

A GLOBAL BURDEN OF LUMPY SKIN DISEASE, OUTBREAKS, AND FUTURE CHALLENGES

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Introduction

The symptoms of lumpy skin disease (LSD), which mostly affects cattle, include fever, lymphadenitis, mucosal irritation, cutaneous nodules, ocular discharge, and edema of subcutaneous cellular tissue. It is a disease that may be spread by different arthropods and results in large economic losses due to the exhaustion of cattle, damage to their hides, sterility, mastitis, and decreased milk output. Up to 20% of death has also been documented (Chihota et al., 2003; Shalaby et al., 2016). Other domestic animals and wildlife have been shown to have LSD both naturally occurring and experimentally infected (Authority, 2015; Usadov et al., 2018; Young et al., 1970). Many species have been shown to have LSD antibodies (Barnard, 1997; Hedger & Hamblin, 1983; Kayesh et al., 2020), although these might be responses to other capripoxviruses (Davies, 1982; Hamblin et al., 1990). A DNA virus that is a member of the poxviridae family is the main cause of lumpy skin disease (Tulman et al., 2001). Three viruses make up the capripoxvirus genus: Goat pox virus (GTPV), Lumpy skin disease virus (LSDV), and Sheep pox virus (SPPV). The LSDV was discovered as an etiological agent in the 1940s (Von Backstrom, 1945; Weiss, 1968). Before the 1980s, when it first appeared in the Middle East and other Near Eastern nations, LSD seemed to be exclusive to Africa (Wainwright et al., 2013). The illness was initially identified in 2014 in Azerbaijan (Zeynalova et al., 2016), and it then expanded to Europe in 2014 and 2016 before reaching Russia in 2017 (Authority, 2017).

LSDV is the cause of lumpy skin disease that includes the Poxviridae family and subfamily Chordopoxvirinae of the genus capripoxvirus. Knopvelsiekte, Pseudo-urticaria, Neethling viral sickness, and LSD are some of the terms used to describe the illness (Al-Salihi, 2014; E Tuppurainen et al., 2017). LSD is a transboundary, vector-borne, non-zoonotic illness that presently only affects ruminants, such as cattle and water buffaloes. Biting flies, mosquitoes, and ticks are the arthropod vectors that transfer illness (Bosch et al., 2017; Lubinga et al., 2013; Tuppurainen et al., 2011). The Lumpy skin disease is marked by swollen lymph nodes, high fever, and nodules on the skin that cause acute anorexia, decreased milk production, and infertility. Overall, it has an impact on the economic worth of animals since it will have an impact on their ability to produce meat and milk, high-quality hides, draught power, and reproductive effectiveness (abortion and infertility) (Rgbe, 2014). It is an illness that must be reported and has a terrible impact on international cattle commerce as well. Although the illness is indigenous to African nations, it has lately been discovered in hitherto undiscovered regions of the globe. Morris (1931) (Morris, 1930) noted that the first LSD case originated in Zambia and afterward spread to southern and northern African nations. Israel, Kuwait, Oman, and Yemen later were affected by it (Wainwright et al., 2013).

According to OIE, this illness is now widespread in a number of African, European, and Asian nations (Tuppurainen et al., 2015). Unknown factors may have contributed to the disease's spread to India, such as cattle crossing international borders or vectors traveling from nearby nations. LSD use has recently been recorded in nations that

border India, and Pakistan including Bangladesh and China. For appropriate planning of efficient disease management, a knowledge of the epidemiology of exotic illnesses becomes essential. The most recent LSD developments are summarised in this overview.

Several significant companies and sectors have indicated that LSD has a direct or indirect socioeconomic effect. The considerable drop in milk output is the first and most noticeable effect of LSD in the South Asian region, which is home to 21% of the world's dairy farm animals (Das et al., 2021). Arthropods, particularly blood-sucking insects (Annandale et al., 2014; Chihota et al., 2001; Irons et al., 2005; ESM Tuppurainen et al., 2017) contaminated feed, contaminated water, and direct transmission through saliva, nasal secretions, and semen in the later stages of the infection are the main vectors for the spread of LSDV. According to several studies, hard ticks may contribute to the spread of viruses (Lubinga et al., 2015; Tuppurainen et al., 2013). It has been shown that the virus and viral antigen responsible for lumpy skin disease is present in the saliva and other tick organs such as the midgut, haemocytes, and salivary glands (Lubinga et al., 2013; Lubinga, Clift, et al., 2014). *Aedes aegypti* is the only dipteran capable of completely transmitting the virus to susceptible cattle (Chihota et al., 2001).

Origin and genetics

The virus that causes lumpy skin disease (LSDV), which affects all domestic animals except dogs, is a member of the Poxviridae family of viruses. Two subfamilies make up the family: Entomopoxvirinae, which infects invertebrate hosts, and Chordopoxvirinae, which infects vertebrate hosts (Quinn et al., 2015). There are ten genera in the Chordopoxvirinae subfamily, including Capripoxvirus. This genus encompasses viruses from three different species, including LSDV, GTPV, and SPPV which infect cattle, goats, and sheep respectfully (King et al., 2012).

The LSDV is brick-shaped, encapsulated having complex symmetry. It also contains double-stranded DNA and reproduces in the cytoplasm of the host cell. It is 320 X 260 nm in size. The alike 2.4 kbp-long inverted terminal repeats that encircle the central coding region are seen throughout the 151 kbp-long LSDV genome, which comprises 156 putative genes. Sheeppox and goatpox virus homologs share 97% of the nucleotides in 30 structural and non-structural genes found in LSDV (Tulman et al., 2001; Tulman et al., 2002). When comparing SPPS, LSDV, and GTPV in Capri poxviruses, similar patterns have been seen. The later evolution of poxviruses leads to a reduction of their host range owing to gene loss. The LSDV 132, N2L, myxoma viruses M004.1 and M003.2, K7L, IL-1 receptor, F11L, and Vaccinia viruses unique gene are among the nine genes encoded by the terminal regions of the LSDV virus. Accumulated mutations in GTPV and SPPV are predicted to have a detrimental effect on the host range and virulence functions of the LSDV 132 gene. The three viruses' genome sequence length is unaffected by this disruption, but the lack of these genes in GTPV and SPPV implies that the host limitation to cows alone may be caused by this disruption (Biswas et al., 2020; Tulman et al., 2002).

Unlike other chordopoxviruses, the LSD virus contains 146 invariant genes that, when processed into proteins, regulate the production of proteins associated with stability and structural development, host range, mRNA synthesis, transcription, nucleotides metabolism, and DNA replication and virulence. The genes responsible for host range and viral virulence are different in the terminal region, and those that do exist have a lower level of amino acid similarity, on average only 43%, whether they are present

or not. The LSDV contains genes that are similar to epidermal growth factors like protein, IL-10, IL-1, binding proteins, the G protein-coupled CC chemokine receptor (GPCR), and more. Other genera of poxvirus include these genes often (Tulman et al., 2001).

The LSD clinical syndrome was first noted in Zambia in 1929. It was formerly assumed that it was the consequence of poisoning or a reaction to insect stings. Additional occurrences are documented from Zimbabwe (Southern Rhodesia), the Republic of South Africa, and Botswana (Bechuanaland) between 1943 and 1945. Up until 1949, a panzootic disease affected over 8 million cattle in South Africa, causing considerable economic losses (Diesel, 1949; Von Backstrom, 1945). Sudan in 1972, West Africa in 1974, and East Africa (Kenya) in 1957 were the first places where LSD was discovered and diagnosed. Between 1981 and 1986, outbreaks of epizootic LSD were also documented in Cameroon, Tanzania, Zimbabwe, Somalia, and Kenya with death rates of 20% in infected cattle (Brenner et al., 2006). Between 1929 and 1986, the illness was only present in a few sub-Saharan African nations (Davies, 1981). The United Arab Emirates (UAE) (2000), Lebanon (1993), Yemen (1995), Bahrain (2003), Kuwait (1991), Israel (2006), the World Organization for Animal Health (OIE), and Oman (2010) (Tuppurainen & Oura, 2012) have all reported finding LSD (Abdulqa et al., 2016; Coetzer & Tuppurainen, 2004).

In comparison to orthopoxvirus, mature capripoxvirions have bigger lateral bodies and a more oval appearance (Munz & Owen, 1966). Their typical dimensions are 320 x 260 nm (Ghaboussi, 1978). The LSD virus flourishes and multiplies to exceptionally high levels in a wide variety of cell cultures, comprising kidneys from lamb and calf, adrenal and thyroid glands, muscle, and testes. The onset of cytopathic effects during early isolation might take up to 11 days (Weiss & Geyer, 1959). There is only a single serotype of the LSD virus, that is hard to distinguish using conventional viral neutralization tests due to its serological similarity to the virus that causes sheep and goat pox (SGP) (Burdin, 1959).

Epidemiology

It is believed that the majority of LSD virus infections are transmitted via means of insects (MacOwan, 1959; Von Backstrom, 1945). The pox virus is very resilient and may live in infected tissue for at than 120 days, and likely for longer. The virus may also be discovered in the blood and nasal saliva, spit semen, and lacrimal secretions, which are regarded as the primary means of LSD transfer. Cross-protection between the LSD virus and sheep or goat pox viruses has been utilized in Kenya and the Middle East to immunize cattle against LSD. It is possible to recover the LSD virus from necrotic skin nodules that have been stored at -80°C for ten years and also from infected tissue culture that has been stored at 4°C for six months. When compared to thick-skinned local breeds like the Afrikaner and Afrikaner crossbreeds, the sickness often appears more strongly in imported Bostaurus breeds, such as Friesian cattle with necrotic skin nodules (Abdulqa et al., 2016). Even though all age groups are vulnerable, the clinical illness is more severe in young animals and cows that are lactating at their peak (Coetzer, 2004). A skin lesion harbouring the virus most likely appears at the injection site within 1-3 weeks following experimental infection by intradermal inoculation, but the incubation period in the field is between 2 and 5 weeks (Abdulqa et al., 2016; Haig, 1957). A significant, financially crippling, and reportable condition called lumpy skin disease reduced cow output as a result of widespread illnesses and persistent weakness. A thorough knowledge of the epidemiological elements of LSD relating to the pathogen, host, and environment may help with

preventative strategies. Particular focus was placed on exposing hosts and the virus to environments that were conducive to the disease's transmission and spread (Hailu et al., 2015). The disease's occurrence, geographic range, and method of transmission in big herds of cattle were all shown to result in significant economic losses (Salib & Osman, 2011; Tuppurainen & Oura, 2012).

Geographical Distribution

The geographic distribution of LSDV, GPV, and SPV are distinctive from one another, and over the last fifty years, SPV and GPV have been geographically limited to Africa and Asia, spreading from Africa to the north of the equator (Kitching et al., 1989). In 1929, LSD first appeared in nations in Sub-Saharan Africa, and over the next seventy years, it expanded to both the north and south. All of the sub-Saharan African nations, as well as Madagascar, are now included in the geographic scope of LSD. It is endemic to all of the African nations and may be found in a variety of ecological zones, including temperate, dry semi-arid, and desert regions (Davies, 1991).

Zambia was the first African country to report using LSD in 1929 (Gari et al., 2011). In 1989, LSD was discovered outside of Africa in Israel, Palestine, Jordan, Lebanon, Kuwait, Saudi Arabia, Iraq, Oman, Yemen, the United Arab Emirates, and Bahrain (Abutarbush et al., 2015; Al-Salihi & Hassan, 2015). In 2014, it was detected in Europe's middle and eastern regions (Sameea Yousefi et al., 2017; Tageldin et al., 2014). In 2016, LSD was proven in South East Europe, the Balkans, and the Caucasus.

Host range

Cattle and Asian water buffaloes (*Bubals bubalis*) are the principal natural hosts for LSDV, with all ages and sexes vulnerable to infection. Calves acquire serious lesions 24–48 hours before their dams (Elhaig et al., 2017). In breeds with thin skin, such as Friesians, particularly at cows' peak lactation, more severe illness symptoms have been seen (evik & Doan, 2017). Although mixed herds of cattle, buffalo, sheep, and goats are more often seen, no epidemiological data on the function of small ruminants as a reservoir for LSDV have been documented (Elhaig et al., 2017). In Egypt, LSDV affects local breeds of all ages, producing mild to severe illness, although severe instances have been discovered in young calves and alien breeds (Salib & Osman, 2011). Additionally, the development of disease outbreaks is greatly influenced by the separation of LSDV from naturally infected water buffaloes (Elhaig et al., 2017; Sharawi & Abd El-Rahim, 2011). Despite interaction with clinically infected cattle that was verified by viral isolation and PCR in a different investigation involving buffaloes, none of the buffaloes tested positive for LSDV by virus isolation and PCR, despite a modest rise in LSDV antibodies in their serum (Elhaig et al., 2017). Arabian Oryx (*Oryx leucoryx*) and spring box have both shown clinical indications of LSD (*Antidorcas marsupialis*). Additionally, giraffes (*Giraffa camelopardalis*) and impalas (*Aepyceros melampus*) have been experimentally infected, although wildlife does not play a large part in the epidemiology of the illness (Gortázar et al., 2022). Although sheep and goats may be experimentally infected with LSDV, the virus cannot be acquired naturally (Wolff et al., 2020).

Pathogenesis

After LSDV infection, the virus replicates, viremia develops, fever sets in, the virus localizes to the skin, and nodules form (Constable et al., 2016). After intradermal inoculation of the virus, the following incidents were experimentally observed:

Four to seven days post-infection (DPI), there is localized swelling at the inoculation site as 1-3 cm nodules or plaques. From 6 to 18 DPI, viral shedding manifests as oral and nasal discharge. The development of generalized cutaneous nodules and regional lymphadenopathy takes place between 7 to 19 DPI (Mulatu & Feyisa, 2018b). Virus found in semen 42 days after fever (Annandale et al., 2014). Different areas of the body often exhibit a unique structure termed "sit-fasts" (necrotic cores detached from the surrounding skin) (Gumbe, 2018), which may develop ulcers (Lubinga, Tuppurainen, et al., 2014; Namazi & Khodakaram Tafti, 2021).

The virus replicates intracellularly in pericytes, macrophages, fibroblasts, and endothelial cells, causing vasculitis and lymphangitis in infected tissues (Khan et al.; Namazi & Khodakaram Tafti, 2021).

Animals that are underweight, lactating cows, and young calves seem to have compromised humoral immunity, making them more vulnerable to natural infections (Babiuk et al., 2008). Animals that have survived a viral infection have exhibited lifetime immunity. Calves from infected mothers are immune to the clinical disease for roughly 6 months because they have acquired maternal antibodies (Tuppurainen et al., 2005). There are no known carriers of LSDV at this time, and infected animals recover from the virus (Namazi & Khodakaram Tafti, 2021; ESM Tuppurainen et al., 2017).

Transmission

Wild ruminants, water buffalo, and cattle are all susceptible to lumpy skin disease. The virus does not seem to infect sheep and goats, according to studies (El-Nahas et al., 2011; Lamien et al., 2011). LSDV may persist in the environment for a very long period at normal temperatures, especially in dried scabs. The virus lives for at least 18 days in air-dried hides, 35 days in desiccated crusts, and 33 days in necrotic skin nodules. (Mulatu & Feyisa, 2018b) the claim that heating the virus to 65°C for 30 minutes or 55°C for two hours would render it inactive. Since the virus may live for a long period in scabs or lesions, skin lesions are believed to be the main site of infection. Additionally, the virus is removed through milk, blood, saliva, lachrymal secretions, nasal, and semen (transmissible to suckling calves) (Namazi & Khodakaram Tafti, 2021).

Arthropods, particularly blood-sucking insects (Annandale et al., 2014; Chihota et al., 2001; Irons et al., 2005; ESM Tuppurainen et al., 2017) that pollute feed and water, as well as direct transfer in the latter stages of the infection through nasal secretions, saliva, and semen, are the main vectors for the spread of LSDV. According to some studies, there is no correlation between infection rates and cattle density, demonstrating that early on in the disease, direct viral transmission is less significant than indirect transmission, which is more important (Magori-Cohen et al., 2012; Namazi & Khodakaram Tafti, 2021).

The fact that most LSD outbreaks occur in the summer when arthropod activity is at its peak, may be evidence that several types of vectors, particularly blood-feeding

insects, are involved in the propagation of the virus (Kahana-Sutin et al., 2017; Namazi & Khodakaram Tafti, 2021; Sprygin et al., 2018).

Several studies have shown that hard ticks may play a part in the spread of viruses (Lubinga et al., 2015; Tuppurainen et al., 2013). The saliva and other tick organs, such as the hemocytes, salivary glands, and midgut, were identified to contain the virus and viral antigen that causes lumpy skin disease (Lubinga et al., 2013; Lubinga, Clift, et al., 2014). Moreover, molecular data were used to demonstrate that ticks mechanically and transstadially transmit the virus (Stubbs et al., 2012). The quick emergence of widespread epidemics, however, is not explained by their prolonged attachment to the host. As a result, it appears that ticks might be serving as viral reservoirs (Kahana-Sutin et al., 2017; Namazi & Khodakaram Tafti, 2021).

The only dipteran capable of fully transmitting the virus to vulnerable cattle is *Aedes aegypti* (Chihota et al., 2001). *Anopheles stephensi* Liston, *Culicoides nubeculosus*, or *Culex quinquefasciatus* Say mosquitoes could not transmit the virus (Chihota et al., 2003). Despite the presence of *Stomoxys calcitrans* in LSD outbreaks and its ability to spread the capripox virus to sheep and goats (Baldacchino et al., 2013; Yeruham et al., 1995), the transmission of LSDV to vulnerable animals has not been accomplished (Chihota et al., 2003). The finding of LSDV in *Culicoides punctatus* implies that this species may be a vector for viral transmission (Şevik & Doğan, 2017). Moreover, it is claimed that the ratio of biting insects to host populations is significantly associated with the chance of transmission (Gubbins et al., 2008; Namazi & Khodakaram Tafti, 2021).

In trials, both PCR and viral isolation revealed the persistence of the lumpy skin disease virus in bovine semen (Annandale et al., 2010; Givens, 2018; Irons et al., 2005). Additionally, semen contributed to the virus' spread among inseminated heifers (Annandale et al., 2014; Namazi & Khodakaram Tafti, 2021).

Clinical Signs and symptoms

Numerous researchers around the world have noted the following clinical signs, including viral excretion through nasopharyngeal and oropharyngeal release, viral localized on the skin, vasculitis, pyrexia, infected sperms, growth of abscesses harm the host's hide, decreased fertility of bull, destruction to livestock pelts, decreased milk yield and body mass, lymphangitis, and geographic swollen lymph viremia, virus multiplication, (Abutarbush et al., 2015; Annandale et al., 2014; Tasioudi et al., 2016; E Tuppurainen et al., 2017). Animals' different vital organs, particularly their digestive system, genital tract, snout, trachea, nasal mucosa, oral pad, abomasum, larynx, inner lips, testicles, spleen, liver, heart, renal, gingiva, udders, abomasum, uterine tubes, lungs, teats, and, are heavily infected (Al-Salihi & Hassan, 2015; Zeynalova et al., 2016).

While the incubation timespan for this illness is anywhere from 2-5 weeks in the wild, it has been shown to be as little as 7 days to a maximum of 14 days in lab circumstances. There are three clinical manifestations of lumpy skin disease: chronic, acute, and subacute. In the mild type of illness, intermittent fever is first noticed, then 1-2 skin nodules appear. Emaciation, epiphora, and agalactia are the most challenging clinical manifestations to manage. As the condition progresses, the skin may show signs of nodular lesions, erythema, and pruritis. Oro-nasal mucosae, pubic, and perineal lesions develop, causing severe pain (Salib & Osman, 2011). A large number of nodules appear all over the skin in large numbers in the severe form,

which lasts with about a week before they firm quickly and begin to be encapsulated by thin hemorrhagic rings. The nodules may scatter towards other mucosal membranes as they continue to scab over. The dermal layer and musculature are currently attached to these blister configurations, which have an apparent escalating deterioration template and the existence of epithelioid eosinophilic nodules, according to the histology of these lesions (Biswas et al., 2011). During 2 to 3 weeks, these exposed components dry up and harden, creating pain for moving animals. The development of fistulas, also known as "sit fasts," is caused by the ongoing degradation of these sites and the inadequate re-epithelialization that results. It has been observed that fly maggots or screwworm fly larvae often infest these wounds. Suppuration and subsequent chronic diseases further increase the risk of developing serious septic shock and ultimately dying from it (Abutarbush, 2017; Y. R. Khan et al., 2021). In certain situations, lymphadenitis can occur as well. Pneumonia is brought on by the necrosis material that continues to shed into the lungs when these clusters penetrate the nasal tract particularly. In severe circumstances, LSD is known to impair ovulation and result in fetal abortion in cattle. Painful patches around the genitalia in bull may cause infertility (Al-Salihi, 2014; Y. R. Khan et al., 2021).

Diagnosis Methods

Laboratory testing and field presumptive diagnosis are the foundation of diagnosis. Skin lesions with certain characteristics are diagnostic. Visceral nodules are seen in the lungs, musculoskeletal system, gut, uterine tubes, and teats during autopsy exams. Clinical and pathological tests show that the subcutaneous tissue and epidermis have been filled with cells. The area that is affected contains fibroblasts, lymphocytes, macrophages with few plasma cells, and neutrophils. The virus is isolated by allowing it to develop on the Vero kidney cells from an African green monkey and chorioallantoic membrane of chicken embryonated eggs. LSDV causes hemorrhagic pock sores and CAM to thicken. Additional evidence comes from observable cytopathological changes in muscle cell cultures, such as cellular rounding, shrinkage, and empty patches in the cell sheet, although definitive diagnosis needs OIE-recommended laboratory testing (LSDV is being identified by virus isolation, serological tests, real-time PCR or PCR). The presently used typical test for LSDV detection is the virus neutralization assay (Manual, 2010; Saiyad et al., 2022).

Laboratory diagnosis of LSDV is often performed using genomic techniques such LSDV specific real-time PCR 10 and Capri poxvirus generic gel-based PCR and immunological testing including enzyme-linked immunosorbent assay (ELISA) and virus neutralization test (VNT) (Sudhakar et al., 2020). Real-time PCR and conventional PCR are used to distinguish between LSDV vaccine isolates and wild-type pathogens.

Differential Diagnosis

Differential diagnoses of LSD in cattle are required. Clinical samples must be examined for the following diseases in cattle: bovine popular stomatitis virus (BPSV) causes bovine popular stomatitis, cowpox virus (CPXV) causes cowpox, pseudo-cowpox virus (PCPV) causes pseudo-cowpox, bovine pox virus (BPXV) causes buffalo pox, and bovine herpes virus type 2 causes pseudo-lumps on the injury (BHV-2) (Sudhakar et al., 2020). The response is confined in the epithelium, the lesions are delicate with a depressed apex, and the pseudo-LSD or Alleptron herpes virus infection is moderate. In addition to these disorders, parasitic, bacterial, fungal, and

inflammatory responses may also result in nodules that are often challenging to distinguish, especially in rare instances.

There have been notable LSD field epidemics in China, Hong Kong, Vietnam, Russia, and Turkiye due to the advent of vaccine-like recombinant LSDVs, which has crucial implications for LSD laboratory diagnosis, such as EEV-based DIVA tests and GPCR (Tuppurainen et al., 2020).

Treatment and Control

According to the OIE, immunization is the sole method of reducing LSD emergence and spread in endemic nations (Lu et al., 2021). Removing sick cattle out from the flock and administering supportive care such as anti-inflammatory medicines, vitamin injections, and antibiotics is standard practice. These treatments often lower the likelihood of pyrexia, additional infectious diseases, and inflammation, which increases the animal's appetite.

The cattle business is on the brink of collapse as a result of the recent breakout of illness in Pakistan. Here are some suggestions for preventing LSDV disorders in Pakistan. Along with removing diseased animals, outlawing the trade in live animals, and imposing travel restrictions, vaccination is the greatest method for controlling the spread of the virus in afflicted areas. NSAIDs may aid in avoiding secondary infections in addition to antibiotics. Testing for LSDV is required on bulls used for breeding. The disease's consequences may be lessened with the help of skilled nursing.

Economic losses of LSD

LSD is a new, very hazardous illness that is rapidly spreading in many nations, including Pakistan. In many wealthy and developing nations, LSD has resulted in significant economic losses. In neighboring regions (Pakistan, Turkiye, Ethiopia, etc.), strong fever and mastitis conditions caused abortion, a 10-85% milk reduction, a decline in growth and progress, compromised hides, a significant drop in infertility, the death of susceptible hosts, disordered eating behaviors, increased vaccination prices, and a decline in the quality and quantity of epithelium and hides that are unbearable including both large and small farmers (Jamil et al., 2022; Sajid et al., 2012).

The integrity of LSDV

Although LSDV can withstand extreme heat and drought, it cannot survive direct sunlight. According to reports, the virus takes 30 minutes to become inactive at 65 °C or when it comes into direct contact with lipophilic detergents, compared to 2 hours at 55 °C. There are several disinfectants that are very efficient against LSDV, including formalin, phenol, quaternary ammonium compounds, sodium hypochlorite, ether, chloroform, and iodine compounds (Alkhamis & VanderWaal, 2016; Mulatu & Feyisa, 2018a).

LSD preventive and therapeutic approaches

It is possible to employ a "homologous" strain of the LSDV vaccination that is live attenuated, such as the "Neethling strain." It is possible to employ a live attenuated vaccine strain of the "heterologous" goat pox or sheep pox virus (Manual, 2010). There should be a mass awareness campaign concerning the symptoms and clinical signs of LSD use and the productivity losses it causes. When unusual occurrences are found, they should be reported as quickly as possible to the veterinary authorities. It

is not advisable to bring animals suspected of having febrile nodular skin disease to farms. There should be an effort made to reduce the number of vectors in the affected areas. Unaffected animals can avoid infection by using mosquito repellent to eliminate mechanical transmission of LSD.

Due to the growing trade in various animal products and live animals, poverty in rural communities, a lack of diagnostic tools, and restricted availability of effective vaccinations in endemic areas, and other factors, LSDV is rapidly spreading across Pakistan. By implementing various control strategies, vector-borne illnesses may be managed. LSDV may be controlled with antibiotic medication. (Salib & Osman, 2011) on experiment bases LSDV can be controlled, they found that a weird mix of supportive treatment, antibacterial agents, anti-inflammatory drugs, and antiseptic treatments had positive outcomes. Within two to three weeks, mastitis, myiasis, pneumonia, diarrhea, and lameness may all improve (Salib & Osman, 2011).

Because LSDV therapy is highly expensive and not available to every farmer in the nation, adequate preventative measures should be taken to effectively manage the significant economic losses of milk reduction and skin damage, and hide the loss. In a different research, (Gari et al., 2010) found that immunization may lessen the loss of lactation and animal products owing to myiasis, fever, mortality, and miscarriage (Jamil et al., 2022).

Health And Economic Impact

LSD has a direct or indirect socioeconomic impact that has been reported by a number of important industries and sectors. In South Asia, where 21% of the world's dairy farm animals reside, the initial and most noticeable effect of LSD is the substantial decrease in milk production (Das et al., 2021). A Turkish analysis found that per lactation, an affected cow's average milk production decreased by 159L (Chihota et al., 2001). Despite the likelihood of secondary bacterial infection, meat from LSD-infected animals is still allowed to enter the food chain. Due to LSDV infection, it was observed that indigenous breeds and Friesian cattle in Ethiopia produced less meat annually by an estimated 1.2% and 6.2%, respectively (Radostits et al., 2007). Additionally, any scars, breaches, or lesions in the raw skin or hide of cattle may reduce the quality of leather, as was the case with animal skins that had been significantly impacted by LSD (Neamat-Allah, 2015). In 56% of cases, Bangladeshi leather is produced from cows, which is well acknowledged for its higher quality (Siddiky, 2017), accounting for 3.5% of the country's yearly exports (Şevik & Doğan, 2017). India is the tenth-largest exporter of leather products in the world, bringing in \$8.500 billion yearly (Gari et al., 2011). Animals cannot be effectively used for draught because of pyrexia and lameness. Artificial insemination with diseased bull semen can spread LSD to breeding cattle, lowering the pregnancy rate (Das et al., 2021; E Tuppurainen et al., 2017).

The expenditures associated with trade restrictions, vaccination, quarantine and therapeutic interventions, feed, and labor, stamping out, maintaining farm biosecurity, and other factors are included in the indirect economic effect of LSD. Farm owners must pay an extra price for supplemental feed for diseased animals during their recovery time in addition to the extended period for fattening (Green, 1959). The cost of treating the infected cattle with broad-spectrum antibiotics and anti-inflammatory drugs as part of the LSD treatment in Jordan was calculated at US\$ 35.04 (Paul et al., 2013). Sometimes it is necessary to eradicate a large population of diseased animals, as was the case in Greece (Hong, 2018) and Bulgaria, which experienced the worst

economic disaster with losses of almost US\$ 8000 per herd (Dwivedi et al., 2019). The probability of LSD spreading quickly across the production and marketing routes is significant since it is a transboundary infectious disease (Tadesse Degu & Fesseha, 2020). Considering culling rates and the number of bulls in danger, a risk assessment study for LSD conducted on an Ethiopian bull market estimates a financial loss of US\$6,67,785.6 (Abutarbush et al., 2016). It is not always logical to implement quarantine in a peripheral farming program in order to save money. Estimated quarantine costs in the USA were \$145,000 (2010 US dollars) (Agianniotaki et al., 2017), which included personnel costs, cost of food, diagnostic tests costs, costs associated with throwing out test positives, and miscellaneous costs. Israel spent about \$750,000 to contain the initial LSD outbreak by slaughtering all suspicious animals nearby and carrying out the ring vaccination (Casal et al., 2018; Das et al., 2021; Rossiter & Al Hammadi, 2009).

Future Recommendations

The disease has become a major concern to small-scale farmers. The disease was prevalent in greater Africa until the nineteenth century, then spread to the Russian Federation, Eastern Europe, the Middle East, and, more recently, Asia (Das et al., 2021; Saltykov et al., 2022). The scientific community has taken notice of LSD's repeated attacks on sensitive regions. It is to be understood that now is the crucial moment to plan for emergencies in order to prevent this transboundary disease from spreading abundantly. The focus should be placed on vector control, movement restriction, strict quarantine, advanced vaccination campaigns, appropriate veterinary treatment, and general management of farm sanitation facilities to prevent the invasion and spread of the disease (Das et al., 2021).

Scientific Ethics Declaration

The authors say that they are responsible for the scientific, moral, and legal aspects of this chapter, which is published in Current Studies in Health and Life Sciences 2023.

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