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Author: Borg, Karl

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A REVIEW OF WILDLIFE DISEASES FROM SCANDINAVIA[1]

Karl Borg
Morbydalen 1, 182 32 Danderyd, Sweden

ABSTRACT: The epidemiological and historical aspects of some important and representative wildlife diseases from Scandinavia are discussed. In noninfectious diseases, examples include cataract in moose (*Alces alces*), atherosclerosis in hybrid hares (*Lepus timidus* × *L. europaeus*), and ethmoid tumors in moose. The epizootiological and historical aspects of the recent epizootics of myxomatosis in European rabbits (*Oryctolagus cuniculus*) and rabies and sarcoptic mange in red foxes (*Vulpes vulpes*) are reviewed. The decline and subsequent increase in population abundances of tetraonids including the capercaillie (*Tetrao urogallus*), black grouse (*Lyrurus tetrix*), and hazel hen (*Tetrastes bonasia*) are discussed, and an hypothesis on predation by foxes is presented as a possible explanation for these population fluctuations. The potential impact of environmental pollution on wildlife populations is emphasized with reference to mercury in wildlife from Sweden and the possible effects of cadmium and selenium resulting from acidification. A bibliography of important references is presented pertaining to these and other diseases of wildlife from Scandinavia.

Key words: Wildlife diseases, Scandinavia, review, cataract, atherosclerosis, ethmoid tumors, myxomatosis, rabies, sarcoptic mange, environmental pollution.

INTRODUCTION

In Europe, studies on wildlife pathology date from the beginning of the present century. These include studies by Horne (1911) on tularemia from lemmings (*Lemmus lemmus*) in Norway and a general survey of wildlife diseases by Olt and Strose (1914). More recently, there have been a number of surveys including studies by Christiansen (1949) in Denmark, Reydellet (1960) in France, Andersson (1959) in Finland, Krause (1939) in the Federal Republic of Germany, Vaccari et al. (1960) in Italy, Holt (1965) in Norway, Varela (1967) in Portugal, Bergman and Klarin (1925) and Borg (1956, 1975) in Sweden, Bouvier (1960) in Switzerland, Keymer (1958) and McDiarmid (1962) in the United Kingdom, and Valentincic (1960) in Yugoslavia. In addition, there are a large number of publications on specific wildlife diseases.

Wildlife pathology is a comprehensive discipline. The disease problems in a particular country are determined in part by the endemic fauna, geographical location, climate, topography, etc., and also by the educational background and interests of the resident wildlife pathologists. In the northern latitudes, wildlife diseases are associated often with starvation and metabolic disturbances resulting from qualitative and quantitative nutritional deficiencies during severe winters. In the more southerly latitudes, infectious and parasitic diseases are often responsible for wildlife mortalities.

In addition to providing valuable information on wildlife species and their ecology and population biology, many wildlife diseases are important domestic animal pathogens and human zoonoses. Many species of wildlife are excellent indicator organisms for environmental pollution and other stressers. The present paper discusses some examples of major wildlife diseases with particular reference to those occurring in wildlife from Scandinavia.

NONINFECTIOUS DISEASES

Cataract occurs often in moose (*Alces alces*) (Kronevi et al., 1977). This may be congenital or caused by a variety of postnatal factors such as senility, trauma, metabolic diseases, nutritional deficiencies, radiation, inflammatory diseases, and

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chemical substances. Most cases submitted to the National Veterinary Institute, Stockholm/Uppsala, Sweden indicated that cataract was a result of congenital and/or senility as well as from ergot (*Claviceps purpurea*) intoxication. The question as to whether or not moose with cataract and other eye disorders with resultant impaired vision may have contributed to the increasing number of road accidents involving these animals remains to be resolved.

Atherosclerosis

Although lesions do occur, clinical cases of atherosclerosis are uncommon in wildlife from Sweden (Poungshompoo, 1985). However, the lesions can be induced in hybrids of the hares *Lepus timidus* and *L. europaeus* which have been developed as an animal model for human atherosclerosis. In the hybrids of these two species of hares the allele for the enzyme glucose-6-phosphate dehydrogenase on both X-chromosomes are not identical which allows tracing of the origin of the vessel lesions (Lee et al., 1981). These hybrids are produced on a hare farm in Sweden.

Ethmoid tumors

Ethmoid tumors are infiltrative neoplasms of the mucous membrane covering the ethmoid bone. From 1947 to 1982 Borg and Nilsson (1985) reported these neoplasms from 35 moose and four roe deer (*Cervus capreolus*). These neoplasms extensively infiltrate the soft and hard tissues of the ethmoid region and may extend into the brain cavity and subcutaneous tissues of the forehead. Clinical signs included a nasal suppurative and/or bloody discharge, external outline aberrations and disorders related to central nervous system injury. These tumors were found only in female moose.

Ethmoid tumors were described originally in cattle and horses from Sweden and Norway (Stenstrom, 1909, 1915; Magnusson, 1915; Horne and Stenersen, 1916). Mutiple cases occurred in some herds indicating that these neoplasms may have a viral etiology. The geographical distribution of cases of ethmoid tumors in cattle and moose was similar, indicating a mutual transmission. Because these neoplasms have disappeared from cattle but not from moose in Scandinavia, it appears that moose may be the primary reservoirs for the disease.

Although ethmoid tumors have been observed in Scandinavia since the early 1900's, they have been discovered only recently elsewhere. They were found in cattle from India (Rajan et al., 1972), Brazil (Tokarnia et al., 1972) and the Dominican Republic (Pospischil et al., 1979). A Herpesvirus was isolated from tumor tissue in cattle from India (Moreno-Lopez, 1984, pers. comm.).

Various species of birds have been implicated in the transmission of a number of viral infections (Eccles, 1924; Rasmussen, 1938; Thompson, 1961; Borg and Bakos, 1963; Kull, 1966; Borg, 1985). Examples include foot and mouth disease in artiodactyls by starlings (*Sturnus vulgaris*), myxomatosis in European rabbits (*Oryctolagus cuniculus*) by buzzards (*Buteo buteo*) and crows (*Corvus* spp.), viral enteritis in mink (*Mustella vison*) by gulls (*Larus* spp.), and tick-borne encephalitis by blue throats (*Luscinia svecicia*). Apparently, there may be a greater than casual association between the bluethroat and the occurrence of ethmoid tumors in moose from Scandinavia and cattle from India. In spring and summer these birds reside in moose habitat. In autumn, they migrate to southern India where they overwinter. If ethmoid tumors are caused by a virus, the bluethroat may be responsible for transmission of the viruses and possibly introduced the disease to India from Scandinavia.

INFECTIOUS DISEASES

Myxomatosis

In 1938, myxomatosis appeared unexpectedly for the first time in the European

rabbit population of southern Sweden. Not until 20 yr later, it was realized that the disease was introduced purposely for rabbit control (Borg, 1962). Three individuals were involved in its introduction: Professor Gustav Hulphers, Gustaf Lundberg, and Johan Berg von Linde. Because Professor Hulphers was acquainted with Dr. Georges Bouvier in Lausanne, Switzerland, it seems likely that the Lausanne strain was used. Initially, there was some mortality, but after a few months the disease disappeared from the rabbit population in southern Sweden.

Using the Lausanne strain, another intentional release of Myxomavirus in France in 1952 resulted in an extensive epizootic that spread over the European rabbit's distribution in western Europe. In 1953, the disease appeared in England where it was evidently released by a British farmer. By 1961 the virus had reached Sweden. It is still not known whether or not it was intentionally introduced or spread by natural means.

The myxomatosis epizootic caused high mortality in rabbits and subsequent severe ecological ramifications. In many forested areas the vegetation recovered. In France, many munition-works and hat factories using rabbit fur were closed; as were a number of boarding-houses dependent on weekend rabbit hunters (Reydellet, 1953). In Great Britain, the myxomatosis epizootic coincided with the use of dieldrin for the treatment of seeds. The treated seeds were ingested by pheasants (*Phasianus colchicus*) and wood pigeons (*Columba palumbus*) which became intoxicated. These birds became easy prey for foxes (*Vulpes vulpes*) and replaced the rabbits as a food resource. Thus, instead of starvation due to loss of the rabbit food resource base, many foxes succumbed to secondary dieldrin poisoning.

As a newly introduced disease in Scandinavia, the myxomatosis epizootic provided many opportunities for epizootiological studies. For example, it became obvious that buzzards and crows were important in the spread of the infection (Borg and Bakos, 1963).

Rabies

The present rabies epizootic in Europe probably originated from a known endemic focus in central Poland. Beginning in 1935 and with a remarkably constant range extension of 30–50 km/yr, it spread over much of central Europe. By 1964 it reached Denmark where it was controlled successfully (Muller, 1971). A reduction in the number of foxes to ≤ 1 fox/5 km^2 resulted in the eradication of rabies in this fox population. Later, rabies was discovered in Arctic foxes (*Alopex lagopus*), reindeer (*Rangifer tarandus*), and seals (*Phoca* spp.) on Svalbard Island (Spitsbergen, Norway). This island is very sparsely populated which makes it difficult to ascertain when and how the virus was introduced. There may be no relationship with the concurrent rabies epizootic in the European continent and that in Svalbard Island. Rabies may have been introduced by Arctic foxes following polar bears (*Ursus maritimus*) and scavenging on their prey during their migration.

Sarcoptic mange

A variety of *Sarcoptes scabiei* that was very pathogenic to red foxes and infected other hosts such as dogs, lynx (*Lynx lynx*), martens (*Martes martes*), and human beings appeared in Finland in 1967 (Henriksson, 1971), in Sweden in 1972 (Borg et al., 1976) and in Norway in 1975 (Holt, 1976). Foxes succumbed 2–4 mo after infection and the fox populations were severely reduced. Consequently, small game populations increased considerably. In Denmark and on the European continent, sarcoptic mange has been endemic for a long period of time. However, this variety of *S. scabiei* in foxes usually has less severe consequences on individual foxes and on the fox population.

In the red fox population in Sweden,

approximately 95% of the foxes consistently remain within a defined home range and exhibit marked territoriality. However, a few of these animals may move considerable distances. Ellstrom (1981, pers. comm.) found an individual originally marked at Soderhamm that was recovered 3 yr later at Lulea, about 730 km distance from the original capture site. These individual foxes that wander considerable distances from their original home ranges represent a small, but very important, component of the fox population since they may be very important in the introduction of epizootic diseases such as rabies and sarcoptic mange into a new region.

OTHER MORTALITY FACTORS

In the late 1930's there was a marked decrease in numbers of capercaillie (*Tetrao urogallus*), black grouse (*Lyrurus tetrix*), and hazel hen (*Tetrastes bonasia*) in Sweden. Although many factors were examined such as climatological conditions (Hoglund, 1955) and pathogenic protozoan infections including toxoplasmosis and leucocytozoonosis (Borg, 1953, 1961; Morner and Wahlstrom, 1983), the above reduction in tetraonid populations could be explained only partially. However, during the 1930's modern forestry management techniques created very favorable habitat for small rodents resulting in substantial increases in their numbers. Since small rodents are the main food resource base for foxes, their numbers increased also. Hunting statistics indicated a four- to five-fold increase in the annual numbers of hunter-killed foxes. It was during this period that the tetraonid populations declined dramatically.

Food habit studies, based on stomach contents, indicated that tetraonids and their eggs constituted only a minor part of the diet of red foxes in Sweden. While this may have been the situation for individual foxes, the tetraonids may not have been refractory to the four- to five-fold increase in pressures from predation by foxes. This seems to be confirmed by the fact that following the substantial reduction in numbers of foxes due to the sarcoptic mange epizootic in the early 1970's, the tetraonid populations have recovered in many places (Borg, 1983).

ENVIRONMENTAL POLLUTION

Effluents from industry, mining and agriculture may affect wildlife directly or indirectly, and with severe ecological consequences. In Sweden, we became aware of the problems of mercury in the environment as early as 1950, 12 yr prior to the publication of Rachel Carson's book "Silent Spring." In spite of early warnings, not much attention was directed toward this discovery (Borg, 1958; Borg et al., 1969; Riley, 1984).

Presently, acidification is one of the more serious problems in Scandinavia. It resulted in the formation of soluble metal salts in the environment, leading to a considerable accumulation of cadmium and other metals in the tissues of wildlife. Although toxic levels apparently have not yet been reached in terrestrial wildlife, the residues constitute a potential hazard to humans. Organs such as kidneys and livers of wildlife should not be used for human consumption.

Because of acidification, other minerals such as selenium salts have become less accessible. However, selenium levels in wildlife seems to still be adequate based on glutathione peroxidase levels in the tissues of many wildlife species. Alternatively, it should be mentioned that past non-selective release of selenium containing waste from ceramic industries into the environment may have resulted in some toxicity to certain species of wildlife. In a number of cases, on one or more extremities of roe deer and moose exposed to excessive amounts of selenium in their diet, we noted excessive growth of the claw horn and dew claw. Also, in certain small areas of the human skin it was noted that indi-

vidual hairs seemed to have a very rapid growth rate. Although the evidence is circumstantial, these clinical signs are consistent with excessive dietary intake of selenium.

CONCLUDING REMARKS

The study of wildlife disease and pathology should not be considered as an exclusive and isolated discipline, but as an important and valuable contribution to medical research for the benefit of mankind and society. Specifically, this may be summarized as follows. Results of wildlife disease research in terms of comparative pathology should be applied to general veterinary and human medicine where applicable. The significance of certain species of wildlife should be recognized as important indicator organisms of environmental pollution, and this information should be translated to potential hazards to the human population. For zoonotic diseases, it is important to ascertain their transmission from wildlife to domestic animals and man, particularly their dissemination by birds and other widely ranging and migratory species that may spread the organisms over extensive geographic areas. Also, epizootics in wildlife species provide unique opportunities to investigate mechanisms of establishment, maintenance, and transmission of infectious agents in natural populations. Certainly, it is important to clarify the ecological implications and consequences of epizootic diseases in wildlife to agriculture and forestry.

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ABOUT THE AUTHOR

Karl Yngve Borg, a native of Sweden, was born 16 September 1917. He received the Doctor of Veterinary Medicine degree in 1945 and the Doctor of Philosophy degree in the field of Veterinary Science in 1953, both from the Royal Veterinary College, Stockholm. Dr. Borg's distinguished career began in 1945 when he joined the staff of the National Veterinary Institute, Stockholm. In 1969 he became Professor and Head of Wildlife Pathology. Professor Borg has

published some 185 scientific papers on wildlife diseases, including zoonoses and the impact of environmental pollution on wildlife. He is a consultant expert of the Swedish Board of Chemical Products and Honorary Secretary of the International Union of Game Biologists. His many awards include Decorated Knight of the Royal Swedish North Star Order and Recipient of the Gold Medal of His Majesty the King of Sweden, the Gold Medal of Princess Eugenie of Sweden, the Silver Plaque of the Central Union of Swedish Sportsmen, the Gold Medal of the General and National Societies of Animal Protection, the Gold Medal of the Swedish Veterinary Association, and the Gold Plaque of the Royal Swedish Academy of Sciences. Long a member and supporter of the Wildlife Disease Association, Professor Borg was elected an Honorary Member of the Wildlife Disease Association in 1985 following his retirement.