

Lumpy skin disease

Authors: Prof. JAW Coetzer and Dr. Eeva Tuppurainen

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EPIDEMIOLOGY

Available evidence suggests that there is only one immunological type of LSDV: virus isolates collected over an extended period from a large number of natural cases originating from outbreaks of the disease in South Africa, Kenya and Malawi showed complete cross-neutralization with the prototype “Neethling” strain.

Cross-protection between LSDV and sheep- or goat pox viruses has been exploited by the use of sheeppox virus for the immunization of cattle against LSD in Kenya and in the Middle East.

Lumpy skin disease virus is remarkably stable. It can be recovered from skin nodules kept at -80°C for ten years and from infected tissue culture fluid stored at 4°C for six months. The virus can persist in necrotic skin nodules for up to 39 days but this period may be much longer.

Periodic epidemics occur in most African countries, particularly in those of the sub-Saharan region.

Imported *Bos taurus* breeds with thin skins, such as Friesland cattle, usually show more severe signs of the disease than thick-skinned indigenous breeds including the Afrikaner and Afrikaner cross-breeds. Similarly, all age-groups are susceptible. Cows in the peak of lactation as well as young animals show usually more severe clinical disease.

Very little is known about the susceptibility of wild ruminants to LSDV. Using novel molecular diagnostic techniques and sequencing, clinical LSD has been confirmed in springbok in South Africa. Clinical disease suspected to be LSD has been described in five Asian water buffalo (*Bubalus bubalis*) in Egypt and in an Arabian Oryx (*Oryx leucoryx*) in Saudi Arabia, springbok (*Antidorcas marsupialis*) in Namibia and oryx (*Oryx gazella*) in South Africa. Giraffe (*Giraffa camelopardalis*) and impala (*Aepyceros melampus*) are highly susceptible to experimental infection, whereas two young African buffaloes (*Syncerus caffer*) did not develop clinical signs or seroconverted. A random sample of 440 sera taken from culled buffalo in the Kruger National Park in South Africa between the ages of one and 20 years were all negative for serum-virus neutralizing antibody to the prototype Neethling strain. From the clinical trial, and the failure to detect antibody in a random sample of sera, it was concluded that the African buffalo is not susceptible to the virus or only slightly so. More recently, LSDV DNA was detected in samples collected from naturally-infected water buffaloes in Egypt. However, the PCR method used in this study was not able to differentiate between LSDV and sheep- and goat pox viruses.



The mode of transmission of LSD has not been established fully, although circumstantial evidence suggests that biting insects play a role in the dissemination of infection. It is usually more prevalent during the wet summer and autumn months, particularly in low-lying areas and along water courses, but outbreaks may also occur during the dry season and winter months.

The virus has been recovered from *Stomoxys* spp. and *Musca confiscae* and mechanical transmission of LSDV has been demonstrated by *Aedes aegypti* mosquito. The three common African hard tick species, namely, brown tick (*Rhipicephalus appendiculatus*), the bont tick (*Amblyomma hebraeum*) and the African blue tick (*Rhipicephalus (Boophilus) decoloratus*), have recently been incriminated in the transmission and epidemiology of LSD. *R. appendiculatus* and *A. hebraeum* males were able to transmit the virus mechanically and successful transovarial transmission of LSDV by *R. decoloratus* species was demonstrated. *A. hebraeum* ticks over-winter as nymphal stage and *R. decoloratus* in gravid females and eggs. Therefore, the survival of the virus in *R. decoloratus* eggs and larvae and *A. hebraeum* nymphs/adults may at least in part explain how LSD overwinter during inter-epidemic periods.

Some scientists are of the opinion that the low titre of LSDV present in the blood of infected animals during the viraemic stage is not sufficient for mechanical transmission to occur by biting flies feeding on blood alone and that biting flies have to feed on skin lesions to obtain enough virus for transmission to take place. The virus may remain viable in scab or tissue fragments for several months.

Direct transmission by contact between animals is inefficient. However, transmission did occur when common drinking troughs were used, thus confirming the suspicion that infected saliva might contribute towards the spread of the disease. The disease is transmissible to suckling calves through infected milk.

Lumpy skin disease virus has been demonstrated to persist in bovine semen for up to 42 days post-infections (dpi) and viral DNA was detected until 159 dpi. The virus was isolated from the semen of bulls with inapparent disease.