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Viral diseases of northern ungulates

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Abstract: This paper describes viral diseases reported in northern ungulates and those that are a potential threat to these species. The following diseases are discussed: bovine viral diarrhoea/mucosal disease (BVD/MD), alphaherpesvirus infections, malignant catarrhal fever (MCF), poxvirus infections, parainfluenza type 3 virus infection, Älvsborg disease, foot-and-mouth disease, epizootic haemorrhage disease of deer and bluetongue disease, rabies, respiratory syncytial virus infection, adenovirus infection, hog-cholera, Aujeszky's disease and equine herpesvirus infections. There are no significant differences in antibody prevalence to BVDV among deer in habitats with high, intermediate and low density of cattle. In addition, sequence analysis from the BVDV isolated from roe deer (*Capreolus capreolus*) showed that this strain was unique within BVDV group I. Disrinct BVDV strains might circulate in free-ranging roe deer populations in Germany and virus transmission may be independent of domestic livestock. Similar results have been obtained in a serological survey of alpha-herpesviruses in deer in Germany. Malignant catarrhal fever was studied in fallow deer (*Cervus dama*) in Germany: the seroprevalence and positive PCR results detected in sheep originating from the same area as the antibody-positive deer might indicate that sheep are the main reservoir animals. Contagious ecthyma (CE) is a common disease in domestic sheep and goats caused by the orf virus. CE has been diagnosed in Rocky Mountain bighorn sheep (*Ovis canadensis*), mountain goats (*Oreamnos americanus*), Dall sheep (*Ovis dalli*), chamois (*Rupicapra rupicapra*), muskox (*Ovibos moschatus*) and reindeer (*Rangifer tarandus*). Most parainfluenza type 3 virus infections are mild or clinically undetectable. Serological surveys in wildlife have been successfully conducted in many species. In 1985, a new disease was identified in Swedish moose (*Alces alces*), designated as Älvsborg disease. This wasting syndrome probably has a multi-factorial etiology. Foot-and-mouth disease virus (FMDV) can infect deer and many other wild artiodactyls. Moose, roe deer and the saiga antelope (*Saiga tatarica*) are the main hosts of FMDV in the Russian Federation. In addition, serological evidence of a FMD infection without clinical disease was detected in red deer in France. Epizootic haemorrhage disease of deer (EHD) and bluetongue (BT) are acute non-contagious viral diseases of wild ruminants characterised by extensive haemorrhage. Culicoides insects are the main vectors. EHD and BT only play a minor role in Europe but both diseases are widespread in North America.

Key words: holarctic, cervid, moose, muskoxen, reindeer, viral disease.

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Introduction

This paper documents viral diseases in northern ungulates including those which are a potential threat to these species. The following species are discussed: reindeer/caribou (*Rangifer tarandus*), roe deer (*Capreolus capreolus*), red deer (*Cervus elaphus*), wapiti (*Cervus elaphus canadensis*), fallow deer (*Dama dama*), sika deer (*Cervus nippon*), moose (*Alces alces*), white-tailed deer (*Odocoileus virginianus*), mule deer (*Odocoileus hemionus*), muskox (*Ovibos moschatus*), bison (*Bison bison*), chamois (*Rupicapra rupicapra*), ibex (*Capra ibex*), bighorn sheep (*Ovis canadensis*), mountain goat (*Oreamnos americanus*), Dall sheep (*Ovis dalli*), wild boar (*Sus scrofa*), pronghorn (*Antilocapra americana*) and Przewalski's wild horse (*Equus przewalskii*).

Bovine virus diarrhoea/mucosal disease (BVD/MD)

Bovine virus diarrhoea virus (BVDV) belongs to the genus *Pestivirus* within the family Flaviviridae.

There is a close antigenic relationship to classical swine fever and Border disease virus of sheep (Horzinek, 1991). BVDV is a major pathogen of cattle with world-wide economic impact (Thiel *et al.*, 1996). Signs in cattle include transient acute infections which may be inapparent or mild, or mucosal disease which is inevitably fatal. Transplacental infection leads to abortion, foetal malformations and development of persistently viremic calves depending on the state of development of the foetus and the biotype (cytopathogenic or non-cytopathogenic) of the virus (Moenning & Liess, 1995; Brownlie, 1990). It is unknown whether this applies to wild ruminants as well (Depner *et al.*, 1991) although viremic individuals in wild ungulates were demonstrated by experimental infection (Morton *et al.*, 1990; Hyera *et al.*, 1993; Van Campen *et al.*, 1997).

Primary clinical signs in wild ruminants are erosion and ulceration of the oral mucosa, haemorrhagic enteritis and general physical impairment. Clinical signs include weakness, lack of fear, impaired hearing and vision, dehydration and emaciation. Pyrexia, anorexia, salivation and nasal discharge usually also occur, while some cases have skin lesions and may be lame due to interdigital ulceration and inflammation of the coronary bands (Richards *et al.*, 1956; Romvary, 1965; Wiesner, 1987; Neumann *et al.*, 1980; Morton *et al.*, 1990; Nettleton, 1994).

Serological surveys in free-ranging populations have been successfully conducted in more than 40 species on several continents (Nettleton, 1990).

In the United Kingdom, serological evidence of BVDV was found in fallow deer by McDiarmid (1975) and by Lawman *et al.* (1978) (8%). Lawman *et al.* (1978) also described seropositive red deer (16%) and sika deer (9%). In France, Baradel *et al.* (1988) determined an antibody prevalence of 0.7% in roe deer, 5.5% in chamois and 7.5% in ibex. Seropositive reindeer were found by Stuen *et al.* (1993) in Norway (9%) and by Rehbinder *et al.* (1992) in Sweden (6%). In the former West-Germany, antibodies against BVDV have been found in approximately 7% of red deer (Weber *et al.*, 1978, 6.6%; Frölich, 1993, 7.7%), and in roe deer (Weber *et al.*, 1978, 5.9 %; Frölich, 1993, 10%). However, in the former East-Germany, only 0.6 % of cervid sera were determined to be seropositive (Dedek *et al.*, 1988). Of wild species other than deer, Dahle *et al.* (1993) and Oslage (1993) found BVDV neutralizing antibodies in 0.8% and 1.2% of wild boar sera which were not identical to classical swine fever positive sera. Kahrs *et al.* (1964) and Friend & Haltermann (1967) reported white-tailed deer seropositive to BVDV. In several national parks in the US, the overall seroprevalence in mule deer was 59% (Aguirre *et al.*, 1995). High seroprevalence was also detected in wapiti (54%) in several national parks in the US by Aguirre *et al.* (1995) and in Alberta by Kingscote *et al.* (1987) (52%). 18% of moose in Canada (Thorsen & Henderson, 1971) and 12% in Alaska (Kocan *et al.*, 1986) have been reported to be seropositive. In Canada, 69% of caribou tested in 1978 and 60% tested in 1979 had specific antibodies against BVDV (Elazhary *et al.*, 1981), whereas Zarnke (1983) only found 3% seroprevalence in caribou in Alaska. In pronghorns, seropositive reactors were found in Canada (4%) (Barrett & Chalmers, 1975) and Idaho (Stauber et al., 1980). Seropositive reactors were reported in bighorn sheep by Clark *et al.* (1985) (10%) and Parks & England (1974).

Virus isolations: Romvary (1965) first isolated a noncytopathogenic BVDV from the spleen of a roe deer. Schellner (1977) isolated BVDV from spleen, intestinal lymph nodes, and abomasal mucosa of roe deer suffering from abomasitis and severe enteritis. Neumann *et al.* (1980) isolated the virus from farmed fallow deer. Weber *et al.* (1982) detected noncytopathogenic BVDV in three farmed fallow deer. Diaz *et al.* (1988) demonstrated BVDV in one fallow deer. Isolation of a noncytopathogenic BVDV from the spleen of a red deer was reported by Nettleton *et al.* (1980). A pestivirus differing from BVDV was isolated from red deer by Baradel *et al.* (1988). Cytopathogenic BVDV was isolated from two seronegative roe deer from northern Germany by Frölich & Hofmann (1995).

Clinical and pathological findings: Shope *et al.* (1955) described conditions which indicated the existence of a BVDV infection in a New Jersey deer herd. In several sick or dead white-tailed deer and mule deer from various parts of North Dakota, Richards *et al.* (1956) observed profuse salivation, nasal discharge, and in one case corneal opacity. Pathological changes included reddening and ulceration of the digestive tract. Romvary (1965) described lesions typical of BVD in six free-ranging roe deer in Hungary. In Sweden, histological changes and lesions characteristic of BVD were observed in captive fallow deer (Diaz *et al.*, 1988), as well as free-ranging moose and roe deer (Feinstein *et al.*, 1987).

Transmission: the role of pestiviruses in wild ruminant populations and the interactions between wild ungulates and domestic livestock are not well understood (Nettleton, 1990; Aguirre *et al.*, 1995; Frölich, 1995). Transmission in cattle may be either horizontal, mainly oronasal via direct contact between infected and susceptible animals, or vertical. The virus is shed in secretions or excretions including nasal discharge, saliva, semen, urine, tears and milk. Faeces are usually a poor source of virus (Brownlie *et al.*, 1987). Indirect transmission by vectors also occurs (Meyling *et al.*, 1990). Tarry *et al.* (1991) reported on the possibility of insect (*Stomoxys calcitrans, Haematopota pluvialis*) transmission. The natural mode of transmission of BVDV to wild ungulates and the question of whether wild ungulates can serve as a reservoir is not yet clear. Experimental infection with BVDV in wild ruminants was demonstrated by Richards *et al.* (1956), Morton *et al.* (1990), Hyera *et al.* (1993), and Van Campen *et al.* (1997). Whether persistent BVDV infections occur in wild ruminant species as in domestic ruminants is not yet proven but there is some indication that this might happen (Hyera *et al.*, 1993). Neumann *et al.* (1980) and Kocan *et al.* (1986) assumed a causal relationship between the spread of BVDV in cattle and its occurrence in deer. Romvary (1965) diagnosed BVD in roe deer living adjacent to a cow farm where BVD had previously caused severe losses. In contrast, Weber *et al.* (1982) and Liebermann *et al.* (1989) assumed an independent infection process in wild ruminants with BVDV. Pastoret *et al.* (1988) supposed that wild species do not play a major role in transmitting infection to domestic cattle. In free-ranging deer, the highest seroprevalence (60%-70%) was detected in Canadian caribou by Elazhary *et al.* (1981) although these caribou had had no direct contact with domestic ruminants for 25 years. Frölich (1995) found no significant difference in antibody prevalence among deer in habitats with high, intermediate and low densities of cattle. The sequence analysis of the BVDV isolated from roe deer (Frölich & Hofmann, 1995) showed a unique position of this roe deer strain within the BVDV group I (Fischer *et al.*, 1998). This study indicated that distinct BVDV strains might circulate in free-ranging roe deer populations in Germany and that virus transmission is independent of domestic livestock (Fischer *et al.*, 1998).

Alphaherpesvirus infections

Serological surveys performed in different species of deer revealed the presence of alphaherpesviruses related to bovine herpesvirus-1 (Nettleton *et al.*, 1988). Such viruses include BHV-1, which causes infectious bovine rhinotracheitis (IBR) and pustular vulvovaginitis (IPV) in cattle (Ludwig & Gregersen, 1986), the herpesvirus of red deer (HVC-1) (Inglis *et al.*, 1983; Reid *et al.*, 1986), the *Rangifer* herpesvirus (RanHV-1) isolated from reindeer (Ek-Kommonen *et al.*, 1986) and that from goats (caprine herpesvirus-1; CapHV-1) (Engels *et al.*, 1992). The clinical symptoms in deer associated with these herpesvirus infections include conjunctivitis, lacrimation and corneal lesions. Ulceration of the nostrils and a serous or purulent nasal discharge may also occur (Inglis *et al.*, 1983; Nettleton *et al.*, 1986; Reid *et al.*, 1986).

Serological surveys in free-ranging populations have been successfully conducted in many northern ungulates. Thiry *et al.* (1988) found a low seroprevalence in free-ranging populations of roe deer and red deer in France and Belgium: none of the roe deer in Belgium and less than 1% of those in France were seropositive for herpesviruses. In red deer, 1% were positive in France and 11% in Belgium. In Italy, 2% of fallow deer were seropositive for BHV-1 (Giovannini *et al.*, 1988). In the southern part of former West Germany, Weber *et al.* (1978) found antibodies against BHV-1 in 9% of fallow deer, 2.5% of red deer and 1.5% of roe deer samples. In the former German Democratic Republic, 13% of red deer, 1% to 3% of roe deer and 3% of fallow deer sera were seropositive for BHV-1 (Kokles, 1977; Kokles *et al.*, 1988). Higher antibody prevalence of alphaherpesviruses was found in Britain: antibodies against BHV-1 were detected in 16% (Lawmann *et al.*, 1978) and against HVC-1 in 29% (Nettleton *et al.*, 1986) of red deer. In Alaska, serological evidence of exposure was reported for reindeer (Dieterich, 1981) and caribou (Zarnke, 1992).

Alphaherpesvirus infections also commonly appear in ungulates from Scandinavia: In reindeer in Finland antibodies against BHV-1 were found by Ek-Kommonen *et al.* (1982) (18%) and Hyllseth *et al.* (1993) (10% to 46%). In Norwegian reindeer a seroprevalence of 9% to RanHV-1 was found by Stuen *et al.* (1993) and of 32% in studies by Hyllseth *et al.* (1993).

The mode of infection in free-ranging ungulates is not yet clear. Direct contact is normally required for the natural transmission of herpesviruses in

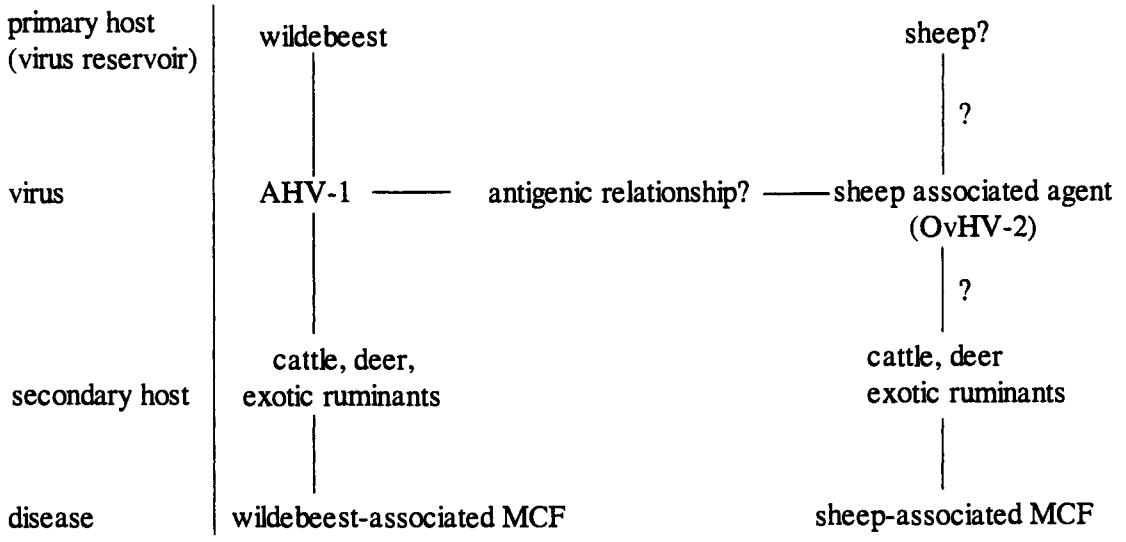

Fig. 1. Malignant Catarrhal Fever (MCF), modified after Rolle & Mayr (1993).

ungulates. In Germany, no association has been found between cattle density and antibody prevalence against alpha-herpesviruses in deer. In these deer populations, contact with cattle is obviously not essential (Frölich, 1996). This contrasts with the hypothesis of Weber *et al.* (1978) and Lawmann *et al.* (1978) who assume a transmission from a domestic to wild host and vice versa. Nettleton *et al.* (1988) and Kokles (1977) assume that herpesviruses of free-ranging deer have not so far posed a threat to other domestic livestock and that alpha-herpesviruses from deer occur only in their natural hosts and do not cross to other species. Baradel *et al.* (1988) stated that there may have been a separate parallel evolution of viruses in wild and domestic ruminants. Likewise Ronsholt *et al.* (1987) showed that cattle are not susceptible to the topical HCV-1 isolate which would not, therefore, appear to represent a health hazard to cattle.

Malignant Catarrhal Fever (MCF)

MCF affects many species of ruminants. However, there is great variation in susceptibility to infection (English, 1981; Hunter, 1981; Seal *et al.*, 1989; Reid, 1992; Mackintosh, 1993; Murphy *et al.*, 1994). Based on the reservoir ruminant species from which the causative viruses arise, the two major epizootiological entities of the disease that have been described are wildebeest-associated (WA) and sheep-associated (SA) MCF (Fig. 1). The etiologic agent for WA-MCF has been isolated, characterised as a gammaherpesvirus, and named alcelaphine herpesvirus 1 (AHV-1) (Plowright *et al.*, 1960; Bridgen *et al.*, 1989) whereas the putative SA-MCF agent has not yet been isolated (Reid, 1992). Based on its antigenic and base-sequence homology to AHV-1, the putative agent of the SA-MCF has been tentatively classified as ovine herpesvirus 2 (OvHV-2) (Roizman, 1992). MCF has been described in several species of deer (Westbury, 1984) and cervids are generally regarded as highly susceptible to MCF (Plowright, 1986; Buxton, 1988). The range of clinical signs observed in MCF affected ruminants has been diverse (Westbury, 1984; Blake *et al.*, 1990). The disease tends to be peracute or acute with animals succumbing before the more florid lesions, characteristic of protracted cases, develop (Reid & Buxton, 1989). However, MCF in ruminants can also be present as subacute or chronic disease with clinical signs becoming progressively more marked with duration of illness (Buxton, 1988).

In contrast to many reports in captive ruminants (e.g. Pierson *et al.*, 1974; Westbury, 1984, Krogh & Jensen, 1988) only single cases of MCF in free-ranging ruminants have been published. The disease was diagnosed in two free-ranging moose from Sweden showing CNS symptoms. Subsequently one was shot and the other one found dead and both were diagnosed as MCF based on histopathology (Warsame & Steen, 1989). Frölich *et al.* (1998)

Table 1. Family Poxviridae, modified after Rolle & Mayr (1993).

genus	host range
orthopoxvirus	e.g. humans, bovidae, various wild aminal species
avipoxvirus	birds
capripoxvirus	ruminants
leporipoxvirus	leporidae
suipoxvirus	pigs
molluscipoxvirus	humans
yatapoxvirus	primates
parapoxvirus	predominantly ruminants

investigated 329 samples from three species of free-ranging deer, including 253 roe deer, 22 red deer and 54 fallow deer, in which only fallow deer were antibody-positive. The few reports of the disease in free-ranging deer in Europe may reflect a lack of surveillance and awareness of the disease in wild cervids in Europe.

Few data are available on the transmissibility of SA-MCF agent in free-ranging ruminants. The cause of cervid MCF has not been determined in most cases (Buxton & Reid, 1980; Oliver *et al.*, 1983) although it is thought to be a virus carried by clinically normal sheep (Buxton, 1988). Recent studies have further implicated OvHV-2 or a similar virus (Tham, 1997; Tomkins *et al.*, 1997). Circumstantial evidence also suggests that there may be sources of virus other than sheep in SA-MCF as a number of outbreaks have been observed in which no contact with sheep was reported (Straver and van Bekkum, 1979). Domestic goats and rabbits (*Oryctolagus cuniculus*) have been mentioned as possible reservoirs (Blood *et al.*, 1979). In studies of Frölich *et al.* (1998) the seroprevalence and positive PCR results detected in sheep samples, which originate from the same area as the antibody-positive fallow deer, might indicate that in this case sheep are the main reservoir animals.

Poxvirus infections

Poxvirus infections in holarctic wild ungulates are mainly caused by species of the genus *Parapoxvirus* within the family of Poxviridae (Table 1). There are three accepted members of the parapoxvirus genus, orf virus (OV) papular stomatitis virus (PSV), and pseudocowpox virus (PCV).

Contagious ecthyma (CE), otherwise known as orf, is a common disease in domestic sheep and goats caused by the OV. CE can also affect several wild ungulates including Rocky Mountain bighorn sheep (Lance *et al.*, 1981), mountain goats (Blood, 1971), Dall sheep (Smith *et al.*, 1982), chamois and muskox (Falk, 1978; Kummeneje & Krogsrud, 1978), as well as reindeer (Kummeneje & Krogsrud, 1979). Lance *et al.* (1983) conducted experimental infections in mule deer, white-tailed deer, pronghorns, and wapiti. Parapoxvirus has also been isolated from red deer (Robinson & Mercer, 1995).

Clinical signs: OV enters its host through skin abrasions (Bruner & Gillespie, 1973). In bighorn sheep and Dall sheep the disease is characterised by lesions of the lips, mammary gland and teats, muzzle, legs, vulvae, and occasionally the eyes and hooves. Most lesions start as discrete reddened swellings, followed by pustules and ulcers in 3 or 4 days. Lesions usually disappear in 2 to 4 weeks. Most infections are seen in lambs but mild cases are observed in adult ewes and rams (Blood, 1971). Clinical signs appear to be more severe in hot weather and to improve in cold weather (Dieterich *et al.*, 1981). In reindeer CE is benign (Kummeneje, 1979). However, during an outbreak of CE in Finnish semi-domesticated reindeer in winter 1992-1993, 400 individuals died as a result of secondary bacterial infection and starvation (Tryland *et al.*, 1995). The clinical picture showed cauliflower-like papillomas mainly around the mouth and lips. Similar lesions on the lips, muzzles and nostrils and to a lesser extent on the neck, eye lids, chest and in the perianal region could be observed in CE affected muskox (Kummeneje & Krogsrud, 1978). The affected animals often suffered from dyspnoe and feeding problems as well as from secondary bacterial infections (Kummeneje & Krogsrud, 1978). During an outbreak of CE in a herd of captive muskox in Norway all members of the herd showed signs of infection. Five of eight males died while the 11 females only showed small warts. CE never reoccurred in the animals which survived this outbreak (Mathiesen *et al.*, 1985). In red deer the virus produced lesions similar to those seen with OV in sheep (Robinson & Mercer, 1995).

Parhological findings: The papilloma-like lesions of CE affected muskox in Greenland were grossly and histologically similar to common warts (Kummeneje & Krogsrud, 1978). Biopsy of lip nodules in a captive muskox in Alaska revealed proliferating tubes of keratinized epidermis projecting upward over a core of dermis. Many cells in these epithelial papillae had ballooning degeneration and

papillae had partially necrotic patches of epithelium. Numerous inflammatory cells were seen throughout the biopsy with neutrophils predominating on the surface and in the epidermis. Bacterial colonies were seen in the necrotic areas. Lymphocytes and macrophages predominated in the dermal tissue (Dieterich *et al.,* 1981). Superficial dermatitis with eosinophilic intracytoplasmatic inclusion bodies were seen in biopsy material of teat lesions of an Alaskan Dall sheep (Smith *et al.,* 1982).

CE is very common in sheep and goats in Norway and transmission of parapoxvirus from these animals to reindeer or muskox seems highly likely. It is believed that the infection of a muskox herd in Greenland was of ovine or caprine origin (Kummeneje, 1979). However, the presence of a reindeer strain of the virus cannot be excluded (Kummeneje, 1979) and the identity and the host specificity of newly found parapoxvirus isolates still has to be investigated (Büttner *et al.,* 1995). Transmission occurs by contamination of abrasions of mucous membranes or skin, with exudate or scabs. The virus is very stable in dried scabs. Indirect transmission of the infection via objects such as knifes and barbed wire has been reported (Leavell *et al.,* 1968; Johannessen *et al.,* 1975). Lambs infected on the mouth may transmit the infection to their mothers during suckling. Regular use of salt blocks by bighorn sheep also appears to be important in maintaining the disease (Blood, 1971).

The genus *Orthopoxvirus* within the family Poxviridae causes diseases in a wide range of species. Little is known about the occurrence of orthopoxviruses in wildlife species (Tryland, 1998). Mayr *et al.* (1995) detected orthopoxvirus-specific antibodies in wild boar in Germany.

Parainfluenza type 3 virus (PIV-3) infection

PIV-3 commonly causes respiratory infection with little or no clinical manifestation. However, in association with other viral and bacterial pathogens and stress-inducing situations, it causes a severe pneumonia in cattle called «shipping fever». Parainfluenza viruses are classified in the genus *Paramyxovirus* within the family Paramyxoviridae. Four serotypes of parainfluenza virus have been described but almost all infections in livestock are caused by serotype 3. The virus is shed in nasal and ocular secretions. Persistently infected animals have not been reported (Woods, 1968; Frank & Marshall, 1973; Kingsbury *et al.,* 1978). Most parainfluenza infections are mild or clinically undetectable. Development of clinical disease is usually dependent on interaction with infectious and environmental factors. The virus, together with *Pasteurella* species, can lead to pneumonia and death. Fever is accompanied by lachrymation, serous to mucopurulent nasal discharge, depression and dyspnoea. Recovery is the general rule, but when secondary bacterial infection occurs, dyspnoea and depression may be severe or fatal (Lopez *et al.,* 1976; Lehmkuhl & Cutlip, 1982). Serological surveys have been conducted in many species: in white-tailed deer antibodies against PIV-3 were found in Minnesota (20 %; Ingebrigtsen *et al.,* 1986) and Canada (around 80%; Sadi *et al.,* 1991). A high seroprevalence also was detected in pronghorns (49%) in southeastern Alberta (Kingscote & Bohac, 1986) and in free-ranging bison (67%) in Alaska (Zarnke, 1983). In 8 national parks in western US, the overall prevalence in wapiti was 46% and for mule deer 32% (Aguirre *et al.,* 1995). Clark *et al.* (1993) detected that 10% of bighorn sheep had been exposed to PIV-3.

In Germany and Italy, Kokles *et al.* (1988), Giovannini *et al.* (1988) and Maglione *et al.,* (1992) determined antibodies against PIV-3 in fallow deer, red deer, and roe deer, with a prevalence of 9% to 20%. However, in a serological survey in reindeer in Norway all 326 sera tested for antibodies against PIV-3 were negative (Stuen *et al.,* 1993).

Virus isolates of PIV-3 were possible from nasal swabs or secretions from fallow deer, mule deer, and pronghorns in Alberta (Thorsen *et al.,* 1977).

Älvsborg disease

In 1985, a new disease was identified in Swedish moose and designated Älvsborg disease. The name 'Älvsborg' originates from a region in Southern Sweden, where the disease was first observed. Between 1985 and 1995, Älvsborg disease killed more than a thousand moose. The actual number of affected moose temains unknown (Rehbinder *et al.,* 1991; Steen *et al.,* 1993; Merza *et al.,* 1994). The disease affects all age classes of moose. The post mortem picture of Älvsborg disease is characterised by erosive, ulcerative and necrotic lesions in the mucous membranes of the digestive tract, atrophied lymphoid organs and emaciation (Merza *et al.,* 1994).

This wasting syndrome probably has a multifactorial etiology. The pathological changes, as well as

serological findings, indicate the possible presence of BVDV (Cedersmyg, Steen, Frank, Frölich and Rehbinder, unpubl. data). Moreover, a retrovirus (*Alces* leucotropic oncovirus, ALOV) has been isolated (Merza *et al.*, 1994). Retroviruses are known to cause wasting diseases and immunodepression in domestic ruminants. In addition, unusually low levels of copper, chromium and zinc had been observed in the livers of these moose. Undernutrition and malnutrition resulting in starvation and emaciation is considered an important factor having a profound and adverse effect on trace element levels (Cedersmyg, Steen, Frank, Frölich and Rehbinder, unpubl. data). In conclusion, Älvsborg disease is regarded as a multifactorial disease but the etiology of this disease is not yet fully elucidated.

Foot-and-mouth disease (FMD)

FMD is a highly contagious acute viral infection almost exclusively of ruminants and pigs. It is characterised by high morbidity and low mortality. A variety of other wildlife species becomes infected periodically but there is little evidence that they are important for viral maintenance or transmission to cattle. FMDV belongs to the family Picornaviridae, and is the only member of the genus *Aphthovirus.* Seven serological types have been found: A, O, C, SAT-1, SAT-2, SAT-3 and Asia 1 (Thomson, 1994). The virus is resistant to external influences and may survive for many weeks. FMDV can be transmitted by the airborne route and may be transported over considerable distances (Hedger, 1981).

Clinical symptoms: In all species, foot lesions develop in the interdigital space. Secondary bacterial infections of foot lesions frequently occur, particularly where animals are kept in muddy, unhygienic conditions. Moreover, FMD is characterised by the development of lesions in the mouth. The young of domestic species susceptible to FMD may die suddenly as a result of myocarditis. This is referred to as «tiger-heart disease» (Thomson, 1994). The respiratory tract is the usual route of infection. Virus is excreted not only during the clinical manifestations of disease. In some species infection may take place and virus may be excreted in the total absence of clinical signs (Hedger, 1981). An overview about the occurrence of FMDV in wild artiodactyl animals in the holarctic region was presented by Rea-Min *et al.* (1997). Moose, roe deer and saiga antelope are the main hosts of FMDV in the Russian Federation (Kruglikov *et al.,* 1985). In addition, serological evidence of FMD infection without clinical disease was detected in one of 88 free-ranging red deer in France by Barrat *et al.* (1988).

Epizootic haemorrhage disease of deer (EHD) and bluetongue disease (BT)

EHD is an acute non-contagious viral disease of wild ruminants characterised by extensive haemorrhage. EHD and BT are caused by antigenically related though distinct viruses and are clinically and pathologically rather similar. Culicoides insects are the main vector (Alexander & Buxton, 1994).

The clinical course is usually acute and rapidly fatal, while wapiti develop only a mild disease. The symptoms of EHD are characterised by extensive hemorrhages, progressive weakness, terminal coma and death. Animals develop a mucopurulent nasal discharge, conjunctivitis and coronitis. Widespread haemorrhages of the mucous membranes and intestinal serosa are typical at post-mortem examinations (Wallach & Boever, 1983; Alexander & Buxton, 1994; Rolle & Mayr, 1993).

The clinical signs for BT are similar to EHD; namely excessive salivation with a purple-blue discoloration of the tongue, caused by circulatory disorders. Hemorrhages in the pulmonary artery are pathognomic. Congenital malformations and abortion have been reported following exposure to BT in the first trimester of pregnancy (Wallach & Boever, 1983; Dedek & Steineck, 1994).

Although EHD and BT only play a subordinate role in Europe (BT only occurs in Spain and only a few reports of EHD exist for Great Britain), transmission to other parts of Europe may be possible (Dedek & Steineck, 1994). In North America, EHD was first recognised as a specific disease in white-tailed deer in the mid 1950s when die-offs occurred in New Jersey and Michigan. Since then, many outbreaks have occurred (e.g. Alberta, North and South Dakota, Missouri, Nebraska, Texas and Washington) and serological studies have been performed in various parts of the US (e.g. Chalmers *et al.,* 1964; Fay *et al.,* 1956; Fosberg *et al.,* 1977; Hoff *et al.,* 1973; Prestwood *et al.,* 1974; Trainer & Karstad, 1970; Shapiro *et al.,* 1991; Nettles *et al.,* 1992; Fischer *et al.,* 1995; Stallknecht *et al.,* 1995; Stallknecht *et al.,* 1996; Farnell *et al.,* 1999).

In the US, BT was noted as a disease of white-tailed deer in 1955 (Fay *et al.,* 1956). In 1976, an extensive wildlife die-off due to bluetongue occurred in the Missouri River Basin. During that

die-off, at least 4100 pronghorn antelopes and deer died in Wyoming (Thorne *et al.*, 1982). Since then, outbreaks have occurred and serological studies have been performed in various parts of the US (e.g. Kocan *et al.*, 1982; Dulac *et al.*, 1988; Stallknecht *et al.*, 1991; Pearson *et al.*, 1992).

Rabies

Rabies is an acute infectious disease of the central nervous system caused by a virus that generally persists in natural hosts as a salivary gland infection in carnivores. The virus is usually transmitted from animal to animal and from animal to man by biting. All warm-blooded animals are susceptible. An animal with clinical signs of rabies should be killed and its brain examined for Negri bodies. A Negri body is well differentiated by Sellers's stain as a magenta (purplish red) round or oval body with blue to black, basophilic, internal bodies (Sikes, 1981).

The arctic fox (*Alopex lagopus*) serves as a reservoir and vector for rabies in most Arctic regions, and outbreaks of disease coincide with population peaks and migrations. Cases in other species are only sporadic (Ødegaard & Krogsrud, 1981).

Deer are susceptible to infection with rabies if bitten by a rabid carnivore but are represent hosts incidental to the epizootiology of the virus (Ødegaard & Krogsrud, 1981). Sporadic cases of rabies have been diagnosed in reindeer (Ødegaard & Krogsrud, 1981; Prestrud *et al.*, 1992) and moose (Lis, 1991; Anonymous, 1996; 1997; Muller *et al.*, 1998). Rabies is also known to occur in fallow deer and red deer (Anonymous, 1992 a; b; Cac *et al.*, 1992). A relatively high number of cases of rabies, however, were reported in roe deer from several European countries (Schulz, 1986; Blancou & Barrat, 1988; Duricic *et al.*, 1988; Birlbauer *et al.*, 1990; Lis, 1991; 1996; Anonymous, 1991; 1992 b, 1994). A change in behaviour may suggest a rabies infection in free-ranging cervids but a clinical diagnosis cannot be carried out with certainty in a living herbivore. The terms «furious rabies» and «dumb rabies» which point to certain behavioural features are not appropriate for non-carnivorous animals. In addition, especially in ruminants, the inflammatory changes of the brainstem are often inconspicuous and may be confined to a few brain vessels with cuffing lymphocytes and a very small glial nodules, commonly called Babes' nodules (Jubb *et al.*, 1993).

Adenovirus infection

Adenovirus infections are probably widespread although most are subclinical. Bovine adenovirus infections have been associated with a variety of respiratory and alimentary tract diseases but their role in the causation of these diseases remains uncertain (Thomson, 1994). Antibodies to bovine adenovirus have been found in red deer, fallow deer, roe deer, and sika deer in Great Britain. The reaction rate was highest in fallow deer but no clinical disease associated with the infection has been noted. In Hungary, type 6 bovine adenovirus was responsible for an outbreak of respiratory disease in a group of captive fallow deer. One buck which died showed acute tracheitis and interstitial pneumonia (Alexander & Buxton, 1994). In France, Barrat *et al.* (1988) found antibodies against bovine adenovirus in 33 of 89 serum samples collected from wild red deer.

Thousands of mule deer were killed by a haemorrhage disease and an apparently novel adenovirus was associated with this epizootic in California (USA) during 1993-1994. A systematic vasculitis with pulmonary edema and haemorrhage enteropathy or a localised vasculitis associated with narcotising stomatitis/pharyngitis/glossitis or osteomyelitis of the jaw were common necropsy findings in the animals that died (Woods *et al.*, 1997). Artificially infected mule deer showed identical histological findings to free-ranging animals which died naturally (Woods *et al.*, 1997).

Respiratory syncytial virus (RSV) infection

The virus is classified as a member of the genus *Pneumovirus* in the family Paramyxoviridae. Infection with bovine respiratory syncytial (BRS) vitus is undetectable in the majority of animals but in some it does cause mild to severe respiratory tract disease characterised by fever, coughing, serous nasal and ocular discharges and dyspnoea. It is one of several viruses which are primary pathogens in the bovine respiratory disease complex (Van Vuuren, 1994). However, RSV does not appear to be a problem in farmed, park, or free-living deer. Under experimental conditions, virus isolated from sheep was transmitted to white-tailed deer calves. Clinical disease was not recognised although lung lesions, similar to those found in lambs, developed. Virus was recovered from the lower respiratory tract but transmission to deer did not occur (Alexander & Buxton, 1994). However, serological evidence of RSV in wildlife is available from different countries. In

North America, antibodies against BRS virus were found in free-ranging white-tailed deer, mule deer, bighorn sheep and mountain goats (Clark *et al.*, 1985; Dunbar *et al.*, 1985; Johnson *et al.*, 1986). In eight national parks in the western US, 54% of wapiti were seropositive (Aguirre *et al.*, 1995). However, Kingscote *et al.* (1987) and Hein *et al.* (1991) found no serological evidence in wapiti collected in Alberta and Central Washington, respectively. In Italy, six of 43 sera of free-ranging fallow deer (Giovannini *et al.*, 1988) and 7% of red deer were positive for antibodies against BRS virus (Maglione *et al.*, 1992).

Hog cholera

Hog cholera is an acute, highly fatal disease affecting wild boar of all ages. It is characterised by sudden onset, high morbidity and very high mortality. Transmission is accomplished by direct contact or by ingestion of virus-contaminated feed or water. Young animals which recover are permanently stunted. Clinical signs include anotexia, diarrhoea, neurological symptoms and high fever. The disease may last from 24 hours to 16 days. Wild hogs lose their shyness and develop polydipsia as a result of high fever. Post mortem lesions are characterised by petechial hemorrhages on serosal surfaces and in the renal cortex. Chronically infected individuals may show «button ulcers» 10 mm in diameter which are associated with the intestinal mucosa (Wallach & Boever, 1983; Loepelmann & Dedek, 1991; Dedek & Steineck, 1994). Presently, hog cholera officially occurs in wild boar in six European countries: Germany, Italy, Austria, France, Slowakia and Czechia.

Aujeszky's Disease

Although many species of domestic animals are susceptible to infections by pseudorabies virus (PrV), pigs are considered to represent the main host reservoir. Only limited data exist about natural infection in wildlife. During 1991-1994 European wild boar were serologically and virologically investigated for the occurrence of PrV-infections in Eastern Germany by Müller *et al.* (1996). 281 (8.9%) of the tested sera were positive in ELISA. Reactivity was confirmed by presence of neutralizing antibodies in 220 sera and by immunoblotting. Based on epidemiological analysis the authors concluded that PrV-infections occurred in wild boar populations of the examined region for a number of years with increasing prevalence. Interestingly, pseudorabies had been eradicated in domestic pigs in this area in 1985. Four PrV could be isolated from epidemic areas. Molecular biological analysis using restriction length polymorphism showed considerable differences to PrV-strains occurring in domestic animals. Thus, the infections in the wild boar population appear to be endemic and persist completely separately and without affecting the domestic pig population (Müller *et al.*, 1996).

Clinical signs include a brief course of hyperexcitability, ataxia, coma, and progressive paralysis. The disease is relatively mild in adult animals, causing heavy mortality only in the young (Wallach & Boever, 1983). Aujeszky's disease has not been reported as causing natural disease of free-living deer (Alexander & Buxton, 1994).

Equine herpesvirus infections

The horse is natural host to five herpesviruses of which three are classified as alphaherpesvirinae and two as gammaherpesvirinae (Roizman, 1996). The three equine alphaherpesviruses so far known are: equine herpesvirus type 1 (equine abortion virus, EHV-1), equine herpesvirus type 3 (equine coital exanthema virus, EHV-3) and equine herpesvirus type 4 (rhinopneumonitis virus, EHV-4). Equine herpesvirus type 2 (EHV-2) and the related equine herpesvirus type 5 (EHV-5) are gammaherpesviruses (Telford *et al.*, 1993). Until now, no literature is available about the occurrence of antibodies against EHV in reintroduced Przewalski's wild horse or other free-ranging equids in the holarctic region.

Conclusions

For some diseases (e.g. BVD, EHD, BT, CE and alphaherpesvirus infections) serological studies as well as virus isolation in wildlife have been performed quite intensively in different countries. For other viral diseases antibodies could be detected only in a few cases but virus isolation or DNA detection was not possible in free-living ungulates (e.g. MCF). For some agents like EHV even antibody detection was not possible. However, the mode of transmission for most diseases remains unclear which may reflect a lack of surveillance of viral diseases in wild ungulates.

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