WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

A Review on Lumpy Skin Disease

[1]Monika Barkusing Rajput, [2]Monika Daulat Vinchu, [3]Tejas Dilip Wadje, [4]Pravin Bhausaheb Kadam

Department of Pharmacy, S. N. D. College of Pharmacy, Nashik

[1] monikajput0345@gmail.com, [2]monikavinchu@gmail.com, [3]wadjetejas@gmail.com, [4]pravinkadam610@gmail.com

ABSTRACT-

The lumpy skin disease is double standard DNA virus , an major infection transboundry affecting cattle and buffalo of all breeds and age groups is lumpy skin disease. lumpy skin disease major affect on the economic worth of animal . for lumpy skin disease two methods are used for dignosis which are the presumptive diagnosis and comfirmative daignosis . Their are various species which causes the lumpy skin disease on live stock .Lumpy skin disease belongs to the genus capripoxvirus within the family poxviridae . in the lumpy skin disease the transmition of virus may occurs by insect, vector and nonvector . the virus can be enter in body through skin or gastrointestinal mucosa .High fever is initial symptoms of lumpy skin disease .Lumpy skin disease can not be treted by antiviral medication but it can be treat by vaccination which is given to prevent the disease .

keywords - lumpy skin disease and its diagnosis methods

INTRODUCTION-

lumpy skin disease (LSD**) is** an major infectious transboundary infection affecting cattle and buffalo of all breeds and age groups is.[8] It is One of the main health issues affecting the cattle industry in the majority of developing nations, It mostly affects cattle and is brought on by the lumpy skin disease virus, of which the Neethling strain serves as the prototype and is mechanically transferred by insect vectors.[9] Exanthema nodularis bovis, LSD, pseudo-urticaria, Neethling virus illness, and knopvelsiekte are all names for the infectious condition lumpy skin disease. It is brought on by a virus (LSDV) from the genus Capripoxvirus in the family Poxviridae. Antigenically, it is very similar to the sheep and goat pox virus.[10] There are multiple antigenically diverse forms of LSDV, including non-enveloped, enveloped, intracellular mature viruses, cell-associated enveloped viruses, extracellular enveloped viruses.[11] The condition is distinguished by confined, firm skin nodules that cover the entire body, lesions in the mouth, pharynx, and respiratory system, pyrexia, and enlarge lymph nodes. [7]Overall, it affects the cattle sector because it causes secondary bacterial infections and it lowers the economic worth of animals since it reduces their ability to produce meat and milk, high-quality hides, draught strength, and reproductive efficiency (abortion and infertility).[4]

History -

In 1929, North Rhodesia (Zambia) received a report of lumpy skin disease (LSD), which at the time was reportedly attributed to an allergic reaction in cattle brought on by biting insects.[8] The disease was first discovered in Europe in Cyprus in 2014, Greece in 2015, and the Balkan countries in 2016, with the initial outbreaks occurring in Egypt in 1988.Lumpy skin disease, which severely limits cattle productivity and is rapidly expanding throughout the Middle East, was only found in Africa until 1989 . Additionally, due to thedisease's expansion outside of its typical range, countries in Europe and Asia are now at risk from it.[7] In a succession of epizootics, the disease has continued to spread across the majority of the African continent, as previously noted by Davies (1991 b) and House (1990). Senegal,The diseasMozambique, and Mauritius all reported LSD use in 2001.[5]the disease was only present in countries on the Sub-Saharan African continent until the 1980s (from 1929 to 1984), yet it has been predicted that it may spread outside of this region. LSD epidemics were recorded in the Middle East in Oman in 1984 and 2009, Kuwait in 1986 and 1991, Egypt in 1988 and 2006, Israel in 1989 and 2006, Bahrain in 1993 and 2002-2003, United Arab Emirates in 2000.[5] Central Ethiopia experienced one of the continent's LSD outbreaks from 2007 to 2011.in Adama,Wenji, Mojo, and Welenchiti, four districts. In total, 1,675 outbreaks with 62,176 cases and 4,372 fatalities were documented over a 5-year period from 2007 to 2011.[5]The disease was confined to larger Africa until 1988 before gradually spreading to the MiddleEast, Eastern Europe, and ultimately the Russian Federation. After thereafter, the pandemic continued to spread, and in 2019 fresh cases in South and East Asia were recorded The first event was reported on July 14, 2019, making Bangladesh the first hotspot in South Asia, according to an OIE report. The disease then extended to further regions of China, India, Nepal, and Bhutan in 2020.fatalities were documented over a 5-year period from 2007 to 2011.The disease was confined to larger Africa until 1988 before gradually spreading to the Middle East, Eastern Europe, and ultimately the Russian Federation. After thereafter, the pandemic continued to spread, and in 2019 fresh cases in South and East Asia were recorded The first event was reported on July 14, 2019, making

Bangladesh the first hotspot in South Asia, according to an OIE report.[8] The disease then extended to further regions of China, India, Nepal, and Bhutan in 2020.[12]

Etiology-

LSD virus (LSDV) belongs to the genus capripoxvirus within the family Poxviridae. LSDV genome is ~151 kbp in length. Two other capripoxvirus-s, sheepox virus (SPV) and goatpox virus (GPV) which cause devastating disease in sheep and goats respectively, are also antigenically similar to LSDV.[1] All 3 members of the genus Capripoxvirus are antigenically similar, sharing a common precipitating antigen, which permits the use of heterologous virus for protection.[2]The virus has a double-stranded DNA genome of about 151 kbp. It is enveloped, linear, ovoid shaped virion measuring 220-450 nanometer (nm) by 140-266nm LSDV is a brick shaped enveloped virus, 320 × 260 nm size LSDV contains homologues genes such as interleukin-10 (IL-10), IL-1 binding proteins, G protein-coupled CC chemokine receptor (GPCR), and epidermal growth factor-like protein which are found in other poxvirus genera .[4] it is surviving in necrotic skin nodules for up to 33 days or longer, desiccated crusts for up to 35 days and at least 18 days in air-dried hides. It can remain viable for long periods. LSDV genes share a high degree of colinearity and amino acid identity (average of 65%) of its genomic region with genes of other known mammalian poxviruses, particularly suipoxvirus, yatapoxvirus, and leporipoxviruses.[5]

Virus is recoverable for at least 18 days from air-dried hides kept at room temperature and from infected tissue culture fluid stored at 4 °C for six months.120 The virus was reported to persist in necrotic skin nodules for up to 33 days but this period may be much longer in the environment.[6] LSDV can persist in skin plugs for about 42 days. It is likely that the viral type inclusion body protein in infected cells may protect the vision after the scab has disintegrated, although not yet proven.[7]

Transmission-

1.Direct and indirect modes of transmission, non-vectored

2. Insect transmission

3. Tick transmission

Lumpy skin disease virus (LSDV) is a contagious, infectious, vector-borne viral disease which primarily affects cattles (Bos Taurus, Bos indicus and Asian water buffalo). Arthropods, are mechanical vector, the main means of spreading the LSD virus. Iatrogenic transmission of LSDV can also occur. Other routes of spread are through artificial insemination, direct or indirect contact, and other means. LSDV can be mechanically transmitted by a variety of flying and non-flying blood-feeding insects, and they play a significant part in transmission. [29]

Vector Transmission- The role of arthropod vectors in virus transmission has been experimentally confirmed. Various arthropods can transmit LSDV, although they do not replicate in vectors. Transmission is therefore mechanical and not biological in nature.[29]

The most commonly suspected vector species for the spread of Lumpy skin disease is the common stable fly (S. calcitrans). (Weiss, 1968; Kitching and Mellor, 1986; Kahana-Sutin et al., 2017; Yeruham et al., 1995; Davies, 1991)[29]

Potential vectors identified thus far:

Mosquitos: Aedes aegypti, Anopheles stephensi, Culex quinquefasciatus

Flies: Stomoxys calcitrans, Haematobia irritans, Prostomoxys sp., Haematopota spp., Biomyia fasciata

Midges: Culicoides nubeculosus

Ticks: Rhipicephalus appendiculatus, Rhipicephalus decoloratus, Amblyomma hebraeum [31]

Non-vector transmission through Direct and indirect modes - There is very little evidence that LSDV can be transferred by direct contact. Generally, Direct contact has been proved to be an ineffective pathway for LSDV transfer, but real experimental evidence is minimal. Early experimental research and on-the-ground observations in South Africa led to the conclusion that LSDV transmission via direct contact undoubtedly happens, albeit at low rates and efficiency. (Diesel, 1949; Weiss, 1968).[29]LSDV has been isolated from infected semen in experiments, however transmission of LSDV through semen (natural mating or artificial insemination) has not been scientifically demonstrated. [30]

Transmission without a vector Despite being ineffectual, Non-vectored LSDV transmission occurs when clinically affected animals come into touch with contaminated materials, with no biological or mechanical vectors required. Saliva, nasal, and ocular discharges contain infectious LSDV that spreads disease by contaminating communal eating and drinking areas. [14]

Pathogenesis-

Lumpy Skin Disease Virus (LSDV) enters in the host body through the skin or gastrointestinal tract mucosa. LSD triggers an immediate burst of many confined cutaneous nodules, which is accompanied by a feverish reaction.[14] After subcutaneous or intradermal inoculation of cattle with LSDV, a localized swelling and expansion of the regional lymph nodes appear four to seven days later. [5]

High fever is the initial clinical symptom detected in cattle after incubation, followed by Localized swelling and the formation of inflammatory nodules at the site of inoculation. These skin nodules are circumscribed, hard, circular, and elevated, and they involve the skin, subcutaneous tissue, and on rare instances, even the underlying muscles. Following subcutaneous or intradermal LSDV inoculation of calves, localized swelling at the site of inoculation developed 4 to 7 DPI (DOT PER INCH) ranging in size from 1 to 3 cm and covered up to 25% of the skin surface. Enlargement of the regional lymph nodes and generalized eruption of skin nodules usually follows 7 to 19 DPI. Following a febrile reaction, viremia and low amounts of viral shedding in oral and nasal secretions were identified between 6 and 15 and 12 and 18 DPI, respectively. Additionally, evidence of LSDV is found in saliva, semen, and skin nodules until at least 11, 42, and 39 days following the onset of a fever, respectively. [28]

Diagnosis -

Based on the presumptive diagnosis and confirmative diagnosis, Lumpy skin disease (LSD) can be diagnosed. Preclinical indicators, clinical history, symptoms infected animals, and the frequency of disease outbreak in neighbouring locations can all be used to make a presumptive diagnosis of LSD.[14]

The most diagnostic histopathological findings seen during presumptive diagnosis include congestion, persistent fever, emaciation, low mortality, lymphoid growth, lachrymation, necrotic pox lesion around the muzzle, and skin lesion in the legs.[16]

When a nodular skin lesion first appears, a laboratory test for the virus is used to confirm the diagnosis. Laboratory tests such as the Enzyme-Linked Immunosorbent Assay (ELISA), Indirect Fluorescent Antibody test (IFAT), Indirect Immunofluorescence test, Virus Neutralization Test (VNT), Serum Neutralization Test (SNT), Electron Microscopy, Agar Gel Immunodiffusion Test (AGIDT), and Western Blotting can all be used to detect the disease.[14]

The most traditional and often used confirmatory test for the diagnosis of Lumpy Skin Disease (LSD), which is specifically brought on by Capripoxvirus, is Polymerase Chain Reaction (PCR). Utilizing the RT-PCR technology, the DNA of the Lumpy skin disease virus (LSDV) can be quickly detected. Capripoxviral antigen and antibody can be found using the Virus Neutralization Test (VNT) and electron microscopy examination. In compared to the Indirect Fluorescent Antibody Test (IFAT) and the Virus Neutralization Test (VNT), the Enzyme-Linked Immunosorbent Assay (ELISA) Test is more accurate and sensitive.[14]

Table 1- Diagnosis Method - [21]

A Field Presumptive Diagnosis	**Confirmative diagnosis**
1) Pox knob of mucuous membrane of tongue ,oronasal, pharynx,trachea. **2) Pleuritis** **3) Necrosis of skin disease** **4) Enlargement of lymph nodes** **5) Synovitis and tendosynovitis with fibrin in the synovial fluid .** **6) pox lesion in urinary bladder.** **7) Intracytoplasmic eosinophilic inclusion** **8) Perivascular fibroplasias and cellular Infiltrates** **9)Contagious disease with generalised skin nodules** **10).Oedema and areas of focal lobular atelectasis in lungs** **11) Vasculitis, thrombosis, infarction**	**1.Enzyme-linked Immunosorbent Assay** (detection of antibodies against capripoxvirus using P32 monospecific polyclonal antiserum protein.) **2. Indirect Fluorescent Antibody test (IFAT)** (cross reaction with parapoxviruses) **3. Western Blotting** (sensitive and specific system for detection of antibody and structural proteins to capripoxvirus) **4. Virus Neutralization Test (VNT)** (cross reaction with capripoxviruses) **5 Agar Gel Immunodiffusion Test (AGIDT)** (cross reaction with Bovine popular stomatitis and pseudocowpox virus). **6. Electron Microscopy** (demonstration of virus in negatively stained preparation of biopsy specimen taken from affected skin or mucus membrane)

Differential Diagnosis- [10],[14],[20]

To prevent confusion, Lumpy Skin Disease (LSD) must be distinguished From other skin conditions that are similar.

LSD and certain illnesses, including

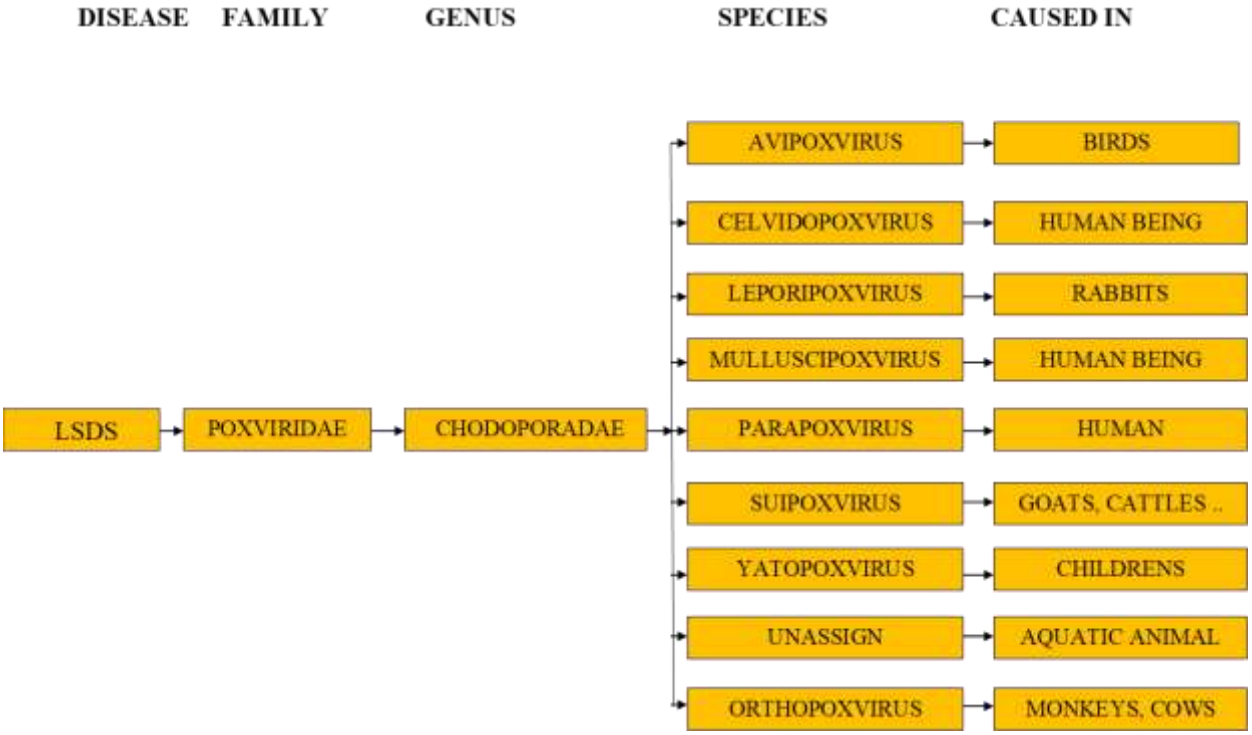
- **Pseudo Lumpy Skin Disease**
- **Pseudo Cow Pox**

- **Bovine virus diarrhoea**
- **Insect bites**
- **Urticaria**
- **Photosensitization**
- **Dermatophilosis**
- **Demodicosis (Demodex) of skin or foot or mouth**
- **Besnoitiosis**
- **Snotsiekte**
- **Oncocercariasis**
- **Rinder pest.**

Immunity-

The attenuated south African vaccine strain has been shown to protect against clinical disease but experiences during the outheaks in 1990 have challenged the assertion that immunity to LSD is life long. in natural infection, very young calves, lacting cows, and malnourished animal seem to develop more severe disease that may be due to an impaired humoral immunity. a lifelong cell mediated immunity is developed in most animal that recover from clinical disease. Animal will develop protective immunity from 10 to 21 days post vaccination and then required an annual booster dose.In natural,very young calves,lactating cows and malm ourished animal seem to develop more sever disease that may be due to an impaired humoral immunity. immunity after recovery from natural infection is life long; calves of immune cows acquire matuternal antibody and are resistant to clinical disease for about six months. The host immunity against LSDV is mainly cell mediated and therefore, serogical testing may no be sensitive enough to detect mild and long standing infection or antibody in vaccinated animal. [2],[17],[26],[27],[28]

Classification of Lumpy Disease Virus- [10],[14],[16]



Preventional treatment -

Unfortunately, lumpy skin condition cannot be treated with any specific antiviral medications. Supportive care for cattle is the sole treatment available. This can involve utilising wound care sprays to treat skin lesions and antibiotics to stop secondary skin infections and pneumonia. Animals suffering from pain can be given anti-inflammatory medications to maintain their appetite. Although intravenous fluid delivery might be advantageous,

it might not be practical in the field. The lack of treatment options for lumpy skin disease virus emphasises the need of using effective vaccination for preventing disease.[18]

The techniques for LSD prevention and control are similar to those used for the majority of viral infections. These can be handled through vaccination, hygienic measures, vector control, and restrictions on the movement of infected animals. Effective control and prevention measures must be implemented in order to control the disease.[16]

These preventional treatment and control strategies primarily include:

Restrict the movement of infected animals with LSD: Animals that are infected should not be transported at all to stop the spread of transboundary disease. To stop the rapid spread of disease inside a country, animals with such lesions should be isolated for inspection.[16]

Restrict vector movements: LSD thought to be transmitted primarily by blood-feeding insects; hence quarantine and movement restriction alone are not very effective to control LSD unless supported by mass vaccination. Therefore, efficient insect control may reduce the rate of LSDV transmission. Vectors movement due to prevailing winds may cause disease transmission. Vector control methods like use of vector traps, insect repellents and providing insect proof housing for animals, use of insecticides can also be used for preventing the disease.[19]

Vaccination: For LSD, a live attenuated vaccination is available. in endemic regions, Live vaccines help control losses from lumpy skin disease. Companies created vaccines based on various strains of the LSD virus. Since LSD is closely related to the virus that causes sheeppox and goatpox, the vaccine for both diseases can also be used to prevent lumpy skin disease.[16]

Economic Importance-

Even though the mortality rate caused by LSD is relatively low, the disease is of significant economic significance attributed to production losses.[15] Considering that the disease's mortality rate is often low, its high morbidity has a greater economic impact than its low mortality rate. Significant losses result from restricted body growth, damaged hides, male and female infertility, mastitis, decreased milk production, and abortions.[16] The overall trade of live animals and animal products is affected by the decrease in animal welfare. The meat industry, dairy industry, leather, and other businesses reliant on cattle and its byproducts may suffer significant financial losses as a result.[16] Direct losses to the government come from vaccination programmes as well as control measures to manage trade restrictions, vector control, disease surveillance programmes, public awareness campaigns, etc.[24]

LSD is one of the trans-boundary diseases extending beyond its traditional boundaries. Therefore, limiting the movement of live animals and animal products can have a big impact on international trade,[16][24].

The cost of treatment of LSD for native zebu in Ethiopia was estimated to be $6.43 USD per head and $58 USD for Holstein Friesian. The cost of supportive antibiotic treatment for an outbreak in Jordan was estimated to be 27.9 British pounds per person.[16] Due to introduction of LSD in 2019, studies on the economic impact and production losses caused by lumpy skin disease in Bangladesh, China, and India have not been conducted. A projected 1.45 billion USD in direct losses of livestock and production have been estimated. LSD's introduction in 2019 could have a significant impact on the livestock trade in Asian nations. According to a 2017 estimate, exports of live cattle, buffalo meat, meat products, dairy products, and covers totalled USD 5.5 billion to Asian countries (Roche et al. 2020). APEDA (Agricultural and Processed Food Products Export Development Authority) data shows that India’s alone 3,694.29 USD million of which 3175.09 USD million was buffalo meat.[24]

Reference-

1. Isolation and characterization of lumpy skin disease virus from cattle in India, Naveen kumar, 2011

2.Lumpy skin disease in southern Africa: a review of the disease and aspects of control, P Huntera and D Wallaceb.

3. LUMPY SKIN DISEASE: AN EMERGING BOVINE VIRAL INFECTION IN INDIA , N. Ahmed , 2020

4. A review: Lumpy skin disease and its emergence in India, Tania Gupta, 2020

5. Lumpy Skin disease: Review of literature, K. A. Al-Salihi , 2014

6. Lumpy skin disease,J A W COETZER

7. A Review on: Lumpy Skin Disease: Enhance Awareness on the Epidemiological situation and Diagnosis; Prevention and Control Measures in Ethiopia, Girma Zewdie, 2021

8.Lumpy Skin Disease: An Emerging Bovine Viral Infection in India (Nekibuddin Ahmed, Sharmita Doley, Anjan Jyoti Nath, Sayod Ahmed Barlaskar)

9.A Case Report on Clinical Management of Lumpy Skin Disease in Bull (Abdi Fufa Feyisa)

10. Lumpy Skin disease: Review of literature (K. A. Al-Salihi) A review: Lumpy skin disease and its emergence in India (Tania Gupta & Vanita Patial & Diksha Bali & Shivani Angaria & Mandeep Sharma & Rajesh Chahota)

11. Outbreak investigation and molecular diagnosis of Lumpy skin disease among livestock in Saudi Arabia 2016(S. Kasem M. Saleh, Qasim, O. Hashim, A. Alkarar, Abu-Obeida,A, Gaafer, R. Hussien, A. AL-Sahaf, A. Al-Doweriej. F. Bayoumi, A. Hodhood, M. Abdelatif.

12. A review: Surveillance of lumpy skin disease (LSD) a growing problem in Asia (Yasir Razzaq Khan , Ahmad Ali , Kashif Hussain , Muhammad Ijaz , Ameer Hamza Rabbani ,Rabia Liaquat Khan , Syed Nazar , Muhammad Umair Aziz , Awais Ghaffar ,Hina Afzal Sajid)

13. A Review on: Lumpy Skin Disease: Enhance Awareness on the Epidemiological situation and Diagnosis; Prevention and Control Measures in Ethiopia(Zewdie G)

14 An updated review on lumpy skin disease: perspective of Southeast Asian countries : (Moumita Das, Md. Shahidur Rahman Chowdhury, Sharmin Akter, Apurbo Kumar Mondal, Md Jama, Uddin, Md. Masudur Rahman , Md Mahfujur Rahman,)

15 Hunter P, Wallace D Lumpy skin disease in southern Africa: a review of the disease and aspects of control

16. A review: Lumpy skin disease and its emergence in India Tania Gupta

17. PANKAJ KUMAR, Emergence and transboundary spread of lumpy skin disease in South Asia

18. Babiuk, S. (2018). Treatment of Lumpy Skin Disease. In: Lumpy Skin Disease. Springer, Cham. https://doi.org/10.1007/978-3-319-92411-3_17

19. A Review on: Lumpy Skin Disease: Zewdie G

20. A review: Lumpy skin disease and its emergence in India: (Tania Gupta , Vanita Patial, Diksha Bali, Shivani Angaria, Mandeep Sharma, Rajesh Chahota.)

21. Lumpy Skin disease: Review of literature(K. A. Al-Salihi ,BSC, MSC, Ph.D in Veterinary Medicine and Pathology / Faculty of Veterinary Medicine /The University of Nottingham / UK.)

22. Lumpy skin disease in southern Africa: a review the disease and aspect of control P Huntera and D Wallace

23. Emergnce and transboundary spread of lumpy skin disease in south asia.

24. PANKAJ KUMARI,RASHMI REKHA KUMARIZ,SARITA DEVI3,MANOJ KUMARI TRIPATHI1,JASPRET SINGH1,RAVI KUMAR2,AND MANISH KUMAR4.

25.REVIEW: Lumpy skin disease :endula mulatu1* and abdi feyisa2 .

26. Comparative studies o lumpy skin disease virus in human:samia Ahmed kamal*

27. Lumpy Skin disease: Review of literature (K. A. Al-Salihi)

28. Review: Lumpy Skin Disease (Endalu Mulatu, Abdi Feyisa)

29. Transmission of lumpy skin disease virus: A short review (Sprygina, , Ya Pestovaa , D.B. Wallaceb,c , E. Tuppurainena,b,c , A.V. Kononov)

30. Excretion of lumpy skin disease virus in bull semen (P.C. Ironsa, E.S.M. Tuppurainenb , E.H. Venterb)

31. Introduction and spread of lumpy skin disease in South, East and Southeast Asia (Authors : Xavier Roche, Andriy Rozstalnyy, Damian TagoPacheco, Akiko Kamata, Claudia Pittiglio, Daniel Beltran Alcrudo, Khadak Bisht, Surendra Karki, Jessica Kayamori, Fairouz Larfaoui, Eran Raizman, Sophie VonDobschuetz, Madhur S Dhingra, Keith Sumption)

32. Abutarbush SM, Ababneh MM, Al Zoubi IG, Al Sheyab OM, Al Zoubi MG, Alekish MO, Al Gharabat RJ. (2013). Lumpy Skin Disease in Jordan: Disease Emergence, Clinical Signs, Complications and Preliminary-associated Economic Losses. Transbound Emerg Dis. Oct 21. doi: 10.1111/tbed.12177.

33. Alexander RA, Plowright W and Haig DA. (1957). Cytopathogenic agents associated with lumpy-skin disease of cattle. Bull. Epiz. Dis. Afr. 5:489-492.

34. Ali Meawad Ahmed and Amina A. Dessouki. (2013). Abattoir-Based Survey and Histopathological Findings of Lumpy Skin Disease in Cattle at Ismailia Abattoir. International Journal of Bioscience, Biochemistry and Bioinformatics. 3(4): 372-375.

35. Ali BH and Obeid HM. (1977). Investigation of the first outbreak of Lumpy skin disease in the Sudan. Brit. Vet. J., 1333:184-189.

36. Ali AA, Esmat M, Attia H, Selim A, Abdel-Humid YM. (1990). Clinical and pathological studies on lumpy skin disease in Egypt. Veterinary Record, 127, 549–550.

37. Anonymous D. (1988). Lumpy skin disease.Vol.1.No.l, Paris: O.I.E. Disease Information.

38. Ayre-Smith RA. (1960). The symptoms and clinical diagnosis of lumpy skin disease in Egypt. Vet. Rec., 127:549-550.ANONYMOUS. 1988. Lumpy skin disease. Vol. 1. No. l, Paris:O.I.E. Disease Information .

39. Ayelet G , Haftu R, Jemberie S, Belay A, Gelaye E, Sibhat B, Skjerve E and Asmare K. (2014). Lumpy skin disease in cattle in central Ethiopia: outbreak investigation and isolation and molecular detection of lumpy skin disease virus Rev. sci. tech. Off. int. Epiz. 33 (3), 1-23.

40. APHIS Veterinary Services Centers for Epidemiology and Animal Health. (2006). Lumpy Skin Disease, Israel. (Impact Worksheet). http://www.aphis.usda.gov/vs/ceah/cei/.

41. Balinsky CA, Delhon G, Smoliga G, Prarat M, French RA, Geary SJ, Rock DL, Rodriguez LL. (2008). Rapid preclinical detection of sheeppox virus by a real-time PCR assay. Journal of Clinical Microbiology, 46(2):438-442