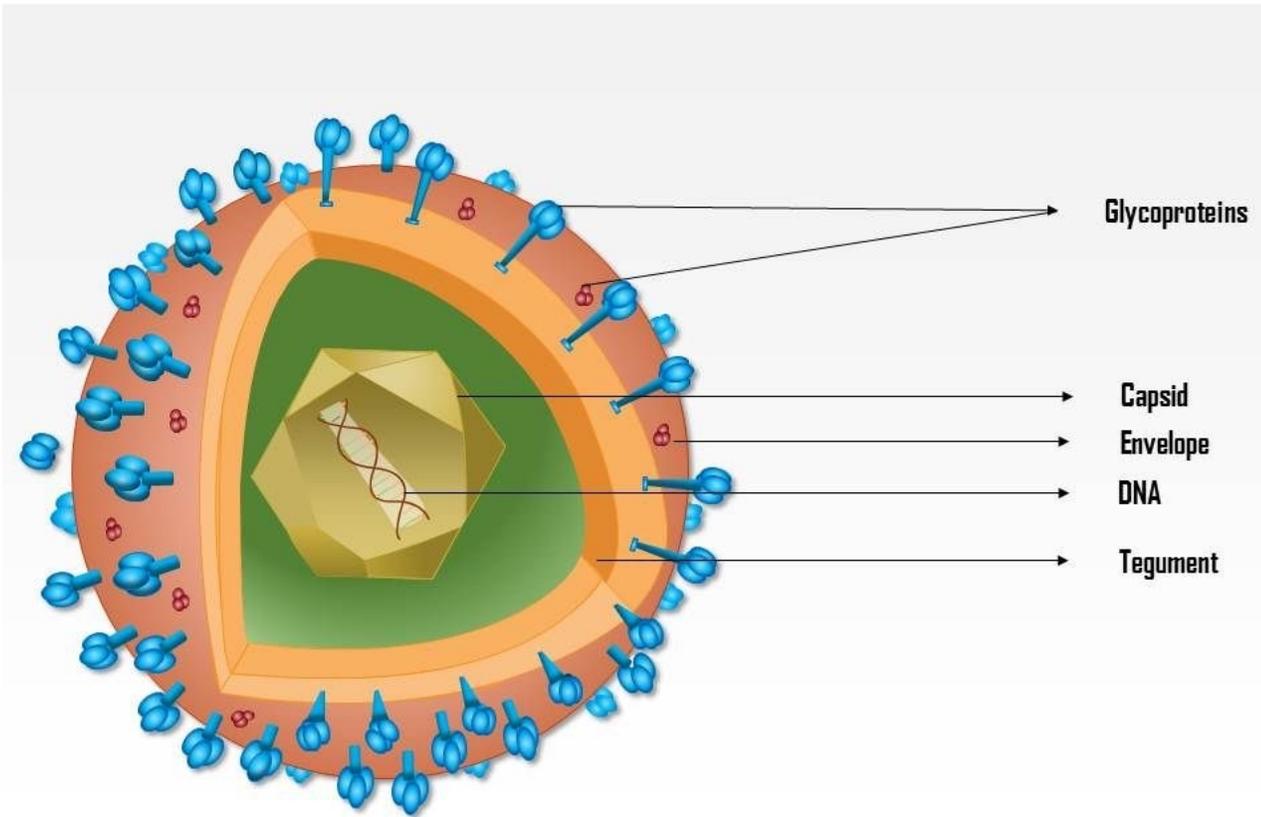
An electron micrograph showing several herpesviruses. Two prominent, spherical, reddish-brown particles with dark, electron-dense cores are visible in the upper center. The background consists of a complex network of brown, granular material, likely cellular debris or membranes, with some larger, irregularly shaped structures. The overall appearance is that of a biological specimen prepared for electron microscopy.

HERPESVIRIDAE FAMILY

Bovine, Equine

Structure of Herpes-Virus

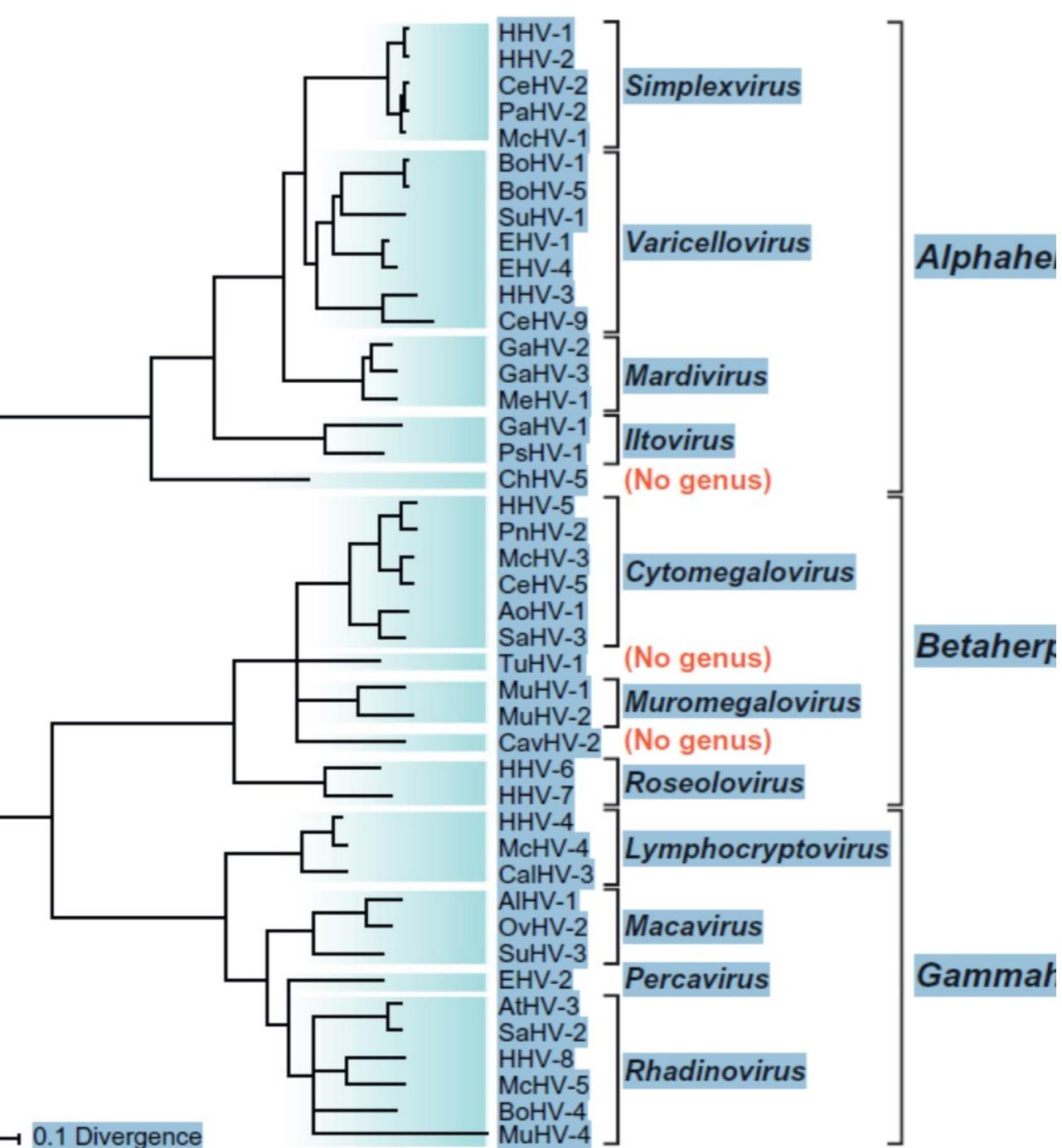


- Double stranded DNA
- Icosahedral
- Enveloped



LATENCY ABILITY

- Herpeviruses become latent in recovered animals. Reactivation during stressful situations induces disease or tumors.



Herpesviridae

- Subfamily
- Alphaherpesvirinae
- Betaherpesvirinae
- Gammaherpesvirinae

ALPHAHERPESVIRINAE

Bovine
Herpes Virus
Type 1,2,5

Equine
Herpes Virus
Type 1,3,4

Caprine
Herpes Virus
Type 1

Canine
Herpes Virus
Type 1

Feline Herpes
Virus Type 1

Porcine
Herpes Virus
Type 1

BETAHERPESVIRINAE

- Viruses that cause infection in humans are included in this subgroup. In this grouping, only SHV2, which causes rhinitis in pigs, is present.

GAMMAHERPESVIRINAE

Cattle;

Bovine Herpes Virus
Type 4 and
Alcelaphine
herpesvirus (BHV-3)

Horses;

Equine Herpes Virus
Type 2 and 5

Sheep;

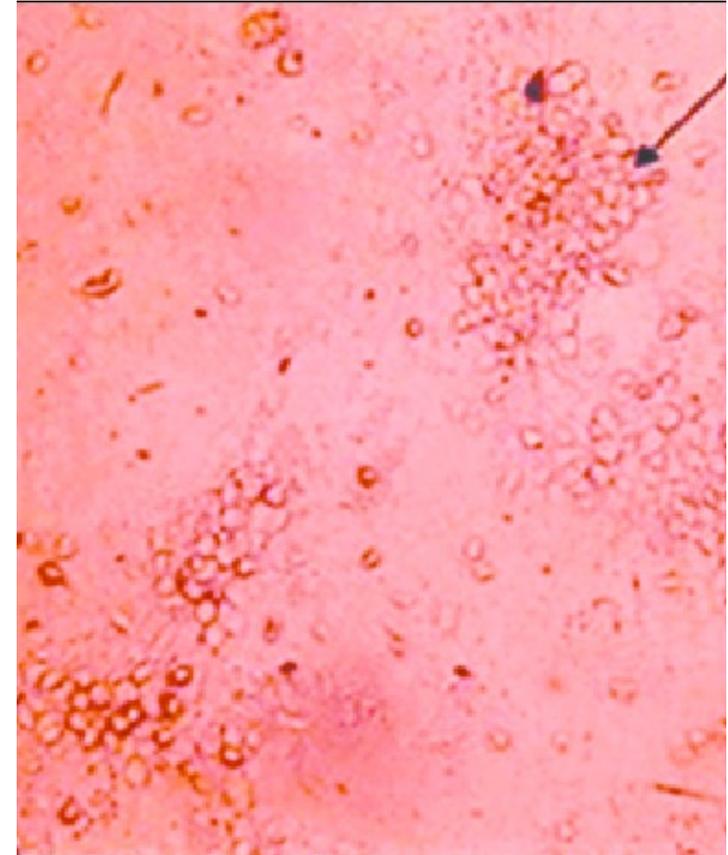
Ovine Herpes Virus
Type 1 and 2

Goats;

Caprine Herpes
Virus Type 2

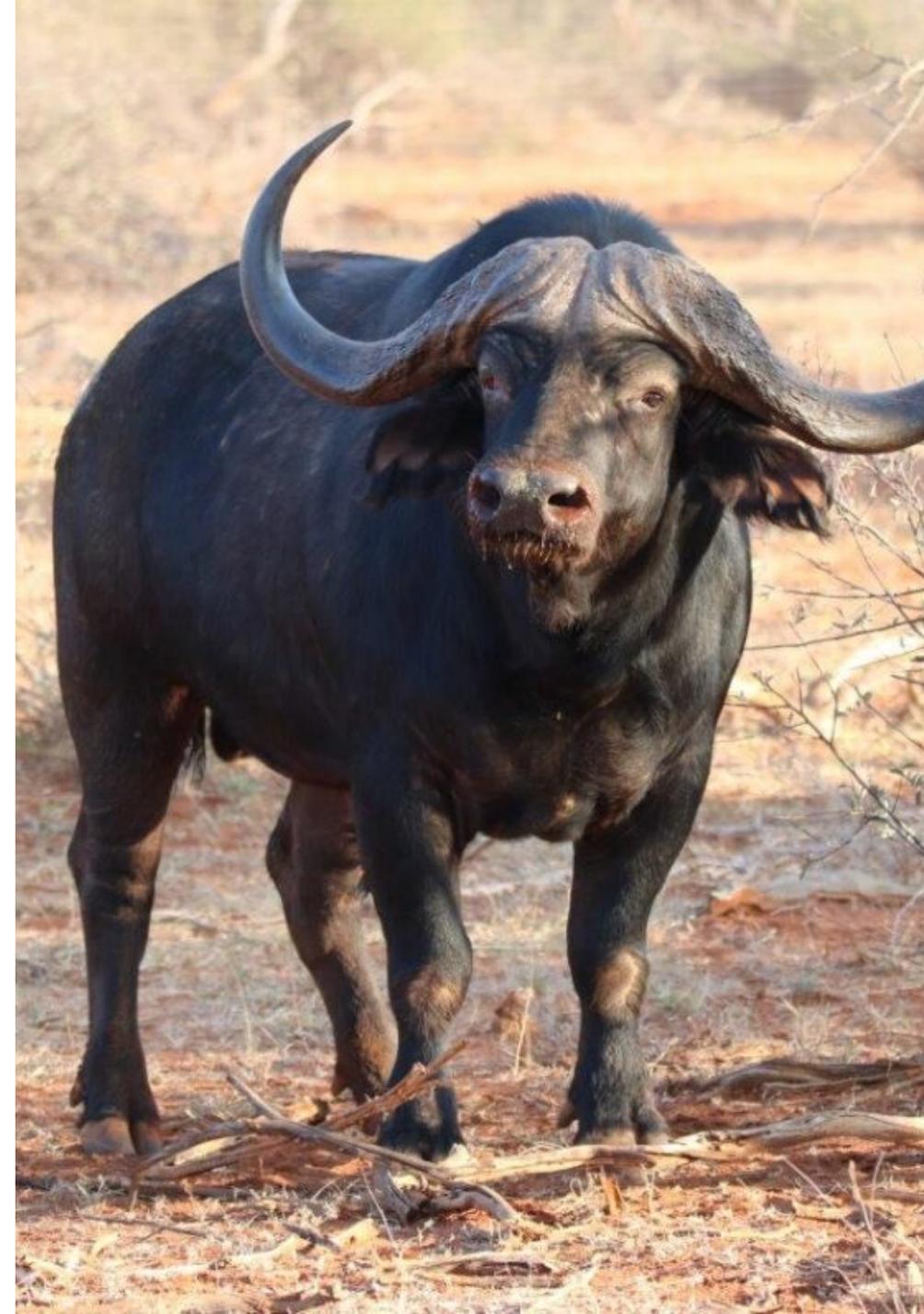
BOVINE HERPESVIRUS 4

- Gamaherpesvirinae
- dsDNA
- Bovine herpesvirus 4 (BHV-4) has been called 'passenger' virus because it causes mainly subclinical disease.
- BHV-4 infection is associated with cattle diseases of the genital tract, such as vulvovaginitis, post-partum metritis and abortion.



SUSCEPTIBLE SPECIES

- Ruminants, American bison, African buffalo, sheep.
- BHV-4 has also been isolated from lions and from cats.





TRANSMISSION

- Direct or Indirect.
- Indirectly, through contaminated objects primarily comprising respiratory or vaginal secretions.
- The respiratory route is the main route of transmission of the virus.



SYSTEMS AFFECTED

- mammary gland diseases of large ruminants
- reproductive diseases of large ruminants
- reproductive diseases of small ruminants

Pathogenesis

The virus replicates within mucosal cells and then invades the mononuclear cells, causing generalised infection and crossing the placenta to infect the unborn fetus.

Like other herpesviruses, BHV-4 can undergo latency, often residing in the trigeminal ganglia similarly to BHV-1 (IBR).

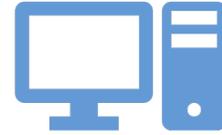
Recovered cattle often become latent carriers. Both acute and latent infections are highly prevalent in endemic BHV-4 areas.

Clinical Signs

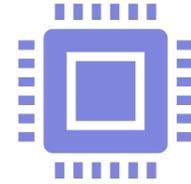
Abortion and births of weak or dead lambs, calves and kids.

In dairy cattle, BHV-4 can cause mastitis and associated udder lesions and milk changes.

Diagnosis



Virus Isolation



PCR.



Antibodies to BHV-4 can be detected using ELISA and Indirect Immunofluorescence.

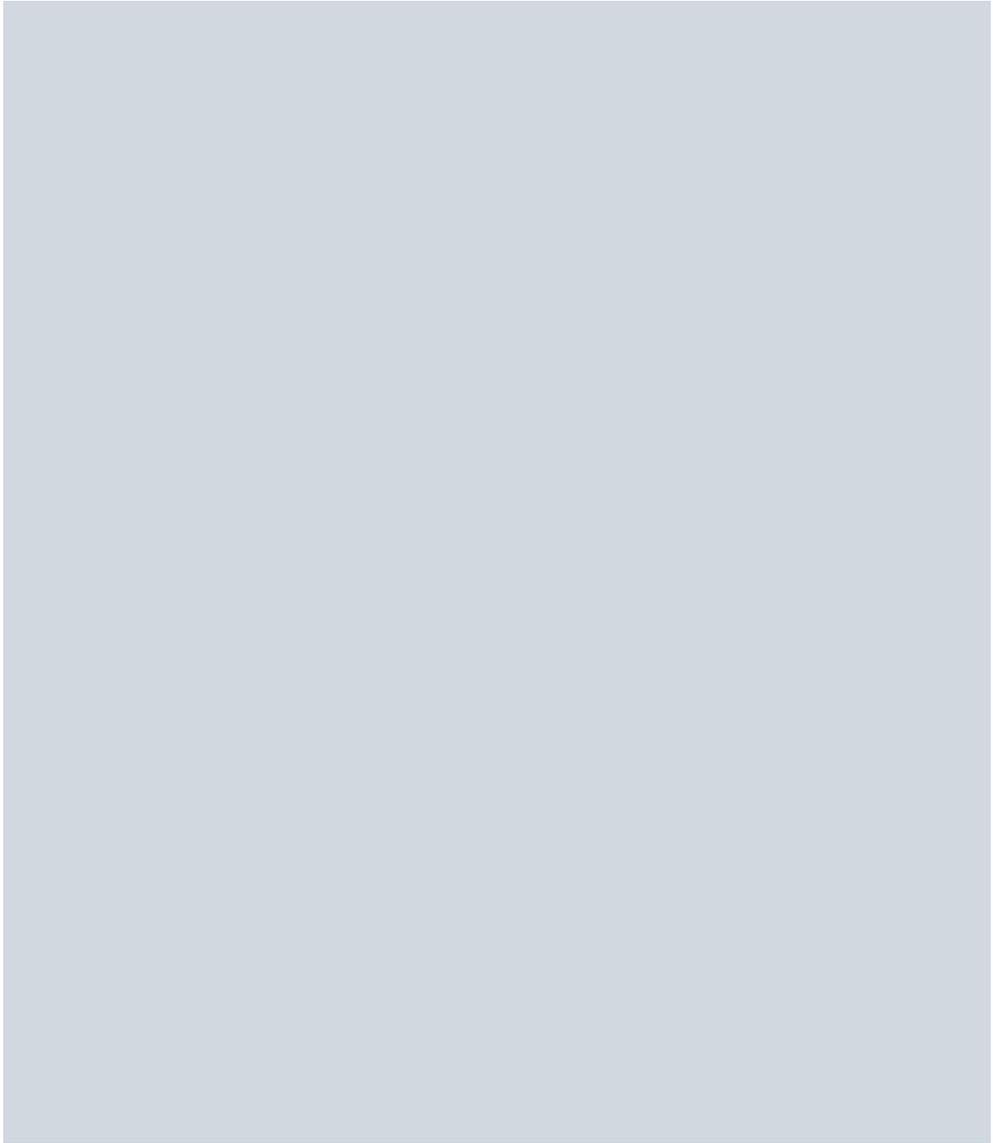
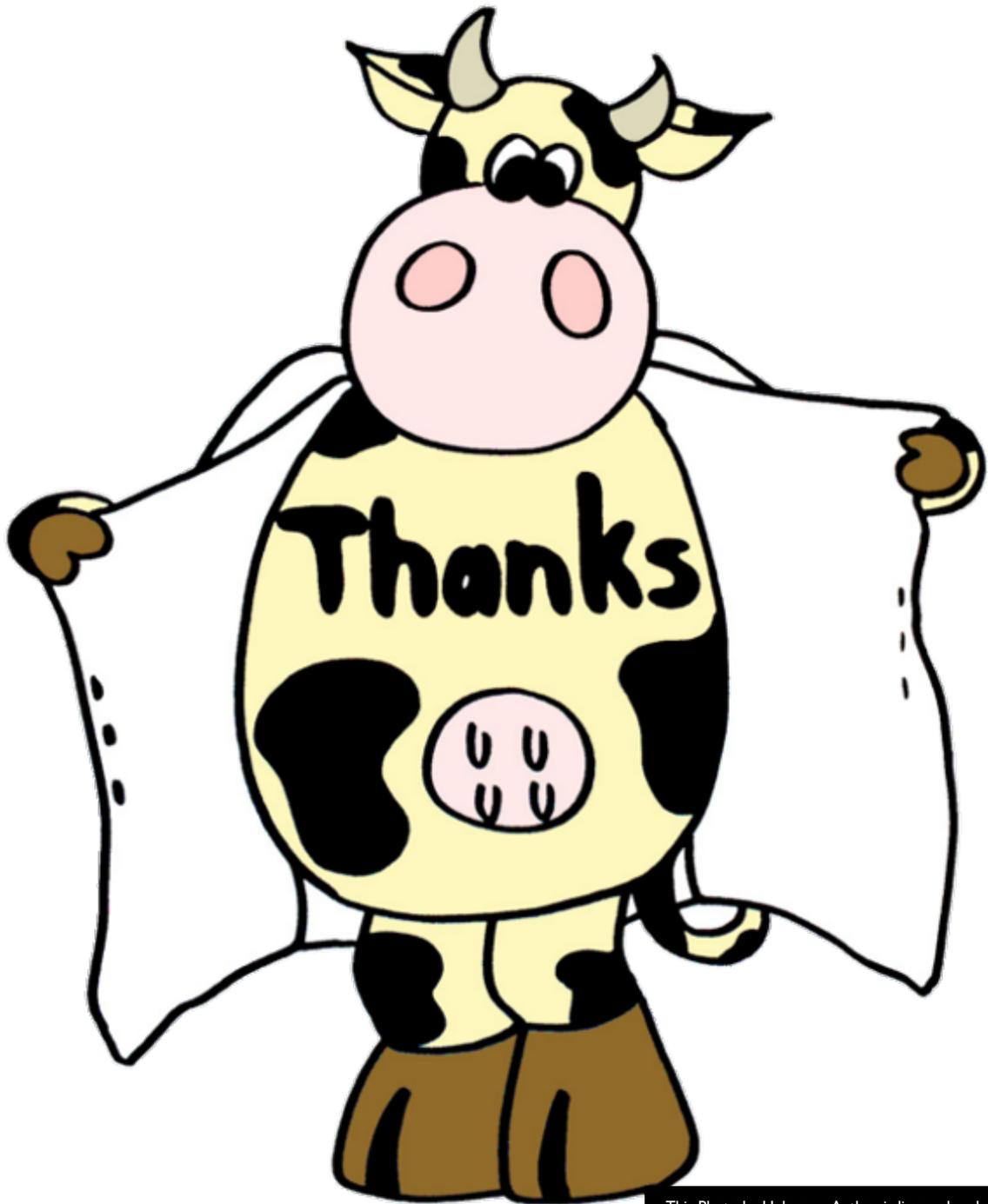


Immunoperoxidase assays are also available.



Prevention

- Hygienic measures
- Seropositive animals should be removed.
- Infected cows that calve should be isolated.





CORYZA GANGRENOSA
BOVUM

- Malignant Catarrhal
Fever

ETIOLOGY

Gammaherpesviruses
of
the *Macavirus* genus.

MCFV, contains at
least 10 members, five
of which are currently
known to cause
disease.

MALIGNANT
CATARRHAL
FEVER (MCF)
IS AN
INFECTIOUS
SYSTEMIC
DISEASE



- Affecting mainly ruminants
- The disease in cattle is mostly sporadic

Wildebeest-associated MCF:

Sheep-associated MCF:

Caprine-associated MCF:

Unknown origin:

Roan antelope origin:



Wildebeest-associated MCF: alcelaphine herpes virus 1 (AIHV-1). Endemic in wildebeest populations worldwide.

Sheep-associated MCF: ovine herpesvirus 2 (OvHV-2). Endemic in most sheep populations worldwide.

Caprine-associated MCF: caprine herpesvirus 2 (CpHV-2): Endemic in most domesticated goat populations worldwide and causes MCF in cervids.

Unknown origin: causes MCF in white-tailed deer (MCFV-WTD).

Roan antelope origin: Hippotragine herpesvirus-1 was used to experimentally induce MCF in rabbits.

AIHV-1:

- Most cases of wildebeest-associated MCF occur when susceptible animals are exposed to parturient wildebeest or young calves, or pasture contaminated by them.

OvHV-2

- Some of the lambs are infected in utero with most lambs becoming infected perinatally.
- Transmission is only from sheep to susceptible hosts, not horizontally between infected hosts. Aerosol transmission is main route. Close contact with sheep is needed.

CpHV-2



P.I.A.D.C. Cow with MCF

IN SYMPTOMATIC CATTLE, THE CASE FATALITY RATE FROM MCF VIRUSES IS 80-90 PERCENT, AND IN SYMPTOMATIC BISON, DEER, AND WATER BUFFALO, IT REACHES 100 PERCENT.

CLINICAL SIGNS

Peracute form:



Intestinal form:



Cutaneous (Skin) form:



Neurologic form:

CLINICAL SIGNS

Peracute: asymptomatic, or depression followed by diarrhea and dysentery may develop 12–24 hours prior to death

In general: high fever, serous lachrymation and nasal exudate progressing to profuse mucopurulent discharge, inappetance, and decreased milk yields

Skin ulceration and necrosis in the udder and teats.



IN RECOVERED ANIMALS, RESIDUAL CORNEAL
OPACITY IS COMMON, ALTHOUGH TOTAL HEALING
IS ALSO POSSIBLE.

PROGRESSIVE BILATERAL CORNEAL OPACITY,
STARTING AT THE PERIPHERY, IS
CHARACTERISTIC.



Bovine, muzzle. Multiple shallow erosions are filled with dried nasal exudate.



Bovine, muzzle. The muzzle is hyperemic, multifocally covered by adherent mucopurulent exudate, and contains many shallow erosions.



Bovine, oral mucosa. There is a gingival hyperemia and focal erosion



Corneal opacity causes blindness



the surface of the muzzle has been sloughed in this animal



Affected cattle are profoundly depressed with a high fever

FORMS OF MCF

Peracute: asymptomatic, or depression followed by diarrhea and dysentery may develop 12–24 hours prior to death.

Intestinal: Fever, Diarrhea, hemorrhagic gastroenteritis

Cutaneous (Skin) form: Skin lesions (erythema, exudation, cracking, crust formation). Skin ulceration and necrosis may be extensive or restricted to the udder and teats.

Neurologic form: Hyperaesthesia, incoordination, nystagmus and head pressing.



Cow with MCF



**Malignant catarrhal fever
erosions on the tongue**

- High fever, increased serous lachrymation and nasal exudate progressing to profuse mucopurulent discharge, inappetance, and decreased milk yields.
- Eye lesions
- Hemorrhagic enteritis.
- Salivation and oral hyperaemia, erosions of the tongue, hard palate, gums.
- Enlarged lymph nodes, and limb joints may be swollen

POST MORTEM

inflammation and necrosis of respiratory, alimentary, or urinary mucosal epithelium;

subepithelial lymphoid infiltration;

generalized lymphoid proliferation and necrosis;

vasculitis.

Mucosal ulcerations and hemorrhage.

Prominent white nodules representing intramural and perivascular proliferation may be apparent, particularly in the kidneys.



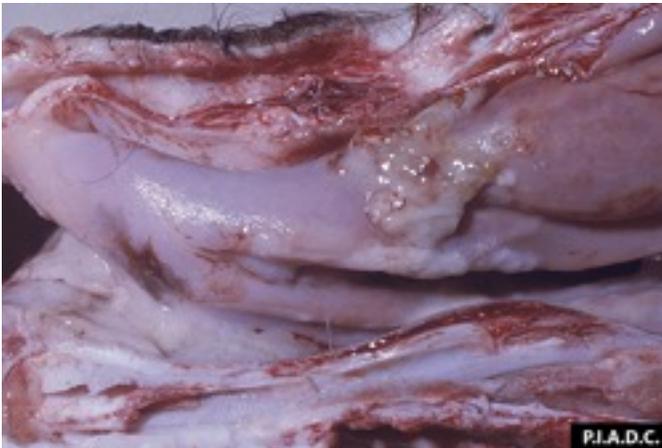
Bovine, hard palate. There are multiple coalescing mucosal erosions.



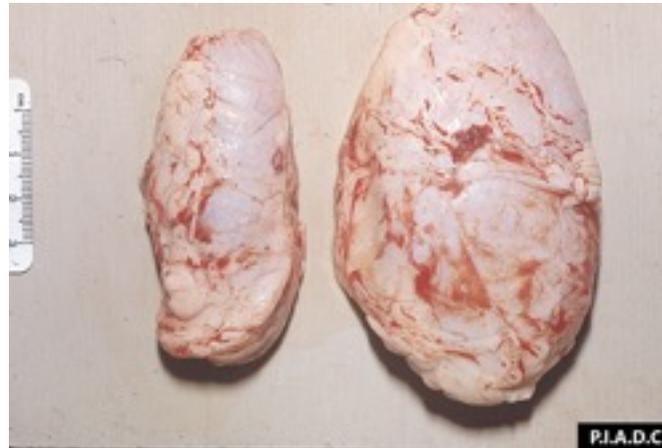
Bovine, skin. There are numerous raised plaques (multifocal dermatitis).



Bovine, head, sagittal section. Mucoid exudate multifocally covers the nasal and pharyngeal mucosa.



Bovine, nasal turbinate. There is a small amount of mucoid exudate.



Bovine, prescapular lymph nodes: Moderately (left) to markedly enlarged (right) due to MCF.



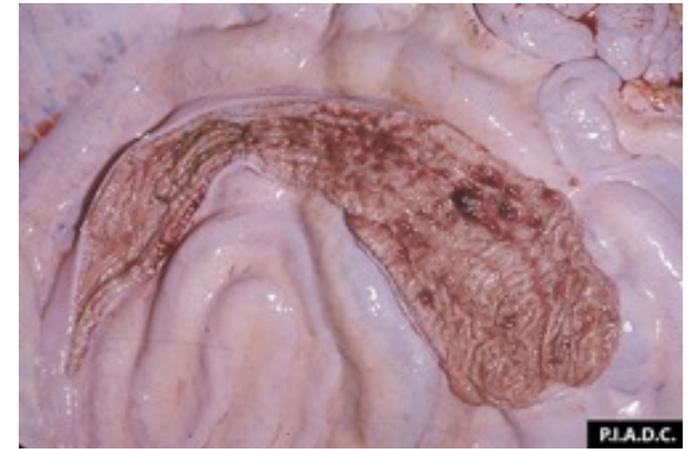
Bovine, prescapular lymph node. There are foci of hemorrhage (and necrosis) in the cortex, and the medulla is edematous.



Bovine, omasum. Omasal leaves contain multiple pale foci of necrosis; on the right there are several ulcers.



Bovine, cecum and ileum. There are scattered small foci of mucosal hemorrhage and erosion.



Bovine, spiral colon. There are multiple mucosal hemorrhages.



Bovine, kidney. Multiple pale foci in the cortex are foci of interstitial nephritis.

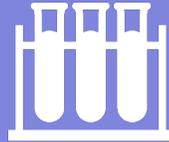


Bovine, colon. There is severe longitudinal linear congestion of the mucosa.

DIAGNOSIS



PCR



Serological tests

Virus neutralisation
(ELISA)
Immunofluorescence



Virus isolation

CONTROL AND PREVENTION



No treatment



No vaccine



Separation of carriers from susceptible species.



When huge numbers of powerful shedders are present, such as in lamb feedlots, distances of more than one kilometer may be required to protect particularly vulnerable animals like bison.



REFERENCES

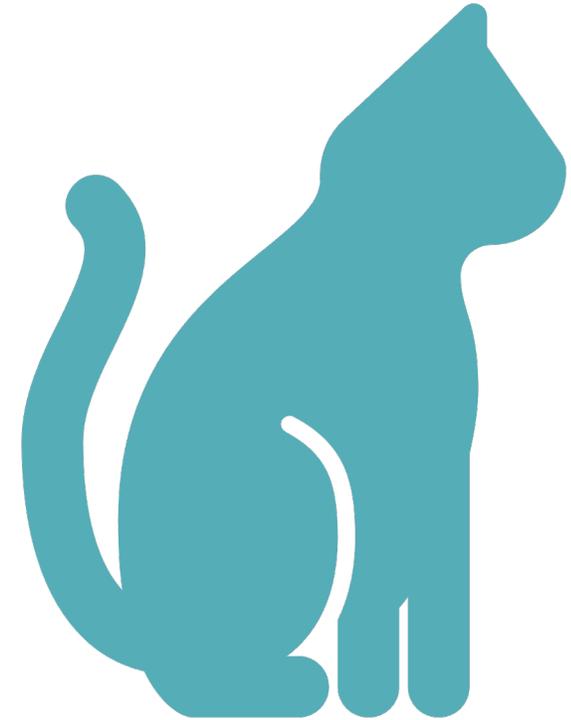
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- <http://www.msdtvetmanual.com/generalized-conditions/malignant-catarrhal-fever/overview-of-malignant-catarrhal-fever>
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FELINE
HERPESVIRUS
1

FELINE VIRAL RHINOTRACHEITIS (FVR) IS AN CATS.

- Feline herpesvirus 1 (FHV-1)
- Ocular and upper respiratory disease in cats and kittens, and a potential cause of eosinophilic dermatitis.



TRANSMISSION

contact with discharge from an infected cat's eyes, mouth or nose.

litter boxes, food and water

An infected pregnant cat might also pass the virus on to kittens in utero.

Some cats who become infected with feline herpes are latent carriers.

CLINICAL SIGNS

Watery or mucopurulent nasal discharge

Spasm of the eyelid muscle resulting in closure of the eye
(blepharospasm)

Eye discharge

conjunctivitis

Keratitis

Fever

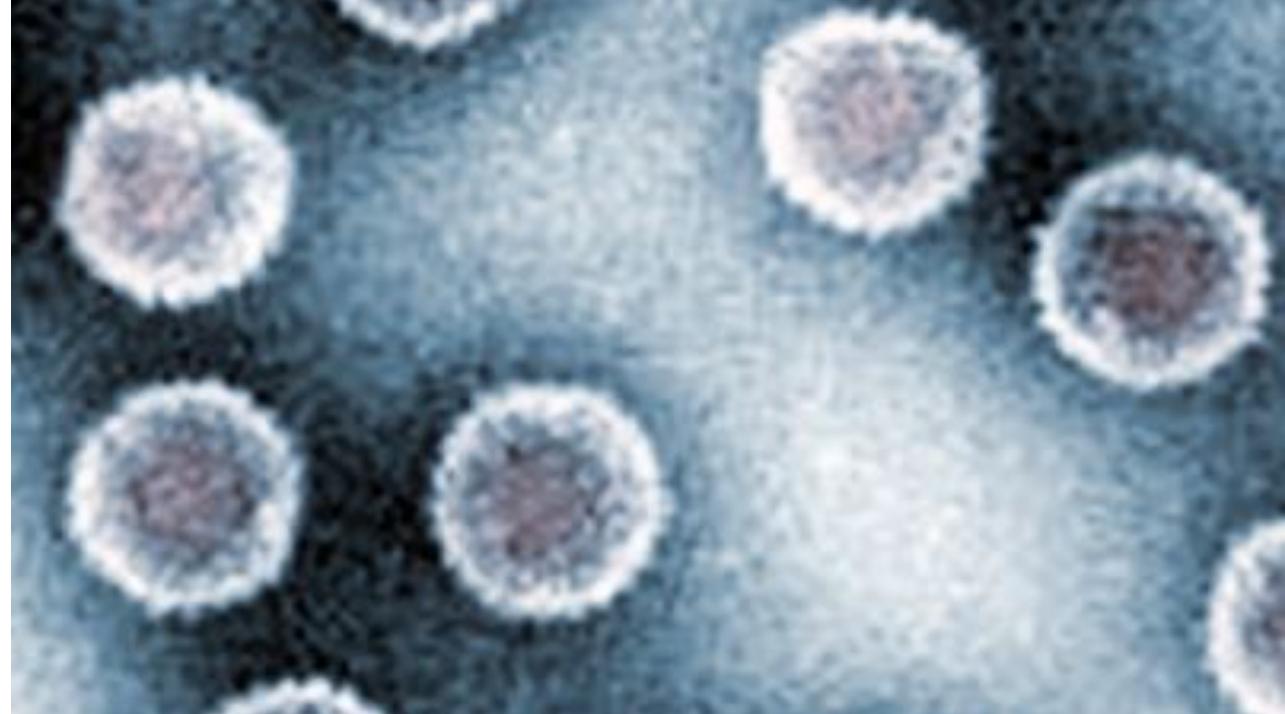
General malaise

Loss of pregnancy

Cats weakened by the virus may also develop secondary infections.



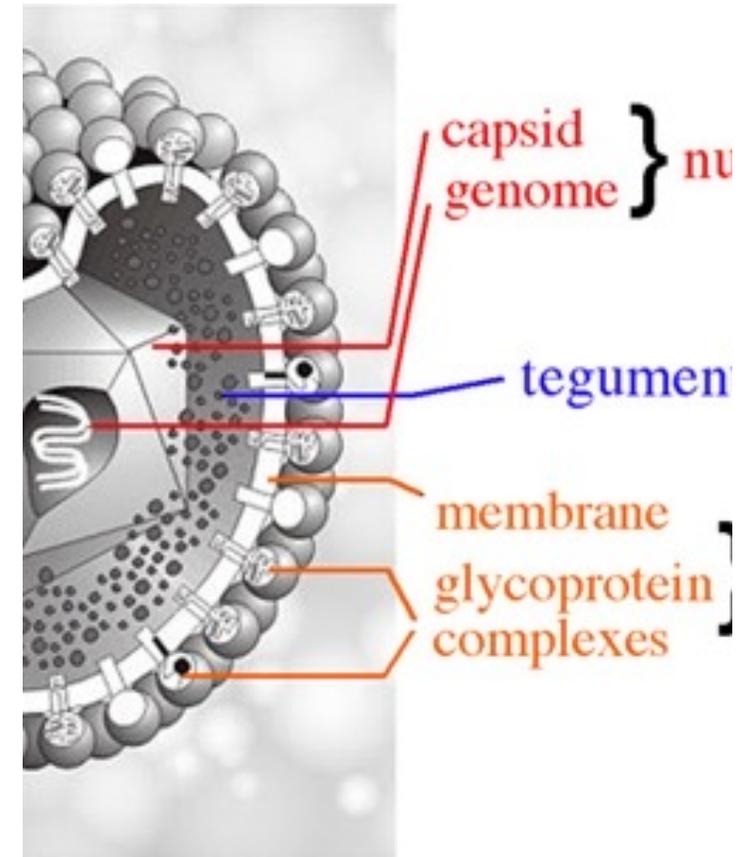
BHV-1



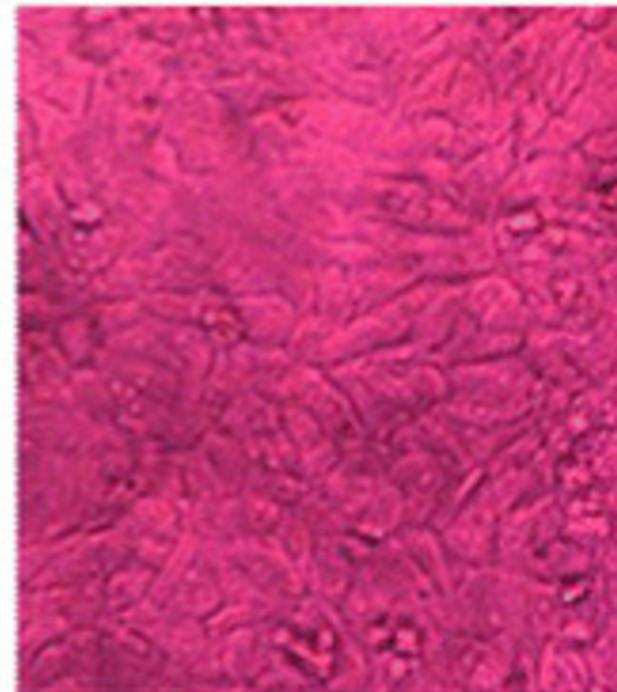
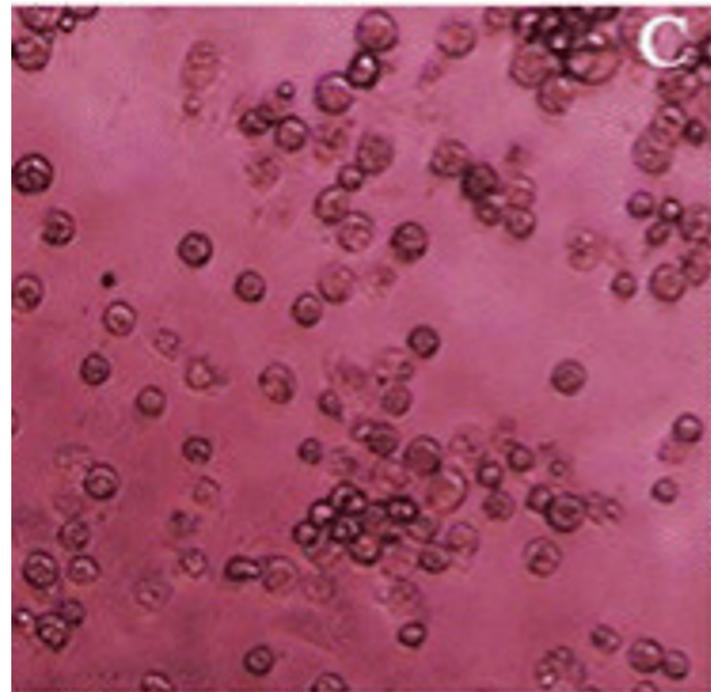
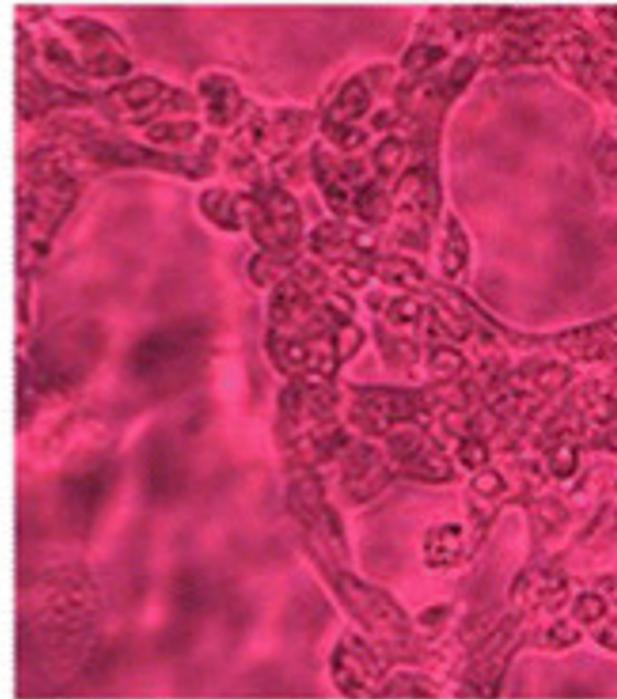
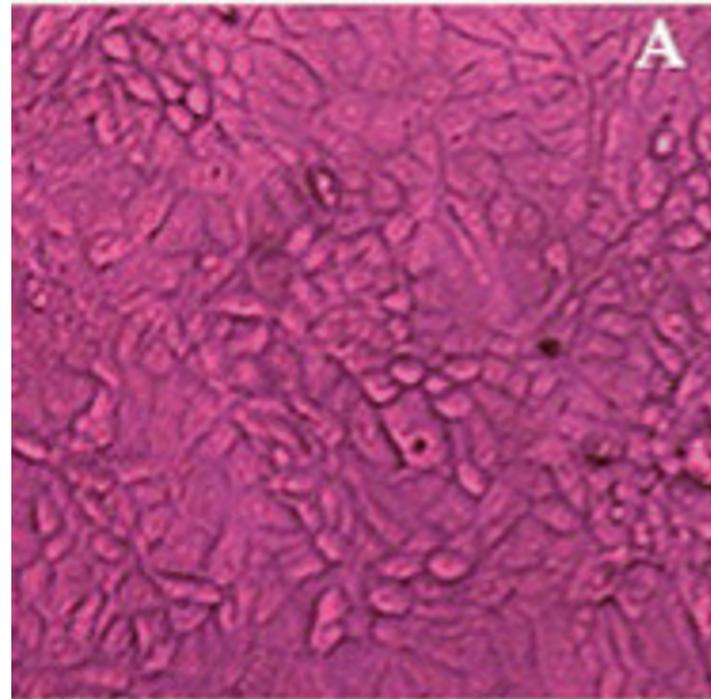
Infectious Bovine Rhinotracheitis (IBR),
Infectious Pustular Vulvovaginitis (IBV),
Infectious Balanoposthitis (IPV)

ETIOLOGY

- Family Herpesviridae, subfamily Alphaherpesvirinae
- Enveloped, Icosahedral
- DNA
- Only a single serotype of BHV-1 is recognized; however, three subtypes of BHV-1 have been described on the basis of endonuclease cleavage patterns of viral DNA:
 - BHV-1.1 (respiratory subtype),
 - BHV-1.2 (genital subtype),
 - BHV-1.3 (encephalitic subtype) BHV-5
- Three major glycoproteins, gB, gC, and gD induce neutralising antibody responses



- Virus could be isolated Bovine tissue cultures; MDBK.



Boubaker–Elandalousi, Ramzi, et al. "Non-cytotoxic Thymus capitata extracts prevent Bovine herpesvirus-1 infection in cell cultures." BMC veterinary research 10.1 (2014): 231.

Transmission

BHV-1 infections are widespread in the cattle of all ages.

IBR is a highly contagious.

Infection occurs by inhalation and requires contact between animals spreading quickly through the group.

Artificial insemination with semen from subclinically infected bulls.

Milk

Cattle with latent BHV-1 infections.

FEEDLOT CATTLE, BREEDING CATTLE

In feedlot cattle, the respiratory form is most common.

The viral infection alone is not life-threatening but predisposes to secondary bacterial pneumonia, which may result in death.

In breeding cattle, abortion or genital infections are more common.

PATHOGENESIS

The virus enters the animal via the nose and replicates in mucous membranes of the upper respiratory tract and in the tonsils.

It subsequently disseminates to conjunctivae and by neuronal axonal transport reaches the trigeminal ganglion. (Latent infection)

After genital infection, BoHV-1 replicates in mucous membranes of the vagina or prepuce and becomes latent in sacral ganglia.

The viral DNA remains in the neurons of the ganglia, probably for the entire life of the host.

CLINICAL SIGNS

Respiratory infection

- Fever (40° – 42° C),
- depression,
- cough, nasal and lacrimal discharge are common.
- Dyspnea, possibly with open-mouthed breathing,
- Subcutaneous emphysema may occur.
- Secondary bacterial pneumonia is a frequent occurrence.
- Abortion





Genital infection

- vesicles, pustules, hyperaemia or oedema and erosions or ulcers in the mucosa of the vulva and vagina or on the penis and prepuce.
- infectious pustular balanoposthitis (IPB).







IBR with severe palpebral congestion

Conjunctivitis

- The conjunctival form of BHV-1 infection, which resembles 'pink eye', is relatively uncommon.
- In some cases, the only sign of infection is conjunctivitis.

DIAGNOSIS

PCR,

virus isolation,

Immunoperoxidase

fluorescent antibody
staining.

PREVENTION AND CONTROL

- Immunization with modified-live or inactivated virus vaccines generally provides adequate protection against clinical disease.
- Both IM and intranasal modified-live vaccines are available, but the IM types may cause abortion in pregnant cattle.
- The intranasal vaccines can be used in pregnant cattle.
- Only inactive vaccines are used in our country.



gE(-) marker vaccine

This vaccine is obtained by deletion mutant of the glycoprotein E-encoding gene.

This gene is responsible for pathogenicity and is not essential for virus replication.



virus

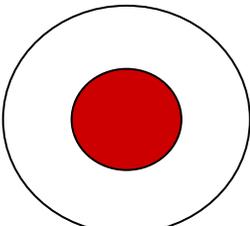
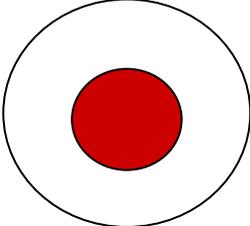


Vaccine virus

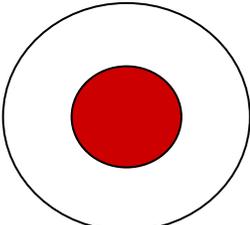
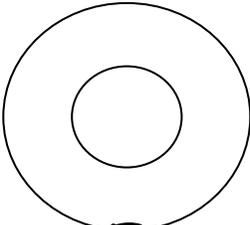


MARKER VACCINE

- In serum after vaccination with the gE(-) marker vaccine
- antibody to all glycoproteins except gE detection is possible.



infected



vaccinated

gE

gB

A close-up, profile view of a brown horse's head, showing its eye, ear, and muzzle. The horse is looking downwards and to the right. The background is solid black, which makes the horse's features stand out. The text is overlaid on the right side of the horse's head.

EQUINE HERPESVIRUS INFECTION

(Equine viral rhinopneumonitis, Equine abortion virus)

Etiology

Alphaherpesviridae , enveloped, double stranded DNA, latent infections and carrier states are seen with both virus types.

EHV-1 is the most important viral cause of abortion in horses. Abortion storms.

The neurologic form of EHV-1 has demonstrated increasing morbidity and mortality since 2000.

Therefore, the USDA has designated neuropathic EHV-1 as a potentially emerging disease.

Transmission

Transmission occurs by direct or indirect contact with infectious nasal secretions, aborted fetuses, placentas, or placental fluids.

Respiratory transmission is most common route of exposure.

Direct transmission

- Aborted fetuses, fetal membranes and/or fluids are significant sources of the virus.
- Infected foals are highly contagious and can transmit infection to other horses via the respiratory route through shedding virus into the environment.

Indirect transmission

- Virus can be viable for several weeks in the environment once it has been shed by the horse.
- Virus contaminated fomites are a significant factor in EHV spread.

Also, following infection, horses may become latent carriers of EHV; virus may be reactivated after stress or high doses of corticosteroids.



Pathogenesis

The pathogenetic mechanisms of EHV-1 and EHV-4 differ significantly.

EHV-4 infection is restricted to respiratory tract epithelium and associated lymph nodes; EHV-1 strains develop cell-associated viremia and prefer vascular endothelium, especially the nasal mucosa, lungs, placenta, adrenal, thyroid, and CNS.

CLINICAL SIGNS

The incubation period ranges from 2 to 10 days.

Fever is often biphasic and can be transient.

The upper respiratory tract infection occurs after the first fever episode. A systemic viremia is frequently followed by the second febrile episode (6-7 days).

Respiratory Disease

Fever (38.9-41.7°C)

Coughing

Nasal discharge

Enlargement of the mandibular and/or
retropharyngeal lymph nodes

Lethargy, anorexia

Conjunctivitis

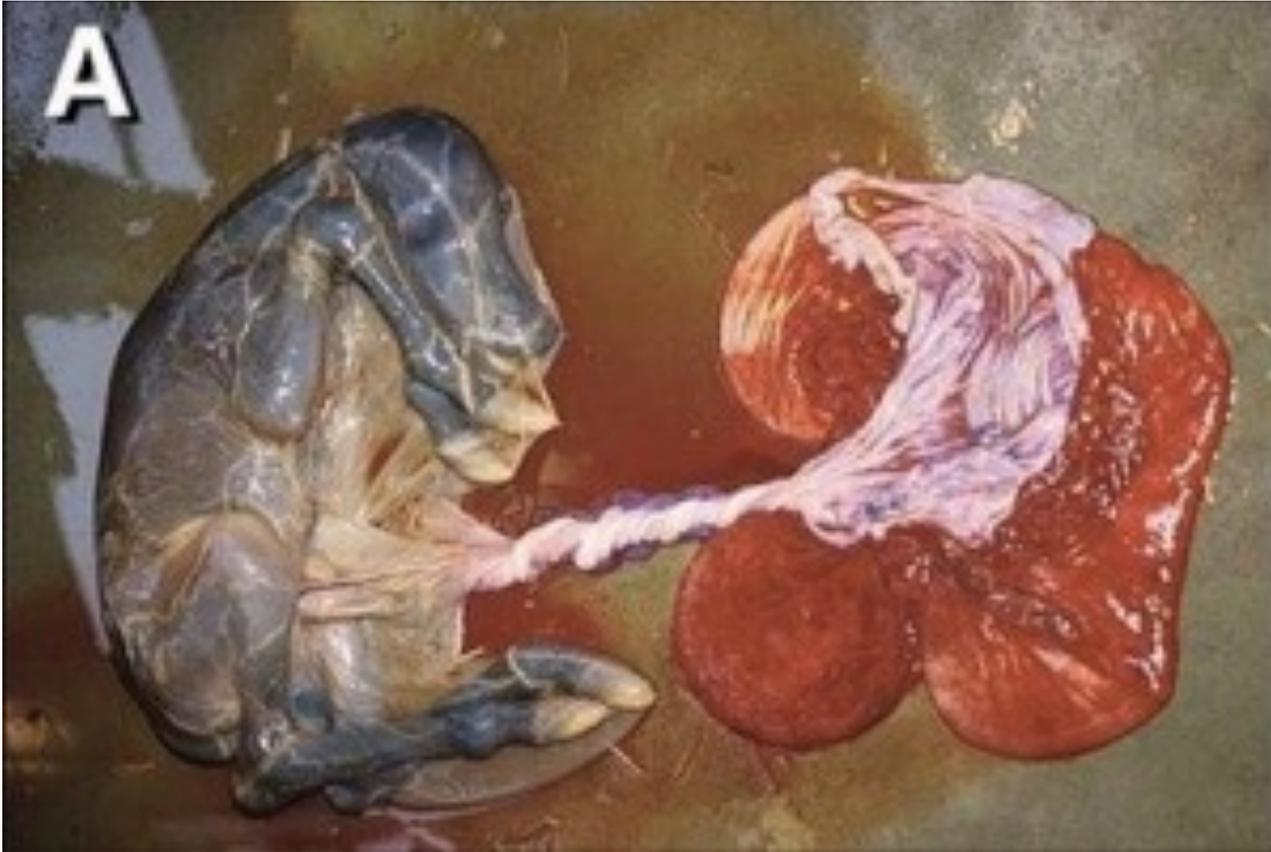
Ocular disease including uveitis and keratitis

Neonatal foals infected in utero are usually abnormal from birth

and exhibit any combination or all of the following:

- o Fever
- o Lethargy
- o Weakness
- o Jaundice
- o Respiratory distress/stridor/pneumonia
- o CNS signs (occasionally)
- o Death commonly occurs within 3 days.

Older foals: nasal discharge is the most common sign of disease.



Abortion:

Typically occurs in late pregnancy (7+ months); very occasionally as early as 4 months.

Neurologic Disease

Incoordination of
the hind (and
occasionally fore)
limbs :

Ataxia or wobbly
gait

Urine
retention/dribbling

Bladder atony

Recumbency with
inability to rise

Neurologic signs
are often preceded
by fever and/or
respiratory signs

Prevention and Control

New horses should be isolated for 21 days. Especially pregnant mares.

Pregnant mares should be maintained in a group away from the weanlings, yearlings, and horses out of training.

In an outbreak of respiratory disease or abortion, affected horses should be isolated and appropriate measures taken for disinfection of contaminated premises.

No horse should leave the premises for 3 wk after recovery of the last clinical case.

- Vaccination (EHV-4 and EHV-1) should begin when foals are 4–6 mo old.
- A second dose is given 4–6 wk later, and a third dose at 10–12 mo of age. Booster vaccinations may be indicated as often as every 6 mo through maturity (5 yr of age).

