The Family **Reoviridae** contains six genera, three of which infect animals: (i) orthoreovirus (reovirus), (ii) orbivirus, and (iii) rotavirus. The original isolates were called" reovirus", an acronym for "respiratory enteric orphan virus", because earlier they were not found linked with any disease

The most important pathogens are in the orbivirus genus, which causes bluetongue and African horse sickness. The rota viruses cause enteritis in many different species.

BLUE TONGUE

(Commonly known as: "catarrhal fever of sheep" and "soremuzzle",)

ETIOLOGY:

Bluetongue (BT) is a viral disease of sheep, (occasionally cattle and goats), transmitted by insect vectors and is primarily caused by arthropod-borne **Orbivirus** of reoviridae family, a non-enveloped, double-stranded RNA (ds RNA) viurses.

The disease is characterized by catarrhal stomatitis, rhinitis, enteritis, and lameness.

SPREAD

Bluetongue is transmitted by biting insects of the genus Culicoides. Bluetongue is most severe in sheep and is endemic in India. Sheep of all ages are susceptible, but adults seem to be affected more often than lambs. Morbidity is 30-50 % however mortality is <10%.

Most domestic species are highly susceptible and incidence in sheep, cattle, buffaloes and goats have been reported in India. Rate of positivity in buffaloes was higher than in cattle, and the incidence was higher in males than in females.

Infection also reportedly transmitted by insemination from an infected bull, since the virus is present in the **semen.** The virus has also been recovered from bovine semen (Semen Stations) associated with structural abnormalities of the spermatozoa.

PATHOGENESIS

Following Biting Insects depositing the BT virus in skin, viral replication initially occur in skin & haematopoietic cells. This results in viraemia and subsequent replication in and damage to endothelial cells throughout the lymphatic system of body, leading to capillary permeability and fragility, subsequent disseminated intravascular coagulation and necrosis of tissues supplied by damaged capillaries.

Endothelial cell damage is responsible for the widespread gross and microscopic lesions. Endothelial cells become swollen and later become necrotic, causing oedema, haemorrhage, thrombosis, and infarction.

SIGNS

High fever (105° F) is the first sign, depressed and lethargic; associated reddening of the nasal and oral mucosa and excessive salivation (is clear/strigy, not frothy). A watery to seroud nasal discharge from the nostrils later becomes mucoid, muco-purulent to bloody, and may dry to form crusts,partially block the nasal passage. Reddening of conjunctiva along with increased tear production also seen.

Oedematous swellings appear in the lips, tongue, ears, face, and inter-mandibular space. Oedema and cyanosis of the tongue (bluish) are so striking that they have given the disease its name. However, they are not always present.

Petechiae soon appear on Oral and nasal mucosa, where the epithelium becomes thickened and is shed, leaving excoriations (abrasions) and bleeding points. As the fever subsides, flushing (redness) of the skin and feet appears, and the coronets become warm and tender. This results in stiffness and lameness.

The disease may terminate in severe emaciation, prostration (lying down), and muscular weakness (sometimes with torticollis, *i.e.,* twisted neck drawing the head to one side), which may last 3 weeks or so. This is followed by pulmonary oedema and death from pneumonia.

LESIONS

The lesions of bluetongue originate from replication of the virus in endothelial cells, and are characterized by oedema, hyperaemia, haemorrhage, and infarction.

There is extensive oedema of the subcutis around the head and neck. Changes around the **mouth**, (characteristic of disease), consist of hyperaemia, oedema, cyanosis, and multiple haemorrhages, especially on the muzzle, tongue, and cheeks, with erosion and even ulceration of the epithelium. The dental pad, hard palate, gums, oesophagus, reticulum and rumen also have similar lesions.

Skin near the hoof reveals, intense hyperaemia of the corium, mostly concentrated at the tips of the dermal papillae, and associated with oedema and infiltration with neutrophils.

The **muscles** usually contain foci of gross haemorrhage, associated with microscopic evidence of necrotic changes in muscle bundles. Haemorrhage and necrosis also occur in the myocardium, particularly in papillary muscles of left ventricle.

Bluetongue virus infection during **pregnancy** may result in foetal infection, causing severe cerebral abnormalities in both sheep and cattle, and its severity depends on stage of pregnancy. In ewes exposed at 40-60 days of pregnancy and cattle of 60-120 days, a severe necrotizing encephalopathy occurs, which at birth is seen as **hydranencephaly** (hydrocephalus). Later in pregnancy, focal necrotizing lesions develop, seen as **porencephaly** (cavities in brain) at birth.

Necrosis of the **retina** may also develops, causing lambs or calves born with retinal dysplasia in addition to the cerebral abnormalities.

DIAGNOSIS

Diagnosis can be made on the basis of a (i) history, symptoms and gross lesions, and (ii) Isolation and identification of the virus (from blood or spleen) are necessary to confirm diagnosis.

In Sheep, must be differentiated from photosensitization, contagious ecthyma, foot-and-mouth disease, *Oestrus ovis* infestation, ulcerative dermatitis, and sheep-pox.

In cattle, disease must be differentiated from FMD, Rinderpest, vesicular stomatitis, infectious rhinotracheitis, mycotic stomatitis, and the bovine-virus diarrhoea disease complex.