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Lumpy Skin Disease Spread in India: A Review

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ABSTRACT

The transboundary illness lumpy skin disease, which is significant commercially, affects cows and buffaloes. The lumpy skin disease virus (LSDV), a constituent of the Poxviridae family and genus Capripoxviridae, causes it to manifest (Neethling strain). The virus has been compared to Allerton and Bovine Mammilitis Virus, which results in lesions that resemble those found in the early stages of LSD. Some of the key clinical signs are typical skin nodules on the body, increased body heat, nasal discharge, excessive salivation, breast tissue inflammation, lessened milk supply, cachexia, depression, and unwillingness to move. The contagion is widespread in African countries and the Middle East regions. Asia and other areas have recently started to experience expansion. LSD morbidity rates of 13.93 percent in cattle have recently been observed in India (2022) during six months. The economic impact of this viral disease is quite concerning due to the severe morbidity and high mortality rates it produces in unprotected cattle and calves. It could also be used as a tool in economic bioterrorism and threaten international trade. The COVID-19 outbreak and the subsequent implementation of sanctions have hastened the disease's spread in India. The current study aims to provide recent developments on a range of disease-related topics, including transmission, epidemiology, diagnosis, and preventative and therapeutic approaches. This review's findings emphasize the significance of LSDV diagnostics using molecule and strain monitoring. The information would also aid in developing discriminative and prevention strategies for LSDV in India.

INTRODUCTION

In many developing parts of the developing world, raising livestock is one of the main ways to enhance lifestyle. The livestock industry is quite dynamic and accounts for 40% of all agricultural output globally. It also supports livelihood and food security. According to the FAO, India has the world's largest livestock sector, which accounts for 15% of all livestock worldwide. Owns the most cattle, with around 535.78 million. According to India's 20th livestock census, India ranks top in both overall buffalo population (109.85 million) and milk output. A significant factor in the fall in cattle productivity is animal illnesses. LSD is one of the developing viral diseases with the greatest economic impact [1]. A high temperature, enlarged lymph nodes, and restricted nodules distinguish it. Cattle of various ages and breeds are affected by this disease, which has a high rate of morbidity but a low risk of death. Decreased milk supply, sterility, etc., result in large-scale economic losses. Being a vector-borne disease, its rapid spread is challenging to control. To protect India's susceptible cattle from new viral infections like LSD, diagnostic and preventive measures are needed.

2. History

LSD has a long history. In Zambia (then Northern Rhodesia), the clinical signs of LSD were first recorded in 1929 [1, 2, 3]. The disease reoccurred irregularly until 1943, when it spread to Botswana, Zimbabwe (Southern Rhodesia), and South Africa in 1945 [4]. Later in 1998, there was a report of an outbreak of lumpy skin disease (LSD) in cattle in the "ElMenia Governorate" of Upper Egypt [5]. El-Kohly et al.. (2008 documented a recurring outbreak of LSD in Egypt [6]. Following these occurrences, massive LSD epidemics were recorded in Turkey, Oman, and Greece throughout three years from 2013 to 2014 [7, 8, 9]. Even while capripoxviruses are far less frequent than they were fifty years ago, they continue to spread in new regions [10].

Previously, LSD symptoms were assumed to be the result of poisoning or an allergy to bee stings. Countries like Botswana, Zimbabwe, and the Republic of South Africa reported similar clinical symptoms, where the disease's contagious nature was disclosed [11]. Currently, LSD is causing significant losses to cattle production in various regions of Europe and Asia. According to an FAO risk assessment estimate, the illness was documented in seven countries, beginning with “China and India in August 2019, Nepal in June 2020, Taiwan in July 2020, Bhutan and Vietnam in October 2020, and Hong Kong in November 2020” [1].

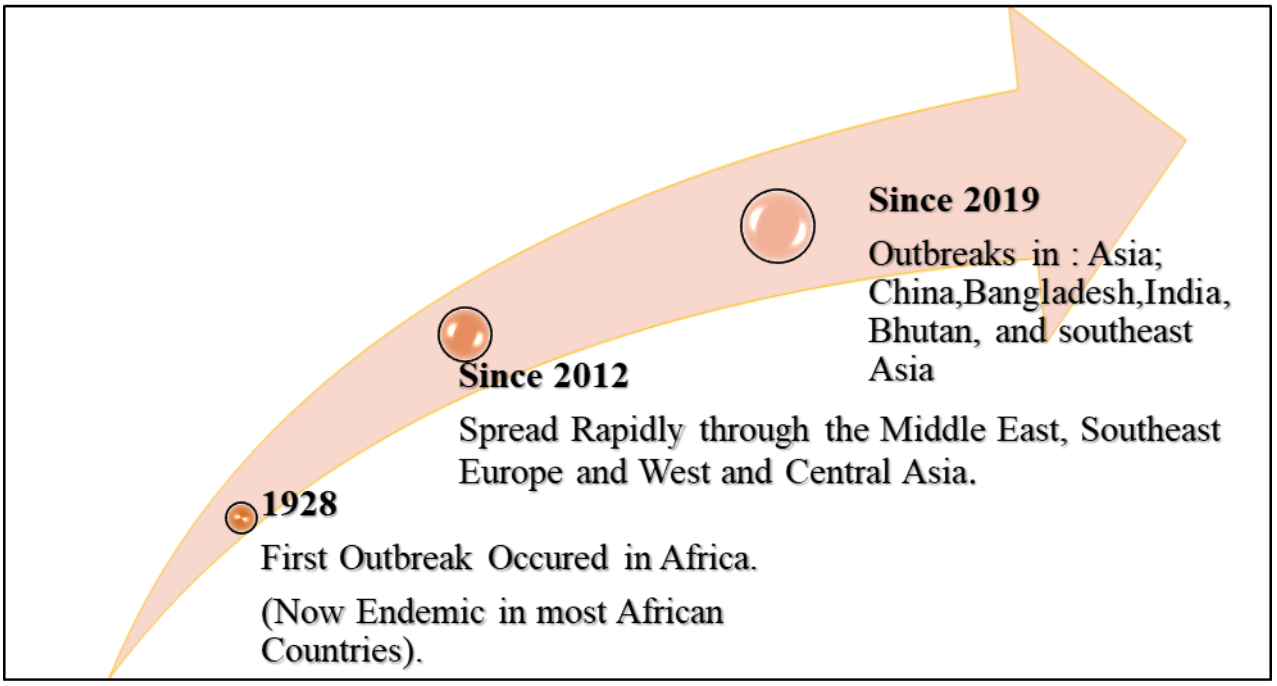


Fig 1. Influence of LSD worldwide post initial outbreaks [11].

[***Source***: *Lumpy Skin Disease infecting cattle in India; here's all you need to know*. (2022, September 30).

2.1 India LSD timeline:

India, which has the world's largest cow population (303 million head), witnessed the infection spread for the first time in 15 states in just 16 months. In August 2019, five districts were dealing with exotic cowpox when the first LSD pandemic in Odisha was reported. There were 182 affected animals among 2,539 cattle, with a 7.1% apparent morbidity rate and no deaths. 29.87 percent of the cattle tested positive for capripoxvirus using a generic PCR, while 37.66 percent of the calves tested positive for LSDV in PCR analysis. Out of 102 samples, 17 asymptomatic in-contact cattle samples and 60 suspected LSD samples were discovered [12].

Furthermore, Kumar et al., (2021) collected samples from "Ranchi, Jharkhand, India", which included an organised cattle dairy farm and small dairy units in villages such as "Tussum, Nagri, Khemra, and Gadgaon", indicating disease spread in nearby geographical areas at the same time [13].

3. Recent outbreaks and impact:

In India, LSD made a comeback in 2022. In Gujarat's Kutch region, the initial incidence of Lumpy Skin Disease was recorded in April. Since then, it had expanded to Punjab, Himachal Pradesh, Andaman & Nicobar, and Uttarakhand by early August. It first appeared in Rajasthan at the end of July. Then it amplified to Haryana, Uttar Pradesh, and Jammu & Kashmir. It was reported in the following weeks in Delhi, Jharkhand, Madhya Pradesh, and Maharashtra. As of September 11, the virus had roughly tainted 16 lakh livestock across 197 districts. It surpassed 50,000 deaths- mostly cows- of the roughly 75,000 cattle the disease had claimed had been documented in Rajasthan [14].

4. Etiology and genetic basis of lumpy skin disease:

The Lumpy skin disorder is caused by a member of the family "Poxviridae", specifically the genus "Capripoxvirus". The family Poxviridae encompasses LSDV [15]. The family Poxviridae consists of two subfamilies: Chordopoxvirinae, which includes poxviruses that infect vertebrates, and Entomopoxvirinae. LSDV is classified within the subfamily Chordopoxvirinae [17]. It can be identified using serological tests and has multiple antigens in common with the viruses that cause sheep and goat diseases [18, 19].

The individuals in this family are among the largest viruses [18]. The virus has an oval shape, which has been confirmed through electron microscopy analysis [20]. The diameters range from 220 to 450 nanometres in length and from 140 to 266 nanometres in width. The LSDV genome consists of double-stranded DNA [18]. The sequence of the LSDV genome is known. 156 putative genes are included in the 151 kbp LSDV genome [21]. The RNA polymerase 30 kDa component, primarily crucial in replication, is encoded by the RPO30 gene [18, 22]. The 'G-protein-coupled chemokine receptor', which is involved in host immunity modulation, is encoded by the host range gene known as GPCR [23]. Potential promoter activity has been identified in a 56 bp region of the LSDV genome that mimics known early and late promoters of the poxvirus [24]. Despite having significant links to other Chordopoxvirinae members, it possesses a specific gene collection that determines the viral host range and pathogenicity. [18]

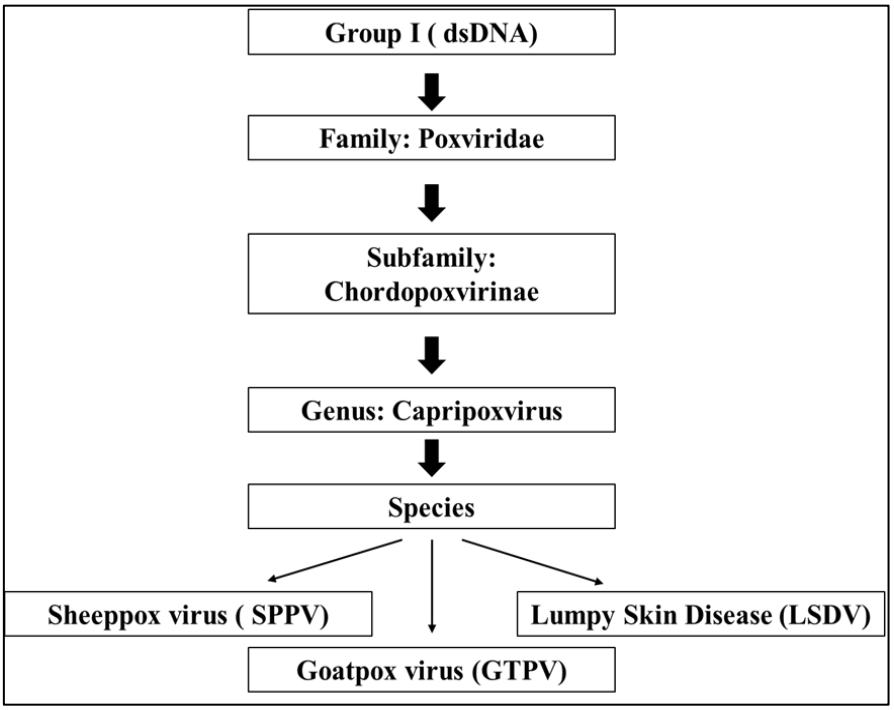


Fig 2: Classification of Capri pox viruses [18].

[***Source and image credits***: Al-Salihi, K. A. (2014, December 1).

Heat treatment at 55°C and 65°C for 30 minutes makes LSDV susceptible. At 55 °C, the virus deactivates in two hours, but at 65 °C it takes 30 minutes. Extremely alkaline or acidic surroundings are favourable to the virus. Nonetheless, it can withstand transient pH changes of 6.6 to 8.6 at 37 °C for five days without significantly lowering titers [25]. World Health Organisation (WHO) suggests that "Iodine (1:33 dilution), formalin (1%), quaternary ammonium compounds (0.5%), phenol (2% for 15 minutes), ether (20%), chloroform, and sodium hypochlorite (2-3% concentration) are all effective disinfectants against this virus" [26].

Research has shown that the virus may be retrieved from skin nodules preserved at minus 80°C even ten years after they were harvested. The infected tissue sample can remain viable for viral culture after six months when conditioned at 4 °C [27].

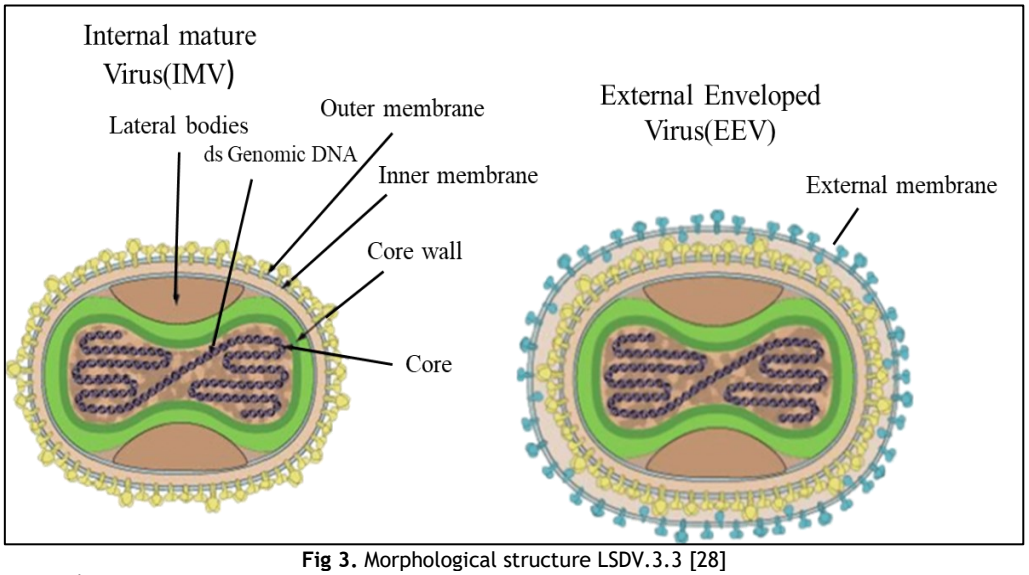


Fig 3. Morphological structure LSDV.3.3 [28]

[***Source and image credits***: Gumbe, A. A. F. (2018). Review on lumpy skin disease and its economic impacts in Ethiopia.]

5. Epidemiology:

5.1 Geographical Spread in India:

In 2019, the illness first arrived in India. Odisha reported possible occurrences of Lumpy Skin Disease in August of that year. However, the first outbreak of the disease to be confirmed in a lab was reported in November 2019 [2,14]. Later, it became widespread, covering major states in India [2].

5.2 Transmission:

5.2.1 Host susceptibility and risk factor:

Lumpy skin disease is not 'zoonotic' in nature. '*B. taurus'* bovines with thin skin 'high-producing' are typically more seriously affected than zebu-type or crossbred cattle. 'It can cause illness in domestic buffaloes rarely; however, many cattle are susceptible to lumpy skin disease, which can lead to several illnesses [29, 30, 31]. Although both sexes are susceptible, all breeds and age groups are affected by LSD [7]. There have been reports of domestic buffalos with lower morbidity rates from LSD infection than from cattle [32].

The prevalence of LSD significantly increases with the existence of a warm, humid agro-climate that is home to numerous vector populations [33]. Numerous research studies indicate a correlation between the occurrence of LSD and the addition of new animals

to a herd, as well as husbandry practices, including group grazing and drinking sites. However, no direct correlation has been established between the disease and cattle migrations. [34, 35].

5.2.2 Transmission method:

Although there isn't a specific way that LSD is spread, there is circumstantial evidence that suggests that biting insects could spread the sickness. Additionally, it is generally known that arthropod vectors are the primary means of LSDV transmission, as opposed to clinically ill animals using contaminated objects or materials coming into contact with susceptible animals. The virus can also be conveyed to vulnerable cattle through infected animals' blood, saliva, semen, and milk. Additionally, nodules that form on the Mucoid membranes of the mouth, udder, rectum, and genitalia are potential sources for releasing sufficient viruses to serve as infection-causing agents [31, 36].

Although actual experimental data is sparse, direct contact has generally been demonstrated to be an ineffectual pathway for the transmission of LSDV. There exists the possibility of LSDV transmission by direct touch, albeit at low rates and efficiency [37, 38 39]. Capripoxviruses, which include sheeppox (SPP) and goatpox (GTP), can be transmitted through direct contact with virus-containing droplets and aerosols [40, 41].

Additionally, pollutants, fomites, and vectors pose indirect transmission. One example of how different genera within the Poxviridae family have distinct transmission methods both within and between genera is the capripoxvirus family [42]. Sharing water supplies and adding new animals to a herd appears to enhance the chance of LSD outbreaks [37, 43, 44]. More testing is required to confirm if contaminated milk or skin lesions on the mother's udder and teats are the sources of indirect transfer from the mother to the calf [37, 45].

Arthropods can also transmit LSD, although this depends on several factors, including vector competence, host availability, bite frequency, and chance of feeding activity. [37, 46]. The transmission of LSD to cattle is largely observed in mosquito species such as *Aedes natronius*, *Culex mirificus*, *Culex quinquefasciatus*, and *Anopheles stephensi* [37, 47, 48]. Biological transmission is more closely associated with a single or small group of vector species than the mechanical mode of transmission [49]. As a result, LSD transmission is a complicated process that needs more empirical data.

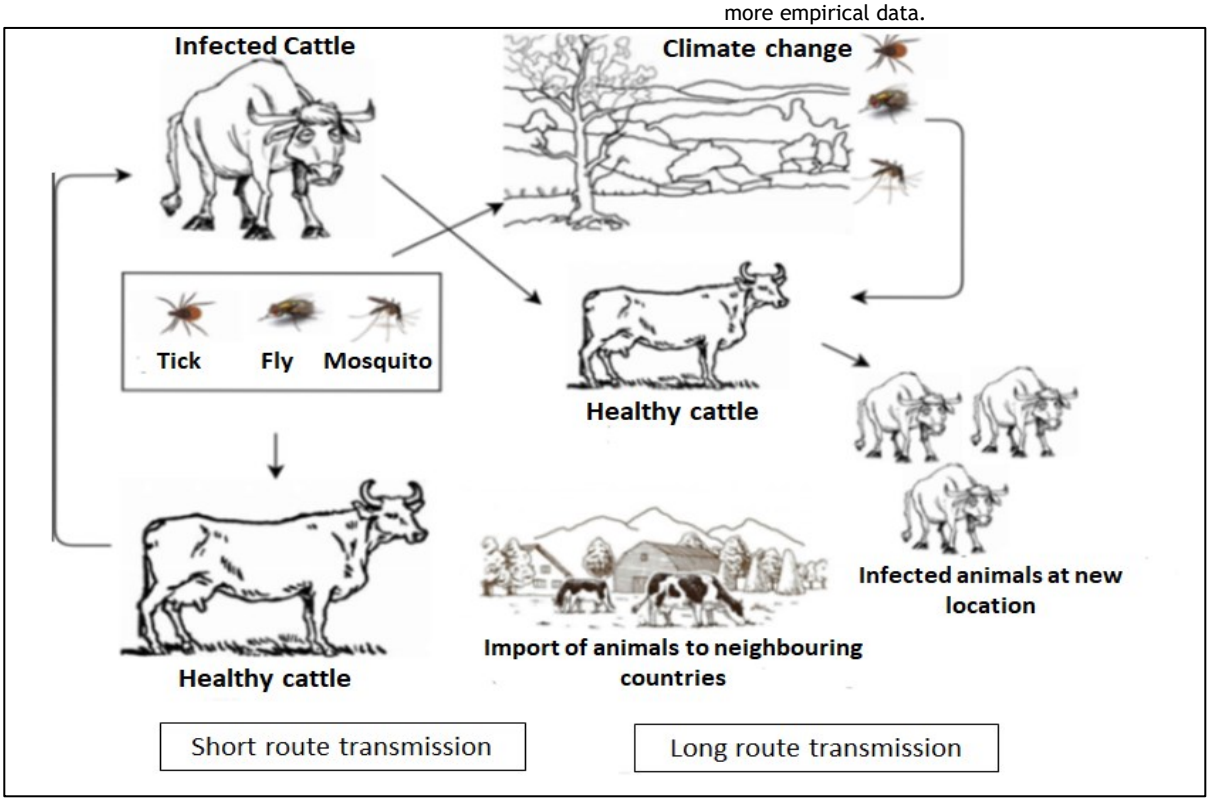


Fig 4. Transmission of LSDV [31].

[***Source and image credits***: Gupta, T., Patial, V., Bali, D., Angaria, S., Sharma, M., & Chahota, R. (2020). A review: Lumpy skin disease and its emergence in India. Veterinary Research Communications, 44(3–4), 111–118. https://doi.org/10.1007/s11259-020-09780-1]

5.3 Pathogenesis:

Compared to other viral illnesses, the pathophysiology of LSD is not well understood [50]. LSD has a gradual pathophysiology that begins with viral injection and progresses to local implantation and multiplication, viremia, and viral dissemination to certain tissues and organs [51, 52]. A period of incubation of LSDV is usually 5 weeks in the event of spontaneous infections, however, it fluctuates between four and seven days clinically [53]. LSDV, like other Capripoxviruses, shows a "tissue trophism for keratinocytes" [54]. Ultimately, there is ulceration with exudation, which causes scab and crust development, as well as varying degrees of haemorrhage, congestion, and oedema in the surrounding area [55, 49]. The virus particles may be passed on to additional infection regions, including the lungs, liver, kidney, and other lymph nodes [55]. Thus, in a nutshell, the pathogenic pathway of viruses often involves implantation at the entry point, reproduction in cells, dissemination to target organs, and diffusion into the surrounding environment [56].

Histopathological changes during the initial stage include lymphangitis and vasculitis with concomitant infarction and thrombosis. As regional lymph nodes grow, they become clogged, leading to pyaemia and cellulitis as shown in Fig 6 [57]. LSD infection can cause hyperplasia, including intracytoplasmic inclusion bodies including eosinophilic cells, macrophages, lymphocytes, and vasculitis due to virus trophism [58, 59, 60]. Initial serum exudation from LSD skin nodules is possible, but they eventually acquire an infarct. Oedema, haemorrhages, and congestion are possible conditions in the adjacent tissues. Enlarged lymph nodes are typical in the necrotic cores, as are subsequent bacterial infections [61]. Several virus-encoded factors are created during infection and have an impact on disease aetiology [62, 63].

5.4 Clinical Manifestation:

The two febrile stages (biphasic fever) that characterize clinical symptoms develop after a variable incubation period of 4 to 12 days. The infected animal's temperatures rise to 40 to 41.5°C, which persist for 6 to 72 hours or more [64]. Other signs of

infection in animals include tearing, elevated secretions from the nose and mouth, and a decreased urge to move. Although they are independent of the sex or age of the animal, the intensity of the clinical manifestations differs according to the group management approach. In a few rare instances, lesions cover the affected animal's skin [18]. The typical lesions range in size from 5 to 50 mm and are spherical and irregular. They look like firm, slightly elevated areas of skin covered by confined patches of upright hair. Additionally, the animal develops disease sores on its nares and muzzle [18].

The smaller nodules frequently consolidate to create larger lesions and peel off, causing a condition termed "sit-fasts", which provide the nidus source of further vector attraction and secondary bacterial infections [65]. Because of the implication of the entire skin and muscles, extreme pain causes the animal to become unwilling to move; at times [66, 67]. Severe cases can cause inflammatory lesions in the mouth, trachea, larynx, and oesophagus [67].

The acute stage of the sickness frequently results in abortion [69, 70, 71, 72, 73]; LSD skin abrasion has been observed on both living calves and aborted babies. Infertility is an issue after an LSD infection. The majority of infected cows undergo a cessation of ovulatory activity, usually due to weak body conditions, and females remain anoestrous for several months. Lesions can be seen on the genitalia of infected bulls. After two to three weeks, the skin wounds slowly appear more tenacious [18].

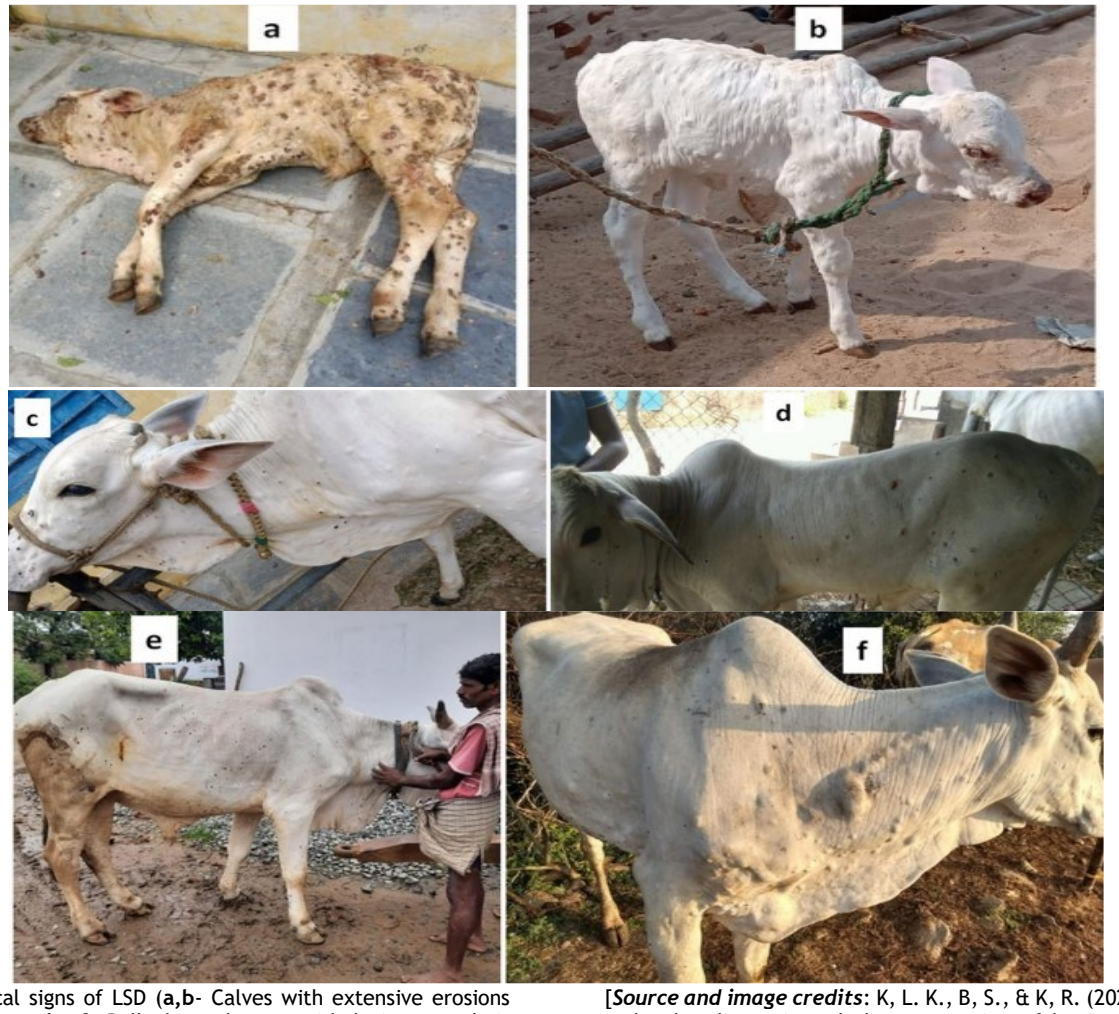


Fig 5. Clinical signs of LSD (**a,b**- Calves with extensive erosions and nodules, **c,d,e,f-** Bullocks and cows with lesions on their faces, necks, and in some cases, their entire bodies giving them a punched-out look with enlarged lymph nodes)[74].

[***Source and image credits***: K, L. K., B, S., & K, R. (2021). Clinico-molecular diagnosis and characterization of bovine lumpy skin disease virus in Andhra Pradesh, India. Tropical Animal Health and Production, 53(4). https://doi.org/10.1007/s11250-021-02872-3].

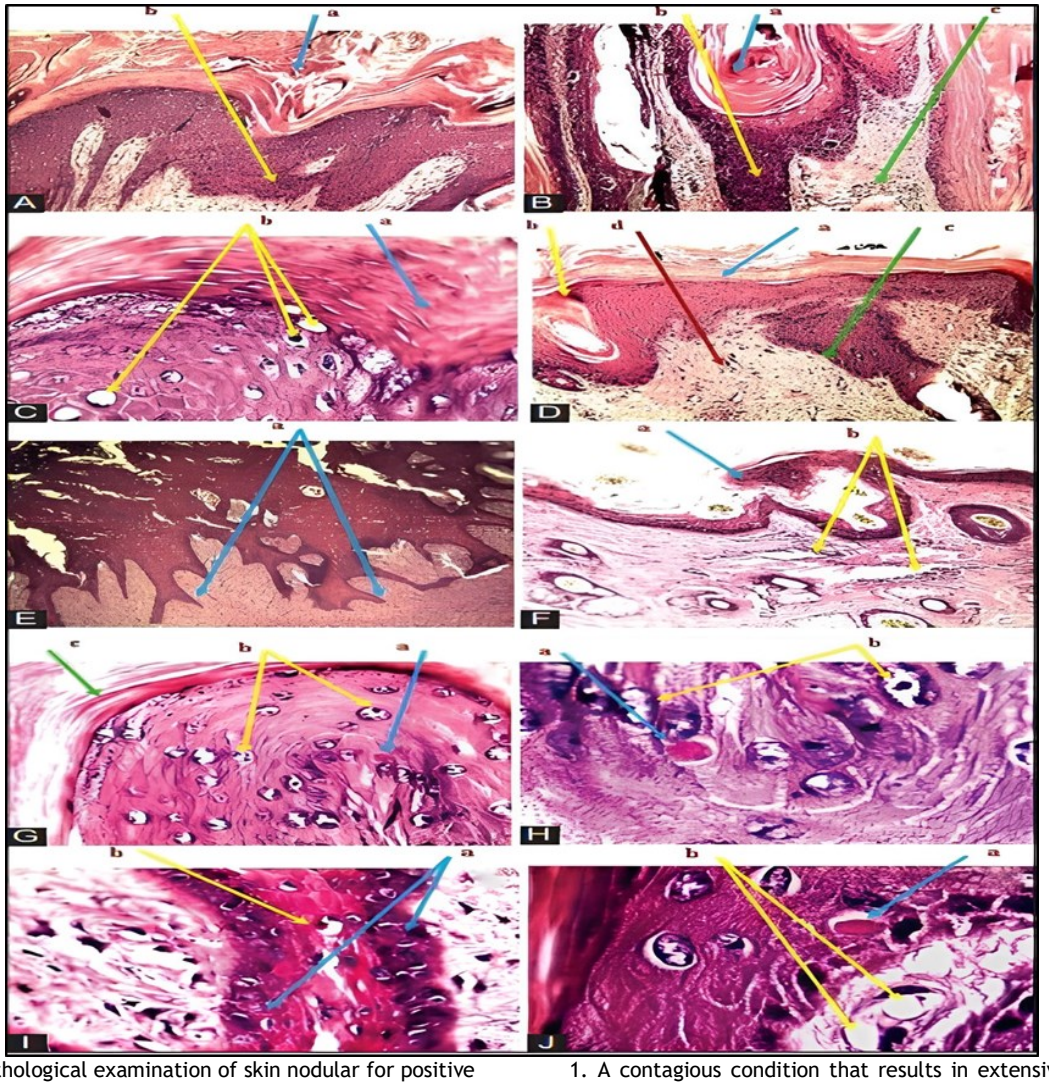


Fig 6: "Histopathological examination of skin nodular for positive lesions: (A): (a) Epidermal hyperkeratosis. (b) Stratum basale hyperplasia. (B): (a) Hyperkeratosis. (b) Hyperplasia. (c) Presence of inflammatory cell infiltration. (C): (a) Hyperkeratosis. (b) Granulocyte vacuolation and swelling. (D): (a) Hyperkeratosis. (b) Acanthosis. (c) Hyperplasia. (d) Inflammatory cell infiltration. (E): (a) Dermal-directed hyperplasia. (F): (a) Acanthosis. (b) Inflammatory cell infiltration within the dermis. (G): (a) Presence of intracytoplasmic inclusion bodies. (b) Granulocyte vacuolation. (c) Hyperkeratosis. (H): (a) Eosinophilic inclusion bodies. (b) Granulocyte vacuolation. (I): (a) Eosinophilic inclusion bodies. (b) Proliferation of basal cells within the epidermis. (J): (a) Eosinophilic inclusion bodies. (b) Granulocyte vacuolation" Gharban et al., 2019 [59].

[***Source and image credits***: Gharban, H. A., Al-Shaeli, S. J., Al-Fattli, H. H., & Altaee, M. N. (2019). Molecular and histopathological confirmation of clinically diagnosed lumpy skin disease in cattle, Baghdad Province of Iraq. Veterinary World, 12(11), 1826].

6. Diagnosis:

Mortality, morbidity, and clinical signs can be used to make a preliminary diagnosis of LSD. Examples include [18, 36]:

1. A contagious condition that results in extensive skin nodules [18, 36].
2. Low mortality and ongoing fever [18, 36].
3. Lungs with oedema and isolated lobular atelectasis [75].
4. Synovitis where the synovial fluid contains fibrin [76].
5. Painful lesions may appear on the testicles and bladder [77].

6.1 Histopathological characteristics:

The biopsy of early skin lesions is advised for histopathology and held in buffered formalin at 10% [63]. The most important histological characteristics are the following [63]:

1. Nodules that affect the subcutaneous tissue, skin, and muscle and Vasculitis, necrosis, oedema, haemorrhage, and congestion are commonly present along with adjacent musculature.
2. Lymphocyte augmentation, oedema, blockage, and bleeding.
3. Condition of Cellular infiltrates, thrombosis, and vasculitis [63].

- **LSD can be definitively diagnosed based on [78]:**

1. Transmission electron microscopy (TEM) [79].
2. Fluorescent antibody tests [78].
3. Agar gel immunodiffusion (detecting the precipitating antigen of capripoxvirus) [80].
4. ELISA [80].
5. PCR technique [80].

Table 1
Diagnosis of LSDV [Namazi et al., 2021] [63]

Techniques	Purposes					
	Animals from infection	Animals before migration, free of infection	Contribution to eradication policies	Confirmation of clinical cases	Prevalence of infection surveillance	Immune status post-vaccination
	Detection of the pathogen					

Virus Isolates	+	++	+	+++	+	-
PCR	++	+++	++	+++	+	-
Electron microscopy	-	-	-	+	-	-
	Immune response detection					
Virus neutralization	++	++	++	++	++	++
Electron microscopy	+	+	+	+	+	+

6.2 Differential diagnostic evaluation:

Over the years, misdiagnosis and underreporting have likely become more prevalent as a result of veterinarians in new areas confirming cases of lumpy skin disease without prior expertise with the condition [81].

Many illnesses might have symptoms that resemble LSD. It is critical to have a reliable diagnosis to provide sensitive herds with optimal prevention and management approaches. To differentiate LSD from pseudo-LSD resulting from infections caused by bovine herpesvirus-2 (BoHV-2), urticaria, insect bites, besnoitiosis, nocardiosis, demodicosis, onchocerciasis, pseudo-cowpox, bovine papular stomatitis, cowpox, foot and mouth disease, bluetongue, mucosal disease, malignant catarrhal fever, and infectious bovine rhinotracheitis, a differential diagnosis is necessary [82]. Other symptoms of hypersensitivity (photosensitisation, insect and spider bites) may cause ambiguity with LSD lesions. These disorders can be differentiated using PCR and other antigen or antibody-specific assays [83]. Histopathology allows for ruling out infections caused by viruses, bacteria, or fungal causes of nodular formation in clinical instances. Cytopathic effects, such as "eosinophilic intracytoplasmic inclusion bodies", are well-recognised in cases of LSD [84].

The following conditions can be confused with LSD [18]:

1. Bovine virus-induced diarrhoea.
2. An allergy to insect bites, rinderpest, and malignant catarrhal fever in cattle.

7. Treatment:

No specific treatment exists for lumpy skin disease [85]. A variety of drugs and supplementary therapies are administered to treat infections in animals [18, 82,].

1. The sick animal needs to be kept apart from the herd [18].
2. Many skin-routine drugs can be tested to treat skin contusions. Animals can be scrubbed with potash water [18].
3. To prevent allergies, antihistamine medications should be administered via vaccination [18, 82,].
4. The spread of secondary bacterial infections must be avoided [18, 82,].
5. Control the movement or quarantine of freshly imported animals for a minimum of three to four weeks before allowing them into the farm during an epidemic [86].
6. Utilization of intravenous fluid delivery to prevent dehydration [87].

Various medicinal drugs can be employed to mitigate the impact of LSD. These medicines comprise antibiotics like "enrofloxacin, oxytetracycline, and penicillin", as well as the non-steroidal anti-inflammatory drug "Meloxicam" and steroidal anti-inflammatory drug "dexamethasone" solution [88, 89, 90].

8. Prevention and Control:

Quarantine measures for specific periods following the import of cattle from infected countries is an effective method of preventing LSD [1, 63]

1. Vector control by using insecticides and traps [91].
2. Sanitary prophylaxis by the restriction on the import of livestock, carcasses, hides, skins etc during the epidemic period [92].
3. Good provisions of drainage and channels for urine discharge [1, 63].
4. Cleaning of cattle shed regularly and trying to make the shed dry [1, 63].
5. Regular monitoring of cattle to detect the symptoms earlier [1, 63].

8.1 Vaccines for LSD:

The World Organisation for Animal Health (WOAH) states that "proper immunisation can prevent LSD by giving cattle strong immunity against LSDV". All cattle breeds, ages, and pregnant animals should be able to use the vaccines without risk, and they should be properly produced and labelled. To contain the epidemic in endemic areas, live attenuated vaccinations of both homologous (vaccinating cattle with a vaccine based on LSDV) and heterologous (vaccinating cattle with a vaccine based on the sheep pox/goat pox virus) have been utilized recently [93]. The WOAH Manual does not include inactivated LSDV vaccines, yet some producers have created them for nations that are prepared to utilise them. In at-risk, disease-free nations, these vaccinations may be chosen as a preventive measure since they provide immunity that lasts shorter [94]. By administering under-attenuated vaccinations to animals that had previously contracted a virulent field strain of LSDV, recombinant LSDV vaccines can be created [95]. The popular "South African Neethling strain and the Kenyan sheep and goat pox (KSGP) O-240 and O-180 strains" compose the attenuated vaccine on the market. The vaccine viruses are derived from 61 serial passages in lamb kidney (LK) cell cultures, 20 passages in the chorioallantoic membrane of chicken embryos, and three additional passages in LK cells [96].

The development of vaccines against lumpy skin disease (LSD) is essential to halting the virus's spread in cattle. One well-known vaccination called the Lumpy Skin Disease vaccination, is made by Onderstepoort Biological Products Ltd. and uses the LSDV "Neethling" strain. It is usually given intramuscularly or subcutaneously. Jordan Bio-Industries Centre (JOVAC) produces a different vaccine that is likewise intended for cattle and is given as a single subcutaneous dosage of 0.5 to 1 mL. The Lumpi-ProVacind vaccine, which is made in India by Indian Immunologicals Limited, likewise employs the LSDV strain and offers immunity after just one injection, lasting roughly a year. To lessen the effects of LSD epidemics, a complete control approach that involves vaccination, movement restrictions, and vector management is implemented. This includes the use of these vaccines [93, 97].

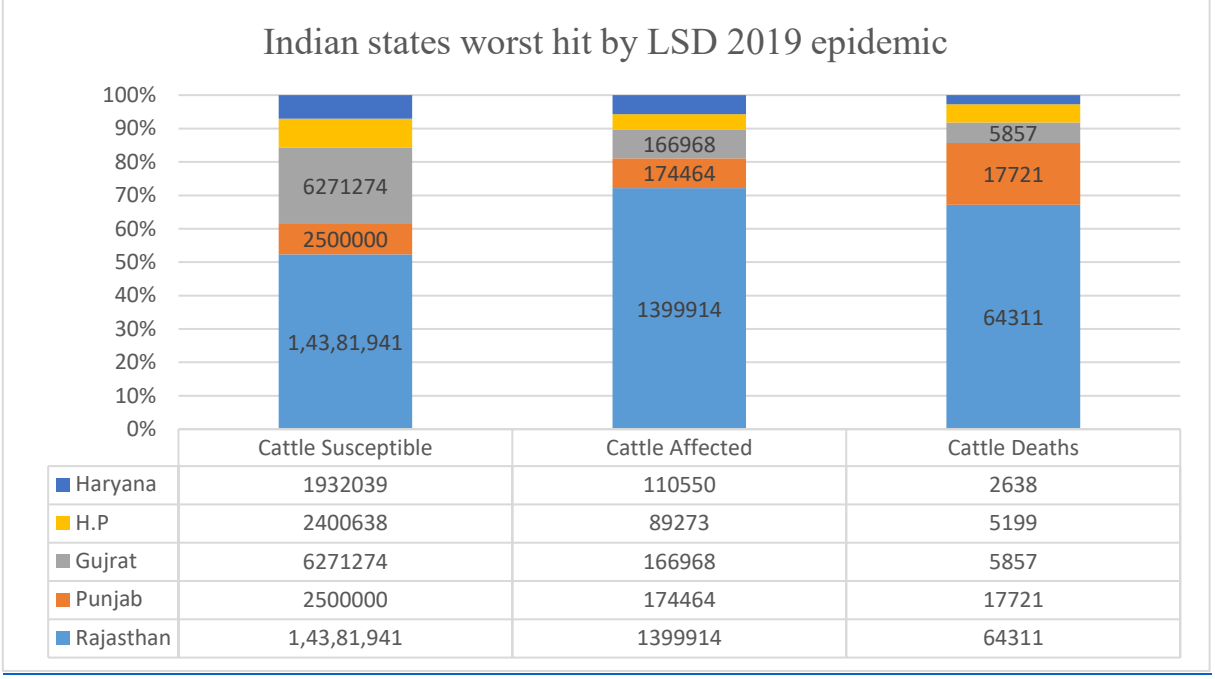
Although vaccinations should be administered in accordance with manufacturer guidelines, effective vaccination campaigns also require adherence to certain standard guidelines. Before entering a property, it is advised that mature cattle, calves from

vaccinated animals, or recently acquired animals receive an annual vaccination [98].

9. Monetary Effects in India's Context:

According to the FAO's risk assessment analysis, the direct losses in livestock and output from LSD might cost South Asia and Asia up to $1.45 billion. India's livestock industry suffered grave losses as a result of a recent LSD outbreak. Starting from September 2022, LSDV had affected over 16 lakh animals in 197 districts. More than 50,000 deaths, mostly in cows, had been reported among the roughly 75,000 cattle that the disease devastated [99].

In India, lumpy skin disease (LSD) has resulted in large financial losses. Over 97,000 cattle died as a result of the 2022 outbreak in just three months, which affected milk output and the incomes of animal keepers. Further compounding economic losses, the condition can result in a temporary decrease in milk production, temporary or permanent sterility in bulls, and skin damage [100].



	Cattle Susceptible	Cattle Affected	Cattle Deaths
Haryana	1932039	110550	2638
H.P	2400638	89273	5199
Gujrat	6271274	166968	5857
Punjab	2500000	174464	17721
Rajasthan	1,43,81,941	1399914	64311

Note: The data in the above figure is obtained from https://indianexpress.com/article/india/lumpy-skin-disease-punjab-haryana-hp-together-see-over-25000-deaths-8172890/.

Fig 7. A statistical overview of Indian states affected by LSD in the 2019 epidemic [97].

[***Source and credits***: Sharma, H. (2022, September 26). Lumpy skin disease: Nearly 1 lakh cattle deaths, toll almost double in three weeks. Retrieved from https://indianexpress.com/article/india/lumpy-skin-disease-punjab-haryana-hp-together-see-over-25000-deaths-8172890/]

There were 20 times as many fatalities from lumpy skin disease in 2022 as there were from India's three most serious cattle diseases in FY 2019–20: foot and mouth disease (4,881 deaths), hemorrhagic septicemia (98 deaths), and anthrax (84 deaths). Only rinderpest, which was eradicated internationally in 2011, has ever caused such devastation in cattle [99].

CONCLUSION

The Neethling poxvirus, a part of the genus Capripoxvirus and family Poxviridae, is associated with lumpy skin disease (LSD), a widespread skin ailment that impacts cattle. Most African countries have LSD, which was first described in Zambia. It is also widely used in the Middle East, Asia, and Europe right now. 2019 saw the discovery of LSD in the Indian state of Odisha. Since then, it has returned numerous times, most recently in 2022. The virus is mostly disseminated by mechanical vectors of arthrophages, and wildlife serves as a reservoir for outbreaks of LSDV, which are frequently connected with the rainy and hot seasons. The host, environment, and pathogen are the disease's three key risk factors. The degree to which the clinical symptoms of LSD may be acute or subacute depends on factors including the breed, age, and sex of the cow. The sickness affects young animals and cows that are heavily nursing more severely. LSD can be recognized using suitable molecular and serological approaches. The illness can have a negative financial impact on global commerce in both live animals and animal byproducts.

LSD can be decreased effectively by vaccination, reducing animal mobility, and the removal of sick and exposed animals. According to the foregoing conclusion, the following suggestions are made [63]:

1. The executive branch should create tactical measures, such as limiting the movement of livestock, implementing a strategic vaccination campaign, and removing infected and infected animals from populations, to effectively control and eradicate the illness.
2. Implementing a quarantine mechanism before adding additional animals to the herd.
3. Common knowledge among the community is that private grazing plots and irrigation sources should be used by herd owners to prevent herd mixing and contact.
4. To create mass vaccination of cattle and the use of rings as the primary means of controlling LSD.
5. Control measurements require an accurate on-time diagnosis.
6. In endemic areas, an annual vaccination program using a parallel strain of the LSDV is required [63].

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