established where tabanids, *Stomoxys, Simulium, Chrysops,* and other biting flies are prevalent. It could be introduced into the United States of America and become established, creating a problem of enormous economic significance.

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Bluetongue and Related Diseases

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Identification

A. *Definition.* Infectious, non-contagious viral diseases of ruminants transmitted by insects and characterized by inflammation and congestion of the mucous membranes, leading to cyanosis, edema, hemorrages and ulceration.

B. *Etiology.* Bluetongue (BT) virus is the type species of the genus *Orbivirus* in the family Reoviridae, which are icosahedral, double-stranded RNA viruses. Orbiviruses are arthropod-borne viruses approximately 50 to 70 nanometers in diameter characterized by extreme lability at low pH and resistance to inactivation by lipid solvents such as ether, chloroform, and deoxycholate.[9,20,22,45,88,975,116,117,118] Temperature sensitivity of BT virus is peculiar as the virus has been found to survive in a preserved defibrinated blood sample for 25 years at room temperature[91] and is very stable at refrigerator temperatures but freezing has a deleterious effect on the virus.

Twenty serotypes of BT virus have been found in the world.[21,44,46,91,104] Serotypes 10, 11, and 13 exist in the U.S. and other parts of the world including Africa,[44] but serotype 17 has only been found in the U.S.[5] Epizootic Hemorrhagic Disease (EHD) virus, an orbivirus related to BT virus, have been found in North America.[5] Two serotypes of EHD or EHD-like virus have also been isolated from gnats in Nigeria, but it is not known if these represent new serotypes.[86] Ibaraki virus was isolated from cattle in Japan in the early 1960's [94] and has been found to be very closely related if not identical to EHDV.[16]

C. *History.* In the late 1800's BT was described as a disease of imported European breeds of sheep in South Africa although it had apparently been recognized since the first importations of sheep in the 1700's.[20,43,47,48] In 1934, BT virus was isolated and identified from cattle in South Africa with a disease described as "Pseudo-Foot-and-Mouth."[8] During the 1940's, BT appeared throughout medeastern Asia in Palestine, [33,60] Syria, and Turkey,[35,120] and in 1943, the most severe epizootic of BT known occurred on the island of Cyprus.[30,109] The disease appeared in Portugal and Spain in the mid 1950's in both sheep and cattle.[63,101] In the United States, a clinical entity called "mycotic stomatitis" in cattle was described between 1889 and 1904 which was identical to the description of BT in cattle in South Africa and elsewhere, and which is now recognized as being caused by BT or EHD virus.[85] BT was described in Texas sheep in 1948 and reported as "Soremuzzle" but it wasn't until 1952 that the virus was isolated and identified in California sheep.[79,80] BT virus was first recovered from U.S. cattle in 1959 from cases clinically diagnosed as "mycotic stomatitis".[14]

Epizootic Hemorrhagic Disease (EHD) virus was first recovered from a white-tailed deer in a major dieoff in New Jersey in 1955.[110] The virus has since been recovered from cattle with clinical disease identical to BT in the U.S.[84] and has been isolated from gnats in Nigeria.[62,86]

Ibaraki virus, an orbivirus closely related to EHD virus, was isolated from a major epizootic in cattle in Japan in 1959.[93,94,95]

Signs

A. *Clinical features.* Bluetongue in U.S. sheep is much less severe than the disease seen in Africa or Mideastern Asia.[20] In the U.S., bluetongue epizootics usually occur in the late summer months and early fall until the first frost in the more temperate areas. In the southern states the disease

can occur at almost any time of the year, but most frequently in the late spring or early summer months. All ages of sheep are susceptible, but in endemic areas usually only lambs will be affected. Among fully susceptible sheep 80 to 100 percent of the flock may become sick. Mortality may be quite variable and is frequently associated with secondary stress factors rather than the specific effects of the virus. Onset of BT in sheep begins with an increased respiration rate, fever, salivation and frothing of the mouth, hyperemia and congestion of the oral mucous membranes, and depression. The increased respiration rate in sheep occurs shortly before or at the onset of the fever and is usually associated with the peak viremia. Peak fevers usually occur about one day after the onset of respiratory distress. Rectal body temperatures frequently reach 41.6 C (107° F) but usually will be between 40 to 41 C (104 to 106° F). Salivation and frothing at the mouth usually begin at the time of the peak fever or shortly afterwards. Hyperemia and congestion of the mucous membranes of the mouth, lips, and nasal mucosa develop soon following the appearance of salivation. Hyperemia can vary from a slightly increased pinkness of the mucous membranes to a deep scarlet coloration and eventually cyanosis with a blue discoloration resulting in the name "bluetongue." Many sheep will spontaneously recover after a few days but in the more acutely affected animals, congestion and edema of the oral mucous membranes become more intense and eventually necrotic ulcerations of the lips, dental pad, tip of the tongue and gingiva may develop, and laminitis may occur. Laminitis can be extremely variable in its appearance. In some cases it may be the only sign observed and in other cases it may not be observed at all. After the acute disease subsides many sheep will be unthrifty and some may develop chronic pneumonia.[37,38,64,67,77,79,81] In Africa the disease is much the same except for the degree of severity of the signs and the proportion of sheep that develop the more severe form of the disease.[38,43,47,48,111,112,113] Inflammation of the oral mucous membranes often leads to massive submucosal or subepithelial hemorrhages. Necrosis of muscle fibers is usually extensive and sheep may die during the acute stage of the disease with massive hemorrhages in the heart.

Bluetongue in cattle is primarily inapparent with clinical disease in only 5 to 10 percent of the infected cattle. Mortality is low and usually the result of secondary infections. Descriptions of the disease in South Africa, Portugal, Israel, and the United States are very similar.[8,14,60] BT virus is easily transmitted to cattle, but acute clinical signs in experimentally infected cattle are rare.[3] In experimentally infected cattle, fever and leukopenia usually are seen 6 to 8 days after infection.[51,70,71] Onset of clinical BT in cattle is first observed as hyperemia of the mucous membranes and of the exposed epithelium, especially on the udder and teats. Milk production may drop in dairy cows.

Cattle will usually be stiff and lame with a myositis which they will warm out of if driven. Peak fevers develop in cattle usually about the same time as the hyperemia of the mucous membranes and may reach 41 C (106° F) but fevers from 39.5 to 40.5 C (103 to 105°) are more common. Shortly after onset of the febrile response, cattle begin salivating profusely and hyperemia and congestion of the oral mucous membranes will become more extensive. Ulcerations may develop on the gingival or buccal mucosa or on the tongue. The most frequently seen lesion is a superficial ulcer on the dental pad. Necrosis of the epithelium of the muzzle may occur causing a "burnt muzzle" in the cattle. Frequently this will be the first sign noticed by a farmer. Laminitis usually develops in cattle about the same time as the oral ulcerations and is characterized by hyperemia and swelling of the sensitive laminae of the corium without any initial surface lesions. If the animal is forced to walk on hard ground, there may be extensive damage to the sensitive laminae and the hoof may be lost. A few sick cattle will develop an extensive pityriasis and necrosis of the skin with sloughing and growth of new underlying epithelium. Lactating dairy or beef cattle will frequently develop scabs on the teats due to irritation of the sensitized epithelium by a nursing calf or milking machine. The most significant losses in cattle are frequently due to infertility, abortions, deformed and weak calves in chronically infected herds.[71] The acute clinical signs of stomatitis and laminitis are rarely seen in these herds.[69]

In goats BT infection is usually inapparent.[20,113] Clinical BT has been reported in goats in Israel[5] and India[105] similar to the disease in sheep. There does appear to be some difference in susceptibility of goats to different strains of BT and there may also be some differences in susceptibility of various breeds of goats.[66]

In white-tailed deer, BT and EHD produce an acute to peracute highly fatal hemorrhagic disease resembling the most severe forms of BT seen in sheep in Africa.[20,55,98] BT in pronghorn antelope also resembles the disease in white-tailed deer, but the mortality may be somewhat lower. BT has also been found in mule deer, but the disease is milder than in white-tailed deer. BT in bighorn sheep resembles the disease in domestic sheep.[103] In the North American elk (Wapiti) BT appears to be very similar to the disease in domestic cattle.[89,115]

Epizootic Hemorrhagic Disease in white-tailed deer has caused massive dieoffs throughout various areas of the U.S. and Canada. The disease in white-tailed deer is observed in the field as sudden deaths in deer and deer being found dead along streams with hemorrhage from the body portals and swollen black or blue tongues. Recovered deer will be extremely gaunt and emaciated and may have separations of the hoof or sloughed hooves.[56,110,123] Epizootics of BT in white-tailed deer have been reported occurring simultaneously in similar or adjacent geographic areas. EHD has been isolated from cattle during BT-like epizootics.[84] Clinical signs, morbidity, and mortality of EHD in cattle appear to be identical to BT. Although deaths have been reported in pronghorn antelope and mule deer in association with dieoffs in white-tailed deer in which EHD virus was recovered neither natural or experimental

infection of these or other wild ruminants has been reported.[17] Sheep develop a transitory inapparent infection when experimentally infected with EHD virus but have not developed overt clinical disease. Descriptions of Ibaraki disease in Japanese cattle are identical to the descriptions of BT in cattle from other parts of the world. Ibaraki virus was originally thought to be a BT virus.[93] Workers in Japan have reported that the virus does not cause disease in sheep.[95]

B. *Incubation period.* Experimentally infected sheep and cattle develop the initial clinical signs of BT 6 to 8 days after inoculation of virus or by bites of the biological vector. Incubation of BT and EHD in white-tailed deer can be quite variable and range from 5 to 10 days or more. In the field, new cases usually cease to appear 10 to 14 days after the first frost when an epizootic is interrupted by freezing weather.

Diagnosis

A. *In the field.* In sheep the high morbidity with fever, edema of the head and neck, and swollen congested mucous membranes with ulcerations is usually sufficient to make a clinical diagnosis of BT. In cattle the characteristic low morbidity makes the disease more difficult to diagnose. Differentiation between BT and EHD infection cannot be made in cattle, deer, or other ruminants affected by both viruses.

B. *Laboratory.* Laboratory confirmation is necessary to differentiate BT from EHD viral infection. Viral isolation may be by sheep inoculation (for BT only),[81] inoculation of embryonating chicken eggs by the intravenous route[27,30] or direct inoculation of cell cultures (best for EHD).[4] The best samples for isolation of BT or EHD virus is fresh whole blood collected from an animal during the peak febrile stage of infection. Ten to 50 ml. of blood should be collected in heparin or other anticoagulant such as sodium citrate. Those blood samples should either be shipped promptly to the laboratory on wet ice or if they cannot be shipped to arrive at the laboratory within 48 hours after collection, they should be centrifuged and the washed RBC's should be sent to the laboratory on wet ice.[64] To wash the RBC's and buffy coat resuspended in normal saline containing 1 percent phenol, then recentrifuged. The supernate should be removed and discarded and the RBC's resuspended in sufficient phenolized saline to restore the sample to its original volume. From dead animals red bone marrow or spleen is preferred for viral isolation. To collect bone marrow samples, the entire femur of a young animal or the sternum of a mature animal can usually be shipped to the laboratory on wet ice.

Diagnosis by serological tests for BT or EHD can be by either a complement fixation[10] or an immunodiffusion test.[50] Paired serums 2 to 4 weeks apart are necessary to make a presumptive diagnosis. There is some cross reaction between BT and EHD virus in both these tests so interpretation of results can often be confusing. Latently infected carrier cattle may be serologically negative.

Prognosis

Mortality due to BT in sheep is extremely variable and prognosis usually depends more on secondary stress factors than on the disease itself. Healthy, relatively parasite-free sheep will usually have low mortality unless they are severely stressed by shipping or cold wet weather. Most cattle will recover from BT or EHD, but pregnant cattle may have a wide variety of problems in their calves. Congenital infection of calves may result in fetal death with resorption, abortion, stillborn calves, live deformed calves, apparently healthy calves which survive only a few days or weeks, or live apparently normal calves which may remain latent carriers of BT virus for the remainder of their lives.[71,72,73] Bulls infected either as adults or infected *in utero* may shed BT virus in their semen[75] and can transmit the virus to cows by natural breeding. Recovered sheep or cattle probably remain immune to infection with the same virus type for life, but may be sensitized and have a more severe response if infected with virus of another type.

Epizootiology

A. *Geographic Distribution.* BT virus has been found throughout most of Africa and Asia along the eastern end of the Mediterranean Sea, Pakistan, and India.[1,30,33,43,105,106,120] In South America BT has been reported from Peru, but the diagnosis was never confirmed.[108] Recently a BT virus was recovered from Culicoides gnats trapped in Australia.[102] Only the United States has reported and confirmed BT on the North American continent, although there have been unconfirmed reports of BT in Mexico and serological evidence in British Columbia, Canada.

In the United States BT virus has been isolated from sheep, cattle, or deer in 30 of the conterminous states. Serological evidence of BT has been found in every state except Alaska and Rhode Island.

EHD virus has been found primarily in North America in the U.S. and Canada.[123] Isolates identified as EHD have been recovered from gnats in Nigeria.[62,86]

Ibaraki virus has only been found in Japan and serological evidence has been found in Taiwan and Bali, Indonesia.[49]

B. *Transmission.* Transmission of BTV and EHDV is principally by small blood sucking gnats of the genus *Culicoides. C. pallidipennis* is the principal vector of BTV in Africa and Asia,[18,92] and *C. variipennis* is the principal vector of BTV and EHDV in the United States.[25,27,54] Two other species have been found infected with BTV in Kenya.[92] The vectors of Ibaraki virus have not been reported.

Mechanical transmission can occur by transfer of blood from an infected animal to a susceptible one. Transfer could be intentional to transmit the disease or unintentional by use of contaminated instruments or by contaminated mouth parts of biting insects.

Vertical transmission from an infected dam to her offspring has been demonstrated in cattle,[51,71] sheep, [33,107]

and elk (Wapiti).[115] Vertical transmission from infected bulls through the semen to his progeny has also been demonstrated, as well as venereal contact transmission to the cow bred by the bull. Transmission through the semen has only been demonstrated by natural service, but possibly could also occur by artificial insemination.[75]

C. *Hosts.* All domestic and wild ruminants are considered susceptible to infection with BT virus to some degree. Cattle are probably the principal reservoir host, although goats may play an important role in some parts of the world.[5,105] Other wild ruminant species, such as the North American Elk (Wapiti), may also be reservoirs but their importance is diminished by their limited geographic distribution and movement.

Control and Eradication

A. *Preventive Measures.* As long as cattle move freely in the U.S., there is little that anyone can do to prevent epizootics of BT or EHD from occurring or to prevent their own herd or flock from becoming infected. Quarantines of infected herds are useless because arthropodborne viruses are area-wide problems and if one herd in an area is infected, then nearly every other herd in the area may also be infected. Quarantine of an entire infected area during the vector season may be effective if it could be done. **It is very important that U.S. livestock producers protect themselves against introduction of more pathogenic strains of virus as those presently found in the U.S. have relatively low pathogenicity.** In order to prevent introduction of new types of virus, there must be control, not only of domestic livestock imported into the U.S., but any wild ruminants which may carry the virus as well. Quarantining or restricting movement of animals from a zoological park will not prevent a vector from picking up an arthropod-borne disease and transmitting it to domestic livestock or indigenous wild species of animals.

B. *Immunization.* A live-attenuated BT vaccine is currently available for use in sheep only.[57] The vaccine has only serotype 10 BT virus in it so it is not effective against serotypes 11, 13, or 17. The vaccine will also cause abortions and deformities in lambs if pregnant ewes are vaccinated.[107]

C. *Sanitation and disinfection.* Vector control is probably one of the most effective ways of controlling BT epizootics. *Culicoides variipennis* larvae are found in soft, silty muddy areas along the edges of ponds, streams or ditches with slow moving water, stock tank overflows, or septic tank overflows, particularly when these areas are heavily contaminated with fecal material.[52] Changing contours of ponds, ditches, or streams from gently sloping muddy banks to steep graveled banks will aid in controlling *C. variipennis.* Correcting stock tank or septic tank overflows will also reduce numbers of the gnats.

Insecticides are of limited value although general applications of insecticides around feedlots may be of some benefit in controlling *Culicoides spp.* Environmental impact of insecticide application must always be considered before their use.

It has been found that the ability of *C. variipennis* to transmit BT is genetically controlled and some insects are unable to transmit the virus.[52] Potential future control of BT or EHD could involve genetic alteration of wild populations of *C. variipennis* to a non-transmitting population.

D. *Treatment.* There is not a specific treatment for BT or EHD. Antibiotics are effective in reducing secondary infections and antihistamines apparently provide some symptomatic relief, but corticosteroids are contra-indicated. General supportive therapy of sick animals by confinement to small pens or barns with ready access to soft feed and water will reduce losses.

E. *Reporting.* Any case of stomatitis from any cause should be reported to State or Federal veterinary officials, particularly if associated with lameness. Unless BT is very common in the area, all cases should be laboratory confirmed and any unusually severe cases must be promptly reported and confirmed.

Public Health Aspects

Natural Bluetongue, EHD, or Ibaraki virus infections have not been reported in man. Colorado Tick Fever (CTV) virus is an Orbivirus that does affect man in the U.S.A.

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