

Malignant Catarrhal Fever: An Emerging Disease in the African Buffalo (*Syncerus caffer*)

S. Pfitzer¹, R. Last², I. Espie³ and M. van Vuuren⁴

¹ Magudu Veterinary Consulting Room, Pongola, South Africa

² Vetdiagnostix – Veterinary Pathology Services, Cascades, South Africa

³ National Zoological Gardens, Pretoria, South Africa

⁴ Department of Veterinary Tropical Diseases, Faculty of Veterinary Science, University of Pretoria, Pretoria, South Africa

Correspondence:

M. van Vuuren. Department of Veterinary Tropical Diseases, Faculty of Veterinary Science, University of Pretoria, P Bag X04, Onderstepoort 0110, Pretoria, South Africa. Tel.: +27 12 5298329; Fax: +27 12 5298312; E-mail: Moritz.vanvuuren@up.ac.za

Summary

Within the tribe Bovini in the subfamily Bovinae, the water buffalo (*Bubalus Bubalis*), American bison (*Bison bison*), European bison (*Bubalus bonasus*) and yak (*Bos grunniens*) are recognized as species highly susceptible to malignant catarrhal fever (MCF). In contrast, the lack of reports describing clinical MCF in the African buffalo (*Syncerus caffer*) whether free ranging or captive has led to a perception that African buffaloes are resistant to MCF. During the last decade, several cases of MCF in African buffaloes were confirmed in South Africa and experience with seven of these cases is described in this report. Detection of viral nucleic acid in blood or tissues was successful in six African buffaloes that suffered from clinical signs compatible with MCF. Four were positive for infection with ovine herpesvirus type 2 (the causative virus of sheep-associated MCF), and two were positive for alcelaphine herpesvirus type 1 (causative virus of wildebeest-associated MCF). Histopathological examination of tissue samples from all the animals yielded typical lesions that were consistent with those described for MCF in domestic cattle. Developments in the management of African buffaloes translocated from their traditional habitats have likely contributed to the identification of another susceptible host in the subfamily Bovinae.

Keywords:

malignant catarrhal fever; African buffalo;
ovine herpesvirus type 2; alcelaphine
herpesvirus type 1

Introduction

Malignant catarrhal fever is a lymphoproliferative disease with a high mortality in susceptible species once typical clinical signs have appeared. Since the first isolation of alcelaphine herpesvirus type 1 (AlHV-1), one of the causal herpesviruses of malignant catarrhal fever (MCF) from blue wildebeest (*Connochaetus taurinus*) (Plowright et al., 1960), several closely related *gammaherpesviruses* have been described, and the list of susceptible indicator species and reservoir hosts have likewise increased. One of these viruses, namely ovine herpesvirus type 2 (OvHV-2) is endemic in sheep populations worldwide, but has never been propagated *in vitro*. Although it is non-pathogenic in sheep, it is the most important cause of MCF in the northern hemisphere and Oceania.

Two *gammaherpesviruses* carried subclinically by African antelope species were isolated, respectively, in 1973 and 1991, namely alcelaphine herpesvirus type 2 (AlHV-2) from hartebeest (*Alcelaphus buselaphus*) and hippotragine herpesvirus type 1 (HipHV-1) from roan antelope (*Hippotragus equinus*) (Reid and Rowe, 1973; Reid and Bridgen, 1991). The latter viruses are non-pathogenic in their carrier hosts and other species. A virus isolated from scimitar-horned oryx calves (*Oryx dammah*) may prove to be a new species (Flach et al., 2002), but remains unclassified. Two other *gammaherpesviruses* recently recognized, include the MCF virus of white-tailed deer (*Odocoileus virginianus*) (Kleiboeker et al., 2002), and caprine herpesvirus type 2 that is prevalent in clinically healthy goats (Li et al., 2001). Both these viruses are pathogenic for deer (Keel et al., 2003).

The economic importance of at least four of these *gammaherpesviruses* lies in their ability to cause MCF in either domestic cattle, buffaloes and farmed deer (McAllum et al., 1982; Wilson et al., 1983; Sutherland et al., 1987; Orr and Mackintosh, 1988; Brown and Bloss, 1992; Tham, 1997), pigs (Løken et al., 1998), and a formidable list of wild artiodactyl species in captivity or semi-captivity (Barnard et al., 1994). Although AIHV-1 and OvHV-2 can cause disease in many species of wild ruminants, under free-ranging conditions, MCF has never been reported in wild animals in Africa, notwithstanding the fact that wildebeest frequently roam with a wide variety of ungulates, such as the African buffalo (*Syncerus caffer*). In zoological collections, however, a wide variety of species have been reported to develop clinical signs. More than 150 ruminant species are susceptible to MCF virus infection, and clinical disease has been described in over 30 of these species almost exclusively in captivity.

Within the family *Bovidae*, in the subfamily *Bovinae*, it has been known since 1973 that the American bison (*Bison bison*) is highly susceptible to infection with OvHV-2 and development of MCF (Ruth et al., 1977). The latter has been described as the most important acutely fatal infectious disease in the commercial bison industry (O'Toole et al., 2002). Similarly, water buffaloes (*Bubalus bubalis*) and yaks are known to be highly susceptible (Reid and Van Vuuren, 2004). In contrast, the African buffalo has anecdotally and historically been regarded as a species resistant to MCF. This perception was based on the absence of references (documented or anecdotal) describing either clinical or subclinical infections with *gammaherpesviruses* in free-ranging and captive African buffaloes. Plowright (1963) working in East Africa reported that three buffaloes were not susceptible to MCF. In a serological survey described by Barnard et al. (1994), free-ranging African buffaloes were seronegative for MCF. Hamblin and Hedger (1984) similarly reported that no antibodies against AIHV-1 were detected in 848 African buffalo sera collected in several countries in Africa. More recently, Pagamjav et al. (2005) reported the absence of the gammaherpesvirus DNA polymerase gene in the blood of 34 African buffaloes in South Africa.

Notwithstanding the fact that African buffaloes in many conservancies live in close association with wildebeest (*Connochaetus* spp.), the reservoir host of alcelaphine herpesvirus type 1, clinical MCF has never been described in free-ranging buffaloes on the African continent.

Materials and Methods

Animals

The cases of MCF in African buffaloes included in this report were historical cases diagnosed during the past 10 years by private practitioners and institutions.

Case no. 1

An adult buffalo cow with a good calving history was nearly 12 years old when she developed clinical signs compatible with MCF in the Pretoria National Zoological Gardens during March 2003. Polymerase chain reaction (PCR) carried out on EDTA blood of this buffalo was positive for OvHV-2 (Table 1).

Case no. 2

An adult bull was introduced into the Pretoria National Zoological Gardens 5 years prior to development of MCF in May 2003 at the age of 8 years. Despite the fact that clinical signs and pathology were indicative of MCF, PCR results of clotted blood were negative (Table 1).

Case no. 3

A male buffalo was born and raised in a boma as part of the buffalo bovine tuberculosis (bTB) eradication scheme in South Africa. He was sold as a bTB-free animal and re-introduced into a wildlife reserve in northern Kwazulu-Natal province that stocked both blue and black wildebeest. This animal was reported sick several months after introduction in 2007, 3 days prior to its death. PCR results were positive for AIHV-1 (Table 1).

Case no. 4

An African buffalo cow on a game farm in the Free State Province where it co-mingled with other game species

Table 1. Summary of African buffaloes diagnosed with malignant catarrhal fever

| Signalment | Region | Habitat | Year | Virus identified with PCR | Sample type |
|-----------------|------------------------|------------|------|---------------------------|-------------------------------------|
| Adult cow | Gauteng Province | Captive | 2003 | OvHV-2 | EDTA blood |
| Adult bull | Gauteng Province | Captive | 2003 | Negative | Clotted blood |
| Subadult male | Kwazulu-Natal Province | Free-range | 2007 | AIHV-1 | Fresh kidney |
| Adult cow | Free State Province | Free-range | 2007 | OvHV-2 | Formalin (spleen, lymph node, lung) |
| Adult bull | Limpopo Province | Free-range | 2009 | AIHV-1 | Formalin (liver, lung, kidney) |
| Adult cow | Kwazulu-Natal Province | Free-range | 2011 | OvHV-2 | EDTA blood |
| Subadult female | Free State Province | Free-range | 2012 | OvHV-2 | EDTA blood |

including wildebeest fell sick and died in 2007. PCR results were positive for OvHV-2 (Table 1).

Case no. 5

An adult buffalo bull from Limpopo Province that was found sick and chemically immobilized for treatment in 2009. PCR results were positive for AIHV-1 (Table 1).

Case no. 6

An adult cow was translocated with a small group of disease-free buffaloes onto a game farm in northern Kwazulu-Natal province. She calved 6 months later. Buffaloes on the farm grazed together with Merino sheep on harvested maize fields until it was depleted at the end of July 2011. The cow became sick towards the end of August and was euthanized at the end of September. PCR results were positive for OvHV-2 (Table 1).

Case no. 7

A subadult female buffalo that became sick during August 2012. Supportive treatment likely contributed to full recovery of this animal 6 weeks later. A follow-up PCR test for OvHV-2 nucleic acid was positive 3 months following the start of clinical signs (Table 1).

Nucleic acid detection

Blood and tissue samples were submitted to the Biotechnology Division of the Onderstepoort Veterinary Institute for nucleic acid detection as previously reported (Bremer et al., 2005).

Histopathological examination

Histopathological examination of formalin-fixed tissue samples were carried out in the pathology laboratory, Faculty of Veterinary Science, Onderstepoort, South Africa (cases 1 and 2) and Vetdiagnostix laboratory, Pietermaritzburg, South Africa (cases 3 to 6).

Results

The first confirmed case was a 12-year-old buffalo cow housed at the Pretoria National Zoological Gardens. She had calved about 4 months prior to developing clinical signs. The first signs were non-specific and included a temperature of 39.2°C, listlessness and a dry muzzle. She was immobilized 3 days later and treated with antimicrobials and vitamins. Over the next 3 days, she seemed to improve and then became listless again while still eating. Ten days after the start of clinical signs, she developed bilateral mucopurulent nasal discharge, excessive tear production and bilateral blepharospasm as well as corneal oedema due

to keratitis. She also showed nervous signs such as high-stepping gait and moderate ataxia. PCR was carried out and found positive for OvHV-2. Two weeks after the start of clinical signs, the cow was euthanized and a post-mortem examination carried out. The examination did not reveal any of the typical gross lesions that are usually found in cases that succumb to MCF and therefore did not provide strong evidence for the positive PCR results and the typical clinical signs observed prior to euthanasia. Histopathological examination revealed mild multifocal lymphocytic perivascularitis in the brain and mild perivascular lymphocyte infiltration in the kidneys.

The second animal confirmed in the Pretoria National Zoological Gardens during 2003 was an 8-year-old bull. During May 2003, this buffalo was seen coughing and a nasal swab was examined for tuberculosis but was negative. Ten days later, a bronchoscopy was carried out under anaesthesia and puss as well as ulceration of the bronchi and a diphtheritic membrane in the trachea was found. Corneal opacity was noticed (Fig. 1). A bronchial wash was carried out, and material was examined and cultured. A mixed growth of *P. aeruginosa*, *K. pneumonia*, *Corynebacterium* and *Pasteurella* species was obtained. Blood was collected for PCR, but yielded a negative result. Four days later, the animal developed blepharospasm and excessive lacrimation of both eyes. The next day the bull was immobilized again and treated with antimicrobial and anti-inflammatory drugs. Further samples for PCR were collected. The mucopurulent nasal discharge and corneal opacity got worse (Fig. 2). The animal remained eating and seemed to improve with treatment but was losing weight. Further PCR tests for MCF were negative. On 6 June 2003 after further treatment and without much improvement, the animal was euthanized and a post-mortem examination



Fig. 1. Keratitis and corneal opacity in a buffalo bull suffering from sheep-associated malignant catarrhal fever.



Fig. 2. Epiphora and mucopurulent nasal discharge.

carried out. The lesions were highly indicative of MCF. The nasal cavity revealed severe acute diffuse fibrinopurulent rhinitis with hyperaemia of the mucous membranes and turbinate bones. Bronchi and lungs showed severe diffuse congestion with oedema and fibrinopurulent bronchopneumonia. Mild focal erosions could be seen in the abomasal mucosa, and the kidneys had developed a moderate diffuse nephrosis with mild congestion and swelling of the cortex as well as multifocal subacute to chronic lymphocytic nephritis with small disseminated raised nodules. Post-mortem lesions in the brain were dominated by multifocal subacute lymphocytic perivasculitis with infiltration of lymphoid cells around some small blood vessels. In the spleen, lymphoid hyperplasia was visible. Peripheral and organ lymph nodes showed lymphoid hyperplasia. The liver revealed a moderate diffuse subacute inter-lobular hepatitis with infiltration of lymphoid cells into portal tracts and scattered foci of fibrinoid necrosis of blood vessels in the portal tracts.

The first wild African buffalo confirmed with AIHV-1 infection was a subadult male that was noticed to be weak and separated from the herd 3 days prior to its death in 2007. This young bull was translocated to a wildlife reserve in Kwazulu-Natal province that also stocked blue and black wildebeest several months before and originated from a captive breeding project. The rectal temperature was recorded at 39.5°C shortly before death. Gross lesions included moderate anaemia, nephritis and perinephritis with adhesions, chronic hepatitis, intestinal serosal congestion and haematuria. Histopathological examination was characterized by a multifocal necrotizing vasculitis (arteritis and/or phlebitis) with dense perivascular cuffing by lymphocytes, lymphoblastic type cells and histiocytes. Morphological changes reported included a lymphohistiocytic interstitial nephritis, lymphohistiocytic

cholangiohepatitis, focal lymphocytic endocarditis, mural oesophageal necrotizing vasculitis, cerebral lymphocytic perivasculitis and nodular lymphocytic abomasitis. Wildebeest-associated MCF virus (AIHV-1) nuclei acid was detected in fresh unfixed renal tissue on PCR analysis.

The fourth animal included in this report hailed from the Free State, a province that is not included in the African buffalo's traditional range. This buffalo cow died in 2007 within 2 days of being seen sick. Gross post-mortem examination revealed widespread visceral haemorrhages particularly in the bladder and gall bladder. The liver was very enlarged and friable. Histopathological changes were dominated by multisystemic fibrinoid and necrotizing vasculitis with lymphoplasmacytic and histiocytic perivascular cuffing and vascular adventitial accumulates of lymphocytes. Morphological pathological changes included a mild lymphoplasmacytic interstitial nephritis, severe necrotizing ischaemic lymphadenitis and vasculitis, lymphoplasmacytic cholangiohepatitis with phlebitis, necrotizing splenitis with vasculitis, pulmonary vasculitis and severe lymphocytic enteritis with vasculitis. Immunohistochemical stains for bovine viral diarrhoea virus were negative, and the PCR assay was positive for OvHV-2.

The second confirmed case of wildebeest-associated MCF occurred in an adult bull in the Limpopo Province in 2009. Clinical examination of this bull revealed foul-smelling diarrhoea; pneumonic lung sounds on auscultation with noticeable dyspnoea. The animal died during the reversal of the immobilizing agent. Gross post-mortem examination revealed anaemia, severe pseudomembranous tracheitis, bronchopneumonia, necrotic enteritis, hepatomegaly and splenomegaly.

Histopathological examination revealed widespread lymphohistiocytic perivascular cuffing at multiple visceral sites accompanied by vasculopathy. Morphological pathological changes observed included pseudomembranous tracheitis with leukocytoclastic vasculitis, lymphoplasmacytic enteritis, lymphohistiocytic interstitial nephritis with fibrinoid vasculitis, lymphohistiocytic interstitial pneumonia with necrotizing vasculitis and lymphohistiocytic cholangiohepatitis. Wildebeest-associated MCF virus (AIHV-1) nuclei acid was detected in formalin-fixed liver, lung, kidney and trachea.

The sixth animal was again of particular interest as it was a free-ranging buffalo cow in a traditional buffalo region. This cow was translocated from another game farm 1 year previously. She had calved about 6 months before she showed signs of disease. This group of buffaloes was allowed to graze in a harvested maize field together with South African mutton Merino sheep of 1–4 years of age. Buffaloes and sheep were grazing in the field together for about 3 months until about July 2011. First signs that the

cow was not well appeared at the end of August 2011. She seemed depressed and separated from the herd. The cow was immobilized but apart from slight serous discharge that had dried on her nostrils, other clinical signs were absent. She was treated with antimicrobials and anti-inflammatory drugs, and blood was sent for a general medical profile. No significant changes could be established in the blood profile. The cow seemed better 1 week after treatment and ate a lot according to the farmer, but she kept losing weight and stayed separated from the herd. In middle September, the cow was put into an enclosure for closer supervision. She had obstructed airways, and the temperature was 39.2°C. She became worse a few days later, and a bilateral corneal opacity started to become visible. PCR results came back positive for OvHV-2. It was decided not to put the animal down as buffaloes are very valuable animals, and there was still hope that she might recover. However, at the end of September 2011, she was euthanized. A post-mortem examination was carried out, and the gross pathology and histopathology supported the PCR results. Histopathological examination revealed generalized vasculopathy with lymphoplasmacytic vascular cuffing, smooth muscle hypertrophy and fibrin leakage, especially in the lung. Abomasal mucosal infarction and ulceration were also visible.

The only survivor in the group of buffaloes included in this report is a buffalo cow that became sick at the age of 8 months. The cow presented with a temperature of 41 °C, nasal discharge and crusting of the muzzle. Epiphora was present, and mucopurulent discharges were observed in the corners of the eyes, but corneal opacity did not develop. A moist dermatitis was visible around the anus and down in the inguinal area for about 20 cm. Although the main differential diagnoses were infectious bovine rhinotracheitis and mucosal disease, blood was collected for PCR and yielded a positive result for ovine herpesvirus type 2. The animal was given supportive treatment in the form of antimicrobial and anti-inflammatory drugs as well as vitamins. She recovered completely without any sequelae. Blood was collected 3 months after the first bleed and again tested positive for OvHV-2 nucleic acid, confirming persistent infection.

Discussion

The first reference to possible MCF in the African buffalo was described by Barnard (1994) when he cited Tustin who worked with two African buffalo calves held in a small camp at the Onderstepoort Veterinary Research Institute during 1966. PCR assays were not available at the time, and viral isolation was not attempted. During the decade of the 1990s, two buffaloes died in the Pretoria National Zoological Gardens (PNZG) with lesions suspicious for MFC, but

confirmation by means of nucleic acid detection was not carried out (Espie, I., unpublished results).

The first laboratory confirmation of MCF in an African buffalo occurred in 2001 when a 2-year-old animal in the Western Cape Province (not included in this report) tested positive for OvHV-2 by means of PCR (Neser, J., unpublished results). During 2003, an adult buffalo cow suffering from clinical signs of MCF in the PNZG yielded positive PCR results for sheep-associated MCF. Paradoxically, the post-mortem examination could not confirm lesions typically seen with clinical MCF despite the fact that clinical signs were typical. An adult bull in the PNZG that died 2 months later yielded typical histopathological lesions, but negative PCR results. However, the history, clinical signs, gross pathology and microscopic lesions left little doubt that the animal suffered from MCF. The PCR was performed on clotted blood which may have contributed to the negative results as the sensitivity of the test is lower when clotted blood is used due to of inhibitory factors.

The clinical signs observed in both the captive and free-ranging buffaloes were very similar to the signs observed in cattle. In African buffaloes, the bilateral keratitis was also characterized by bilateral corneal opacity, and the nasal discharge that initially appears serous, later changes to mucopurulent. It dried on the muzzle causing obstruction of the nostrils and often difficult breathing. The animals became weak and disinclined to drink or eat. It was noticeable that the two buffaloes confirmed with wildebeest-associated MCF died after a short illness, whereas the sheep-associated cases survived for several weeks before succumbing to the disease. It is generally accepted that cattle are more susceptible to the effects of wildebeest-associated MCF, and this may also be the case in African buffaloes.

Histopathologically, these cases were characterized by fibrinoid necrotizing vascular changes, arteritis as well as phlebitis and marked hyperplasia of lymphoid cells in lymphoid tissues. There were widespread perivascular, vascular adventitial and interstitial accumulation of these cells in non-lymphoid organs, similar to the pathology described in domestic cattle with MCF and experimental infection of bison (Liggitt et al., 1980; Schultheiss et al., 1998, 2000; Reid and Van Vuuren, 2004). Severe intestinal congestion and haemorrhage reported in bison were not observed in these buffalo cases (Liggitt et al., 1980; Schultheiss et al., 2000). The fibrinoid necrotizing vasculitis of intestinal mucosal and submucosal vessels reported here appears to be more common in buffalo than bison and was frequently documented in domestic cattle (Schultheiss et al., 1998; Brown et al., 2007). These vascular lesions were evident in all cases reported here except case No 1 and quite distinct from the more subtle vascular lesions described in bison, deer and elk, where fibrinoid vascular necrosis is usually absent and the number of vascular and perivascular

lymphoid cells usually low (Liggitt et al., 1980; Schultheiss et al., 2000; Brown et al., 2007). However, mild cases of sheep-associated MCF have also been described in cattle (O'Toole et al., 1997). Schultheiss et al. (2000) stated that subtle inflammatory lesions that might be considered non-diagnostic in cattle appear to be important in bison. The mild lesions found in case no. 1 are therefore compatible with sheep-associated MCF in bison and rarely in cattle.

During the past two decades, disease-free buffaloes (buffaloes free from bovine tuberculosis, foot-and-mouth disease, theileriosis and brucellosis) became widely distributed throughout South Africa. This translocation has resulted in African buffaloes being exposed to sheep which otherwise may never have occurred (except in zoological gardens). The fact that one buffalo survived clinical disease caused by OvHV-2 is in accordance with reports that animals suffering from the wildebeest-associated disease very seldom if ever survive, whereas the sheep-associated disease can lead to more chronic cases and the odd survivor (Michel and Aspeling, 1994; O'Toole et al., 1997; Penny, 1998). Skin lesions observed in the one surviving buffalo is also in accordance with the observation that the sheep-associated disease is mainly if not exclusively responsible for skin lesions.

The incidence of clinical disease in buffalo still appears to be low, possibly mirroring the situation in cattle where large numbers of animals may be exposed to the MCF virus but only a few develop overt infection (Løken et al., 2009). The severity of the pathology in buffalo is also similar to that of domestic cattle, and this might be related to the susceptibility of the particular species to infection. Highly susceptible species such as bison, elk and deer possibly succumb to infection before there has been adequate time to develop severe pathology. Survival of case 7 would support some resistance in African buffaloes, yet the mild histopathology described in some cases suggests that there may be a spectrum of susceptibility. In the ever-changing landscape of game farming with increased co-mingling with domestic livestock and introduction of African buffalo into areas that are not part of their traditional range, the epidemiology of MCF in African buffaloes is still very much an emerging phenomenon.

References

- Barnard, B. J. H., J. J. van der Lugt, and E. Z. Mushi, 1994: Bovine malignant catarrhal fever. In: Coetzer, J. A. W., R. Tustin, G. Thompson, and N. P. J. Kriek (eds), *Infectious Diseases of Livestock*, 1st edn, pp. 946–957. Oxford University Press, South Africa.
- Bremer, C. W., H. H. Swart, F. A. Doboro, B. Dungu, M. Romito, and G. J. Viljoen, 2005: Discrimination between sheep-associated and wildebeest-associated malignant catarrhal fever virus by means of a single-tube duplex nested PCR. *Onderstepoort J. Vet. Res.* 72, 285–291.
- Brown, C. C., and L. L. Bloss, 1992: An epizootic of malignant catarrhal fever in a large captive herd of white-tailed deer (*Odocoileus virginianus*). *J. Wildl. Dis.* 28, 301–305.
- Brown, C. C., D. C. Baker, and I. K. Barker, 2007: Alimentary system. In: Maxie, M. G. (ed.), *Pathology of Domestic Animals*, 5th edn, pp. 152–158. Saunders Elsevier, USA.
- Flach, E. J., H. Reid, I. Pow, and A. Klempt, 2002: Gamma herpesvirus carrier status of captive artiodactyls. *Res. Vet. Sci.* 73, 93–99.
- Hamblin, C., and R. S. Hedger, 1984: Neutralizing antibodies to wildebeest-derived malignant catarrhal fever in African wildlife. *Comp. Immunol. Microbiol.* 7, 195–199.
- Keel, M. K., J. G. Patterson, T. H. Noon, G. A. Bradley, and J. K. Collins, 2003: Caprine herpesvirus-2 in association with naturally occurring malignant catarrhal fever in captive sika deer (*Cervus Nippon*). *J. Vet. Diagn. Invest.* 15, 179–183.
- Kleiboeker, S. B., M. A. Miller, S. K. Schommer, J. A. Ramos-Vara, M. Boucher, and S. E. Turnquist, 2002: Detection and multigenic characterization of a herpesvirus associated with malignant catarrhal fever in white-tailed deer (*Odocoileus virginianus*). *J. Clin. Microbiol.* 40, 1311–1318.
- Li, H., J. Keller, D. P. Knowles, and T. B. Crawford, 2001: Recognition of another member of the malignant catarrhal fever virus group: an endemic gammaherpesvirus in domestic goats. *J. Gen. Virol.* 82, 227–232.
- Liggitt, H. D., A. E. McChesney, and J. C. DeMartini, 1980: Experimental transmission of bovine malignant catarrhal fever to a bison (*Bison bison*). *J. Wildl. Dis.* 16, 299–304.
- Løken, T., M. Aleksandersen, H. Reid, and I. Pow, 1998: Malignant catarrhal fever caused by ovine herpesvirus-2 in pigs in Norway. *Vet. Rec.* 143, 464–467.
- Løken, T., A.-M. Bosman, and M. Van Vuuren, 2009: Infection with ovine herpesvirus type 2 in Norwegian herds with a history of previous outbreaks of malignant catarrhal fever. *J. Vet. Diagn. Invest.* 21, 257–261.
- McAllum, H. J. F., N. M. Mavor, and P. Hemmingsen, 1982: A malignant catarrhal fever-like disease in red deer (*Cervus elaphus*). *N. Z. Vet. J.* 30, 99–101.
- Michel, A. L., and I. A. Aspeling, 1994: Evidence of persistent malignant catarrhal fever infection in a cow obtained by nucleic acid hybridisation. *J. S. Afr. Vet. Assoc.* 65, 26–27.
- O'Toole, D., H. Li, D. Miller, W. R. Williams, and T. B. Crawford, 1997: Chronic and recovered cases of sheep-associated malignant catarrhal fever in cattle. *Vet. Rec.* 140, 519–524.
- O'Toole, D., H. Li, C. Sourk, D. L. Montgomery, and T. B. Crawford, 2002: Malignant catarrhal fever in a bison (*Bison bison*) feedlot. *J. Vet. Diagn. Invest.* 14, 183–193.
- Orr, M. B., and C. G. Mackintosh, 1988: An outbreak of malignant catarrhal fever in Père David's deer (*Elaphurus davidianus*). *N. Z. Vet. J.* 36, 19–21.
- Pagamjav, O., T. Sakata, E. M. Ibrahim, C. Sugimoto, S. Takai, J. T. Paweska, T. Yamaguchi, J. Yasada, and H. Fukushima, 2005:

- Detection of novel gammaherpesviruses in wild animals in South Africa. *J. Vet. Med. Sci.* 11, 1185–1188.
- Penny, C., 1998: Recovery of cattle from malignant catarrhal fever. *Vet. Rec.* 142, 227.
- Plowright, W., R. D. Ferris, and G. R. Scott, 1960: Blue wildebeest and the aetiological agent of bovine malignant catarrhal fever. *Nature* 188, 1167–1169.
- Reid, H. W., and A. Bridgen, 1991: Recovery of a herpesvirus from a roan antelope (*Hippogtragus equines*). *Vet. Microbiol.* 28, 269–273.
- Reid, H. W., and L. Rowe, 1973: The attenuation of a herpesvirus isolated from hartebeest (*Alcelaphus buselaphus cokei*, Gunther). *Res. Vet. Sci.* 15, 144–146.
- Reid, H. W., and M. Van Vuuren, 2004: Bovine malignant catarrhal fever. In: Coetzer, J. A. W., and R. C. Tustin (eds), *Infectious Diseases of Livestock*, 2nd edn, pp. 895–908. Oxford University Press, South Africa.
- Ruth, G. R., D. E. Reed, and C. A. Daley, 1977: Malignant catarrhal fever in bison. *J. Am. Vet. Med. Assoc.* 171, 913–917.
- Schultheiss, P. C., J. K. Collins, L. E. Austgen, and J. C. DeMartini, 1998: Malignant catarrhal fever in bison, acute and chronic cases. *J. Vet. Diagn. Invest.* 10, 255–262.
- Schultheiss, P. C., J. K. Collins, T. R. Spraker, and J. C. DeMartini, 2000: Epizootic malignant catarrhal fever in three bison herds: differences from cattle, and association with ovine herpesvirus-2. *J. Vet. Diagn. Invest.* 12, 497–502.
- Sutherland, R. J., R. E. Oliver, B. W. Saunders, and W. S. Poole, 1987: Changes in blood coagulation parameters of red deer (*Cervus elaphus*) experimentally infected with malignant catarrhal fever. *N. Z. Vet. J.* 35, 150–154.
- Tham, K. M., 1997: Molecular and clinicopathological diagnosis of malignant catarrhal fever in cattle, deer and buffalo in New Zealand. *Vet. Rec.* 141, 303–306.
- Wilson, P. R., M. R. Alley, and A. C. Irving, 1983: Chronic malignant catarrhal fever: a case in a sika deer (*Cervus Nippon*). *N. Z. Vet. J.* 31, 7–9.