

MALIGNANT CATARRHAL FEVER (MCF)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Many species of <i>Artiodactyla</i> , including bovids, cervids, giraffids, suids	<p>Direct contact with infected individuals and bodily fluids (nasal and ocular secretions).</p> <p>Fomites Water (possible)</p> <p>Aerosol transmission also important for some of the viruses.</p> <p>Transmitted only between carriers and clinically susceptible animals. Affected animals do not transmit MCF to their conspecifics.</p>	<p>Mucous membrane ulceration and inflammation, high fever, oral and nasal exudates, corneal opacities, and lymph-adenopathy</p> <p>Additional signs may include: neurologic signs, diarrhea, arthritis, and skin lesions.</p>	<p>Typically fatal in susceptible species</p> <p>Up to 25% of cattle may develop chronic disease with a waxing and waning course. Up to 5% may clinically recover, but most eventually die.</p>	None, usually ineffective	<p>Separate clinically susceptible species from known carrier species such as sheep, goats, and wildebeest in known infected areas to prevent direct transmission</p> <p>Prevent fomite transmission</p> <p>Use precautions to prevent aerosol transmission</p>	No

Fact Sheet compiled by: Danelle M. Okeson

Sheet completed on: 20 August 2010; updated 22 September 2012.

Fact Sheet Reviewed by: Hong Li; Naomi S. Taus

Susceptible animal groups: Cervidae, Bovidae, Giraffidae, Suidae

Causative organism: Herpesviruses classified in the genus *Macavirus*. At least 10 viruses have been identified that are categorized within the MCF virus group. Some have been associated with MCF in clinically susceptible species.

Alcelaphine herpesvirus 1 (AIHV-1; classic African MCF/wildebeest-associated), carried by wildebeest; susceptible species = Cervidae and Bovidae

Ovine herpesvirus 2 (OvHV-2; sheep-associated), carried by domestic and wild sheep, considered endemic in domestic sheep; susceptible species = ruminant species and swine. Most MCF cases in domestic cattle and bison in the US are due to OvHV-2. European breeds of cattle (*Bos taurus*), are relatively resistant, but Bali

MALIGNANT CATARRHAL FEVER (MCF)

cattle, bison, and some cervid species such as Pere David's deer are highly susceptible.

Caprine herpesvirus 2 (CpHV-2), carried by domestic and exotic goats, considered endemic in domestic goats; clinically susceptible species = white-tailed deer, Sika deer.

A herpesvirus referred to as "malignant catarrhal fever virus-white tailed deer" (MCFV-WTD), carrier unknown; susceptible species = white-tailed deer.

Ibex-MCFV, carried by Nubian ibex (*Capra nubiana*); prior to a case in a captive bongo (*Tragelaphus euryceros*) the virus was not considered pathogenic.

Alcelaphine herpesvirus 2 (AIHV-2), identified in but non-pathogenic in Jackson's hartebeest; clinically susceptible species = Barbary red deer (*Cervus elaphus barbarus*)

Other herpesviruses categorized in the same group as the pathogenic MCF viruses have been identified in aoudad, roan antelope, musk ox, gemsbok, but do not yet appear to cause disease under natural conditions.

Zoonotic potential: No

Distribution: Disease may occur worldwide in situations in which clinically susceptible species are in contact with carrier species.

Incubation period: It varies depending on several factors such as amount of virus transmitted and host. In field outbreaks, the incubation period for bison is about 40 to 70 days. Cattle have become ill in as few as 9 days, while other evidence suggests that some cattle may be subclinically infected for 20 months or more before developing the disease. The latter case could be due to a long period of subclinical infection followed by viral reactivation leading to clinical disease.

Clinical signs: These vary with susceptibility of affected species. Highly susceptible species may have a peracute course with few to no clinical signs or sudden death after non-specific signs such as depression, weakness, and diarrhea. Acute disease may involve high fever and a loss of appetite. Clinical signs may include: mucous membrane ulceration and inflammation, high fever, oral and nasal exudates, corneal opacities (common in domestic cattle), and lymphadenopathy. Additional signs may include: neurologic signs, diarrhea, arthritis, and skin lesions may also develop.

Carrier species do not typically develop clinical signs.

Post mortem, gross, or histologic findings: These may vary with disease severity and course, but often include "inflammation and epithelial necrosis in the gastrointestinal, respiratory, and urinary tracts, with lymphoproliferation, infiltration of nonlymphoid tissues (particularly the renal cortex and periportal areas of the liver) by lymphoid cells, and vasculitis".

Diagnosis: PCR (polymerase chain reaction) is the method of choice for viral detection.

Serological tests for antibodies include competitive inhibition ELISA (cELISA), immunoperoxidase test (IPT), neutralization test (NT) and others. Detection of antibodies indicates infection, not necessarily disease.

Material required for laboratory analysis: Antibody testing by cELISA - serum or plasma.

Antemortem detection of viral DNA by PCR - whole blood in EDTA.

Postmortem detection of viral DNA by PCR - preferred samples - lymph node or spleen; but lung, kidney, and intestine among others are also acceptable.

Relevant diagnostic laboratories:

Washington Animal Disease Diagnostic Laboratory (WADDL), Pullman, Washington. National Veterinary Services Laboratories (NVSL) in Ames, Iowa.

Treatment: No treatment is available or usually ineffective. Supportive care may be administered, but disease is often acute and fatal in highly susceptible species. Some animals may die without clinical signs. Occasional reports of recovery in treated cattle exist, but some cattle may also recover without treatment.

Prevention and control: Separate clinically susceptible species from carrier species such as wildebeest, domestic and exotic sheep, and domestic and exotic goat species. Bovids, particularly bison and water

MALIGNANT CATARRHAL FEVER (MCF)

buffalo, are highly susceptible to MCF. Exotic members of the bovidae family such as bongo antelope have died from MCF traced back to an exotic goat species. Wildebeest-associated MCF has occurred in domestic cattle in the U.S. when the two species were housed together.

Cervids should not be mixed with sheep, goats, or wildebeest.

Prevent direct contact and fomite transmission. Transmission of the wildebeest-associated form (AIHV-1) and the sheep-associated form (OvHV-2) is believed to occur primarily from either direct contact with infected body fluids or secretions, or via fomites such as water sources, feeders, caretakers, and birds.

Calving is considered a high risk period for transmission.

Use precautions to prevent aerosol transmission. Transmission of the disease over relatively short distances has occurred, indicating that direct contact is not absolutely necessary. Aerosol transmission is a significant mode of transmission of OvHV-2 in domestic sheep.

Suggested disinfectant for housing facilities:

Herpesviruses causing MCF are typically “fragile and quickly inactivated in harsh environments”, so common disinfectants are likely effective.

Notification: The wildebeest-associated and sheep-associated forms are reportable diseases under USDA-APHIS-VS National Animal Health Reporting System. MCF clinical signs may appear similar to foreign animal diseases such as rinderpest and foot and mouth disease.

Measures required under the Animal Disease Surveillance Plan: Currently none

Measures required for introducing animals to infected animal: Clinically susceptible species should not be introduced to carrier species. Clinically susceptible species should be physically separated from carrier species. In addition, separate keeper staff and equipment should be used to prevent fomite transmission.

Conditions for restoring disease-free status after an outbreak: See prevention and control measures.

Experts who may be consulted:

Hong Li, DVM, PhD

Animal Disease Research Unit, USDA-ARS

Washington State University

Phone: (509) 335-6002

Fax: (509) 335-8328

3019 ADBV, WSU

Pullman, WA 99164-6630

hli@vetmed.wsu.edu or Hong.Li@ARS.USDA.GOV

References:

1. Barnard, B.J.H., and H.E. Van de Pypekamp. 1988. Wildebeest-derived malignant catarrhal fever: unusual epidemiology in South Africa. *Onderstepoort J. Vet. Res.* 55: 69-71.
2. Crawford, T.B., H. Li, S.R. Rosenberg, R.W. Nordhausen, and M.M. Garner. 2002. Mural folliculitis and alopecia caused by infection with goat-associated malignant catarrhal fever virus in two sika deer. *J. Am. Vet. Med. Assoc.* 221: 843-847.
3. Gasper, D., B. Barr, H. Li, N. Taus, R. Peterson, G. Benjamin, T. Hunt, and P.A. Pesavento. 2012. Ibex-associated malignant catarrhal fever-like disease in a group of bongo antelope (*Tragelaphus euryceros*). *Vet. Pathol.* 49(3): 492-497.
4. Heuschele, W.P. 1993. Malignant catarrhal fever. *In: Fowler, M.E. (ed.). Zoo and Wild Animal Medicine Current Therapy 3.* W.B. Saunders Company, Philadelphia, Pennsylvania. Pp. 504-506.
5. Heuschele, W.P., and H.W. Reid. 2001. Malignant catarrhal fever. *In: Williams, E.S., and I.K. Barker (eds.). Infectious Diseases of Wild Mammals, 3rd ed.* Iowa State University Press, Ames, Iowa. Pp. 157-164.

MALIGNANT CATARRHAL FEVER (MCF)

6. Li, H., C.W. Kunha, and N.S. Taus. 2011. Malignant catarrhal fever: understanding molecular diagnostics in context of epidemiology. *Int. J. Mol. Sci.* 12: 6881-6893.
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3211016/pdf/ijms-12-06881.pdf>
7. Li, H., K. Gailbreath, L.C. Bender, K. West, J. Keller, and T.B. Crawford. 2003. Evidence of three new members of malignant catarrhal fever virus group in muskox (*Ovibos moschatus*), Nubian ibex (*Capra nubiana*), and gemsbok (*Oryx gazella*). *J. Wildl. Dis.* 39: 875-880.
8. 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.
9. Loken, T., M. Aleksandersen, H. Reid, and L. Pow. 1998. Malignant catarrhal fever caused by ovine herpesvirus-2 in pigs in Norway. *Vet. Rec.* 143: 464-467.
10. MCF web site – College of Veterinary Medicine, Washington State University
<http://www.vetmed.wsu.edu/mcf>. Accessed 2 July 2013.
11. MCF Fact sheet – Center for Food Security and Public Health, Iowa State University
http://www.cfsph.iastate.edu/Factsheets/pdfs/malignant_catarrhal_fever.pdf. Accessed 2 July 2013.
12. Okeson, D.M., M.M. Garner, N.S. Taus, H. Li, and R.L. Coke. 2007. Ibex-associated malignant catarrhal fever in a bongo antelope (*Tragelaphus euryceros*). *J. Zoo Wildl. Med.* 38: 460-464.
13. O'Toole, D., H. Li, C. Sourk, D.L. Montgomery, and T.B. Crawford. 2002. Malignant catarrhal fever in a bison (*Bison bison*) feedlot, 1993-2000. *J. Vet. Diagn. Invest.* 14: 183-193.