

Malignant Catarrhal Fever

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Snotsiekte*

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Importance

Malignant catarrhal fever (MCF) is a serious, often fatal, disease that affects many species in the order Artiodactyla (even-toed ungulates) including cattle, bison, deer, moose, exotic ruminants and pigs. At least ten MCF viruses have been recognized, including two well-known viruses carried by sheep and wildebeest. Six of these viruses have been linked to disease, while the others have been found, to date, only in asymptomatic carriers. Each MCF virus is highly adapted to its usual host, and does not normally cause disease in that species, but can cause fatal infections if transmitted to susceptible animals.

Malignant catarrhal fever occurs in many countries worldwide. Sheep-associated MCF is the predominant form outside Africa. It is a particular problem in species such as farmed bison, deer and Bali cattle, although it occasionally affects relatively resistant hosts such as pigs and European breeds of cattle. Wildebeest associated MCF is an important disease among cattle in Africa, while zoos can be affected by either of these two forms, as well as by less common MCF viruses carried in various exotic ruminants. Malignant catarrhal fever is difficult to control, as the infections are widespread and asymptomatic in the reservoir species, and the incubation period can be long in susceptible animals. The only reliable methods of control are to separate susceptible species from carriers or breed virus-free reservoir hosts.

Etiology

Malignant catarrhal fever is caused by viruses in the genus *Macavirus* of the family Herpesviridae (subfamily Gammaherpesvirinae). There are two major groups of MCF viruses. The Alcelaphinae/Hippotraginae group contains alcelaphine herpesvirus 1 (AIHV-1), AIHV-2, hippotragine herpesvirus 1 (HiHV-1) and a virus carried in oryx (MCFV-oryx). The Caprinae group includes ovine herpesvirus 2 (OvHV-2), caprine herpesvirus 2 (CpHV-2), MCF virus-white tailed deer (MCFV-WTD), and the viruses carried by asymptomatic ibex (MCFV-ibex), muskox (MCFV-muskox) and aoudad (MCFV-aoudad). Most MCF viruses are named after their reservoir hosts; however, MCFV-WTD was found in sick white-tailed deer, and its carrier is unknown.

The two most important viruses are OvHV-2, which causes sheep-associated MCF, and AIHV-1, which causes the wildebeest-associated form of this disease. CpHV-2, MCFV-WTD, MCFV-ibex and AIHV-2 are also known to be pathogenic. No illness has been associated yet with MCFV-muskox, MCFV-oryx or MCFV-aoudad, which are carried in muskox (*Ovibos moschatus*), gemsbok/ South African oryx (*Oryx gazella*), and aoudads (*Ammotragus lervia*), respectively. Nevertheless, it is likely that these viruses can also cause MCF in some species of animals. HiHV-1, which was found in asymptomatic roan antelope (*Hippotragus equinus*), appears to be very similar or identical to MCFV-oryx.

Species Affected

MCF viruses are usually carried asymptotically by their reservoir hosts, but can cause disease in other species. Wildebeest (*Connochaetes* spp.) are the carriers for AIHV-1. The blue wildebeest (*Connochaetes taurinus*) is the major reservoir host, but black wildebeest (*Connochaetes gnou*) are also carriers. All or most wildebeest appear to be infected by this virus. Domesticated sheep (*Ovis aries*) are the reservoir hosts for OvHV-2, and most individuals are infected. At least some species of wild sheep, such as Dall's sheep (*Ovis dalli*) and mouflon (*Ovis musimon*) are also carriers. Goats are the carriers for CpHV-2. Most goats are thought to be infected with this virus, based on high seroprevalence to MCF viruses; however, goats can also be infected asymptotically with OvHV2, and serological tests cannot distinguish these two organisms. Alcelaphine herpesvirus-2 (AIHV-2) is carried subclinically in hartebeest (*Alcelaphus buselaphus*) and topi (*Damaliscus korrigum*). Nubian ibex (*Capra nubiana*) are known to be reservoir hosts for MCFV-ibex. The reservoir host for MCFV-WTD is uncertain, but goats may carry this virus.

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MCF viruses cause illness in various members of the order Artiodactyla. Most susceptible animals belong to the subfamily Bovinae (e.g. cattle, bison, water buffalo, and exotic ruminants such as antelope, guar and banteng) and family Cervidae (e.g. deer, reindeer, moose), but other species such as giraffes (family Giraffidae) and pigs (family Suidae) are also affected. A species can be susceptible to one MCF virus, but relatively resistant to others.

Most ruminants other than antelope of the subfamilies Alcelaphinae and Hippotraginae should be considered susceptible to AIHV-1. Significant numbers of cattle become ill if they are exposed to this virus.

European breeds of cattle (*Bos taurus* and *Bos indicus*) tend not to become sick when they are exposed to OvHV-2. When cases do occur, they often affect only a single animal. Water buffalo (*Bubalus bubalis*) and some species of deer are more susceptible to this virus than cattle, and American bison (*Bison bison*), Père David's deer (*Elaphurus davidianus*), white-tailed deer (*Odocoileus virginianus*), axis deer (*Axis axis*) and Bali cattle (*Bos javanicus*) are highly susceptible. OvHV-2 can also affect pigs, giraffes and exotic ruminants. OvHV-2 infections were recently reported in horses (order Perissodactyla) on a farm where these animals shared their feed with goats.

CpHV-2 associated disease has been seen in cervids including moose (*Alces alces*), roe deer (*Capreolus capreolus*), sika deer (*Cervus nippon*), white-tailed deer and pronghorn antelope (*Antilocapra americana*), as well as in water buffalo. Other MCF viruses have been linked only rarely to clinical cases. MCFV-WTD was found in sick white-tailed deer. The ibex-associated MCF virus has caused disease in several bongo antelope (*Tragelaphus euryceros*) and an anoa, and a virus resembling AIHV-2- was found in sick Barbary red deer (*Cervus elaphus barbarus*).

Although most cases of MCF have been reported in domesticated or captive animals, there are also reports of illness in wild animals such as moose and deer. A variety of ungulates, hamsters and rabbits can be infected experimentally with OvHV-2 and AIHV-1. In unusual cases, MCF viruses might be able to cause disease in their normal hosts. MCF-like disease was reported in domesticated sheep that were infected experimentally with high doses of OvHV-2. Rare clinical cases have been tentatively attributed to OvHV-2 in domesticated goats, and in Stone's sheep (*Ovis dalli stonei*) and Barbary sheep (*Ammotragus lervia*) at a zoo.

Zoonotic potential

There is no evidence that any of the MCF viruses can infect humans.

Geographic Distribution

MCF viruses can be found worldwide, but illness occurs only where a carrier species can pass a virus to susceptible hosts. AIHV-1 associated disease is mainly seen in sub-Saharan Africa, in areas where wildebeest are

present. This virus is reported to be the most important MCF virus in some parts of Africa, although OvHV-2 associated disease also occurs.

OvHV-2 is the major cause of MCF in domesticated animals outside Africa. Sheep-associated MCF is common among Bali cattle in Indonesia; however, cases in cattle are infrequent in countries where *Bos taurus* and *Bos indicus* are the predominant species. OvHV-2 is a serious concern in countries with bison and cervid farms, as these species are very susceptible. Infections in pigs were, at one time, reported mainly from Norway, but sick pigs have now been found in other European countries and North America, and are likely to occur elsewhere.

OvHV-2 is reported to be the major cause of MCF among ruminants in zoos and wildlife parks. Several other MCF viruses, including AIHV-1, MCFV-ibex and other viruses carried in exotic species can also cause disease in these locations.

Transmission

MCF viruses, like other herpesviruses, establish lifelong, latent infections. In wildebeest, AIHV-1 occurs in both cell-free and cell-associated forms. These animals shed cell-free virus in nasal and ocular secretions for a short period after they become infected. After this time, the virus occurs mainly in the cell-associated form, which is transmitted only rarely to other animals. (However, cell-free virus can be isolated from the nasal secretions of some animals that are stressed or given corticosteroids.) AIHV-1 is spread mainly by wildebeest calves, which can become infected *in utero*, by direct contact with other wildebeest, or in aerosols during close contact. Virus shedding is most intense during the first 3-4 months of life, and most calves are thought to become infected from their cohorts. Contamination of pastures may also contribute to transmission. Neutralizing antibodies usually develop by approximately 3 months of age, and shedding declines after this time. After the age of six months, wildebeest shed little virus except when they are stressed or during parturition.

Most cases of wildebeest-associated MCF are seen when susceptible animals are exposed to parturient wildebeest or young calves. This usually occurs after close contact, but transmission has been reported when the animals were separated by at least 100 meters. Inhalation is thought to be the primary means of transmission for all MCF viruses, although ingestion might also be possible. Cell-associated AIHV-1 is very fragile, and infectivity disappears after 72 hours in the environment. Cell-free virus has been reported to survive for more than 13 days in humid environments. MCF viruses are inactivated quickly by sunlight.

Like AIHV-1, OvHV-2 seems to be excreted mainly in nasal and ocular secretions. The virus is shed intermittently for short periods, typically lasting less than 24 hours. While sheep of all ages may transmit OvHV-2, one study found that the highest virus levels and most frequent shedding occurred in 6-9 month old lambs. Most lambs in naturally

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infected flocks do not seem to become infected until they are at least two months of age. Circumstantial evidence (antibodies to MCF viruses in a survey of some gnotobiotic and specific pathogen-free sheep), and the identification of virus-infected cells in colostrum and milk, suggest that vertical transmission of OvHV-2 is possible. However, transmission by these routes seems to be uncommon. Viral DNA has also been reported in the semen of rams.

Cases of sheep-associated-MCF increase during the spring lambing season. Because there are no reports that sheep shed more virus during parturition, and shedding does not seem to be common in very young lambs, some authors suggest that this phenomenon might be caused by improved virus survival at cool temperatures, or seasonal variations in stock densities. Other sources suggest that the dynamics of infection might vary between flocks. As OvHV-2 has never been cultured, its survival in the environment is unknown. In general, enveloped viruses such as herpesviruses survive better under moist conditions.

Susceptible animals usually become infected with OvHV-2 when they are in close contact with sheep, but cases have been reported when sheep and cattle were separated by 70 meters. One outbreak in bison occurred in herds up to 5 km from a lamb feedlot.

CpHV-2 transmission in goats seems to resemble OvHV-2 transmission in sheep. In one infected goat herd, 94% of the goats became infected by the age of 10 months. In this herd, CpHV-2 DNA was first detected in goat kids at 3 months of age, approximately 50% had seroconverted by 7 months of age, and more than 80% by 9 months. There was no evidence of infection at birth, and no goats became infected if they were removed from the infected herd at one week of age. Adult goats also became infected readily when they were exposed to a CpHV-2 infected herd.

Ruminants that develop malignant catarrhal fever are usually dead end hosts. A few instances of animal-to-animal transmission have been suspected in cattle, pigs and OvHV-2 infected deer, although there is no definitive evidence that this is possible. In pigs, large amounts of OvHV-2 DNA have been found in the semen of asymptomatic boars and in the nasal mucosa and skin of sick animals. Case reports have also suggested that some non-reservoir hosts might be able to transmit MCF viruses to their offspring. One recent study suggested that horizontal transmission does not occur between bison.

Subclinical infections with OvHV-2 have been reported in some incidental hosts including cattle, bison, cervids and pigs. Recrudescence might be possible in these animals, although it seems to be uncommon even in animals that are stressed.

Disinfection

Many common disinfectants can inactivate MCF viruses. If heavy organic debris is present, the World

Organization for Animal Health (OIE) recommends 3% sodium hypochlorite.

Incubation Period

The incubation period varies with the virus, host and other factors, and is incompletely understood. Cattle became sick 11-34 days after inoculation with AIVH-1, and 11-73 days after the administration of blood from OvHV-2 infected, sick cattle. In bison exposed to sheep, the incubation period is often a month or more, with peak losses reported to occur 40-70 days after exposure. The incubation period was 2-6 weeks in pregnant bongos affected by ibex-associated MCF in a zoo, and 21 to 40 days in one-week-old piglets inoculated intranasally with OvHV-2.

Latent infections and recrudescence might also be possible. Epidemiological evidence suggests that some cattle may be infected subclinically for more than 20 months before developing disease. In one outbreak in subclinically infected bison, clinical cases tended to occur 3-14 days after the animals were stressed by handling.

Clinical Signs

Malignant catarrhal fever can present with a variety of clinical sign in different species, depending on their susceptibility to the virus. Subclinical infections are usual in the reservoir hosts. Asymptomatic infections have also been reported in some incidental hosts such as cattle, bison, deer and pigs.

Almost any organ can be affected in clinical cases, although the gastrointestinal tract, eye and central nervous systems are often involved in domesticated ruminants. Peracute disease, which tends to occur in highly susceptible species, progresses very rapidly, with few clinical signs before death. In some animals, death may be preceded by 12-24 hours of depression, weakness, diarrhea or dysentery. Clinical signs are more apparent in animals that survive longer, such as cattle, which may be ill for a week or more before dying. In addition to high fever and inappetence, cattle often have bilateral corneal opacity, beginning at the corneoscleral junction and progressing inward. Serous oculonasal discharge is common early; later, this discharge becomes mucopurulent. The muzzle and nares are usually encrusted, and dyspnea, open-mouthed breathing and salivation may be seen. The oral mucosa is often hyperemic, and may contain multifocal or diffuse areas of necrosis. Erosions may be found at the tips of the buccal papillae. The superficial lymph nodes are often markedly enlarged in cattle. The skin is sometimes erythematous or ulcerated, and hardened scabs may develop, particularly on the perineum, udder and teats. In some animals, the horn and hoof coverings may be loosened or sloughed. The joints may be swollen, and milk production often drops. Diarrhea, hemorrhagic gastroenteritis or hematuria may also be seen, although these signs are less common than in bison and deer. Occasionally, animals develop neurological signs,

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especially in the terminal stages. Although many or most animals die, chronic infection or recovery is possible. Persistent eye lesions may be seen in surviving cattle. Skin lesions have been reported in cattle without other clinical signs, and may resolve spontaneously.

The clinical signs are similar in other species, but often vary in some aspects. For example, corneal opacity is reported to be inconsistent in water buffalo with MCF, although mild to moderate conjunctivitis seems to be common. Bison often die rapidly, without developing purulent rhinitis or keratoconjunctivitis. Anorexia, depression, corneal opacity, lacrimation, mild serous nasal and ocular discharge, coughing, salivation, diarrhea and neurological signs may be seen in some cases, but in many bison herds, the symptoms are subtle. Hematuria and hemorrhagic enteritis are more common than in cattle, but lymphadenomegaly is usually minimal. In the last stage of the disease, bison often develop an edematous band at the corneoscleral junction; this lesion is difficult to recognize in live animals. Bison attempt to mask the clinical signs until they are near death. Inhalation pneumonia is common in the last stage of the disease, and some sick bison may be attacked by herd mates, resulting in trauma. Recumbent animals generally die within a few hours.

In deer, malignant catarrhal fever is often peracute, with death within a few days, and the characteristic signs may not be seen. In other outbreaks or in more resistant species, more typical MCF symptoms including depression, loss of condition, a rough hair coat, nasal discharge, corneal opacity, transient loose stools, hemorrhagic diarrhea and bloody urine have been reported, with some animals surviving for up to three weeks after the onset of disease. Unusually, skin lesions were the primary complaint in some deer infected with CpHV-2. In white-tailed deer infected with this virus, the major lesions were widespread alopecia; thickening, crusting, hyperkeratosis, and focal ulceration of the skin; weight loss; and impaired vision. The hoof walls were shed in some animals. Similarly, sika deer infected with CpHV-2 developed skin lesions including extensive alopecia, as well as weight loss and diarrhea. One sika deer had seizures but no other clinical signs before it was euthanized. Neurological signs including abnormal behavior, apathy and incoordination have been reported in wild moose and roe deer.

Most OvHV-2 infections reported in pigs have been acute or peracute, with high fever and dyspnea the most consistent signs. Subclinical infections and chronic cases lasting for several weeks have also been reported. In some but not all outbreaks, pigs developed crusting rhinitis or foul-smelling nasal discharge, erosions on the nasal and oral mucosa, corneal edema or uveitis. Other reported signs were hematuria, reproductive losses (abortions, stillbirths and smaller-than-normal litters) reddened foci on the skin and neurological signs. In some outbreaks, only pregnant animals were affected. One-week old, experimentally infected piglets developed fever, apathy, anorexia, adipisia,

skin rashes and seizures, but there was no evidence of nasal or ocular discharge, corneal opacity or diarrhea. However, these piglets may have been euthanized before some clinical signs developed. Although OvHV-2 infections in pigs are often fatal, some pigs have recovered.

Sporadic cases have been documented in other species. Possible MCF was reported in an OvHV-2 infected alpaca with apathy, dehydration, anorexia and emaciation. In a zoo, 3 periparturient bongo antelope with MCF became acutely inappetent and developed respiratory distress, dying within 24–72 hours. Illnesses attributed to possible MCF in Stone's sheep (*Ovis dalli stonei*) in a zoo were sudden death in one animal, and neurological signs including hind limb weakness, unusual aggression to humans and seizures in another. Three goats infected with OvHV-2 developed a fever and neurological signs, and one animal had diarrhea and bilateral corneal opacity. Unusually, PCR evidence of OvHV-2 infections was reported in a sick, 6-month-old foal and asymptomatic adult horses on a farm in Brazil. The foal had neurological signs and severe dyspnea, which progressed rapidly to death.

Post Mortem Lesions [Click to view images](#)

Malignant catarrhal fever is characterized by inflammation and epithelial necrosis, with lymphoproliferation, infiltration of nonlymphoid tissues by lymphoid cells, and vasculitis.

The extent of the lesions varies with the severity and course of the disease. In cattle that die suddenly, there may be few abnormalities other than hemorrhagic enterocolitis. In less acute cases, the carcass may be dehydrated, emaciated or normal. Diffuse or focal bilateral corneal opacity is common, and corneal ulcers are sometimes present. The muzzle is often raw and encrusted with a serous, mucopurulent or purulent nasal discharge. Hyperemia, edema and small focal erosions or ulcers may be found on the nasal mucosa. Dermatitis and skin ulcers may be found in some animals. The lymph nodes are usually markedly enlarged in cattle, although the degree of involvement varies. On cut surface, they may be firm and white, hemorrhagic or necrotic. Petechiae or ecchymoses may be present on various serosal surfaces. Prominent raised white foci, 1-5 mm in diameter, may be seen in some tissues, particularly the kidney. These nodules are sometimes surrounded by a thin hemorrhagic zone. The gastrointestinal tract can contain erosions and hemorrhages; in severe cases, the intestinal contents may be hemorrhagic. However, lesions in the gastrointestinal tract may be difficult to identify, especially when the carcass is autolyzed. The upper respiratory tract often has catarrhal exudates and erosions, and a diphtheritic membrane may be present. Ecchymotic hemorrhages, hyperemia and edema are common in the mucosa of the urinary bladder. In more chronic cases, the small arteries in multiple organs can be very prominent and tortuous, with thickened walls.

Similar lesions have been reported in other species, but some species-specific differences have been noted. In

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bison, vasculitis tends to be milder than in cattle and the lymph nodes are less likely to be markedly swollen, but hemorrhagic cystitis and hemorrhagic colitis are more common. Bison that die with few clinical signs may have advanced lesions on necropsy. Unique features of MCF in 3 periparturient bongos included necrotizing cholangiohepatitis and neutrophilic, necrotizing myocarditis, together with more characteristic lesions. Severe perirenal hemorrhage and multiple renal infarcts were seen in Stone's sheep thought to have MCF, together with petechial hemorrhages in multiple tissues, and serosanguineous to fibrinous effusion in some body cavities.

Diagnostic Tests

Sick animals

Malignant catarrhal fever is often suspected based on microscopic lesions in tissues collected at necropsy. Because some MCF viruses cannot be isolated from infected animals, polymerase chain reaction (PCR) assays are often the method of choice for confirmation. Lymphoid tissues such as spleen and lymph node are optimal for this test, but other tissues can also be used. Most PCR tests detect AIHV-1 and/or OvHV-2, but some can also detect other MCF viruses. Epidemiological information (e.g., contact with sheep but not wildebeest) should guide the choice of PCR assay.

Infections with viruses in the Alcelaphinae/Hippotraginae group, such as AIHV-1, can also be diagnosed by isolating this virus from the blood of live animals, or from the lymph nodes, spleen and other affected tissues at necropsy. AIHV-1 is inactivated quickly in dead animals, and samples should be taken as soon as possible. The most useful samples are collected immediately after euthanasia of a dying animal. The viability of the host cells must be maintained after sample collection, as the virus cannot be recovered from dead cells. AIHV-1 can be isolated in bovine thyroid cells or other susceptible cell lines, and can be identified by immunofluorescence or immunocytochemistry. OvHV-2 and CpHV-2 cannot be isolated in cell culture.

Serological tests for MCF viruses include virus neutralization (VN), immunoblotting, enzyme-linked immunosorbent assays (ELISAs) and immunofluorescence or immunoperoxidase tests. All of these assays are based on antigens from alcelaphine herpesviruses (mainly AIHV-1), which can be propagated in cell culture. While they also detect antibodies to other MCF viruses (e.g., OvHV-2 and CpHV-2), such tests cannot distinguish reactions to different viruses. Most of the serological tests can be used in sick animals; however, the VN test cannot be employed, as these animals do not usually develop neutralizing antibodies. Cross-reactions with other herpesviruses (e.g., bovine herpesvirus-4) are possible, especially in tests that use polyclonal antibodies. Most, but not all, sick cattle and bison are seropositive. Because healthy animals can also have antibodies to MCF viruses, serology should be used in conjunction with histopathology and clinical findings.

It is difficult to substantiate a diagnosis of MCF in the reservoir host for a virus. Critical components include histopathology and the elimination of other possible causes of the illness.

Reservoir hosts

PCR can be used to document the absence of infection in some reservoir hosts (for example, when producing OvHV-2 free sheep). Highly sensitive PCR assays should be employed. The peripheral blood leukocytes of most infected sheep contain sufficient OvHV-2 DNA to be detected by PCR, but this is not the case in infected oryx or black wildebeest. PCR was reported to be positive in 85% of goats with antibodies to CpHV-2.

Serology can also be used to identify infected reservoir hosts. Seroconversion may take more than 4 weeks in animals that received a low dose of virus. Maternal antibodies can be a problem in lambs under 4 months of age. All of the serological tests, including VN, can be used in wildebeest. VN is not useful in sheep and goats infected with OvHV-2, CpHV-1 or related viruses, as they have low or no titers of neutralizing antibodies to AIHV-1.

In wildebeest calves, cell-free virus can be found in nasal secretions for a short period after infection. The virus can also be isolated from peripheral blood leukocytes at this time. It is less likely to be successful in older wildebeest, except when they are immunosuppressed (e.g., by stress or drug treatment). Cell-associated AIHV-1 can be isolated by establishing cultures of tissues from wildebeest.

Subclinically infected, susceptible species

The levels of OvHV-2 DNA are very low in subclinically infected bison and cattle, and may not be detected readily by PCR. However, such infections are not expected to be clinically significant, as these animals are expected to be dead end hosts for the virus. In surveillance, such animals can be detected more readily by serology than PCR.

Treatment

No specific antiviral therapy is available. Antibiotics to control secondary infections and supportive therapy may occasionally help, but many affected animals die.

Control

Disease reporting

Veterinarians who encounter or suspect malignant catarrhal fever should follow their national and/or local guidelines for disease reporting. In the U.S., malignant catarrhal fever is a reportable disease in many states. State authorities should be consulted for more specific information.

Prevention

Malignant catarrhal fever can be prevented by separating susceptible animals from sheep, goats, wildebeest or other suspected reservoir hosts. Wildebeest seem to transmit AIHV-1 readily, and should always be

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Morbidity and Mortality

separated from cattle. Susceptible animals should not be allowed on pastures where these animals have recently grazed, particularly near the time wildebeest calve. To prevent sheep-associated MCF, it is important to separate sheep from bison, some deer and other highly susceptible species. Although this form of MCF can also occur in European breeds of cattle, cases are uncommon, and separation of cattle and sheep is not always necessary. Some flocks of sheep have been associated with recurring transmission to cattle, lasting for years, and separation or culling of such flocks should be considered.

In zoos and wild animal parks, control is complicated by the number of potentially susceptible animal species and MCF virus carriers, which are often poorly characterized. In addition to OvHV-2 and AIHV-1, there have been at least two incidents involving transmission from Nubian ibex to bongo. Increased susceptibility (e.g., during pregnancy) or increased virus shedding (e.g., recently acquired animals stressed by a new environment) might be factors in some outbreaks.

The minimum distance needed for separation is unknown. Although most infections occur when the carrier host and susceptible animals are in close contact, transmission of AIHV-1 has been reported when animals were separated by a distance of at least 100 meters. OvHV-2 was reported in cattle separated from lambs by 70 meters. This disease was also reported in bison herds up to 5 km from a lamb feedlot. Separation by longer distances is likely to be more important when the host is highly susceptible and the concentration of virus is high. Transmission on fomites must also be avoided, particularly when the species is highly susceptible. In some cases, the production of virus-free hosts may also be considered. OvHV-2 free domesticated sheep and mouflon have been produced by early weaning, isolation and testing for virus. CpHV-2-free goats were produced by separating them from the infected herd at 7-10 days of age. One outbreak at a zoo was successfully controlled by the removal of infected sheep species, followed by the reintroduction of virus-free mouflon sheep derived from the infected flock. Commercial vaccines are not currently available for any species.

During outbreaks, susceptible animals should be separated immediately from the suspected source of the virus to prevent further cases. Although there is some uncertainty about the possibility of horizontal transmission in some species, most incidental hosts are thought to be dead end hosts. The current consensus is that sick animals they do not need to be culled or separated from other susceptible animals. Because the incubation period can be very long, cases can continue to occur for months, even after the animals have been separated from the source of virus. Stress reduction can help prevent disease in subclinically or mildly affected animals.

AIHV-1, OvHV-2 and CpHV-2 seem to be carried in many, most or all individuals of the reservoir species, and the virus spreads readily within infected herds. In wild wildebeest, an intense epizootic occurs during the perinatal period, and all calves are thought to become infected with AIHV-1 before they reach 6 months of age. Uninfected wildebeest are thought to be rare in zoos, if they exist. Similarly, most sheep are infected with OvHV-2, although infection usually seems to occur later than in wildebeest, and OvHV-2 free herds have been created by early weaning. Most goats in CpHV-2 infected herds are thought to carry this virus by the age of 10 months. The prevalence of MCF viruses in wild sheep and goats is poorly understood, and may vary with the species or population. For example, antibodies to MCFV were found in 95% of a group of wild Dall's sheep in Alaska. but one population of bighorn sheep (*Ovis canadensis*) had no evidence of infection. Illness in reservoir hosts is thought to be possible, but very rare.

In MCF-susceptible species, AIHV-1 infections are often associated with exposure to wildebeest herds around the time of calving. Wildebeest-associated MCF is uncommon when all wildebeest in the herd are older than 6 months. Outbreaks of sheep-associated MCF (OvHV-2) have been seen most often during lambing season, although the reason is still unclear. Certain sheep flocks may continue to infect other animals for years. However, cases of MCF can be unpredictable, and sometimes develop in animals that were exposed to carriers without incident for years. The conditions leading to such outbreaks are often speculative. Stress might increase virus shedding, and environmental conditions such as high humidity might increase virus survival. In one outbreak, concentration of the virus by a barn fan was suggested as a possible contributing factor. Pregnancy might increase animals' susceptibility. In some pig herds, most or all of the affected animals were pregnant. Similarly, MCF occurred in 3 periparturient bongo antelope exposed to a healthy adult male Nubian ibex, but neither a bongo calf exposed to this ibex, nor male bongo exposed to other Nubian ibexes became ill.

In Africa, morbidity from AIHV-1 is approximately 6-7% in most cattle herds, although it can be as high as 50%. In European breeds of cattle, sheep-associated MCF usually occurs sporadically in only one to a few animals in the herd. The morbidity rate from this form is usually less than 1%. A few outbreaks affecting 16-50% of the herd have been reported, but this is unusual. In highly susceptible species such as farmed cervids and bison, both the number of affected animals and the case fatality rate are higher. Morbidity rates as high as 50-100% have been reported in bison, in outbreaks where there is close contact with sheep. The case fatality rate from MCF viruses is 80-90% in symptomatic cattle, and approaches 100% in symptomatic bison, deer and water buffalo. Residual corneal opacity is

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often seen in recovered cattle, but complete recovery is also possible.

Healthy animals, including those from MCF-susceptible species, may have antibodies to MCF viruses. Subclinical infections have been reported in bison, deer, cattle, pigs, wild cervids and other species. In one study, 24% of healthy bison had antibodies to MCF viruses, and OvHV-2 DNA was found in the blood of 11% of the seropositive bison. Genetic susceptibility to illness has been identified in bison. On Norwegian farms where outbreaks had occurred previously in cattle and/or swine, antibodies to OvHV-2 were found in 25% of the cattle and 43% of the pigs. Up to 35% of some wild cervid populations are also reported to have antibodies to MCF viruses.

Internet Resources

The Merck Veterinary Manual

<http://www.merckvetmanual.com/>

World Organization for Animal Health (OIE)

<http://www.oie.int>

OIE Terrestrial Animal Health Code

<http://www.oie.int/international-standard-setting/terrestrial-code/access-online/>

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

<http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/>

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