

BLUETONGUE AND EPIZOOTIC HEMORRHAGIC DISEASE

(Sore muzzle, pseudo foot-and-mouth disease, muzzle disease)

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Definition [top](#)

Bluetongue (BT) and epizootic hemorrhagic disease (EHD) are insect-borne viral diseases of ruminants characterized by acute or subacute clinical courses in

susceptible ruminants. The BT virus (BTV) and EHD virus (EHDV) have also been associated with congenital disease in sheep and cattle.

Etiology [top](#)

Bluetongue and epizootic hemorrhagic disease are caused by orbiviruses in the family *Reoviridae*. Other orbiviruses include Ibaraki, Palyam, Eubenangee, and Tilligerry. The viruses are resistant to lipid solvents, which is typical of nonenveloped viruses. The viruses are relatively acid-labile, and slow freezing at -10 to -20 oC (14 - 4o F) is deleterious to the virus.

Worldwide, 24 serotypes of BTV and 9 serotypes of EHDV have been identified. Five serotypes of BTV and two serotypes of EHDV have been isolated in the United States. However, only BTV serotypes 10, 11, 13, and 17 and EHDV serotypes 1 and 2 are currently active. BTV serotype 2, originally isolated from animals imported into Florida, may not have established itself in the United States; however, epidemiologic studies will be required to resolve this issue.

Acetic acid is an effective disinfectant.

Host Range [top](#)

The host range of BTV is very broad and includes all ruminant species tested to date. However, the expression of clinical disease varies in different species. Sheep are fully susceptible to BTV. EHDV typically also infects most ruminant species; however, sheep appear to be a poor host and rarely develop signs of EHDV infection.

Geographic Distribution [top](#)

The geographic distribution of the orbiviruses is extensive, but current knowledge is still incomplete. Virus distribution is based on the presence of certain *Culicoides* species, including *C. variipennis*, *C. imicola*, *C. brevitarsis*, and others. Orbivirus infections are common in tropical, subtropical, and temperate climates. Areas with year-round vector activity could easily maintain the virus by a continuous vector-host cycle. Virus persistence in areas with severe winters is not understood. Reintroduction of virus into the area in the warm months by transportation of infected animals or infected *Culicoides* being carried on the wind is probably common. Some research reports suggest that overwintering of the virus in these areas occurs by mechanisms such as (1) prolonged viremias (up to 3 months) in certain animals, (2) transplacental transmission of BTV in the late fall and early winter to the late-term developing fetus with subsequent birth of a viremic calf,

and (3) overwintering of the virus in *Culicoides* that may survive through the winter at very low population densities. Virological and serological testing has suggested that BTV exists in North, Central, and South America; Africa; and parts of Asia; Europe; the Middle East; and the South Pacific; EHDV is probably similarly distributed.

Transmission [top](#)

Transmission of orbiviruses is primarily by *Culicoides* species, which are biological vectors. Limited experimental studies have also demonstrated that ticks are capable of mechanically or biologically transmitting BTV; however, their role in the epidemiology of BTV is probably minimal. Virus can also be transferred from viremic dams (sheep and cattle) to the developing fetus. Although BTV can be found in the semen of certain rams and bull, it is only isolated at the time of peak viremia; this presence of virus would appear to be the result of blood cells in the semen. Extensive field and experimental studies suggest that transmission of BTV via semen is of not importance in the epidemiology of BTV. The potential for BTV transmission also exists owing to poor management practices such as using the same needle or infected surgical equipment on several animals (mechanical transmission).

Incubation Period [top](#)

The incubation period of BT in sheep is usually 7-10 days; however, viremia may appear as early as 3 to 4 days after infection. In cattle, viremia occurs as early as 4 days postinfection, but clinical signs are uncommon. Development of clinical BT in cattle may be the result of hypersensitization. Under laboratory conditions such animals develop clinical signs 10 to 12 days following reexposure to the virus. The incubation of BTV infection in deer is 7 to 12 days. No information is available on the incubation periods for EHDV.

Clinical Signs of Bluetongue [top](#)

BT in Sheep [top](#)

The classic signs of BT in sheep are those of an acute to subacute infection by a virulent strain of virus in fully susceptible animals belonging to the fine wool or mutton breeds. However, the signs of BT are variable. Not all strains of BTV that infect sheep cause clinical disease. In some flocks, no clinical sign is apparent, whereas in other flocks infected by the same virus up to 30 percent may develop signs of disease.

The first sign of illness which begins 7 to 8 days after infection, is a rise in temperature to 106-107° F (41.6-41.7° C). Temperatures may be elevated for 4 to 12 days following the initial rise. Within 24 hours of the initial rise in temperature, excessive salivation and frothing at the mouth develop and are associated with hyperemia and swelling of the buccal and nasal mucosa (Fig. 33). If forced to move, sheep may pant like a dog. During the next 2 to 3 days, erosions and ulcerations may develop in the buccal mucosa. By 4 to 7 days in severe cases, extensive ulcerations may be covered by gray necrotic tissue on the dental pad and dorsal surface of the tongue. In addition, affected animals being fed rough feed (stemmy alfalfa) may have more severe lesions of the oral mucosa.

Hyperemia is often observed around the coronary bands of the hooves. Often the hooves are tender and varying degrees of lameness are apparent. In more severe cases, the animals stand with an arched back (Fig. 34). If the animals are driven during this time, they may slough their hooves. Animals that recover may have a dark line in the wall of the hoof.

The lesions in the mouth, the reluctance to move, and the necrosis of striated musculature lead to weakness, depression, and rapid weight loss. These can result in prostration and eventual death in severely affected animals. Sheep that recover from severe infections may have a break in the wool 3 to 4 weeks after the fever has subsided. This can lead to partial or complete shedding of wool.

Bluetongue virus infection of pregnant ewes in the first trimester can cause fetal death and resorption, abortion, or birth of "dummy" lambs. Attenuated BTV vaccines can also cause reproductive failure.

BT in Cattle [top](#)

Bluetongue virus infection in cattle usually does not cause any clinical sign of disease. Subclinical disease is only evidenced by changes in the leukocyte and lymphocyte subpopulation counts in the peripheral blood and a mild acute eosinophilic dermatitis. A consistent fluctuation in rectal temperature is indicative of viremia and a mild disease. Occasionally, field outbreaks of BT disease occur in which as many as 30 percent of the cattle have clinical signs. Experimental data support the contention that clinical BT in cattle occurs as result of prior sensitization to a related orbivirus followed by a later second exposure. After secondary exposure, clinical signs become apparent in 10-12 days. Clinical signs consist of mild hyperemia in the buccal cavity and around the coronary band; vesicular lesions, which lead to ulcerations, in the buccal mucosa; erect hair over the cervical and dorsal thoracic areas; and a definite hyperesthesia. In addition, the dermis becomes thickened with prominent folds apparent in the cervical areas,

and a dry crusty exudate leads to matting of hair in affected areas. These lesions may persist for 10 to 20 days. Similar lesions have been reported on teats of cattle with clinical BT. Hoof lesions may be associated with lameness. In some instances severe breaks in the hooves occur 40 to 60 days after infection and are usually followed by foot rot.

Bluetongue virus infection can cause reproductive failure in cattle. Some infected bulls will become temporarily sterile following acute infections. After recovery, production of sperm resumes, and the bulls are capable of settling cows.

Certain strains of BTV are capable of causing fetal death, resorption and or abortion; cell-culture-adapted live virus may be more effective than field virus in establishing fetal infection. Teratogenic effects of BTV in the bovine fetus include hydranencephaly and cerebral cysts that result in "dummy" calves.

Critical factors for fetal infection include the stage of embryonic or fetal development when infection occurs, the immune status of the dam, and the strain (s) of BTV causing infection. The most susceptible period for fetal infections occurs between 60 and 140 days gestation in nonimmune dams. Experimental studies suggest 15 to 20 percent of viremic dams will transmit virus to their fetuses. In areas where strains of BTV are endemic, there is little evidence that BTV has adverse affects on reproduction.

BT in Goats [top](#)

Bluetongue infection of goats is typically an inapparent infection similar to that described for cattle.

Clinical Signs of EHDV Infection [top](#)

EHD in Sheep [top](#)

The EHD virus does not appear to cause significant clinical disease in sheep.

EHD in Cattle [top](#)

The EHD virus rarely causes disease in cattle. However, Ibaraki virus (an EHDV serotype) has been associated with sporadic outbreaks of severe disease in cattle in Japan. Mortality rates have been as high as 10 percent. Clinical signs consist of fever, erosive and ulcerative lesions of the oral and esophageal mucosa, stiffness, lameness, and thickened, edematous skin. In addition, there has been a report of

a combined EHDV and BTV disease of cattle. In pregnant cows, EHDV infection can result in fetal resorption or hydranencephaly if infection occurs between days 70 and 120 of gestation.

EHD in Deer [top](#)

An EHD virus infection in white-tailed deer usually follows a peracute course leading to death. Often, deer are found dead around waterholes which suggests that they had a high fever and were dehydrated.

Gross Lesions [top](#)

BT in Sheep [top](#)

The lesions of BT in sheep vary greatly depending on (1) the strain of virus, (2) individual animal and breed susceptibility and (3) environmental (stress) factors. Prominent lesions include facial edema, edematous ears, and dry, crusty exudate over the nostrils (Fig. 33). Lesions in the oral cavity include focal petechial (pinhead-size) hemorrhages that progress to gray necrotized debris over erosions and ulcerations on the lips; on the dorsal, lateral, and ventral surfaces of the tongue; and on the dental pad. The buccal mucosa may be cyanotic. Hyperemia and occasional erosions can occur on papillae and laminae in the reticulum and omasum.

Lesions in the respiratory system include cyanosis and edema of the nasal mucosa and pharynx, and there may be tracheal hyperemia and congestion. Froth in the trachea is present only when there is pulmonary congestion and edema.

Lesions in the vascular system cause hyperemia, edema, and hemorrhages. A characteristic lesion is hemorrhage at the base of the pulmonary artery. Petechial and ecchymotic (larger than pinhead-size) hemorrhages may be observed at times in the endocardium. Focal gray-white areas of necrosis are often found in the papillary muscles and less frequently in other areas of the myocardium.

The most prominent changes in the skin include dermal and subcutaneous edema of the head and ears. Sometimes an irregular rash (exanthematous eruptions) may progress into serous and crusty exudates on the skin. Hyperemia is prominent at the coronet of the hoof. Often, this reddening is accompanied by petechial or ecchymotic hemorrhages that extend down the horn.

A yellow gelatinous exudate is common in the fascia (connective tissue) along and between skeletal muscles. On the cut surface of the heavy muscle there may be

focal hemorrhages and areas that appear dry and gray-white.

Newborn lambs with congenital BT have hydranencephaly or porencephaly. These lesions are characterized by fluid-filled cavities, either occupying the whole of the cranial vault or as cystic cavities in the gray and white matter of the cerebral cortex. Cerebellar dysplasia (abnormal development) (Fig. 35) with rudimentary medial and lateral lobes may be present. The spinal cord may be dysplastic (abnormal development) and lack white matter. Skeletal deformities may consist of scoliosis (lateral curvature of the spine) and torticollis (twisted neck).

BT in Cattle [top](#)

Gross lesions in cattle differ in some respects from those observed in sheep. The most prominent lesions involve the skin, mouth, and hooves. Skin lesions are characterized by marked edema that leads to thick folds — particularly in the cervical areas. Lesions may form in the folds as serous exudate accumulates and dries. Dry, crusty exudate is present on the skin over the cervical and thoracic areas. The crusty material results from vesicular eruptions and ulcerations.

The external nares may have erosions covered by crusty exudate that sloughs. Lesions in the mouth start as vesicles and proceed to ulcers covered with grayish necrotic debris. These lesions are more common on the buccal mucosa and dental pad and rarely the tongue. Hyperemia occurs at the coronary band. In some instances, fissures occur 6 to 8 weeks following infection.

In utero BTV infection may lead to fetal death and resorption, abortion, hydranencephaly, or cerebral cysts.

BT in Deer [top](#)

Bluetongue in susceptible deer causes widespread hemorrhages throughout the body. These lesions are associated with intravascular thromboses and hemorrhages varying in size from petechial to ecchymotic. In chronic BT, deer may develop severe fissures and even sloughing of hooves. Ulcers covered with gray necrotic debris are found in the buccal mucosa, dental pad, and tongue.

EHD in Deer [top](#)

In susceptible deer EHDV causes lesions very similar to those caused by BTV. The widespread hemorrhages in mucous membranes, skin, and viscera are the result of disseminated intravascular clotting. The Ibaraki strain of EHDV can cause widespread vascular lesions similar to those described for BTV in cattle.

Degenerative changes (focal hemorrhage or dry and gray-white appearance, or both) in striated musculature are prominent in the esophagus, larynx, tongue, and skeletal muscles.

Morbidity and Mortality [top](#)

In sheep, BT can range from inapparent to severe, depending on the breed, strain of virus, and environmental stress on the animals. Morbidity can reach 100 percent; mortality can vary from 0 to 50 percent. Many animals will recover within a few days to 2 weeks.

In cattle, BTV and EHDV infection is usually subclinical. Although morbidity can approach 5 percent, cattle typically recover within a few weeks. However, lameness and unthriftiness may persist for prolonged period.

The morbidity and mortality for BTV infection in other species are as follows:

Goats - - minimal clinical signs

White-tailed deer - - morbidity approaching 100 percent and a mortality of 80-90 percent

Pronghorn antelope - - morbidity approaching 100 percent and a mortality of 80-90 percent

Bighorn sheep - - morbidity approaching 100 percent and a mortality of 0 to 50 percent

North American elk - - similar to cattle; the disease is usually subclinical.

Diagnosis [top](#)

Field Diagnosis [top](#)

Tentative diagnosis of BT can be made when (1) clinical signs appear in populations known to be susceptible, (2) the occurrence of disease coincides with a prevalence of insect vectors, (3) necropsy of sheep reveals characteristic gross lesions, and (4) a flock history of recent wasting (loss of weight) and pododermatitis (foot rot).

Specimens for the Laboratory [top](#)

Preferred samples for confirmatory diagnosis include sterile heparinized blood samples from animals with clinical signs or spleen or bone marrow, or both, from dead animals. Samples from aborted and congenitally infected newborn animals should include heparinized blood and, if possible, spleen, lung, brain, and serum. If possible, the heparinized whole blood (erythrocytes and white cells) should be washed in saline containing antibiotics and resuspended in saline prior to shipping. This procedure will reduce the antibody that may neutralize the virus if blood-cell lysis occurs.

Specimens should be shipped refrigerated, not frozen. Freezing makes virus isolation difficult.

Laboratory Diagnosis [top](#)

Confirmatory diagnosis is based on isolation and identification of virus from blood or tissues. Diagnosis for lambs and calves infected in utero is based on serology (if no colostrum has been ingested) or virus isolation, or both.

Differential Diagnosis [top](#)

Differential diagnoses include plant photosensitization, foot-and-mouth disease, vesicular stomatitis, bovine virus diarrhea, malignant catarrhal fever, infectious bovine rhinotracheitis, parainfluenza-3, contagious ecthyma, and actinobacillosis.

Vaccination [top](#)

Vaccination has been the primary means of controlling BT disease in sheep. To date, only modified-live (attenuated) virus vaccines have been used. Because of the multiplicity of BTV serotypes and variable cross-protection between serotypes, vaccination has resulted in varying degrees of success. The serotypes incorporated into the vaccine must be the same as those causing infection in the field. The practice of administering multiple virus serotypes in a single vaccination is argued against by some scientists because (1) an immune response (virus neutralizing antibody) is typically only induced to one, or at best two of the serotypes incorporated in the vaccine and (2) reassortment between the genome segments of the multiple vaccine viruses may occur in the host of a vector feeding on such a vaccinated animal. Although simultaneous infection of sheep, cattle, or *Culicoides* can result in creation of reassortant viruses, there is no evidence that this process has resulted in generation of new serotypes. However, such reassortant events may result in altered virulence and biological transmissibility.

No inactivated or subunit vaccines are currently available, though several experimental vaccine preparations have been studied, including inactivated virus vaccines, subunit vaccines prepared by purification of natural VP2 (viral protein responsible for inducing virus neutralizing antibody), and recombinant VP2 expressed in a baculovirus system.

No vaccine is available for EHDV.

Control and Eradication [top](#)

Vaccination can be used in endemic areas.

Vector control measures to impede the spread of BTV infection are not commonly used. However, certain measures have potential effectiveness such as water management (reduction of *Culicoides* breeding sites), use of insecticides and larvacides (spraying breeding areas), and insect repellents in which animals are dipped.

The only applicable treatment available is to minimize animal stress and administer broad-spectrum antibiotics to combat secondary infection.

Public Health [top](#)

There is only one documented human infection, and that was in a laboratory worker

GUIDE TO THE LITERATURE [top](#)

1. Bluetongue Symposium. 1975. Aust.Vet.J.,51.
2. International Symposium on Bluetongue and Related Orbiviruses. 1985. Prog. Clin.Biol.Res.,78.
3. International Symposium on Bluetongue, African Horsesickness and Rrlated Orbiviruses. CRC Press, 1992.
4. BEKKER, J. G., DeKOCK, W., and QUINLANN, J.B. 1934. The occurrence and identification of bluetongue in cattle—The so-called pseudo foot-and-mouth disease in South Africa. Onderstepoort J. Vet. Sci. Anim. Indust., 2:393-507.

5. BOWNE, J.G. 1971. Bluetongue disease. *Adv. Vet. Sci. Comp. Med.*, 15:146.
6. CAMPBELL, C.H., BARBER, T.L., and JOCHIM, M.M.: Antigenic relationship of Ibaraki, bluetongue, and epizootic hemorrhagic disease viruses. *Vet. Micro.*, 3:15-22.
7. GIBBS, E.P.J., and GREINER, E.C. 1989. Bluetongue and Epizootic hemorrhagic Disease. in Epidemiology of Arthropod- Borne Viral Diseases, T.P. Monath, ed., Boca Raton, FL: CRC Press, pp.2: 39-70.
8. GORMAN, B.M. 1979. Variation in orbiviruses. *J. Gen. Virol.*, 44:1-15.
9. GOULD, A.R., and PRITCHARD, L.I. 1990. Relationships amongst bluetongue viruses revealed by comparisons of capsid and outer coat protein nucleotide sequences. *Virus Res.*, 17:31.
10. HOWELL, P.G., and VERWOERD, D.W. 1971. Bluetongue virus. *Viol. Monographs*, 9:35-74.
11. HUISMANS, H., and ERASMUS, B.J. 1981. Identification of the serotypespecific and group-specific antigens of bluetongue virus. *Onderstepoort J. Vet. Res.*, 48:51-58.
12. JONES, R.H., and FOSTER, N.M. 1978. Heterogeneity of *Culicoides variipennis* field populations to oral infection with bluetongue virus. *Am. J. Trop. Med. Hyg.*, 27: 178-183.
13. JOCHEIM, M.M., and JONES, S.C. 1976. Plaque neutralization of bluetongue virus and epizootic hemorrhagic disease virus in BHK-21 cells. *Am. J. Vet. Res.*, 37:1345-1347.
14. KARSTAD, L., and TRAINER, D.O. 1967. Histopathology of experimental bluetongue disease of white-tailed deer. *Can. Vet. J.*, 8:347-254.
15. LUEDKE, A.J. 1969. Bluetongue in sheep: Viral assay and viremia. *Am. J. Vet. Res.* 30:499-509.
16. LUEDKE, A.J., BOWNE, J.G., JOCHIM, M.M., and DOYLE, C. 1964. Clinical and pathological features of bluetongue in sheep. *Am. J. Vet. Res.*, 25:963-970.
17. MacLACHLAN, N.J., and OSBURN, B.I. 1983. Bluetongue virus-induced hydranencephaly in cattle. *Vet. Pathol.*, 20:563-573.

18. MURPHY, F.A., BORDEN, E.C., SHOPE, R.E., and HARRISON, A. 1971. Physicochemical and morphological relationships of some arthropodborne viruses to bluetongue virus—A new taxonomic group. Electron microscopic studies. *J. Gen. Virol.*, 13:273-278.
19. NELL, E.M. 1971. Cattle and culicoides biting midges as possible overwintering hosts of bluetongue virus. *Onderstepoort J. Vet. Res.*, 38:65.
20. OSBURN, B.I., MCGOWAN, B., HERON, B., LOOMIS, E., BUSHNELL, R., STOTT, J., and UTTERBACK, W. 1981. Epizootiologic study of bluetongue: Virologic and serologic results. *Am. J. Vet. Res.*, 42:884-887.
21. OSBURN, B.I., SILVERSTEIN, A.M., PRENDERGAST, R.A., JOHNSON, R.T., and PARSHALL, C.J. 1971. Experimental viral-induced congenital encephalopathies. I. Pathology of hydranencephaly and porencephaly caused by bluetongue vaccine virus. *Lab. Invest.*, 25:197-205.
22. PEARSON, J.E., and JOCHIM, M.M. 1979. Protocol for the immunodiffusion test for bluetongue. *Ann. Proc. Am. Assoc. Vet. Lab. Diag.*, 22:463-471.
23. RICHARDS, W.P.C., CRENSHAW, G.L., and BUSHNELL, R.B. 1971. Hydranencephaly of calves associated with natural bluetongue virus infection. *Cornell Vet.*, 61:336-348.
24. Roy, P. 1991. Towards the Control of Emerging Bluetongue Disease. London: Oxford Virology Publications, pp. 1-71.
25. SPREULL, J. 1905. Malarial catarrhal fever (bluetongue) of sheep in South Africa. *J. Comp. Path. Therap.*, 18:321-337.
26. STOTT, J. L. , OSBURN, B.I., and MACLACHLAN, N.J. 1984. Diagnosis of bluetongue virus infection in cattle: Virus isolation or serology? *Proc Annu Mtg Am Assoc Vet Lab Diag* 26.
27. VERWOERD, D.W., HUISMANS, H., ERASMUS, B.J. 1979. Orbiviruses. In Comprehensive Virology, Vol 14. H. Fraenkel-Conrat and R.R. Wagner, eds. Plenum Pub.
28. VERWOERD, D.W., ELS, H.J., DeVILLIERS, E.M., and HUISMANS, H. 1972. Structure of the bluetongue capsid. *J. Virol.*, 10:783-794.

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