IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

LUMPY SKIN DISEASE: REVIEW OF LITERATURE.

GUIDED BY –Prof. SWAPNIL SUDAM HARISHCHANDRE
PANKAJ ASHOK TAGAD, SHUBHAM PRAKASH PATIL, NILESH TUKARAM PARAKHE, SUSHMA VISHNU DATIR, VAISHNAVI GODHAJI SAPTE

DR.KOLPE INSTITUTE OF PHARMACY, KOLPEWADI, KOPARGAON, A.NAGAR MAHARASHTRA INDIA.

ABSTRACT:

LSD is an important Tran's boundary disease affecting the cattle industry worldwide. The Objectives of this study were to determine trends and significant change points, and to forecast the Number of LSD outbreak reports in Africa, Europe, and Asia. LSD outbreak report data (January 2005 To January 2022) from the World Organization for Animal Health were analysed. We determined statistically significant change points in the data using binary segmentation, and forecast the number Of LSD reports using auto-regressive moving average (ARIMA) and neural network autoregressive (NNAR) models. Four significant change points were identified for each continent. The year between the third and fourth change points (2016–2019) in the African data was the period with the highest Mean of number of LSD reports. All change points of LSD outbreaks in Europe corresponded with massive outbreaks during 2015–2017. Asia had the highest number of LSD reports in 2019 after the third detected change point in 2018. For the next three years (2022–2024), both ARIMA and NNAR forecast a rise in the number of LSD reports in Africa and a steady number in Europe. However, ARIMA predicts a stable number of outbreaks in Asia, whereas NNAR predicts an increase in 2023–2024. This study provides information that contributes to a better understanding of the Epidemiology of LSD.

KEYWORD: Classification of LSD, Biology of LSDV, Pathogenesis, Histopathological Findings, Diagnosis, Treatment.

INTRODUCTION:

Lumpy Skin Disease (LSD) is an infectious disease in Cattle caused by Lumpy Skin Disease Virus (LSDV) under the family Po-xviridae. Currently the disease has been emerged as a devastating threat for the large Domesticated ruminants in Asia, Europe and the Middle East [1]. The disease is enlisted by the OIE due To its capacity for fast trans-boundary spread [2, 3]. In endemic areas, LSD is a re-emerging transmissible Infection that results significant socio-

economic Impairment to small-scale and courtyard agrarians [4]. Considering the disease burden, morbidity and Mortality cattle are found as more sensitive to the Illness compared to buffalos and other ruminants [5]. Despite the practice of mixed herd farming in many Countries consisting of cattle, sheep, and goats, it is not Yet evidenced that small ruminants act as reservoirs for History of lumpy skin disease The first description of the clinical signs of LSD was in 1929 in Zambia (formerly Northern Rhodesia) (Morris 1931). In the beginning, LSD signs were considered to be the consequence either of poisoning or a hypersensitivity to insect bites. Same clinical Signs were occurred in Botswana, Zimbabwe and the Republic of South Africa between 1943 and 1945, where the infectious nature of the disease was recognized in these Outbreaks. In South Africa, LSD occurred as a panzootic, which affected eight million cattle. The Disease continuous until 1949, and generate massive economic losses (Thomas and Mare 1945; Von Backstrom, 1945; Diesel, 1949). In 1957, LSD was identified in EastAfrica in Kenya. In 1972, the disease was reported in Sudan (Ali and Obeid 1977) and West Africa in 1974. While, it was spreading into Somalia in 1983 (Davies 1991 a and the disease has continuous to spread over most of African continent in a series of Epizootics as previously recorded by Davies (1991 b) and House (1990). In 2001, LSD Was reported in Mauritius, Mozambique and Senegal. Nowadays, LSD occurs in most of African continent (except Libya, Algeria, Morocco and Tunisia) (Tuppurainen and Oura 2012). Until 1980s (From 1929 to 1984) the Disease was limited to countries in Sub-Saharan African continent, albeit it's probable to move beyond this range had been proposed (Davies 1981). In the Middle East, the outbreaks of the LSD, were reported in Oman in 1984 and 2009 (House et al 1990; Kumar 2011; Tageldin 2014). Kuwait in 1986 and 1991, Egypt in 1988 and 2006 (Ali et al 1990; House et al 1990; Davies 1991a; Fayez and Ahmed 2011; Ali and Amina 2013), Israel in 1989 and 2006 (Shimshony 1989; APHIS 2006; Shimshony and Economides 2006), Bahrain in 1993 and 2002-2003, Yemen, United Arab Emirates in 2000 and the West Bank also reported LSD invasion (Shimshony and Economides, 2006; Kumar 2011; Sherrylin et al 2013). In Oman, LSD was re-emerged Once again in 2009 in a farm population of 3200 Holstein animals with 9 high morbidity and mortality rates 30-45 % and 12% respectively (Tageldin et al 2014). In Egypt, Suez Governorate, the LSD was reported in May 1988 (Ali et al 1990). The disease was Arrived in Egypt with cattle imported from-Africa and kept at the local quarantine Station. It spread locally in the summer of 1988 and apparently overwintered with little or no manifestation of clinical disease. Twenty-two out of twenty-six Egyptian Governorates were affected with diseases, then the disease reappeared in the summer of 1989 and continuous for five to six months. This epizootic showed low morbidity rate (2%) due to the vaccination procedure that included nearly two million cattle with a Sheep pox vaccine. However, approximately 1449 animals died. In the summer of 2006, In one farm with a total of 30 cases in dairy cows. LSD outbreak was re-emerged again in several Egyptian governorates, where all age groups and both sex of Egyptian Cattle were infected with severe and serious complications. (Fayez and Ahmed 2011; Ali and Amina 2013). In Israel, the LSD was reported in 1989. This outbreak was subsequently disposed of by the slaughter of all infected cattle as well as contacts. In Addition, ring vaccination with a sheep pox strain was carried out around the focus area which led to limit the distribution of the disease. One of the recent outbreaks of LSD in African continent were occurred in central Ethiopia in 2007 to 2011. These outbreaks were described as active. It was investigated in four districts: Adama, Wenji, Mojo and Welenchiti. The totally 1,675 outbreaks were Reported over 5 years period from 2007 to

2011, with 62,176 cases and 4,372 deaths. The Oromia represented the highest numbers of outbreaks (1,066), followed by Amhara (365) and the Southern Nations, Nationalities and People's Region (123). The 2010 were reported the highest number of outbreaks that were frequently seen between September and December. The morbidity and mortality rates were 13.61% (296) and 4.97 % respectively (Ayelet et al 2014). Syria, Lebanon and Jordan are joined LSD affected countries in 2012 and 2013. The Disease has been reported in Turkey in October 2013, Iran and Iraq in 2014 (Figure 2) (Sherrylin et al 2013; Lumpy skin disease, Iraq 2015).In Jordan, LSD was reported as emerging disease. The outbreak started in mid-April, 2013. Two adult dairy cattle in Bani Kenanah district, Irbid governorate, on the Jordanian border of Israel and Syria, were developed clinical signs suggestive of LSD and confirmed as positive by PCR. The overall morbidity rate was 26%, mortality rate 1.9% and case fatality rate 7.5% (Abutarbush et al 2013). In Iran, the LSD considered as emerging disease that has been identified for the first Time in 2014. In total, six cases were reported in dairy cows. The outbreaks were reported in two villages in the west of the country. The illegal movement of animals and the usual vectors are thought to be the source of the outbreak. (The cattle site 2014). The expectation of the travelling and invasion of the LSD to free neighbours countries Are possible. LSD may invade north and west from Turkey into Europe and the Caucasus and East to Central and South Asia. In addition, Russian Federation to the north and Bulgaria and Greece to the west are considered to be at-risk countries.

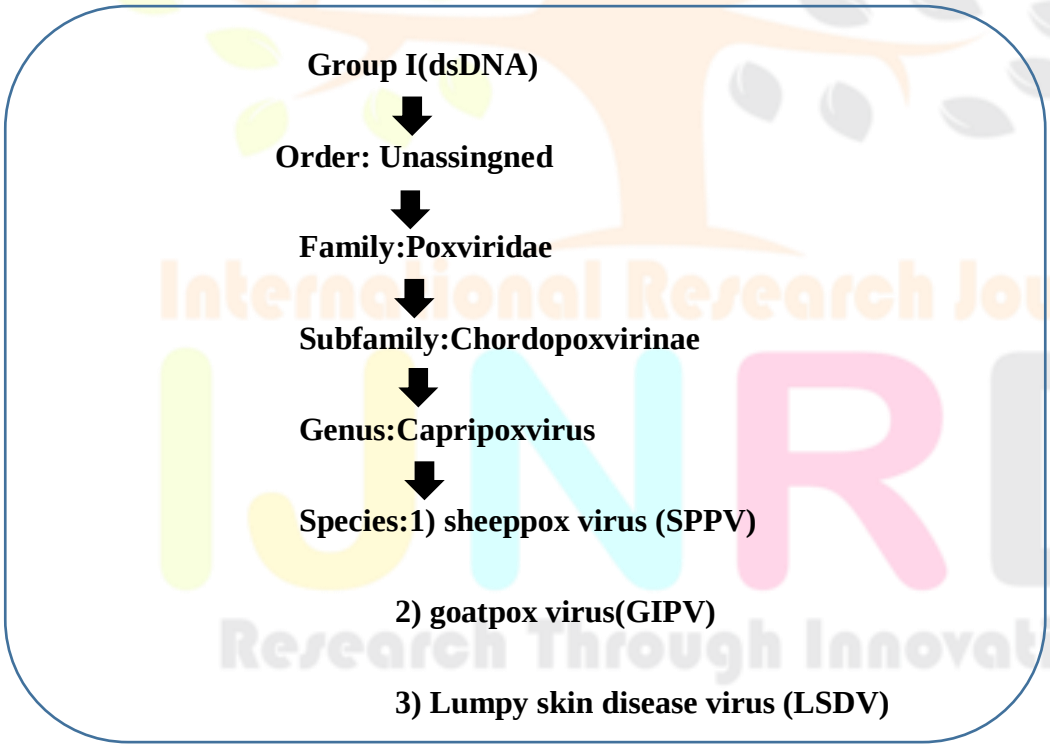

Fig 1. **Classification of Lumpy skin disease virus:**

BIOLOGY OF LSDV:

The virus that causes LSD is an enveloped, linear, ovoid, Double-stranded DNA virus under the family Poxviridae and genus Capri poxvirus [6]. The sole Serotype of LSDV; "Neethling" was first identified in South Africa and represented similar antigenic Properties with goat and sheep pox virus [7]. The virus is characteristically impervious to many physical and chemical agents and remains constant between pH 6.6 and 8.6, but is predisposed to higher alkaline Environment [7]. It undergoes an exclusive survival Capability in necrotic skin nodules (33 days), desiccated Crusts (35 days), sunlight protected infected tissue (6 Months) and air-dried hides at room temperature (minimum 18 days) [8]. Resistance to heat is flexible but most isolates are disabled at 55ºC for couple of Hours, or 65°C for 30 minutes [9]. The virus is Susceptible to highly alkaline or acidic solutions, and Detergents containing lipid solvents [10]. The organism becomes defenceless in daylight while inactivated with Ultraviolet rays and at 55 °C for one hour [20]. Moreover, LSDV shows susceptibility to 20% Chloroform, 1% formalin, ether, 2% phenol, 2–3% Sodium hypochlorite, 0.5% quaternary ammonium Compounds, iodine compounds dilution and the Detergents containing lipid solvents [11].

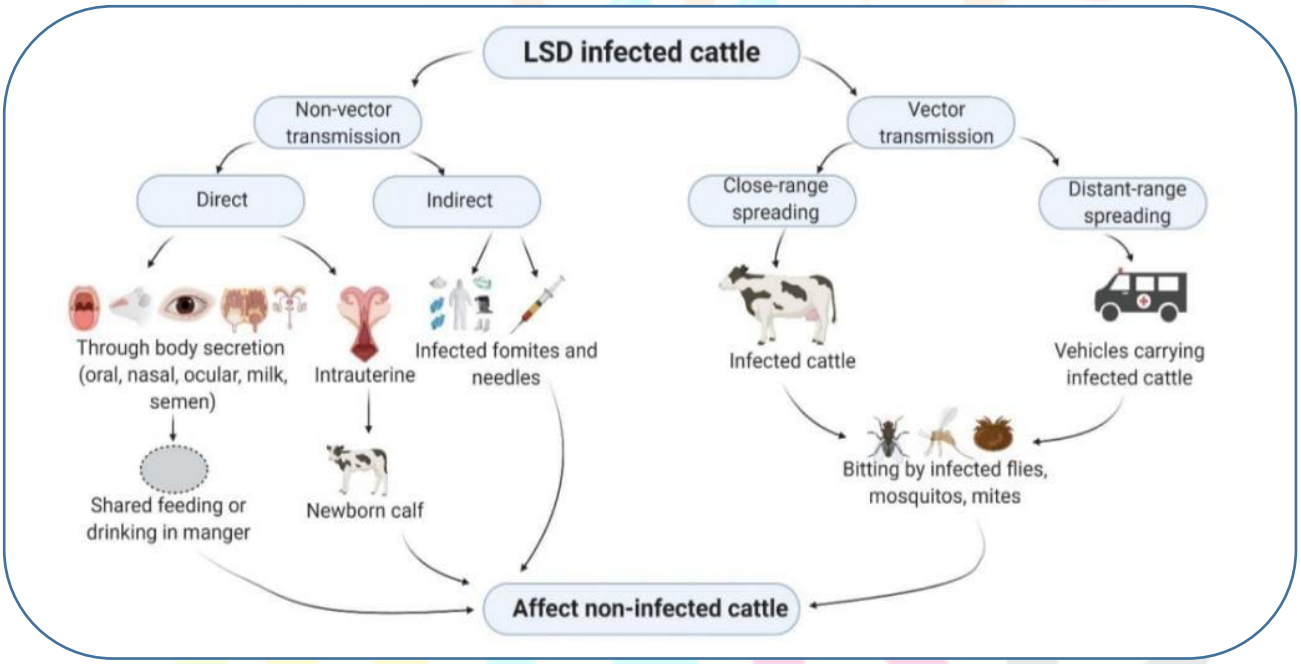

Fig 2: Epitome of possible modes of transmission of LSDV.LSD infected cattle may affect non-infected cattle through vector or non-vector transmission.

PATHOGENESIS:

LSD is manifested by prompt explosion of multiple circumscribed cutaneous nodules and accompanied by a febrile reaction [12]. The spread of viral particles takes place through blood and form generalized lymphadenitis [13]. Viremia occurs after the early febrile condition for almost 4 days. Following skin lesions due to the replication of the virus in certain cells such as fibroblasts, pericytes, and, endothelial cells of lymphatic and blood vessels lesions are produced in those sites [14, 15]. Histopathological changes in acute skin injuries include lymphangitis,vasculitis, thrombosis,

infarction, edema and necrosis [10]. Nodules might be found in subcutaneous tissues and muscle fascia [11]. Neighboring tissue of epidermis, dermis, and core musculature reveal hemorrhages congestion, and edema with distended lymph nodes [2]. A special structure called 'sit-fasts' (necrotic cores detached from the adjacent skin) [8] is usually seen indifferent parts of the body, which may ulcerate [17]. The host immunological status exposes the lower rate of lymphocyte diffusion and phagocytic motion during the subsequent fourteen days of post infection [18].

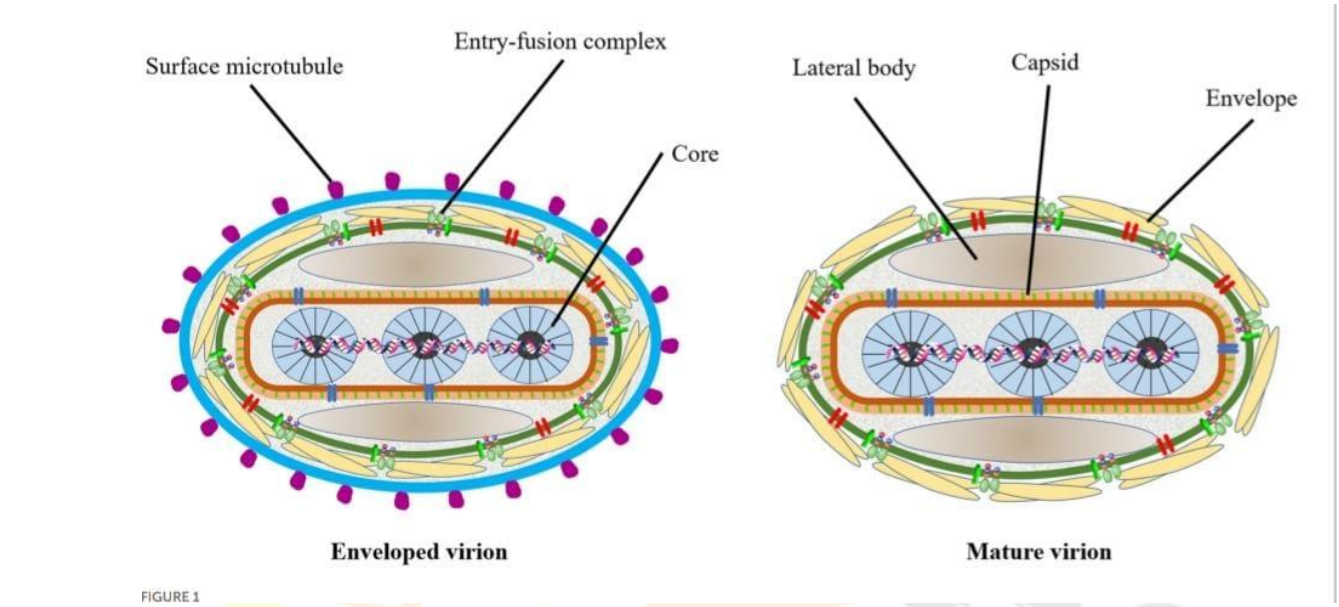

Fig3. The prediction diagram of LSDV Structure mode.

Gross pathological findings:

LSD has well-described gross lesions. Skin nodules are usually uniform in size, firm Round and raised, but some may fuse into large irregular and circumscribed plaques. The Cut surface of the nodules is reddish-gray, in addition, to the accumulation of the reddish Grey serous fluid and edema in the subcutis layer. The resolved lesions appear as Indurated which is called "sitfasts" or seclude or may form deep ulcers. The typical Circular necrotic alimentary lesions may also be seen on the muzzle, nasal cavity, larynx, Trachea, bronchi, inside of lips, gingiva, dental pad, forestomach, abomasum, uterus,Vagina, teats, udder and testes (Ali et al 1990)[19]. Regional lymph nodes are grossly enlarged and can be 3-5 times their usual size, oedematous and having pyaemic foci, in Addition to local cellulitis. Muscle tissue and the fascia over limb muscle may be show Nodular lesion that are grey-white surrounded by red inflammatory tissue. The same Nodules are distributed throughout the carcass. It is about 10-30 mm diameter in the Kidney. Interstitial or bronchopneumonia associated with 10-20 mm diameter lesions are also scattered in the lungs. These lesions result from infiltration of the large Epithelioid 'celles claveleuses', described by Borrel for sheep pox. The lesions are separated from the necrotic epithelium far from the healthy tissue. The necrotic tissue Sloughs away to leave an ulcer that slowly heals by granulation. Severely infected Animals may show secondary bacterial pneumonia, tracheal stenosis, acute and chronic Orchitis, mastitis with secondary bacterial infection, and similar

lesions in the female Reproductive tract (Davies et al 1971; El-Neweshy et al 2012; Kumar 2011).[20,21,22]

Histopathological findings

Histopathological findings of the LSD disease are very characteristic and provide a Basis for diagnosis. The lesions vary considerably depending on the stage of Development. In the acute stage of the disease, it is mostly characterised by lesions of Vasculitis, thrombosis, infarction, perivascular fibroplasia. Inflammatory cell are Infiltrated the infected areas, which includes macrophages, lymphocytes and Eosinophils. Keratinocytes, macrophages, endothelial cells and pericytes may be Revealed Intracytoplasmic eosinophilic inclusions. The epidermis and dermis layers of the infected animal are showing oedema and infiltrated with large epithelioid Macrophage type cells. There are an oedema and infiltration of the epidermis and dermis with large epithelioid Macrophage type cells, which have also been well described for sheep pox. They are found with plasma cells and lymphocytes in early lesions, and in older lesions, Fibroblasts and polymorphonuclear leucocytes with some red cells predominate. Endothelial proliferation is seen in the blood vessels of the dermis and subcutis, with Lymphocytic cuffing of the blood vessels, which lead to the thrombosis and necrosis. Specific intracytoplasmic inclusions may be found in the various epithelial elements, Sebaceous glands and follicular epithelium. These are largely eosinophilic-purple and IP Appear to have a clear halo surrounding them, which is probably a processing artefact. The lesions are substantially the same throughout the body (Burdin 1959; Ali et al 1990; El-Neweshy et al 2012; Ali and Amina 2013)[23]

Fig4: Infected cow

Diagnosis:

The diagnosis of LSD is based on typical clinical signs combined with laboratory Confirmation of the presence of the virus or antigen.

1. A Field presumptive diagnosis of LSD can be based upon the:

A. Morbidity, mortality and clinical signs that reflect LSD such as:

1. Contagious disease with generalised skin nodules

2. A characteristic inverted conical necrosis of skin nodules (sitfast), Enlargement of

Lymph nodes draining affected areas.

3. Persistent fever, emaciation, and low mortality.

4. Pox lesions of mucous membrane of the mouth, the pharynx, epiglottis, tongue and throughout the digestive tract, mucous membranes of the nasal cavity, trachea and lungs

5Oedema and areas of focal lobular atelectasis in lungs

6. Pleuritis with enlargement of the mediastinal lymph nodes in severe cases

7.Synovitis and tendosynovitis with fibrin in the synovial fluid

8.Pox lesions may be present in the testicles and urinary bladder

B. Histopathological features:

Skin biopsies of early lesions are suitable for histopathology and should be preserved in 10 percent buffered formalin. The most diagnostic histopathological features are: Congestion, haemorrhage, oedema, vasculitis and necrosis are always associated with Nodules that are involving all skin layers, subcutaneous tissue, and often adjacent Musculature. Lymphoid proliferation, oedema, congestion and haemorrhage.Vasculitis, thrombosis, infarction, perivascular fibroplasia and cellular infiltrates Intracytoplasmic eosinophilic inclusions may be seen in different cells.

A confirmative diagnosis of LSD can be based upon the:

Laboratory investigations and identification of the agent based on (OIE 8. Terrestrial Manual 2010; OIE 2013): [(24)] Isolation of the virus Confirmation of lumpy skin disease in a new area requires virus isolation and

Identification.

Samples for virus isolation should be collected within the first week of the occurrence of clinical signs, before the development of neutralising antibodies (Davies 1991; Davies et al 1971).Skin biopsies of early lesions (ones where necrosis has not occurred) provide samples that can be used for virus isolation and electron Microscopy. In addition, LSD virus can be isolated from buffy coat from the blood. Sample collected into EDTA or heparin during the viraemic stage of LSD. Samples Should be taken from at least three animals. Samples aspirated from enlarged lymph Nodes can be also used for virus isolation. LSD virus grows in tissue culture of bovine, Ovine or caprine origin. Bovine dermis cells or lamb testis (LT) cells (Primary or Secondary culture), are considered to be the most susceptible cells.

LSD capripoxvirus have been also adapted to grow on the chorioallantoic membrane of embryonated Chicken eggs and African green monkey kidney (Vero) cells, which is not recommended 38. For primary isolation (OIE Terrestrial Manual 2010).

B. Electron microscopy:

Transmission electron microscopic (TEM) diagnosis of LSD can be confirmed within a Few hours of receipt of specimens. TEM demonstration of virus in negatively stained Preparations of biopsy specimens taken from affected skin or mucous membranes. Mature capripox virions have an average size 320 x 260 nm and are a more oval profile and larger lateral bodies than orthopox virions (OIE Terrestrial Manual 2010).

C. Fluorescent antibody tests:

Capripoxvirus antigen can also be identified on the infected cover-slips or tissue culture Slides using fluorescent antibody tests.

D. Agar gel Immunodiffusion

An agar gel immunodiffusion (AGID) test has been used for detecting the precipitating Antigen of capripoxvirus, but has the disadvantage that this antigen is shared by Parapoxvirus.

E. Enzyme-linked immunosorbent assay

It is made by using expressed recombinant antigen to produce P32 monospecific Polyclonal antiserum and the production of monoclonal antibodies (MAbs) (Carn, et Al 1994).Polymerase chain reaction (PCR) and loop-mediated isothermal amplification (LAMP) assay have been used for detection of capripoxviruses with higher sensitivity. (Bowden et al 2009; Balinsky et al 2008).

Serology:

Frozen sera from both acute and convalescent animals are used. Virus neutralisation (cross reacts with all capripoxviruses) and indirect fluorescent antibody test (cross Reaction with parapoxviruses) are commonly used. Enzyme-linked immunosorbent Assay for the detection of antibodies against capripox virus has been developed usingThe expressed structural P32 protein (Carn et al., 1994; Heine et al 1999). Agar gel Immunodiffusion tests (This test may give false-positive reactions due to cross reactionWith bovine papular stomatitis virus and pseudocowpox virus). Western blot analysis Provides a sensitive and specific system for the detection of antibody to capripoxvirus Structural proteins, although the test is expensive and difficult to carry out Differential diagnosis **.**There are many diseases causing similar signs of LSD. It is important to obtain a definite Diagnosis to ensure the best preventative and control measures for susceptible herds.

LSD can be confused with the following diseases:

- Pseudo-lumpy-skin disease
- Bovine virus diarrhoea/mucosal disease
- Demodicosis (Demodex)
- Bovine malignant catarrhal fever (Snotsiekte)
- Rinderpest
- Besnoitiosis
- Oncocercariasis
- Insect bite allergies

Treatment

Lumpy skin disease is caused by virus and, therefore, has no known cure. However, Antibiotics, anti-inflammatory drugs or a shot of vitamins are used in some cases to treat Secondary bacterial infections or to deal with fever or inflammation and improvement Of the animal's appetite Control .Control of Lumpy skin disease by quarantine and movement control is not very effective Because biting flies and certain tick species are most probably the most important Method of transmission of the disease. Although, the control of insects was not effective in preventing the spread of LSD, but use of insecticides together with repellents can be an aid in the prevention of the spread of LSD. LSD outbreaks can be eradicated by Quarantines, depopulation of infected and exposed animals, proper disposal of carcases, Cleaning and disinfection of the premises and insect control. LSD control can only be by vaccination or immunoprophylaxis. Live vaccines help Control losses from lumpy skin disease in endemic areas. According to OIE, four live Attenuated strains of capripoxvirus have been used as vaccines specifically for the Control of LSD (Brenner et al, 2006; Capstick & Coakley 1961 & 1962; Carn et al.1994). These are: a strain of Kenyan sheep and goat pox virus passaged 18 times in lamb Testis (LT) cells or fetal calf muscle cells, Yugoslavian RM 65 sheep pox strain, Romanian sheep pox strain and lumpy skin disease virus strain from South Africa, Passaged 60 times in lamb kidney cells and 20 times on the chorioallantoic membrane Of embryonated chicken eggs. The following vaccines have been used in protection of the animal .Homologous live attenuated virus vaccine (Neethling strain: immunity conferred lasts up to 3 years). Heterologous live attenuated virus vaccine (Sheep or goat pox vaccine, but May Cause local, sometimes severe reactions). This vaccine is not advised in countries Free from sheep and goat pox because the live vaccines could otherwise provide a source of infection for the susceptible sheep and goat populations. There is no new generation recombinant capripox vaccines are commercially available.

Conclusions:

In this work, we used a statistical approach to identify major changes in the data un-derlying LSD outbreak reports. Additionally, we utilized time series models to forecast the number of LSD outbreak reports in Africa, Europe, and Asia during 2022–2024. Although LSD outbreak reports in Africa appear to be decreasing since 2020, it is expected that the number of reports will increase slightly. The number of LSD outbreak reports in Europe isprojected to continue the previous 5-year steady trend. Additionally, the forecast predicts an increase in the number of outbreak reports in Asia. These findings indicate that LSD remains a substantial threat to the cattle industry in various countries; thus, efforts should be made to monitor its spread within and between regions. Additionally, because LSDis regarded as a significant transboundary disease, strict disease prevention and control in every country are critical. Furthermore, coordination among nations to control and eradicate the disease is essential.

REFERENCES:

1. Calistri P, De Clercq K, Gubbins S, Klement E, Stegeman A, Cortiñas Abrahantes J, et al. Lumpy skin disease epidemiological report IV: data collection and analysis. EFSA Journal. 2020; 18(2):6010.

2. Tuppurainen ESM, Babiuk S, Klement E. Lumpy skin disease. Springer international Publishing, USA, 2018, pp 47-51.

[3] Swiswa S, Masocha M, Pfukenyi DM, Dhliwayo S, Chikerema SM. Long-term changes in the spatial distribution of lumpy skin disease hotspots in Zimbabwe. Tropical Animal Health and Production. 2017; 49:195–199.

[4] Negesso G, Hadush T, Tilahun A, Teshale A. Trans-Boundary Animal Disease and Their Impacts on International Trade: A Review. Academic Journal of Animal Diseases. 2016; 5:53–60.

[5] Kardjadj M. Capripoxviruses: Transboundary Animal Diseases of Domestic Ruminants. Annals of Virology and Research. 2016; 2(3):1024.

6) Yilmaz H. Lumpy Skin Disease: Global and Turkish Perspectives. Approaches in Poultry, Dairy & Veterinary Sciences 2017; 1:11-15.

7. Abdulqa HY, Rahman HS, Dyary HO, Othman HH. Lumpy Skin Disease. Reproductive Immunology: Open Access.2016; 01:1–6.

8) Ali A, Gumbe F. Review on lumpy skin disease and its economic impacts in Ethiopia. 2018; 7:39–46.

9) Kreindel S, Masiulis M, Skrypnyk A, Zdravkova A, Escher M, Raizman E. Emergence of lumpy skin disease in Asia and Europe. FAO. 2016; 360:24–26.

10) Aber Z, Degefu H, Gari G, Ayana Z. Review on Epidemiology and Economic Importance of Lumpy Skin[7:48 am, 13/12/2022] Pankaj: Disease. International Journal of Basic and Applied Virology. 2015;4:8–21.

11) Hailu B, Tolosa T, Gari G, Teklue T, Beyene B. Estimated prevalence and risk factors associated with clinical Lumpy skin disease in north-eastern Ethiopia. Preventive Veterinary Medicine. 2014; 115:64–68.

12) Lubinga JC, Tuppurainen ESM, Coetzer JAW, Stoltsz WH, Venter EH. Evidence of lumpy skin disease virus over-wintering by transstadial persistence in Amblyomma hebraeum and transovarial persistence in Rhipicephalus decoloratus ticks. Experimental and Applied Acarology.2014; 62:77–90.

13) Lubinga JC, Tuppurainen ESM, Mahlare R, Coetzer JAW, Stoltsz WH, Venter EH. Evidence of transstadial and mechanical transmission of lumpy skin disease virus by Amblyomma hebraeum ticks. Transboundary and Emerging Diseases. 2015; 62:174–82.

15) [8:05 am, 13/12/2022] Pankaj: Abdulqa HY, Rahman HS, Dyary HO, Othman HH. Lumpy Skin Disease. Reproductive Immunology: Open Access.2016; 01:1–6.

16)Hailu B, Tolosa T, Gari G, Teklue T, Beyene B. Estimated prevalence and risk factors associated with clinical Lumpy skin disease in north-eastern Ethiopia. Preventive Veterinary Medicine. 2014; 115:64–68.

17. Lubinga JC, Tuppurainen ESM, Coetzer JAW, Stoltsz WH, Venter EH. Transovarial passage and transmission of LSDV by Amblyomma hebraeum, Rhipicephalus appendiculatus and Rhipicephalus decoloratus. Experimental and Applied Acarology. 2014; 62:67–75.

18.Hendrickx G, Gilbert M, Staubach C, Elbers A, Mintiens K, Gerbier G, et al. A wind density model to quantify the airborne spread of Culicoides species during north-western Europe bluetongue epidemic, 2006. Preventive Veterinary Medicine. 2008; 87:162–81.

19) 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.

20) Davies FG, Krauss H, Lund LJ, Taylor M. (1971). The laboratory diagnosis of lumpy skin disease. Res. Vet. Sci., 12:123-12

21)El-Neweshy MS, El-Shemey TM and Youssef SA. (2012). Pathologic and Immunohistochemical Findings of Natural Lumpy Skin Disease in Egyptian Cattle.Pak Vet J, xxxx, xx(x): xxx. ©2012 PVJ.

22) Kumar S M. (2011). An Outbreak of Lumpy Skin Disease in a Holstein Dairy Herd in Oman: A Clinical Report. Asian Journal of Animal and Veterinary Advances, 6, 851–859.

23) OIE Terrestrial Manual. (2010). Lumpy Skin Disease. Chapter 2.4.14. (Available at http: //www.oie. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.24OIE Terrestrial Manual. (2010). Lumpy Skin Disease. Chapter 2.4.14. (Available at http: //www.oie. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.