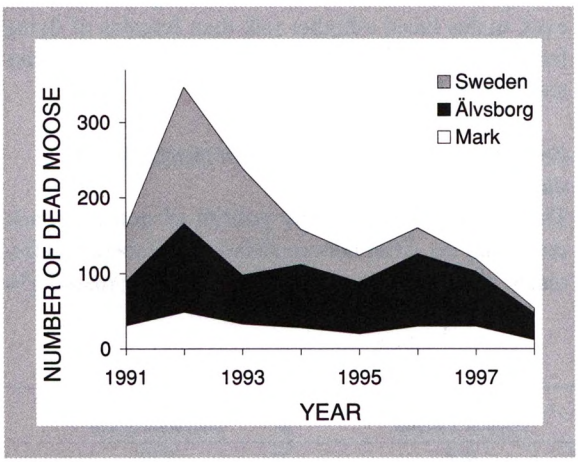

Figure 4. Yearly reports of moose found dead from non-traumatic causes at three geographic levels (Sweden = country, Älvsborg = county and Mark = community). This sample consists of animals with MWS as well as others in which death was non-traumatic and the cause of death unknown.

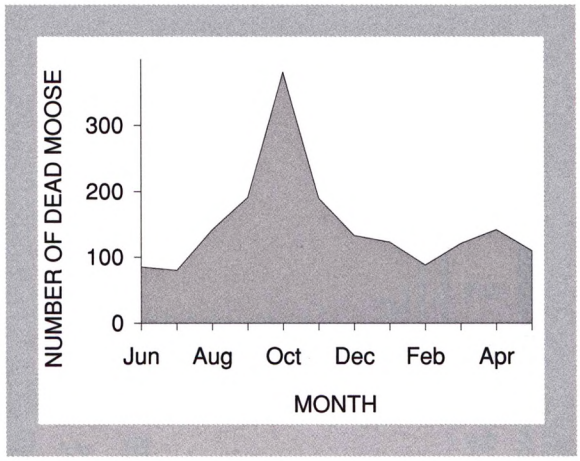

Figure 5. Monthly reports of 1,097 non-traumatic moose deaths during 1991-1998.

Seasonal distribution

The frequency of non-traumatic deaths peaked in October (Fig. 5), but there was no difference in monthly non-traumatic death risks as estimated from the radio-collared moose in Mark (females: Z-test, $P > 0.05$ for all pairs of months; males: Z-test, $P > 0.05$ for all pairs of months; Fig. 6). There was no significant difference in monthly risk of dying from non-traumatic causes between the sexes ($\chi^2 = 1.12$, df = 1, $P = 0.290$). Monthly MWS fractions were not correlated with the number of reports of non-traumatic deaths (Sample A: Spearman correlation, $r_s = 0.29371$, N = 12, $P = 0.3541$; Sample B: Spearman correlation, $r_s = 0.20387$, N = 12, $P = 0.5251$).

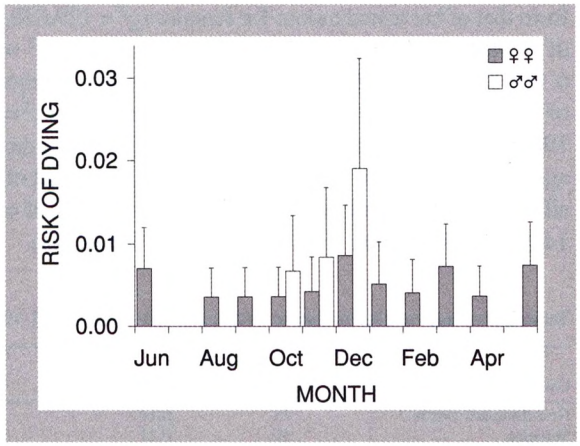

Figure 6. Monthly death risk from non-traumatic causes, as estimated from radio-collared moose in Mark during 1994-1999. Filled bars refer to female and open bars to male animals. Handles indicate standard error.

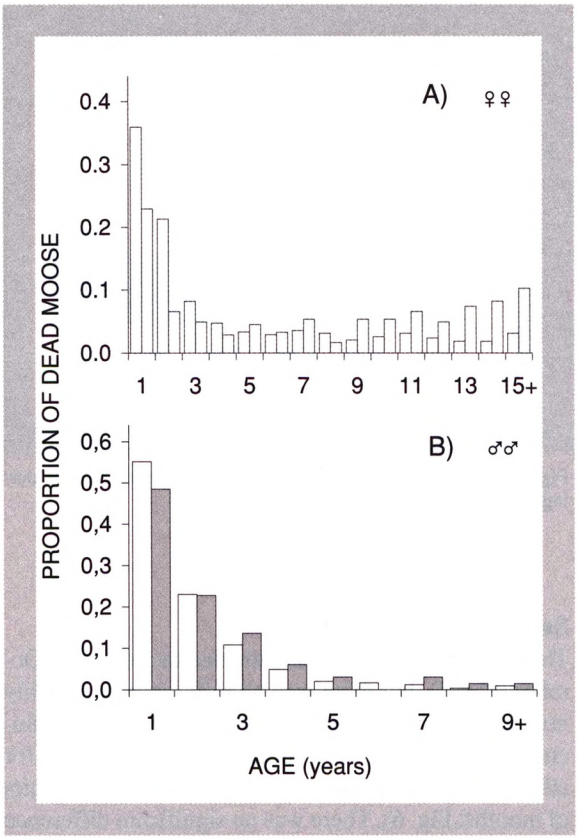

Figure 7. Distribution of moose age at death during 1991-1998. Calves (<1 year) are not included. White bars refer to healthy moose shot or killed by vehicles (females: N = 919; males: N = 842), and grey bars to non-traumatic moose deaths (females: N = 235; males: N = 62).

Age distribution

The age distribution of non-traumatic deaths differed from that of harvested moose for females ($\chi^2 = 129.670$, df = 14, $P < 0.0001$; Fig. 7A) but not for males ($\chi^2 = 6.146$, df = 8, $P = 0.631$; see Fig. 7B). The distribution of harvested moose differed between the sexes ($\chi^2 = 198.232$, df = 14, $P < 0.0001$; see Fig. 7). However, the age distribution of MWS was not different from that of all other non-traumatic deaths (females: $\chi^2 = 19.878$, df = 14, $P = 0.134$; males: $\chi^2 = 8.986$, df = 8, $P = 0.344$).

Report rate of moose carcasses

Of 16 dead radio-collared moose left *in situ*, 13 were reported by the public giving an estimated discovery rate of 81%. The average number of reported non-traumatic deaths during 1991-1998 in Mark was 26. Correcting for unreported moose deaths, this suggests that on average about 32 moose died of non-traumatic causes each year in Mark.

Death-to-discovery time lag

Several of the non-traumatic deaths were discovered without any time lag; in three cases because the moose were euthanised. More than half of the moose reported were discovered within one week of death, and only one of 16 was missed for longer than two months (64 days).

Mortality rate

Estimates from a multiple decrement life table with a synthetic cohort

The fraction of deaths from non-traumatic causes was 13.9% for females and 5.7% for males (Table 2). The probability of dying from non-traumatic causes, both in the presence and in the absence of other death causes, increased with age; in females, for which data were available for moose >10 years old, mortality increased with age (Fig. 8).

On average the yearly risk of dying from non-traumatic causes, in the absence of other causes of death, was 3.5% in females and 3.0% in males. In the presence of other causes of death the probability of dying from a non-traumatic cause was 3.1% in females and 2.9% in males. By pairwise comparisons of the age classes (all ages >8 years combined into one age class), however, males faced a higher risk than females of dying from a non-traumatic cause (Wilcoxon signed-ranks test, N = 9, $P < 0.05$).

Estimates based on the Kaplan-Meir method with a staggered entry design

The age-specific mortality pattern of non-traumatic deaths did not differ between Mark and Robertsfors (females: $\chi^2 = 2.634$, df = 1, $P = 0.105$; Fig. 9A; males: $\chi^2 =$

Table 2. Average number of moose deaths in Mark during 1991-1998.

	Female		Male		Total	
Cause of death	N	Fraction of deaths	N	Fraction of deaths	N	Fraction of deaths
Non-traumatic deaths [1]	23	0.14	9	0.06	32	0.10
Vehicles	19	0.11	9	0.06	28	0.09
Hunting	127	0.75	135	0.88	262	0.81
Total	169	1.00	153	1.00	322	1.00

[1] Labels any dead moose without any sign of injury from vehicles or sports hunting. The number was calculated by dividing the number of reports by the estimated recovery rate of 81%

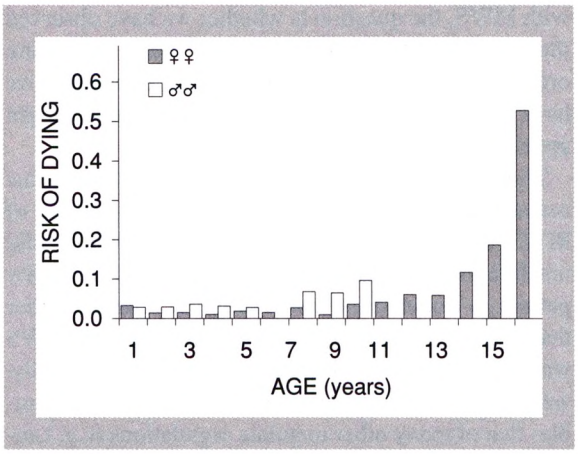

Figure 8. Age-specific risk of dying from non-traumatic causes including MWS, as estimated from data on dead moose reported by the public during 1991-1998 for females (grey bars) and males (white bars).

0.135, df = 1, P = 0.713; Fig. 9B). When data from Mark and Robertsfors were pooled, the non-traumatic age-specific death risk was significantly higher in males than in females ($\chi^2 = 5.173$, df = 1, P = 0.023).

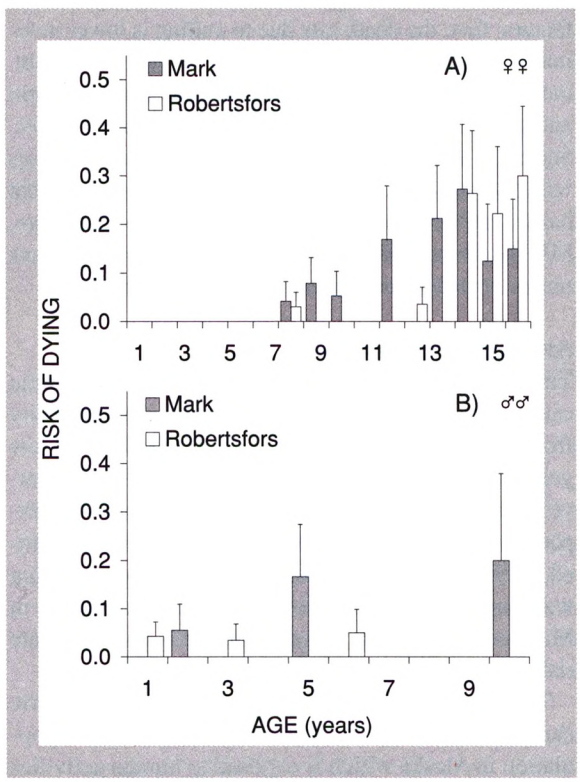

Figure 9. Age-specific risk of dying from non-traumatic causes including MWS, as estimated from data on radio-collared moose from the communities Mark (grey bars) and Robertsfors (white bars). Handles indicate standard error.

Discussion

Methodological consideration

Earlier concerns about the population consequences of MWS were partly due to a reporting bias. Although initial research on MWS may not have been as systematic as our study, it influenced public perceptions about moose dying from non-traumatic causes. Although one might suspect that earlier reports were unreliable, we cannot simply dismiss them (Hilborn & Mangel 1997). In our study, instead of discarding any data we therefore compared different independent estimates. Our analyses reveal that some earlier interpretations of the impact of MWS may have been incorrect.

Spatial, temporal and seasonal pattern of the non-traumatic deaths

The concentration of non-traumatic deaths in southwestern and eastern Sweden (see Fig. 2) and the increasing number of reports in the mid-1980s (Stéen, Frank, Bergsten & Rehbinder 1989) and the early 1990s (see Fig. 4) may reflect disease outbreaks. Alternative explanations of changes in moose abundance and/or in the probability of finding dead moose, however, may be just as plausible. Despite this, it was previously suggested without reservation that the concentration of dead animals in the southern parts of Älvsborg in the 1990s (see Fig. 2) was due to MWS (Frank et al. 1994). Certainly the density of reported moose with signs of MWS in Älvsborg was higher than that in other parts of Sweden (see Fig. 2), but when compared to all non-traumatic deaths, it was not higher than expected. It should be stressed here that the estimates of the MWS fraction during 1991-1994 might be unreliable because of the biased way in which moose were sampled for clinical examination (see the 'Methods' section). Nevertheless, we cannot exclude the possibility that the numerous reports from Älvsborg in the 1990s may indeed have reflected a greater prevalence of MWS there than elsewhere in Sweden. The lack of difference in yearly death risks estimated from radio-collared moose in Mark and Robertsfors suggest that Älvsborg was not more affected by MWS than other parts of Sweden.

During the 1980s, when MWS was first discovered, the moose population in Sweden was the highest recorded in modern times (Cederlund & Markgren 1987). With or without an increase in the mortality rate, higher density should lead to a greater number of moose found dead. A similar pattern was also found for moose sent to the SVA for post-mortem examination, which increased tenfold in the mid-1980s (Stéen et al. 1989). A minor fraction of these clinically examined moose

showed signs of MWS; most, however, had been killed by other diseases or traumas (Stéen et al. 1993). The tenfold increase in moose dying from non-traumatic causes sent to the SVA therefore cannot be explained by MWS. Rather, it appears that the discovery of MWS and the increase in moose deaths in the 1980s were coincidental.

In the early 1990s, for a better individual characterisation of MWS, appeals to the public resulted in numerous reports of non-traumatic deaths (see Fig. 4). During 1994 the practice of necropsying moose found dead stopped, and this most probably caused the subsequent decline in the countrywide reports (see Fig. 4). At that time the public also learned from the media that large numbers of dead moose were being found in Älvsborg, possibly resulting in a continued public inclination in Älvsborg to report non-traumatic moose deaths, and a decrease to do so elsewhere in Sweden.

Between parturition (May-June) and the hunting season (September-December) the Swedish moose population is larger than during the rest of the year. Therefore, if deaths were independent of season, most reports should be expected to be made during summer. The frequency of reported moose found dead from non-traumatic causes peaked in October. The reason for the apparent seasonal pattern, however, is most likely that people spend more time in the field during autumn, hunting or picking berries. The monthly non-traumatic death risk estimated from radio-collared moose in Mark, supports this explanation (see Fig. 6). Although it is possible that MWS may show a seasonal pattern different from that of other non-traumatic deaths, we found no support for this argument because the MWS fraction did not correlate with the seasonal pattern of non-traumatic deaths.

Age-specific mortality rate

Age-dependent mortality is sometimes documented in studies of mammal populations (e.g. Loison, Festa-Bianchet, Gaillard, Jorgensen & Jullien 1999). The role of intrinsic versus extrinsic factors in senescence studies on wild populations has rarely been explored (Finch 1994, Rose 1994). Because senescence has been defined as "an increase with age in that portion of the age-specific mortality that can be attributed to intrinsic changes in the organism" (Stearns 1992), an increase in mortality with age may have origins other than senescence. For example, in game species the largest individuals, and usually therefore the oldest (especially in species where age correlates with size) may exhibit the highest mortality risk if hunters prefer big trophies. This hazard is associated with age but only coincidental to senescence. Keeping in mind the mortality pattern associated with MWS, the question is whether we have observed the effect due to a novel extrinsic factor preceding ordinary senescence in Swedish moose or whether we have just described one way of how senescence might appear in moose.

There are at least three reasons why we suspect that senescence explains the age-specific prevalence of MWS: first, the age-specific mortality rate of moose did not differ between Mark and Robertsfors. If the mortality pattern in Robertsfors has its origin in intrinsic rather than extrinsic factors, the risk of moose dying from MWS was most probably increased by senescence; secondly, we found a mortality pattern for both sexes that resembles that of many other ungulate populations (e.g. Loison et al. 1999), namely that male survival is lower than female survival, with the gender difference increasing with age (see Fig. 8). And thirdly, the age distribution of moose classified to have MWS did not differ from the age distribution of moose reported dead from all other non-traumatic causes.

Sex-specific mortality rate

As previously mentioned, it has been suggested that MWS preferentially affects mature females (Sand & Cederlund 1992). We suggest that this is due to two main factors: first, the death rate due to culling is lower in females than in males (E. Broman & K. Wallin, unpubl. data). This gives rise to a female-biased adult sex ratio and thus makes females more numerous than males. Secondly, the hazard of non-traumatic causes increases with age, and very few males reach the age at which that hazard rate becomes high (see Figs. 7B & 8). Therefore, MWS does not affect females any more than it affects males. Instead, our analyses reveal the opposite.

Average mortality rate

The annual average death rate from non-traumatic causes was <4% for both females and males. Mortality from culling was several times higher. In the past few years the moose population in Mark has declined as harvest rates have increased as a result of a long-term experimental manipulation of moose density, which started in 1997. Our unpublished data suggest that if culling was stopped, the finite rate of increase (λ) of moose in Mark would be 1.19. Therefore, there is no immediate risk of local moose extinction due to MWS.

The observed mortality rate from non-traumatic causes of moose in Sweden is similar to that of a population in Alaska which is exposed to human activities (e.g. traffic, hunting and poaching), wolves *Canis lupus*, and brown bears *Ursus arctos* (Bangs, Bailey & Portner 1989). Since the average mortality rate depends on

both age-specific mortality and the population age structure such comparisons may be equivocal. However, the Alaska population also exhibited a death rate that increased with age.

Moose Wasting Syndrome versus other non-traumatic deaths

In this study we could not estimate the mortality rate directly due to MWS, for two reasons. First, there is no unambiguous criterion for separating MWS deaths from other non-traumatic deaths in moose. Second, even if a valid criterion existed we still have no unbiased data (see the 'Methods' section, Sample B) contemporary to the other requisite of our mortality rate models. Because MWS incidences were included in the total for non-traumatic deaths, however, the non-traumatic mortality rate may provide a reasonable estimate of the maximum impact of MWS.

Prospects

Our analysis reveals that the previous characterisation of MWS at the population level was exaggerated. After more than 15 years of MWS research, this may be a major reason for our inability to convincingly explain what causes MWS (Broman et al. 2002). From the hunter's point of view, management decisions may have been unfortunate since lower harvest quotas motivated by the concern for local extinction may have created density-dependent effects such as smaller calves and less productive females (e.g. Wallin, Bergström & Vikberg 1995, Ferguson, Bisset & Messier 2000).

We speculate that incorrect assumptions about MWS have jeopardised the possibility of solving the scientific and the management problems associated with MWS. If our speculation is correct, it raises further questions. How common are such situations in wildlife management? Do similar problems affect all kinds of research? Perhaps McShea, Underwood & Rappole (1997) are right in saying on the first page of their book, The Science of Overabundance - Deer Ecology and Population Management: "There is, perhaps, no research endeavour that contains more common knowledge than the study of deer ..." and "Common knowledge too often serves as the bible of management decision".

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