

Bluetongue: The Silent Threat Carried by Tiny Biting Midges

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Introduction

Bluetongue is an economically important viral disease of domestic and wild ruminants that occurs across many parts of the world. Despite its striking name, the disease does not spread by ordinary contact between animals. Instead, it is carried from one animal to another by tiny blood-feeding insects known as *Culicoides* biting midges. This makes bluetongue a fascinating example of the close relationship between animals, insects, climate and the environment.

Sheep are generally the species in which severe clinical disease is most frequently recognized, while cattle and goats may often become infected without showing obvious signs. However, clinically silent animals can still play an important epidemiological role because infected cattle may remain viraemic for prolonged periods and serve as a source of virus for feeding midges.

Bluetongue is distributed across Africa, Asia, Australia, Europe, North America and other tropical and subtropical regions wherever competent *Culicoides* vectors are present. Recent outbreaks in Europe and the detection of emerging strains have once again highlighted the changing epidemiology of bluetongue. A 2025 review reported important outbreaks involving BTV-3 and BTV-12 during 2024–2025 and emphasized the expanding geographical importance of bluetongue in association with vector distribution, climate and animal movement.

Etiology

Bluetongue is caused by **bluetongue virus (BTV)**, an RNA virus belonging to the genus *Orbivirus* of the family *Reoviridae*. BTV possesses a segmented double-stranded RNA genome and has considerable genetic and antigenic diversity. The number of recognized serotypes has increased as new viruses have been

identified; recent literature describes at least 26–28 serotypes, although the exact number depends on the classification system and newly recognized strains.

The existence of multiple serotypes is one of the major challenges in bluetongue control. Immunity against one serotype may provide little or no protection against another. Consequently, identification of the circulating virus is extremely important when designing vaccination and disease-control programmes.

Different BTV strains also differ considerably in their ability to produce clinical disease. Some infections remain completely unnoticed, whereas others can cause severe haemorrhagic disease and death, particularly in susceptible sheep. The severity therefore depends not only on the virus but also on the species, breed, age, immune status and environmental conditions of the affected animals.

Transmission

The most important feature of bluetongue transmission is its dependence on ***Culicoides* biting midges**. A female midge becomes infected when it takes a blood meal from a viraemic animal. The virus then replicates within the insect, eventually reaching its salivary glands. When the infected midge feeds on another susceptible

animal, the virus can be transmitted through its bite.

This means that bluetongue is fundamentally different from highly contagious diseases such as foot-and-mouth disease. An infected sheep cannot simply transmit BTV to another sheep by standing next to it. A competent vector is normally required for efficient transmission. Cattle are particularly important in the epidemiology of bluetongue because they can develop prolonged viraemia while showing few or no clinical signs. They can therefore act as an important reservoir of virus for feeding midges. Vector activity is strongly influenced by temperature, rainfall, humidity and suitable breeding habitats. Consequently, bluetongue transmission often increases during periods favourable for *Culicoides* populations. Wind can also contribute to the geographical spread of infected midges. This is particularly important because *Culicoides* are small insects capable of being transported considerable distances under favourable atmospheric conditions. Although vector transmission is overwhelmingly the major route, BTV has also been detected in semen and can occasionally be transmitted vertically from an infected dam to the fetus. These routes are epidemiologically much less important than transmission by biting midges.

Pathogenesis

After an infected midge bites a susceptible animal, BTV enters the bloodstream and initially infects cells associated with the immune and vascular systems. The virus replicates in tissues including lymphoid organs and endothelial cells lining blood vessels. The development of vascular injury and increased vascular permeability is central to the disease process. Damage to small blood vessels can result in leakage of fluid, oedema, haemorrhage and tissue injury. This vascular damage explains many of the characteristic clinical manifestations of bluetongue, including swelling of the face and lips, haemorrhages, congestion of mucous membranes and lesions around the coronary band. The tongue may become swollen and, in severe cases, cyanotic, producing the classical “blue tongue” appearance from which the disease receives its name. However, despite the name, a

blue tongue is not present in every affected animal and should not be considered essential for diagnosis. The inflammatory and vascular changes can also affect the feet. Inflammation of the coronary band may lead to pain, lameness and, in severe cases, difficulty standing or walking. Pregnant animals may suffer reproductive consequences, including abortion or fetal infection. Certain BTV strains may cross the placenta and cause fetal disease or congenital abnormalities. Young animals and particularly susceptible sheep may develop severe disease. In some cases, the virus can affect skeletal and cardiac muscle, and severe myocardial damage may contribute to death.

Clinical Signs

The severity of bluetongue varies considerably. Many infected animals show no obvious clinical signs, while susceptible sheep can develop severe and sometimes fatal disease. This difference between infection and clinical disease is one of the most important features of bluetongue epidemiology. In clinically affected sheep, the disease may begin with **fever, depression, weakness and loss of appetite.** As the disease progresses, swelling and inflammation may develop around the lips, face and tongue. Excessive salivation and nasal discharge may occur, and the animal may have difficulty eating because of painful lesions in the mouth. The tongue may become swollen and, in severe cases, appear bluish because of impaired circulation and cyanosis. However, the classical blue coloration is relatively uncommon compared with the other manifestations of the disease. Oral lesions may include congestion, haemorrhage, erosions and ulceration of the mucous membranes. Animals may become progressively weak and lose body condition.

Diagnosis

Clinical suspicion is an important first step, particularly when sheep develop fever, facial swelling, oral lesions, salivation, lameness and other compatible signs during periods of high *Culicoides* activity. However, clinical signs alone are not sufficient to confirm bluetongue because several other diseases can produce similar lesions and systemic effects.

Laboratory confirmation is therefore essential.

Real-time reverse transcription polymerase chain reaction (RT-qPCR) is widely used for detecting BTV RNA and can provide rapid evidence of active infection. Serological tests, including ELISA, can be used to identify antibodies and assess previous exposure at the herd or population level.

Virus isolation and sequencing can provide additional information, particularly during outbreaks. Molecular characterization is increasingly valuable because it helps determine the serotype and genetic lineage involved and can assist in understanding the movement and evolution of BTV. The choice of sample depends on the stage of infection and diagnostic objective. Blood, particularly whole blood collected appropriately, can be useful for molecular detection because BTV associates with erythrocytes and can remain detectable for prolonged periods. Recent advances in molecular diagnostics have improved the speed and sensitivity with which BTV infections can be identified. Emerging field-deployable molecular assays are also being investigated to make diagnosis more accessible in regions where laboratory infrastructure is limited.

Treatment

There is currently **no specific antiviral treatment that reliably eliminates BTV infection**. Management is therefore mainly supportive and should be undertaken under veterinary supervision. Affected animals should be provided with adequate water, easily accessible feed and comfortable housing. Because oral lesions can make eating painful, soft and palatable feed may help maintain nutritional intake. Animals with severe lameness should be protected from unnecessary movement and trauma.

Secondary bacterial infections may require appropriate treatment based on veterinary assessment. Anti-inflammatory or other supportive medications may be considered depending on the clinical condition and applicable veterinary guidelines. However, treatment of individual animals does not address the major epidemiological problem. Because the virus is transmitted by biting midges, controlling

exposure to vectors and preventing new infections are central to disease management.

Control and Prevention

The control of bluetongue requires a combination of **vaccination, vector surveillance, animal surveillance and movement management**. Vaccination is considered one of the most effective practical approaches for reducing losses in endemic regions. However, because BTV has numerous serotypes and considerable antigenic variation, the vaccine must be appropriately matched to the serotypes or strains of concern. A vaccine against one serotype cannot automatically be assumed to provide adequate protection against another. Vector control can also contribute to disease reduction, although complete elimination of *Culicoides* populations is generally impractical. Management measures that reduce exposure to biting midges can be useful, particularly during periods of intense vector activity. Housing animals during peak vector activity, reducing exposure to vector habitats where practical and maintaining suitable environmental management can help lower transmission risk.

Surveillance should include both **animal surveillance and vector surveillance**. Sentinel animals can help identify virus circulation, while monitoring *Culicoides* populations provides information about periods and areas of increased transmission risk.

Movement of infected animals should be controlled during outbreaks. Introduction of susceptible animals into areas with active vector transmission should be carefully managed, and appropriate quarantine or movement restrictions may be required by veterinary authorities.

Conclusion

Bluetongue may be named after one of its most dramatic clinical signs, but the real story of the disease lies in the invisible relationship between a virus, a biting midge and a susceptible ruminant. The disease is caused by BTV and is transmitted mainly by *Culicoides* midges, with sheep often experiencing the most severe clinical disease while cattle and goats may remain apparently healthy despite infection. The disease can produce fever, facial and oral swelling, salivation, mucosal lesions, lameness, wool



abnormalities and, in severe cases, death. Its economic impact extends beyond mortality through reduced production, reproductive losses, surveillance costs and restrictions on animal movement and trade. Recent research has shown that bluetongue continues to evolve and expand into new epidemiological settings. Evidence from India also demonstrates substantial variation in prevalence and geographical risk, emphasizing the need for targeted surveillance and vaccination strategies. The most effective approach is therefore not a single intervention but a **combination of vaccination, early diagnosis, vector surveillance, animal surveillance, appropriate movement control and improved understanding of climatic and ecological risk factors.**

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