ALCELAPHINE HERPESVIRUS 1 AND OVIS HERPESVIRUS 2

Aetiology Epidemiology Diagnosis Prevention and Control
Potential Impacts of Disease Agent Beyond Clinical Illness References

AETIOLOGY

Classification of the causative agent

Alcelaphine herpesvirus 1 (AlHV-1) and ovine herpesvirus 2 (OvHV-2) are the causes of malignant catarrhal fever (MCF) in several ruminant and cervid species. They are part of the gammaherpesvirinae subfamily in the *Macavirus* genus. The reservoir host of AIHV-1 is the wildebeest (*Connochaetes gnou*, *C. taurinus*); the reservoir host of OvHV-2 is the sheep (wild and domestic). Both viruses cause clinical MCF in bovids, cervids, and pigs. There are ten identified viruses that cause MCF; six are known to cause the disease in animals. AlHV-1 and OvHV-2 are the most-widely studied and understood. Other viruses include caprine herpesvirus-2 (CpHV-2, endemic to goats) and ibex MCFV (endemic to ibex (genus *Capra*)).

For the purpose of voluntary reporting on non OIE-listed disease in wildlife, “Alcelaphine herpesvirus 1 and ovine herpesvirus 2” refers to infection in ruminant and cervid species.

Resistance to physical and chemical action

Temperature:	Infectivity lost at 56°C after 9-15 minutes
pH:	Stable between pH 5.5-8.5
Chemicals/Disinfectants:	Inactivated by detergents, lipid solvents, and 3% sodium hypochlorite
Survival:	Survives best in moist and humid environments; unable to survive outside of host for extended periods of time

EPIDEMIOLOGY

Hosts

- AIVH-1
  - Reservoir hosts: wildebeest (*Connochaetes gnou*, *C. taurinus*)
  - Susceptible hosts: domestic cattle (*Bos taurus*)
- OvHV-2
  - Reservoir hosts:
    - Domestic sheep (*Ovis aries*)
    - Bighorn sheep (*Ovis canadensis*)
    - Mouflon (*Ovis aries orientalis* group)
  - Susceptible hosts:
    - American bison (*Bison bison*)
    - European bison (*Bison bonasus*)
    - Bali cattle (*Bos javanicus*)
    - Water buffalo (*Bubalus bubalis*)
    - Moose (*Alces alces*)
    - Domestic cattle (*Bos taurus*)

- Domestic goats (*Capra hircus*)
- Giraffes (*Giraffa* spp.)
- Domestic swine (*Sus scrofa*)
- Sika deer (*Cervus nippon*)

Transmission

- Inhalation of aerosols
- Ingestion of contaminated food or water
- It is thought that nearly all wildebeest are infected with AlHV-1. Wildebeest calves are particularly infective during the first three months of life, after which neutralizing antibodies result in a decrease in viral load. Adult wildebeest shed the virus at lower levels throughout their lifetime, though levels may increase during calving or stress.
- Shedding of viral particles through nasal discharge is thought to be the primary mode of transmission. Horizontal transmission to lambs is most common via secretions from adult sheep, and most lambs acquire the virus in this way; however, transplacental infection is also possible. Lambs aged 6-9 months are thought to shed most of the virus in a population.

Sources

- Secretions from reservoir hosts

Occurrence

OvHV-2 is thought to be endemic in sheep worldwide. Disease outbreaks of AlHV-1 in cattle occur in Africa, particularly in Kenya, Tanzania, and South Africa, during the wildebeest calving season. In 2008, there was an outbreak of AlHV-1 in cattle in Texas, which was thought to be introduced by wildebeest that were housed on the same farm. Outbreaks associated with OvHV-2 are a problem in bison feedlots in the United States and for Bali cattle in Indonesia, but disease usually only affects a few members of the herd. Since 2015 in China, there has been an emerging outbreak of MCF in sika deer due to both OvHV-2 and CpHV-2.

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE World Animal Health Information System - Wild (WAHIS-Wild) Interface [http://www.oie.int/wahis_2/public/wahidwild.php/Index].

DIAGNOSIS

MCF pathogenesis is not well-understood. It is thought that T-lymphocytes are the primary reason for clinical disease in animals infected with the virus, with CD8+ T-cells accumulating in affected tissues. Regarding OvHV-2 infection in sheep, the virus starts replicating in the alveoli and migrates to nasal turbinates, from which the virus is shed.

Mortality of incidental hosts with clinical MCF is nearly 100%. However, it is believed that many incidental hosts develop an antibody response to the viruses without demonstrating clinical signs of disease. Reservoir hosts of the virus do not generally show clinical signs of MCF, though MCF has been induced in sheep experimentally when inoculated with high doses of OvHV-2.

Wildebeest shed AlHV-1 during the first 3 months of life, acquiring the virus either in utero or soon after birth from wildebeest shedding the virus. Sheep shed OvHV-2 starting 5 months after birth, usually after acquiring

the virus from ocular and nasal secretions of sheep; transplacental transmission or suckling causes infection less often. The incubation period of these viruses in reservoir hosts is 3-4 weeks. Death occurs around 2-7 days after clinical signs, though some animals are able to survive infection and appear to have lifelong immunity.

Clinical diagnosis

Development of clinical signs are similar among incidental hosts. These include lymphadenopathy, nasal and ocular discharge, leukopaenia, fever, depression, torticollis, ataxia, and diarrhoea. Lesions of the oral mucosa and gastrointestinal tract result in haemorrhage and melaena. Bilateral corneal opacities may lead to blindness. Bison may abort their foetuses. Subclinical infections may occur in cattle, bison, and deer. Host species have subclinical, lymphotropic infections.

Lesions

- Bilateral corneal opacities
- Haemorrhage and mucosal erosions of gastrointestinal tract
- Haemorrhagic cystitis of urinary bladder
- Raised white nodules (approximately 1-5 mm in diameter) on internal organs
- Erosions and haemorrhages of laryngeal, tracheal, nasal, and oral mucosa
- Interstitial inflammation of renal cortex
- Lymphadenopathy, sometimes haemorrhagic
- Cattle: lymphocytic arteritis-periarteritis with tunica media necrosis and T-lymphocytes

Differential diagnoses

- All incidental hosts
    - Rabies
    - Toxin exposure (e.g., caustic agents)
    - Vesicular stomatitis virus (VSV)
    - Rinderpest
    - Foot-and-mouth disease (FMD)
    - Bluetongue virus (BTV)
- Cattle
    - Bovine viral diarrhoea (BVDV)
    - Infectious bovine rhinotracheitis (IBR, bovine herpesvirus-1)
    - Footrot
    - Pseudocowpox, cowpox
    - Bovine herpes mammillitis, pseudo-lumpy skin disease (bovine herpesvirus-2)

Laboratory diagnosis

Samples

For isolation of agent

- Whole blood in EDTA
- Peripheral blood leukocytes
- Lymph node

- Intestine
- Kidney
- Lung

Serological tests

- Serum or plasma

Procedures

Identification of the agent

- Viral isolation of AlHV-1 from peripheral blood leukocytes, secretions, or tissues; to date there have been no viral isolations of OvHV-2
- Polymerase chain reaction (PCR)
    - Multiple modalities are utilised diagnostically, including quantitative, nested, real-time, and multiplex techniques

Serological tests

- Antigen capture enzyme-linked immunosorbent assay
- Competitive-inhibition ELISA (cELISA)
    - Can detect antibodies to AlHV-1 and OvHV-2
- Virus neutralisation
    - Tests are available to detect antibodies to AlHV-1 in wildebeest
    - Not useful for detection of OvHV-2 in sheep because they generally do not produce neutralizing antibodies
- Immunofluorescence assay (IFA)/immunoperoxidase test (IPT)
    - OvHV-2 antibodies may cross-react with antibodies for bovine herpesviruses

For more detailed information regarding laboratory diagnostic methodologies, please refer to Chapter 3.4.13 *Malignant Catarrhal Fever* in the latest edition of the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals.

PREVENTION AND CONTROL

Sanitary prophylaxis

- Move livestock herds away from breeding grounds for wildebeest and wild/free-ranging sheep, especially before calving/lambing seasons
- Remove lambs from flock after birth to prevent OvHV-2 infection in domestic sheep
- Keep sheep and wildebeest separate from susceptible animals in zoologic or game parks

Medical prophylaxis

- While there is no commercially available vaccine available, an experimental attenuated vaccine was successful in protecting cattle in Kenya from AlHV-1 infection (strain C500)

POTENTIAL IMPACTS OF DISEASE AGENT BEYOND CLINICAL ILLNESS

Risks to public health

- There is a lack of evidence to suggest this virus poses a risk to human health.

Risks to agriculture

- Sheep or wildebeest that graze or are housed in the same areas as incidental hosts such as goats, bison, cervids, or cattle may lead to a higher risk of viral transmission to these animals and subsequent ill-thrift, production losses and death due to infection.
- There may be a decrease in milk production from cattle and bison may abort their foetuses, resulting in financial losses to farmers.

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The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Science Department (scientific.dept@oie.int). Last updated 2020. Written by Samantha Gieger and Erin Furmaga with assistance from the USGS National Wildlife Health Center.