

Bluetongue

Sore Muzzle, Pseudo Foot-and-Mouth Disease, Muzzle Disease

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Importance

Bluetongue is an insect-borne viral disease of ruminants. Among domestic animals, clinical disease occurs most often in sheep, and can result in significant morbidity. Affected sheep may have erosions and ulcerations on the mucous membranes, dyspnea, or lameness from muscle necrosis and inflammation of the coronary band. Some sheep may slough their hooves, and surviving animals can lose part or all of their wool. Some strains of the virus can result in mortality rates as high as 70% in highly susceptible sheep.

The bluetongue virus has recently expanded its geographic range. Before 1998, this virus rarely occurred in Europe; however, some serotypes are now regularly found in southern European countries and may be enzootic in this region. In 2006, a serotype 8 virus, which may have come from Africa, caused outbreaks in Germany, Belgium and the Netherlands. Due to the adaptability of its vector, *Culicoides dewulfi*, to European weather conditions, the latter virus has the potential to expand geographically in northern Europe.

Etiology

Bluetongue results from infection by the bluetongue virus, a member of the genus *Orbivirus* and family Reoviridae. Twenty-four serotypes have been identified worldwide; six serotypes (1, 2, 10, 11, 13, and 17) have been found in domesticated or wild ruminants in the United States. Bluetongue viruses are closely related to the viruses in the epizootic hemorrhagic disease (EHD) serogroup.

Species Affected

Bluetongue virus infects many domesticated and wild ruminants including sheep, goats, cattle, buffalo, deer, antelope, bighorn sheep and North American elk. Clinical disease is seen often in sheep, occasionally in goats, and rarely in cattle. Severe disease can also occur in some wild ruminants including white-tailed deer (*Odocoileus virginianus*), pronghorn (*Antilocapra americana*) and desert bighorn sheep (*Ovis canadensis*). In Africa, some large carnivores have antibodies to bluetongue, and, in the United States, a contaminated vaccine resulted in some abortions and deaths in pregnant dogs.

Geographic Distribution

The bluetongue virus has been found in many parts of the world including Africa, Europe, the Middle East, Australia, the South Pacific, North and South America, and parts of Asia. The virus is present in some regions without associated clinical disease. In the United States, the distribution of the vector limits infections to the southern and western states.

Transmission

Bluetongue virus is transmitted by biting midges in the genus *Culicoides*. *Culicoides varipennis* var *sonorensis* is the principal vector in the United States, *C. brevitarsis* in Australia, and *C. imicola* in Africa and the Middle East. *C. imicola* is also the major vector in southern Europe, but *C. dewulfi* has been identified as the vector in the 2006 northern European outbreaks. Other *Culicoides* species can also transmit the virus and may be important locally. Ticks or sheep keds can be mechanical vectors but are probably of minor importance in disease transmission. Cattle are the major amplifying host due to their prolonged viremia and the feeding preferences of many *Culicoides* species.

Bluetongue is not a contagious disease; however, the virus can be spread mechanically on surgical equipment and needles. Bluetongue virus can be found in semen and venereal transmission from bulls is possible, but does not appear to be a major route of infection.

Incubation period

In sheep, the incubation period is usually 5 to 10 days. Cattle can become viremic starting at four days post-infection, but rarely develop symptoms. Animals are usually infectious to the insect vector for several weeks.

Clinical Signs

The vast majority of infections with bluetongue are clinically inapparent. In a percentage of infected sheep and occasionally other ruminants, more severe disease can occur.

In sheep, the clinical signs may include fever, excessive salivation, depression, dyspnea and panting. Initially, animals have a clear nasal discharge; later, the discharge becomes mucopurulent and dries to a crust around the nostrils. The muzzle, lips and ears are hyperemic, and the lips and tongue may be very swollen. The tongue is occasionally cyanotic and protrudes from the mouth. The head and ears may also be edematous. Erosions and ulcerations are often found in the mouth; these lesions may become extensive and the mucous membranes may become necrotic and slough. The coronary bands on the hooves are often hyperemic and the hooves painful; lameness is common and animals may slough their hooves if they are driven. Pregnant ewes may abort their fetuses, or give birth to “dummy” lambs. Additional clinical signs can include torticollis, vomiting, pneumonia or conjunctivitis. The death rate varies with the strain of virus. Three or four weeks after recovery, some surviving sheep can lose some or all of their wool. Recrudescence of clinical disease has been reported in sheep, possibly as the result of persistent infections in ovine $\gamma\delta$ T-lymphocytes.

Infections in cattle are usually subclinical; often, the only signs of disease are changes in the leukocyte count and a fluctuation in rectal temperature. Rarely, cattle have mild hyperemia, vesicles or ulcers in the mouth; hyperemia around the coronary band; hyperesthesia; or a vesicular and ulcerative dermatitis. The skin may develop thick folds, particularly in the cervical region. The external nares may contain erosions and a crusty exudate. Temporary sterility may be seen in bulls. Infected cows can give birth to calves with hydranencephaly or cerebral cysts. Cattle that have clinically apparent disease may develop severe breaks in the hooves several weeks after infection; such breaks are usually followed by foot rot. Infections in goats are usually subclinical, and similar to disease in cattle.

Although many infections in wild ruminants are inapparent, severe disease can occur in some species. In pronghorn antelope and whitetail deer, the most common symptoms are hemorrhages and sudden death.

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

In sheep, the face and ears are often edematous. A dry, crusty exudate may be seen on the nostrils. The coronary bands of the hooves are often hyperemic; petechial or ecchymotic hemorrhages may be present and extend down the horn. Petechiae, ulcers and erosions are common in the oral cavity, particularly on the tongue and dental pad, and the oral mucous membranes may be necrotic or cyanotic. The nasal mucosa and pharynx may be edematous or cyanotic, and

the trachea hyperemic and congested. Froth is sometimes seen in the trachea, and fluid may be found in the thoracic cavity. Hyperemia and occasional erosions may be seen in the reticulum and omasum. Petechiae, ecchymoses and necrotic foci may be found in the heart. In some cases, hyperemia, hemorrhages and edema are found throughout the internal organs. Hemorrhage at the base of the pulmonary artery is particularly characteristic of this disease. In addition, the skeletal muscles may have focal hemorrhages or necrosis, and the intermuscular fascial planes may be expanded by edema fluid.

In deer, the most prominent lesions are widespread petechial to ecchymotic hemorrhages. More chronically infected deer may have ulcers and necrotic debris in the oral cavity. They may also have lesions on the hooves, including severe fissures or sloughing.

Morbidity and Mortality

In sheep, the severity of disease varies with the breed of sheep, virus strain and environmental stresses. The morbidity rate can be as high as 100% in this species. The mortality rate is usually 0-30%, but can be up to 70% in highly susceptible sheep. Similar morbidity and mortality rates are seen in bighorn sheep. Bluetongue is usually severe in whitetail deer and pronghorn antelope, with a morbidity rate as high as 100% and a mortality rate of 80-90%.

Most infections in cattle, goats and North American elk are asymptomatic. In cattle, up to 5% of the animals may become ill, but deaths are rare. In some animals, lameness and poor condition can persist for some time.

Diagnosis

Clinical

Bluetongue should be suspected when typical clinical signs are seen during seasons when insects are active. A recent history of wasting and foot rot in the herd supports the diagnosis.

Differential diagnosis

The differential diagnosis includes foot-and-mouth disease, vesicular stomatitis, peste des petits ruminants, plant photosensitization, malignant catarrhal fever, bovine virus diarrhea, infectious bovine rhinotracheitis, parainfluenza-3 infection, contagious ecthyma (contagious pustular dermatitis), sheep pox, foot rot and *Oestrus ovis* infestation. In cattle and deer, EHD can also result in similar symptoms.

Laboratory tests

Bluetongue can be diagnosed by isolating the virus in embryonated chicken eggs or cell cultures. Appropriate cell cultures include mouse L, baby hamster kidney (BHK)-21, African green monkey kidney (Vero), and *Aedes albopictus* (AA) cells. Isolation in embryonated eggs is more sensitive than isolation in cell culture.

Bluetongue virus can also be isolated by inoculation into sheep, and sometimes suckling mice or hamsters. Animal inoculation is more sensitive than virus isolation in cell culture, and may be particularly valuable when the virus titer is very low. Bluetongue viruses can be identified to the serogroup level by immunofluorescence, antigen-capture enzyme-linked immunosorbent assay (ELISA) or the Immunospot test, as well as other techniques. These viruses can be serotyped with virus neutralization tests.

Polymerase chain reaction (PCR) techniques are widely used to identify the bluetongue virus in clinical samples. These techniques allow for rapid diagnosis and can identify the serogroup and serotype.

Serology is sometimes used for diagnosis. Antibodies appear 7 to 14 days after infection and are usually persistent. Available serologic tests include agar gel immunodiffusion (AGID), competitive ELISA, and virus neutralization. The AGID and indirect ELISA tests can identify serogroup-specific antibodies. A newer monoclonal antibody-based competitive ELISA can also distinguish antibodies to viruses in the bluetongue serogroup from antibodies to the EHD serogroup. Virus neutralization tests can determine the serotype specificity of antibodies, but are cumbersome. Complement fixation has largely been replaced by other tests, but is still used to detect antibodies to bluetongue virus in some countries.

Samples to collect

A human infection has been documented in one laboratory worker; reasonable precautions should be taken while working with this virus.

Blood samples (for virus isolation) and serum should be collected from several live febrile animals, as early as possible after infection. The blood should be collected into an anticoagulant. Spleen, bone marrow, or both are the tissues of choice at necropsy. Blood and serum should be collected from lambs with congenital disease; spleen, lung and brain tissue should also be sent, if they are available. All samples should be transported cold but not frozen, and sent to the laboratory as soon as possible.

Recommended actions if bluetongue is suspected

Notification of authorities

Bluetongue is a reportable disease in many states. State authorities should be consulted for more specific information.

Federal: Area Veterinarians in Charge (AVIC):
http://www.aphis.usda.gov/animal_health/area_offices/
State Veterinarians:
<http://www.aphis.usda.gov/vs/sregs/official.html>

Control

Bluetongue is transmitted by insect vectors and is not contagious by casual contact. Disinfectants cannot

prevent the virus from being transmitted between animals; however, where disinfection is warranted, sodium hypochlorite or 3% sodium hydroxide are effective. Insect control is important in limiting the spread of the disease; synthetic pyrethroids or organophosphates are effective against *Culicoides*. Moving animals into barns in the evening can also reduce the risk of infection. Although the bluetongue virus does not infect equids, horses and stables should be considered in any control scheme, as *Culicoides* can feed on horses, and manure piles are ideal breeding sites for these vectors.

In countries where bluetongue is endemic, vaccines are also used for control. Attenuated vaccines, which are available in countries including the U.S., are generally serotype specific. Multivalent live vaccines are also sold in South Africa. During the vector season, the viruses in attenuated vaccines can be transmitted to unvaccinated animals, and could reassort with field strains, resulting in new viral strains. In addition, vaccines can cause fetal malformations in pregnant ewes.

Public Health

Bluetongue is not a significant threat to human health. However, one human infection has been documented in a laboratory worker and reasonable precautions should be taken while working with this virus.

For More Information

- Manual for the Recognition of Exotic Diseases of Livestock
<http://www.spc.int/rahs/>
- The Merck Veterinary Manual
<http://www.merckvetmanual.com/mvm/index.jsp>
- United States Animal Health Association.
Foreign Animal Diseases
http://www.vet.uga.edu/vpp/gray_book02/fad/index.php
- World Organization for Animal Health (OIE)
<http://www.oie.int>
- World Organization for Animal Health (OIE). Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/eng/normes/mmanual/a_summry.htm

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*Link defunct as of 2006