

Review Article

Epidemiology and diagnostic methods of lumpy skin disease: A Short Review

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Abstract

Lumpy skin disease (LSD) is a severe viral disease that is having an impact on the cattle industry. The disease is now widespread in the majority of African countries, and it has lately expanded beyond the continent into the Middle East area. The disease's symptoms include an initial period of fever, followed by swollen lymph nodes, circumscribed firm skin nodules, and ulcerative lesions. It occurs in all agroclimatic situations, although it is more common in low-lying areas and beside watercourses. It is transmitted by insect vectors among cattle that share comparable pasture and watering sites and gather in the same barn. In this article, the lumpy skin disease virus, its epidemiology, and diagnostic methods are reviewed.

Introduction

Lumpy skin disease is a devastating viral disease that affects cattle and Asian water buffalo and is caused by the lumpy skin disease virus (LSDV). According to the OIE, it is one of the most economically significant viral diseases and is classified as a disease of international concern. The disease is on the OIE's list of notifiable terrestrial animal diseases because of its great economic importance [1-3]. It causes significant economic losses by decreasing milk production, emaciation, and undergrowth in infected animals, irreparable damage to hides, abortion, infertility, and secondary bacterial infections, which can sometimes result in death [2].

LSDV is a member of the *Poxviridae* family's *Chordopoxvirinae* subfamily and the genus *Capripoxvirus* (CaPVs). The *Poxviridae* family is distinguished by its large and complex genome, which is made up of a single, linear molecule of ds DNA that codes for about 200 proteins and is divided into two subfamilies: *Chordopoxvirinae*, which is responsible for vertebrate *poxviruses*, and *Entomopoxvirinae*, which is responsible for insect *poxviruses*. The genus *Capripoxvirus* includes viruses including lumpy skin disease virus (LSDV) and sheep and goat *poxviruses* (SPPV and GTPV) [3,4].

LSDV can only complete its replication cycle in ruminant hosts due to its narrow host range. The disease mostly affects cattle of all ages, both sexes, and breeds, with lactating and pregnant cows being more severely affected [5,6]. However, research suggests that young animals are more vulnerable to the severe form of the disease; *Bos Indicus* is less susceptible to clinical disease than *Bos Taurus*, and Asian water buffaloes have also been shown to be susceptible. Despite the fact that LSD has not been detected in goats or sheep, skin lesions have developed in sheep, goats, giraffes, impalas, and Grant's gazelles housed in proximity to sick cattle [7]. LSDV outbreaks in previously disease-free areas are often linked to the entry of cattle from the affected zone, as well as high temperatures and humidity [8,9].

There has been little research on the diagnostic procedures and epidemiological aspects of LSD, and there is a lack of public knowledge about the disease's relevance. A detailed investigation of the epidemiological characteristics of LSD and its diagnostic procedures may aid in disease control and prevention [10].

Literature review

History of lumpy skin disease: The first clinical

manifestations of LSD were discovered in 1929 in Zambia (now Northern Rhodesia) in the form of skin nodules. It was thought to be either plant poisoning or an allergic reaction to an insect bite at the time [1,2]. Another epidemic of the disease occurred in Botswana in October 1943, and it was named “Ngamiland cattle disease” since it initially appeared in Ngamiland. There was evidence during this period that the disease was caused by an infectious agent [3].

Between 1943 and 1945, the disease was spread to other African countries, including Zimbabwe (Southern Rhodesia) and the Republic of South Africa, where evidence of infectious agent transmission was identified by inoculation of cattle with the suspension of the skin nodules and given the name “Lumpy Skin Disease [4].” The disease spread as a panzootic in South Africa, affecting eight million cattle. During the following decades, LSD progressively spread northwards, and it is now found across the entire African continent except Morocco, Libya, and Algeria [2,3].

In East Africa, LSD was discovered in 1957, 1972, 1974, and 1983 in Kenya, Sudan, West Africa, and Somalia, respectively. Between 1981 and 1983, the disease was seen in Ethiopia’s North Western, Western, and Central regions, with high levels of morbidity and fatality. LSD outbreak was first reported in Egypt in May 1988 [2]. LSD outbreaks have been recorded in Oman in 1984 and 2009. Kuwait reported LSD invasions in 1986 and 1991; Egypt in 1988 and 2006; Israel in 1989 and 2006; Bahrain in 1993 and 2002–2003; Yemen, and the United Arab Emirates in 2000 [5]. In 2013, Turkey reported the first confirmed case of LSD in Europe [11].

Lumpy skin disease virus

Except for dogs, the Poxviridae family comprises the largest viruses capable of spontaneously causing disease in most domestic animals. Chordopoxvirinae, the poxviruses of vertebrates, and Entomopoxvirinae, the poxviruses of insects, are the two subfamilies [5]. Lumpy skin disease virus is a member of the Capripoxvirus genus and the Chordopoxvirinae subfamily. The two additional viral species in this genus are the Sheeppox virus and Goatpox virus (Figure 1). There is only one serotype of LSDV, which is the Neethling virus (a prototype strain of LSDV), and it is closely related antigenically to sheep and goat poxvirus [6].

The LSDV virus is a double-stranded DNA virus that measures 300x270x200 nm in size and has a genomic size of approximately 151 kbp with 146 genes. It’s a brick-shaped virus with complicated symmetry [8,13]. Their capsid, or nucleocapsid, is brick or oval-shaped containing the genome and lateral bodies (Figure 2) [6]. *Capripoxviruses* (CaPVs) are phylogenetically distinct, but they share a high nucleotide sequence identity. Based on the P32 genomic sequence, their phylogenetic analysis showed that members of the genus could be divided into three different clusters: GTPV, SPPV, and LSDV. At the 55th position of P32, sheep poxvirus contains an extra aspartic acid that is absent in GTP and LSD viruses [14].

Lumpy skin disease virus (LSDV) is genetically related to SPPV and GTPV [10]. The genome is conserved and shares

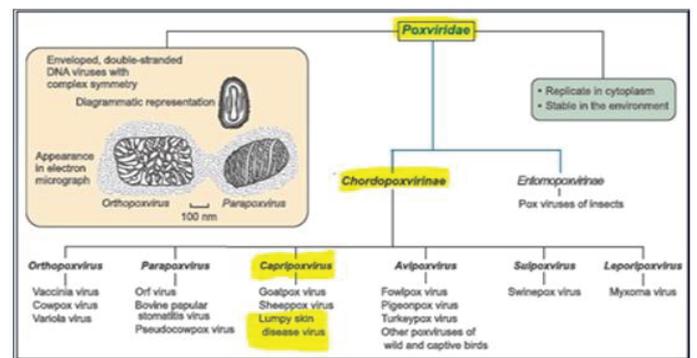


Figure 1: Poxviridae diagrammatic presentation [12].

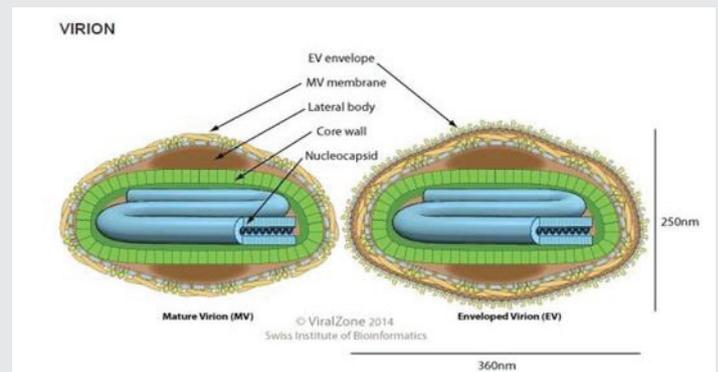


Figure 2: Capri pox virus structure [16].

97% of its sequence with viruses from goat pox and sheep pox. Serologic cross-reaction and cross-protection among members account for significant DNA cross-hybridization between species. Cross interactions between poxvirus species are well known [3].

The virus that causes lumpy skin disease is very persistent at ambient temperatures, especially in dried scabs. It can survive in necrotic skin nodules for up to 33 days or longer, in dehydrated crusts for up to 35 days, and in air-dried hides for at least 18 days. Sensitivity to heat differs among strains [16] Figure 3.

Epidemiology of lumpy skin disease

Occurrence of the disease: LSD is prevalent in most African countries, notably in the Sub-Saharan area [5,17]. It extended to South-East Europe, the Balkans, the Caucasus, Russia, and Kazakhstan after 2012 [7]. Field outbreaks may be severe and widespread infections with high rates of morbidity and mortality, while others may have few affected animals and few or no reported deaths. But in general, outbreaks are more severe when the infection is first introduced to a region and then decreased, most likely due to the development of widespread immunity. Morbidity rates during epizootics approach 80%, but are closer to 20% in endemic areas [9].

Hosts and susceptibility

LSDV is highly host-specific (domestic cattle and water buffaloes) with the exception that some strains may replicate

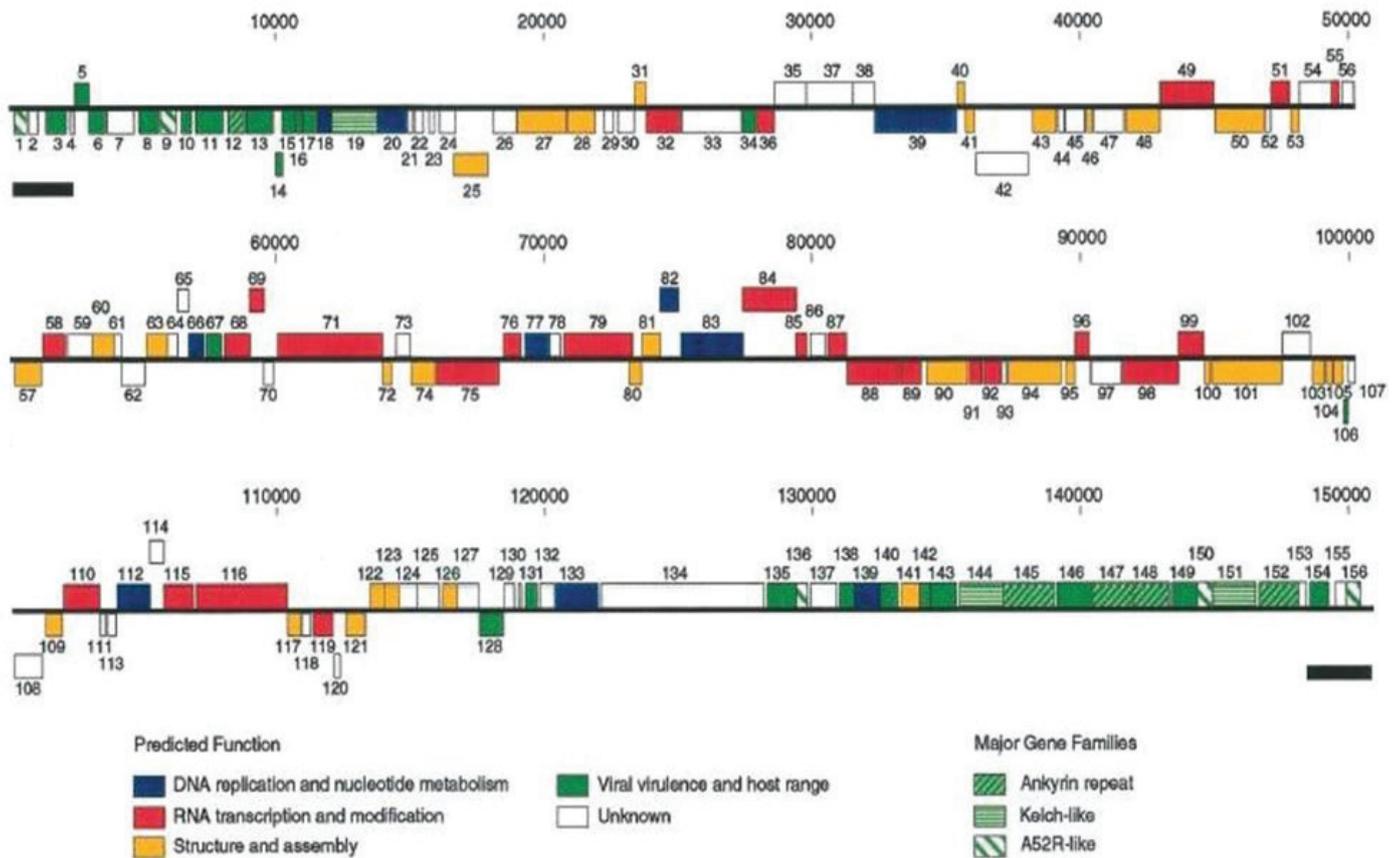


Figure 3: Linear map of the LSDV genome [13].

in sheep and goats. However, no epidemiological investigations have found small ruminants to be viral reservoirs as of yet [5,11]. Domestic cattle and Asian water buffalo are the most vulnerable animals during LSD outbreaks in the field [5,18]. Some wild animals, such as the giraffe (*Giraffe Camelopardalis*) and the impala (*Aepyceros melampus*), are also vulnerable to experimental infection, but their role is unknown [19]. Rather than the virus's virulence, the host animal's susceptibility is determined by its immunological state, age, and breed [20].

Sources of the virus

Nodules that occur on the mucous membranes of the eyes, nose, mouth, rectum, udder, and genitalia also ulcerate and release enough viruses which can serve as sources of the virus. Approximately half of the infected animals may develop clinical signs; the majority of experimentally infected animals will become viremic and a source of the virus. LSD virus was found in saliva for 11 days, semen for 22 days, and skin nodules for 33 days in experimentally infected cattle, but not in urine or faces [2]. Because *Capripoxviruses* are very resistant to physical and chemical conditions, they may survive in lesions or scabs for extended periods of time and have a great affinity for dermal tissues [21].

Transmission

In most of Sub-Saharan Africa, LSD has been seen to occur after seasonal rains, when the number of certain arthropod

species increases [21]. The study that looked at the risk variables involved with the development of LSD in Ethiopia discovered that a warm and humid agro-climate, which supports an abundance of vector population, was linked to a higher incidence of LSD [22]. LSDV can be mechanically transmitted by a number of hematophagous arthropod vectors, according to evidence from several sources. The disease is high, with 50-60% attack rates where mosquito populations are abundant and low, 5-15% morbidity in arid areas where there are fewer potential mechanical vectors [2,23].

Mechanical transmission of some poxvirus species by insect vectors such as *Stomoxys calcitrans* may occur due to high viral loads in skin lesions [24]. Invasive blood-feeding arthropods, such as mosquitoes and sand flies, are suspected to be associated with LSD outbreaks characterized by generalized lesions [25]. *Stomoxys calcitrans* and *Biomyia fasciata* were caught after being fed on sick cows, and the LSD virus was isolated from them [26]. Chihota et al found that *Aedes aegypti* female mosquitos can mechanically transmit LSDV from infected cattle to susceptible cattle [27]. Such a vector feeding regularly and changing hosts between feedings is likely to transmit LSDV mechanically [26]. Chihota et al identified the LSDV genome in mosquitoes (*Anopheles stephensis* and *Culex quinquefasciatus*) and biting midges (*Culicoides nubeculosus*) feeding on LSD-positive animals, but did not observe LSDV transmission by these insects.



Direct and indirect contact might be minor sources of infection (e.g., through infective saliva contaminated feed and water). Poxviruses are extremely resistant and can survive in infected tissue for more than 120 days, or longer. The virus is also identified in blood, nasal discharge, lachrymal secretion, semen, and saliva, which are thought to be the primary routes of LSD transmission [28]. Because the LSD virus can survive for long periods of time in both milk and semen, other potential transmission vectors include nursing cow milk and infected bull semen [29].

Clinical signs and pathogenesis

Large skin nodules covering all regions of the body, fever, swollen lymph nodes, lack of appetite, decrease in milk production, depression and unwillingness to move, nasal discharge, and lachrymation are all symptoms of the disease. In the field, it has a 2 to 4-week incubation period [2]. The nodules that grow on the skin range in size from 2 to 7 cm in diameter, appearing as well-circumscribed regions of erect hair, round, firm, and slightly raised from the surrounding skin, and they are especially noticeable in short-haired animals [30].

During the acute stage of skin lesions, histopathological changes such as vasculitis and lymphangitis with subsequent thrombosis and infarction, which result in edema and necrosis are seen. Serum may have leaked at first, followed by a distinctive inverted greyish pink conical zone of necrosis from LSD skin nodules. Congestion, hemorrhages, and edema are present in adjacent tissue. Secondary bacterial infections are prevalent in necrotic cores, as are enlarged lymph nodes [2].

Diagnosis of lumpy skin disease

LSD is frequently diagnosed in the field based on the disease's typical clinical characteristics. LSD should be considered clinically when there are distinctive skin nodules, fever, and enlargement of superficial lymph nodes. Thus, the differential diagnosis of LSD is mainly based on distinctive clinical indications. Milder and subclinical forms, on the other hand, need fast and dependable laboratory testing to confirm the diagnosis [31]. Detecting viral DNA using conventional or real-time polymerase chain reaction (PCR) is the most often utilized way of diagnosing LSD. Other different molecular assays are also favored diagnostic methods or serology-based diagnostic tests that identify antibodies to the LSD virus [3,7,11].

Virus isolation

Virus isolation is critical in the confirmation of clinical disease and determination of the isolate. This is the method used in the samples to test the virus's viability [11]. To propagate LSDV, a number of primary cells or cell lines of bovine, ovine, or caprine origin are utilized. The virus may also grow on the chorioallantoic membrane of embryonated chicken eggs and African green monkey kidney (Vero) cells [7]. It grows slowly in cell cultures, and the cytopathic effect (CPE) is generally detectable five to seven days after inoculation [16,32]. LSDV induces a specific cytopathic effect (CPE) and intracytoplasmic inclusion bodies in cell culture,

which differs from infection with Bovine herpesvirus 2, which causes pseudo-lumpy skin disease and causes syncytia and intranuclear inclusion bodies in cell culture [3,7].

Molecular detection methods

Molecular diagnostic testing is critical for monitoring the spread of these viruses and controlling disease outbreaks. LSD virus confirmation in the laboratory may be done quickly utilizing a *Capripoxvirus*-specific PCR approach or by demonstrating characteristic *Capripoxvirions* in biopsy material or dried crusts using transmission electron microscopy (TEM). The genome has been detected utilizing *Capripoxvirus*-specific primers for the attachment protein and fusion protein genes, and multiple conventional and real-time PCR technologies have been developed for use on blood, tissue, and sperm materials [3,14,33].

For *Capripoxvirus*, the real-time PCR approach using primers and a probe was verified [2,34]. Molecular assays employing loop-mediated isothermal amplification to identify capripoxvirus genomes have been shown to have sensitivity and specificity comparable to real-time PCR, with a simpler approach and a cheaper cost [35,36].

Serological tests

Serological assays for LSDV include the indirect fluorescent antibody test (IFAT), viral neutralization, enzyme-linked immunosorbent assays (ELISA), and immunological blotting (Western blotting) [33]. The only serologically approved test available is the Virus neutralization test (VNT). Neutralizing antibodies occur 3-4 days after the onset of clinical symptoms and reach maximal titer levels in 2-3 weeks. The agar gel immune diffusion test (AGID) and IFAT are less specific than VNT due to cross-reactivity with antibodies to other poxviruses. Western blotting is sensitive and specific, but it is difficult and expensive to perform. Some ELISAs for antibody detection have been identified, but none have been verified sufficiently to advise for use [7,21].

Differential diagnosis

The main differential diagnosis is pseudo-LSD induced by bovine herpesvirus 2 (BoHV2). Pseudo-lumpy skin disease (caused by herpes virus-2) cutaneous lesions involve only the epidermis and produce a scab after sloughing; systemic symptoms do not occur. This is usually a milder clinical disease with superficial nodules that resemble only the early stages of LSD. Histopathological features of BoHV-2 infection that are not found in LSD include intra-nuclear inclusion bodies and viral syncytia [7,9].

Other differential diagnoses include photosensitization, dermatophilosis, dermatophytosis, bovine farcy, actinobacillosis, actinomycosis, urticaria, insect bites, nocardiasis, besnoitiosis, demodicosis, onchocerciasis, cowpox, and pseudo-cowpox (for integumentary lesions). Bluetongue, foot and mouth illness, malignant catarrhal fever, bovine viral diarrhea, bovine popular stomatitis, and infectious bovine rhinotracheitis are all possible diagnoses for mucosal lesions [14,37].



Economic importance

Capripoxviruses are growing as a global threat to sheep, goats, and cattle [21]. Lumpy skin disease causes significant economic losses due to decreased feed intake, milk production, weight conversion, abortion and infertility, damaged hides, temporary or permanent infertility in males and females, mastitis, and mortality rates of up to 40%, even as mortality rates rarely exceed 3%. Furthermore, the disease is a major notifiable disease that impedes international trade [2,12,21,38].

The disease's economic impact was mostly owing to its high morbidity rate rather than its fatality rate [38]. As a result, the financial impact of these losses on herd owners, consumers, and industries that produce animal goods and byproducts is significant [22,30].

Control and prevention

LSD treatment is only symptomatic, with antimicrobial therapy used to prevent subsequent bacterial infections [39]. Because movement restrictions and the removal of affected animals are typically ineffective, vaccination is the only practical and economically viable strategy for controlling the spread of the disease and improving cattle productivity in endemic areas [7,11,33]. Vaccinating animals every year might keep LSD under control [40].

Inactivated vaccines are less effective, so several live attenuated vaccines have been developed and used across the world. These vaccines are inexpensive and give enough

protection provided sufficient herd immunity (above 80%) is maintained by yearly immunizations [41]. Four live attenuated CaPV strains have been employed as vaccines for the control of LSD in endemic regions, helping to reduce losses from lumpy skin disease [13,14]. These are: a strain of the Kenyan sheep and goat pox virus; the Yugoslavian RM 65 sheep pox strain; the Romanian sheep pox strain; and a lumpy skin disease virus strain from South Africa [5].

Animals that have recovered from infection with any of the Capripoxvirus strains studied so far, whether bovine, ovine, or caprine, share a major neutralizing site and are resistant to infection with any other strain. Immunity against poxviruses is both humoral and cell-mediated [42]. Cattle can be protected against LSD by employing Capripoxvirus strains originating from sheep or goats, such as the Romanian sheep pox strain utilized in Egypt. 14. Strict quarantines and the avoidance of the introduction of infected animals into healthy herds, isolation, and prohibition of animal movements, slaughtering of all sick and infected animals (depopulation of infected and exposed animals), proper disposal of carcasses (incineration), cleaning and disinfection of the premises, and insect control can all help to control an outbreak [2,36] Table 1.

Conclusion and recommendation

Lumpy skin disease is one of the most economically significant transboundary, viral diseases of domestic cattle. It is economically significant in animals because of chronic debility, decreased milk production and weight, damaged skins, abortion, and mortality [2]. LSD is currently present in

Table 1: Most commonly used vaccines registered for use in cattle against lumpy skin disease (LSD) [42].

Manufacturer	Product Name and Virus Strain	Target Species	Titre, Dose, Administration	Presentation Doses/Vial
Onderstepoort Biological Products (OBP) South Africa	Lumpy Skin Disease Vaccine for Cattle (LSD Neethling strain)	Cattle	Not known 2 ml SC	25/50
Intervet (Pty) South Africa/MSD Animal Health	Lumpyvax™ (LSD SIS Neethling type strain)	Cattle	10 ^{4.0} TCID ₅₀ /dose 1 ml SC	20/100
MCI Santé Animale Morocco	Bovivax-LSD™ (LSD Neethling strain)	Cattle	10 ^{3.5} TCID ₅₀ /dose 2 ml SC	25/50/100
Jordan Bio-Industries Center (JOVAC) Jordan	LumpyShield-N™ (LSD Neethling strain)	Cattle	10 ^{4.0} TCID ₅₀ /dose 1 ml SC	5/10/25/50/100
Middle East for Vaccines (MEVAC) Egypt	MEVAC LSD (LSD Neethling strain)	Cattle	10 ^{3.5} TCID ₅₀ /dose 1 ml SC	10/25/50
National Veterinary Institute (NVI) Ethiopia	Lumpy Skin Disease Vaccine (LSD Neethling strain)	Cattle	10 ^{3.0} TCID ₅₀ /dose 1 ml SC	5/20/100
Kenya Veterinary Vaccines Production Institute	Lumpivax™ (Live attenuated LSDV)	Cattle	TCID ₅₀ not known 2 ml SC	50/100/150
Pendik Veterinary Control Institute/ Ministry of Agriculture, Turkey	Penpox-M™ Live SPPV (Bakirköy SPPV strain)	Cattle	10 ^{2.5} TCID ₅₀ /dose 3 ml SC	
Vetal Company Turkey	Poxvac™ (Bakirköy SPPV strain) Lumpyvax™ (LSD Neethling strain)	Sheep Cattle Cattle	10 ^{2.5} TCID ₅₀ /dose 3ml SC 10 ^{3.5} TCID ₅₀ /dose 2 ml SC	20/50/100/200 10/25/50/100
Dollvet Turkey	Poxdoll™ (Bakirköy SPPV strain) LSD-NDOLL (LSD Neethling strain)	Cattle Sheep Goats Cattle	10 ^{2.5} TCID ₅₀ /dose 3ml SC 10 ^{3.5} TCID ₅₀ /dose 3ml SC	50/100 10/25/50/100
FGBI-Federal Centre for Animal Health, The Russian Federation	Sheep Pox Cultyral Dry™(Arriah SPPV strain)	Sheep Cattle	Not known	50/100



the majority of African and Middle Eastern countries. LSD is often diagnosed based on specific clinical signs and differential diagnoses. Milder and subclinical forms, on the other hand, require quick and accurate laboratory testing to prove the diagnosis [31]. The disease's economic impact was mostly due to its high morbidity rate rather than its mortality rate [38].

Based upon the above conclusion, the following recommendations are forwarded:

- a. The disease's global expansion requires special attention.
- b. Action plans for effective control and prevention should be developed to reduce the disease's economic losses.
- c. If LSD is introduced into a disease-free country, rapid identification and culling of infected herds, as well as ring vaccination, should be undertaken.
- d. Additional research into control strategies is required.

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