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# Lumpy skin disease (LSD) in cattle caused by the lumpy skin disease virus (Neethling virus): A member of the genus *capripoxvirus* within the family *poxviridae*

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**Abstract**
Lumpy skin disease (LSD) causes major economic losses in the livestock industry of most African countries. It is caused by Lumpy Skin Disease Virus (LSDV), a member of the *Poxviridae* family, with the prototype Nettling strain. LSDV belongs to the *capripox* virus family which includes sheep pox virus and goat pox virus. It is spread by insect vectors between pastures and watering places and cattle congregating in the same barn. LSD is an enzootic infectious, eruptive and rarely fatal disease of cattle characterized by skin nodules. Cattle and water buffalo are the only animal species affected, with high morbidity rates but low mortality, however, mortality is high in calves. LSD reduces milk and beef production, causes abortion in females and infertility in males. The origin of LSD was in Zambia in 1929. LSD is considered an endemic disease in the African continent. However, the disease has moved out of Africa since the 1980s. The disease is reported in LSD-free countries (Jordan, Syria, Lebanon, Turkey, Iran and Iraq) with potential economic losses to the livestock industry. Therefore, mass vaccination along with other appropriate control measures is the most effective way to limit the spread and economic impact of lumpy skin disease.

**Keywords:** Clinical signs, LSD, LSDV, lesions, transmissions

## Introduction
Lumpy skin disease (LSD, Pseudo-urticaria, Neethling virus disease, exanthema nodularis bovis, and knopvelsiekte) is an infectious disease. It is caused by a virus (LSDV) of the Poxviridae family, *capripox* virus, (Alexander *et al*., 1957) [5]. *Capripoxvirus* (CaPVs) is one of eight species in the *Chordopoxvirinae* subfamily of Poxviridae and includes Lumpy Skin Disease Virus (LSDV), Sheep Pox Virus (SPPV), and Goat Pox Virus (GTPV). These viruses are responsible for economically important diseases of domestic ruminants in Africa and Asia, (CFSPH, 2008) [26]. LSD causes significant economic losses in affected countries, but while all stakeholders in the livestock industry experience loss of income, poor, small and marginal farmers suffer the most, (FAO, 2017) [17].
LSD is a disease of domestic cows and buffaloes. Although LSD has not been detected in goats or sheep. Sheep, goats, giraffes, impala, and Grant's gazelles housed near sick cattle develop skin lesions, (OIE, 2016) [23]. Lumpy skin disease (LSD) is a borderline high-impact cattle pox disease. Clinical symptoms include lesions in mouth, pharynx and respiratory tract, skin and visceral nodules (2-5 cm in diameter), mucous membrane and internal organs, mild fever for 2-3 days, weakness, enlarged lymph nodes, swelling of the skin and sometimes death, (OIE, 2016; Buller R.M., 2005) [23, 10]. Nodules are circumscribed, firm, round, large, and involve the skin, subcutaneous tissue, and sometimes muscle. It is a vector-borne disease transmitted by various biting and biting blood-feeding arthropods such as mosquitoes, flies and ticks. Mortality rates up to 20%, economic losses due to condition deterioration, reduced milk production, abortions, infertility, secondary bacterial infections and damaged coats, (Anonymous, 1988) [32].
Although infected animals recover within 2-3 weeks, milk production decreases in lactating cattle for several weeks. The morbidity rate is about 10-20% and the mortality rate is about 1-5%, (Advisory and guidelines). The double-stranded DNA virus, LSDV, like other Poxviridae, has a large size, a property that has made this virus widely used as a vector to

express foreign viral genes and to produce recombinant multivalent vaccines. Another potentially beneficial property of LSDV is its remarkable stability and ability to survive for long periods of time at ambient temperature, particularly in dried form. The virus is also very resistant to inactivation.

**History and Geographic Distribution:**

The disease was first identified in Northern Rhodesia (Zambia) in 1929. Lumpy skin disease (LSD) was known, a skin disease of cattle called "pseudo-urticaria" found in what was then known as Northern Rhodesia. The disease was probably a lumpy skin disease, but at the time it was thought to be caused by insect bite wounds, (Mac-DONALD, 1931) [21]. The disease appeared in the following years and plant poisoning was also the cause, (L.E. Roux, 1945) [20]. Another epidemic of this disease occurred in October 1943 in Botswana and was named "Ngamiland Cattle Disease" because it was initially observed in Ngamiland. During this period there was evidence that the disease was caused by an infectious agent, (Abdulqa H.Y., 2016) [1].

Additional cases occurred between 1943 and 1945 in Botswana, Zimbabwe, and the Republic of South Africa. An estimated 8 million cattle were affected by panzootic infection in South Africa in 1949, causing enormous economic losses. LSD spread across Africa between the 1950s and 1980s, affecting cattle in Kenya, Sudan, Tanzania, Somalia, and Cameroon. The disease was first reported in Israel in 1989, and since then sporadic outbreaks have occurred in several countries in the Middle East. In 2012, the disease appeared on Israel's northeastern border and spread to an unprecedented scale in the Middle East, (OIE, 2016) [23]. Outbreaks were reported from Lebanon, the Palestinian Autonomous Territories, Jordan, Kuwait, Saudi Arabia, Iraq, and Iran. The first entry of the disease was reported in Turkey in 2013, where LSD is currently endemic, (Bowden T.R. *et al*., 2008).

**Hosts and susceptibility**

LSD has a narrow vertebrate host range. Lumpy skin disease is host-specific, causing natural infection in cattle and Asian water buffalo (*Bubulus bubalis*), although the prevalence of disease in buffalo (1.6%) is significantly lower than in cattle (30.8%), (Spickler, 2008) [26]. Some LSDV strains can replicate in sheep and goats. Although mixed herds of cattle, sheep and goats are common, to date there is no epidemiological evidence on the role of small ruminants as reservoirs for LSDV, (Al-Salihi K., *et al*., 2014) [7]. Lumpy skin disease does not affect humans. Clinical signs of LSD have been observed following experimental infection in impala (*Aepyceros melampus*) and giraffe (*Giraffa Camelopardalis*), (El-Nahas E.M., 2011) [15]. The disease has also been reported in the Arabian oryx (*Oryx leucoryx*) and (*Greth a*,) springbok (*Antidorcas marsupialis*). The susceptibility of wild ruminants or their potential role in the epidemiology of LSD is not known.

**Sources of the virus**

Nodules found on the mucous membranes of the eyes, nose, mouth, rectum, udder, and genitalia also ulcerate and release large amounts of LSDV that can serve as a source of virus. Virus can be separated from this material for up to 35 days and likely for longer, (Tuppurainen E.S., 2012) [30]. LSDV can be isolated from blood, saliva, eye and nasal secretions, and semen. LSDV is detected in the blood (viraemia), usually at lower levels 7 to 21 days after infection than in skin lesions. Ejaculation may be prolonged; LSDV has been isolated from the semen of experimentally infected bulls 42 days after inoculation. There has been one report of placental transmission of LSD. LSD does not cause chronic disease. It does not indicate a delay and the disease does not recur.

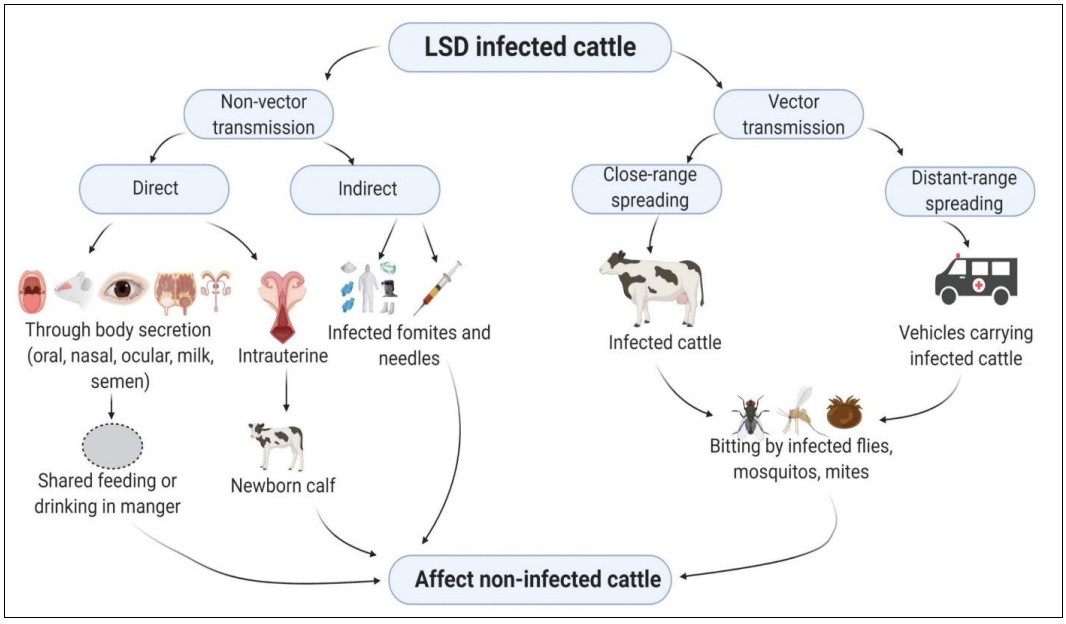


**Fig 1:** Transmission

The main means of transmission is believed to be through arthropod vectors. Consequently, hosts become infected with the virus through the bites of mosquitoes (e.g., *Culex mirificens* and *Aedes natrionus*), biting flies (e.g., *Stomoxys calcitrans* and *Biomyia fasciata*), and male ticks (*Riphicephalus appendiculatus* and *Amblyomma hebraeum*). Could play a role in virus transmission. The importance of different 2 arthropod vectors may vary in different areas depending on the abundance and feeding behavior of the vector, (Tuppuraine E.S. and Alexandrov T., 2017) [28]. Infected bulls can excrete the virus in semen; however, LSD has not been transmitted through infected semen, (Annandale C.H., 2014) [8]. It is not known whether infection can be transmitted by fomites, for example by ingestion of food and

water contaminated with infected saliva, (Ali H., 2012) [6]. Animals can be infected by experimental inoculation with skin nodules or blood material. Direct contact is thought to play a minor, if any, role in the transmission of the virus.

**Clinical signs**

The time between inoculation and the first observation of typical clinical signs in experimentally infected cattle is 7 to 14 days, regardless of route of infection, and 2 to 5 weeks in natural cases. (Carn V.M., 1995) [11]. Clinical symptoms may include nodules on the skin, mucous membranes and internal organs, fever, emaciation, enlarged lymph nodes, nasal discharge, lacrimation, abortion, skin edema, and sometimes death. In acutely infected animals the first symptoms are fever, followed by the formation of swellings or nodules on the skin that give the disease its name. These nodules are 2-7 cm in diameter and are mainly found on the head, neck, udder and perineum. Nodules, which may number in the hundreds, are painful, affect the skin, subcutaneous tissue, and sometimes the underlying muscles, and become necrotic, causing deep pitting, (Alemayehu G., 2013) [4]. Other symptoms include a significant decrease in milk yield, depression, weakness, excessive salivation, and eye and nasal discharge. Pox lesions also occur in the mouth, nose, testicles, and bladder. Superficial lymph nodes, the draining area of the affected skin are significantly enlarged and the limbs become swollen, making the animal reluctant to move. Secondary bacterial infections commonly affect the teats, tendons, and joints, and may cause fly strikes at the site of skin lesions, (EFSA. 2015) [14]. Miscarriages can occur and aborted fetuses have sometimes been reported to be included in the nodules. Infected bulls and cows may become temporarily or permanently sterile, (Constable P.D., 2017) [13].

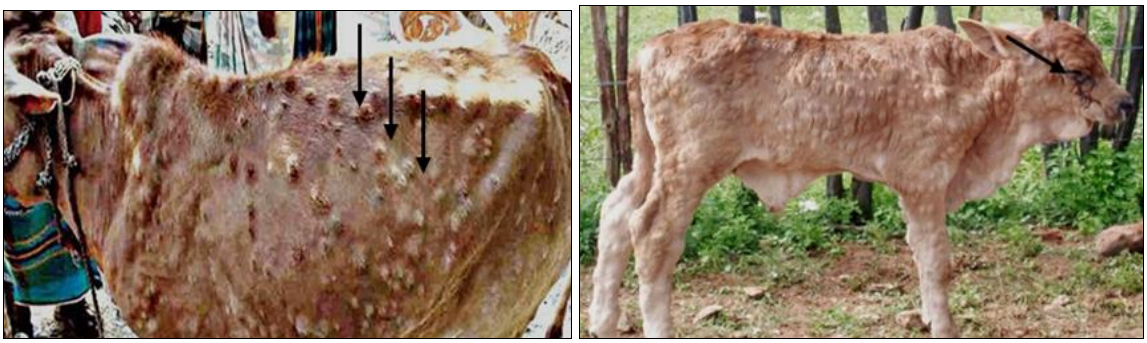

**Fig 2:** Diagnosis of lumpy skin disease

**Clinical Diagnosis**

Although the field incubation period is poorly described, in experimental infections, fever occurs 6–9 days after inoculation and the first skin lesions appear at the inoculation site 4–20 days, (Hosamani M. *et al*., 2004) [19]. LSD should be suspected when the characteristic skin nodules, fever and enlarged superficial lymph nodes are seen. Painful nodules of 2–7 cm in diameter develop over the entire body, particularly on the head, neck, udder and perineum between 7 and 19 days after virus inoculation, (Abera Z., 2015) [2].

**Differential Diagnosis**

Although severe LSD is highly characteristic, mild forms include pseudo-lumpy skin disease/bovine herpes mammalities (bovine herpes virus 2), dermatophilosis, ringworm, insect or tick bites, besnoitiosis, hypoderma, bovis infestation, photosensitization, and bovine herpes. It can be confused with such diseases as popular stomatitis (*Parapoxvirus*), urticaria and skin tuberculosis. Most of these diseases can be distinguished from lumpy skin disease by the duration of the disease, as well as clinical signs, including histopathology and other laboratory tests, (Radostits O.M. *et al*., 2006) [25].

**Control and prevention**

Control and prevention of lumpy skin disease relies on four tactics – movement control (quarantine), vaccination, culling campaigns and management strategies. Specific national control plans vary from country to country and advice should therefore be sought from the relevant authorities and veterinarians, (EFSA, 2015) [14]. Lumpy skin disease can be introduced into new areas by infected animals, contaminated skin and other animal products, or infected insects. Early identified outbreaks have sometimes been eradicated through quarantine, population, and cleaning and disinfection of infected premises, but in some large outbreak's vaccination was an important component of eradication plans, (Murray L., 2013) [22]. Quarantine and movement control are unlikely to completely prevent transmission when LSDV is vector-borne; however, they may prevent infected animals from transmitting the virus to distant centers. Insect control is commonly used during outbreaks of lumpy skin disease, although its effectiveness is still unclear. Sometimes an insecticide treatment of the carcasses helps prevent the flies from contracting the virus. This is especially important if the carcasses are transported from non-infected areas. Vaccination is the most effective means of control, and live homologous vaccines containing a Neethling-like strain of LSDV are recommended, (Thomas L., 2002) [27].

**Treatment**

There is no specific treatment for lumpy skin disease, but supportive care, including antibiotics, anti-inflammatory drugs or vitamins, is used in some cases to treat secondary bacterial infections or to improve fever or inflammation and the animal's appetite. Wound dressings have been used to reduce fly strikes and secondary infections. Complete recovery may take several months and may be prolonged if a secondary bacterial infection occurs. Treatment is directed at preventing or controlling secondary infection. Animals severely affected by the LSD virus can take up to 6 months to fully recover.

**Conclusion**

Clinically the disease is characterized mainly by distinctive nodular lesions on the skin and underlying tissues of affected animals with occasional involvement of various parts of the body; Conjunctiva, alimentary, respiratory and urogenital

tracts. As a result of lesions, loss of hide quality, chronic debilitation, reduced milk production, weight loss, infertility, abortion and death cause tremendous economic losses. It can also have a dramatic impact on rural livelihoods, which are heavily dependent on cattle, with significant losses in production. The effects of the disease are also devastating at the national level as its presence has triggered severe trade restrictions.

Based on the above findings, the following recommendations are put forward:

1. Global expansion of the disease requires special attention.
2. Timely accurate diagnosis is essential for control measurements.
3. An annual vaccination strategy with a uniform strain of LSDV is mandatory in endemic areas.
4. Vector control and restrictions on animal movement are important during active periods of pest movement.
5. Bulls used for breeding must be diagnosed with LSDV.

**References**


1. Abdulqa HY, Rahman HS, Dyary HO, Othman HH, Lumpy skin disease. Am J Reprod Immunol. 2016;1:2476-1974.
2. Abera Z, Degefu H, Gari G. Assessment of Distribution and Associated Risk Factors of Lumpy Skin Disease in Selected Districts of West Wollega Zone, Western Ethiopia. Ac J Ani Dis. 2015;4:130-140.
3. Advisory. Lumpy skin disease, Govt. of India, Animal Husbandary and Dairying department of Animal Husbandary, New Delhi; c2021.
4. Alemayehu G, Zewde G, Admassu B, Risk assessments of lumpy skin diseases in Borena bull market chain and its implication for livelihoods and international trade. Trop Animal Health Prod. 2013;45(5):1153-9.
5. Alexander RA, Plowright W, Haig DA. Cytopathogenic agents associated with lumpy-skin disease of cattle. Bull. Epiz. Dis. Afr. 1957;5:489-492.
6. Ali H, Ali AA, Atta MS, Cepica A. Common, emerging, vectorborne and infrequent abortogenic virus infections of cattle. Transbound Emerg Dis. 2012;59:11-25.
7. Al-Salihi K. Lumpy skin disease: Review of literature. MRVSA. 2014;3:6-23.
8. Annandale CH, Holm DE, Ebersohn K, Venter EH. Seminal Transmission of Lumpy Skin Disease Virus in Heifers. Transbound Emerg Dis. 2014;61:443-448.
9. Bowden TR, Babiuk S, Parkyn GR, Copps JS, Boyle DB. *Capripoxvirus* tissue tropism and shedding: A quantitative study in experimentally infected sheep and goats. Virology. 2008;371(2)380-393.
10. Buller RM, Arif BM, Black DN, Dumbell KR, *et al*. Poxviridae. In Virus Taxonomy: Eight Report of the International Committee on the Taxonomy of Viruses. Elsevier Academic Press, Oxford; c2005.p. 117-133.
11. Carn VM, Kitching RP. The clinical response of cattle experimentally infected with lumpy skin disease (Neethling) virus. Arch Virol. 1995;140:503-513.
12. CFSPH. Center for Food Security and Public Health, Iowa State University. Lumpy Skin Disease; c2008.
13. Constable PD, Hinchcli KW, Done SH, Grundberg W. Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats. 11th edn. Elsevier, UK; c2017.p. 1591.
14. EFSA. Scientific Opinion on Lumpy Skin Disease. EFSA Panel on AHAW. EFSA Journal. 2015;13(1):3986.
15. El-Nahas EM, El-Habbaa AS, El-Bagoury GF, Radwan MEI. Isolation and identifi cation of lumpy skin disease virus from naturally infected buffaloes at Kaluobia, Egypt. Glob Vet. 2011;7:234-237.
16. European Food Safety Authority. Scientific Opinion, Scientific Opinion on lumpy skin disease; EFSA Panel on Animal Health and Welfare (AHAW); EFSA Journal. 2015;13(1):3986.
17. FAO. (Food and Agriculture Organization of The United Nations. Animal Production and Health, Lumpy Skin Disease, Rome; c2017.
18. Greth A, Gourreau JM, Vassart M, Vy NB, *et al*. *Capripoxvirus* disease in an Arabian Oryx (Oryx leucoryx) from Saudi Arabia. Journal of Wildlife Diseases. 1992;28(2):295-300; 15 ref.
19. Hosamani M, Mondal B, Tembhurne PA, *et al*. Differentiation of sheep pox and goat poxviruses by sequence analysis and PCR-RFLP of P32 gene. Virus Genes. 2004;29(1):73-80.
20. Le Roux PL. Notes on the probable cause, prevention and treatments of pseudo-urticaria and associated septic conditions in cattle. Northern Rhodesia Department of Animal Health, Newsletter; c1945. p. 1-4.
21. Mac-Donald RAS, Pseudo-urticaria of cattle. In: Ann. Rep. Dept. Anim. Health; c1931.
22. Murray L, Edwards L, Tuppurainen ES, *et al*., Detection of *capripoxvirus* DNA using a novel loop mediated isothermal amplification assay. BMC Vet Res. 2013;1 9:90.
23. OIE. (World Organisation for Animal Health. Lumpy skin disease. OIE Man. Diagnostic Tests Vaccines Terr. Anim; c2016. p. 1-14.
24. OIE. Terrestrial manual chapter 2.4.13. Lumpy Skin Disease; c2017.
25. Radostits OM, Gay CC, Hinchcliff KW, Constable PD. Veterinary Medicine E-Book: A textbook of the diseases of cattle, horses, sheep, pigs and goats. Elsevier Health Sciences. Edition 10th. Sounders Elsevier; c2006.
26. Spickler Anna Rovid. Lumpy skin disease. At http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php; c2008.
27. Thomas L, Lumpy-skin disease, a disease of socioeconomic importance. J Med Virol. 2002;76:6054-6061.
28. Tuppuraine ES, Alexandrov T, Beltran-Alcrudo D, Lumpy skin disease field manual-A manual for veterinarians. FAO Animal Production and Health Manual. 2017;20:1-60.
29. Tuppurainen E. Diagnostic assays for the detection of lumpy skin disease virus and antibodies. EMPRES. 2017;47:7-9.
30. Tuppurainen ES, Oura CA. Review: lumpy skin disease: an emerging threat to Europe, the Middle East and Asia. Transbound Emerg Dis. 2012;59(1):40-8.
31. World Organization for Animal Health. Lumpy Skin Disease. Terrestrial Manual. Retrieved from www.oie.int; c2016.
32. Attiya H, Snir M, Warmuth MK. Computing on an anonymous ring. Journal of the ACM (JACM). 1988 Oct 1;35(4):845-75.