



Review article

A review on current epidemiology and molecular studies of lumpy skin disease virus-an emerging worldwide threat to domestic animals

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ABSTRACT

In South Asia, cattle are afflicted by the expanding virulent condition known as Lumpy Skin Disease (LSD), and sheep pox and goat pox are caused by the Capri virus. These illnesses endanger worldwide trade. Due to inadequate immunisations and poverty in rural areas, Capricorn poxviruses are spreading. This is due to the economic repercussions of the COVID-19 epidemic, debilitating sanctions in endemic countries, a growth in the legal and criminal trade of live animals and animal products, and global climate change. Skin spores are the main route of infection; however, the virus is also excreted through bodily fluids and semen. As a result, the virus is transmitted to susceptible hosts by biting flies, mosquitoes, and other insects. Insects can be transstadial and transovarial. Lumpy skin disease lesions can swell and rupture after 7 to 14 days in experimental settings, but it usually takes 2 to 5 weeks in a normal infection. Lumpy skin disease is characterised by hard, constrictive, few (mild forms) to numerous (severe forms) skin nodules that may encompass respiratory, urogenital, and other organ mucous membranes. Consequently, milk output decreases, and in countries that raise cattle, there are more abortions, cases of temporary or permanent infertility, hide damage, and mortality, all of which result in a financial loss. The best method for limiting the spread and monetary impacts of lumpy skin disease is mass immunisation and other management measures. This review provides the latest information on lumpy skin disease's viral biology, transmission, clinical, and pathological aspects.

Keywords- LSD, Capri poxvirus, Covid-19, Immunizations, Skin sores, Global climate change.

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INTRODUCTION

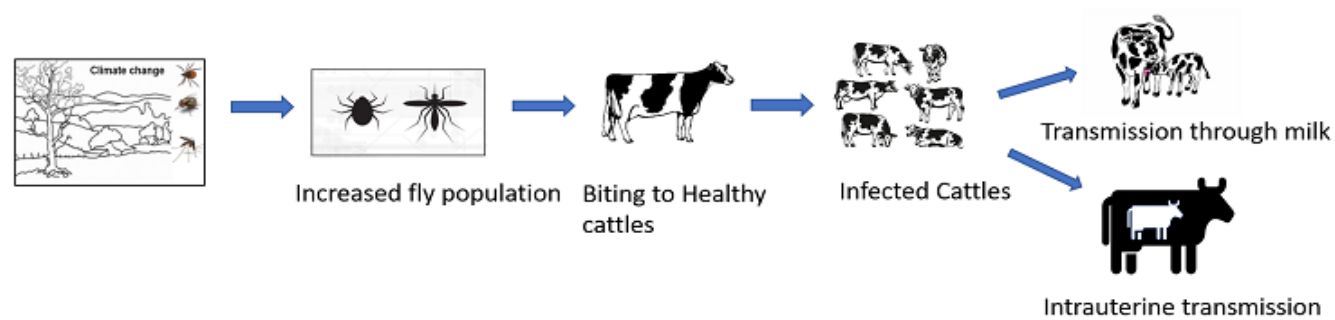
Domestic cattle and domestic water buffalo may get the pox disease, which is spread by vectors, and form nodules on their skin. The disease was formerly exclusive to Africa and the Middle East, but in 2015 it spread to southern Russian Federation territories, including the Balkans and the Caucasus. The poor, small-scale, and backyard farmers are struck the hardest by Lumpy Skin Disease (LSD) outbreaks, despite the fact that all players in the cattle sector lose money. Cattle output, milk yields, and animal body health are all severely affected by the illness. Hides become ruined, and animals either abort or become infertile as a result. The expense of a complete or partial stamp-out would be in addition to the losses sustained from operations. As a result of trade and movement restrictions placed on cattle, losses are suffered indirectly. Acute or sub-acute sickness can be caused by LSD in cattle and water buffalo, making it a severe danger to the stockbreeding industry^[1,2]. All bovines are vulnerable,

but calves and lactating cows are especially so^[3, 4]. This transboundary infection has been added to the list of diseases that need to be reported to the World Organization for Animal Health (OIE) because of the potential for rapid spread and the significant economic harm it could inflict. Recent cases in formerly disease-free nations highlight the urgency of addressing the spread, as well as any potential for eradication, of this pandemic^[5,6]. The lumpy disease virus is a large, enveloped, double-stranded Deoxyribonucleic Acid (DNA) virus (150 kbp) that shares a common ancestor with the Capri poxvirus genus sheep pox (SPPV) and goat pox (GPPV) viruses^[7-9]. Among the Poxviridae family of viruses that attack domestic ruminants, this one has the greatest economic impact. The virus's genome and lateral bodies are contained within the nucleocapsid, or capsid, which has a brick-like or oval shape. Due to substantial DNA cross-species hybridization, members experience serologic cross-

reaction and cross-protection. However, despite the prevalent misconception that Capri poxviruses are host-specific, some SPPV and GTPV strains can spontaneously cross-infect and cause sickness

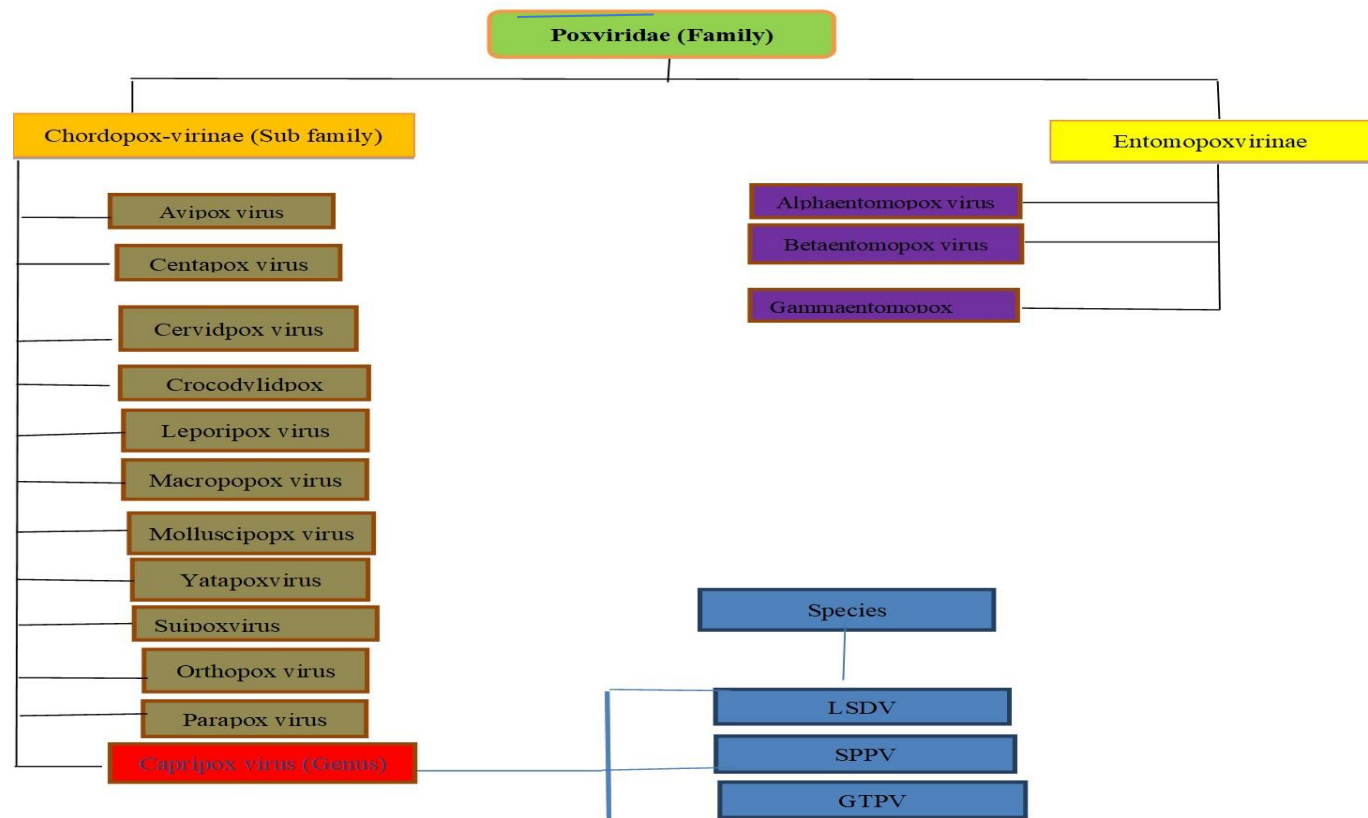
in both host species. Although LSDV may infect 2 sheep and goats in a lab, no instances of infection in the wild have yet to be seen [10].

Figure 1: Graphical abstract



Virology

Figure 2: Taxonomy of LSDV



Epidemiology and historical outbreaks

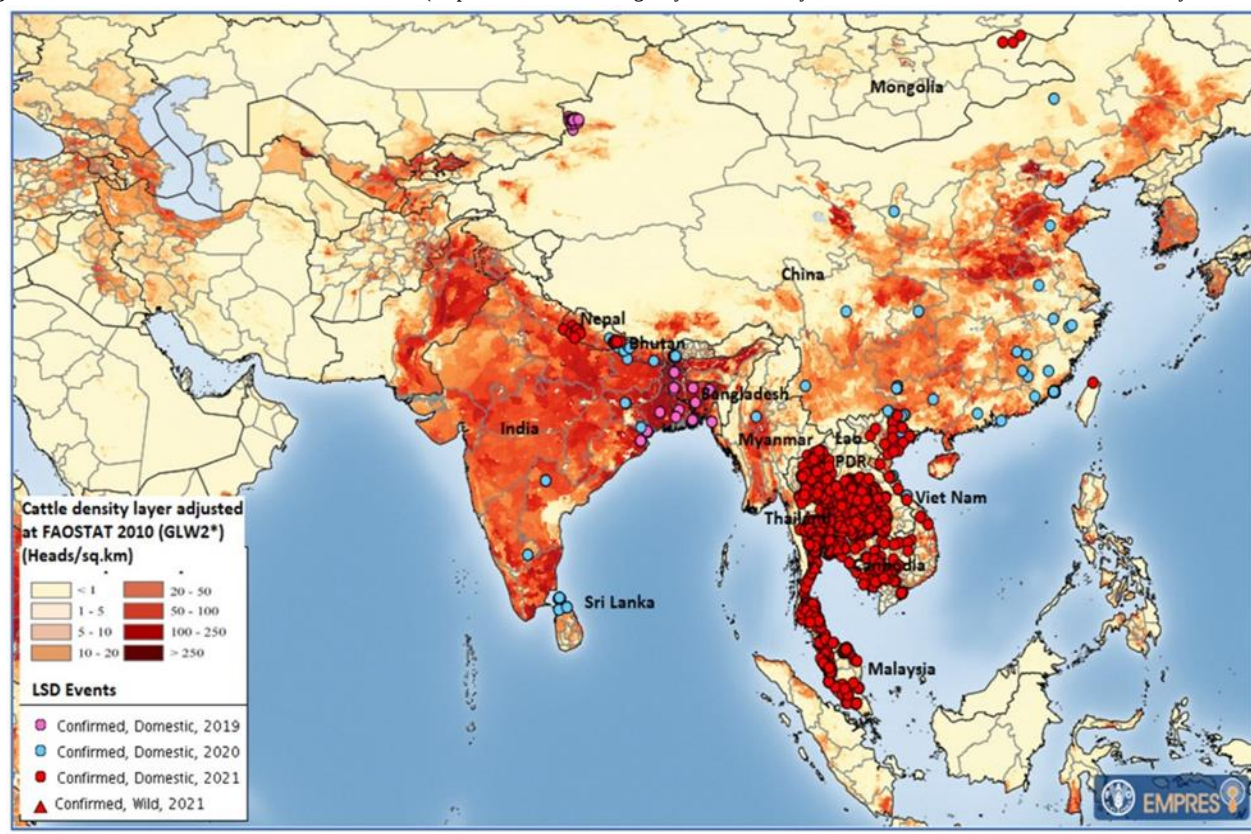
Most often, there will be a gap of several years between LSD epidemics. It is unclear how or where the virus can survive between outbreaks, and it is also unknown if it has a particular reservoir. Neither of these questions can be answered [12,13]. Even though outbreaks are frequently seasonal, they can occur at any time since, in many afflicted places, no season is completely devoid of vectors. Large-scale LSD outbreaks are often brought on by an increase in the number of naive (i.e., susceptible) animals, a surplus of active blood-feeding vectors, and unrestricted animal movement. The first example is typically related to the introduction of a new animal or animals into an existing herd or into close proximity to an

existing herd [14,15]. There is a death rate of up to 15% and a morbidity rate of 45%. Age, breed, and immunological health are among the variables that affect the host's susceptibility. In general, European cow breeds that are known for their high milk production are more vulnerable to the disease than native animal species from Africa and Asia. Cows that produce a lot of milk are typically the most seriously impacted by the disease [16]. Cattle that are symptomatic and virally positive are frequently found among sick animals, both in laboratory settings and in the wild. As a result, in order to put a stop to the disease's progression, it is absolutely necessary to consider the possibility that an affected herd contains infected animals that do not

exhibit any obvious clinical signs ^[17,18]. This is so that animals can employ blood-feeding vectors to disseminate the virus to additional hosts in this circumstance. The migration of cattle from contaminated

locations that have not been vaccinated or have not developed immunity creates a significant danger of spreading the disease^[19].

Figure2: LSD outbreaks in Asia from 2019 to 2021 (map source: FAO Emergency Prevention System Global Animal Disease Information System data).



Economic importance of LSD

The morbidity and death rate associated with LSD might vary greatly depending on the sensitivity of the host as well as the presence of insect vectors. When compared to native animals from Africa and Asia, the milk-producing European cow breeds that are generally more sensitive to the disease and suffer more severe effects from it. In regions where the illness is prevalent, the morbidity rate is typically somewhere around 10%, although it can range anywhere from 3% to 85%. In spite of the fact the condition is not associated with a significant mortality rate (1-3%), the economic losses that follow an LSD eruption are larger. It leads to significant economic losses as a result of a reduction in feed intake, milk output, weight conversion, abortions and infertility, and damaged hides. In addition to this, the sickness is a serious, notifiable condition that impedes international trade ^[22-24]. Recently, the virus that causes lumpy skin disease has been under scrutiny as a possible agent of agricultural terrorism ^[25]. This is because of the virus's potential to travel out of Africa and into the rest of the world. According to research conducted by Abutarbush et al. during an outbreak in Jordan, the typical cost of supportive antibiotic therapy was calculated to be 27.9 British pounds per individual ^[26]. Based on the results of a

questionnaire study that was administered to livestock producers in the Oromia regional state of Ethiopia, it was determined what the financial cost of therapeutic LSD would be ^[27]. The yearly financial cost comprised the average production losses, which were caused by illness and death and resulted in a loss of milk production, beef and, traction power, as well as expenditures associated with treatment and vaccination at the herd level. It was calculated that the average financial cost of infected herds was 6.43 United States dollars per head for indigenous zebu and 58 United States dollars per head for Holstein Friesian or crossbred cattle ^[28].

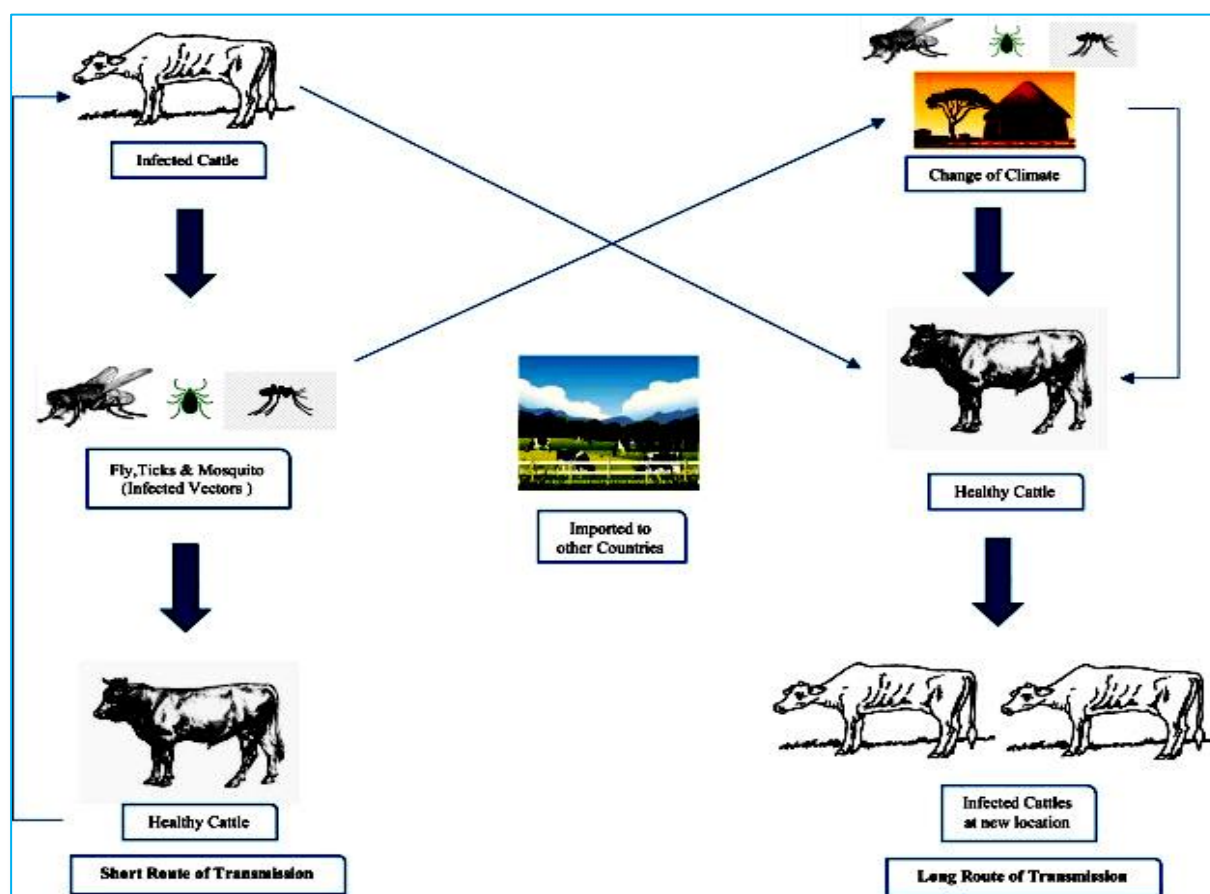
Transmission

There is a good chance that the primary vector will change depending on the habitat in which you are located ^[32]. There is evidence that common stable fly (*Stomoxys calcitrans*), the *Aedes aegypti* mosquito, and certain African tick species of the *Rhipicephalus* and *Amblyomma* spp. are capable of spreading the Lyme disease virus (LSDV). Although this risk has not been sufficiently studied, there is a potential risk of viral transfer from infected carcasses to uninfected living animals via insects. Direct contact is not usually considered to be a source of infection, but it is plausible that it may ^[33,34]. It is possible for infected animals to

simply remain viremic for a few days, but in more severe cases, viremia can persist for as long as two weeks. Infected animals will secrete infectious LSDV in their saliva, as well as in their nasal and ocular secretions, if they have lesions on their skin, mucous membranes in their mouths, and nasal canals^[35,36]. These spills could contaminate public spaces where animals gather to eat and drink. To this day, infectious LSDV has been found in the saliva and nasal discharge of patients up to 18 days after their first infection^[37,38]. More investigation is required to determine how long the infectious virus remains in the discharge after it has been expelled. Crusts provide excellent protection for infectious LSDV, particularly after crusts have detached from skin lesions where they were attached. Even though there are no experimental data available, it is highly

likely that natural or agricultural habitats will remain polluted for an extended period of time if they are not cleaned and disinfected thoroughly^[39]. Field experience has shown that when naive cattle are transferred to LSDV-infected holdings after being stamped out, they get sick within a week or two. This indicates that the virus remains either in vectors, in the environment, or in both of these places. After infection, the virus is still present in the bull's sperm, which means that either natural mating or artificial insemination may result in the disease being transmitted to females^[40]. It is well known that infected cows who are pregnant will give birth to calves that have skin lesions^[41]. Both contaminated milk and skin lesions that appear on the teats have the potential to spread the infection to nursing calves^[42,43].

Figure3: LSD transmission of different vectors



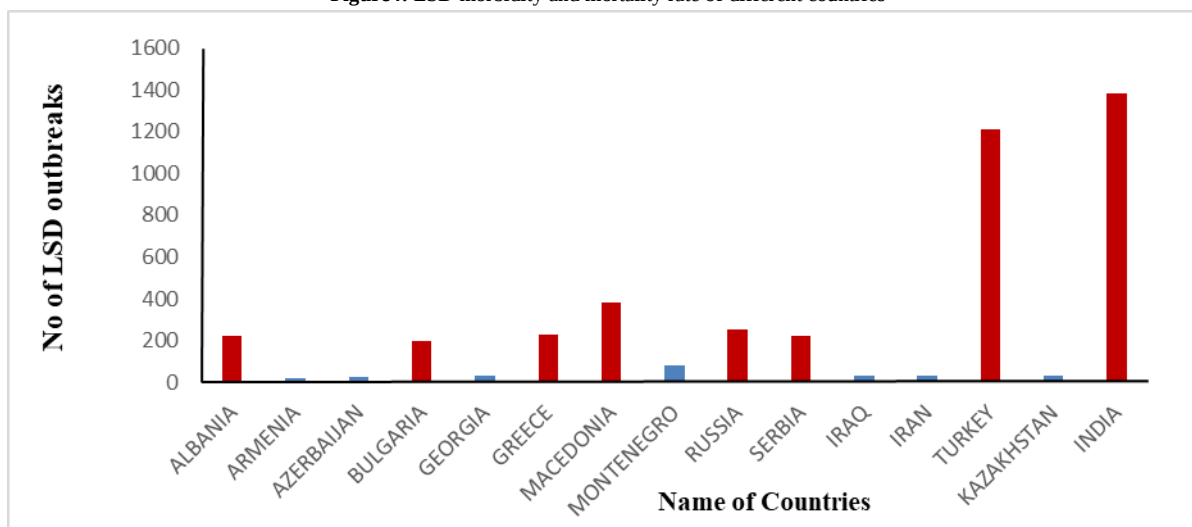
Role of wildlife in the disease spread

Seropositivity can show whether or not animals are involved in the disease's transmission^[44]. In animals, it seems that mild clinical cases are often neglected since it is difficult, if not impossible, to monitor the skin lesions^[45]. African buffaloes, blue wildebeest, eland giraffes, impalas, and larger kudus are just some of the other animals that have tested positive for the virus^[44,45]. Researchers identified the illness in an Arabian oryx. However, the

part that wildlife plays in LSD's epidemiology is still poorly understood^[46].

Morbidity and mortality rate of different country

The length of time it takes for an LSDV infection to become apparent in the wild is unknown. The death rate is typically around 10% (occasionally up to 40%), whereas the morbidity rate ranges from 5% to 45% (sometimes up to 100%). The severity of the illness on an animal depends on its age, breed, immunological condition, and stage of development^[47].

Figure4: LSD morbidity and mortality rate of different countries**Clinical manifestations and pathology**

Experimentally infected animals incubate for four to seven days, whereas naturally infected animals may take five weeks^[48].

Clinical signs include:

Lachrymation and nasal discharge are first seen.

Subscapular and pretemporal lymph nodes are swollen.

Fever may last a week(>40.50C).

Milk yield plummets.

nodular skin lesions (10-50 mm).

Mild instances have few lesions (Fig. 5A and 5B).

Infected animals with numerous lesions. Head, neck, perineum, genitalia, udder, and limbs are preferred places (Figs. 5C and 5D).

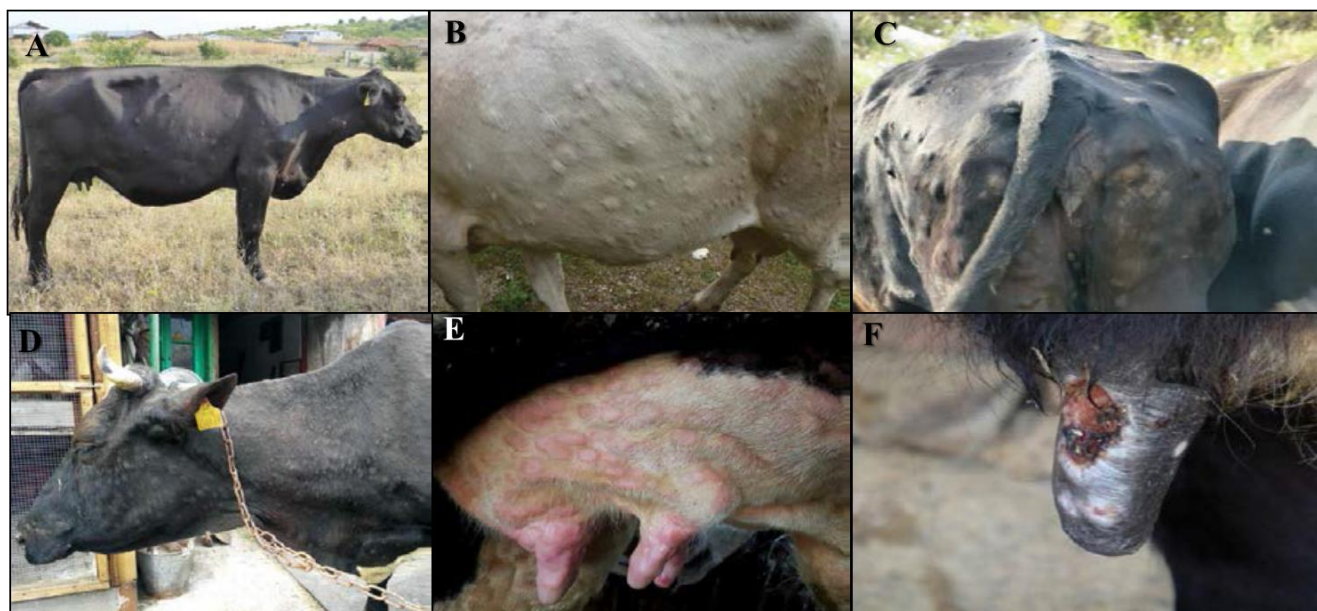
Deep nodules involve skin, subcutaneous tissue, and occasionally muscles. Necrotic plaques in the oral and nasal mucous membranes induce purulent nasal discharge and excessive salivation. Lesion centers ulcerate and scab over (Fig.5E and 5F). Nodules might last months.

Ulcerative lesions in one or both eyes' corneas can cause blindness.

Lesions on the legs and joints can cause subcutaneous infections, secondary bacterial infections, and lameness.

Subcutaneous lesions are revealed after skinning an animal with numerous skin lesions

Pox lesions are observed throughout the digestive and respiratory systems postmortem. Tracts and internal organs.

Figure 5: A)Mild LSD with full-body lesions, B) Cattle with severe lesions and swollen lymph nodes, C) Skin lesions in the perineum and genitalia, D)Severe LSD with head, neck, limbs, and body lesions, E) LSD case with udder and teat nodules, F) Ulcerative lesions in the teat

Diagnostic techniques

Consistent lab Confirmation of the virus's or antigen's presence along with the characteristic clinical indications of LSD can be diagnosed by looking for generalized nodular skin lesions and swollen superficial lymph nodes in afflicted animals. Before a diagnosis can be determined, a number of distinct diagnostic procedures (Table 1) that each call for a special kind of material must be completed in the lab. Electron microscopy examination and serum or virus neutralization testing are the gold standard procedures for identifying Capricorn pox viral antigen and antibody, respectively. Real-time Polymerase Chain Reaction (PCR) or traditional methods can be used to confirm an LSD clinical diagnosis. Gel-based PCR requires more time and effort than real-time PCR. It is a low-tech solution, yet it works well and is cost-effective. Calves that had been experimentally infected underwent comparative diagnostic testing, and the results showed that polymerase chain reaction (PCR) was the

most quick and accurate method for detecting viral DNA in both blood and skin samples. On the downside, it takes a long time to utilize; studies have shown that detecting viremia using viral isolation takes 1–12 days, whereas PCR takes 4–11 days. To propagate, LSDV needs just a tissue culture medium and cells from the bovine dermis or lamb testis (primary or secondary culture, respectively). This virus causes a cytopathic effect 1 and intra cytoplasmic inclusion bodies, in contrast to BHV-2, which produces syncytia and intra nuclear inclusion bodies. Since host defence against LSDV is primarily cell mediated, serological testing may not be sensitive enough to detect small and persistent infections or antibodies in vaccinated animals. It has proven difficult to build successful antibody enzyme-linked immuno sorbent assay. The indirect fluorescent antibody test (IFAT) for diagnosing and screening for LSD may be prohibitively expensive and time-consuming when compared to the ELISA approach.

Table1: Diagnostic Techniques

Test Objective	Methods	Epidemiological Analysis	Pre-movement screening	Assist in elimination	surveillance for infection prevalence	Prevalence of infections surveillance	Immune state after vaccination in specific animals or people or populations postvaccination
Agent attribution	virus segregation	+	++	++	+++	+	–
	PCR	++	+++	++	+++	++	–
	Electron microscopy	–	–	–	++	–	–
Identifying immunological responses of immune response	IFAT	++	+	+++	+	+	+
	VN	+++	++	++	++	++	++

Treatment, Prevention and Control

Treatment, prevention, and control Antibiotics are used as a symptomatic treatment for LSD in order to prevent further bacterial issues. Salib and Osman's clinical studies showed that a combination of antimicrobials, anti-inflammatory medications, supportive care, and antiseptics was effective in avoiding LSD problems and saving lives. The majority of adverse reactions to the experiment, including 1 corneal opacity (keratitis), mastitis, diarrhea, lameness, pneumonia, and myiasis, resolved within a few days to weeks. However, because treating LSD (and its complications) can be expensive and doesn't guarantee a full recovery, preventing the disease is preferable. This is due to the disease's potential to result in large economic losses due to damage to hides, milk loss from mastitis, and the loss of animal products due to death, miscarriage, fever, and myiasis. The importance of vaccination in eliminating lumpy skin disorders (LSD) in hotspots is shown by research by Gari et al. on the economic and epidemiological impacts of lumpy skin disorders in Ethiopia. Additionally, the authors observe that vaccination can reduce LSD financial losses by 17% per head in local zebu herds and by 31% per

head in herds of Holstein Friesian or crossbred animals. Due to mobility issues and the occasionally ineffectiveness of evacuating sick animals, vaccination is the only effective way to control unendemic areas. The economic cost of an LSD outbreak can be mitigated by the adoption of effective vaccinations as soon as possible. Capricorn poxviruses are well-known for the cross-protection they offer to one another. Therefore, either a single homologous (Neethling LSDV strain) or heterologous (sheep pox or goat pox virus) live attenuated vaccine can protect cattle from LSD infection. Vaccines against strains such as the LSDV Neethling strain, KSGPV O-240 and O-180, RM65 from Yugoslavia, SPP from Romania, and GTP from Gorgan are already available. In a recent study on the effectiveness of three CaPV strains against LSD in Ethiopia, Gari et al. discovered that the Gorgan GTP vaccine can successfully protect cattle against LSDV, while the Neethling and KSGP O-180 vaccines were ineffective and suggested the need for additional molecular characterization for those ineffective vaccines. Due to the possible safety difficulties connected with the use of the live attenuated LSDV vaccine, it is advised that nations that have

never had an LSD epidemic use the same vaccination used to protect sheep from sheep pox during LSD outbreaks. During an outbreak, eradication methods, including quarantine, the killing of diseased and in-contact animals, proper corpse disposal, cleaning and disinfecting of premises, and pest management, must be implemented as soon as is practical. The import of animals, bodies, skins, and semen from endemic areas must also be subject to severe regulations in disease-free countries^[49,50].

CONCLUSION

Viruses of the genus CaPV are responsible for the vector-borne illness known as lumpy skin disease (LSD), which was formerly exclusive to Africa. In recent years, however, it has been quietly expanding into other areas, including Europe. Animals with distinct nodular lesions typically have damaged skin and underlying tissues, although they may also develop in the conjunctiva, alimentary, respiratory, and urogenital tracts. The symptoms of the lesions, which include a loss in hide quality, chronic debility, decreased milk output, decreased body weight, infertility, abortion, and death, result in enormous financial resources being wasted on their treatment. Significant output losses could have a devastating impact on rural communities that rely heavily on livestock for income. The national level is also affected by the disease's existence, which has resulted in severe trade restrictions. Therefore, the following suggestions are made to encounter such frightening events: A description of the clinical-hematological and biochemical profiles of these animals, as well as the typical clinical manifestations of LSD in cattle, is required.^[51,52]

- Control measurements require precise, timely diagnosis.
- Vaccination against the LSDV using a strain that causes no immunogenic response is required annually in endemic regions.
- When insects are most active in their migration, it is crucial to implement measures to prevent vectors and limit animal movement.
- LSDV testing is required for all breeding bulls.

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DECLARATIONS

Conflict of Interest:

The authors declare no potential conflicts of interest.

Ethical approval:

This Article does not contain any studies with human participants or animals performed by the author.

REFERENCES

1. Balinsky CA, Delhon G, Smoliga G, et al, 2008. Rapid preclinical detection of sheeppox virus by a real-time PCR assay. *Journal of Clinical Microbiology* 46 (2), Pages- 438–442, DOI: 10.1128/JCM.01953-07.
2. Gallardo C, Okoth E, Pelayo, 2011. African swine fever viruses with two different genotypes, both of which occur in domestic pigs, are associated with ticks and adult warthogs, respectively, at a single geographical site. *Journal of General Virology* 92(2), Pages- 432-44, DOI: 10.1099/vir.0.025874-0.
3. Bowden TR, Babiuk SL, Parkyn GR, et al, 2008. Capripoxvirus tissue tropism and shedding: A quantitative study in experimentally infected sheep and goats. *Virology*, 371 (2), Pages- 380–393, DOI: 10.1016/j.virol.
4. Chaleampo Loymunkong, Kitsada Panyawut, Metas Chaleesean, et al, 2022. Potential waterbased extracts from fresh and dried auricularia auriculajudaeon the inhibitory effect of human papillomavirus type 16 and cervical cancer cell line in caski. *Journal of medical pharmaceutical and allied sciences*, V 11 - I 6, Pages - 5469 – 5475, DOI: 10.55522/jmpas. V11I6.4183.
5. EFSA AHAW Panel (EFSA Panel on Animal Health and Welfare), 2015. Scientific Opinion on lumpy skin disease. *EFSA Journal* .13 (1). Pages-68 -73, DOI: 10.2903/j.efsa.2015.3986.
6. EFSA. 2016. Urgent advice on lumpy skin disease. EFSA Panel on Animal Health and Welfare. ADOPTED. *EFSA Journal*. Pages-45-73, DOI: 10.2903/j.efsa.2016.4573.
7. El-Nahas EM, El-Habbaa AS, El-Bagoury et al, 2011. Isolation and identification of lumpy skin disease virus from naturally infected buffaloes at Kaluobia, Egypt. *Global Veterinaria*, 7, Pages-234-237, DOI:10.1186/s12917-022-03398-y.
8. Dubey A, GhoshNS, RathorVPS, et al, 2022. Sars- COV-2 infection leads to neurodegenerative or neuropsychiatric diseases. *International Journal of Health Sciences*, 6(S3), Pages-2184–2197, DOI.org/10.53730/ijhs. v6nS3.5980.
9. DubeyA, SinghR, KumarA, et al, 2022. A Critical Review on Changing Epidemiology of Human Monkeypox-A Current Threat with Multi-Country Outbreak. *Journal of Pharmaceutical Negative Results*. Pages- 660–671, DOI: 10.47750/pnr.2022.13.S01.82.
10. Gari G, Abie G, Gizaw D, et al, 2015. Evaluation of the safety, immunogenicityand efficacy of three capripoxvirus vaccine strains against lumpy skin disease virus. *Vaccine*33. Pages - 3256–3261, DOI: 10.1016/j.vaccine.2015.01.035.
11. Gari G, Bateau-Coroller F, Le Goff C, et al, 2008. Evaluation of indirect fluorescent antibody test (IFAT) for the diagnosis and screening of lumpy skin disease using Bayesian method. *Veterinary Microbiology* 129 (3-4). Pages- 269–280, DOI: 10.1645/0022-3395(2002)088[0594:EOAIFA]2.0.CO;2.
12. Gelaye E, Lamien CE, Silber R, et al, 2013. Development of a cost-effective method for capripoxvirus genotyping using snapback primer and dsDNA intercalating dye. *PLoS One*, 8 (10). Pages-230-235, DOI: 10.1371/journal.pone.0075971.

13. Gelaye E, Belay A, Ayelet G, et al, 2015. Capripox disease in Ethiopia: genetic differences between field isolates and vaccine strain, and implications for vaccination failure. *Antiviral Research*, 119. Pages- 28-35, DOI: 10.1016/j.antiviral.
14. Gelaye E, Mach L, Kolodziejek J et al, 2017. A novel HRM assay for the simultaneous detection and differentiation of eight poxviruses of medical and veterinary importance. *Sci Rep*, 7, Pages -428-92, DOI: 10.1038/srep42892.
15. Haegeman A, Zro K, VandenbusscheF, et al, 2013. Development and validation of three Capripoxvirus real-time PCRs for parallel testing. *Journal of Virology. Methods*, 193 (2), Pages- 446–451, DOI: 10.1016/j.jviromet.2013.07.010.
16. Ireland DC, Binopal YS, 1998. Improved detection of Capri poxvirus in biopsy samples by PCR. *Journal of Virological Methods*, 74 (1), Pages- 1–7, DOI: 10.1016/s0166-0934(98)00035-4.
17. Lamien, CE, Le Goff C, Silber R, et al, 2011. Use of the Capripoxvirus homologue of Vaccinia virus 30 kDa RNA polymerase subunit (RPO30) gene as a novel diagnostic and genotyping target: Development of a classical PCR method to differentiate goat poxvirus from sheep poxvirus. *Veterinarian Microbiology*. 149 (1-2), Pages 30–39, DOI: 10.1016/j.vetmic.2010.09.038.
18. Lamien CE, Lelenta M, Goger W, et al, 2011. Real time PCR method for simultaneous detection, quantitation and differentiation of Capri poxviruses. *Journal of Virological. Methods*, 171 (1), Pages -134–140. DOI: 10.1016/j.jviromet.
19. Le Goff, C, Lamien CE, Fakhfakh E, et al, 2009. Capripoxvirus G-protein-coupled chemokine receptor: a host-range gene suitable for virus animal origin discrimination. *Journal of general virology*. 90, Pages, 1967–1977. DOI: 10.1099/vir. 0.0 10686-0.
20. Menasherow S, Erster, O, Rubinstein-Giuni M, et al, 2016. A high-resolution melting (HRM) assay for the differentiation between Israeli field and Neethling vaccine lumpy skin disease viruses. *Journal of Virology Methods*, 23, Pages -12–15, DOI: 10.1016/j.jviromet.
21. Menasherow S, Rubinstein-Giuni, M, Kovtunen A, et al, 2014. Development of an assay to differentiate between virulent and vaccine strains of lumpy skin disease virus (LSDV). *Journal of Virology Methods*, 199, Pages - 95–101, DOI: 10.1016/j.jviromet.
22. Stubbs S, Oura CAL, Henstock, M, et al, 2012. Validation of a high-throughput real-time polymerase chain reaction assay for the detection of capripoxviral DNA. *Journal of Virology Methods*, 179 (2), Pages -419–422, DOI: 10.1016/j.jviromet.
23. Tuppurainen ESM, Venter EH, Coetzer JAW, et al, 2005. The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques. *Onderstepoort Journal of Veterinarian. Research*, 72 (2), Pages -153–164, DOI: 10.4102/ojvr.v72i2.213.
24. EFSA, 2015. European Food Safety Authority. Scientific Opinion on Lumpy Skin Disease. EFSA Panel on Animal Health and Welfare (AHAW). *EFSA Journal* 13: Pages -3986, DOI: 10.2903/j.efsa.2015.3986.
25. Abutarbush SM, Ababneh MM, Al Zoubil IG, et al, 2013. Lumpy Skin Disease in Jordan: Disease Emergence, Clinical Signs, Complications and Preliminary-associated Economic Losses. *Trans bound Emerging Disease* 62, Pages - 549-554, DOI: 10.1111/tbed.12177.
26. Babiuk S, Bowden TR, Dalman B, et al, 2008. Quantification of lumpy skin disease virus following experimental infection in cattle. *Trans bound Emerging Disease* 55, Pages -299-307, DOI: 10.1111/j.1865-1682.2008.01024.x.
27. Abutarbush SM, Ababneh MM, Al Zoubil IG, et al, 2013. Lumpy Skin Disease in Jordan: Disease Emergence, Clinical Signs, Complications and Preliminary-associated Economic Losses. *Trans bound Emerging Disease* 62, Pages -549-554, DOI: 10.1111/tbed.12177.
28. Gari G, Bonnet P, Roger F, et al, 2011. Epidemiological aspects and financial impact of lumpy skin disease in Ethiopia. *Preventive Veterinarian Medicine* 102, Pages - 274-283. DOI: 10.1016/j.prevetmed.
29. Coetzer, JA W, 2004. Lumpy skin disease. In J. A .W. Coetzer, & R. C. Tustin (Eds.), *Infectious diseases of livestock*, 2nd ed., Pages - 1268–1276.
30. Tasioudi KE, Antoniou SE, Iliadou P, Sachpatzidis A, et al. 2016. Emergence of lumpy skin disease in Greece, Transboundary and Emerging Diseases, 63, Pages -260–265, DOI: 10.1111/tbed.12497.
31. Sevik M, Dogan, M, 2017. Epidemiological and molecular studies on lumpy skin disease outbreaks in Turkey during 2014–2015. *Transboundary and Emerging Diseases*, 64(4), Pages – 1268. DOI: 10.1111/tbed.12501.
32. Yeruham, I, Nir, O, Braverman, Y, Davidson, M, et al, 1995. Spread of lumpy skin disease in Israeli dairy herds. *Veterinary Record*, 137. Pages - 91–93, DOI: 10.1136/vr.137.4.91.
33. Gari G, Waret-Szkuta A, Grosbois V, et al, 2010. Risk factors associated with observed clinical lumpy skin disease in Ethiopia. *Epidemiology and Infection*, 138, Pages -1657–1666, DOI: 10.1017/S0950268810000506.
34. Sevik M, Dogan, M, 2017. Epidemiological and molecular studies on lumpy skin disease outbreaks in Turkey during 2014–2015. *Transboundary and Emerging Diseases*, 64(4), Pages – 1268, DOI: 10.1111/tbed.12501.
35. Chihota CM, Rennie LF, Kitching et al, 2003. Attempted mechanical transmission of lumpy skin disease virus by biting insects. *Medical and Veterinary Entomology*, 17, Pages – 294–300, DOI: 10.1046/j.1365 2915.2003.00445.x.
36. Tasioudi K E, Antoniou S E, Iliadou P, et al. 2016. Emergence of lumpy skin disease in Greece, 2015. *Transboundary and Emerging Diseases*, 63, Pages – 260–265, DOI: 10.1111/tbed.12497
37. Chihota CM, Rennie LF, Kitching RP, et al, 2001. Mechanical transmission of lumpy skin disease virus by *Aedes aegypti*

- (Diptera: Culicidae). *Epidemiology and Infection* 126, Pages – 317–321, DOI: 10.1017/s0950268801005179.
38. Elhaig MM, Selim A, Mahmoud M, 2017. Lumpy skin disease in cattle: Frequency of occurrence in a dairy farm and a preliminary assessment of its possible impact on Egyptian buffaloes. *Onderstepoort Journal of Veterinary Research* 84, Pages – 1393, DOI: 10.4102/ojvr.v84i1.1393.
 39. Tuppuraine ES, Stoltz WH, Troskie M, et al, 2011. A Potential Role for Ixodid (Hard) Tick Vectors in the Transmission of Lumpy Skin Disease Virus in Cattle. *Transbound Emerging Disease* 58, Pages – 93-104, DOI: 10.1111/j.1865-1682.2010.01184.x.
 40. Tuppuraine ES, Coetzer JA, Venter EH, 2005. The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques. *Onderstepoort Journal of Veterinary Research*, Pages – 153-164, DOI: 10.4102/ojvr.v72i2.213.
 41. Bhanu Prakash V, Indrani BK, Hosamani A, et al, 2006. The current status of sheep pox disease. *Comp Immunology Microbiology Infectious Disease* 29, Pages – 27-60, DOI: 10.1016/j.cimid.2005.12.001.
 42. Tageldin MH, Wallace DB, Gertdes GH, et al, 2014. Lumpy skin disease of cattle: an emerging problem in the Sultanate of Oman. *Tropical Animal Health Product* 46, Pages – 241-246, DOI: 10.1007/s11250-013-0483-3.
 43. Babiuk S, Bowden T, Boyle D, et al, 2008. Capripoxvirus es: an emerging worldwide threat to sheep goats and cattle. *Transbound Emerging Disease* 55, Pages – 263-272. DOI: 10.1111/j.1865-1682.2008.01043.x.
 44. Gari G, Bonnet P, Roger F, et al, 2011. Epidemiological aspects and financial impact of lumpy skin disease in Ethiopia. *Preventive Veterinary Medicine* 102, Pages – 274-283, DOI: 10.1016/j.prevetmed.
 45. Abutarbush SM, 2017. Lumpy Skin Disease (Knopvelsiekte, Pseudo- Urticaria, Neethling Virus Disease, Exanthema Nodularis Bovis). In: Bayry J (eds.) *Emerging and Re-emerging Infectious Diseases of Livestock*. Springer International Publishing, Gewerbestrasse 11, 6330 Cham, Switzerland, Pages –309-326, DOI: 10.1002/vms.3.434.
 46. Gari G, Abiea G, Gizawa D, et al, 2015. Evaluation of the safety, immunogenicity and efficacy of three capripoxvirus vaccine strains against lumpy skin disease virus. *Vaccine* 33, Pages-3256-3261, DOI: 10.1016/j.vaccine.
 47. Tuppurainen E, Oura C, 2012. Review: Lumpy skin disease: An emerging threat to Europe, the middle east and Asia. *Transbound Emerging Disease* 59, Pages – 40-48, DOI: 10.1111/j.1865-1682.2011.01242.x.
 48. Booth TF, Davies CR, Jones LD, et al, 1989. Anatomical basis of Thogoto virus infection in BHK cell culture and in the Ixodid tick vector, *Rhipicephalus appendiculatus*. *Journal of Genetic Virology* ,70, Pages - 1093–1104, DOI: 10.1099/0022-1317-70-5-1093.
 49. Rouby S, Aboulsoud, E, 2016. Evidence of intrauterine transmission of lumpy skin disease virus. *Veterinary Journal*, 209, Pages - 193–195, DOI: 10.1016/j.tvjl.
 50. Stram Y, Kuznetsova L, Friedgut O, et al, 2008. The use of lumpy skin disease virus genome termini for detection and phylogenetic analysis. *Journal of Virological Methods*, 151, Pages-225–229, DOI: 10.1016/j.jviromet.
 51. Zheng M, Liu Q, Jin, N Y, et al, 2007. A duplex PCR assay for simultaneous detection and differentiation of Capripoxvirus and Orf virus. *Molecular and Cellular Probes*, 21, Pages-276–281, DOI: 10.1016/j.mcp.2007.01.005
 52. Manoj Kumar Prajapati , Ayush Jain, Anuj Jain, et al, 2022. Clinical and demographical profile hiv and tb confection with special emphasis on the sputum and CBNAAT positivity in vindhya region of India. *Journal of medical pharmaceutical and allied sciences*, V 11 - I 6, Pages - 5485 – 5488, DOI: 10.55522/jm pas.V11I6.4593.

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