

Evidence of parapox-, alphaherpes- and pestivirus infections in carcasses of semi-domesticated reindeer (*Rangifer tarandus tarandus*) from Finnmark, Norway

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Abstract: During March to May 2000, 48 carcasses of semi-domesticated reindeer (*Rangifer tarandus tarandus*) were collected on winter pastures and calving grounds from two herds in western Finnmark and two herds in eastern Finnmark, northern Norway. The animals were autopsied and blood and tissue samples were collected for serology (alphaherpes- and pestivirus; virus neutralization test) and polymerase chain reaction (PCR; parapoxvirus; *B2L* gene) investigations. Autopsy revealed that 39 of 48 animals (81%) had died of emaciation. Parapoxvirus-specific DNA was detected in samples from 6 of 48 animals (12.5%; liver, parotid salivary gland and/or pulmonary lymph nodes). A DNA sequence of 376 base pairs from a PCR amplicon obtained from a liver sample from one animal showed 98-99% identity with orf virus strain Orf-11 and reindeer parapoxvirus isolates from Norway and Finland (1992 and 1994), 92-93% similarity with pseudocowpoxvirus and 87% similarity with bovine papular stomatitis virus. Alphaherpes- and pestivirus antibodies were detected in 10% and 33% of the animals, respectively. These results indicate that parapoxvirus, presumably orf-virus, is present among reindeer also in Finnmark, although contagious ecthyma has never been reported in reindeer in this important reindeer herding area. Furthermore, they show that herpes- and pestiviruses are still endemic in reindeer herds in Finnmark. The nature of these viruses and their impact on reindeer health and reproduction and reindeer herding economy should be further addressed, as well as the possibility that these viruses may be transferred between reindeer and domestic animals in this region.

Key words: infectious disease, virology, virus infection, wildlife.

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Introduction

Finnmark County, northern Norway, constitutes the most important reindeer herding area in Norway, with about 165 000 semi-domesticated reindeer (*Rangifer tarandus tarandus*) and approximately 760 man-labour year in the period 2003-2004 (Heatta, 2005). The reindeer pastures are divided in districts and animals from different districts may have limited contact. The reindeer herding is semi-nomadic, moving between winter pastures located mainly inland and summer pastures, usually in coastal ar-

reas. During the snow-free period and especially in coastal areas, reindeer often graze farmed fields and may also encounter cattle, sheep and goats in the field, thus facilitating interspecies transmission of infectious agents. Mortality of semi-domesticated reindeer has many causes, such as predators, restricted food availability, diseases and accidents. In Finnmark, the annual losses of animals have been around 5-18% and 11-49% for adults and calves, respectively, in the period from 1999-2004 (Heatta

2005). Predators, such as golden eagle (*Aquila chrysaetos*), wolverine (*Gulo gulo*), lynx (*Lynx lynx*) and brown bear (*Ursus arctos*) may cause a substantial part of these losses (Andersen, 2004). Some of the reported mortality is caused by hitherto unknown factors. Persistent virus infections, such as parapox-, alphaherpes- and pestivirus infections may have impact on reindeer reproduction and survival.

Parapoxviruses (family *Poxviridae*) cause contagious ecthyma in sheep, goats and other species, including reindeer. A severe outbreak of contagious ecthyma occurred in reindeer in Norway in spring 2000 (Tryland *et al.*, 2001). Outbreaks have also occurred in Finland since the winter 1992-93 (Oksanen & Norberg, 1994; Tikkanen *et al.*, 2004), and reindeer with clinical symptoms similar to contagious ecthyma have previously been observed in Sweden (Norkvist *et al.*, 1973). It has been shown that the causative virus of contagious ecthyma in reindeer in Norway is orf-virus, a virus species causing similar clinical disease in sheep and goats (Klein & Tryland, 2005). In Finland, virus isolates from early disease outbreaks in reindeer have been classified as orf-virus, whereas isolates from recent contagious ecthyma cases have been characterised as pseudocowpoxvirus, a closely related parapoxvirus (Tikkanen *et al.*, 2004). Contagious ecthyma in reindeer have not yet been reported from Finnmark, but the disease is present among sheep in this region.

Herpesviruses belonging to the subfamily *Alphaherpesvirinae* may cause diarrhoea and mucosal lesions in the mouth, nose and the upper respiratory tract in cattle and other ruminants, as well as abortions (Straub, 1990; Murphy *et al.*, 1999). Herpesvirus has been isolated from reindeer in Finland

(Ek-Kommonen *et al.*, 1986) and Sweden (Rockborn *et al.*, 1990). A previous serological investigation of reindeer in Finnmark revealed neutralizing herpesvirus antibodies in 10-46% of the animals from all the 7 investigated herding areas (Stuen *et al.*, 1993). A recent screening of wild reindeer in southern Norway revealed anti-herpesvirus antibodies in 28.5% of the animals ($n=831$; Lillehaug *et al.*, 2003). Alphaherpesvirus are also associated with ocular diseases, such as conjunctivitis and corneal lesions, and infection of the upper respiratory tract of deer (Inglis *et al.*, 1983).

It has been shown that reindeer is susceptible to experimental infection with bovine viral diarrhoea virus (BVDV; family *Flaviviridae*, genus *Pestivirus*), developing clinical symptoms consistent with BVD in cattle (Morton *et al.*, 1990). A pestivirus was recently isolated from a captive reindeer in a zoo (Pestivirus reindeer-1; Duisburg Zoo, Germany), and characterization of the isolate has indicated that the virus is closer related to a pestivirus in sheep (Border disease virus; BDV) than to BVDV (Avalos-Ramirez *et al.*, 2001; Becher *et al.*, 2003). In a previous serological investigation of reindeer in Finnmark, neutralizing pestivirus antibodies were detected in animals from 6 of 7 investigated herding districts, with a total seroprevalence of 17% (Stuen *et al.*, 1993). Pestivirus antibodies have also been detected in reindeer in Finland (58%; Neuvonen *et al.*, 1983) and Sweden (6%; Reh binder *et al.*, 1992), and in caribou (*R. tarandus*) in Canada (60%; Elazhary *et al.*, 1979) and Alaska (3%; Zarnke, 1983).

To assess factors that may influence mortality in reindeer in Finnmark, 48 carcasses were collected from winter pastures and calving grounds in 2000. The present study reports investigations on

Table 1. Age and sex distribution of 48 reindeer carcasses found on winter pastures and calving grounds in western (herds A and B) and eastern Finnmark (herds C and D). The animals were autopsied and investigated for the presence of parapoxvirus-specific DNA (PCR) and antibodies against herpes- and pestivirus (serology).

Age group ^a	Male	Female	Total	Herd
Foetus	1	0	1	A
Newborn calves (< 2 weeks old)	12	3	15	B, C, D
Yearlings (9-12 months old)	12	7	19	A, B, D
Young animals (21-24 months old)	0	4	4	A, D
Adults (> 33 months)	0	9	9	A, B
Total	25	23	48	

^a Age groups was determined on the basis of the set of teeth (Skjenneberg & Slagsvold, 1968).

samples from the reindeer carcasses with regard to presence of parapoxvirus-specific DNA in tissues (polymerase chain reaction; PCR) and antibodies against herpes- and pestivirus in blood (serology).

Material and methods

Reindeer carcasses ($n=48$; Table 1) were collected on winter pastures and calving grounds representing four herding districts in Finnmark, Norway, from March to end of May 2000. Two of the districts were located in western Finnmark (A: $n=19$, B: $n=7$), whereas two were located in eastern Finnmark (C: $n=6$, D: $n=16$) (Table 1). Some of the carcasses were collected frozen in the field, whereas those collected in the spring were frozen before transport for autopsy. The degree of cadaverosis was evaluated as none ($n=1$), moderate ($n=26$), moderately severe ($n=14$) and severe ($n=2$), whereas no information exists for 5 animals, including the foetus. Age groups (newborn; < 2 weeks, yearling; 9-12 months, young; 21-24 months, and adult; > 33 months) was determined by assessing the set of teeth (Skjenneberg & Slagsvold, 1968). The cause of death was determined through autopsy. The diagnosis emaciation was based on three major macroscopic changes; no visible fat in the abdomen, serous atrophy of pericardial fat, and semi-transparent gelatinous bone marrow in femur (Josefsen *et al.*, unpubl.). During autopsy, a total of 281 tissue samples were collected from tonsils ($n=47$), parotis salivary gland ($n=46$), lung ($n=48$), pulmonary lymph nodes ($n=36$), liver ($n=48$), and spleen ($n=48$), as well as from lesions in the mucosa of the mouth ($n=3$) and abomasum ($n=5$). DNA was extracted from the tissues by a QIAamp tissue kit (QUIAGEN, GmbH, Düsseldorf, Germany). PCR primers (PPP-1 and PPP-4) from the gene encoding the B2L envelope antigen of orf virus (strain NZ-2) were used (Inoshima *et al.*, 2000). The PCR was performed with an annealing temperature of 62 °C (Tryland *et al.*, 2001) in a Gene Amp PCR system 9700 (Perkin Elmer Corp., Norwalk, Connecticut, USA). Amplicons were analysed by gel electrophoresis (Gibco BRL Horizontal Gel Electrophoresis, Horizon® 1114; Life Technologies™, Paisly, Scotland) in 2% agarose gel (Ultra pure agarose gel; Life Technologies) using TAE buffer (0.04 M Tris-acetate, 1.0 mM ethylenediamine tetra-acetic acid), with ethidium bromide for staining of DNA. PCR

amplicons were prepared for sequencing by removing primers and dNTP using ExoSapIT reagent (Amersham Pharmacia; Uppsala, Sweden; 1 µl/5µl PCR product, 45 min at 37 °C, followed by 20 min at 80 °C for enzyme inactivation). Cycle sequencing was conducted in both directions using Big Dye 3.1 reagents (ABI BigDye® Terminator Version 3.1, Applied Biosystems, Oslo, Norway). Two µl 125 M EDTA, 2 µl 3 M sodium acetate, and 50 µl ethanol was added to the 20 µl sequencing product. Electrophoresis of the cycle sequencing extension products were conducted in an ABI Prism® 377 DNA Analyzer (Applied Biosystems). Raw sequence data were edited by Chromas software (Version 2.3; Technelysium Pty Ltd., Tewantin, Qld., Australia) and BioEdit Sequence Alignment Editor (Version 7.0.4; Tom Hall, Department of Microbiology, North Carolina State University, North Carolina, USA; <http://www.mbio.ncsu.edu/BioEdit/bioedit.html>). The reindeer parapoxvirus DNA sequence obtained in this study has been submitted to the GenBank nucleotide sequence database (accession number DQ028479; Reindeer parapoxvirus putative virion envelope antigen gene).

Blood samples were collected during autopsy from large vessels and centrifuged (1500 g for 15 min) and the supernatant were frozen at - 20 °C. Blood supernatant from 43 animals were investigated for pestivirus specific antibodies by virus neutralization test (VNT) against BVDV (Løken *et al.*, 1982). Samples from 41 animals were tested for alphaherpesvirus specific antibodies by VNT against reindeer herpesvirus 1 (RanHV-1; Rimstad *et al.*, 1992).

Results

The foetus was aborted due to unknown cause, one animal was hit by car, two animals were euthanized due to trauma and emaciation, six animals were killed by predators (lynx, wolverine and eagle), whereas 39 animals (81%) had died from emaciation and had no other pathological signs. The PCR and serology results are presented in Table 2. Parapoxvirus-specific DNA was detected in liver, parotid salivary gland and/or pulmonary lymph nodes from 6 of 48 animals (13%); one newborn calf, three yearlings, and two adult reindeer. The DNA sequence of 376 base pairs of an amplicon obtained from a liver sample from one animal (Table

Table 2. Semi-domesticated reindeer from herds A (western Finnmark) and C and D (eastern Finnmark), Norway, with evidence of parapoxvirus (virus-specific DNA in tissue samples; PCR), alphaherpesvirus and pestivirus infection (antibodies).

Animal number	Herd	Age ¹	Cause of death	Parapoxvirus DNA in tissue	Antiherpesvirus antibodies	Pestivirus antibodies
1	A	Young	Emaciation			+
4	A	Yearling	Emaciation	Liver		
6	A	Yearling	Emaciation			+
7	A	Yearling	Emaciation		n.i.	+
8	A	Adult	Emaciation			+
11	A	Adult	Emaciation	Gl. parotis ³		n.i.
12	A	Yearling	Emaciation	Liver		+
16	A	Adult	Emaciation	Lung ln. ⁴		+
17	A	Adult	Emaciation			+
18	A	Young	Emaciation			+
30	C	Newborn	Low body weight/-fat ²	Gl. parotis ³	+	+
32	C	Newborn	Lynx		+	
37	D	Young	Car strike			+
41	D	Newborn	Low body weight/-fat ²		+	+
42	D	Newborn	Low body weight/-fat ²		+	
43	D	Newborn	Low body weight/-fat ²			+
45	D	Yearling	Lynx	Gl. parotis ³ Lung ln. ⁴		
46	D	Yearling	Died in transport			+
47	D	Yearling	Emaciation			+

¹ Age: Newborn (< 2 weeks), yearling (9-12 months), young (21-24 months), adult (> 33 months).

² Weak-born; presumably dead of hypothermia.

³ Glandula parotis (salivary gland).

⁴ Lung lymph node.

n.i.: not investigated.

2, animal 4) showed a high degree of identity with orf virus strain Orf-11 as well as with other orf-virus isolates and other parapoxviruses (Table 3). Parapoxvirus specific DNA was detected in animals from herds A, C and D. Four of the 41 investigated animals (10%) had anti-alphaherpesvirus antibodies. All were 2-3 days old calves, representing herds C and D (eastern Finnmark), whereas no animals from herds A and B (western Finnmark) had alphaherpesvirus antibodies. Among the 15 newborn calves tested, this constitutes a seroprevalence of 27%. One of the calves with anti-alphaherpesvirus antibodies was killed by lynx, whereas the three others had low body weight and little or no body fat and were regarded as weekly born. Fourteen of 43 animals (33%) had neutralizing antibodies against

pestivirus; 3 newborn calves, 5 yearlings, 3 young animals (21-24 months old) and 3 adults, representing herds A, C and D.

Discussion

Most of the animals (81%) had died of emaciation, and although lesions in the oral and abomasal mucosa were detected in three and five individuals, respectively, we had no indications of virus infections as the primary cause of death. Outbreaks of contagious ecthyma in semi-domesticated reindeer in Finland and Norway have been severe with relatively high morbidity and mortality (Oksanen & Norberg, 1994; Tryland *et al.*, 2001). Since this disease never has been reported in reindeer in Finnmark, it has been concluded that reindeer in this region

Table 3. Genetic similarity between the DNA sequence obtained from the PCR product (376 base pairs; B2L-gene) from a liver sample from a reindeer (*Rangifer tarandus tarandus*; animal 4) and corresponding DNA sequences from other parapoxvirus isolates (GenBank).

Host species	Country of origin	Virus species (year of isolation)	GenBank accession number	Similarity (%)
Sheep	Scotland	Orfvirus (Orf-11; ref. strain)	AY605958	99
Reindeer	Norway	Orfvirus (1999)	AY605963	99
Reindeer	Finland	Orfvirus (1992)	AY453659	99
Reindeer	Finland	Orfvirus (1994)	AY453661	99
Reindeer	Norway	Orfvirus (2000)	AY605964	98
Cattle	Norway	Pseudocowpox (1979)	AY605960	92
Cattle	Norway	Pseudocowpox (1992)	AY605961	92
Cattle	Norway	Pseudocowpox (1983)	AY605970	93
Musk ox and Shetland sheep	USA	Orfvirus (2001)	AY424973	87

have not yet been exposed to parapoxvirus. However, due to the findings of parapoxvirus-specific DNA in tissues from the carcasses investigated in this study, it is now clear that parapoxvirus is present among reindeer also in this region, and that the virus may be present in various tissues, and not only the skin and mucosa were clinical lesions usually are present. Furthermore, these findings suggest asymptomatic parapoxvirus infections in reindeer, a phenomenon recently reported in cows in Japan (Sentsui *et al.*, 1999). This implies that reindeer individuals may be persistently infected and that environmental stress factors, such as relocations, corraling, handling, transport, and restricted food quality and availability may influence whether a clinical outbreak occur or not. This may have implications for the management of reindeer, by reducing the amount of stress factors to a minimum in efforts to avoid outbreaks of contagious ecthyma. Based on the DNA sequences amplified from the B2L gene and the demonstrated similarity with published parapoxvirus gene sequences, we assume that the virus asymptotically present in reindeer in Finnmark is orf-virus, although isolation and a more thoroughly characterization of the virus is needed. Since no reindeer-specific parapoxvirus has been isolated, a transmission of virus between reindeer and sheep and goats seems likely, which should be kept in mind when sharing corrals, transports and other equipment between different animal species and herds.

Of the 30 reindeer <12 months tested, anti-al-

phaherpesvirus antibodies were found in 4 animals (13%), all being 2-3 days old calves representing herds C and D, whereas none of the 13 reindeer >12 months tested had anti-alphaherpesvirus antibodies. It is not certain whether the antibodies were produced by the calves caused by an intra-uterine infection, or whether they had been passively transferred from the mother via colostrum. In a previous investigation on anti-alphaherpesvirus antibodies in slaughtered reindeer from Finnmark, a seroprevalence of 63% were found in animals <12 months of age ($n=225$; Stuen *et al.*, 1993). The discrepancy between that study and our results for this age group may represent changes over time, but might as well reflect the limited number of animals tested or differences between the herds being represented. Herpesviruses may establish itself as life-long latent infections that may be reactivated and cause abortions and weak offspring in several animal species (Straub, 1990; Engels & Ackermann, 1996; Murphy *et al.*, 1999). Three of the four calves with anti-alphaherpesvirus antibodies were weak-borne, which may have connection to a herpesvirus infection. They were borne during periods with cold weather (2-10 °C below zero in combination with wind and snow) and it is likely that hypothermia has contributed to their death. In reindeer, alphaherpesvirus infection may cause mucosal lesions in the upper alimentary tract and nose, and it has been suggested that such lesions may be the port of entry of bacteria such as *Fusobacterium necrophorum*, causing severe necrobacillosis in rein-

deer (Rockborn *et al.*, 1990). It was recently demonstrated that a herpesvirus isolated from a Canadian elk (*Cervus elaphus nelsoni*) established latency in cattle upon experimental infection, showing a possible link between wildlife and livestock (Deregt *et al.*, 2005). The overall impact of such infections on the reproduction (abortions and calf mortality) and survival of reindeer is unknown.

Anti-pestivirus antibodies were found in 14 individuals, representing herds A, C and D. Animals <12 months had a seroprevalence of 27%, which for this age group were higher than previously found in slaughtered reindeer from Finnmark (6%; Stuen *et al.*, 1993). This may indicate that a higher percentage of animals that die on winterpasture is exposed to pestivirus as compared to slaughtered animals, but the number of animals tested are too restricted to draw any firm conclusion. Following a national eradication campaign, BVDV is about to vanish from the Norwegian cattle population (Nyberg, 2002). Most BVDV infections in adult cattle are sub-clinical, although some animals show mild diarrhea, nasal and ocular discharge, erosive stomatitis and a drop in milk yield (Murphy *et al.*, 1999). The infection may also cause abortions, retardation of foetal growth ("weak calf syndrome"), congenital defects, and life-long infected and virus shedding calves that produce no antibodies against the virus (Murphy *et al.*, 1999). Such persistently infected calves may develop a more severe syndrome, mucosal disease, which may cause diarrhea, dehydration and emaciation (Murphy *et al.*, 1999). No pestivirus has been isolated from wild or semi-domesticated reindeer, and whether the virus promoting the production of anti-pestivirus antibodies in reindeer is BVDV or a different and serologically cross-reacting pestivirus is unknown (Stuen *et al.*, 1993; Lillehaug *et al.*, 2003). It is also unknown whether the endemic pestivirus in reindeer cause clinical disease.

Some of the animals had antibodies to both alphaherpesvirus- and pestivirus, or to one of the viruses in combination with the presence of parapoxvirus DNA in tissue samples (Table 2). All the three viruses may establish persistent infections and herpesviruses are generally known to establish life long latent infections. In addition, a pestivirus infection, like BVDV, may cause immunosuppression (Brackenbury *et al.*, 2003) that may have effect

on reactivation of latent infections as well as the establishment of new infections. An important stress factor for reindeer during winter is lack of food and emaciation, which was found as the direct cause of death for more than 80% of the investigated animals in this study (Josefsen *et al.*, unpubl. data). Restricted food availability and food quality may be a stress factor present over prolonged time periods during winter, which may influence the susceptibility to and the outcome of virus infections.

In contrast to the situation for parapoxvirus, which seems to be transferred from domestic animals (sheep and goats) to reindeer, there are several factors that suggest that reindeer are hosting their own alphaherpes- and pestivirus variants. Herpesvirus has been isolated from reindeer in both Finland (Ek-Kommonen *et al.*, 1986) and Sweden (Rockborn *et al.*, 1990) and due to characterisations by means of serology (Lyaku *et al.*, 1992), genomic restriction fragment length polymorphism (RFLP; Rimstad *et al.*, 1993) and PCR and RFLP on amplicons from the glycoprotein-B and -D genes (Lyaku *et al.*, 1996; Ros & Belák, 1999), it seems that reindeer herpesvirus (RanHV-1) is a virus variant closely related to herpesvirus in deer (cervid herpesvirus; CerHV-1) and goats (caprine herpesvirus; CapHV-1). Also the fact that the disease BVD in cattle are about to be eradicated and that the syndromes caused by herpesvirus in cattle (infectious bovine rhinotracheitis and infectious pustular vulvovaginitis and balanoposthitis) have not been registered in cattle in Norway since 1993 (Nyberg *et al.*, 2003), are strong indications of the existence of distinct herpes- and pestivirus species in reindeer. In addition, high seroprevalences of antibodies, both against pesti- and herpesviruses, found in reindeer populations in this and previous studies (Stuen *et al.*, 1993; Lillehaug *et al.*, 2003) are supporting the theory that specific reindeer viruses exists. Furthermore, herpes- and pestivirus antibodies have been found in wild reindeer in both Norway (Lillehaug *et al.*, 2003) and Greenland (Anonymous, 1999). These are two reindeer populations with very limited contact with cattle, which also supports this view. Also other wild cervid populations in Norway and other European countries are hosting alpha-herpesviruses and pestiviruses (Thiry *et al.*, 1988; Frölich & Hoffman, 1995; Anonymous, 1999; Frölich, 2000; Lillehaug *et al.*, 2003). Pestivirus isolates from a zoo-reindeer,

a giraffe and a bison are tentative new species of the genus (Avalos-Ramirez *et al.*, 2001; Becher *et al.*, 2003), and recent characterisation of pestivirus isolates from roe deer from Germany (Fischer *et al.*, 1998), Pyrenean chamois (*Rupicapra pyrenaica pyrenaica*) (Arnal *et al.*, 2004), and pronghorn antelope (*Antilocapra americana*) from USA (Vilcek *et al.*, 2005) indicates that these viruses do not fit with the pestivirus species identified so far, and that several distinct pestiviruses might exist among wildlife species.

Whether the pesti- and alphaherpes viruses in semi-domesticated reindeer may be transferred to and cause disease in livestock remains unknown. If so, reindeer may represent an important virus reservoir and it may be difficult to eradicate such disease agents from livestock. If these viruses are non-pathogenic or only cause mild disease problems for domestic animals and/or wildlife species, they may still be important factors hampering serological diagnostic tests for livestock by causing false positive results due to serological crossreactions (Lyaku *et al.*, 1992; Moennig & Plagemann, 1992).

We do not know if parapox-, alphaherpes- and pestivirus infections cause clinical disease and economical losses for the reindeer husbandry in Finnmark. However, this investigation indicates that these viruses are present and endemic in reindeer of this region. The nature of these viruses and their impact on reindeer health and reproduction and reindeer herding economy should therefore be further addressed, as well as the possibility that these viruses may be transferred between reindeer and domestic animals in this region.

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References

- Andersen, F. G. (ed.). 2004. *Totalregnskap for reindriftsnæringen for reindriftsåret 1. april 2003-31. mars 2004*. Reindriftsforvaltningen, Alta, Norway.
- Anonymous. 1999. *Report on the Animal Health Situation in Greenland 1999* (22. September 2005: <http://www.foedevarestyrelsen.dk/FDir/Publications/2000640/kapl.asp>).
- Arnal, M. C., Fernández-de-Luaco, D., Riba, L., Maley, M., Gilray, J., Willoughby, K., Vilcek, S. & Nettleton, P. F. 2004. A novel pestivirus associated with deaths in Pyrenean chamois (*Rupicapra pyrenaica pyrenaica*). – *Journal of General Virology* 85: 3653-3657.
- Avalos-Ramirez, R., Orlich, M., Thiel, H-J. & Becher, P. 2001. Evidence for the presence of two novel pestivirus species. – *Virology* 286: 456-465.
- Becher, P., Avalos Ramirez, R., Orlich, M., Cedillo Rosales, S., König, M., Schweizer, M., Stalder, H., Schirrmeier, H. & Thiel, H. J. 2003. Genetic and antigenic characterization of novel pestivirus genotypes: implications for classification. – *Virology* 311: 96-104.
- Brackenbury, L.S., Carr, B.V., & Charleston, B. 2003. Aspects of the innate and adaptive immune responses to acute infections with BVDV. – *Veterinary Microbiology* 96: 337-44.
- Deregt, D., Tessaro, S. V. & Gilbert, S. A. 2005. Serological evidence of latency in cattle experimentally infected with elk herpesvirus. – *Veterinary Record* 156: 610-611.
- Ek-Kommonen, C., Pelkonen, S. & Nettleton, P. F. 1986. Isolation of a herpesvirus serologically related to bovine herpesvirus 1 from a reindeer (*Rangifer tarandus*). – *Acta Veterinaria Scandinavica* 27: 299-301.
- Elazhary, M. A. S. Y., Roy, R. S. & Fréchette, J. L. 1979. Serological evidence of IBR and BVD infection in caribou (*Rangifer tarandus*). – *Veterinary Record* 105: 336.
- Engels, M. & Ackermann, M. 1996. Pathogenesis of ruminant herpesvirus infections. – *Veterinary Microbiology* 53: 3-15.
- Fischer, S., Weiland, E. & Frölich K. 1998. Characterization of a bovine viral diarrhoea virus isolated from roe deer in Germany. – *Journal of Wildlife Diseases* 34: 47-55.
- Frölich, K. 2000. Viral diseases of northern ungulates. – *Rangifer* 20: 83-97.
- Frölich, K. & Hofmann, M. 1995. Isolation of bovine viral diarrhoea virus-like pestiviruses from roe deer (*Capreolus capreolus*). – *Journal of Wildlife Diseases* 31: 243-246.
- Heatta, J. I. (ed.). 2005. *Ressursregnskap for reindriftsnæringen for reindriftsåret 1. april 2003-31. mars 2004*. Reindriftsforvaltningen, Alta, Norway.
- Inglis, D. M., Bowie, J. M., Allan, M. J. & Nettleton, P. F. 1983. Ocular disease in red deer calves associated

- with a herpesvirus infection. – *Veterinary Record* 113: 182-183.
- Inoshima, Y., Morooka, A. & Sentsui, H. 2000. Detection and diagnosis of parapoxvirus by the polymerase chain reaction. – *Journal of Virological Methods* 84: 201-208.
- Klein, J. & Tryland, M. 2005. Characterisation of parapoxviruses isolated from Norwegian semi-domesticated reindeer (*Rangifer tarandus tarandus*). – *Virology Journal* 2 (1): 79 [http://www.virologyj.com/content/2/1/79].
- Lillehaug, A., Vikøren, T., Larsen, I. L., Åkerstedt, J., Tharaldsen, J. & Handeland, K. 2003. Antibodies to ruminant alpha-herpesviruses and pestiviruses in Norwegian cervids. – *Journal of Wildlife Diseases* 39: 779-786.
- Lyaku, J. R., Sinclair, J. A., Nettleton, P. F. & Marsden H. S. 1992. Production and characterization of monoclonal antibodies to cervine herpesvirus-1. – *Veterinary Microbiology* 32: 229-39.
- Lyaku, J. R. S., Vilcek, S., Nettleton, P. F. & Marsden, H. S. 1996. The distinction of serologically related ruminant alphaherpesviruses by the polymerase chain reaction (PCR) and restriction endonuclease analysis. – *Veterinary Microbiology* 48: 135-142.
- Løken, T., Hyllseth, B., & Larsen, H. J. 1982. Border disease in Norway. Serological examination of affected sheep flocks. – *Acta Veterinaria Scandinavica* 23: 46-52.
- Moennig, V. & Plagemann, P. G. 1992. The pestiviruses. – *Advances in Virus Research* 41: 53-98.
- Morton, J. K., Evermann, J. F. & Dieterich, R. A. 1990. Experimental infection of reindeer with bovine viral diarrhoea virus. – *Rangifer* 10: 75-77.
- Murphy, F.A., Gibbs, E. P., Horzinek, M. C. & Studdert, M. J. 1999. Herpesviridae – In: *Veterinary virology*, 3rd ed. Academic Press, London, UK, pp. 301-325.
- Neuvonen, E., Veijalainen, P., Retulainen, S. & Ek-Kommonen, C. 1983. Onko poroilla virustauteja? – *Poromies* 6: 16-17.
- Nordkvist, M. 1973. Munvårtsjuka - en ny rensjukdom? – *Rennäringsnytt* 8-9: 6-8.
- Nyberg, O. 2002. 2002 – siste året med BVD? Husdyrfor-søksmøtet, pp. 77-80.
- Nyberg, O., Tharaldsen, J. & Heier, B. T. 2003. *The surveillance and control programme for infectious bovine rhinotracheitis (IBR) and infectious pustular vulvovaginitis (IPV) in Norway. Annual Report 2003*. National Veterinary Institute, Norway, pp. 53-56. (27. November 2005: http://1.1.1.1/477469076/455192136T051122110145.txt.binXMysM0dapplication/pdfXsysM0dhttp://www.vetinst.no/Arkiv/Pdf-filer/NOK-2003_2/09-2003%20IBR-IPV.pdf).
- Oksanen, A. & Norberg, H. 1994. Smittsom munnskurv. – *Reindriftnytt* 3-4: 13-17.
- Rehbinder, C., Bělak, S. & Nordkvist, M. 1992. A serological retrospective study in reindeer on five different viruses. – *Rangifer* 12: 191-195.
- Rimstad, E., Krona, R. & Hyllseth, B. 1992. Comparison of herpesviruses isolated from reindeer, goats, and cattle by restriction endonuclease analysis. – *Archives of Virology* 123: 389-97.
- Rockborn, G., Rehbinder, C., Klingeborn, B., Lefler, M., Klintevall, K., Nikkilä, T., Landén, A. & Nordkvist, M. 1990. The demonstration of a herpesvirus, related to bovine herpesvirus 1, in reindeer with ulcerative and necrotizing lesions of the upper alimentary tract and nose. – *Rangifer Special Issue* 3: 373-384.
- Ros, C. & Belák, S. 1999. Studies of genetic relationships between bovine, caprine, cervine and rangiferine alphaherpesviruses and improved molecular methods for virus detection and identification. – *Journal of Clinical Microbiology* 37: 1247-1253.
- Sentsui, H., Murakami, K., Inoshima, Y., Shibahara, T. & Yokomizo, Y. 1999. Isolation of a parapoxvirus from a cow treated with interferon- γ . – *Veterinary Microbiology* 70, 143-152.
- Skjenneberg, S. & Slagsvold, L. 1968. Reindriften og dens naturgrunnlag. Universitetsforlaget Oslo, Norway, pp. 1-332.
- Straub, O. C. 1990. Infectious bovine rhinotracheitis virus – In: Dinter, Z. & Morein, B. (eds.). *Virus infections of ruminants*. Elsevier Science Publishers, Amsterdam, The Netherlands, pp. 71-108.
- Stuen, S., Krogsrud, J., Hyllseth, B. & Tyler, N. J. C. 1993. Serosurvey of three virus infections in reindeer in northern Norway and Svalbard. – *Rangifer* 13: 215-219.
- Thiry, E., Vercouter, M., Dubuisson, J., Barrat, J., Sepulchre, C., Gerardy, C., Meersschaert, C., Collin, B., Blancou, J. & Pastoret, P. P. 1988. Serological survey of herpesvirus infections in wild ruminants of France and Belgium. – *Journal of Wildlife Diseases* 24: 268-273.
- Tikkanen MK, McInnes CJ, Mercer AA, Buttner M, Tuimala J, Hirvela-Koski V, Neuvonen E, Huovilainen A. 2004. Recent isolates of parapoxvirus of Finnish reindeer (*Rangifer tarandus tarandus*) are closely related to bovine pseudocowpox virus. – *Journal of General Virology* 85 (Pt 6):1413-1418.
- Tryland, M., Josefson, T. D., Oksanen, A. & Aschfalk, A. 2001. Parapoxvirus infection in Norwegian semi-domesticated reindeer (*Rangifer tarandus tarandus*). – *Veterinary Record* 149: 394-395.
- Vilcek, S., Ridpath, J.F., Van Campen, H., Cavender, J.L. & Warg, J. 2005. Characterization of a novel pestivirus originating from a pronghorn antelope. – *Virus Research* 108: 187-193.

Zarnke, R. L. 1983. Serologic survey for selected microbial pathogens in Alaskan wildlife. – *Journal of Wildlife Diseases* 19: 324-329.

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Abstract in Norwegian / Sammendrag:

I løpet av perioden mars-mai 2000 ble 48 reinsdyrkadavre (*Rangifer tarandus tarandus*) samlet inn fra vinterbeiter og kalvingsområder fra to flokker i Vest-Finnmark og to i Øst-Finnmark, Norge. Dyrene ble obdusert, og blod og vevsprøver ble samlet for påvisning av antistoffer mot alfaherpes- og pestivirus i blod (serologi) og tilstedeværelse av parapoxvirus-DNA i vev (Polymerase kjedereaksjon, PCR; parapoxvirus B2L genot). Obduksjonen viste at 39 av de 48 dyrene (81%) hadde dødd av avmagring. Parapoxvirus-spesifikt DNA ble funnet i prøver av lever, spyttkjertel (Gl. parotis) og/eller lungelymfeknuter fra 6 av de 48 dyrene (12,5%). En DNA sekvens på 376 basepar fra PCR-opppermeringsproduktet fra en leverprøve hadde 98-99% likhet med orf-virus (Orf-11) og parapoxvirus isolert fra reinsdyr i Norge og Finland (1992 og 1994), 92-93% likhet med pseudocowpoxvirus og 87% likhet med bovint papulær stomatittvirus, hvorav de to siste parapoxvirusartene er assosiert med storfe. Disse resultatene viser at også reinsdyr i Finnmark er infisert av parapoxvirus, til tross for at sykdommen munnskurv ikke ennå er rapportert hos rein i dette fylket. Alfaherpes- og pestivirus antistoffer ble funnet hos henholdsvis 10% og 33% av dyrene. Dette er i samsvar med tidligere funn på slaktedyr, og viser at disse virusinfeksjonene er endemiske hos rein i Finnmark. Det er viktig å avklare hvilken rolle disse virusinfeksjonene spiller for reinsdyrenes helse og næringens økonomi. Videre bør det avklares i hvilken grad disse virusstypene er i stand til å smitte mellom rein og husdyr.

