



Bluetongue Backgrounder

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Causative agent

Bluetongue (BT) is caused by an orbivirus member of the Reoviridae family. Reoviridae are 60 to 80 nanometers (nm) in diameter, unenveloped, double-stranded RNA viruses. They have an icosahedral shape, indicating that the nucleocapsid has 20 symmetrical faces. Reoviruses are unique in that the capsid has three layers. African horse sickness and equine encephalosis viruses are also orbiviruses. There are 24 immunologically distinct serotypes of the BT virus.

Natural distribution

BT primarily affects sheep, and susceptibility to disease varies with breed. European breeds are more susceptible to infection with BT virus. White-tailed deer and pronghorn are very susceptible to the BT virus, and may be more severely affected than sheep. Cattle, goats, North American elk, elephants, and dromedary camels may become infected with the BT virus, but infection is inapparent and they potentially serve as reservoirs.

BT was first recognized in the late 1800s in South Africa, but was not described in detail until the early 1900s. It was first reported in the United States in the 1950s. Since 1999, outbreaks have occurred in Belgium, Bulgaria, Croatia, France, Germany, Greece, Italy, Kosovo, Luxembourg, Macedonia, Morocco, the Netherlands, Spain (Balearic Islands), and Yugoslavia.

Distribution of the BT virus is generally limited to geographic areas between the latitudes of 40°N and 35°S, but it has been documented to exist as far north as 53°N. The virus is present where its vectors exist, but clinical disease is not always associated with presence of the virus. Because the virus is transmitted by an insect vector, disease is associated with the seasonal activity of the vector. In the United States, BT is generally observed in the South and Southwest in the late summer and early fall.

The World Organization for Animal Health (OIE) classifies BT as a notifiable disease because of its potential for rapid spread and substantial impact on the international trade of animals and animal products. The United States Agricultural Bioterrorism Act of 2002 recognized BT as an agent that could pose a severe threat to animal health, animal and human health, or animal products in the United States. The BT virus exists in the United States, and serves as an impediment to cattle movement from the United States to Canada, Australia, New Zealand, and the European Union.

Transmission

BT is an infectious disease, but it is not contagious; that is, the disease is caused by an infectious agent, but it is not spread from infected animals to susceptible animals by direct contact. Insect vectors of BT virus are members of the *Culicoides* family, or midges. Because the virus is transmitted by arthropods, it is considered to be an arbovirus. Prior to 2006, all bluetongue outbreaks were linked to an African insect vector, and the incidence of cases would decrease with climate-induced insect vector death. An exotic strain of the bluetongue virus caused an outbreak in Sardinia, Italy in November 2006; although believed to be African in origin, this strain had not previously affected animals in Europe. During the 2006 outbreak in Belgium, France, Germany, Luxembourg, and the Netherlands, the insect vector was identified as a biting midge that had adapted to the climate of Northern Europe; this may have substantial impact on the potential for further spread of the disease in Europe. In addition, the possibility that endemic areas will be established in Northern Europe may increase seasonal disease recurrence and provide additional challenges for disease control measures.

After an adult midge takes a blood meal from an animal infected with the BT virus, approximately 2 weeks are required for the virus to amplify before it can be effectively transmitted to a

susceptible animal during a subsequent blood meal. The BT virus does not survive in the larval form of the insect. Once infected, midges remain infected for life.

The incubation period for BT in sheep is 5 to 20 days. Deer incubate the virus for 7 to 12 days prior to onset of clinical signs. The incubation period in cattle is 3 to 10 days, and the virus is present in the animal's red blood cells for 60 to 80 days.

A pregnant cow can transmit BT virus to its calf *in utero*. Cows can become infected via the insect vector or via virus-infected semen, but the latter route of transmission is not believed to play a major role in the epidemiology of BT.

Only one case of human infection with BT virus has been reported; this infection occurred after exposure to the virus in a laboratory.

Clinical Signs

Severity of clinical signs depends on the animal's age, breed, and health status, in addition to the viral serotype and number of infecting viral particles. Clinical signs of BT in sheep include fever (up to 107.6°F), depression, emaciation, and swelling of the muzzle, eyelids, and ears. The mucosal surfaces of the mouth become inflamed and ulcerated, and this is followed by erosion and sloughing. The tissues may become so swollen that they become deprived of oxygen; the tongue thereby develops a characteristic blue or purple color that gives the disease its name. Swallowing and breathing are often compromised by severe swelling, and nasal discharge or blood-tinged drool may be observed. Lameness, caused by inflammation of the coronary bands of the hooves, may be observed. The hooves may slough when disease is severe, or if animals are moved over long distances while ill. Fetal resorption or abortion may be observed if ewes are infected during the first trimester of pregnancy. If death occurs, it is generally within 8 to 10 days. For surviving animals, the recovery period is prolonged and may be associated with hair loss, sterility, cracked hooves and muzzle, and delayed growth. A dark line may be observed in the hoof wall, and a break in the wool may be observed 3 to 4 weeks after the fever has resolved.

White-tailed deer and pronghorn infected with BT virus develop fulminating hemorrhagic disease, and sudden death may occur. Clinical signs observed in these species are identical to those observed with infection by epizootic hemorrhagic disease (EHD) virus.

Although infection with the BT virus is most often clinically inapparent in cattle, clinical disease does occur. Clinical signs observed are similar to those observed in sheep. Foot lesions may resemble those observed with foot-and-mouth disease [\[Insert link to FMD background\]](#), and tongue swelling may mimic that observed with mycotic (fungal) stomatitis. Hyperesthesia (increased skin sensitivity), erect hair, and thickened skin may also be observed. Reproductive failure may occur, and infected bulls may be temporarily sterile. Clinical disease in cattle is most likely precipitated by prior sensitization to a related orbivirus. Goats are more resistant to the BT virus than sheep or cattle, but may also develop similar clinical signs.

Infection in sheep and cattle with BT virus that is contracted in utero can induce hydranencephaly (a congenital condition in which fluid-filled cavities are present in place of the cerebrum) and brain cysts, resulting in "dummy calves." Natural infection with BT virus strains derived from live virus vaccines has occasionally induced these conditions.

Other diseases and conditions with clinical signs similar to those observed with BT include contagious ecthyma, foot-and-mouth disease, vesicular stomatitis, photosensitization, pneumonia, foot rot, plant poisoning, coenurosis, and pests des petits ruminants.

Diagnosis

A tentative diagnosis of BT can be made based on characteristic lesions in susceptible populations during high risk season. However, confirmation of BT requires virus isolation and identification. BT virus can be isolated, using cell culture, sheep inoculation, or inoculation of embryonated eggs. Immunofluorescence, antigen capture enzyme-linked immunosorbent assay, and immunospot testing are immunologic methods of identification. Serum neutralization allows identification, but serotyping with this test is complicated by cross-reactions among serotypes. Polymerase chain reaction (PCR) allows faster identification; this test only requires several days to complete, whereas traditional methods require 3 to 4 weeks for results. Serologic tests available for

diagnosis of BT include competitive enzyme-linked immunosorbent assay (CELISA), agar gel immunodiffusion (AGID), and complement fixation (CF).

Heparinized blood samples from living animals are usually sufficient for virus isolation and identification. It is preferred that blood cells be washed in saline containing antimicrobials and resuspended in physiologic saline prior to submission; the washing procedure reduces the amount of antibodies that may neutralize the virus (and therefore impede identification) if cells rupture. Spleen, liver, red bone marrow, blood from the heart, and lymph nodes can be harvested at necropsy and submitted for virus isolation and identification. Samples should be preserved at 4 C, and not frozen.

Treatment

Bluetongue is a reportable disease. State or federal animal health officials should be immediately notified when clinical signs of BT are observed. There is no effective treatment.

Morbidity and Mortality

Morbidity of BT in sheep can approach 100%. The case fatality rate (the number of affected animals that die from the disease) in sheep with BT is variable, but can reach 70% during some outbreaks. Morbidity in cattle and North American elk is 5% or less, and death is rare.

Morbidity in white-tailed deer, pronghorn antelope, and bighorn sheep approaches 100%. White-tailed deer and pronghorn exhibit case fatality rates of 80 to 90%, whereas bighorn sheep exhibit case fatality rates of 50% or less.

Prevention and Control

Housing animals indoors between dusk and dawn (the time of highest vector activity) can decrease transmission of BT virus by decreasing animals' exposure to the insect vector, but is not commonly practiced. Avoiding areas of high vector activity and density decreases the likelihood that animals will be bitten by potentially infected insects. Water management that reduces opportunities for midges to breed and the use of insecticides and insect repellents are beneficial in reducing risk of transmission. In addition, management practices that minimize animal stress optimize animals' health and resistance to BT.

Cases or outbreaks of BT in nonendemic areas necessitate strict quarantine and isolation. Traditional measures for control include restriction of animal movement, vector control, measures to decrease exposure to the vector, and slaughter of viremic animals.

Modified live virus vaccines are available; for vaccination to be effective, the serotype of the vaccine must match that causing infection in the field. Attenuated vaccines are capable of inducing reproductive failure in sheep.

BT virus is susceptible to ethanol, formalin, acetic acid, phenylphenol, and sodium hypochlorite solutions, and heat above 50 C. The virus is able to survive for months at 4 C and 20 C, and the presence of protein (including blood) increases its survival.