

VESICULAR STOMATITIS

AETIOLOGY

Classification of the causative agent

Vesicular stomatitis virus (VSV) is a member of the family Rhabdoviridae, genus *Vesiculovirus*. There are two distinct immunological classes of VSV recognised: New Jersey (NJ) and Indiana (IND). There are three subtypes of the IND serogroup based on serological relationships: IND-1 (classical IND) IND-2 (cocal virus) and IND-3 (alagoas virus).

Resistance to physical and chemical action

Temperature:	Inactivated by 58°C for 30 minutes
pH:	Stable between pH 4.0 and 10.0
Chemicals/Disinfectants:	Sensitive to formaldehyde, ether and other organic solvents; chlorine dioxide, formalin (1%), 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, 2% sodium carbonate, 4% sodium hydroxide, and 2% iodophore disinfectants, all effective disinfectants.
Survival:	Inactivated by sunlight; survives for long periods at low temperatures

EPIDEMIOLOGY

- Although the VSV has been extensively studied at the molecular level, many unknowns remain regarding its epidemiology
- VSV is known to be transmitted directly via the transcutaneous or transmucosal route
- Certain VS viruses have been isolated from sand flies, black flies mosquitoes and other insects to both pigs and cattle
 - seasonal variation (disappearance at end of rainy season in tropical areas and at first frost in temperate zones) also supports vector-borne transmission
 - hypotheses that the VS virus is a plant virus present in pasture
- In endemic areas, VSV maintains long-term, stable cycles between sand flies and subclinical susceptible hosts; evidence of neutralising antibodies in domestic and wild animals in these areas
- Morbidity rate variable, up to 90% in a herd
- Low mortality rate

Hosts

- Domestic hosts: equidae (horses, donkeys, mules) bovidae, suidae and South American camelids
 - sheep and goats tend to be resistant with few clinical signs.
- Wild hosts: white-tailed deer and numerous species of small mammals in the tropics
- Human (minor zoonosis)
- Experimental host range includes laboratory animals (mice, rats, guinea-pig) deer, raccoons, bobcats, and monkeys

Transmission

- Mechanism of transmission of VSV is unclear
- Contamination by transcutaneous or transmucosal route
- Arthropod transmission: sand flies (*Phlebotomus*, *Lutzomyia* spp.), mosquitoes (*Aedes* spp.), black flies (family Simuliidae)
- Experimental transmission of VS NJ has been demonstrated to occur from black flies (*Simulium vittatum*) to domestic swine and cattle

Sources of virus

- Saliva, exudate or epithelium of open vesicles
- Arthropod vectors
- Plants and soil (suspected)

Occurrence

The disease is limited to the Americas; however, it has been described in France (1915 and 1917) and in South Africa (1886 and 1897). Strains of the serotype NJ and subtype IND-1 are endemic in livestock in areas of southern Mexico, Central America, Venezuela, Colombia, Ecuador and Peru. Sporadic activity of NJ and IND-1 VSV has been reported in northern Mexico and western United States. IND-2 has only been isolated from mammals sporadically in Argentina and Brazil. The IND-3 subtype (Alagoas) has been isolated only in Brazil. While VS is not diagnosed in livestock every year in the USA, it is considered to be endemic in feral pigs on Ossabaw Island, Georgia.

DIAGNOSIS

Incubation period varies from 2–8 days with an average of 3–5 days. VSV vesicles can develop within 24 hours post-inoculation. In humans, the incubation period can vary from 24 hours to 6 days but is usually 3–4 days. For the purposes of the OIE *Terrestrial Animal Health Code*, the incubation period for VS is 21 days.

Clinical diagnosis

The signs are similar to those of foot and mouth disease (FMD), with which it can easily be confused (but horses are resistant to FMD and susceptible to VS)

- VS cannot be reliably clinically differentiated from the other vesicular diseases, such as foot and mouth disease (FMD), vesicular exanthema of swine (VES), and swine vesicular disease (SVD) when horses are not involved. An early laboratory diagnosis of any suspected VS case is therefore a matter of urgency.
- The incidence of disease can vary widely among affected herds; 10–15% of the animals show clinical signs and these are usually adult animals
- Cattle and horses under 1 year of age are rarely affected
- First manifestation of disease is usually excessive salivation
- Blanched raised or broken vesicles of various sizes in the mouth:
 - Horses: upper surface of the tongue, surface of the lips and around nostrils, corners of the mouth and the gums
 - Cattle: tongue, lips, gums, hard palate, and sometimes muzzle and around the nostrils
 - Pigs: snout
- Lesions involving feet of horses and cattle are not exceptional
- Teat lesions occur in dairy herds
- Foot lesions and lameness are frequent in pigs
- Recovery in few days up to 2 weeks
- Complication: loss of production and mastitis in dairy herds due to secondary infections, lameness in horses
- Morbidity rates vary between 5 and 70 %; mortality is rare
 - higher mortality has been observed with NJ strains in swine

Lesions

- Vesicles, ulcers, erosions, and crusting of muzzle and lips; limited to the epithelial tissues of mouth, nostrils, teats and feet

The pathogenesis of the disease is unclear, and it has been observed that the humoral-specific antibodies do not always prevent infection with VS serogroup viruses.

The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated April 2013.