DISEASE AND GENETICAL INVESTIGATIONS OF FENNOSCANDIAN CERVIDS - A REVIEW

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ABSTRACT: Fennoscandia (Norway, Sweden, Finland, and Denmark) is inhabited by many wild and/or semi-domesticated populations of cervids: moose (*Alces alces*), red deer (*Cervus elaphus*), roe deer (*Capreolus capreolus*), fallow deer (*Dama dama*), and reindeer (*Rangifer tarandus*). Cervids, especially moose, roe deer and reindeer, are an important natural resource of considerable economic value for tourism, hunting, and animal husbandry. Severe contagious livestock diseases have not been present in the cervids. Today however, increased trade of domestic and wild animals has lead to an obvious risk of foreign diseases entering Fennoscandia. Routine investigations of wildlife diseases date from the beginning of the present century in Fennoscandia. Besides these studies, research projects today are specifically concerned with investigating wildlife diseases. In this paper we: (1) give an overview of past and present investigations of diseases of cervids in Fennoscandia; (2) summarize the development of disease management from past to present; and (3) describe a direction for future research in Fennoscandia with respect to the dynamic problems associated with disease management of wild cervids beginning to arise in a rapidly changing Europe.

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Fennoscandia is basically Norway, Sweden, and Finland, but, in a wider sense, also Denmark. These countries have differing environment, flora, and fauna, and are inhabited by wild and/or semi-domesticated populations of cervids, including indigenous moose (*Alces alces*), red deer (*Cervus elaphus*), reindeer (*Rangifer tarandus*), roe deer (*Capreolus capreolus*), and imported fallow deer (*Dama dama*), sika deer (*Cervus nippon*), and white-tailed deer (*Odocoileus virginianus*). Fallow deer were imported to Sweden during the 16th century, and the sika deer during the 20th century (Bjärvall and Ullström 1985). These species are mainly kept as fenced game animals, but individuals that have escaped or been released now form small groups of feral animals. The white-tailed deer was donated by Finnish immigrants in the U.S.A. to their presumably hungry countrymen in 1934 (Bjärvall and Ullström 1985). The animals were first kept fenced, but individuals escaped and form a free-living population in southern Finland. Moose, red deer, and roe deer naturally migrated from the south into Fennoscandia after the last ice age some 10,000 years ago, whereas reindeer emigrated into the region about 12,000 years ago (Curry-Lindahl 1963).

Wild cervid populations, especially moose, roe deer, and reindeer, have been and are veryimportant as a natural resource with a substantial economic value to tourism and the outdoor experience, for hunting, and animal husbandry. For example, in Sweden meat production from harvested

moose comprises 4-5% of the total production in the country annually (R. Bergström, *pers. comm.*). The moose population in Fennoscandia (none are present in Denmark) is estimated to be approximately 450,000 animals (G. Cederlund and W. Faber, *pers. comm.*). The wild red deer population is approximately 79,500 (Brainerd *et al.* 1995, Vestergaard and Bavngaard 1996, K. Wahlström, *pers. comm.*). Roe deer have increased dramatically in recent years and number 1.5 million (G. Cederlund, *pers. comm.*). The white-tailed deer population in southern Finland consists now of over 20,000 animals (Piiroinen 1997).

Most reindeer are semi-domesticated, i.e., every individual is ear-marked and belongs to one owner; the total number fluctuates annually between 700,000 and 800,000, of which approximately 300,000 to 400,000 are slaughtered yearly (Nieminen 1993). There are approximately 30,000 wild mountain reindeer (*R. t. tarandus*) in southern Norway, and about 1,000 wild forest reindeer (*R. t. fennicus*) in Finland (Nieminen 1993). Norwegian wild reindeer are hunted yearly, whereas the hunt of forest reindeer has so far been limited to some individuals causing disturbance.

Fennoscandia has been isolated and protected from animal diseases by vast bleak areas in the North, by surrounding seas, and, in modern times, except for Denmark, by strict quarantine regulations. Severe contagious diseases that are a threat to livestock as well as to wild ungulates have not been present. Today as Denmark, Sweden and Finland have joined the European Union, import and export trade with agricultural products and animals, both domestic and wild, have increased. The Soviet Union has dissolved and trade with Russia will probably increase. All this has lead to an obvious increased risk of exotic animal diseases entering Fennoscandia.

Investigations of wildlife diseases have a long history in Fennoscandia. Studies on wildlife diseases date from the beginning of the present century, including Norway (Horn 1911), Sweden (Hülphers 1911; Hülphers *et al.* 1943, 1944; Borg 1956), Denmark (Christiansen 1935, 1949), and Finland (Andersson 1959). In Denmark, wildlife disease studies began in the 1930's, and by the 1950's problems were associated with biocides (Clausen 1985). In Sweden, routine investigations on wildlife carcasses begun by the Swedish Hunters' Association in 1941 continue today by the Swedish National Veterinary Institute (SVA). Besides these studies, research projects specifically concerned with investigating wildlife diseases are also being funded. Moreover, meat from all wild and fenced cervids to be sold to the public in Fennoscandia has to be inspected for diseases and meat hygiene by a meat control veterinarian (Official Journal of the European Communities 1991, 1992; Statens Livsmedelsverks Författningssamling 1994, 1996; Norges Offentlige Utredninger 1996).

MOOSE

The Past

As early as 1798, Svartz provided information for the The Swedish Academy of Science on a falling sickness involving hunted moose that fell without being wounded (Helander 1996).

Parasitic diseases. - Hülphers *et al.* (1943, 1944) reported on the occurrence of parasites including abomasal nematodes (*Trichostrongylus* spp.) and trematodes (e.g., *Fasciola hepatica*). In a post-mortem survey of moose conducted in the 1960's, 25 different parasites were identified (Nilsson 1971). *Onchocerca*, later shown to be species-specific for moose (*Onchocerca alcis*, Bain and Rehbinder 1986), was rare in the necropsied material but found in up to 98% of slaughtered moose (Roneus *et al.* 1984). The parasite is lo-

cated in the capsules of the joints and is spread by vectors, such as blackfly and mosquitos (Roneus *et al.* 1984). *Cysticercus tenuicollis*, the cyst of the tapeworm *Taenia hydatigena* in canids, occurs beneath the serosa of internal organs in moose. It is generally non-pathogenic (Borg 1975). Another trematode that has been reported is *Dicrocelium dendriticum*, the small liver fluke, which can cause proliferation and calcification of the bile ducts (Borg 1975).

Bacterial and fungal diseases. -- Bovine tuberculosis caused by *Mycobacterium bovis* is a zoonotic disease and was reported in Swedish moose during the 1940's (Kämpe and Horntwedt 1940, Hermansson 1943, Hülphers *et al.* 1943); it was believed that the moose were infected by contact with domestic cows. Sweden was declared free from tuberculosis by 1958 (Szewzyk *et al.* 1995). Avian tuberculosis (*Mycobacterium avium*) was recorded in moose in the 1980's (M. Stéen, *unpubl. data*).

Staphylococcus spp. infections causing hepatitis, chronical nephritis and arthritis (Hülphers *et al.* 1943, 1944) were reported. *Clostridium* spp. infections leading to septicaemia and enteritis, as well as serological evidence of tularemia caused by *Francisella tularensis* were found in Swedish moose during the 1950's to 1970's (Borg 1975). *Aspergillus* granuloma in the brain was infrequently diagnosed.

Viral diseases. -- Tickborn encephalitis was reported by Svedmyr *et al.* in 1965 in Swedish moose, a zoonosis that in humans can cause severe encephalitis. In the moose a nonpurulent meningo-encephalitis was diagnosed and the virus was isolated. A serological survey in the moose population showed antibodies to the virus (Borg 1975). Malignant catarrhal fever was noted in a couple of moose from the far north of Sweden in the early 1950's (Borg 1975). The disease is fatal, caused by a herpes virus, and sheep are believed to be the carriers. It causes inflammation in the mucous membranes, pus in the openings of the body, and diarrhea.

Neoplastic diseases. -- The occurrence of tumors was reported early in disease surveys, for example, benign fibroma and hygroma (Hülphers *et al.* 1943, 1944). In 1966 Hansen and Borg reported leukosis, a systemic disease in the hemopoietic system, with abnormal proliferation of the neutrophils causing general enlargement of lymphoid organs to occur in moose. Other neoplasms included ethmoid tumors, an expansive, infiltrative growing cancer of carcinomatous or sarcomatous nature derived from the mucous membrane of the ethmoid bone, and carcinomas and sarcomas in different organs (nostrils, tongue, salivary glands, sinuses, thorax, lungs, liver, kidneys, adrenal glands, testicles, and skeleton) (Magnusson 1915, Borg and Nilsson 1985)

Other diseases. -- Keratitis, cataracts, and/or glaucoma causing reduced vision and blindness have been recorded relatively commonly in moose (Kronevi *et al.* 1977, Borg 1987).

Non-infectious diseases. -- Hydrocephalus, with an abnormal amount of cerebrospinal fluid in the brain cavity causing pressure and diminishing of the tissue, resulting in neurological disorders and abnormal reactions, has been recorded. Trauma, as well as inherited malformations in the skeleton and extremities, mandibles and skull were reported early (e.g., *cyphos*, *lordos*, *scolios campylognathi brachygnathi*, *hernia ceribri*, *palatoschisis*, *adactyli*, *micromeli*, *perodactyli condrostrofi*, *phocomeli*, *syndactyli*, and *polydactyli*). Other congenital defects as hermaphrodites and pseudohermaphrodites were also reported (Hülphers *et al.* 1944, Borg 1975). Peruque antlers in which the velvet had not shed were observed; these were attributed to pituitary adenoma, and disturbed functions of the testicle. Indiges-

tion causing tympany, as well as dystocia, prolapse of female genitals and uterus were reported in moose (Borg 1975).

Environmental diseases. -- Few cases of intoxication have been detected, with the exception of ergotism causing gangrene by the fungus *Claviceps purpurea* (Borg 1975, 1987).

In Norway and Sweden, areas have been and are suffering from acid rain, due to air pollution from England and the European continent (Brodin 1993). Cadmium (Cd) levels were therefore measured in moose organs to obtain information about Cd levels in moose and in the environment (Frank and Petersson 1984). Cadmium is a non-essential trace element with toxic effects on the central nervous system (CNS), renal, and reproductive systems. Cadmium levels in moose tended to increase with age (Mattsson *et al.* 1981, Frøslie *et al.* 1984), and when levels found in moose were relatively high (≥20.5 µg/dry weight), humans were recommended not to consume moose liver and kidneys (Mattsson *et al.* 1981, Scanlon *et al.* 1986).

Genetical and physiological investigations. -- Genetic studies of alleles at polymorphic loci in moose in the 1970's and 1980's indicated that moose had a considerable genetic heterogeneity and a complex population structuring within Scandinavia (Ryman *et al.* 1980, Chesser *et al.* 1982)

The Present

Between 1985 and 1997 the disease pattern of moose radically changed in Fennoscandia, whereby diseases with impacts on a population level rather than individuals have arisen (M. Stéen, *pers. comm.*). Part of the explanation might be that the moose population increased dramatically from the 1970's to 1980's (Cederlund and Bergström 1996), especially in Sweden and Norway. In the 1980's more than 200 moose per year were necropsied in Sweden (M. Stéen, *unpubl. data*), compared to a total of 420 between 1947 and 1982 (Borg 1991).

Parasitic diseases. -- The increase in necropsies which occurred in the 1980's allowed for the detection of Elaphostrongylosis, a neurotopic disease caused by a previously unknown species of meningeal and tissue worm, *Elaphostrongylus alces* n. sp., Stéen, Chabaud, Rehbinder 1989 (Stéen *et al.* 1989, 1994, 1997; Stéen and Johansson 1990). The disease caused severe neurologic disorders and death in moose in Norway and Sweden during the mid-1980's and has been associated with slow growth and underdeveloped calves on an island in the Stockholm archipelago of Sweden (Stéen and Rehbinder 1986, Stuve 1986, Stéen and Roepstorff 1990, Stéen 1991, Olsson *et al.* 1995). Pathologically the parasite induces inflammatory reactions in the CNS (Stéen and Rehbinder 1986, Stéen 1991). In Norway the parasite was recorded in approximately 35% of the moose population (Stuve 1986). The susceptible portion of the population in both Norway and Sweden was calves and yearlings, in which neurological disturbances and death occurred (Stéen and Rehbinder 1986, Stuve 1986, Stéen and Roepstorff 1990, Stéen 1991, Olsson *et al.* 1995). The infection rate declined with age (M. Stéen, *unpubl. data*).

Elaphostrongylus alces first stage larvae are passed in feces and larval development takes place in an intermediate host, a gastropod, where it develops into an infectious larval stage (Olsson *et al.* 1993). The gastropods are ingested by moose while feeding, the animal becomes infected, and the nematodes migrate to the CNS. In contrast to other *Elaphostrongylus* spp. in deer, *E. alces* is located in the epidural and not the subdural space of the CNS.

Hundreds of moose in Sweden and Norway have died in association with infections

of the parasite (Stéen 1989, 1991); however in Finland *E. alces* seems to be of no importance to the moose population (S. Nikander, *unpubl. data*).

Other parasites observed in Sweden and Norway recently are the moose bot fly (*Cephenemyia ulrichii*) and ked (*Lipoptena cervi*)(Andersson 1985, Stéen *et al.* 1988, Mehr 1993, Nilssen and Haugerud 1994). There is reason to believe that these parasites have migrated from Finland where they are common (Frey 1914, Mikkola *et al.* 1982). The parasite fauna of Finnish moose differs somewhat from that of the Swedish and Norwegian moose populations. The lungworm *Bicaulus alcis* is common in Finland, but is not known from Norway (S. Nikander, *unpubl. data*) and is rare in Sweden (M. Stéen, *unpubl. data*).

Trypanosoma cervi, a blood parasite, has recently been recorded in Swedish moose (Dirie *et al.* 1990). Further, the ticks *Ixodes ricinus* and *Haemaphysalis punctata* (Jaenson *et al.* 1994) have been found on moose. Ticks from Swedish moose were negative for the spirochete *Borrelia burgdorferi* which causes the zoonosis, Lyme disease, in humans (Mejlon and Jaenson 1993).

Viral diseases. -- Malignant catarrhal fever has again been recorded in moose in recent times (Warsame and Stéen 1989). A well known disease in moose is fibropapilloma; the fibropapilloma virus (EEPV) viral transcription pattern in EEPV (C 127 cells) was described in moose by Eriksson (1993).

Other diseases. -- In Sweden during the mid-1980's fatal wasting syndrome was recorded in moose, particularly in the southwest (Rehbinder *et al.* 1991, Stéen *et al.* 1993). Initially the syndrome seemed particularly prevalent in adult animals, however later investigation found that calves have also died from this condition. Mortality in association with the syndrome seems to increase with age, and females over 4 years of age are most prone to the syndrome (Cederlund *et al.* 1994). The symptoms are locomotive aberrations, weakness, listlessness, blindness or impaired vision, and fearlessness (Rehbinder *et al.* 1991, Stéen *et al.* 1993). The post-mortem findings include erosive, ulcerative, or necrotic lesions in the mucous membranes, atrophied lymphoid organs, and emaciation (Rehbinder *et al.* 1991, Stéen *et al.* 1993).

During the last decade more than 1400 moose have been found sick or dead in association with this syndrome. Attempts have been made to identify the etiology agents, involving bacteria, parasites, viruses, dietary deficiencies, trace element deficiencies, and/or the effects of pollution as causes of this syndrome. Thus far however, the etiology of the syndrome remains unknown. A retrovirus has been isolated and antibodies against a pestivirus have been detected in sick animals in connection with the disease, but these have not been proven to be the causative agent (Merza *et al.* 1994, Stéen *et al.*, *unpubl. data*).

Osteoporosis has been seen in association with the wasting syndrome in Sweden (M. Lind, Uppsala Univ., *unpubl. data*), and is also recorded in both Norway and Finland (Stuve 1994, K. Nygrén, Finnish Game and Fisheries Res. Inst., *pers. comm.*). This condition has been reported most commonly in young animals in Norway, while in Sweden older age groups are affected. In Norway as well as in Sweden, mineral deficiencies, acid precipitation, and overbrowsing have been investigated and discussed as potential causes (Pehrson and Faber 1993, Punsvik and Jerstad 1994, Arnemo 1995a, Arnemo and Soveri 1995, Storaas and Punsvik 1996, Brainerd 1997). The hematology and blood chemistry have also been studied in relation to the wasting syndrome and the parameters diverge from healthy animals (Kockum-Adolfsson 1995).

Environmental diseases. -- In connection with the investigation of the wasting syndrome in moose, trace elements have been investigated. Compared to a decade earlier, when moose throughout Sweden and Norway were investigated for heavy metals and trace elements, low levels of copper (Cu), iron (Fe), zinc (Zn), and chromium (Cr), and increased levels of molybdenum (Mb) were detected in organs from sick and apparently normal animals in southwest Sweden (Scanlon *et al.* 1986; Frank 1989, 1995; Frank *et al.* 1994; Petersson and Galgan 1994).

The Chernobyl accident in the Ukraine of the former Soviet Union in 1986, which caused deposition of radioactive fallout over Fennoscandia, was devastating for the moose hunt in many areas. Levels of radioactive cesium (^{137}Cs) were high in harvested moose and these animals were considered unfit for human consumption. The authorities recommended them to be destroyed (Nelin 1994). In 1995, 9 years after the accident, there were still areas with ^{137}Cs levels in moose that exceeded the limit for human consumption (Palo and Wallin 1996).

Genetical and physiological investigations. -- Physiological studies have been done on apparently healthy moose to obtain basic physiological values. This has produced knowledge of blood morphology, hematological, and blood chemical values (Kockum-Adolfsson *et al.* 1997, Kockum-Adolfsson, *unpubl. data*), and how immobilization influences blood parameters (Arnemo 1995b, Kockum-Adolfsson *et al.*, *unpubl. data*).

In connection with these diseases an interest arose to determine whether or not moose are paricularly susceptible to infectious diseases. The genetic diversity of the major histocompatability complex (MHC), which encodes cell surface proteins that bind and present intracellular peptides to lymphocytes, has been studied (Mikko and Andersson 1995). Both European and North American populations of moose exhibit very low levels of genetic diversity at the expressed MHC class DRB locus.

Thus, it is believed that a lower variety of peptides can bind efficiently to the MHC molecule. This could suggest that moose inherited a low capacity to resist infectious diseases, but no evidence to support this theory exists. Recently a follow-up study of the MHC class II DRB in moose was conducted (Mikko 1997). The investigation includes moose from Sweden, Norway, Canada, and Alaska. Limited levels of polymorphism were found in all populations, although somewhat higher in the Scandinavian populations than in the North American. A total of 7 alleles were detected, which is about the same number as found in reindeer. The striking feature of the moose DRB alleles, as compared with reindeer, is that they are very similar to each other, i.e., the genetic distance between them is minute (Mikko 1997).

Tests used for analysis of cell-mediated immunity in domestic animals have been tested in moose and been found to work (Kockum-Adolfsson 1997). Other examination of healthy animals has revealed that moose can have atherosclerosis (Poungshompoo 1985).

The Future

Moose are distributed throughout Fennoscandia except Denmark and are hunted annually. Because of this moose are a valuable source of biological material collected for environmental investigations over a long span of years. Organs will be studied in Sweden, Norway, and Finland concerning environmental pollutants, heavy metals, and trace elements. Also demographic data will be collected and serological as well as enzymatic studies concerning infectious diseases and general health con-

ducted (Jaren *et al.* 1995, Statens Veterinärmedicinska Anstalt 1996, Swedish Environmental Protection Agency 1996a). In Norway wildlife management and a moose-forest-community project investigating the health of the moose population relative to demographic and genetic parameters is prioritized (Sæther *et al.* 1992, Storaas and Punsvik 1996, Brainerd 1997).

REINDEER

The Past

Parasitic diseases. -- Linnaeus (1707-1778, in Skjenneberg and Slagsvold 1968) was familiar with warbles (*Hypoderma tarandi*) and throat bot fly (*Cephenemyia trompe*) in reindeer. Later these parasites were studied by Bergman (1916, 1917) and are still investigated by parasitologists and ecologists today. Sucking and biting lice (*Solenopotes tarandi*, *Trichodectes tarandi*) were described in Swedish reindeer by Mjöberg (1915). Other parasites that still inflict reindeer are *Dictyocaulus* spp., the lungworm, the tapeworms *D. dendriticum krabbei*, *T. hydatigena*, and *Linguatula arctica*, a parasite in the sinuses (Haugerud *et al.* 1993). *Echinococcus granulosus* is prevalent in Scandinavian reindeer, and in Norway 23% were infected (Skjenneberg and Slagsvold 1968). An eradication program was conducted in northern Norway (Kummeneje *et al.* 1981).

Elaphostrongylosis, caused by *E. rangiferi*, is a well known disease in reindeer in Fennoscandia. In the 1960's and 1970's it caused numerous deaths in Swedish and Norwegian reindeer herds. Loss of calves in the populations were infrequently high (Roneus and Nordkvist 1962, Bakken and Sparboe 1973). Swedish reindeer were also found to have a high frequency of visceral parasitic granulomas of *O. tarsicola* origin (Rehbinder *et al.* 1979). Nordkvist (1971, 1980) studied parasite control, and control has become routine in many areas; e.g., in Finland over 80 % of the overwintering animals are treated yearly with ivermectin (Ivomec®). It is supposed to help the animals survive the winter and produce healthy offspring. Meat inspection has allowed for a more systematic disease control, and has been extensively utilized. For example, Kummeneje (1976, 1977) studied verminous pneumonia in connection with pasteurellosis (*Pasteurella multocida*), as well as a follow-up on an *E. granulosus* eradication program in northern Norway (Kummeneje *et al.* 1981).

Bacterial and fungal diseases. -- Linné reported in 1732 and 1759 on gangrene in the clefts and under the skin ending in septicemi and pyemi (in Qvigstad 1941; in Skjenneberg and Slagsvold 1968). Necrobacillosis (*Fusobacterium necrophorum*) has been a scourge for reindeer herding and was reported by Horn in 1897 and Bergman in 1919 (in Qvigstad 1941; in Skjenneberg and Slagsvold 1968). Since then the disease has been reported infrequently (Skjenneberg and Slagsvold 1968). Anthrax (*Bacillus antracis*) was reported from reindeer in Sweden in 1749 by Gissler (1759) (in Qvigstad 1941) and in Norway in 1893 (Skjenneberg and Slagsvold 1968). Reindeer pest, caused by *Clostridium septicum*, had severe impacts on reindeer populations in the 18th and 19th centuries in Sweden and Finland (Qvigstad 1941, Skjenneberg and Slagsvold 1968), and also infected wild reindeer and thousands of animals died (Skjenneberg and Slagsvold 1968). In the early part of this century, pasteurellosis was encountered in hundreds of Swedish as well as Norwegian reindeer and in the 1980's it was seen in Finnish reindeer (Westerling 1993).

Other diseases. -- Infectious keratitis and conjunctivitis have been reported as early as1909 by Bergman (in Skjenneberg and Slagsvold 1968). Rehbinder (1977)

found that in reindeer herds, especially reindeer calves suffered from the disease.

Genetical and physiological investigations. -- Røed and Whitten (1986) investigated genetic variation in different subspecies of tundra reindeer and caribou. Their results indicated that Alaskan caribou (*R. t. granti*) and Eurasian reindeer evolved from a common ancestral population different from the ancestral population of Peary caribou (*R. t. pearyi*) and Svalbard reindeer (*R. t. platyrhynchus*).

The Present

Most reindeer in Fennoscandia are semi-wild, and thousands of human families depend on reindeer husbandry. Husbandry legislation and disease management are therefore important.

Parasitic diseases. -- *Elaphostrongylus rangiferi* has caused neurological disorders in goats, sheep, and muskox (*Ovibos moschatus*) sharing pasture with reindeer as well as in experimentally infected sheep, goats, and moose (Holt *et al.* 1990, Handeland 1991, Handeland and Sparboe 1991, Stéen 1991, Handeland and Skorping 1993, Handeland *et al.* 1993, Stéen *et al.* 1997). Recently, *E. granulosus* has been discovered in reindeer in northern Finland and Sweden (Oksanen and Laaksonen 1995, D. Christensson, *pers. comm*). Dirie *et al.* (1990) and Rehbinder (1990) reported on some vector borne parasites in reindeer including *Megatrypanum trypanosomes*, *Setaria tundrae*, *O. tarsicola*, and *Lappnema auris*.

Bacterial and fungal diseases. -- Pinkeye has recently been detected in corralled Finnish reindeer. *Moraxella ovis* was recorded from affected eyes (Oksanen *et al.* 1996).

Viral diseases. -- In Swedish reindeer a disease characterized by ulcerative and necrotic lesions of the upper alimentary tract and nose, and caused by a herpes virus related to bovine herpes virus type 1, has been detected (Rockborn *et al.* 1990). Antibodies against herpes virus are also common in Finnish reindeer (Ek-Kommonen *et al.* 1982). Norwegian reindeer commonly have antibodies against both reindeer herpes virus and bovine virus diarrhea virus (BVDV) (Stuen *et al.* 1993). Parapoxvirus infection causing severe suffering has recently been found in modern semi-domesticated reindeer (Büttner *et al.* 1995).

Other diseases. -- As reindeer husbandry has developed to emphasize productivity, pastures have been overgrazed. This has forced Finnish and Swedish reindeer owners to feed the flock during winters, and in many cases to corral them. While starvation can be prevented, it is obvious that corralling increases physical contact between animals, which may facilitate the spread of infectious diseases. The emerging infectious diseases have affected the feeding politics in Finland; although the need for supplementary feeding is recognized, it is now often preferred to feed the animals in the forest and fells instead of in corrals.

Environmental diseases. -- Fallout of radiocaesium ^{134}Cs and ^{137}Cs from the Chernobyl accident contaminated large areas of reindeer pasture. High Cs levels severely affected reindeer meat production, and activity concentrations up to 96 kBq/kg fresh weight have been recorded. In Norway contaminated areas seemed to have reached the same levels as in Sweden, while Finland was scarcely affected. Wild reindeer in Norway had levels up to 70 kBq/kg. A significant decline in concentration of Cs has occurred from 1986 to 1992 in both Sweden and Norway (Åhman 1994).

Genetical and physiological investigations. -- The handling procedures before slaughter were investigated by Wiklund (1996) to check the effects on blood metabolites, muscular, and abomasal lesions, all used as stress markers. Tradi-

tional handling using a lasso to catch reindeer was found to be more stressful compared to reindeer shot undisturbed, helicopter herding, selection by hand, or road transport by truck. The physical condition and nature also influenced the stress tolerance. Reindeer meat was found to be extremely tender regardless of stress.

Genetic investigation of MHC class II *DRB1* in reindeer in Norway including Svalbard has been conducted. Polymorphism and heterozygosity were found in Norwegian reindeer while the Svalbard reindeer were extremely homozygous (Mikko 1997). These MHC data are consistent with the extensive studies of protein polymorphism conducted by Røed (1985).

The Future

The importance of managing wild reindeer populations and their reintroduction to habitats from where they were eradicated are stressed in Norway (Sæther *et al.* 1992, Jaren *et al.* 1995, Storaas and Punsvik 1996). In Finland, the wild forest reindeer population is expected to grow and become an important game species (Kojola 1996). In general, reindeer husbandry will continue to be important to the native Saami people. Important issues at present include the development of management tools for pasture resource management and regulation of reindeer populations to actual pasture resources. Other important issues will be to improve conditions for coexistence with other natural resource uses including nature conservation in areas of reindeer husbandry, especially regarding populations of the major predators brown bear (*Ursus arctos*), lynx (*Lynx lynx*), wolverine (*Gulo gulo*), and wolves (*Canis lupus*)(Ö. Danell, *pers. comm.*).

ROE DEER

The Past

During the years 1941 to 1944, 24 roe deer were necropsied in Sweden and reported by Hülphers *et al.* (1943, 1944). They died from parasites, starvation, hemorrhagic enteritis, hematoma, and trauma.

A compilation of necropsy information from 1947 to 1982 on approximately 4,000 roe deer showed a wide range of diseases and other mortality causes; 50.5% died from starvation, 25% from trauma including predation, and 24.5 % from diseases (infections, parasites, organic diseases, intoxications, tumors, peruque antlers, malformations, etc.). Most of the animals died during the first 4 months of the year (Borg 1970, 1991).

Parasitic diseases. -- In the 24 roe deer necropsied by Hülphers *et al.* (1943, 1944), *Trichostrongylus* spp. infection seemed to be the cause of death in 18 cases, some in connection with the lungworm *D. viviparus*. Deer bot fly (*Cephenemyia stimulator*) was reported by Christiansen (1935) in Danish roe deer.

Common parasites in animals found dead between 1947 and 1982 were lice (*Damalinia* spp.), and ked (*Lipoptena cervi*)(Borg 1991). Nilsson (1971) found 39 parasite species on 462 roe deer carcasses during 1966 to 1968. The most common were *Ostertagia* spp., *Spiculopteragia* spp., *Nematodirus filicollis*, *Nematodirella alcidis*, and *Dicrocoelium dendriticum*, and all in the digestive system.

In 1988 Korsholm reported on nematode infection in roe deer and the roe deer as a reservoir for bovine nematodes. He concluded that roe deer can be important for transmitting gastro-intestinal nematodes (e.g., *Ostertagia* spp.) to livestock. Other organisms found in roe deer are *Pneumocystis carinii* (Protista, Sarcodina, Rhizopodasica), and the blood parasite *Babesia capreoli* (Christensson and Järplid 1979, Settnes *et al.* 1986).

Bacterial and fungal diseases. -- In-

fectious disease in roe deer was reported as early as 1919 with a suspected case of avian tuberculosis (Borg 1991), which is not an uncommon disease in roe deer. The carriers are probably pigeons (*Columba* spp.) and pheasants (*Phasianus colchicus*), and the disease is characterized by tubercles in the lymphoid system and internal organs. Listeriosis has frequently been seen in roe deer (Borg 1970), and the bacterium (*Listeria monocytogenes*) is widespread in nature. The disease is a zoonosis, and human fetuses are especially sensitive since *L. monocytogenes* can cause abortion or result in disabled children. The disease in deer is a systemic disease and can create inflammation in the CNS, signified by circling movements. Roe deer can also be a silent carrier of *L. monocytogenes*.

Another disease, pink eye, affects the eyes and sometimes results in blindness. The etiology was not determined, but the bacterium *Moraxella bovi* was suspected to be involved. Other pathogenic species of bacteria and fungi found were *Clostridium, Corynebacterium, Erysipelothrix, Pasteurella, Salmonella, Streptococcus, Fusobacterium,* and *Actinomyces*. Hillermarks disease, with similarities to paratuberculosis, is caused by a *Mycobacterium* sp. and was reported by Borg (1975) to be a rather uncommon disease among roe deer. The disease resulted in enlargement and necrosis of the body lymph nodes. In cells of epitheloid nature and in the villi of the small intestine accumulation of tuberculine bacterias could be seen (Borg 1975). Metritis probably in connection with dystocia was also reported.

Viral diseases. -- Malignant catarrhal fever, reported in northern Sweden in moose in the early 1950's, was later recorded in roe deer in these same areas (Borg 1975).

Neoplastic diseases. -- Cancer and tumors were common findings, with both benign and malign origin (e.g., carcinoms, sarcoms, melanoms, dysgerminoms, semioms, synovioms, liomyoms, glioms, osteoms, adenoms, granulosa cell tumors, and ethmoidal tumors)(Borg 1991).

Non-infectious diseases. -- Among roe deer, trauma was a common cause of death, and approximately 13% had been in an accident. They had traces of fights with other males or jabbing injuries where branches were stuck into the body cavity. Foreign bodies such as sharp material, wires, and plastic sometimes had caused ruptures in the body cavity and other disorders. Since roe deer are often hunted with shotguns and the bullets at longer distances only penetrate the skin, healed or fresh gun-shot wounds were recorded in 3% of the necropsied animals (Borg 1991). Of the roe deer killed by predation, over half (56.6%) suffered from disease and the rest were healthy animals. Carnivores preying on roe deer were identified as dogs, red fox (*Vulpes vulpes*), lynx, wolverines, pine marten (*Martes martes*), and Golden eagle (*Aquila chrysaetos*)(Borg 1991).

Malformation and congenital defects were seen (e.g., subaortic septal defect, *diplocardi*, cryptorcism, overgrowths of the clefts, and dermoid cysts). Other finds in the genitals were *prolapsus uteri* and *vovolus uteri* (Borg 1975, 1991).

Peruque antlers were a rather common feature in the material, and 39 animals had that malformation of 4,168 necropsied (Borg 1991). As in moose these animals had diseases as well as cancer in the testicles, the thyroid, and the pituitary gland. Roe deer males, as in cervids in general with the exception of reindeer, bear antlers, but occasionally female roe deer can also produce antlers. A disturbance in the hormone balance can be the cause, but is not proven. These females were also often pregnant.

Environmental diseases. -- Roe deer have been prone to ^{137}Cs, similar to moose,

because they select food that is highly contaminated by radioactive fallout, for example mushrooms (Karlén *et al.* 1991, Cederlund and Liberg 1995).

Genetical and physiological investigations. -- Female roe deer are reproductive from the age of 15 months, but there are exceptions with younger animals coming into estrus. The rut occurs at the end of July and beginning of August when females mate, and fawns are born at the end of May or beginning of June the year after (Borg 1991). Investigations of fetus size were also performed, and in January the length was on average 20 mm, in February 50 mm, in March 100 mm, in April 170 mm, and in May 250 mm (full size). Mummified fetuses were sometimes found in reproductive organs (Borg 1975, 1991). Poungshompoo (1985) reported, as in moose, that healthy roe deer could have atherosclerosis.

The Present

Over the last decade there has been a rapid increase in the population of roe deer in Fennoscandia. The present population, especially in Sweden, is the densest in modern time (Cederlund and Liberg 1995). As seen in moose, diseases are affecting the population rather than individuals (M. Stéen, *pers. comm.*).

Parasitic diseases. -- In an investigation of the population demography of a previously non-hunted population of roe deer with a density of 500 animals/1,000 ha, the infection of parasites and other diseases of 200 harvested animals were also studied (Stéen 1992). Parasites found with an impact on the animals were parasites in the abomasum (*Spicutoptergia* sp., *Ostertagia* spp.), small intestine (*Trichostrongylus* spp., *Nematodirus* sp.), cecum (*Trichuris* sp.), and large intestine (*Chabertia* sp., *Oesophagostomum* sp.)(M. Stéen, *unpubl. data*).

A significant relationship between age and parasite infection could be seen with the highest infection in juveniles. The parasite rate decreased with age (> 2 years) and increased again in roe deer over 6 years of age. Body condition in adult roe deer could also significantly be related to the amount of parasites (Tillbom 1995). The roe deer have been investigated for *Eimeria* spp., coccidians, and *E. rotunda*, *E. capreoli*, *E. ponderosa*, and *E. superba* were found. Additionally 3 unknown species were detected. The relevance for disease in roe deer of this parasite is not known (Törnlöv *et al.* 1994).

The tick fauna has recently been investigated, and *I. ricinus* and *H. punctata* were found to infest roe deer (Jaenson *et al.* 1994). The prevalence antibodies to and the spirochete *B. burgdorferi* have been investigated in both Denmark and Sweden. In Denmark antibodies were detected in 52 % of the roe deer sera. The spirochete *B. burgdorferi* was not found in ticks infesting roe deer in Sweden (Mejlon and Jaenson 1993, Webster and Frandsen 1994).

Other diseases. -- Currently, diarrhea is the most common disturbance in the roe deer population, of which the etiology is still unknown, but comparisons have been made with the wasting syndrome in moose (Mörner 1995). Diaz *et al.* (1996) reported on ulcerative, necrotizing lesions, and diarrhea in roe deer similar to those seen in moose. The diarrhea seems to be moderate, but affected animals have extreme emaciation with atrophic muscles. Mörner (1995) has also made an epidemiological study. Organs will also be collected to investigate virus antibodies and be included in a monitoring program concerning environmental pollutants and heavy metals (Mörner 1995).

Environmental diseases. -- Henriksen (1994) reported that roe deer became sick and died when they fed on rape seed oil. The clinical signs were of CNS origin, with apathy, circling, blindness, and loss of natu-

ral fear. The time of incubation is usually more than a week. Histologically, lesions in the brain parenchyma are seen, with necroses in *cortex cerebri*. The pathogeneses are thought to be induced by thiamin deficiency in the rumen.

Genetical and physiological investigations. -- Roe deer have also been investigated concerning the genetic polymorphism at MHC class II DRB loci. Limited levels of polymorphism were found in roe deer. The number of alleles is also lower than in the moose but the genetic distance between the alleles is greater. Therefore the total amount of variation is not as low as for moose. Results indicate that the roe deer in Norway and Sweden have experienced a population bottleneck (Mikko 1997).

The Future

Authorities throughout Fennoscandia wish to lower roe deer numbers below present levels, in the hopes of reducing browsing damage, to forest plantations, and car accidents. According to Cederlund and Liberg (1995), roe deer have an inherently high reproductive rate, and thus hunting as a regulating factor has not likely influenced the roe deer population increase which has occurred in Sweden in the last decade.

Moreover, hunting as a regulating factor is difficult to measure in a population with high reproductive rate, but under certain circumstances, it can be used to help control roe deer numbers. In particular, climate and predation cause high stochasticity that reduces the effectiveness of the hunt. If environmental factors are optimal for population growth (potential rate of increase of >50%), hunting may have only a marginal effect (Cederlund and Liberg 1995). Predators that can have an impact on roe deer numbers are red fox, lynx, and wolves, where red fox are most important (Cederlund and Liberg 1995).

DEER

The Past

Parasitic diseases. -- Parasites are common and belong to the same species as reported in moose and roe deer. The signs are retardation in growth and increased susceptibility to other diseases. Fallow deer seem to be more refractory to parasites than the other deer (Clausen 1986, Stéen 1988, Englund 1992). *Elaphostrongylus cervi* was found in red deer in Sweden and Denmark, as well as in Norway (Eriksen *et al.* 1989, Stéen and Johansson 1990, Gibbons *et al.* 1991). The parasite in red deer is not considered to be pathogenic, although Borg (1979) reported Elaphostrongylosis in a red deer. White-tailed deer were exported to southern Finland from the USA prior to World War II. Some of those animals escaped into areas where large moose populations are free-ranging. A study was conducted between 1966 and 1967 to determine if *Parelaphostrongylus tenuis* occurred in Finnish white-tailed deer, however no parasites were found (Andersson *et al.* 1970). Fortunately, *P. tenuis* was not introduced into Fennoscandia.

Bacterial and fungal diseases. -- Pseudotuberculosis, listeriosis, and abscesses with *Corynebacterium* sp. were reported in Danish farmed deer as well as paratuberculosis (Clausen 1986, Stéen 1988).

Viral diseases. -- Malignant catarrhal fever has been detected in Danish farmed red deer. Sera from farmed Danish deer, both fallow and red deer, have been investigated for virus, but found negative (Stéen 1988).

Fallow deer in Sweden with clinical signs of BVDV were investigated by Diaz *et al.* (1988); the pathological lesions were typical for the disease and the virus was detected in 1 animal.

Neoplastic diseases. -- A nasal tumor

has been reported by Stéen *et al.* (1985) in a free-living fallow deer. The tumor arose from the olfactory mucosa and was composed of two different cancers, malignant schwannoma and a carcinoma. *Fusobacterium necrophorum* was involved.

Environmental diseases. -- Heavy metals and trace element levels have been investigated in Norwegian red deer and the trace elements were not found to differ from domestic animal quantities (Frøslie *et al.* 1984, 1986).

Genetical and physiological investigations. -- A study of the taxonomy and osteology of prehistoric and recent populations of red deer (*Cervus elaphus atlanticus*, *C. e. elaphus*, *C. e. hippolaphus*) in Norway, Sweden, and Denmark was done by Ahlén (1965). He concluded that there were little differences between the Danish and Swedish deer whereas differences between the Danish and Norwegian and between the Swedish and Norwegian populations were quite clear. He considered the Norwegian stock had been separated from the Swedish for a considerable time (4,000-8,000 years ago) and that the Norwegian deer presented more primitive characters than Swedish and Danish deer, with the Norwegian stock representing the earliest. The Norweigan deer have been dated to appear in Norway about 5,000 years ago (Markgren 1990). The Swedish and Danish populations should be considered as 1 instead of 2 subspecies, and the Danish *C. e. hippelaphus* should be transferred to *C. e. elaphus*.

The variation in body weight along 2 climatic gradients in red deer was investigated by Langvatn and Albon (1986) in Norway. They discovered that deer of both sexes and all age classes were significantly heavier in the northern parts compared with the south and in the inland compared with the coast. In another study on fallow deer, atherosclerosis was found, as had been in moose and roe deer (Poungshompoo 1985).

The Present

Parasitic diseases. -- In red deer as well as in fallow deer infestations with ticks, *I. ricinus* and *H. punctata*, have been found (Jaenson *et al.* 1994). The prevalence of antibodies to the tickborn spirochete *B. burgdorferi*, were high in Danish deer, 38% in fallow deer and 27% in red deer. Recently in 1996, *Onchocerca flexuosa* have been found to infest subcutis in farmed red deer. Fallow deer in the same enclosure were not infected (A. Stark and S. Bornstein, National Vet. Inst., *pers. comm.*). The infection has an impact on meat hygiene control.

Bacterial and fungal diseases. -- Bovine tuberculosis (*Mycobacterium bovis*) was first diagnosed in fallow deer in Denmark in 1988 and in Sweden in 1991. The disease was imported with farmed fallow deer from England in 1987 and 1988, respectively (Stéen 1988, Clausen and Korsholm 1991, Bölske *et al.* 1995). The deer farms in contact with the infected consignment of deer were placed under restrictions. These movement restrictions of animals are combined with tuberculine skin tests, slaughter, and meat inspections (Stéen 1988, Bölske *et al.* 1995). Thirteen herds including red and fallow deer in Sweden have been positive for bovine tuberculosis, in fallow deer only, and subsequently destroyed up to 1997 (L. Englund, National Vet. Inst., *pers. comm.*). A voluntary testing program based on 3 tuberculine tests of a whole herd, combined with compulsory movement restrictions on all farmed deer, are implemented in Sweden, and as far as we know there has not been any spread of the disease to wild ungulates in Sweden. Tuberculosis in Denmark was eradicated through a control and eradication program concluded in 1994 (L. Englund, *pers. comm.*). In 1988 however, a free-living red

deer shot and necropsied was positive for the disease (Clausen and Korsholm 1991). In Denmark, as well as in Sweden, tuberculosis in the bovine population was eradicated by 1959 and 1958, respectively (Stéen 1988, Szewzyk *et al.* 1995). Randomly, red deer as well as fallow deer have been tested for tuberculosis in Finland with negative results (Westerling 1994). In Norway, the disease is not known to be found in wild or farmed red deer (Jaren *et al.* 1995).

Mycobacteriosis, i.e., infection with *M. avium* in the body lymph nodes (e.g., mesenteric lymph nodes), spread by bird droppings on dirty feeding grounds are prevalent in farmed deer in Sweden (L. Englund, National Vet. Inst., *pers. comm.*). Necrobacillosis caused by *S. necrophorus* is not unusual in farmed deer. The disease is spread on permanent, dirty feeding places. The bacteria infect through lesions in the mucous membranes in the mouth and cause necroses in the mouth and tongue. The infection can spread through the digestion canal and give necroses and abscesses in the rumen and internal organs (Englund 1992).

In Denmark red deer have died from clostridios-enterotoxaemi caused by the bacteria *Clostridium perfringens* type A. Focal necroses in the mesenterial lymph nodes and spleen and granulomes in the liver occurred (Dietz 1994). In Sweden 200 farmed deer have been investigated for Leptospirosis (*Leptospira* spp.), but found negative (Englund 1992).

Viral diseases. -- Swedish farmed deer, including fallow and red deer, have been investigated for antibodies against certain virus diseases (e.g., infectious bovine rhinotracheitis (IBR) and BVDV). None were positive for IBR, although quite a few deer were positive for antibodies cross-reacting with BVDV. Clinical disease was not seen in those animals (Englund 1992).

Other diseases. -- White-tailed deer in Finland have been investigated for diseases, although no serious infectious diseases have been found in the population. The main cause of death is starvation (Nikander and Soveri 1988).

Genetical and physiological investigations. -- Chemical capture of free-living red deer with medetomidine-ketamine has been tested regarding stress. Hematological and serum biochemical comparison between immobilized free-ranging and unmedicated captive deer showed that chemical capture induced very little stress in red deer (Arnemo 1994). Chemicals for immobilizing, both neuroleptic and anaesthetic for field use have been tested in red deer (Arnemo *et al.* 1993).

Genetic polymorphism at MHC class II DRB exon 2 has recently been investigated in red and fallow deer (Swarbrick *et al.* 1995). In both species 2 loci were found contrary to other cervids investigated, where only 1 locus was found. Complete monomorphism was found in each of the fallow deer loci whereas extensive *DRB* diversity was found in red deer (Mikko 1997).

The Future

Knowledge in Sweden about red deer and their disease management is limited. Therefore, at present it is hard to predict a management program for the red deer in Sweden.

In Norway, farmed deer have been considered a threat to the wild fauna, by spreading diseases and genetic interference with wild deer. On exception permission is given to establish and farm deer, but only using free-ranging deer captured within Norway, and import of farmed deer is prohibited. Included in the regulations for farming deer is that hunting enclosed deer is prohibited (Jaren *et al.* 1995).

In Norway and Denmark there are good numbers of free-ranging red deer that are hunted annually (Brainerd *et al.* 1995,

Vestergaard and Bavngaard 1996). In Norway, up to 20,000 red deer are shot per year, and no hunting restrictions are planned.

CHALLENGES FOR THE 21ST CENTURY

In documents concerning wildlife of Fennoscandia, it is discussed that research heading into the 21st century should highlight strategy programs; e.g., the effect of hunting and management of cervid populations (Swedish Environmental Protection Agency 1996b). Great effort will be focused on how cervid density impacts dynamics and natural losses in the populations. Moose and roe deer in Sweden as well as moose and red deer in Norway have increased dramatically in recent decades, and knowledge is lacking how these large populations change the ecosystem. Thus the impact these cervid populations have on biological diversity is to receive considerably more attention in the future (Swedish Environmental Protection Agency 1996b).

In Norway they also stress the importance of maintaining populations of healthy native cervids. They conclude that it is not important to reach population carrying capacity, but rather to keep the populations at such a number that their condition and health is guaranteed; therefore they are introducing a generalized monitoring of wild animal health (Sæther *et al.* 1992, Jaren *et al.* 1995, Storaas and Punsvik 1996).

Generally speaking, animal health in Fennoscandia concerning both domestic and free-ranging wild animals has been good. However, the risk for introducing "new diseases" into this region through trade is high, owing to regulations the common market of today has. In Europe and in Russia, diseases are present that in Fennoscandia have never occurred or have been eradicated. Diseases can easily be imported and impact the vast cervid populations. With the large herds of wild ungulates, especially cervid, present in Fennoscandia today, it would seem almost impossible to eradicate any introduced disease that became established. Further, such diseases once present in wild populations and in animals that can move long distances (e.g., moose) will become a source for diseases in humans (zoonoses) and domestic animals. Diseases that are important zoonoses and can be spread via cervids are for example echinoccosis (*E. granulosus*) and bovine tuberculosis. Diseases that could be of importance for wild and domestic ungulates in Fennoscandia and are reported to exist in Europe and Russia are for example brucellosis (*Brucella* spp.), leptospirosis, paratuberculosis (*Mycobacterium paratuberculosis*), IBR/infectious pustulous vulvovaginitis (IBR/IPV) (Bovine herpes virus 1), foot and mouth disease (*Aphtovirus*), and bluetongue (*Orbivirus*)(Swedish Board of Agriculture 1994, Edquist *et al.* 1995).

In Fennoscandia today, there is a tradition of widespread, generic, epizootical observation in addition to hunters who willingly collect wildlife carcasses. This allows veterinarians, wildlife researchers, and managers to maintain relatively good control over wildlife diseases, both endemic and introduced, in this region.

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