

REVIEW ARTICLE

	<h2>JOURNAL OF PHARMACOLOGY AND BIOMEDICINE</h2> <p>Published by RB Science Home Page: www.jpbiomed.com</p>
-----------------------------------------------------------------------------------	--------------------------------------------------------------------------------------------------------------------------------------------------------

Mirror to review of Lumpy skin Disease

Vikramjit singh^a, Mandeep Kaur^a, Amit Chawla^{a*}, Payal Rani^b, Mangilal^a, Sachin Poonia^a, Mohit^a, Lovepreet^a

a) Maa Saraswati Institute of Pharmaceutical Sciences, Sitto Road, Abohar, Punjab, India, 152116.

b) Associate Professor, Seth GL Bihani SD college of Tech. Edu. Sriganganagar, Rajasthan, India, 335001.

Article History	ABSTRACT
Received on: 23/10/2024	<p>Lumpy Skin Disease is a disorder which is affecting most of the animals and thus is spreading to nation worldwide and is becoming as a epidemic in current times and serves as a platform to turn attention towards its rising disorder. It is a carrier spread disease transmitted by different arthropods. This article provides detailed information about the LSD, LSDV, diagnosis (not by regular blood tests but by the use of special techniques such as fluorescence, antibodies tests, ELISA), treatment of this fatal disease by both pharmacological and non- pharmacological factors, methods to reduce the risk of lumpy skin disease. Still there is no permanent cure of this disease but healthcare professionals are prescribing some medicines to prevent the spread of this life threatening disease. But quarantine of the infected animal is the first aid to be provided in case of absence of medication. Lumpy skin disease is a life threatening infectious and occasionally fatal disease of cattle. Sources of transmission of lumpy skin disease are cutaneous nodules. It is believed to be pandemic in Africa, mainly a disease of cattle with 20% survival rate and 2% chances of case fatality. But in few cases strong antibiotic therapy seems to be useful. Before live attenuated vaccination camps took their full effect, the disease continued spreading from region to region, mainly showing seasonal spread despite applying control and spread measures. Till now, the most likely vectors for LSDV transmission are blood-sucking arthropods such as flies, mosquitoes and ticks. New clinical studies reveal that common house fly, also play a major role in LSDV transmission, but this has not yet been tested in a clinical setting.</p>
Revised on: 05/11/2024	
Accepted on: 06/11/2024	
Published on: 27/12/2024	
<p>Keywords</p> <p><i>Epidemic</i></p> <p><i>Spread</i></p> <p><i>Diagnosis</i></p> <p><i>Pharmacological treatment</i></p> <p><i>Non- Pharmacological treatment</i></p> <p><i>Quarantine of the infected animals</i></p>	

* Corresponding Author
Mr. Amit Chawla
Email: amitchawla84@gmail.com

Scan QR to visit website



JOURNAL OF PHARMACOLOGY AND BIOMEDICINE

ISSN No. 2456-8244

Publication Hosted by
jpbiomed.com

Introduction

LSD is a disease of cattle and water buffalo. It is a carrier spread disease transmitted by different biting and biting due to blood feeding insects. They caused huge loss to the economy due to barrenness, decreased mammary secretion and thus leads to mortality at an average percentage of 30%.^[1,2,3]. All ages of cattle are affected but mainly young ones are trapped.^[3]

This is the only reason why this disease is quite noticeable due to its rapid spread^[4]. One of the mythological cause to be believed by people in this hardest times of LSD is people of villages generally disrespect the cattle on the roads after use by convocating of decreased milk secretion in animals. Now days, the huge spread of the disease worldwide is a matter of great concern which might be due to carrier of capripox virus to India by any means of food products etc.^[5]

LSDV consists of a double-stranded DNA containing around 150 base pairs (kbp) enclosed in a glycoproteinaceous lipid envelope known as capsid. Capripoxvirus, mainly related to the sheep and goat.^[6,7]

It is mainly associated to both sheep and goat pox virus. However, these viruses cannot be differentiated using routine tests. LSD is mainly a disease of cattle. It is a vector-borne disease transmitted by biting of different blood feeding insects (Arthropods).

The clinical signs of LSD depends on the strain of pathogen and the host cattle breed. However, the disease moved outside and spread to the nation worldwide and lead to the major economy loss to the countries and thus ultimately lead to decrement in GDP of that nation. The incubation period is believed

to be 2 to 5 weeks. Fever is the first appear symptom which is seen in the first 2 days of attack of virus. As soon as this symptom is clinically seen then appearance of nodules on the skin and porous sheaths of cell is also observed which demonstrates that animal is infected of LSDV^[4]. Diagnosis is based on the clinically observed symptoms.

As its detection can't be done by the routine regular blood tests but now recently it is done possible by the help of TEM, ELISA, PCR test. Followed by these tests, Southern Blotting, Western Blotting is also emerging as a powerful tool to identify the characteristics of this fatal virus. However, proper and supportive treatment should be given to infected animals to keep away pathogenic symptoms and to control all upcoming consequences. Building up of the immunity of the infected animals is one of the effective methods to control the disease or building up of immunity of animals. and the effective vaccines are produced from the Neethling strain virus

Outer envelope of the virus is brick or oval in morphology containing the genomic material (DNA,RNA). It is believed to be affecting of goats and sheeps but till now, no such cases have been reported. These kinds of viruses are considered to be host specific in nature.

The Causative Organism:

The genus Capripoxvirus of the family Poxviridae is the causative agent of Lumpy skin disease. Lumpy skin disease virus (LSDV) is closely related to sheep and goat poxviruses but till now, no such cases of infection of goats and sheeps are reported.

Viability of LSDV is different at different temperatures. It remains viable at 55°C/2 hours and 65°C/30 minutes. It can be regenerated

or isolated from skin nodules and kept at -80° C for 10 years. Infected cell mass can be kept at 4° - 8° C for 6-10 months. Virus is mainly active t alkaline pH. LSDV is prone to ether (20%), to phenol (2% / 15 minutes), LSDV has homeostasis, have ability to withstand in the environment for long periods at optimum temperature, especially in humid & dried conditions. It may remain alive for 33 days after death of that particular tissue. Due to its long remaining in the environment it spreads to other species rapidly. Moreover, it is prone for sunlight, surfactants with higher lipophilic region and dark, humid & dry conditions. After many researches, researchers have become successful in identifying the genomic sequence of LSDV.

In its genomic material, it contains an interleukin (IL-10), (IL-1), some amt. of GPCR. After too many researches analysts finally reviewed the complete structure of genomic sequence of a typical capripox virus. Molecular structure of genes of sheep pox virus and goat pox virus are also been identified. It also resembles leprosy virus in many ways too.

Along with viral infection in cattle, viral fever, replication of viral DNA, and continuous invasion to the nearby tissues, developments of skin lesions and nodules may also occur. [8]

Experimentally, after dermal injection of the virus, post day infection (DPI) were noticed after fixed interval of days, which day by day became complicated :

- 4 to 7 DPI: localized swelling which were observed as nodules at the site of injection
- 6 to 18 DPI: oozing/squeezing out of the virus via oral and nasal secretion.
- 7 to 19 DPI: development of generalized skin lesions over many areas of skin.

- After 42 days: presence of virus in semen which was observed during copulation or coitus with female cattle or in the genomic sequence of the developing embryo in the cattle, finally leading to the abnormalities in the foetus.[9]

Internal division of the virus in agranulocytes (WBC) and uterine lining leads to swelling of nerves and swelling of lymph nodes in infected mass or group of cells.[9]

It seems that young calves, lactating cows and underweight animals are more at risk to natural infections, mainly due to improper development of immunity.[10] Animals that have recovered from infection by the virus have shown longtime power of immune system. Baby of cows which are born from their infected mothers are resistant to this disease for nearly 6 months because of the acquired maternal antibodies.[11] Genetic transmission from one generation to another are not yet reported but carriers are observed.

Direct and indirect modes of transmission:

Earlier it was revealed that direct transmission is not the way to transmit LSDV, but after clinical studies it was demonstrated that transmission is possible by direct contact but at low rates and efficacy.

This evidence was supported by continuous outbreak of activity of LSDV at different temperatures. But in cold & dry seasons, the no. of arthropods causing this disease are limited to a great extent. By restricting the movement of cattle, its transmission can be reduced to a huge extent.

Moreover, sharing of water sources and the introduction of new animals into a bathing herd also appears to increase the risk of LSD

transmission. Diagnostic methods and tests available at that crucial times were of low efficiency but relatively emerged with time. viuses, main mouthparts of the arthropods are involved but the whole body doesn't get involved. [13]

Carn and Kitching (1995) studied the direct-contact transmission route of LSDV by performing seven separate experiments, where in each experiment one uninfected cow was housed in close contact with two infected animals for a month. No one of thembe in touch with showed early signs and symptoms anddelayed hypersensitivity. Although these early observations are accurate, they are mainly based on the observation of clinical signs.

During their experiment, the early infected animals, were seen with more oftransmitted infection in bothgp. of animals was only seen in 2 of the 7 experiments, and in 1procedure only in 1 of the viral acquired animals, and it is still not known either they had any nodes in their buccal or pulmonary passage membranes or were defaecatingpathogen in their oral discharge or their tracheal discharge.[12]

They finally derived the evidence that direct contact do not have much hand in spread, because no mutual beneficial association was observed between no. of cows in the herd,spread rates, whereas the seen pattern of transmission was demonstrable by indirect transmission, most likely to be spread by arthropods. Non-infected animals were removed out of herd as as possible in order to prevent the spread to other healthy animals. Nasal and oral discharge of infected ones were properly discarded.

Insect transmission:-

Main method of transmission by arthropod vectors has been reported for several viruses. Mainly it spreads by the mosquitoes, fleas and other bloodfeeding insects.

In the cases of insect mode of transmission of

Upto the recent information regarding LSDV, only this method of transmission is to the great concern but few of the research studies implied that along with this method of spreading, this may be also due to some biological factors. Some of the tests (non-engorged *Culicoides punctatus* females were selected) were too carried out nto demonstrate the activity of virus in the host cell DNA of the animal.[14]

The mainway of spread is not linked with 1 of carrier agencies. In hypothesis, any localized carrier species that mainly emphasizes on cows and changing of hosts could carry fatal virus in its buccal cavity. It is complex for LSD that artificially induced infection of cattle requires growth of harmful virus at high sophisticated levels via both IV and ID routes, although, only 65-70% of the animals typically develop a severe pathological disease.[15,11]

Therefore, successful transmission mostly requires several of bites from blood-suckinginsects to transfer onto the virus residing in their unhygienic mouthparts. However, there are no cases on the role of insect saliva and its impact on the attacked cell response against LSDV at the carrier'sfeeding site, which maydecrease the no. of virus required for spread. A general early condition for an insect to act as a major carrier is its availability at high number.[13]

In cases of obstructed feeding before to resida-tion, the feeding carrier needs to find another host, thus, providing anchance to spread to other body parts. It is still to predict whether this mode of transmission is simply achieved by oral route of contamination or more

difficult attractions are required. In malaised affected animals, skin nodes are believed to have high rates of virus [10], providing a useful site of infection for arthropods. For blood sucking insects, such as mosquitoes, which get their nutrition directly from nerves, the level of viral infection in LSD infected cell is quite low, and viral stage lasts for less than 15 days. [11]

On the other backward side, these arthropods cultivate the pathogen directly into the blood which may in turn lead to increment in their infectivity.

Aedes aegypti is believed to fully transmit the virus to those cattle which have less immunity [16]. Bloodfeeding insects such as *Culex* and *Anopheles* were not revealed to spread the pathogen. [16]

Till now, as per the data of WHO *Stomoxys calcitrans* has transmitted the capripox virus to sheep and goats but no cases are revealed yet. [17,18]

In clinical studies, the residues of pathogen was observed in bovine sperms by PCR and virus quarantine procedure. [20,21,1] Also, it led to the spread of the virus to the embryo developing in the uterus of cattle and thus finally leading to the teratogenicity. [20]

History of lumpy skin disease:-

The first case of the clinical signs of LSD was in 1929 in Northern Rhodesia. In the beginning, LSD signs were considered to be caused by poisoning or a hypersensitivity reactions inside a human body and also susceptible to insect bites. Same clinical pathogenesis and symptoms were occurred in, Zimbabwe and the various parts of South Africa between 1943 and 1945, where the infectious nature of the disease was recognized. In South Africa, LSD ap-

proximately affected eight million cattle. The disease continued until 1949, and generated massive economic losses and lots of suffering to people.

Clinical signs :

The clinical signs of Lumpy Skin Disease have two phases, which is appeared after variant incubation period 4-12 days (usually 7 days). The temperature of the infected animals raises to 40-41.5°C, which may alive for 6-72 hr. or more and may rarely be up to 10 days. Trapped ones may show excess tears out of eyes, increased nasal secretion, changes in locomotor activity of animals. The symptoms of LSD are varied in species to species but do not depend on sex or age. The nodules formed are suddenly erupted within 1-2 days. The erupted nodules may cause spread of the pathogen to other animals too. The head, neck and the limbs are the main detection sites. The whole of the skin of the infected animal is covered with lesions infrequent cases. Typical LSD nodules are round, irregular, appear as raised area of skin. The healthy skin is clearly recognized by the adjacent skin reaction. The affected skin oozes out of serum. The nodules may too develop oedema. They slowly harden and form a (dimple) indentation in the centre.

The disease lesions are also developed in the windpipe or trachea which might cause difficulty in breathing. Larynx, trachea, alimentary tract particularly the abomasum of stomach may also develop ulcers that lead to develop severe swelling of gastric mucosa. If trachea and voice boxes are involved then animal may suffer from difficulty in speaking and also respiration related disorders.

After 2-3 weeks, the skin lesions become more harder and harder and ultimately leads to ne-

crisis and thus squeezing out of foul smelling secretions . Some nodules formed may cause Pain, stiffness, sitting discomfort and thus changes in locomotor activity. After eruption of nodules they may leave a full skin thickness hole in the skin, which heals by granulation slowly by time to time which in meanwhile leads to bacterial invasion.. The limbs are swelled to several times their normal size due to inflammation.

Cattle with gestation period are observed to induce abortion as this fatal virus may transfer to developing foetus and thus lead to abnormalities in new born. As a consequence of this disturbances in the normal menstrual cycle were also seen and thus they remained infertile for a long period of time. While in some cases it may lead to complete barrenness of the animal. Infected bulls also faced loss of copulation due to thus life threatening disease. The recovered animals suffered from weakness and sexual disorders within a period of 6 months. LSD is, however, a serious disease affecting production, although the proportion of animals developing chronic complications may be low; less than 5% of those affected.

Diagnosis:

- **Histopathological features :**

Biopsies (Skin Sample) of starting knots are taken and should be preserved in 10-20% formalin. The most diagnostic histopathological features are:

1. Haemorrhage, oedema, nervosis and cell death are always associated with skin lesions.
2. Interaction of eosinophils with cytoplasmic bodies inclusions may be observed in different cells.

- **Isolation of the virus :**

Its diagnosis requires separation as well as identification of pathogen. Samples for virus separation should be collected within the first week of appearance of symptoms.

Testing samples should be extracted from at least 5 animals. Samples aspirated from enlarged lymph nodes can be also used for virus isolation. LSD pathogen develops in nutrient media of bovine side. LSD capripoxvirus have been also made to develop on the chorioallantoic membrane of developing chicken eggs .

- **Fluorescent antibody tests :**

Antigen can also be identified on the infected glass slides or application of smears using phenomenon of fluorescence.

- **Agar gel immune diffusion:**

Agar gel Immune Diffusion Tests has been used for detection of capripoxvirus.

- **Enzyme-linked immunosorbent assay :**

It is formed by using recombinant technology to develop antisera vaccines and to produce somewhat cloning of antibodies (Monoclonal). In this test, Sample containing proteins are used for detection.

Serology :

Freezed sera from both infected and healthy animals are selected. Live attenuation of virus and the various antibody test. ELISA is commonly used against virus neutralized.

1% (w/v) Agarose gel is too used by diffusion. Along with ELISA, western blotting and southern blotting techniques are used for the detection of antibodies to proteins but now commonly used because test is expensive and large scale is required to carry out this test.

Risk factors :

They are directly linked with the morphology of herd, no. of animals visiting the herd, out of which how many are infected and how many are normal and healthy, along with that the thing matters how many of the visiting cattle could be a possible carrier for transmission of virus. Also the introduction of new animals in the herd is a great matter of concern.

Changing of water in the herd regularly, distance to the herd, transfer of diseased animals to the proper care area should be properly done in order to control the transmission of disease throughout that particular area.^[22,14]To be likely, the wind factor plays an important role in contributing to the disease spread.^[16]

Cattle of all ages and varieties of breeds of the cattle, as both the sexes are linked with the disorder.^[3] Atmospheric factors are also involved such as rainfall, mosquitoes prevailing in the environment. But the clinical researches further revealed that :- age and sex of the animal do not play much significant role in the transmission of the disease.^[23]

Role of wildlife in the disease spread:

Along with the infection of cattle and water buffalo with the virus of LSD, some or few of the wild animals are also susceptible to the virus and may spread it to the wildlife also. But these cases are not as easily revealed because notice of skin lesions are difficult and thus late identification of the virus.^[24]

Giraffe, springbok, impala are seen more susceptible to virus.^[25,26] Other species of cattle whose sera has been tested positive for the pathogen include African buffaloes and cows, American cows and buffaloes etc.^[24,29]

At the end, the role of wildlife in the transmission of LSD is not yet known.^[28]

Economic impact :

This life threatening disease has led to the considerable reduction in the milk yield which is a huge loss to the economy. Due to this, growing age children are devoid of many essential micro and macronutrients which are required in their growth and are lots of them are suffering from malnutrition.

Other results of the disease include temporary or permanent barrenness, abortion, death of viral infected animals and thus caused high mortality rate.^[30]

The causative agent, capripoxvirus, can induce sheep and goat as well, and these diseases have economic significance, given that they act as a major barrier to trade and may be recognized as an economic bioterrorism agent.

Treatment : Treatment can be broadly classified into 2 classes:-

1. Non-Pharmacological Treatment
2. Pharmacological Treatment.

1. **Non-Pharmacological Treatment :** It includes treatment of this disease without the use of drugs. They are common household methods to control the spread of the disease. They are quite easy to use, no special physician or RMP is required. They include following common household methods such as:

- Cleaning of place under animals regularly from time to time.
- Keep away from infected animals.
- Regularly wash area under animals
- Use mosquito repellent spray regularly 3 times per day in order to prevent biting of insects to animals.

- Oral and nasal discharge of infected animals must be properly damped off into the earth by pitting in order to minimize the spread of the virus.
- Proper disposal of rejected meal of animals in order to prevent the disease (in case if animal is a carrier and can be susceptible to spread the virus).
- Proper vaccination must be ensured time to time.
- Proper analgesics and antipyretics must be given to animals time to time.

Supportive care:

Supportive care for cattle infected by LSD may involve the use of the following:

- **Wound healing sprays** : These sprays treat skin eruptions that are caused by nodules to prevent infection. These are those products which provide a great skin care that is available in an easy-to-use aerosol container.
- **Antibiotic:** An antibiotic is given along with treatment to prevent infection and side disorders such as pneumonia, which is seen during the treatment of LSD.
- **NSAID's and analgesics** : These are the drugs that mitigate pain, thus encouraging sick cattle to eat the medicine. Villagers used to administer the drug enrolled in a chapatti or a bread slice in order to prevent expulsion of the drug.
- **IV fluids** : They are those formulations which are directly introduced into the blood. But many veterinarians don't recommend the use of this route of administration of drugs because of a lack of practicality and

efficiency.

Although this standard of treatment does not directly point out the disease, it seems to be quite efficient and safe, which is proved from the low mortality rate of cattle and buffaloes (1 to 3%).

If researchers want a particular point of information, an infant calf who is infected with LSDV in India has successfully recovered through a regular change in lifestyle and also in the rug therapy that is already prescribed.

For 21 days, the treatment plan included antibiotic, NSAID's, multivitamins, anti-anaphylactic drugs, topical antibiotic to be rubbed on the skin nodules, mosquito repellent sprays (DDT, BHC).

All those drugs which are prescribed together to cure the calf and bring back to normal health.

Miscellaneous :

They include other methods that are neither pharmacological nor non-pharmacological such as:- Quarantine and Culling. For instance, quarantine is relatively ineffective when transmission through flying insects is possible. The disposal of bodies is the main issue with slaughter.

Conclusion:

From the above article, we concluded that:- It's spread can be overcome by quarantine the affected animal, cleaning the place below animals regularly from time to time, spray by the use of mosquito repellent spray such as DDT, BHC at least 3 times a day. Sometimes the use of wound healing sprays, administration of some antibiotics after a fixed interval of time, providing antipyretics in order to prevent fever etc.

References:

1. Givens, M. D. (2018). Review: Risks of disease transmission through semen in cattle. *Animal*, 12(S1), s165–s171
2. Tuppurainen, E. S. M. ,Alexandrov, T. , & Beltran-Alcrudo, D. (2017). Lumpy skin disease field manual - A manual for veterinarians. *FAO Animal Production and Health Manual*, 20, 1–60
3. Tuppurainen, E. S. , Stoltsz, W. H. , Troskie, M. , Wallace, D. B. , Oura, C. A. , Mellor, P. S. , Coetzer, J. A. , & Venter, E. H. (2011). A potential role for ixodid (Hard) tick vectors in the transmission of lumpy skin disease virus in cattle. *Transboundary and Emerging Diseases*, 58, 93–104
4. Tuppurainen, E. S. M. ,&Oura, C. A. L. (2012). Review: Lumpy skin disease: An emerging threat to Europe, the Middle East and Asia. *Transboundary and Emerging Diseases*, 59, 40–48
5. Sprygin, A. ,Pestova, Y. , Wallace, D. B. , Tuppurainen, E. , &Kononov, A. V. (2019). Transmission of lumpy skin disease virus: A short review. *Virus Research*, 269, 197637
6. Bhanuprakash, V. ,Indrani, B. K. , Hosamani, M. , & Singh, R. K. (2006). The current status of sheep pox disease. *Comparative Immunology, Microbiology and Infectious Diseases*, 29, 27–60
7. Buller, R. M. , Arif, B. M. , Black, D. N. , Dumbell, K. R. , Esposito, J. J. , Lefkowitz, E. J. , McFadden, G. , Moss, B. , Mercer, A. A. , Moyer, R. W. , Skinner, M. A. , &Tripathy, D. N. (2005). Family Poxviridae. In Fauquet C. M., Mayo M. A., Maniloff J., Desselberger U., & Ball L. A. (Eds.), *Virus taxonomy: Classification and nomenclature of viruses. Eighth Report of the International Committee on Taxonomy of Viruses* (pp. 117–133).
8. Constable, P. D. ,Hinchcliff, K. W. , Done, S. H. , &Grundberg, W. (2017). *Veterinary medicine: A textbook of the diseases of cattle, horses, sheep, pigs, and goats* (11th ed., p. 1591).
9. Coetzer, J. A. W. (2004). Lumpy skin disease. In Coetzer J. A. W., & Tustin R. C. (Eds.), *Infectious diseases of livestock*, (2nd ed., pp. 1268–1276).
10. Babiuk, S. , Bowden, T. R. , Boyle, D. B. , Wallace, D. B. , & Kitching, R. P. (2008). Capripoxviruses: An emerging worldwide threat to sheep, goats and cattle. *Transboundary and Emerging Diseases*, 55, 263–272.
11. Tuppurainen, E. S. M. , Venter, E. H. , &Coetzer, J. A. W. (2005). The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques. *Onderstepoort Journal of Veterinary Research*, 72, 153–164.
12. Magori-Cohen, R. ,Louzoun, Y. , Herziger, Y. , Oron, E. , Arazi, A. , Tuppurainen, E. , Shpigel, N. Y. , &Klement, E. (2012). Mathematical modelling and evaluation of the different routes of transmission of lumpy skin disease virus. *Veterinary Research*, 43.
13. Kahana-Sutin, E. ,Klement, E. , Lensky, I. , & Gottlieb, Y. (2017). High relative abundance of the stable fly *Stomoxys calcitrans* is associated with lumpy skin disease outbreaks in Israeli dairy farms. *Medical and Veterinary Entomology*, 31, 150–160.
14. 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), 1268–1279.
15. Carn, V. M. ,& Kitching, R. P. (1995). An investigation of possible routes of transmission of lumpy skin disease virus (Neethling). *Epidemiology and Infection*, 114, 219–226.
16. Chihota, C. M. , Rennie, L. F. , Kitching, R. P. , & Mellor, P. S. (2003). Attempted mechanical transmission of lumpy skin disease

- virus by biting insects. *Medical and Veterinary Entomology*, 17, 294–300.
17. Baldacchino, F. ,Muenworn, V. , Desquesnes, M. , Desoli, F. , Charoen viriyaphap, T. , &Duvallet, G. (2013). Transmission of pathogens by *Stomoxys* flies (Diptera, Muscidae): A review. *Parasite*, 20, 26.
 18. Yeruham, I. , Nir, O. , Braverman, Y. , Davidson, M. , Grinstein, H. , Hay movitch, M. , & Zamir, O. (1995). Spread of lumpy skin disease in Israeli dairy herds. *Veterinary Record*, 137, 91–93.
 19. Annandale, C. H. , Irons, P. C. , Bagla, V. P. , Osuagwuh, U. I. , & Venter, E. H. (2010). Sites of persistence of lumpy skin disease virus in the genital tract of experimentally infected bulls. *Reproduction in Domestic Animals*, 45, 250–255.
 20. Annandale, C. H. , Holm, D. E. , Eber sohn, K. , & Venter, E. H. (2014). Seminal transmission of lumpy skin disease virus in heifers. *Transboundary and Emerging Diseases*, 61, 443–448.
 21. Irons, P. C. ,Tuppurainen, E. S. , & Venter, E. H. (2005). Excretion of lumpy skin disease virus in bull semen. *Theriogenology*, 63, 1290–1297.
 22. Gari, G. ,Waret-Szkuta, A. , Grosbois, V. , Jacquiet, P. , & Roger, F. (2010). Risk factors associated with observed clinical lumpy skin disease in Ethiopia. *Epidemiology and Infection*, 138, 1657–1666.
 23. Ochwo, S. ,VanderWaal, K. , Munsey, A. , Nkamwesiga, J. , Ndekezi, C. , Auma, E. , &Mwiine, F. N. (2019). Seroprevalence and risk factors for lumpy skin disease virus seropositive ity in cattle in Uganda. *BMC Veterinary Research*, 15, 236.
 24. Barnard, B. J. H. (1997). Antibodies against some viruses of domestic animals in South African wild animals. *Onderstepoort Journal of Veterinary Research*, 64, 95–110.
 25. Lamien, C. E. ,Lelenta, M. , Goger, W. , Silber, R. , Tuppurainen, E. , Matijevic, M. , Luckins, A. G. , & Diallo, A. (2011). Real time PCR method for simultaneous detection, quantitation and differentiation of capripoxviruses. *Journal of Virological Methods*, 171, 134–140.
 26. Le Goff, C. , Lamien, C. E. , Fakhfakh, E. , Chadeyras, A. , Aba-Adulugba, E. , Libeau, G. , Tuppurainen, E. , Wallace, D. B. , Adam, T. , Silber, R. , Gulyaz, V. , Madani, H. , Caufour, P. , Hammami, S. , Diallo, A. , & Albina, E. (2009). Capripoxvirus G-protein-coupled chemokine receptor: A host-range gene suitable for virus animal origin discrimination. *Journal of General Virology*, 90, 1967–1977.
 27. Davies, F. G. (1982). Observations on the epidemiology of lumpy skin disease in Kenya. *Journal of Hygiene (London)*, 88, 95–102.
 28. Tuppurainen, E. S. , Venter, E. H. , Shisler, J. L. , Gari, G. , Mekonnen, G. A. , Juleff, N. , Lyons, N. A. , De Clercq, K. , Upton, C. , Bowden, T. R. , Babiuk, S. , &Babiuk, L. A. (2017). Review: Capripoxvirus diseases: Current status and opportunities for control. *Transboundary and Emerging Diseases*, 64(3), 729–745.
 29. Fagbo, S. ,Coetzer, J. A. W. , & Venter, E. H. (2014). Seroprevalence of Rift Valley fever and lumpy skin disease in African buffalo (*Syncerus caffer*) in the Kruger National Park and HluhluweMfolozi Park, South Africa. *Journal of the South African Veterinary Association*, 85, 1075.
 30. Alemayehu, G. ,Zewde, G. , &Admassu, B. (2013). Risk assessments of lumpy skin diseases in Borena bull market chain and its implication for livelihoods and international trade. *Tropical Animal Health and Production*, 45, 1153–1159.

Cite this article as

Chawala A, Singh V, Kaur M, Rani P, Mangilala, Poonia S, Mohit, Lovepreet Mirror to review of Lumpy skin Disease. *J Pharmacol Biomed.* 2025; 9(1): 748-757