Algunos resúmenes sobre el tema de Lengua azul en Ovinos

Studies on the transmission of Venezuelan equine encephalitis virus by Colombian simuliidae (Diptera). Homan EJ, Zuluaga FN, Yuill TM, Lorbacher H.
The ability of Simulium mexicanum and Simulium metallicum to serve as biological or mechanical vectors of an enzootic and an epizootic strain of Venezuelan equine encephalitis (VEE) virus was examined. Guinea pigs were inoculated with the epizootic Cordoba strain or the enzootic RPVP407 strain of VEE virus. Wild-caught adult Simuliidae were fed on the viremic guinea pigs and the virus content of groups of flies was determined at daily intervals post-engorgement to test for viral replication. Flies were refed on suckling mice at greater than or equal to 8 days post-engorgement to test for biological transmission. Other flies were interrupted while feeding on viremic guinea pigs and refed on suckling mice to test for mechanical transmission. Neither S. mexicanum nor S. metallicum appear to be efficient vectors of either strain of VEE virus, although occasional mechanical transmission was obtained. Titers of virus in flies decreased rapidly after engorgement and from 3-12 days post-engorgement virus was detected only in 5%-25% of both species of flies. Although earlier field evidence implicated both S. mexicanum and S. metallicum as vectors of epizootic VEE, we conclude that it is highly unlikely that they play an important role as vectors of the virus in nature.

College of Veterinary Medicine, University of Florida, Gainesville 32610.
A study of the epidemiology of bluetongue viruses is in progress with the collaboration of 11 Central American and Caribbean countries. To date, over 200 bluetongue virus isolates have been obtained from cattle and sheep in sentinel groups distributed in the participating countries. Bluetongue serotypes identified include 1, 3, 6, and 12, virus types not previously recorded in the Western Hemisphere. Although the clinical impact of bluetongue virus infections in this hyperendemic environment appears to be minimal, the ubiquity of infection causes restrictions on the export of ruminant livestock and germ plasm. The stability of the Caribbean region ecosystem and the long-range implications of the interface with the northern temperate bluetongue virus ecosystem are reviewed.

Serological observations on the epidemiology of bluetongue virus infections in the Caribbean and Florida. Gibbs EP, Greiner EC.
Serological surveys of cattle, sheep and goats have confirmed that infection with bluetongue virus (BTV) is common in Florida, Puerto Rico and St. Croix in the USA, in the Caribbean countries of Jamaica, St. Kitts/Nevis, Antigua, St. Lucia, Barbados, Grenada, Trinidad and Tobago, and the Bahamas, and in the South American countries of Guyana and Suriname. In most countries, over 50% of ruminant livestock have antibody to BTV as assessed by the bluetongue immunodiffusion
A sentinel animal system operating in Florida and 4 islands in the Caribbean has established that the transmission of BTV is seasonal, with most animals becoming infected in late summer and fall. In Florida, it appears that there may be some years when little virus transmission occurs among cattle. Examination of sera from yearling animals and sentinels in the region for antibody to the range of serotypes of BTV recognized worldwide, has resulted in a) the isolation of BTV type 2 from cattle in Florida - the 1st time this virus has been identified in the Western Hemisphere - and b) the recognition that the range of serotypes of BTV present in the Caribbean may be different from those in the USA. No clinical disease has been associated with BTV during the period of these studies (1979-83).

Serological survey of ruminant livestock in some countries of the Caribbean region and South America for antibody to bluetongue virus.
Gibbs EP, Greiner EC, Alexander FC, King TH, Roach CJ.
A serological survey of 6250 sera from cattle, sheep and goats in seven Caribbean and two South American countries showed that antibody to bluetongue virus was widely distributed in each species throughout the survey area. Overall prevalences of antibody were 70 per cent in cattle, 67 per cent in sheep and 76 per cent in goats as assessed by an immunodiffusion test. Within countries the percentage prevalences were Jamaica 77, St Kitts/Nevis 70, Antigua 76, St Lucia 82, Barbados 61, Grenada 88, Trinidad and Tobago 79, Guyana 52 and Surinam 84. No clinical cases of bluetongue have been confirmed in the area surveyed and there are no virus isolates available to indicate which serotype(s) of virus is/are causing the infection(s).

Bluetongue virus isolations from vectors and ruminants in Central America and the Caribbean.
Interamerican Bluetongue Team.
Mo CL, Thompson LH, Homan EJ, Oviedo MT, Greiner EC, Gonzalez J, Saenz MR.
School of Veterinary Medicine, University of Wisconsin, Madison 53706.
A regional prospective study of the epidemiology of bluetongue virus (BTV) serotypes covering 11 countries in Central America and the Caribbean took place between 1987 and 1992. Active surveillance revealed BTV infection to be endemic in the absence of confirmed indigenous cases of bluetongue. During the 6-year span of the study, over 300 BTV isolations were obtained from cattle and sheep. Results of the earlier years of the study were summarized, and surveillance activities in the concluding months of the study from November 1990 to February 1992 were evaluated. Forty-five BTV isolations were made during this time, 44 from sentinel cattle and 1 from a ram with clinical signs compatible with contagious ecthyma. Virus isolation from potential vectors also was attempted, yielding a further 9 BTV isolates from parous Culicoides insignis and C pusillus, 2 BTV isolates from blood-engorged C filarifer, and 1 epizootic hemorrhagic disease virus type-2 isolate from parous C pusillus. Our extensive network of sentinel herds in the region detected BTV-1 as the predominant serotype in Central America in 1991, after an apparent absence of 1 year in the sentinel animals. Other serotypes in Central America at that time included BTV-3 and BTV-6. In Puerto Rico and the Dominican Republic, BTV-4 became the predominant serotype, without detection of BTV-8 and BTV-17, which were common in recent years of the study. The serotypes found in the Caribbean Basin continued to have marked differences from those in North America. The
importance of viewing bluetongue as an infection, the distribution of which is determined principally by ecologic factors, is emphasized.

Regional Bluetongue Team.Homan EJ, Mo CL, Thompson LH, Barreto CH, Oviedo MT, Gibbs EP, Greiner EC.
University of Wisconsin-Madison, School of Veterinary Medicine 53706.

Results of a prospective serologic and virologic study of ruminant livestock in Central America and the Caribbean islands revealed bluetongue virus (BTV) to be enzootic in the 9 countries participating in the study. Bluetongue virus serotypes 1, 3, 6, and 12 were isolated from sentinel animals. To the authors' knowledge, these are the first isolations of BTV from the region studied and the first isolations of these serotypes in the Western Hemisphere. Clinical disease attributable to BTV infection was not observed in sentinel animals. The incidence pattern, with respect to age and geographic location, was determined. The need to evaluate the epizootiologic features or arthropod-borne viruses (arboviruses) on a regional ecologic basis is stressed.

Serological survey of ruminants in some Caribbean and South American countries for type-specific antibody to bluetongue and epizootic haemorrhagic disease viruses.Gumm ID, Taylor WP, Roach CJ, Alexander FC, Greiner EC, Gibbs EP.

The results of a serological survey of ruminant livestock in some countries of the Caribbean and South America for type-specific antibody to bluetongue virus are reported. Using the microneutralisation test with the international serotypes 1 to 22 of bluetongue virus, antibodies to several types were detected. Analysis of the data indicated that in 1981-82 bluetongue virus types 6, 14 and 17, or viruses closely related to them, were infecting ruminants in this region of the world. Antibody to the related virus of epizootic haemorrhagic disease (serotype 1) was also detected in cattle. The difficulty in interpreting the epidemiological significance of data generated by a serological survey of this kind is discussed.


A serotype of bluetongue virus (BTV) hitherto unrecognized in the Western Hemisphere was isolated from cattle in the United States. Clinical disease was not seen in the cattle which were part of a sentinel herd system in Florida designed for studying the epizootiology of BTV. The isolation of serotype 2 was the first recovery of a different serotype of BTV in the United States since 1967. At least 21 serotypes of BTV have been reported worldwide; the 5 serotypes of BTV now recognized in the United States are 2, 10, 11, 13, and 17.
Blood samples were obtained from sentinel beef cattle at monthly intervals, and the sera were tested for antibodies, using a bluetongue virus (BTV) immunodiffusion test (IDT) and virus-neutralization test (VNT), for 5 BTV serotypes (2, 10, 11, 13, and 17) and 2 epizootic hemorrhagic disease virus (EHDV) serotypes (1 and 2). The cattle tested were transported from Tennessee to Texas in 1984 and 1985. All cattle were seronegative by the BTV IDT at the initial bleeding in Texas in 1984 and 1985. In 1984, 16 of 40 (40%) cattle seroconverted as assessed by results of the BTV IDT. In the 16 seropositive cattle in 1984, neutralizing antibodies were detected to BTV serotypes 10 (n = 7), 11 (n = 3), and 17 (n = 11), and EHDV serotypes 1 (n = 1) and 2 (n = 7). In 1984, no cattle seroconverted to BTV-2 or BTV-13. In 1985, 10 of 36 (27.8%) cattle seroconverted as assessed by results of the IDT. Of the 10 seropositive cattle in 1985, neutralizing antibodies were detected to BTV serotypes 10 (n = 10), 11 (n = 10), 13 (n = 7), and 17 (n = 5), and EHDV serotypes 1 (n = 1) and 2 (n = 7). In 1985, no cattle seroconverted to BTV-2. Clinical diseases attributable to BTV or EHDV was not detected in these cattle in 1984 or 1985.

ANIMAL HEALTH DISEASE CARDS

Bluetongue
Names Pathogen(s)
Preferred Name: Bluetongue virus
Spanish: Lengua azul

Overview
Bluetongue virus is an arbovirus (arthropod borne) that naturally infects domestic and wild ruminants, camelids and some other herbivores such as elephants. Bluetongue virus is transmitted by several species of Culicoides (biting midges). Bluetongue is almost exclusively a disease of sheep, although white-tailed deer, pronghorn and desert bighorn sheep may suffer disease in North America. In cattle and goats clinical disease is rare, and, when present, is much milder than in sheep (Verwoerd and Erasmus, 1994).

Bluetongue can cause spectacular disease outbreaks and is placed in the Office International des Epizooties (OIE) List A disease category. Affected sheep may die after acute or chronic disease, or may recover with weight loss and/or wool breaks.

Bluetongue was first described in South Africa after Merino sheep from Europe were introduced in the late eighteenth century (Verwoerd and Erasmus, 1994). The disease was considered confined to South Africa and for many years research efforts on the virus and the disease were exclusively undertaken in that country, mostly at the Onderstepoort Veterinary Institute. The viral nature of the
disease was established, as was its insect spread and multiple virus serotypes (Howell, 1960; 1970). There are now 24 serotypes of bluetongue virus recognized worldwide.

Bluetongue virus is the type species of the genus orbivirus in the family Reoviridae. Initially the virus was classified as an arbovirus but it appeared to share some properties with reoviruses and was provisionally classified as a reovirus. However, bluetongue virus differs in some respects from reoviruses and, along with a number of other related viruses, was classified in a separate genus by Borden et al. (1971).

**Animals Affected**

Bluetongue is an arbovirus, infecting vertebrates and invertebrates cyclically.

Bluetongue virus naturally infects domestic and wild ruminants, camelids and some other herbivores such as elephants. Historically, the primary cycle may have involved species of African antelope, but this role has now been taken over by cattle (Erasmus, 1990).

Midges of the genus Culicoides act as biological vectors of bluetongue virus. Of the approximately 1400 species of Culicoides world-wide, less than 20 are considered actual or possible vectors (OIE, 1998; Mellor, 1990). The most well-studied vector species are C. variipennis and C. insignis in the USA, C. fulvus, C. wadai, C. actoni and C. brevitarsis in Australia, and C. imicola in Africa and the Middle East (Erasmus, 1990). It is possible that additional vector species will be identified in countries such as China and Bulgaria where bluetongue has been recognized only recently.

**Animals Affected Table**

<table>
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<tr>
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<th>Animal Category</th>
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Epidemiology

Bluetongue virus is an arbovirus that has evolved a life cycle where alternate cycles of virus replication in vertebrate and invertebrate hosts are essential for virus persistence. There is no evidence of vertical transmission of the virus in the invertebrate host. Observations on the placental transmission of virus in the vertebrate host are contradictory (Roberts, 1990) and therefore any vertical transmission in vertebrates is considered to be of no consequence to virus ecology. There is little evidence of direct or indirect contact transmission in either host, other than rare instances of seminal transmission in vertebrates (OIE, 1998). The virus cannot be spread by meat, milk or dairy products. Cattle are the primary vertebrate hosts (Erasmus, 1990) and a small number of species of Culicoides midges are the only insect hosts (Mellor, 1990). The rare recovery of bluetongue virus from other insects is of no ecological significance.

The insect vectors of bluetongue virus breed in moist conditions in a variety of habitats, particularly damp, muddy areas and in faecal and plant matter. They have nocturnal feeding habits, preferring still, warm conditions, pastures and open pens. At least some species preferentially feed on cattle. Females take a blood meal prior to egg laying, feed at roughly 4-day intervals and live for about 2 to 3 weeks. The eggs hatch in 2 to 3 days and depending on the temperature, the larval stage lasts 12 to 16 days. Adults emerge 2 to 3 days after pupation and take a blood meal 1 day later and they also mate during this time (Roberts, 1990). The activities of the midge are influenced by temperature and the optimum lies between 13o and 35oC (Sellers, 1981).

As summarized by Gibbs and Greiner (1994), bluetongue is a common, generally subclinical infection of ruminants throughout the tropics and subtropics, within a number of separate ecosystems. Seasonal incursions of the virus into more temperate latitudes, sometimes accompanied by disease, may occur under favourable climatic conditions at certain key locations. There is evidence that infected midges are carried on the wind for long distances (Sellers, 1981). It has been postulated that the major epidemics of bluetongue, in regions where disease occurs only sporadically, can often be traced to windborne carriage of infected Culicoides from distant areas (Gibbs and Greiner, 1988).

Critical in the understanding of the epidemiology of bluetongue is knowledge of the virus competence of the Culicoides species in different ecosystems, but vector competency research is a very specialised discipline. Not only may different populations of a species of midge have varying susceptibilities to a strain of virus, but a single strain of the vector may have differing susceptibility to different virus serotypes (Mellor, 1990).
Competent midges may be infected when biting viraemic vertebrates. The chance of infection depends in part on the genotype of the midge, the strain of virus, the level of viraemia, and environmental factors (Mellor et al., 2000). The extrinsic incubation period (the period between feeding on infected blood and the appearance of virus in the saliva of the midge) is 1-2 weeks.

The colonised USA vector, C. variipennis, is able to ingest approximately 10-4ml of blood (Mellor, 1990), whereas the most widely distributed Australian vector, C. brevitarsis, has a blood meal volume of around 10-4.5ml (Muller et al., 1982). Therefore viraemia must be of the order of 104 infectious units of virus per ml or greater for feeding midges to have much chance of infection. OIE (1998) summarized reported peak levels of viraemia, in virus infectious units per ml of blood, as 104.4 to 106.3 for cattle, 106.4 to 108.0 for sheep and 106.0 for goats, though levels reached are mostly much lower. Viraemia peaks in the first two weeks after infection, before the appearance of serum antibody. Virus titres then drop rapidly and are very low if infections persist for a month or more.

The duration of viraemia in the infected vertebrate is an important factor in the transmission of bluetongue virus to biting, competent midges. Bluetongue is no longer considered a persistent infection of ruminants, especially cattle (MacLachlan, 1994). Singer et al. (2001) analyzed a large volume of existing data on the length of bluetongue viraemia of cattle and concluded that this was equal to or less than 9 weeks in >99% of adults. OIE (1998) report the viraemia of most cattle as less than 4 weeks with fewer than 1% exceeding 8 weeks. The maximum viraemia reported for sheep is 54 days (Koumbati et al., 1999), but this is exceptional.

Distribution

In 1943 bluetongue disease was reported in Cyprus, and outbreaks were subsequently reported in Israel, the USA, Portugal, Spain, Pakistan and India (Verwoerd and Erasmus, 1994). Over the past 30 years evidence of regular virus activity, but not necessarily disease, has been found in most countries in the tropics and subtropics with substantial populations of ruminants. The virus may be found in a geographic band between latitudes 40oN and 35oS. The presence of bluetongue virus within this band, whether year round or seasonal, depends on the climatic zone type. Genetic studies (topotyping) indicate that the virus exists in discrete, stable ecosystems, probably the result of co-evolution of different strains of the virus and vectors (OIE, 1998). Numerous countries in the tropics and subtropics have bluetongue virus unknowingly circulating subclinically in cattle and other ruminants. A properly designed serological survey would reveal the presence of the virus. The virus is endemic in areas of some countries, being more or less continuously active. Depending on climatic factors affecting the vector, in most years the virus will seasonally extend to adjacent areas (Gibbs and Greiner, 1988). In exceptionally favourable years the virus will spread even further, such as to Portugal and Spain in 1956, to British Columbia in 1988, to Bulgaria, continental Greece and Tunisia in 1999 and to Algeria, Sardinia, Corsica, Majorca, Minorca, Sicily and continental Italy in 2000.

Distribution Table

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**Central America**

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Economic Importance and Impact

Bluetongue can be a costly infection for several reasons. The clinical disease in sheep can be severe, resulting in deaths, weight loss and wool break. In some countries where disease is endemic (South Africa and some States of the USA), vaccination is a recurring cost. However the greater cost of bluetongue is to infected countries which export live animals, germplasm and some animal products such as foetal calf serum. Here the presence of bluetongue virus, even if wholly subclinical, causes loss of trade due to restrictions on the source of animals, and the costs of health testing. It has been estimated that in the late 1970s, the ban on US cattle semen exports resulted in an annual loss of $24 million (Gibbs and Greiner, 1988).

Bluetongue is included in the OIE List A diseases, largely because of dramatic outbreaks of disease in Cyprus in 1943 and Portugal and Spain in 1956. The Cyprus outbreak was due to a particularly virulent strain of the virus causing between 60 and 70% losses in some flocks (Gambles, 1949). The
Iberian outbreaks were also spectacular. Within the first 4 months 46,000 sheep had died in Portugal and 133,000 in Spain (Roberts, 1990). This listing of bluetongue in the most serious of animal diseases exacerbates the trade sensitivity and associated costs to countries with the infection, and has been challenged by some (Gibbs and Greiner, 1994).

**Disease Course**

Natural bluetongue infection is usually subclinical. Bluetongue disease is the result of a complex interaction between the animal, the virus and the environment. Bluetongue is almost exclusively a disease of sheep, with European breeds most susceptible. Most breeds of sheep, especially in regions where the virus is endemic, are resistant to disease though there is increasing information that native breeds in India and China can be clinically affected. Outbreaks of disease typically occur either when susceptible sheep are introduced to endemic areas, or when infected midges carry the virus from endemic regions to adjacent areas containing populations of immunologically- negative, susceptible sheep.

Many strains of bluetongue virus appear incapable of causing significant disease following natural or experimental infection of breeds of sheep known to be susceptible to disease. Experimental reproduction of disease can be inconsistent, except with the most virulent strains of virus. This could be because exposure of sunlight can have a marked influence on the severity of disease (Erasmus, 1990). Passage of virulent field virus in cell cultures rapidly reduces virus virulence (Gard, 1987).

After introduction by the bite of an infected midge, bluetongue virus first replicates in the local lymph nodes and subsequently induces a primary viraemia which seeds other lymph nodes, spleen, lung and vascular endothelium (Gibbs and Greiner, 1988). Circulating virus associates with blood cells, mostly with erythrocytes and platelets, though virus associated with mononuclear cells is critical for dissemination of virus throughout the animal. Later in viraemia, the virus is exclusively associated with erythrocytes (MacLachlan, 1994). Virus particles appear to be sequestered in invaginations of the erythrocyte membrane, allowing prolonged viraemia in the presence of neutralizing antibodies (OIE, 1998).

Fever is usual but not invariable. Other common clinical signs include oedema (of lips, nose, face, submandibulum, eyelids and sometimes ears), congestion (of mouth, nose, nasal cavity, conjunctiva, skin and coronary bands), lameness and depression. The oedema of lips and nose can give the sheep a 'monkey-face' appearance. There is frequently a serous nasal discharge, later becoming mucopurulent. The congestion of the nose and nasal cavity produces a 'sore muzzle' effect, the term used to describe the disease seen in sheep in the USA before its bluetongue virus aetiology was realized. The mouth is sore and the sheep may champ to produce a frothy oral discharge. Sheep are not strictly anorexic, but eat less because of oral soreness and will hold food in their mouths to soften it before chewing. Affected sheep occasionally have swollen, congested, cyanotic tongues. Lameness, due to coronary band congestion, may occur early in the disease and lameness or torticollis, as a result of skeletal muscle damage, may occur later (OIE, 1998).

If fever occurs, sheep are first pyrexic 4-10 days after infection. The other clinical signs soon follow with acute deaths occurring during the second week following infection. Many of these deaths are the result of pulmonary oedema and/or cardiac insufficiency. Further sheep may die from chronic disease 3 to 5 weeks after infection with bacterial complications, especially pasteurellosis. Under-nutrition arising from lameness and depression may be contributing factors. The production loss due to bluetongue may be the result of deaths, unthriftiness during prolonged convalescence, wool breaks and possibly reproductive wastage (OIE, 1998).
Although the frequency of infection of cattle with bluetongue virus is generally higher than in sheep, disease in cattle is rare. Clinical infection is actually a hypersensitivity reaction, including fever, stiffness or lameness and increased respiratory rate. There may be lacrimation and increased salivation. The skin of the muzzle is often inflamed, and may crack and peel. The lips and tongue may be swollen, with ulcers on the oral mucosa. Similarly, the skin of the neck, flanks, perineum, and teats may be affected (Erasmus, 1990).

Hydranencephaly and congenital deformities may develop in bovine and sheep foetuses of bluetongue virus-infected dams, the severity of lesions depending on the stage of gestation. Foetuses seem to be most susceptible during the period of active brain development (Erasmus, 1990). It is clear that cell culture-adapted virus more readily crosses the placenta than unadapted virus, suggesting that the occasional instances of natural virus-induced teratogenesis may be due to strains of virus derived from live virus vaccines (MacLachlan, 1994).

Bluetongue in dogs associated with use of a contaminated vaccine was reported by Akita et al. (1994). Only pregnant bitches were affected.

Pathology

All of the pathology of bluetongue can be assigned to vascular endothelial damage resulting in changes to capillary permeability and fragility, with subsequent disseminated intravascular coagulation and necrosis of tissues supplied by damaged capillaries. These changes result in oedema, congestion, haemorrhage, inflammation and necrosis.

In animals dying acutely, the oral mucosa is hyperaemic and petechiae or ecchymoses may be present. Excoriations may be in areas subject to mechanical abrasion; the edges of lips, dental pad, tongue and cheeks opposite the molar teeth. There may be hyperaemia in the fore-stomachs. The lungs may be hyperaemic with severe alveolar and interstitial oedema, froth in the bronchi, and excess fluid in the thoracic cavity. The pericardial sac may have petechiae and excess fluid. A variable sized haemorrhage in the tunica media near the base of the pulmonary artery is almost pathognomonic. Subepicardial and subendothelial haemorrhages, particularly involving the left ventricle, are common. Generalized damage to the cardiovascular system is evidenced by widespread hyperaemia, oedema and haemorrhage (Erasmus, 1990).

Animals that die later than 14 days after infection often show dramatic degeneration and necrosis of the skeletal musculature. Muscles lose pigmentation and the inter-muscular fasciae are infiltrated with a clear gelatinous fluid (Erasmus, 1990).

Microscopic examination of mucosal lesions shows mononuclear cell infiltration, degeneration and necrosis of epithelial cells in which large acidophilic intra-cytoplasmic masses accumulate. Affected muscles have oedema, haemorrhage, hyaline degeneration and necrosis. Infiltration by neutrophils, macrophages and lymphocytes is present in acute cases (Verwoerd and Erasmus, 1994).

Diagnosis

There is a summary of recommended procedures for bluetongue serology and virus isolation (Afshar and Gard, 1995), while Afshar (1994) provides details of the diagnosis methods. The isolation and identification of bluetongue virus is also described in detail by Clavijo et al. (2000).

The recommended tests for the detection of bluetongue serogroup-specific antibodies are agar-gel-immunodiffusion and competitive ELISA, with the latter becoming more popular because of its greater accuracy and adaptation to conventional laboratory rapid testing and reading technology.
The recommended test for the detection of serotype-specific antibodies is the virus neutralization test.

Bluetongue virus is usually isolated from tissues, or preferably red blood cells washed free of any antibody, in embryonated chicken eggs. Bluetongue virus may be detected in the inoculated eggs by antigen or nucleic acid detection procedures or by passage to susceptible cell cultures. Cell culture isolates are identified as bluetongue viruses by tests based on group-specific antibodies and using fluorescent or enzyme conjugates. Isolates placed in the bluetongue serogroup are typed by virus neutralization tests using serotype-specific antisera.

Differential diagnosis should include contagious ecthyma, foot and mouth disease, photosensitization, pneumonia, polyarthritis, footrot, foot abscesses, plant poisonings, peste des petits ruminants, coneurosis and epizootic haemorrhagic disease of deer.

**Disease Treatment**

There is no treatment for bluetongue disease. The recovery of affected animals will be aided by the provision of shade, water, feed and shelter.

**Disease Prevention and Control**

Bluetongue is a disease of sheep, but cattle are the principal vertebrate reservoirs of the virus. Once established, it is impossible to actively eradicate bluetongue virus. The virus will circulate, generally subclinically, in cattle and other ruminants, and in midges. In countries marginally suitable for virus persistence, the virus may be maintained for several years before dying out. Bluetongue entered Portugal and Spain in 1956 and appears to have persisted in Portugal until 1959 and in Spain until 1960 (Roberts, 1990). In seasonally infected areas, the onset of cold weather will reduce midge populations to ineffective levels and cause the virus to retreat to regions of year-round activity.

The bluetongue virus cycle could be interrupted by the immunization of vertebrate hosts, especially cattle, removal of vectors or prevention of vector attack. Understandably, the immunization of animals that will not suffer from the disease is not acceptable to farmers. The control of midges by the application of insecticides and larvicides to insect resting and breeding sites, or systemically to cattle, has not been fully investigated but is likely to have local success only. Protecting sheep from exposure to midges is a more practical approach and can be achieved by moving sheep from insect resting and breeding sites, stabling animals overnight or the use of insect repellents. Mixing cattle with sheep will draw vectors with a host preference for cattle from sheep, but may raise the virus infection level of the midge population.

Prophylactic immunization of sheep is the most practical and effective control measure, especially when the threat is from an epidemic due to a single serotype, such as the type 10 outbreak in Portugal and Spain in the 1950s. However, multiple serotypes of virus are usual in endemic situations (Hawkes, 1996), requiring multivalent vaccines because bluetongue vaccines are serotype specific. However, multivalent vaccines have attendant problems resulting from interference between virus strains, differences in immunogenicity and growth rates between various strains, as well as differences in the response of individual animals to the components of such vaccines (Verwoerd and Erasmus, 1994). Additionally, there is growing concern by some scientists about the use of live attenuated bluetongue vaccines. Murray and Eaton (1996) summarized these concerns into four areas. These areas are: the known teratogenicity of attenuated virus for the developing foetus; the propensity for vaccine virus to be excreted in the semen of bulls and rams; the possibility that vaccine virus will infect vectors and establish in the environment; and the generation of
recombinant progeny virus with novel genetic and biological properties after the reassortment of genes from wild and vaccine virus in the vaccinated animal or the vector.

Alternatives to live attenuated vaccines are described by Murray and Eaton (1996). Vaccines based on inactivated whole virus, recombinant virus-like particles or recombinant core-like particles all show promise, but require more research. If a commercial product of any of these is achieved, it will likely cost considerably more than a live attenuated vaccine.

Live attenuated bluetongue vaccines have wide use in South Africa, and more limited use in USA and a few other countries. The vaccines are compromises between attenuation and immunogenicity and may have residual pathogenicity for some vaccinated sheep. The application of the vaccines has to be well managed. Colostral immunity in young sheep can interfere with the development of active immunity to the vaccine and breeding ewes and rams should be vaccinated before mating.

**Zoonoses and Food Safety**

Bluetongue is not a zoonosis.

**Taxonomic Tree**

Kingdom "Viruses" [01"VIR], Family Reoviridae [41REOV], Genus Orbivirus [51ORBI]

**Pathogen Characteristics**

Bluetongue virus is an icosahedral-shaped particle consisting of a segmented double-stranded RNA genome, encapsidated in a double-layered protein coat. Removal of the outer protein layer activates a viral-associated RNA polymerase which transcribes the ten genome segments into 10 mRNAs which are in turn translated into at least seven structural (VP1-VP7) and three non-structural (NS1-NS3) proteins (Huismans and Dijk, 1990). The virions have a diameter of 68-70 nm, comprising an outer capsid around a 54 nm core (Verwoerd and Erasmus, 1994).

The genome segments vary in size from 0.5 kDa to 2.7x10 3kDa, and the viral proteins range in size from 25,000 to 144,000 daltons. VP2 and VP5 form the outer capsid and the other five structural proteins are in the core. VP2 is primarily responsible for the induction of type-specific neutralizing antibodies and its variable sequence results in the 24 recognized serotypes of bluetongue virus (Verwoerd and Erasmus, 1994). The 24 serotypes are designated BLU 1- BLU 24. Variations in other proteins of the virus are responsible for the innumerable strains of the virus and for their varied biological properties. This genetic diversity of bluetongue virus is a consequence of both drift and reassortment of individual gene segments (OIE, 1998).

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